The relationship between childhood trauma and schizotypy and the pathways underlying this association

Tjasa Velikonja

Submitted for the award of PhD in
Mental Health Sciences

University College London

June 2014

'I, Tjasa Velikonja confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.'

Abstract

There is a growing body of literature demonstrating an association between childhood trauma and schizotypy (e.g. Afifi et al. 2011;Myin-Germeys et al. 2011). However, more research is required to build on methodological limitations of previous studies, explore the possible differential effects of specific trauma types and expand the focus from a single contributor (e.g. psychological, biological) by considering the additive/interactive contributors to schizotypal symptomatology. The aim of the thesis was to explore the relationship between a range of childhood traumatic experiences and schizotypy whilst also incorporating several social, psychological and genetic factors underlying these relationships. Participants were recruited as a part of a cross-sectional case-control study conducted in the London Boroughs of Lambeth and Southwark. The thesis covers a subsample of controls (N=212), healthy volunteers, aged 18-64 and residents in the same geographical area. Data were gathered using an in-depth standardised interview regarding childhood abuse (Childhood Experience of Care and Abuse - CECA) and The Structured Interview for Schizotypy – Revised (SIS-R) measuring a range of schizotypal symptoms and signs. The study found a linear association of total trauma and schizotypy *(adj. β=.88, p=0.004)*, with the strongest associations observed for psychological *(adj. OR=4.85, p=0.039)* and physical abuse *(adj. OR=3.56, p=0.003)*. These particular types of trauma had an especially robust effect on positive schizotypal traits *(psychological: adj. OR=3.79, p=0.013; physical abuse: adj. OR=2.32, p=0.042)*, which are attenuated forms of positive symptoms of schizophrenia (e.g. hallucinations, delusions). Negative beliefs about self/others and depression were the main mediators of these associations. A strong relationship was found for genetic risk of psychosis and increased schizotypy *(adj. β=3.41, p=0.015)*. Other moderators of the childhood trauma - schizotypy association were intrusive life events (*adj. β=4.20, p=0.045)*. This study provides further insights into the association between childhood trauma and schizotypy and gives clues to pathways underlying this association.

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Acknowledgments

Introduction

As psychotic disorders, such as schizophrenia, place a major burden on the individual, family and society, it is important to find ways to identify as early as possible who is at risk for such disorders. One way to do this would be to develop a better understanding of the aetiology of subclinical manifestations of psychosis such as schizotypy. Therefore, the importance of schizotypy research lies in the potential to identify the fundamental features of liability to psychosis and could have substantial implications for prevention, clinical assessment and treatment formulation.

Literature suggests that the psychosis phenotype is expressed at subclinical levels (e.g. van Os et al. 2009). The term 'psychosis continuum' denotes the gradual transition from subclinical attenuated psychotic experiences or schizotypal traits to clinically relevant psychotic disorders as opposed to a sharp categorical division between disorder 'absent' or 'present'. This proposition is based on the pioneering work of Rado (1960) followed by Meehl's (1962) conceptualisation of schizotypy. According to their views, schizotypy represents a fundamental liability to schizophrenia and underlies a range of clinical manifestations ranging between healthy variation and severe mental illness.

The multidimensionality of the schizotypy construct resembles schizophrenia symptomatology (e.g. Lenzenweger et al. 1991) and can be clustered as three dimensions: a positive cluster (e.g. unusual perceptions, magical thinking, ideas of reference), a negative cluster (e.g. restricted affect, social isolation) and a disorganized cluster (e.g. odd behaviour, odd speech) (Claridge et al. 1996;Vollema and Vandenbosch 1995). Similarly, the 'schizophrenia prodrome' (period preceding the diagnosis of schizophrenia) also reflects attenuated schizophrenia symptoms, making these constructs not easily distinguishable (Bedwell and Donnelly 2005).

Nevertheless, the prodromal construct is defined as a more recent onset and escalation in symptom severity usually associated with psychosocial impairment, but schizotypal personality disorder (and schizotypy) reflects more chronic, stable symptomatology (Miller et al. 2003). However, because of the non-specific nature of the early manifestations of the psychotic disorder, the concept of schizophrenia prodrome was refined by Yung and colleagues (2003) who proposed the three categories of recognizing individuals at 'Ultra-High-Risk' (UHR) of developing psychosis. These sub-categories also included individuals with attenuated psychotic symptoms and those with a family history of a psychotic disorder or schizotypal personality disorder along with a significant deterioration in mental state and/or functioning. Schizotypy, schizophrenia 'prodrome' and 'UHR' groups all lie on the psychosis-spectrum of disorders, suggesting the constructs are not necessarily mutually exclusive and can also co-occur (Woods et al. 2009) *(see section 1.1.1 for full definitions of these constructs).*

Traditionally schizotypy has been considered to imply genetic vulnerability to schizophrenia (Rado 1953) but its expression has been argued to greatly depend on the environment to which the individual is exposed (e.g. Raine et al. 1995). There is now emerging evidence showing an association between childhood trauma and increased schizotypy load (Berenbaum et al. 2003;Johnson et al. 2001;Myin-Germeys et al. 2011;Steel et al. 2009). Similar findings are observed for childhood trauma and psychotic-like experiences (e.g. Janssen et al. 2004;Shevlin et al. 2007b) and psychotic disorders (e.g. Bendall et al. 2008;Morgan and Fisher 2007), consistent with an aetiological continuum underlying psychosis.

However, these reports on the association between childhood trauma and schizotypy have several methodological limitations and do not permit firm conclusions to be drawn *(as shown in Chapter 2).* Specifically, the studies utilised crude assessments of trauma with no consideration of contextual factors, did not

assess the multidimensionality of schizotypal personality and did not explore the interaction between genes and environmental factors. There is a need to build on these limitations by using the highest quality level of assessments, including the measurement of different trauma types and considering the contextual factors of traumatic experiences (e.g. trauma severity, frequency, age of occurrence). Moreover, schizotypy and schizophrenia have been postulated to be aetiologically heterogeneous (e.g. Tsuang et al. 2001) and pathways to schizotypal symptoms might thus be more complex than initially suggested (mainly relying on a single contributor – for example childhood trauma). Therefore, the focus on interaction between genes and environmental factors is not only necessary in understanding the developmental path to schizotypal traits but it may also provide valuable clues to the aetiology of psychotic disorders. Research into the aetiology of schizotypy provides an opportunity to identify the liability to psychosis prior to the development of clinical illness, without the possible interference of factors usually associated with research using clinical samples (e.g., impact of medication, chronicity of illness etc.). Most importantly, this might permit early detection and help with early intervention of individuals considered at 'ultra-high-risk' for developing psychosis. This is particularly likely to be the case as schizotypal traits have been shown to be the most reliable predictor of transition to psychosis amongst UHR individuals (Mason et al. 2004).

The main aim of this thesis is to explore the relationship between different types of childhood trauma and schizotypy. Also, it aims to unravel the underlying mechanisms that account for the childhood trauma - schizotypy association by inclusion of genetic *(e.g. family history of mental disorders)*, social *(e.g. adult adversity)* and psychological factors *(e.g. core negative beliefs about self and others)*. This exploration of the pathways leading to schizotypal traits would also

assist in applying the appropriate clinical interventions to detect and prevent the progress of symptoms.

Brief definition of the terms

Childhood Trauma

Childhood trauma is a broad concept encompassing a range of childhood experiences such as physical, sexual or psychological abuse, physical or emotional neglect, peer bullying, parental loss, parental separation, witnessing domestic violence, amongst others. This area has received a lot of attention since the early 1960s when Kempe and his colleagues (1962) introduced the *"battered child syndrome"*. However, there is still a lack of operational definitions and systematic procedures to assess these traumatic experiences, hindering the true understanding (etiological, developmental etc.) of this complex phenomena on later outcomes (Manly et al. 1994). The American Academy of Paediatrics (AAP) defines childhood trauma as *"a repeated pattern of damaging interactions between parent(s) [or, presumably, other significant adults] and child that becomes typical of the relationship (Kairys and Johnson 2002 p.1)"*. Therefore, trauma stands for an event or more enduring conditions that exceeds one's ability to integrate the emotional experience and can affect physical and mental wellbeing throughout an individual's life (Reviere 1996). Nevertheless, these experiences are not rare. A prospective, longitudinal USA study showed that *68%* of individuals by the age of 16 have experiences at least one type of trauma (Copeland et al. 2007). Overall the prevalence of traumatic experiences in general population studies is between *20%* and *80%* (Fairbank and Fairbank 2009). The prevalence not only depends on the culture-specific understanding and identification of the traumas (Raman and Hodes 2012) but also on a variety of situations in social and cultural context (e.g. poverty,

war), where societal harm compromises children's well-being beyond the control of a parent (Korbin 1991). In addition, the prevalence depends on the disparate criteria of trauma used across the studies (Goldman and Padayachi 2000). Early trauma has been linked to a range of childhood and adult psychopathology (e.g. depression (Bifulco et al. 1991), personality disorders (Grover et al. 2007), post-traumatic stress disorder (PTSD) (Vranceanu et al. 2007), anxiety and other mood disorders (Kessler et al. 1997)) but particularly to psychotic-like symptoms and psychotic disorder (e.g. Morgan and Fisher 2007;Varese et al. 2012b). In order to evaluate the true scope of psychological effects of traumatic experiences, a whole range of factors need to be considered: the severity and frequency of traumatic experiences (Janssen et al. 2004;Kilcommons and Morrison 2005;Read et al. 2005), duration of the trauma (e.g. Thornberry et al. 2001), the age (developmental stage) when trauma first occurred (e.g. Fisher et al. 2010), the type of traumatic exposure (e.g. Johnson et al. 1999) and a combination of traumas experienced by the individual (Ney et al. 1994).

For the purposes of this thesis, the types of childhood trauma focused on were determined by those included in the Childhood Experiences of Care and Abuse (CECA) interview measure (Bifulco et al. 1994) with the addition of Bullying Questionnaire (see Arseneault et al. 2006); the measures used within the larger study this sample was drawn from. The CECA covers four main types of childhood trauma: household discord, physical abuse, psychological abuse and sexual abuse *(see section 3.4.3 for exact definition of trauma types according to CECA measure)*:

- **Household discord.** Household discord refers to a general discord between the parents/carers at home (e.g. overt quarrelling, arguments) or violence between them. It also includes a general tense atmosphere in the household caused by discords after which parents have stopped talking to each other for long periods of time (days or weeks) (Lifespan Research Group 2009).

- **Physical abuse.** Butchart and colleagues (2006) defined physical abuse as intentional use of physical force against the child which includes for example hitting, beating, strangling and other forms of physical violence, potentially causing *"harm for the child's health, survival, development or dignity" (p.10).* Perpetrators are usually limited to parents or other adults living within the same household as the child.

- **Psychological abuse.** Psychological abuse refers to singular incidents or repeated forms of cruelty towards the child or inadequate provision of a developmentally appropriate/supportive environment (Butchart et al. 2006). It can cause persistent adverse effects that are damaging to a child's social, emotional, physical and cognitive development. The perpetrator is usually a parent, carer or a close other who has a power and responsibility over the child. Psychological abuse is characterised by behaviours such as *"humiliating/degrading, terrorizing, extremely rejecting, depriving of basic needs or valued objects, inflicting marked distress/discomfort, corrupting/exploiting, cognitively disorienting, or emotionally blackmailing (Moran et al. 2002 p.220)"* the child. Emotional abuse and psychological abuse are not synonyms but have been often used interchangeably in the literature. Both types of abuse are sometimes hard to be distinguished and consequently the attempts to have clear distinctive definitions prove difficult. O'Hagan (1995) stressed that emotional abuse affects the child's emotional wellbeing and emotional development whereas psychological abuse damages their mental wellbeing and impairs mental development. In this thesis only psychological abuse will be investigated.

- **Sexual abuse.** Sexual abuse is defined as forcing or enticing a child or a young person into sexual activity, to which a child is unable to consent to

and/or is not developmentally prepared for. A perpetrator can be any adult or older child exploiting their power and trust over the victim (Butchart et al. 2006). This includes contact forms of abuse such as penetration (e.g. rape) or non-penetrative acts (e.g. touching) or non-contact forms of abuse (e.g. forced to watch sexual images) (NSPCC 2010).

- **Bullying.** Bullying is defined as behaviour that causes distress or harm and occurs between individuals of the similar age (Olweus 1996a). It is an intentionally hurtful behaviour in physical (e.g. hitting), verbal (e.g. name calling, threats) or emotional forms (e.g. intentional isolation from peer group) (NSPCC 2010) and can have detrimental long-term effects on mental health (Goldstone et al. 2012;Hawker and Boulton 2000;Sourander et al. 2007).

A recent NSPCC[1] survey of child abuse (up to the age of 17) and neglect in the UK estimated the prevalence of domestic violence between adults (defined as physical violence or threatening behaviour from an adult (ex)partner towards the parent) at *12.0%* for children under 11 and *17.5%* for children between 11 and 17 (Radford et al. 2011). Within this nationally representative UK survey, the prevalence of the childhood physical abuse was estimated to be around *1.3%* for children under 11 and *6.9%* for children between 11 and 17*;* and sexual abuse was approximately *1.2%* for under 11s and *16.5%* for 11-17s (Radford et al. 2011) consistent with some international studies (Dinwiddie et al. 2000;Pereda et al. 2009). A USA study using a general population sample reported a prevalence of sexual abuse at *21.6%*, similar to the prevalence of physical abuse (*20.6%* (Edwards et al. 2003)). Bullying has produced more mixed results, with the prevalence ranging between *40%* and *80%* (Juvonen and Graham 2001). In the UK it has been estimated to be around *28.0%*

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[1] *National Society for the Prevention of Cruelty to Children, UK*

(for under 11s) or *59.5%* (11-17s) (Radford et al. 2011). Mainly because of the problem of its definition, the prevalence of psychological abuse is difficult to measure, possibly resulting in an underestimation of this type of trauma (*4%* (Moran et al. 2002)). Moreover, it is also important to stress that different types of abuse are highly co-morbid (Dong et al. 2004), adding to the complexity of their assessment.

Psychosis

Psychosis is a broad term, covering a range of mental disorders, characterized by gross impairment in reality testing (Shahrokh and Hales 2003). The symptoms consist of hallucinations, delusions and disorganised thinking, speech or behaviour (American Psychiatric Association 1994). Psychotic disorders include schizophrenia, schizoaffective disorder, depressive psychosis, manic psychosis, and many others. With the pioneering work of Kraepelin (1919) *"dementia praecox"* or schizophrenia was believed to be a distinctive degenerative disease. However, the boundaries of the disorder are now assumed to be more elusive than initially suggested. The symptoms and signs of psychosis are now proposed to lay on the continuum between normal mental states and disorders such as schizophrenia *(continuum hypothesis, see thesis section 1.1.5)* (Johns et al. 2004;Tien 1991;van Os et al. 2000). As such clinically relevant psychotic disorders might only be a subcategory of the psychosis phenotype (Allardyce et al. 2007). Further support for the continuum hypothesis comes from studies exploring the aetiology of psychosis, as the exposure to the interaction effect of multiple genes and environments can only result in a range of phenotypic expressions, varying between healthy variations and clinical states (van Os et al. 2009).

The latest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatric Association 2013) continues to use the categorical approach to the assessment of psychotic disorders. However, it goes

beyond the previous editions in the way it incorporates the underlying dimensional structure to psychosis (Heckers et al. 2013). Instead of assuming the clear boundaries between the entities (categorical view), the more dimensional approach instead focuses on symptom severity (Barch et al. 2013). Psychotic disorders are defined within eight domains. Besides the five domains forming a part of the 'A'[2] criteria for Schizophrenia (delusions, hallucinations, disorganised speech, disorganised or abnormal motor behaviour and negative symptoms), the other domains include depression, mania and impaired cognition (American Psychiatric Association 2013). The differential diagnoses are often based on the symptom severity within each domain rated on a five-point rating scale (0: not present to 4: present/severe) (Barch et al. 2013). A score of 2 or higher is necessary for fulfilling the 'A' criteria for Schizophrenia. These psychotic symptoms are also associated with social and occupational dysfunction *('B' criteria for Schizophrenia diagnosis) (American Psychiatric Association 2013)*. Also, the duration of the symptoms must be at least 6 months *('C' criteria)* with 1 month of active symptoms (Tandon et al. 2013). This is in contrast to the criteria of schizophrenia found in the International Classification of Diseases (ICD-10) (World Health Organisation 1992) where the main focus is on first-rank Schneiderian symptoms (e.g. thought withdrawal, thought broadcasting, auditory hallucinations) (Schneider 1959). However, the new proposals have been made for the new edition of the ICD (ICD-11), aiming towards more synchronisation with the DSM-5 classification system (Gaebel et al. 2013). The DSM-5 *(American Psychiatric Association 2013)* defines the following disorders under the title *'Schizophrenia Spectrum and Other Psychotic Disorders'*: delusional disorder, brief psychotic disorder, schizophreniform disorder, schizophrenia, schizoaffective disorder, substance/medication-induced psychotic disorder,

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[2] *DSM-5 uses 6-criteria (A-F) for diagnosis of schizophrenia. "Criterion A: Characteristic symptoms: Two (or more) of the following, at least one of these should include 1–3: (1) Delusions (2) Hallucinations (3) Disorganized speech (4) Grossly disorganized or catatonic behavior (5) Negative symptoms, i.e., affective flattening, alogia, or avolition (from DSM-5, Tandon et al. 2013 p.5)".*

psychotic disorder due to another medical condition, catatonia, other specified schizophrenia spectrum and psychotic disorder. Also schizotypal personality disorder is recognised under the umbrella of schizophrenia spectrum disorders, but in the DSM-5 is categorised under *'Personality Disorders'* (Bhati 2013). Psychotic symptoms are not exclusively associated with schizophrenia spectrum disorders but are widely observed in mania (Canuso et al. 2008) and depression (Johnson et al. 1991).

This new approach to psychosis *('deconstructing psychosis')* (Tamminga et al. 2010) originates from neurobiological findings in psychosis research, the criticism over the overlap of different psychotic disorders in traditional Kraepelinian view *(e.g. overlap in risk factor, clinical symptoms (Murray and Dean 2008))* and the high frequency of psychosis-like symptoms observed in the general population (Gaebel and Zielasek 2008). The prevalence of psychotic-like symptoms in the general population ranges between *4%* (Eaton et al. 1991) and *28.4%* (Kendler et al. 1996a) whilst clinically significant psychotic disorders are estimated at *3%* (van Os et al. 2009). The lifetime prevalence of schizophrenia is generally reported between *0.5%* and *1%* (Murray and Dean 2008) but the systematic review of the literature showed that it is probably an overestimation (*0.4%* for lifetime prevalence) (Saha et al. 2005).

Schizotypy and schizotaxia

Phenomenological consistency between schizophrenia and more attenuated forms of psychotic-like symptoms implies that there is an underlying liability to schizophrenia that can manifest itself in different forms (Lenzenweger 2010). Meehl (1962) advocated that 'schizotaxia' essentially described the genetically determined integrative defect, while 'schizotypy' results from schizotaxia and its interaction with

social learning. As such it stands as a latent personality organization, characterised by liability to psychosis. This schizotaxia-schizotypy model *(see thesis section 1.1)* has now taken the fundamental role in research into the aetiology of schizophrenia-spectrum disorders (Lenzenweger et al. 2005). The model suggests that schizotaxic individuals will manifest schizotypy on a continuum ranging between healthy variations and clinical outcomes, depending on genetic and bio-psycho-social factors (Meehl 1962). Schizotypal traits include for example odd beliefs or magical thinking, unusual perceptual experiences, suspiciousness or paranoid ideation, inappropriate or constricted affect and social isolation, resembling schizotypal symptoms outlined in DSM-V (American Psychiatric Association 2013) criteria for Schizotypal Personality Disorder (SPD) *(see section 1.1.1)*. The literature suggests three approaches for identifying schizotypy (Lenzenweger 2006b;Lenzenweger 2010). The first is based on a familial approach and focuses on first degree relatives of individuals with schizophrenia. The second approach assesses schizotypy using clinical instruments (e.g. schizotypal personality disorder in DSM-V) and the third approach assesses liability indicators using valid, reliable quantitative measures (e.g. eye tracking, psychometric inventories).

Considering that schizotypy has its basis in genetic underpinnings, it is very likely that the underlying liability to schizophrenia will manifest itself before the appearance of any psychotic or even prodromal symptoms (Lenzenweger 2010). These detectable biological or neuropsychological vulnerability markers of genetic liability to psychosis are often called 'endophenotypes' (Compton and Harvey 2010). They are found at increased rates in unaffected relatives of individuals with schizophrenia compared to the general population, they are associated with the illness, heritable and state independent (*manifest in an individual irrespective of the presence of the illness)* (Gottesman and Gould 2003).

Yet, the stability of psychotic vulnerability does not have to reflect the sole influence of genetic factors, as exposure to environmental risks may interact with genetic predispositions to increase vulnerability (Ingram and Price 2010). The environmental factors hypothesized as significant influences/risks for the development of schizotypy are early prenatal and postnatal environmental influences affecting brain development (e.g. influenza exposure, prenatal stress), disturbances in early parental bonding/attachment, broader psycho-social adversity (e.g. stress, fewer positive life events) and especially childhood trauma *(the focus of this thesis)* (Raine 2006). Also, the conceptualisation of the psychosis vulnerability as a latent factor suggests that the locus of vulnerability lays within the person (not easily detectable), but it is the presence of a stressful/traumatic event that results in this vulnerability being expressed and thus makes identification of vulnerability markers possible (Ingram and Price 2010). The importance of the combined effect of genetic and environmental factors on susceptibility to the development of the disorder is not new, but more recently this framework was also adopted by research exploring the aetiology of schizophrenia (and other disorders) (Gottesman and Gould 2003).

Outline of the thesis chapters

The thesis consists of seven chapters, as briefly summarised below.

Chapter 1: The first chapter presents the main definitions of the schizotypy and schizotypal personality disorder *(section 1.1.1)* and examines the heterogeneity of schizotypy and psychosis-proneness measures *(section 1.1.3)*. The final section of the chapter presents the evidence for the heritability of schizotypy *(section 1.2)*.

Chapter 2: This chapter explores the empirical literature available on childhood trauma and schizotypy *(section 2.1)* as well as psychotic-like symptoms *(section 2.2)*. The main methodological limitations of previous studies are described *(section 2.1.5)*. This is followed by the exploration of the possible pathways that account for the childhood trauma – schizotypy association *(section 2.3)*.

Chapter 3: This chapter begins with the description of the main objectives and hypotheses of the thesis *(section 3.1)*. The experimental study design *(section 3.2)* is then presented followed by a description of the data collection procedures and all the assessment tools used in the study *(section 3.3 & 3.4)*.

Chapter 4: This chapter describes the socio-demographic characteristics of the recruited sample *(section 4.1)*. The main part of the chapter provides a detailed data analysis procedure for each of the hypotheses *(from section 4.2.1 to 4.2.5)*.

Chapter 5: This chapter presents the findings that help to disentangle the main questions set out with the thesis (from *section 5.1 to 5.5)*.

Chapter 6: This chapter incorporates a comprehensive overview of the thesis' outcomes and their fit with the existing literature *(section 6.1 & 6.2)*, followed by the overview of the conclusions in light of the study's strengths and limitations *(section 6.3)*.

Chapter 7: A brief summary of the main findings of the thesis is provided in Chapter 7 *(section 7.1)*. The clinical implications of the study are outlined *(section 7.2)* along with propositions for future research *(section 7.3)*.

CHAPTER 1
Literature Review:
Schizotypy

Chapter 1 Literature Review - Schizotypy

Main aims of the chapter

- To present the main definitions of schizotypy and schizotypal personality disorder *(section 1.1.1)*;

- To explore the multifactorial nature of the schizotypy concept *(section 1.1.2)*;

- To examine the complexity of schizotypy/psychosis-proneness measures *(section 1.1.3)*;

- To outline the socio-demographic characteristics and schizotypy *(section 1.1.4)*;

- To describe the continuum model of psychosis (and how/where does schizotypy fit in the model) *(section 1.1.5)*;

- To present the evidence of heritability of schizotypy and why environmental factors (childhood trauma) are a necessary component for expression of schizotypal traits *(section 1.2)*.

1.1 *What are schizotypy and schizotypal personality disorder?*

This section begins with the presentation of the definitions of schizotypy and schizotypal personality disorder. It also explores the complexity and heterogeneity of the assessment tools in an attempt to incorporate the multidimensionality of the schizotypy construct. Finally, the continuum model of psychosis is defined and common phenomenological and aetiological underpinnings of schizotypy and schizophrenia are presented.

1.1.1 Definitions of the concepts

Schizotypal personality disorder (SPD) was initially introduced as a specific personality disorder in DSM-III to include the subclinical schizophrenia-like symptoms found in the relatives of patients with schizophrenia (Reider 1979;Spitzer et al. 1979). The latest edition of the Diagnostic and Statistical Manual of Mental Disorders (5th Edition, DSM-V) (American Psychiatric Association 2013) included Schizotypal Personality Disorder (SPD) under *Schizophrenia Spectrum and Other Psychotic Disorders (p.87)* and as such fits better with The World Health Organization's ICD-10 (World Health Organisation 1992) view of the disorder than did its previous edition (American Psychiatric Association 2000). The WHO ICD-10 describes Schizotypal disorder as *"characterized by eccentric behavior and anomalies of thinking and affect which resemble those seen in schizophrenia"* (World Health Organisation 1992 F 21) while DSM-V defines SPD as:

"A. A pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behavior, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Ideas of reference (excluding delusions of reference).

2. Odd beliefs or magical thinking that influences behavior and is inconsistent with subcultural norms (e.g. superstitiousness, belief in clairvoyance, telepathy, or 'sixth sense'; in children and adolescents, bizarre fantasies or preoccupations).

3. Unusual perceptual experiences, including bodily illusions.

4. Odd thinking and speech (e.g. vague, circumstantial, metaphorical, overelaborate, or stereotyped).

5. Suspiciousness or paranoid ideation.

6. Inappropriate or constricted affect.

7. Behavior or appearance that is odd, eccentric, or peculiar.

8. Lack of close friends or confidants, other than first-degree relatives.

9. Excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self.

B. Does not occur exclusively during the course of schizophrenia, a bipolar disorder or depressive disorder with psychotic features, another psychotic disorder, or autism spectrum disorder.

Note: If criteria are met prior to the onset of schizophrenia, add 'premorbid', e.g. 'schizotypal personality disorder (premorbid)." (American Psychiatric Association 2013 p.655-656)

The median prevalence of SPD found in general population studies is 0.9% (Torgersen 2009). In a Norwegian sample it was estimated at 0.6% (Torgersen et al. 2001), 0.7% in a German sample (Maier et al. 1992) and in USA samples it varies between 0.6% (Samuels et al. 2002) and 4.6% (American Psychiatric Association 2013) *(e.g. 0.7% (Moldin et al. 1994), 1.1% (Crawford et al. 2005), 3.3% (Lenzenweger et al. 2007)).* In clinical populations, SPD is estimated to be around 3.9% (American Psychiatric Association 2013). Individuals with SPD do not necessarily transition to schizophrenia, but they still display similar (but quantitatively less severe) neurocognitive, behavioural and social deficits (Berenbaum et al. 2003;Campbell and Morrison 2007;Laurens et al. 2007b;Myin-

Germeys et al. 2003b). Besides fitting in the schizophrenia spectrum disorder category, SPD also mirrors the characteristics of personality disorders (Hummelen et al. 2012). As such, it is observed that individuals with SPD have impairments in work and social relationships (Skodol et al. 2011), affecting the overall quality of an individual's life (Cramer et al. 2006). Studying SPD is important because it can provide a useful insight into prodromal phases of schizophrenia, including its aetiology, neuropathology and treatment (Seeber and Cadenhead 2005).

Schizotypy construct resembles the symptoms outlined in DSM-V criteria for Schizotypal Personality Disorder (SPD) but is occurring at a much lower level of severity. Although literature suggests that SPD is rare, studies have advocated that forty percent of individuals displayed schizotypal traits without meeting the diagnostic criteria for schizotypal personality disorder (e.g. Johns and van Os 2001;Tien et al. 1992). Schizotypal traits can be clustered as three dimensions: a positive cluster (e.g. unusual perceptions, magical thinking, ideas of reference), a negative cluster (e.g. restricted affect, social isolation) and a disorganized cluster (e.g. odd behaviour, odd speech) (Claridge et al. 1996;Vollema and Vandenbosch 1995) *(see section 1.1.2)*. Similar three-cluster model is found in schizophrenia symptomatology (Lenzenweger et al. 1991).

Literature supports two distinct approaches to the assessment of schizotypal personality; the first reflects categorical or clinical conceptualisations, while the other advocates a dimensional or personality perspective (Raine 2006). The dimensional approach to schizotypy, that contrasts (or possibly complements (Raine 2006)) the categorical assessment (echoed in DSM categorization) originated from psychosis-proneness scales (Chapman et al. 1976), with physical and social anhedonia as the core features. Bleuler (1911) was the first to document these schizophrenia-like subclinical symptoms in relatives of people with schizophrenia, followed by Rado (1953), who took the observations further and proposed the concept of 'schizotypy',

suggesting that schizotypal behaviour represents a fundamental liability to schizophrenia. According to his model, the schizotypal personality organization resulted from the mutated genes, causing two main deficiencies: a diminished capacity for pleasure and aberrant awareness of the body (schizotypic body image distortions) (Rado 1953; 1960). Influenced by Rado's theories, Meehl (1962; 1990) advocated an integrated model of 'schizotaxia, schizotypy and schizophrenia'. He proposed that not only genetic factors but also integrated social learning define the schizotypal personality organization (along with the clinical symptomatology) (Lenzenweger 2006a). The term schizotaxia ('taxon') essentially described the genetically determined integrative defect, an aberration in brain functioning (pervasive neuronal slippage[3]), while schizotypy results from schizotaxia and its interaction with social learning and stands as a latent personality organization (not necessary observable) (Meehl 1962). As such, schizotypy can manifest itself in various degrees – from minimal signs and symptoms of schizotypal personality to a diagnosis of schizophrenia, suggesting continuity with the normal population (Claridge 1994). The term 'continuum' *(see section 1.1.5)* therefore implies the gradual transition of conditions as opposed to the existence of two distinguishable categories (disorder 'present' or 'absent') as pioneered by Kraepelin's (1971) fundamental division between sanity and madness.

The term 'schizotypy' is therefore a multidimensional construct (Claridge et al. 1996;Raine et al. 1994;Stefanis et al. 2002;Vollema and Vandenbosch 1995). For some it represents an inherited general vulnerability to psychopathology that falls on a continuum between healthy variation and severe mental illness (Hanssen et al. 2005;Meehl 1962;Rossi and Daneluzzo 2002;Siever et al. 1993;Stefanis et al.

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[3] *According to Meehl (Meehl 1962) schizotaxia does not reflect a simple defect of the synaptic control system in the central nervous system (CNS), but it is the presence of 'slippage' (causing insufficient integration, differentiation of multiple information streams) at the CNS synapse that underlies the experiences of schizotypal individuals across various domains (e.g. perception of self/others) (Lenzenweger 2010).*

2002;van Os et al. 2000;Verdoux and van Os 2002;Vollema et al. 2002;Yung et al. 2003); for others it refers to a range of personality traits putatively placing an individual at greater risk of a psychotic disorder, primarily but not exclusively schizophrenia (e.g.Eysenck and Eysenck 1975;Mason and Claridge 2006). Regardless of whether a 'taxon' (genetic load) or dimensions underpin schizotypal traits, individuals with schizotypal traits might never develop a full-blown psychotic disorder. However, they will display a range of experiences that resemble the positive, negative and disorganised symptoms (Cochrane et al. 2010) that are also observed in schizophrenia patients (Arndt et al. 1991;Chen et al. 1997;Fonseca-Pedrero et al. 2009b;Liddle 1987;Raine 1991), but in less severe forms (Kwapil et al. 2008;Raine 2006). These schizotypal traits are still associated with increased likelihood of transition to clinical psychosis (e.g. Mason et al. 2004).

Meehl (1962) initially hypothesized that anhedonia represents the fundamental factor in the development of schizotypy (a component of schizotaxia), but this view changed in his later publications (Meehl 1990). For example, individuals from community samples scoring high on social anhedonia (reduced ability to experience pleasure from social relationships) exhibited characteristics mirroring schizophrenia patients (Chapman et al. 1994b;Mishlove and Chapman 1985). They showed elevated psychotic-like symptoms (Kwapil et al. 2002a;Mishlove and Chapman 1985) *(e.g. 5 year follow-up study (Gooding et al. 2005) and 10-year follow up (Kwapil 1998))*, cognitive deficits (Gooding et al. 2006;Tallent and Gooding 1999) and were at increased risk of developing a full-blown psychosis (Blanchard et al. 2011;Meehl 1962). Social anhedonia also successfully identified individuals with more positive and negative schizotypal traits in a longitudinal community study (Blanchard et al. 2011). This is surprising considering that social anhedonia is from a conceptual standpoint a part of negative schizotypy (Kwapil et al. 2008). Meehl (2001) later proposed that social anhedonia

might stand just as a secondary consequence of positive schizotypal symptomatology. Evidence inconsistent with this suggestion comes from another study that validated social anhedonia as a core feature of schizotypy (a taxon), independent of the positive schizotypy traits (Horan et al. 2007). In addition, social anhedonia was reported to be significantly associated with elevated trait negative affectivity[4] (Blanchard et al. 2011) *(which in turn relates to exposure and reactivity to stress (Blanchard et al. 2011;Bolger and Schilling 1991;Watson 1988))* and diminished positive affect (Blanchard et al. 2011;Gooding et al. 2002), again frequently found in schizophrenia patients (Berenbaum and Fujita 1994;Blanchard et al. 1998;Horan and Blanchard 2003). While Social Anhedonia Scale (Chapman et al. 1976) scores were associated with schizotypal personality disorder symptoms, including psychotic-like experiences, scores on the Physical Anhedonia Scale (Chapman et al. 1976) showed more mixed results, also implying that physical anhedonia (deficit in the ability to experience pleasure from usually pleasurable physical stimuli) might be associated with more heterogeneous symptoms and not specifically linked to schizophrenia-spectrum disorders (Gooding et al. 2005;Horan et al. 2007;Kwapil et al. 2002a;Rey et al. 2009).

In Meehl's view (1990), schizotypal personality organization is a broad construct and as such not equivalent to schizotypal personality disorder as defined in DSM-V (American Psychiatric Association 2013), although there are phenomenological similarities between the concepts (Lenzenweger 2006a). Studies following Meehl's proposition have documented that a cluster of schizotypal characteristics (e.g. positive-like symptoms and negative/deficit-like symptoms related to schizophrenia) is more prevalent in relatives of schizophrenia patients comparing to those with no familial risk for the disorder (Kendler et al. 1981;Reider

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[4] *Negative affectivity represents an affective state dimension/trait, reflecting persistent patterns of negative emotionality and self-concept (Watson and Clark 1984). It also relates to anxiety/neuroticism within the Big Five personality traits (DeNeve and Cooper 1998) .*

1979). Even though SPD was cited as a phenotypic expression of genetic liability to schizophrenia, some studies failed to support this (Kety et al. 1994;Squires-Wheeler et al. 1989;Squires-Wheeler et al. 1988;Yeung et al. 1993), suggesting that SPD might be a familial/genetic liability that underlies several different diagnostic categories. Some authors have argued that schizotypy is related to a broader range of psychotic disorders - schizophrenia spectrum disorders but not exclusively to schizophrenia (Kendler et al. 1995;Mata et al. 2003). Also, as schizotypy observed in schizophrenia relatives was associated with predominantly positive symptoms (e.g. delusions, hallucinations, thought interference), it was proposed that schizotypy might be more related to Schneiderian[5] first rank symptoms than schizophrenia per se (Mata et al. 2003).

Schizophrenia 'prodrome' and at-risk mental states

Similar to schizotypy, the 'schizophrenia prodrome' construct also reflects attenuated schizophrenia symptoms. However, schizotypy and prodromal symptoms of schizophrenia are independent, yet not always easily distinguishable constructs (Bedwell and Donnelly 2005). The prodromal construct is characterised by a more recent onset and escalation in symptom severity along with a decline in functioning, whilst SPD reflects more chronic, stable symptomatology (Miller et al. 2003). Also, symptoms in at least 5 out of 9 domains (in DSM-V) need to be present for the SPD diagnosis, while the prodromal stage can be identified with fewer symptoms (Woods et al. 2009). The prodromal phase refers to the period preceding the diagnosis of schizophrenia (and can only be confirmed retrospectively) in which an individual

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[5] *Schneiderian symptoms (Schneider 1959) of schizophrenia are symptoms strongly suggestive of schizophrenia. First rank of symptoms include: thought withdrawal, thought broadcasting, thought insertion, delusion of control, auditory hallucinations that comment on one's behaviour, auditory hallucinations in which two voices carry on a conversation, hearing one's thoughts spoken aloud. The presence of one of these symptoms was advocated to be symptomatically sufficient for the schizophrenia diagnosis, however this remains inconclusive (Nordgaard et al. 2008). Nevertheless, Schneider's criteria was widely criticised, with a study carried out in nine countries showing that only 58% of individuals with acute schizophrenia displayed at least one of these symptoms (Murray and Dean 2008).*

displays nonspecific features like anxiety or depressed mood along with attenuated schizophrenia symptoms (Yung et al. 1996; 2003) and it is usually associated with psychosocial impairment (Yung et al. 1996). Moreover, individuals in the prodromal phase are at higher risk of being diagnosed with schizophrenia within a period of 1 to 5 years than those with SPD (Hafner et al. 1998;Yung et al. 2003). In a large-scale epidemiological and neurobiological research project *The ABC Schizophrenia Study* (Hafner et al. 1998), *73%* of the cases of first-episode psychosis began with the prodromal phase, lasting approximately 5 years with an exponential increase in positive symptoms within the last year before the onset of disorder. The DSM-III-R (American Psychiatric Association 1987) listed the following nine markers of prodromal stage: i. social isolation/withdrawal; ii. impairment in role functioning; iii. peculiar behaviour; iv. impairment in personal hygiene; v. blunted or inappropriate affect; vi. digressive, vague, over-elaborate speech or poverty of speech; vii. odd beliefs or magical thinking; viii. unusual perceptual experiences; ix. marked lack of interest/energy. The prevalence of a single item in a general population study varied between *10%* and *50%* (McGorry and Singh 1995). However, the heterogeneity of the concept 'prodrome' and concerns over validity and specificity of these symptoms (see Yung et al. 2010) resulted in omission of this criteria from the DSM-IV (American Psychiatric Association 1994). The prodrome of schizophrenia is also not defined in ICD-10 (World Health Organisation 1992).

As the term 'prodromal' incorrectly implies that the subsequent disorder is inevitable and because of the non-specific nature of these early manifestations, the terms 'At-Risk Mental State' (ARMS) (McGorry and Singh 1995) or 'Ultra-High-Risk groups' (UHR) (Yung et al. 2003) have been proposed. The UHR was defined using three sub-categories (Yung et al. 2003; 2004): i. individuals with a family history of a psychotic disorder or schizotypal personality disorder along with a significant deterioration in mental state and/or functioning; ii. individuals with attenuated

psychotic symptoms or iii. individuals with a recent history of frank psychotic symptoms, resolving spontaneously within one week. Despite the interchangeable use of the terms 'ARMS', 'UHR' and 'prodromal' with all sharing the increased risk for developing schizophrenia *(see Figure 1)*, the 'prodrome' can be partially delineated by its definition – the state needs to be symptomatic, family history of schizophrenia is not a necessary prerequisite and the state implies the greater imminence of risk (Miller et al. 2003).

Besides, a psychosis prodrome and SPD patients both lie on the psychosis-spectrum of disorders, making the distinction between the constructs less straightforward. Furthermore, it was found that *26%* of individuals who fitted in prodromal criteria also met SPD criteria and *67%* individuals with SPD met prodrome conditions (Woods et al. 2009). Also, the transition rate to full-blown psychosis among individuals with SPD and those in the prodromal group was reported to be similar (Woods et al. 2009). However, a study that examined the discriminative validity of the self-report measure of SPD *(SPB-Q, Abbreviated Schizotypal Personality Questionnaire)* and the measure for heightened risk of developing schizophrenia or prodromal stage *(Y-PARQ-B, Abbreviated Youth Psychosis at Risk Questionnaire)* found a moderate to good discriminate validity between the concepts. Also, *75%* of respondents who fitted into the at-risk group using one measure did not reach the criteria using the other measure (Bedwell and Donnelly 2005). Also, findings from the *North American Prodrome Longitudinal Study* (Woods et al. 2009) observed that the SPD group displayed greater impairment of premorbid adjustment than the prodromal group, less clear escalation of the symptoms in the year before baseline, and later conversion to psychosis. Finally, the distinction between the SPD and prodrome suggests they are not necessarily mutually exclusive concepts but can also co-occur (Woods et al. 2009).
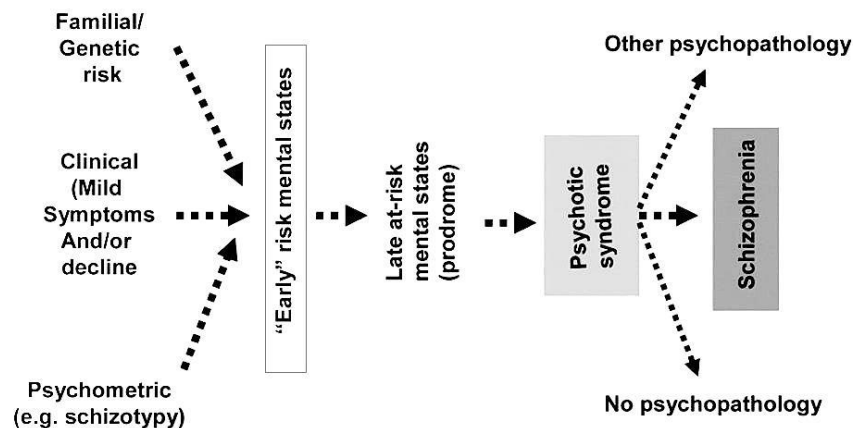
Figure 1: Summary of phases preceding schizophrenia. At-risk mental states are a heterogeneous constructs and can be defined in terms of genetic risk, psychometric risk (schizotypal personality disorder) or clinical risk (mild/sub-threshold symptoms and functional decline) (taken from Keshavan et al. 2011 p.3).

1.1.2   The multifactorial nature of schizotypy

An important question that has been widely discussed in the literature concerns the distinct dimensions of the schizotypy construct. Some authors have defined schizotypy as having a multidimensional structure, composed of distinct but highly correlated factors (Raine et al. 1994;Stefanis et al. 2004b).

Originally it was suggested that schizotypy consists of two separate dimensions: Cognitive/Positive (e.g. unusual perceptions, magical thinking, ideas of reference, suspiciousness) and Interpersonal/Negative symptoms (e.g. restricted affect, lack of close friends, suspiciousness) (Crow 1980;Kendler et al. 1991;Siever and Gunderson 1983). Further evidence indicated that this two-dimensional model parallels schizophrenia symptomatology and can be conceptualized as positive and negative features (Kelley and Coursey 1992;Muntaner et al. 1988;Raine and Allbutt 1989;Venables et al. 1990). Thus, negative schizotypy (e.g. social withdrawal and anhedonia) corresponds to negative symptoms in schizophrenia and positive schizotypy is an attenuated form of positive symptoms of schizophrenia (e.g.

hallucinations, delusions). Some studies have also suggested that the positive dimension itself might be multidimensional (Bergman et al. 1996;Stefanis et al. 2004b), consisting of two factors: a cognitive-perceptual (e.g. magical beliefs, perceptual aberrations) and a paranoia factor (Raine et al. 1994;Stefanis et al. 2004b) or even three factors (including referential thinking as a distinct factor) (Cicero and Kerns 2010b).

Building on a two-dimensional model of schizotypy, which has been criticized by many (Chen et al. 1997;Raine et al. 1994;Reynolds et al. 2000), further factor-analytical studies of the different scales used to measure schizotypy in a community population suggested a three-factor structure – also adding a Disorganized dimension (e.g. odd behaviour, odd speech) (Bergman et al. 1996;Chen et al. 1997;Claridge et al. 1996;Fossati et al. 2003;Gruzelier 1996;Kendler and Hewitt 1992;Liddle 1987;Raine et al. 1994;Reynolds et al. 2000;Rossi and Daneluzzo 2002;Venables and Bailes 1994;Vollema and Vandenbosch 1995). This model resembles schizophrenia symptomatology (Arndt et al. 1991;Fonseca-Pedrero et al. 2011;Lenzenweger et al. 1991;Liddle and Barnes 1990;Rossi and Daneluzzo 2002;Strauss et al. 1974). The three-factor structure of schizotypy has received cross-cultural support (Chen et al. 1997;Gruzelier 1996;Raine et al. 1994) and appeared to be invariant across age and sex (Badcock and Dragovic 2006;Fossatti et al. 2003). Nevertheless, mainly due to the variety of schizotypy measurements used and differences in the populations studied (Raine et al. 1995;Vollema and Vandenbosch 1995), other dimensions of the concept have been proposed. A four-factor structure introduced impulsive noncomformity as an additional factor (such as poor impulsive control, mood swings, nonconformist tendencies) (Mason 1995;Paino-Pineiro et al. 2008;Rawlings et al. 2001;Stefanis et al. 2004b;Suhr and Spitznagel 2001;Venables and Bailes 1994;Vollema and Vandenbosch 1995). Nevertheless, the inclusion of the fourth factor was criticized by some, as impulsive

nonconformity was not observed in relatives of schizophrenia patients (Claridge et al. 1983), it does not reflect cognitions or behaviours found in schizophrenia (Pickering 2004) nor does it predict the increased likelihood of developing psychosis (Chapman et al. 1994a). Furthermore, a five-factor structure (Chmielewski and Watson 2008;Diduca and Joseph 1999) or even six-factor structure (Mass et al. 2007) have subsequently been suggested, but have not received much support in the literature.

1.1.3 The heterogeneity of schizotypy measures

1.1.3.1 Measures of general schizotypy – schizotypy as a fully dimensional trait

An overview of the most commonly used measures of schizotypy is provided in Table 1.

Multidimensional schizotypy measures

Recently, the studies of schizotypy and schizophrenia have not been limited to just the categorical view of psychosis (as employed by the Diagnostic and Statistical Manual of Mental Disorders, DSM-IV-TR) (American Psychiatric Association 2000) but have tried to capture the full array of underlying risk factors and dimensions beyond dichotomous conceptualizations (Gross et al. 2012). A growing body of evidence argues that subtle, sub-clinical experiences are present throughout the general population with psychosis sitting on one extreme pole of the population continuum (Johns and van Os 2001;van Os 2003;Verdoux and van Os 2002). Also, factor-analytical studies consistently suggest that schizotypal personality is a construct consisting of distinct positive, negative and disorganised factors (Bentall et al. 1989;Claridge et al. 1996;Fonseca-Pedrero et al. 2011), further highlighting the need for schizotypy measures that are able to capture the full multidimensionality of

the concept. Some of the measures adopting this fully dimensional view of psychosis and assessing schizotypy from a personality/traits perspective are: The Oxford-Liverpool inventory of Feelings and Experiences (Mason et al. 1995), Schizophrenism scale (Nielsen and Peterson 1976), Rust Inventory of Schizotypal cognitions (Rust 1988), Schizophrenia Proneness scale of the Minnesota Multiphasic Personality Inventory (Butcher et al. 1989) and Structured Interview for Schizotypy (Kendler et al. 1989).

As suggested, the psychosis phenotypes are spread across the continuum depending on symptom intensity, severity and associated social impairment (van Os et al. 2009;Yung et al. 2009). These mild nonclinical phenotypes resemble dimensions found in schizophrenia (Barrantes-Vidal et al. 2010;Kwapil et al. 2012). Therefore, the schizotypy scales provide an opportunity to identify the schizophrenia prodromal indicators (or vulnerability markers) in a nonclinical community population (Claridge and Broks 1984;Lenzenweger 2006b;Raine 2006;Siever and Gunderson 1983), especially aiming at high-risk individuals who may be in need of early intervention. It stands as a useful framework, aimed at the early recognition of schizotypal traits without confounding effects associated with psychotic disorders (symptoms themselves as opposed to the effects of medication, treatment, hospitalisation or stigma etc.). Schizotypy as well as schizotypal personality disorder (SPD) relate to schizophrenia phenotypically and genetically (Silverman et al. 1993;Torgersen et al. 2002) and both show associations with increased risk for schizophrenia (Miller et al. 2002a;Tyrka et al. 1995). Focusing on schizotypy mirrors the psychosis-proneness model, while the SPD conceptualization supports the more (semi)categorical approach adopted in scales like Schizotypal Personality Questionnaire (Raine 1991). However, the factors indicative of liability to the development of psychosis are much broader than SPD criteria (Tsuang et al. 2002a).

Apart from the two very distinct perspectives for measuring schizotypy or psychosis-proneness (fully dimensional vs dichotomous), some of the measures have focused on the normal dimensions of personality (also advocated by Claridge 1997). For example, Eysenck (Eysenck and Eysenck 1975) introduced the Psychoticism Dimension Scale (P-Scale), adopting psychosis-proneness from a personality-theory position. The P-scale was designed to measure psychoticism argued to be a dimension of normal personality and linked to increased vulnerability to psychosis (especially schizophrenia). Despite evidence of parallels between personality and clinical approaches to measurement of psychotic traits, Eysenck's scale has received a lot of criticism. The measure was argued to lack specificity and have a weak predictive validity in terms of identifying individuals who might be at-risk for developing psychosis (Chapman et al. 1994b;Claridge 1983). In contrast, Meehl's (1962) conceptualisation emphasised a taxon ('schizotaxia'), an expression of a pathological process of neurodevelopment which indicates a heightened risk of psychosis (Mason et al. 1997). Both models however are consistent with the dimensional view of psychosis-proneness and do support the multidimensionality of the concept (Bentall et al. 1989;Fonseca-Pedrero et al. 2011;Kwapil et al. 2008).

Measures assessing individual schizotypal traits

Measuring of schizotypy began with the pioneering work of Chapman (1976), whose measures of schizotypy were largely based on Meehl's (1962) view of the concept. The series of Wisconsin Schizotypy scales (WSS) developed by Chapman group included Social Anhedonia and Physical Anhedonia (Chapman et al. 1976), Perceptual Aberration (Chapman et al. 1978), Revised Social Anhedonia scale (Eckblad et al. 1982) and Magical Ideation (Eckblad and Chapman 1983) *(see Table 1)*. The areas that these scales incorporated are consistent with the schizotypy concept reflecting a range of subtle, nonclinical manifestations of schizophrenia

(psychological, physiological deficits found in schizophrenia) (Gooding et al. 2005;Lenzenweger 2010;Vollema and Hoijtink 2000). However, each of the scales has aimed to measure a symptom or trait assumed to be indicative of risk for psychosis, but covered a narrower domain.

As such, Rado (1953) stressed the importance of anhedonia (inability to experience pleasure in physical and social domain) as a core vulnerability factor for schizophrenia spectrum disorders (Lenzenweger 2006a;Meehl 1962). Physical anhedonia seemed to be insensitive to clinical states and is present throughout the course of the illness. Therefore it stands as a substantially heritable trait (Linney et al. 2003;MacDonald et al. 2001) predicting later development of schizophrenia among high-risk groups (Gooding et al. 2005). The evidence comes from the studies showing that physical anhedonia is commonly observed in relatives of schizophrenia patients (Clementz et al. 1991;Kendler et al. 1996b). Additionally, a significant correlation has been documented for self-reported anhedonia in twins in a community population (Kendler and Hewitt 1992). Physical anhedonia is also a stable trait in schizophrenic patients (Rey et al. 1994). However not all schizophrenia patients experience substantial anhedonia, raising the question of whether anhedonia might be associated with one specific subtype of schizophrenia. One study observed that a familial risk for schizophrenia-spectrum disorders was three times higher in anhedonic schizophrenia patients compared to non-anhedonic schizophrenic patients (Schurhoff et al. 2003).

Social anhedonia subjects produced higher scores than controls on four dimensions of the Schizotypal Personality Questionnaire (Raine 1991) - social/interpersonal, disorganisation, paranoid and cognitive/perceptual, and displayed even higher schizotypy scores when compared to physical anhedonia (Rey et al. 2009). That ties with the study where higher social anhedonia also meant increased incidence of schizophrenia-related disorders and more psychotic-like

symptoms (Gooding et al. 2005). Furthermore, even though social anhedonia is originally defined in the context of negative schizotypal features (which would question generalizability to the 'schizotypal population'), those with social anhedonia often show increased positive schizotypal traits (Rey et al. 2009).

Whilst a physical anhedonia score is a stable vulnerability indicator (Blanchard et al. 2001b;Horan et al. 2008), perceptual aberration and magical ideation scores (Chapman et al. 1978;Eckblad and Chapman 1983) may reflect more mediating vulnerability factors (Horan et al. 2008), suggesting that these characteristics have different roles during the developmental processes (potentially) leading to psychotic disorder. Perceptual aberration and magical ideation reflect abnormalities during symptomatic state, therefore fluctuate on the course of the illness and worsen closer to the episode of full-blown psychosis (Horan et al. 2008). Not only have perceptual anomalies been consistently documented as more prevalent in patients with schizophrenia compared with healthy controls (Bunney et al. 1999) *(e.g. hyper-alertness and poor selective attention leading to overload of stimulation (Maher 1974)),* but the Perceptual Aberration Scale is also correlated with impaired functioning (Blanchard et al. 2011;Gross et al. 2012), psychotic-like experiences and schizotypal symptoms (Gross et al. 2012). Miettunen's (2011) prospective birth control cohort study even showed that the Perceptual Aberration Scale had the best concurrent validity for psychosis, including the best discriminate validity among measures of psychopathology.

Ambivalence was introduced by Bleuler (1950), defined by positive and negative characteristics of schizophrenia individuals. Meehl (1962) saw it as one of the four main symptoms of schizotypy, especially predictive of positive schizotypal symptomatology (Kwapil et al. 2000). However, ambivalence received little attention as has not been used consistently due to a lack of adequate operational definitions of the concept (Kwapil et al. 2000). As it was believed that ambivalence measure

might assess more general psychopathology (Kwapil et al. 2000;Raulin 1986), a new revised scale was designed: Schizotypal Ambivalence Scale (Raulin 1986).

Another manifestation of schizotaxia was characterized by cognitive slippage (Meehl 1962) *(Cognitive Slippage Scale – Miers and Raulin 1987)*, relating to the subjects' ability to keep track of their own thoughts. This scale covers the positive and negative domains of thought disorder e.g. incoherence, disorganisation (Miers and Raulin 1987). Furthermore, literature suggests an association between psychosis-prone individuals and impulsive and nonconforming behaviour. As a result, The Impulsive Nonconformity scale (Chapman et al. 1984) was designed to assess the lack of empathy towards others as well as antisocial behaviour, however not everyone with that particular trait will be considered psychosis-prone. As explained, each of the above-mentioned scales capture rather narrow domains, but using all Wisconsin Schizotypy scales (WSS) in combination to increase prediction power (Chapman et al. 1994a) can be a lengthy process.

Even though the Chapman scales have been widely used in psychosis-proneness research, some studies have raised questions about the stability of these scales and low predictive validity in diagnosing psychotic disorders (Kwapil et al. 2000;Meyer and Hautzinger 1999). Also, specific schizotypal traits do not adequately reflect the multidimensional nature of the schizotypy construct. Individual schizotypy items are rarely indicative of increased risk of developing psychosis. Using a range of schizotypal symptoms as assessed by these single measures was shown to improve identification of individuals at higher risk to transition to psychosis (Miller et al. 2002a). However, a more suitable (and methodologically more robust) approach to the assessment of different schizotypy dimensions would be the utilisation of one measure that assesses the multidimensionality of schizotypy as opposed to several single measures.

Table 1: Measures of general schizotypy – including schizotypy as dimensional personality traits (ordered by the year of publishing)

Author	Scale	What it measures/ How it measures	Validity and Reliability
Eysenck and Eysenck (1975)	Psychoticism scale	Measuring general psychoticism	Criticised for its specificity criterion (Claridge et al. 1983) and predictive validity (Chapman et al. 1994b)
Chapman et al. (1976)	Physical and Social Anhedonia Scales	Physical anhedonia - 40-items assessing inability to experience physical pleasure Social anhedonia - 48-items assessing inability to experience interpersonal pleasure/social anhedonia	Internal consistency – (Coefficient alpha) for physical anhedonia 0.66 (for females) to 0.74 (for males); For social anhedonia coefficient alpha 0.82 (females) to 0.85 (males); Correlation between the two scales was 0.60 for males and 0.51 for females (Chapman et al. 1976)
Chapman et al. (1976)	Revised Physical Anhedonia Scale	61-items true/false measuring inability to experience pleasure from physical stimuli (e.g. touching, listening to music)	Internal consistency 0.77 to 0.86 (Kwapil et al. 2008) to 0.94 (Fonseca-Pedrero et al. 2009a); Test-retest reliability 0.65 to 0.84; Correlation with Revised Social Anhedonia 0.40 (Chapman et al. 1995;Kwapil et al. 2008) or 0.30 (Fonseca-Pedrero et al. 2009a); Stability of the scale - Interclass coefficient (ICC)=0.78 (Horan et al. 2008)
Nielsen and Petersen (1976)	Schizophrenism Scale	Assessing cognitive and perceptual aspects of behaviour	(-)
Chapman et al. (1978)	Scale of Perceptual Aberration	35-item (true/false) scale measuring body image distortions and perceptual anomalies in nonclinical population	Internal reliability (alpha=0.85); Good discriminant validity (no association with Physical and Social Anhedonia Scales) (Chapman and Chapman 1980); Stability of the scale - Interclass coefficient (ICC)=0.70 (Horan et al. 2008)
Golden and Meehl (1979)	Checklist of Schizotypal Signs/ Schizoidia scale	7-item scale (short version) derived from Minnesota Multiphasic Personality Inventory (MMPI)	Cannot discriminate between different clinical groups (Miller et al. 1982)
Eckblad et al. (1982)	Revised Social Anhedonia Scale	40-item scale (true/false) assessing deficits in ability to experience pleasure from interpersonal interactions, schizoid indifference, associability, lack of social enjoyment, indifference towards others	Good reliability and validity 0.79 (Mishlove and Chapman 1985); Correlation with Perceptual Aberration (r=0.11 for males, 0.18 for females), Magical Ideation (r=-0.04 for males, 0.19 for females), Physical Anhedonia (r=-0.25 for males, 0.24 for females), Impulsive Nonconformity (r=-0.14 for males, 0.21 for females) - scale largely independent (Mishlove and Chapman 1985); Internal consistency ranges from 0.81 to 0.89; Test-retest reliability from 0.75 to 0.84 (Chapman et al. 1995;Kwapil et al. 2008)
Eckblad and Chapman (1983)	Magical Ideation Scale	30-item scale measuring unconventional beliefs	Good internal consistency (alpha=0.80) & Good convergent validity; Stability of the scale - Interclass coeff. (ICC)=0.83 (Horan et al. 2008)

Table 1 Cont'd

Chapman et al. (1984)	Impulsive Non-conformity Scale	51-item (true/false) measure of impulsive non-conformity	Stability of the scale - Interclass coefficient (ICC)=0.84 (Horan et al. 2008); Convergent validity (correlates with Psychoticism scale r=0.60) (Eysenck and Eysenck 1975)
Raulin (1984)	Intense Ambivalence scale	45-item scale (true/false) assessing intense ambivalence	Excellent internal consistency coefficient alpha=0.87, good test-retest reliability (0.81) (Kwapil et al. 2000); The scale correlates with Perceptual Aberration Scale 0.38 for males and 0.47 for females (Raulin 1984)
Raulin and Wee (1984)	Social Fear Scale	True/false statements assessing social fear	Good internal and test-retest reliability (Raulin and Wee 1984)
Eckblad and Chapman (1986)	Hypomanic Personality scale	48-item (true/false) measure of manic/impulsive behaviour	Correlation r=0.49 with Magical Ideation scale, r=0.43 with Perceptual Aberration Scale (Eckblad and Chapman 1986); Good internal consistency (alpha=0.87)
Raulin 1986 (Raulin 1986)	Schizotypal Ambivalence Scale	19-item scale – revised Intense Ambivalence Scale assessing simultaneous experience of contradictory emotions or the rapid change in emotions	Good internal consistency (alpha=0.84) (Kwapil et al. 2002b)
Miers and Raulin (1987)	Cognitive Slippage Scale	35-item (true/false) scale assessing subtle thought disorder	Good construct validity (Gooding et al. 2001); High internal consistency - 0.87 for males, 0.90 for females (Miers and Raulin 1987), similar in *Gooding et al. 2001*
Kendler et al. (1989)	The Structured Interview for Schizotypy (SIS)	See thesis section 3.4.1	See thesis section 3.4.1
Venables (1990)	Psychoticism and Anhedonia Scales	27-item (yes/no) scale measuring positive and negative schizotypy	Good construct validity
Mason et al. (1995)	Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE)	Multidimensional measure of schizotypy consisting of the following subscales: Unusual experiences, Cognitive Disorganisation, Introvertive Anhedonia, Impulsive Nonconformity 24 to 30 items per subscale (yes/no responses)	High internal consistency (alpha=0.77 or -0.89) (Mason et al. 1995); Cronbach's alpha for Introverted anhedonia=0.82 (Mason et al. 1995), 0.85 (Rawlings and Freeman 1997); For Unusual experiences=0.89 (Mason et al. 1995), 0.77 (Rawlings and Freeman 1997); Cognitive disorganisation=0.87 (Mason et al. 1995), 0.81 (Rawlings and Freeman 1997); Nonconformity=0.77 (Mason et al. 1995), 0.72 (Rawlings and Freeman 1997); Good test-retest reliability (Burch et al. 1998)

1.1.3.2 Measures of attenuated psychotic symptoms

Overall the measures that try to capture the continuum of psychosis proneness have differed from trying to assess the general schizotypy/schizotypal personality traits (Kendler et al. 1989) or focus on more specific psychosis-like symptomatology (Raine and Benishay 1995;Stefanis et al. 2002), especially positive symptoms *(e.g. hallucinations and delusions)* (Launay and Slade 1981;Peters et al. 2004). Some of the commonly used measures incorporating more attenuated psychosis-like symptoms are: the Peters et al. Delusional Inventory PDI-21 (short version) (Peters et al. 2004), Launay-Slade Hallucination Scale (Launay and Slade 1981), Paranoia Scale (Fenigstein and Venable 1992) and Community Assessment of Psychic Experiences (CAPE) (Stefanis et al. 2002) *(see Table 2 for an overview of the measures).* These instruments embrace more diagnostic criteria for assessing psychotic symptoms, outlining symptoms deviant from normal experiences. However, similar to schizotypy measures, the measures of attenuated psychosis-like symptoms do not necessarily assume pathology, as these experiences lay on a continuum and are observed throughout the general population (Johns et al. 2004;Stefanis et al. 2002). Therefore, the measures rather focus on the severity and frequency of these symptoms as well as the associated distress that reflects the clinical significance of these experiences and position individuals at higher risk of developing a full-blown psychosis. They have either been designed to assess just a single symptom (domain) (e.g. Launay-Slade Hallucination Scale, Paranoia Scale) or a range of symptoms (e.g. CAPE). For example, the Launay-Slade Hallucination Scale (Launay and Slade 1981) is a self-report measure assessing a combination of clinical and subclinical hallucinatory experiences (e.g. intrusive thoughts, auditory hallucinations) mainly utilised in studies of non-clinical populations (Morrison et al. 2000;Rankin and O'Carroll 1995). Similarly, Peters et al. Delusional Inventory PDI-21 (Peters et al. 2004) was designed to assess lifetime prevalence of low-level

delusional ideation and as such widely applied to the general population (Peters et al. 1999). Other single domain measures also adopting the psychosis continuum approach include The Paranoia Checklist (Fenigstein and Venable 1992) and the Paranoia/Suspiciousness Questionnaire (Rawlings and Freeman 1997). Both have shown good test-retest reliability and good internal consistency, but having been developed using non-clinical samples the validation in the clinical settings might be more questionable.

On the other hand, beside single domain measures, other scales have tried to gauge psychosis-proneness with an inclusion of a wider array of psychotic-like symptoms. These measures are for example Schizotypal Personality Questionnaire (SPQ) (Claridge and Broks 1984) and the CAPE (Stefanis et al. 2002), building on symptoms outlined by diagnostic criteria of psychosis. The SPQ specifically covers 9 criteria mirroring the DSM-III-R (American Psychiatric Association 1987) schizotypal personality disorder diagnostic criteria and consists of three subscales: cognitive-perceptual, interpersonal and disorganised schizotypy (Raine 1991;Raine et al. 1994). Also, one study using first-episode psychosis patients showed that the positive symptoms of SPQ were associated with genetic vulnerability to schizophrenia (Vollema et al. 2002), therefore the scale could be successfully applied to assess attenuated psychotic-like symptoms in addition to personality disorder.

Measures assessing Prodromal Psychotic Symptoms / At-Risk Mental States

Recently a lot of research has focused on identifying individuals in the prodromal stages of schizophrenia. The main aim is to prevent the illness before the actual onset by detecting and targeting individuals displaying milder, nonclinical symptoms of the disorder. The first type of measures focus on Attenuated Psychotic Symptoms (APS) based on the widely used Positive and Negative Syndrome Scale – PANSS

(Kay et al. 1987) *(e.g. Comprehensive Assessment of At-Risk Mental States –*
CAARMS (Yung et al. 2002), Structured Interview of Prodromal Syndromes – SIPS
(Miller et al. 1999)). The second type assesses symptoms in the Basic Symptoms
approach (BS). This approach is based on a phenomenological view of describing
disturbances such perception, cognition, language or motor function occurring prior
to the illness (Olsen and Rosenbaum 2006a) *(e.g. Bonn Scale for the Assessment*
of Basic Symptoms – BSABS (Gross et al. 1987)). This approach is believed to
reflect the early prodromal stage whereas the 'APS' approach is more likely to
detect individuals in the late prodromal stage. Both types of measures consider the
intensity, frequency and duration of the symptoms along with the degree of
conviction. In comparison to schizotypy symptoms defined as more stable traits, the
prodromal symptoms scales are constructed to measure a deviation in individual's
experiences or behaviour and a significant drop in their functioning. The transition
rates to clinical psychosis among high-risk individuals according to studies
employing the 'APS' approach varied between *9%* and *54%* (Olsen and Rosenbaum
2006b). However, the BS approaches has shown a stronger prediction of later
schizophrenia on a 10-year follow-up interval (Klosterkotter et al. 2001).

These screening tools assessing prodromal states are still under validation.
Also, the preliminary evidence comes from highly selected clinical samples and the
specificity of the symptoms is very low when administered to the general population
(Olsen and Rosenbaum 2006a). The measures have been designed to assess the
prodromal phase of schizophrenia, but the ability of these measures to adequately
evaluate residual psychotic symptoms remains questionable.

Table 2: Measures of attenuated psychotic symptoms (ordered by the year of publishing)

Author	Scale	What it measures/ How it measures	Validity and Reliability
Launay and Slade (1981)	Launay-Slade Hallucination Scale	12-item (Yes/No) scale measuring hallucination experiences	Good test-retest reliability (r=0.84)
Claridge and Broks (1984)	Schizotypal Personality Scale (STA) and Borderline Personality Scale (STB)	37-item scale assessing schizotypal personality (STA) 18-item scale assessing borderline personality	Good reliability and validity
Rust (1988)	Rust Inventory of Schizotypal Cognitions (RISC)	26-item assessing schizotypal cognitions, based on schizophrenia symptoms	Good reliability and validity (Rust 1988)
Raine (1991) Raine and Benishay (1995)	Schizotypal Personality Questionnaire (SPQ) And SPQ-B And also child version SPQ-C (Raine et al. 2011)	SPQ - 74-item self-report scale (Likert type response options), covering 9 criteria mirroring DSM-III-R schizotypal personality disorder diagnostic criteria (Raine 1991) SPQ-B (brief) consists of 22 yes/no items (Cohen et al. 2010), also used for dimensional schizotypy in nonclinical population (Bailey and Swallow 2004;Bedwell et al. 2006;Fonseca-Pedrero et al. 2009b;Mata et al. 2005)	Total reliability SPQ=0.91 (Raine 1991) For SPQ-B good Internal consistency: Ideas of reference (alpha=0.82), social anxiety (alpha=0.87), magical thinking (alpha=0.80), unusual perceptions (0.85), odd speech (0.86) (Cohen et al. 2010); Adequate internal consistency for all three subscales of SPQ-B – coefficient alpha=0.72 to 0.78, however they showed limited convergent and discriminate validity of SPQ-B, similar as SPQ (Raine 1991;Raine and Benishay 1995); Low correlation with Perceptual Aberration Scale (Compton et al. 2009b)
Fenigstein & Vanable (1992)	Paranoia Scale	20-item 5-point scale measure of paranoia	Good test-retest reliability (r=0.70); Good internal consistency (alpha=0.80)
Rawling and Freeman (1997)	Paranoia/Suspiciousness Questionnaire	47-item (yes/no) scale measuring paranoia and suspiciousness	Good internal consistency and test-retest reliability
Bunney et al. (1999)	The Structured Interview for assessing Perceptual Anomalities SIAPA	15-item interview measure, assessing anomalies of five senses, frequency of experiences rated on 5-point Likert scale	Good inter-rater reliability (kappa coefficients from 0.67 to 1.0) for auditory, visual and tactile modalities, olfactory and gustatory poorer - inter-rater agreement (0.28 to 0.72) (Bunney et al. 1999); Internal consistency - Cronbach alpha: auditory=0.84, visual 0.73, tactile=0.66, olfactory=0.46, gustatory=0.68 (Bunney et al. 1999)
Peters et al. (1999)	Peters et al. Delusion Inventory PDI-21 (shortened)(Peters et al. 2004)	21-item measure of all types of delusional beliefs and their multidimensionality, lifetime prevalence	PDI-21 Good internal consistency (Cronbach alpha= 0.82) (Peters et al. 2004), 0.74 (Cella et al. 2011),

Table 2 Cont'd

		assessed with yes/no format (7-factor structure) Likert scale for positive responses to rate the degree of distress, preoccupation and conviction	0.77 (Jones and Fernyhough 2007); Good concurrent validity (Peters et al. 1999)
Freeman et al. (2005)	Paranoia checklist	18-item self-report measure (5-point scale) assesses frequency, distress and degree of conviction and distress	Good internal consistency (alpha=-0.90)
Bell et at. (2006)	Cardiff Anomalous Perceptions	32-item (yes/no) self-report assessment (5-point Likert scale for associated frequency, distress and intrusiveness)	Internal reliability (alpha=0.-89) (Bell et al. 2011); Test-retest reliability (r=0.77); Internal consistency (alpha=0.92)
Stefanis et al. (2002); Konings et al. (2006)	Community Assessment of Psychic Experiences - CAPE	See thesis section 3.4.2	See thesis section 3.4.2

Examples of measures of prodromal symptoms/at-risk mental states

Goss et al. (1987)	Bonn Scale for the Assessment of Basic Symptoms (BSABS)	6 subscales assessing basic symptoms (present/absent), scales of dynamic deficits, cognitive disturbances, coenesthetic experiences, central vegetative disturbances and autoprotective behaviour	Good predictive validity (Klosterkotter et al. 2001)
Hafner et al. (1992)	Interview for the Retrospective Assessment of the Onset of Schizophrenia (IRAOS)	A semi-structured interview, consisting of 5 sections, assessing the social course, symptoms, disability and treatment, for the time period between the first signs of illness and the time of interview	Good validity and reliability
Miller et al. (1999)	Structured Interview for Prodromal Symptoms (SIPS)/ Scale of Prodromal Symptoms (SOPS)	5 items assessing positive symptoms, 4 items for disorganisation, 4 items for general symptoms (SIPS) each on a 6-point severity scale (0 to 6) (SOPS)	Good predictive validity
Yung et al. (2002)	Comprehensive Assessment of At-Risk Mental States (CAARMS)	At-risk mental state defined using Ultra-High-Risk group (UHR) criteria (Yung et al. 2005) A semi-structured interview, 7 subscales (positive symptoms, cognitive change, emotional disturbance, negative symptoms, behavioural change, motor physical change, general psychopathology)	Good predictive validity

Sensitivity and specificity of the early assessments

Evidence shows that schizotypal traits (subclinical expression of psychosis based on Meehl's (1962) definition) or risk indicators of at-risk groups successfully predict the transition to clinical psychosis (Chapman et al. 1994b;Klosterkotter et al. 1997;Lencz et al. 2004;Miller et al. 2002b;Morrison et al. 2002a) even within a 10-year transition period (Chapman et al. 1994b;Klosterkotter et al. 1997). In a 6-month follow-up Yung and colleagues (Yung et al. 1998) observed the transition to clinical psychosis in *40%* of high-risk individuals. Overall, the reports on transition rates vary from *9%* (Carr et al. 2000) to *70%* (Klosterkotter et al. 2001), highly dependent on the study design and the measures used (Olsen and Rosenbaum 2006b). A more recent review of the literature however reported consistent transition risk (independent of the instrument used) of *18%* after 6 months follow-up, *22%* after 1 year and *36%* after 3 years (Fusar-Poli et al. 2012). Some of the studies stressed the predictive value of attenuated psychotic-like symptoms such as perceptual aberrations or odd beliefs (with *46%* of individuals displaying such symptoms having transitioned to clinical psychosis within 6 months) (Miller et al. 1999). Others have documented the high frequency of either negative symptoms preceding the schizophrenia onset (e.g. social isolation (Lencz et al. 2004)), nonspecific symptoms (e.g. depression, anxiety (Yung and McGorry 1996)) or neuropsychological deficits (e.g. disturbances of thought or memory (Klosterkotter et al. 1997; 2001)). A ten-year longitudinal study using the Bonn Scale for the Assessment of Basic Symptoms (BSABS) showed that the presence of prodromal symptoms predicted schizophrenia with a probability of *70%* (the absence of these excluded schizophrenia with the probability of *96%*) (Klosterkotter et al. 2001). Negative symptoms (Tarbox and Pogue-Geile 2011) and neuropsychological deficits (Faraone et al. 2000; 2001) also reflect genetic predisposition to schizophrenia and are commonly observed in relatives of individuals with schizophrenia. Zanelli and colleagues (2010) reported that cognitive

deficits are present across psychotic disorder but most severe forms are specific to schizophrenia. Generally theories suggest that a negative cluster of symptoms can be observed much earlier in the developmental path to psychosis compared to the positive cluster, with *75%* of individuals with schizophrenia reporting negative symptoms 5 years prior to the onset of positive symptoms (Hafner et al. 1998). In contrast, positive and negative symptom clusters might underlie distinct clinical entities (Cornblatt et al. 2002).

Nevertheless, relying on either single symptom or diagnostic at-high-risk categories to identify individuals prior to the illness has raised a lot of concerns. Firstly, these approaches can lead to high 'false positive' rates (Simon et al. 2013;Thompson et al. 2011;Yung et al. 2010;Yung and McGorry 1996) and may have insufficient specificity for schizophrenia (Jackson et al. 1995) therefore lacking clinical utility. Secondly, the observed changes in mental state could imply different pathology (e.g. depression) or the features considered pre-psychotic will either resolve spontaneously (e.g. Huber et al. 1980) or with the support of protective factors (e.g. social support, sufficient coping abilities) (Yung and McGorry 1996). Thirdly, schizophrenia is aetiologically heterogeneous, therefore using a narrower, unitary entity (e.g. prodromal phase) to identify individuals at-risk could be misleading. Fourthly, over-reliance on categorical conceptualisations to predict psychosis (Olsen and Rosenbaum 2006b) does not reflect the continuum hypothesis of psychosis (van Os et al. 2009) *(see section 1.1.5).* Hence the understanding of the interaction between the sensitivity ('true positives') and specificity ('true negatives') of the symptoms/signs predictive of psychosis is still a challenge in psychiatric research.

Nonetheless, the reliable early identification of individuals at risk of developing psychosis provides invaluable clues for clinical practice. There is some evidence that early identification and treatment are associated with more favourable

illness outcome, better social and cognitive functioning, less hospitalisation, lower relapse rates *(for a review of the literature see Lieberman et al. 2001)* but also raises important ethical considerations (Schaffner and McGorry 2001).

1.1.4   Socio-demographic characteristics and schizotypy

Overall studies have found that women tend to score higher on positive schizotypy while men score higher on the negative/disorganised dimension (Claridge and Hewitt 1987;Fossati et al. 2003;Kwapil et al. 2008;Maric et al. 2003;Mason and Claridge 2006;Mata et al. 2005;Raine 1992). While women present higher levels of social anxiety, magical thinking and social paranoia (Fossatti et al. 2003;Mata et al. 2005;Paino-Pineiro et al. 2008;Rawlings et al. 2001), men display higher levels of negative and disorganized symptoms (Fossatti et al. 2003;Mata et al. 2005;Miller and Burns 1995), e.g. physical anhedonia, social anhedonia (Claridge et al. 1996;Goulding et al. 2009;Paino-Pineiro et al. 2008), as well as impulsive non-conformity scores (Claridge 1987;Maric et al. 2003;Raine 1992). These findings were observed in samples of community adults (Mason and Claridge 2006) and adolescent populations (Cyhlarova and Claridge 2005;Venables and Bailes 1994). In general, the gender differences in patterns of symptoms are comparable to findings from clinical samples/schizophrenia patients (Bardenstein and McGlashan 1990;Goldstein and Link 1988).

The reports on associations between age and schizotypy have been mixed (Bora and Arabaci 2009;Fonseca-Pedrero et al. 2008;Fonseca-Pedrero et al. 2011;Mata et al. 2005;Paino-Pineiro et al. 2008;Venables and Bailes 1994). Nevertheless, the studies generally suggest that schizotypal traits tend to have a negative correlation with age (in adult and adolescent populations) (Bora and Arabaci 2009;Cyhlarova and Claridge 2005;Fossatti et al. 2003;Mata et al. 2005).

Especially positive/psychotic-like symptoms (Claridge et al. 1996;Venables and Bailes 1994) and disorganized symptoms showed a significant decrease from the beginning of adulthood onwards in both sexes (Bora and Arabaci 2009;Fossatti et al. 2003). Another study with adolescent girls (aged 12 to 15) failed to confirm these findings, suggesting a positive association between age and total schizotypy scale scores (Fonseca-Pedrero et al. 2008), however due to the possibility of the effect of maturation processes, the interpretation requires some caution (Diduca and Joseph 1999;Wolfradt and Straube 1998). Furthermore, age-related reduction of psychosis-proneness from adolescence to adulthood (Verdoux et al. 1998) could be related to brain maturation (DeLisi 1997;Weinberger 1987) which parallels the increased activity of the dopaminergic system, also showing regression with ageing (Weinberger 1987;Wong et al. 1984).

Differences in schizotypal traits across ethnic groups have been reported for delusional ideation, hallucination experiences and presence of psychotic symptoms, with Afro-Caribbean populations scoring significantly higher than other ethnic groups (Johns et al. 2002;King et al. 2005;Sharpley and Peters 1999). Interestingly, race was the only independent predictor of perceptual aberrations and social anhedonia with African-Americans scoring the lowest out of all ethnic groups (Goulding et al. 2009). The possible cultural differences in schizotypal traits can also be observed, as being a member of certain religious movements has been linked to an increase in schizotypal symptomatology (Sharpley and Peters 1999). Also, few studies have reported an association between religious affiliation and belief in the paranormal (Linney et al. 2003;Thalbourne 1995).

Other socio-demographic factors demonstrating links to higher levels of schizotypal traits were lower social support, poorer overall and social functioning and fewer intimate relationships (Horan et al. 2007;Kwapil 1998;Kwapil et al. 2008). Another study observed that male gender, younger age, unemployment, migrant

status, urban residence, lower income, lower academic attainment and living alone were all associated with the increased likelihood of reporting delusional experiences (Scott et al. 2007).

1.1.5   Schizotypy and the development of psychosis / The continuum model

A growing body of evidence suggests that the psychosis phenotype is expressed at subclinical level in literature referred as schizotypy, psychosis-proneness, psychotic-like experiences or even at-risk mental states (Chapman et al. 1994b;Siever et al. 1993;Stefanis et al. 2002;van Os et al. 2000;Vollema et al. 2002;Yung et al. 2003). The preliminary proposition of the continuum model was documented by Rado (1960) who used the term *'developmental stages of schizotypal behavior'*, that was later conceptualized as a continuum of phenotypic outcomes (Meehl 1962). Rado (1960) suggested that etiologic unity (*"a common schizophrenia diathesis", p.88*) underlies a range of clinical manifestations ranging from schizotypy to schizophrenia.

Evidence for this continuum approach to psychosis has been obtained from several experimental studies (Laurens et al. 2007b;Myin-Germeys et al. 2003b;Yon et al. 2009). However, some controversy remains as to whether the continuum is fully dimensional and relating to all people (Claridge 1972;Claridge and Beech 1995;Claridge and Davis 2003;Rawlings et al. 2008) or quasi-dimensional (Beauchaine et al. 2008;Lenzenweger 1994;Meehl 1962;Meehl 1989) applying only to those individuals with schizophrenia and schizotypy with schizophrenic genes[6]

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[6] *Many studies have reinforced the importance of genetic determinants underlying schizophrenia and elevated schizotypal characteristics were observed in the family members of schizophrenia patients. The prevalence of latent schizophrenia in biological relatives was estimated at 8.2% (Kety et al. 1994) with another study showing that personality disorder among nonpsychotic first-degree relatives of schizophrenia was assessed to range between 4.2% and 14.6% (Tsuang et al. 1999) (see section 1.2).*

(approximately 10% of the population). From a fully-dimensional standpoint, schizotypal features including psychosis-like experiences represent a healthy variation (Claridge et al. 1996) with no discontinuity from the normal population (Claridge 1972; 1994). According to the quasi-dimensional model, beginning with the Meehl's idea of 'schizoid taxon' (Meehl 1962), only those with high schizotypy scores reflect a heightened risk of developing psychotic disorder (suggesting discontinuity with the normal population) (Chapman et al. 1980;Chapman and Chapman 1987). Especially physical and social anhedonia (as a life-long characteristic, not transitory as observed in depression (Meehl 1962)) were highlighted as the main features indicative of psychosis-proneness in a community population (Chapman et al. 1980;Chapman and Chapman 1987). According to Meehl (1962) however, schizotaxia is a necessary (discontinuous view) but not sufficient factor in the development of schizophrenia as it does interact with environmental influences (Lenzenweger 2006b), thus determining the level of psychotic expression (a quasi-continuous model) (Nelson et al. 2013). Consistent with this view, a stress vulnerability model suggests that individuals who are psychosis prone (with high schizotypal levels) and are exposed to psychosocial stress are more likely to develop clinical psychosis (Zubin and Spring 1977). Similarly, fully dimensional model proposes that only high levels of schizotypy are sufficient for the individual to be at risk of developing schizophrenia or other psychotic disorders. Only a combination of genes and aetiological risk factors result in different phenotypic expression along the psychosis continuum (Rawlings et al. 2008). Yung and colleagues (2009) saw the evidence for both models, showing that some traits like bizarre experiences, perceptual abnormalities and persecutory ideas may reflect underlying vulnerability to psychotic disorder (as per quasi-dimensional model), whereas magical thinking is a common/'normal' personality trait (fully-dimensional model).

There is now a growing body of research supporting the fully dimensional model of schizophrenia (Asai and Tanno 2008;Lenzenweger and Maher 2002;Rossi and Daneluzzo 2002;van Os et al. 2000;Verdoux and van Os 2002;Yon et al. 2009) demonstrating that schizotypal personality traits do vary along a normality-abnormality continuum found in both clinical and community groups and adolescent and adult populations (Cyhlarova and Claridge 2005;Johns et al. 2004;Sanchez-Bernardos and Avia 2006). The evidence for the continuum also comes from reports of a high prevalence of psychotic-like symptoms in the general population (Hanssen et al. 2005;Johns et al. 2004;Poulton et al. 2000;Tien 1991;van Os et al. 2000;van Os et al. 2001), estimated between *4%* (Eaton et al. 1991) and *28.4%* (Kendler et al. 1996a), with *17.5%* of the population reporting that they have experienced at least one psychotic symptom (van Os et al. 2000). In another cross-national study, the prevalence of psychotic symptoms ranged widely between *0.8%* and *31.4%* (Nuevo et al. 2012). The lifetime prevalence of hallucinations has been documented to be *10%* for men and *15%* for women (Tien 1991), or *13%* by the age of 26 (using a birth cohort study) (Poulton et al. 2000). Also, the prevalence of delusions before the age of 26 was assessed at *20%* (Poulton et al. 2000). Although these subclinical psychotic-like experiences are commonly observed, they are still evidenced to be risk factors for the onset of full-blown psychotic disorder (Lataster et al. 2009), regardless of what age they occurred at and what other traumatic events had been experienced (Saha et al. 2011). Moreover, the continuity between subclinical and clinical phenotypes (Johns and van Os 2001;Mata et al. 2003;van Os et al. 2000;van Os et al. 2009) is suggested by transitions over time (from subclinical to clinical manifestations (Kwapil 1998;Yung et al. 2003)), even after 15-years (Chapman et al. 1994b;Hanssen et al. 2005;Poulton et al. 2000). However, a prospective longitudinal study showed that childhood psychotic symptoms were not specific to schizophrenia in adulthood but rather a significant indicator of adult psychopathology more broadly (Fisher et al. 2013a).

Although most subclinical psychotic symptoms are transitory, there is evidence for a *30%* persistence rate for psychotic symptoms over an 18 month period (Wiles et al. 2006). However, another study assessed the persistence rate at only *8%*, with another *8%* transferring to clinical disorder two years later (Hanssen et al. 2005). Looking at child populations, *75%* to *90%* of auditory vocal hallucinations were transitory and disappeared over time (Bartels-Velthuis et al. 2011). Despite this high percentage of subclinical symptoms being short-lived, if a person is exposed to environmental risks such as trauma (Spauwen et al. 2006) or cannabis use (Henquet et al. 2005b), this may lead to abnormally persistent levels of psychotic-like experiences (Cougnard et al. 2007;Kuepper et al. 2011). Evidence for phenomenological continuity in form and structure (van Os et al. 2009) is observed in individuals with high schizotypy scores, as they perform in similar ways to individuals with clinically diagnosed psychosis (Berenbaum et al. 2003;Campbell and Morrison 2007;Laurens et al. 2007b;Myin-Germeys et al. 2003b), exhibiting a range of positive, negative and disorganised symptoms (Cochrane et al. 2010) also found in schizophrenia (Arndt et al. 1991;Chapman et al. 1994b;Chen et al. 1997;Gooding et al. 2005;Kendler and Walsh 1995;Liddle 1987;Raine et al. 1994;Raine 2006;Vollema and Vandenbosch 1995).

Beside molecular genetic evidence (Fanous et al. 2007), the genetic relationship between schizotypy and schizophrenia is observed in relatives of individuals with schizophrenia who display more schizotypal traits than those without familial risks for the disorder (Clementz et al. 1991;Kendler and Walsh 1995;Vollema et al. 2002). This also emphasizes that psychotic and personality features share a common genetic basis, implying that the schizotypy concept incorporates psychosis-proneness and personality aberrations (Jang et al. 2005)**.** Reflecting that, schizotypal personality disorder can be viewed either as a premorbid phase of schizophrenia or as a range of personality traits normally distributed in the general

population (Raine 2006). Further support for familial resemblance between schizotypal symptoms and individuals with schizophrenia and their relatives comes from several studies (Erlenmeyer-Kimling 2000;Stone et al. 2001) reporting for example that positive symptoms in schizophrenia patients correlate with higher schizotypy in their relatives (Mata et al. 2000), with higher scores on physical anhedonia and perceptual aberration scores (Clementz et al. 1991;Grove et al. 1991). Moreover, negative symptoms or neuropsychological deficits were also to a lesser degree observed in relatives of schizophrenia patients (Bergida and Lenzenweger 2006;Cochrane et al. 2012;Dinn et al. 2002;Faraone et al. 1995;Noguchi et al. 2008;Sitskoorn et al. 2004;Tsuang 1993), especially affecting attention, long-term verbal memory and executive functions (Faraone et al. 1995; 2000). A similar neuroanatomical signature of schizophrenia and schizotypy was evidenced in smaller volumes of specific brain regions (Seidman et al. 1999) e.g. prefrontal areas (Modinos et al. 2010;Raine et al. 1992), temporal areas (Modinos et al. 2010) and grey volume reduction in prefrontal and temporal areas (Ettinger et al. 2012). In addition, an association was also reported between schizotypy and verbal IQ (Noguchi et al. 2008) as well as working memory (Schmidt-Hansen and Honey 2009). However, the limitations of these findings should be noted firstly as some studies only showed weak correlations (Lenzenweger and Gold 2000;Matsui et al. 2004;Simons et al. 2007;Voglmaier et al. 2000) and secondly because cognitive deficits may also interact with genetic risk for the disorder (Johnson et al. 2003).

Apart from genetic factors, social/environmental factors play a crucial role in schizophrenia and also underlie schizotypy, indicating the aetiological continuity of psychosis (Fanous et al. 2001;Hanssen et al. 2006;Kendler et al. 1993a;Vollema et al. 2002). Clinical and subclinical phenotypes also share the same environmental risk factors such as childhood trauma (Lovatt et al. 2010;Read et al. 2005;Steel et al. 2009) and cannabis use (Arseneault et al. 2004;Barkus and Lewis 2008;Cohen

et al. 2011;Compton et al. 2009a;Henquet et al. 2005b;Skinner et al. 2011;van Os et al. 2002) in a dose-response fashion (Janssen et al. 2004;van Os et al. 2001). Moreover, there is a similar association with demographic factors: ethnic minority status (Cantor-Graae and Selten 2005;Morgan et al. 2009;Sharpley and Peters 1999), an association with urbanicity (Krabbendam and van 2005;van Os et al. 2001), the negative association with age (Bora and Arabaci 2009;Johns et al. 2004), the positive association with single marital status and social disadvantage (Johns and van Os 2001;Peters et al. 1999;Verdoux et al. 1998) and gender differences (Roy et al. 2001).

When looking at the subclinical psychotic symptoms and their relation to the development of schizophrenia, the best all-round predictor was social withdrawal (Miller et al. 2002a). However, a combination of four symptoms and signs in young people as measured by the Structured Interview for Schizotypy was demonstrated to index the highest risk for developing a full-blown psychosis: social withdrawal, psychotic symptoms, socio-emotional dysfunction and odd behaviour. Interestingly, odd behaviour alone was higher in individuals with subclinical symptoms (in the prodromal phase) compared to individuals with schizophrenia, suggesting that either clinical psychotic symptoms overpower or mask the odd behaviour or the treatment itself influences the decrease in this particular trait. Again, this provides additional support to the heterogeneity of schizotypy itself (Miller et al. 2002a).

The fully dimensional model that places schizotypy on a continuum might help us to understand the pathology of schizotypal personality as well as assist in identifying the mechanistic pathways that are leading to the development of psychotic disorders, consequently contributing to effective prevention and/or interventions (Raine 2006;Yon et al. 2009). Importantly though, high levels of schizotypy are not necessarily linked to dysfunctional behaviour with one study showing an association between schizotypy and subjective perception of control a

subject has over positive and negative events (Goulding 2004) while another linked schizotypy to improved creativity (Batey and Furnham 2008;Nelson and Rawlings 2010).

In summary: According to the continuum hypothesis, the psychosis phenotype can be observed at a subclinical level. These subclinical schizotypal characteristics can be clustered into three sub-dimensions, mirroring schizophrenia symptomatology: positive, negative and disorganised cluster. Mainly because of the heterogeneous nature of schizotypy there is a large amount of conflicting evidence with regard to appropriate measures assessing its features, including discrepancies as to which domain is a core of schizotypy and as such more likely to be a reliable predictor of clinical psychosis. Despite the fact that schizotypy implies genetic vulnerability to schizophrenia the importance of environmental factors should not be undervalued.

1.2　*Heritability of schizotypy*

This section covers the exploration of studies examining familial/genetic foundations of schizotypy and consequently its link to schizophrenia. Various schizotypal domains and traits along with evidence for any distinct genetic underpinnings are presented. In brief, this section summarises the studies looking into quantitative and molecular genetics (and interaction with environmental factors) contributing to schizotypy load.

Family studies

As discussed in the previous chapter, there is consistent evidence in the literature supporting genetic determinants underlying schizophrenia, with elevated schizotypal characteristics observed in the family members of schizophrenia patients (Appels et al. 2004;Baron et al. 1985;Battaglia et al. 1995;Chang et al. 2002;Kendler and Gruenberg 1984;Mata et al. 2000;Siever and Davis 2004;Tienari et al. 2003;Torgersen 1984;Torgersen et al. 1993). Also in parallel with these findings are studies that used psychometric measures of schizotypy (Chapman et al. 1978;Chen et al. 1998;Kendler and Hewitt 1992). Overall, positive and negative symptoms in schizophrenia correspond with positive and negative schizotypal dimensions in their pedigrees (Fanous et al. 2001). However negative symptoms in schizophrenia showed associations with more schizotypy factors than positive symptoms. This evidence implies a greater genetic basis for negative symptoms compared to positive symptoms and more apparent phenomenological resemblance between negative symptoms in schizotypy and negative symptoms in schizophrenia (Fanous et al. 2001). On the other hand, it was suggested that siblings of individuals with schizophrenia showed similarity of the first rank symptoms such as though insertion, thought broadcasting, thought withdrawal and delusions of control (Loftus et al. 2000). Recognizing any of these non-clinical phenotypes that have a genetic link to schizophrenia (Gottesman and Gould 2003;Lataster et al. 2009) enhances

identification of the genes affecting schizophrenia risk with less bias related to the disorder or the treatment (van Os et al. 2009). Even though there are inconsistencies in findings, previous studies indicated elevated scores among relatives of people with schizophrenia on all three schizotypy dimensions (Appels et al. 2004;Kendler et al. 1991;Kremen et al. 1997;Yaralian et al. 2000). Heritability of schizophrenia-like traits is noted in some early schizophrenia studies and is conceptualised under different terms like *'latent schizophrenia'* in *'Dementia Praecox'* (Bleuler 1911), Rado's (1953) model of *'schizotypy'* as schizophrenic genotype or Meehl's (1962) *'schizotaxia'*.

Some authors suggested a distinction between schizotypal personality disorder within and outside genetic spectrum of schizophrenia, proposing two distinct SPD subgroups, one genetically related to schizophrenia, the other genetically unrelated (Torgersen et al. 2002). According to this proposition, those with schizotypal personality disorder who are genetically related to individuals with schizophrenia display more negative features (also interpersonal and disorganised features), whilst the subgroup with no family history of schizophrenia is more characterized by positive symptomatology *(e.g. suspiciousness, paranoia, ideas of reference, social anxiety, self-damaging acts, free-floating anger and sensitivity to rejection)* (Torgersen et al. 2002). As negative schizotypy is more pronounced inside the schizophrenia spectrum (especially odd communication and inadequate rapport, social isolation), these symptoms might represent the 'genetic core' of schizotypy related to schizophrenia (Torgersen et al. 2002). This ties in with other studies advocating the negative schizotypy dimension as the main link to schizophrenia (Kendler et al. 1995;Squires-Wheeler et al. 1997). Furthermore, the subgroup inside schizophrenia spectrum disorders was shown to exhibit biological abnormalities similar to individuals with schizophrenia (Siever and Coursey 1985) and impaired language (Condray and Steinhauer 1992) which are also features of 'schizotaxia'

(genetic predisposition to schizophrenia as introduced by Meehl (1962)). Additionally, negative symptoms showed more stability over time (Pogue-Geile and Harrow 1985;Rey et al. 1994) and individuals with negative schizotypy symptoms are more likely to develop psychosis than individuals with positive symptoms (Dworkin and Lenzenweger 1984;Fanous et al. 2001;Verdoux et al. 1996), however Baron et al. (1992) reached the opposite conclusions. Also, not all the studies agreed on heritability of merely negative schizotypy but also observed high heritability of the positive dimension (Battaglia et al. 1999;Kendler et al. 1991).

In general, various factors of schizotypy are demonstrated to be moderately heritable (Claridge 1987;Kendler et al. 1991;Kendler and Hewitt 1992;MacDonald et al. 2001), but the dimensions proposed to have predominantly genetic effects varied across studies. Tarbox and Pogue-Geile's (Tarbox and Pogue-Geile 2011) review of the literature indicated that social-interpersonal schizotypal dimension has a medium genetic effect *(d=0.67)* among relatives of schizophrenia patients, with symptoms like social anxiety, constricted affect and suspiciousness carrying the strongest familial association (Pogue-Geile and Yokley 2010). The cognitive-perceptual dimension on the other hand demonstrated only a small familial association *(d=0.37)*, but possibly raises the issue of underreporting of positive schizotypal symptoms. Disorganized schizotypy symptoms however showed a large genetic component *(d=0.96)* when measured using the interview method but only a small effect *(d=0.22)* when a questionnaire type of measure was utilised. Although language abnormalities and thought disorder, for example, have been previously reported in relatives of schizophrenia patients (Condray and Steinhauer 1992;Docherty and Gottesman 2000;Erlenmeyer-Kimling 2000;Hoff et al. 2005;Zanelli et al. 2010), overall the disorganized dimension has less stability than social-interpersonal and cognitive-perceptual dimension (Bergman et al. 2000).

Heritability of schizotypal traits was assessed to be *0.61* for schizotypal personality disorder (Torgersen et al. 2001) with one study also suggesting the highest heritability for SPD *(at 0.81)* out of 12 personality disorders assessed (Coolidge et al. 2001). More mixed support comes from other studies where heritability of social anhedonia ranged from *27%* (MacDonald et al. 2001) to *67%* (Kendler and Hewitt 1992), while unusual experiences, introvertive anhedonia and cognitive disorganisation were assessed at *50%*, with much lower heritability for delusions *(37%)* (Linney et al. 2003). This difference in findings might also be a reflection of a diversity of measures used. For example heritability of schizotypy was reported to be *50%* when using the Schizotypal Personality Questionnaire (Lin et al. 2007), *53%* using Schizotypal Personality Scale (Claridge and Hewitt 1987), ranging between *27%* (MacDonald et al. 2001) and *40%-50%* (Hay et al. 2001) for the Social Anhedonia scale, *33%* for the Perceptual Aberration scale (MacDonald et al. 2001) and *50% (for unusual experiences, cognitive disorganisation and introvertive anhedonia)* using the O-LIFE (Linney et al. 2003).

Also, studies showed that although trauma significantly predicted the extent and developmental course of subclinical psychotic experiences (Read et al. 2005;Wigman et al. 2012b), psychotic parental pathology was associated with a persistence of such experiences from the ages 10 to 16 (Wigman et al. 2012b). Some argued there is no support for the interaction effects between trauma and parental psychopathology on liability to psychosis (Arseneault et al. 2011;Fisher et al. 2014;Wigman et al. 2012b) yet this evidence has not been consistent (Alemany et al. 2011). Likewise, it is not just psychotic parental pathology that showed an association with mild psychotic experiences, but also the level of more general parental psychopathology that is related to wider range of nonclinical psychotic symptoms (Mortensen et al. 2010;Wigman et al. 2012b) as well as the persistence of such symptomatology (Dominguez et al. 2011).

Beside the suggested 'direct' impact that genetics have on development of schizotypy/psychosis proneness, there is a lot of evidence suggesting more indirect influences of genes. Studies showed a familial transmission of psychological distress in community samples (Eley et al. 2003;Rijsdijk et al. 2003) and distress associated with negative and positive psychotic experiences showed a moderate degree of heritability (Jacobs et al. 2005). Furthermore, deficits in self-monitoring[7] (Knoblich et al. 2004) that can lead to thought insertion and auditory hallucinations (Brebion et al. 2005;Ditman and Kuperberg 2005) were also thought to be influenced by familial risk, with unaffected siblings of schizophrenia patients showing worse self-monitoring than healthy controls (Hommes et al. 2011;Johns et al. 2010;Versmissen et al. 2007).

Using a parental psychopathology as a measure for genetic risks is nonspecific (but well validated) approach (van Os et al. 2008). Considering that a vast amount of genetic variations can be used in the G x E model (each with a very small effect), enormous sample sizes would be required to detect interactions between abuse and these genetic variants (Sullivan et al. 2012).

Twin (heritability) studies

Both symptom dimensions (positive and negative) in psychotic illness and attenuated personality-based variants are influenced by familial etiological factors (Fanous et al. 2001). According to Kendler and colleagues (1997) both dimensions lie in the schizophrenia spectrum with a similar magnitude of their familial relationship. The first study that focused on genetic determinants of schizotypal personality disorder (SPD) showed that *28%* of monozygotic twins vs. *3%* of

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[7] *High 'self-monitors' mainly behave in a manner that is highly responsive to social cues in order to impress others. Low 'self-monitors' on the other hand behave consistent with their own internal states (e.g. beliefs, attitudes) not considering the social context. Self-monitoring enables persons to differentiate self-generated actions from externally elicited stimuli (Gangestad and Snyder 2000).*

dizygotic twins of probands with SPD also had this type of personality disorder (Torgersen 1984). But, the study had several limitations, such as small sample size, a lack of structural measures to assess personality disorders and using a sample of index twin patients (Battaglia et al. 1999). Also, there is a need for a more multidimensional approach to SPD assessment in order to examine the genetic (and environmental) determinants of the schizophrenia-spectrum disorders (Battaglia et al. 1999). Kety and colleagues (1994) assessed the prevalence of latent schizophrenia in biological relatives to be at *8.2%* comparing to *2.5%* in the relatives of controls (statistically significant difference). Furthermore, schizotypal personality disorder among nonpsychotic first-degree relatives of schizophrenia was assessed as ranging between *4.2%* and *14.6%* (Tsuang et al. 1999). Building on many limitations of this study, several twin studies followed, focusing on schizotypy from a continuous perspective (avoiding problems with regard to SPD diagnosis criteria) and all reinforced the importance of genetic factors (Claridge and Hewitt 1987;Kendler et al. 1987;Kendler and Hewitt 1992;Torgersen et al. 2000). As these studies used self-report measures, they were limited in reliability of some key SPD traits like oddness or expression of aloofness (which have moderate to high coefficient of heritability (Battaglia et al. 1999)), therefore more credible evidence supporting the genetic relationship between schizophrenia and SPD comes from studies relying on interview measures (Kendler et al. 1996b;Thaker et al. 1993). Mateu and colleagues (2008) advocated that the conclusions about the role of genetics cannot be based on one study but multiple studies using variety of the measures.

Because of the multidimensional nature of schizotypy it is not surprising that beside some common underlying mechanisms *(single latent factors (MacDonald et al. 2001))* each schizotypal dimension is also influenced by its specific genetic and environmental components (Lin et al. 2007;Linney et al. 2003;Reynolds et al. 2000).

Genetic effects specific to interpersonal-affective and disorganised subscales were observed by Ericson and colleagues (2011). Moreover, the positive and negative components of schizotypy are seen as genetically independent, although might both be related to Cognitive Disorganisation (Linney et al. 2003). A longitudinal study (Ericson et al. 2011) reported the continuity and moderate stability of schizotypal traits at the subjects' first assessment *(aged 11)* largely due to stability of genetic impact. However, both genetic and non-shared environmental influences (also in Kendler and Hewitt 1992;Linney et al. 2003) were reported for latent schizotypy and all subscales at the age of 14 to 16. Importantly, this also implies a substantial modification in schizotypal traits and their aetiologies throughout adolescence (Ericson et al. 2011).

Molecular genetic studies

Briefly touching on the possible molecular genetic underpinnings of schizotypy, genetic links are observed between SPD and *fragile X* syndrome (Freund et al. 1992;Reiss et al. 1988;Sobesky et al. 1996) which is also associated with interpersonal and disorganised features (Freund et al. 1992). *Fragile X* syndrome was also linked to neurocognitive impairments in SPD (Sobesky et al. 1996). *COMT* was another gene identified as a link to schizophrenia and schizotypy (Stefanis et al. 2004c), with increased Val allele associated with negative and disorganised schizotypy scores. Also, a continuum of developmental disruption in *22q11* deletion syndrome has been linked to higher schizotypy as well as declining mental health in early adulthood (Baker and Skuse 2005). Beside the clinical data on neurophysiological and cognitive deficits as endophenotypes of the schizophrenia (Braff and Light 2005), there is also research on personality traits supporting the notion of genetic vulnerabilities to schizophrenia (Bora and Veznedaroglu 2007;Kurs et al. 2005;Ritsner and Susser 2004;Szoke et al. 2002;Van Ammers et al. 1997). As

such, the *ZNF804A* gene, for example, displayed an association with schizotypal personality traits, especially the disorganised subscale (Yasuda et al. 2011). However, this results need to be considered cautiously, until they have been replicated in independent sample. Also, the effect of *DTNBP1* genotype (gene-modulation) on schizophrenia endophenotypes was observed at the population level (Kircher et al. 2009), particularly for the negative symptomatology (Fanous et al. 2005). Different etiological mechanisms for distinct schizotypy factors were advocated by Reynolds and colleagues (Reynolds et al. 2000) e.g. *Neuregulin1* gene polymorphisms were associated with Perceptual Aberration Scale scores but not with scores on three dimensions of the Schizotypal Personality Scale (Lin et al. 2005).

However, genetic analyses have suggested the best fitting model to be additive genetic and environmental factors that affect all three schizotypy dimensions (Cardno et al. 1999;Hay et al. 2001;Kendler and Hewitt 1992;Linney et al. 2003;MacDonald et al. 2001). Also according to Meehl (1962), a subject inherits 'schizotaxia' and may develop a syndrome (sitting on a continuum between schizotypy and schizophrenia) depending on the environmental risks the person is exposed to. Again, this supports the continuum model of psychosis by indicating that schizophrenia and schizotypy have related aetiologies (Gottesman and Shields 1967). A recent study (Alemany et al. 2011) concluded that childhood adversity had an independent effect on positive and negative psychotic-like experiences, with the *BDNF-Val66Met* polymorphism showing only a 'moderation effect' between trauma and later psychosis. An especially robust association between childhood trauma and schizotypy dimensions in subjects with genetic vulnerabilities for schizophrenia further suggests that susceptibility genes may interact with environmental factors (Rutter et al. 1999;Tsuang et al. 2001;van Os et al. 2008;van Winkel et al. 2008) to

induce schizotypal traits (especially positive psychotic-like symptoms) (Schurhoff et al. 2009).

Most importantly, genetic risk is a powerful factor in predicting the development of schizophrenia (Gottesman and Shields 1982;Kallmann 1946), but the misidentification of using the criteria on its own is between *60%* and *80%*, implying the significance of inclusion of multiple variables (and their interaction effects) to increase the predictive power (Carter et al. 2002).

In summary: Overall, studies have demonstrated that schizotypy is moderately heritable. The strongest association was shown for negative schizotypy (believed to be a core feature) but elevated scores on positive and disorganised schizotypy were also evident among relatives of individuals with schizophrenia. The reports on magnitude of familial influences vary greatly depending on study designs and the measures utilised. The molecular genetic analyses have identified a few specific genes that may be related to schizotypy and schizophrenia but these require replication in independent samples. Given that heritability estimates usually fall well short of 100%, it is likely that the aetiology of schizotypy involves both genetic and environmental factors.

CHAPTER 2
Literature Review: Childhood Trauma and Schizotypy

Chapter 2 Literature Review – Childhood Trauma and Schizotypy

Main aims of the chapter

- To explore the empirical literature available on childhood trauma and schizotypy (including psychosis-like symptoms) association *(section 2.1 & 2.2)*;

- To describe the main methodological limitations of previous studies measuring the childhood trauma and schizotypy association *(section 2.1.5)*;

- To investigate the main issues about reliability and validity of the retrospective instruments measuring childhood trauma *(section 2.1.5.1)*;

- To explore the possible pathways that account for the childhood trauma – schizotypy association *(section 2.3).*

2.1 *Childhood trauma and schizotypy association*

This section presents a systematic review of the empirical literature exploring the association between childhood trauma and schizotypy. It also covers several methodological limitations of these studies (including the concerns with regard to reliability and validity of retrospective measures). Overall, it explores the existing evidence of the trauma-schizotypy link and identifies gaps in the literature – the reasoning behind this thesis.

Historical perspective

In the late nineteenth and the early twentieth centuries schizophrenia was considered to be a fully inherited neurodegenerative disease. In the 1950s and 1960s new influential theories emerged. They attributed schizophrenia to being brought up in disturbed families either due to the 'double-bind' interactions in the family (between parents and a child in which the child 'loses' regardless of what he/she does) (Bateson et al. 1956) or 'schizophrenogenic' mothers (Fromm-Reichmann 1950). This search for environmental factors predisposing to schizophrenia gave rise to a lot of criticism from carers' organisations and ultimately rejection of these theories by psychiatrists. This led to the reestablishment of biological psychiatry, further reinforced by the third edition of the DSM (DSM-III) (American Psychiatric Assocation 1980). Bentall (2006) identified various impediments to understanding the aetiological role of the environment in the development of psychosis (or other disorders): overreliance on often meaningless diagnoses, misunderstanding of genetic evidence (no consideration of the gene-environmental interactions) and several ideological and economic interests encouraging the dominance of the biological underpinnings of psychosis.

Despite biological theories having a domineering role through the history of schizophrenia research, the idea that it is not only heredity but psychological and social factors that influence the development of the disorder is not new. In 1845, a

French psychiatrist *Esquirol* documented the importance of adverse events that precipitate illness e.g. domestic problems, disappointments in love (Gelder 1996). Later theories focused mainly on families, especially the parent-child relationship, causing traumatic incidence or *'memory of the trauma'* (Schofield and Balian 1959). Studies showed that individuals who later developed schizophrenia were either coming from 'broken homes', parents were more likely to be mentally ill, rejecting or cruel (Frazee 1953;Wahl 1956), or they had experienced parental death (Barry 1936;Blum and Rosenzweig 1944) or even a death of a sibling (Rosenzweig and Bray 1943).

In the late 1990s a paradigm shift led to an extensive growth of the literature exploring the role of the early childhood environment in the aetiology of psychosis. This area of research extended the role of difficult family interactions to more specific adversities (e.g. physical abuse, sexual abuse) and provided a lot of support to the childhood trauma – psychosis association (Read et al. 2005). These findings however need to be considered in light of many methodological limitations (e.g. Morgan and Fisher 2007).

2.1.1 Introduction to the Childhood Trauma and Schizotypy association

Childhood maltreatment has been previously linked to many disorders including depression (Bifulco et al. 1991;Bifulco et al. 1998;Duncan et al. 1996;MacMillan et al. 2001), anxiety and other mood disorders (Kessler et al. 1997;Swanston et al. 2003), substance misuse (Jonas et al. 2011;Kendler et al. 2000;MacMillan et al. 2001;Spak et al. 1997), eating disorders (Grilo and Masheb 2002;Jonas et al. 2011;Rorty et al. 1994;van Gerko et al. 2005), personality disorders (Afifi et al. 2011;Grover et al. 2007;Lobbestael et al. 2010;Spataro et al. 2004), post-traumatic stress disorder (PTSD) (Duncan et al. 1996;Gearon et al. 2003;Neria et al.

2002;Vranceanu et al. 2007), dissociative disorders (Carrion and Steiner 2000;De Zulueta 2002;Sar et al. 2007), depersonalisation disorder (Simeon et al. 2001) sexual disturbances (Beitchman et al. 1991;Fleming et al. 1999) as well as suicidal ideation (Afifi et al. 2009;Blaauw et al. 2002). However, research has now also demonstrated a robust association with psychosis and schizophrenia-spectrum disorders (Varese et al. 2012b).

Despite the fact that schizotypy is reported to be a phenotypic expression of the genetic vulnerability to schizophrenia, the relationship with full blown psychosis still needs to be clarified (Mata et al. 2000). As previously discussed, the expression of inherited 'schizotaxia' (Meehl 1962) is postulated to greatly depend upon the environment to which the individual is exposed, and can vary from schizotypy to schizophrenia (Mata et al. 2005). There is now a growing body of evidence supporting the association between childhood trauma and increased schizotypy load (Berenbaum et al. 2003;Johnson et al. 2001;Myin-Germeys et al. 2011;Steel et al. 2009). Multiple studies found that increased childhood trauma is experienced by a greater number of schizotypal individuals in comparison with controls (Afifi et al. 2011;Berenbaum et al. 2003;Campbell and Morrison 2007;Lentz et al. 2010;Lobbestael et al. 2010;Myin-Germeys et al. 2011;Raine et al. 2011;Schurhoff et al. 2009;Sommer et al. 2010;Steel et al. 2009). Likewise, general population studies on adolescent and adult populations observed an association between childhood trauma and subclinical psychotic-like experiences (Berenbaum 1999;Campbell and Morrison 2007;Gracie et al. 2007;Janssen et al. 2004;Lataster et al. 2006;Shevlin et al. 2007b), complementing associations found between childhood trauma and psychotic disorders (Bendall et al. 2008;Houston et al. 2011;Morgan and Fisher 2007;Shevlin et al. 2007b).

Observations have shown that this association could not be fully accounted for by parental psychopathology alone (genetic vulnerability) (Fisher et al.

2014;Johnson et al. 1999; 2000). Nevertheless it is important to note that parental psychopathology does not necessarily suggest just genetic links but also reflects other associated psychosocial variables. A high percentage of parental psychosis and depression was noted in severely abused children (Taylor et al. 1991), but abusive parents in comparison to non-abusive parents also displayed lower self-esteem, poorer coping skills, more hostility and impulsivity etc. (Friedrich and Wheeler 1982;Taylor et al. 1991). Moreover, marital discord (Rutter and Quinton 1984), low socio-economic status (Whipple and Webster-Stratton 1991), poor parent-child relationship (Fergusson et al. 1996) and other social factors such as parental substance abuse (Kelleher et al. 1994) observed in such families are potentially acting as confounders of abuse-schizotypy associations.

Overall, the strongest association between childhood trauma and schizotypal traits was among those with pre-existing vulnerability to psychosis (susceptibility genes), suggesting the interaction between genes and childhood trauma on the expression of schizotypal traits, mainly positive traits (Schurhoff et al. 2007;Thompson et al. 2009) and less clearly (Schenkel et al. 2005) or with no effect (Lysaker et al. 2001;Read et al. 2003;Resnick et al. 2003) for the negative traits. This further supports that the childhood trauma – schizotypy association might be specific for people with a psychosis family history, along with the study that found no link between childhood trauma and schizotypy dimension in bipolar- and major depression pedigrees (Schurhoff et al. 2009).

The importance of exploring the relationship between childhood trauma and schizotypy lays in the clues it can provide in the aetiology of psychotic-like symptoms and accordingly psychotic disorders. Mason and colleagues (2004) reported that the most reliable predictor of psychosis in a group of individuals considered 'at-risk' was the levels of schizotypal traits. Also, other studies of an ultra-high risk (prodromal) group (Bechdolf et al. 2010) and a cross-sectional

population survey (Bebbington et al. 2004) reported that a history of sexual trauma not only demonstrated the largest relative risk for psychosis-like symptoms but also predicted transition to psychotic disorder. Schizotypy allows the exploration of nonclinical schizophrenic psychopathology without the effects of any comorbid factor usually associated with research using clinical samples (e.g. the effects of the medication, hospitalisation, stigma etc.). Most importantly, a better understanding of the childhood trauma-schizotypy link could have substantial implications for clinical assessment and treatment formulation. For example, psychotic patients who reported childhood abuse have also experienced earlier hospital admission, higher symptom severity and longer hospitalisations (Goff et al. 1991;Mullen et al. 1993;Read 1998).

Despite the high significance of this research and the increasing number of studies exploring the association between childhood trauma and schizotypal traits, the evidence has not yet been systematically synthesised and assessed. Recently a meta-analysis (Varese et al. 2012b) demonstrated an overall association between childhood trauma and both psychosis-like symptoms and psychotic disorders, but it is unclear whether similar associations can be observed for schizotypy. The systematic review of the available empirical literature *(see Table 3)* presented here therefore examines the association between childhood trauma and schizotypy symptom load mainly in community samples but also in some clinical cases (schizotypal personality disorders). Multiple types of trauma (abuse, neglect, bullying and parental loss or separation) are included in order to assess their possibly differential effects on schizotypy dimensions which can provide further information about some underlying mechanisms that support the childhood trauma-schizotypy association.

2.1.2　　　Empirical literature search strategy

A literature search was conducted in the following databases: *PsycInfo*, *PubMed*, *EMBASE* and *Web of Science*, where two sets of search terms were used. In addition to key words search, only the *PsycInfo* database offers an option to use *MeSH[8] Terms (Medical Subject Headings)* beside the key words, which helped to broaden the search further. The following sets of keywords were used:

1. *trauma** OR *maltreat** OR *abuse* OR *advers** OR *neglect OR bully** OR *victim** OR *parental loss* OR *separat** AND *adolescen** OR *child**

AND

2. *schizoty** OR *psychos** OR *psychotic* OR *illusion* OR *hallucination* OR *delusion* OR *derealisation* OR *depersonalisation* OR *social isolation* OR *hypersensitivity* OR *magical ideation* OR *introversion* OR *referential thinking* OR *suspiciousness* OR *restricted affect*.

The search covered the articles from 1806 to 1st March 2013 and resulted in *17,003* articles in total. After transferring the data into Reference Manager and extracting the duplicates, *13,050* articles were identified for the title screening. Title screening yielded *801* potentially relevant articles, which were narrowed down to *311* after further abstract screening. Through the full-text screening of the remaining articles *25* relevant studies were identified which are used in this analysis. The reasons for excluding articles at each stage are documented in *Figure 2.*

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[8] *MeSH is a controlled vocabulary thesaurus that detects citations, even when authors use different terms for the same concept (National Library of Medicine 2014)*
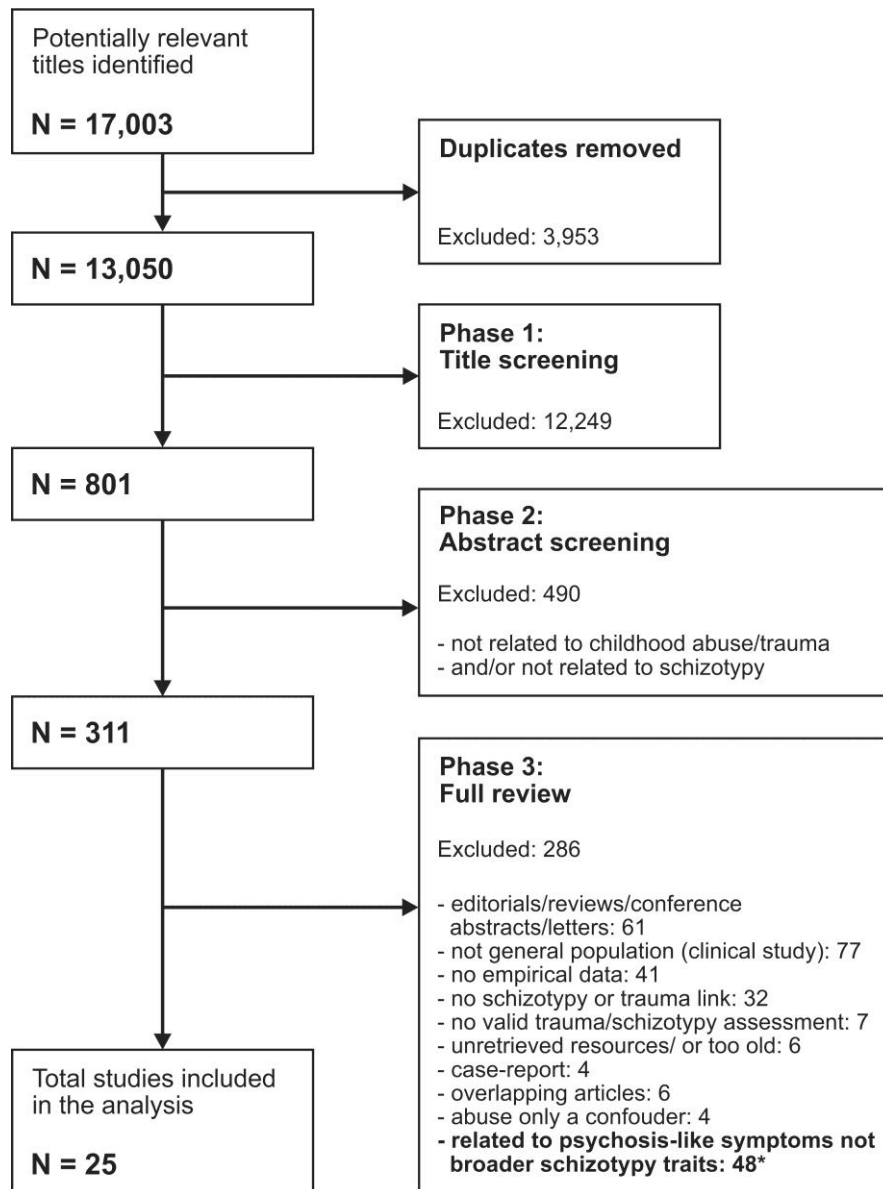
Figure 2: Flowchart of studies included in the literature review.

The section corresponds to the next thesis chapter, exploring the association between childhood trauma and psychotic-like symptoms (Section 2.2)

Inclusion and exclusion criteria

For papers to be included they had to meet the following criteria: (a) an original research paper (including replication studies); (b) written in English language; (c) use a measure of childhood trauma (either emotional, physical or sexual abuse, neglect, bullying, separation from parents or parental loss, or other traumatic experiences e.g. household discord, life- or an injury-threatening event among others) before the age of 18 *(any standardised or non-standardised measure)*; (d)

test whether there is an association with schizotypal traits *(any standardised or non-standardised measure, assessing either single schizotypal trait or multidimensional schizotypy; and any statistical analysis, adjusted or unadjusted statistics)*; (e) use general population/community samples in their studies and not clinical (psychotic) cases, with the exception of schizotypal personality disorders (due to a limited number of non-clinical studies involving schizotypy traits); and (f) include complete information on design and measures used in the study to allow completion of a quality assessment tool designed for this analysis (e.g. exclusion of conference abstracts due to limited information).

Quality assessment tool

Full criteria and scoring for the quality assessment tool are provided in *Appendix I.* This tool was adapted by the author of this thesis from the measure utilised by Beards and colleagues (2013). Briefly, the quality indicators assessed included method of sample selection, the percentage of individuals approached who agreed to participate, the size of the sample, the type of assessment tool used to ascertain a history of childhood trauma and the presence of schizotypal traits, whether different types of trauma were considered separately in the analysis, and whether analyses were adjusted for potentially confounding factors (demographic information and other risk factors such as genetic risk, substance use, depression etc). Each article was assigned a score of 0, 1 or 2 points for each item with a maximum possible score of 14.

2.1.3 Results

Following the inclusion criteria, 25 articles were identified and included in this analysis. These papers are presented in Table 3 in order of the score assigned based on the quality assessment tool.

Table 3: Summary of studies on childhood trauma and schizotypal traits (ordered by quality score) (All abbreviations are listed at the bottom of the table)

Authors	Study Design	Sample Recruited (Age)	% female	Measure of Trauma	Number Exposed	Measure of Schizotypy	Number with the Outcome	Measure of Effect	Quality Score
Afifi et al. (2011) (Canada)	Cross-sectional Representative population-based study	N=34653 NESARC (Above 20 years)	-	Adverse Childhood Experience study (see Dube et al. 2003) and Childhood Trauma Questionnaire (Bernstein et al. 1994) - 5-point scale, Household dysfunction assessed	30% experiences child abuse and/or neglect 40% experienced household dysfunction 52% experienced any childhood adversity	Alcohol Use Disorder and associated disabilities Interview Schedule-Diagnostic and Statistical Manual of mental disorder-fourth edition (Grant et al. 2001)	-	Physical abuse vs no abuse and SPD (OR=1.62, 99% CI=1.28-2.03, p<0.01) Emotional abuse (OR=1.76, 99% CI=1.35-2.31, p<0.01) Sexual abuse (OR=2.05, 99% CI=1.59-2.65, p<0.01) Physical neglect (OR=1.61, 99% CI=1.26-2.05, p<0.01) Emotional neglect (OR=1.35, 99% CI=1.05-1.74, p<0.01)	12
Lentz et al. (2010) (USA)	Cross-sectional Representative population-based study	N=34653 (Above 20 years)	For SPD group only 48.3%	5 childhood events prior to the age of 16 (physical abuse by parent/caretaker; physical abuse by someone other than a parent, witnessing violence at home, neglect by parent/caretaker, sexual assault)	In SPD group Physical abuse by parent/caretaker (N=214, 12.4%) Witnessing violence at home (N=411, 25.1%) Neglect by parent/caretaker (N=203, 12.0%) Sexual assault (N=328, 20.7%)	DSM-IV diagnoses were made using the Alcohol Use Disorders Associated disabilities Interview Schedule IV (AUDADIS-IV) (Grant et al. 2001) Schizophrenia and psychotic episodes by asking participants if they had been previously diagnosed	SPD (N=1534)	Physical abuse by parent/caretaker vs no abuse and SPD (adj.* OR=4.43, 95% CI=3.64-5.40, p<0.001) Witnessing violence at home and SPD (adj.* OR=3.10, 95% CI= 2.65-3.61, p<0.001) Neglect by parent/caretaker and SPD (adj.* OR=4.57, 95% CI= 3.69-5.67, p<0.001) Sexual assault and SPD (adj.* OR=4.15, 95% CI=3.94-5.16, p<0.001) *age, gender, race, marital status, income*	12
Powers et al. (2011) (USA)	Cross-sectional General population study	N=541 (≥18 Median=41)	59.0%	Self-report Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1994) Early Trauma Interview (Bremner et al. 2000)	Adult trauma (40.2%) Childhood trauma (29.8%) Childhood & adult trauma (30%)	The Schedule for Nonadaptive and Adaptive Personality (SNAP) (Clark 1993) - self report 375 true-false items (12 traits scales, 3 temperament scales, 6 validity scales, 13 PD scales) The Personality Disorder Diagnostic scales (see Trull 2005)	-	Childhood physical and emotional abuse correlated with SPD (r=0.15, p<0.001) Physical abuse correlated with unusual perceptions (r=0.11, p<0.01) eccentric behaviour (r=0.15, p<0.001) and social anxiety (r=0.12, p<0.001) Emotional abuse predicted 5 of 8 SPD symptoms when looking at both childhood trauma measures: ideas of reference, excessive social anxiety, a lack of close friends and confidants, unusual perceptual experiences, and eccentric behaviour or appearance Sexual abuse was correlated to eccentric behaviour (r=0.15, p<0.001)	10

Table 3 Cont'd

Lobbestael et al. (2010) (Netherlands)	Cross-sectional Case-control study	N=409 (18-61 Mean=33.54 SD=10.65)	64.0%	Interview for Traumatic Events in Childhood (ITEC) (Lobbestael et al. 2006): sexual, physical and emotional abuse, physical neglect, emotional neglect	87% history of any type of maltreatment	The Structured Clinical Interview for DSM-IV Axis I and II disorders (First et al. 1994;First et al. 1997)	N=250 with Axis II diagnosis	SPD and sexual abuse (ρ=0.19, p<0.01) SPD and physical abuse (ρ=0.25, p<0.01) SPD and emotional abuse (ρ=0.29, p<0.01) SPD and emotional neglect (ρ=0.21, p<0.01) SPD and physical neglect (ρ=0.11, p<0.01) When looking at unique effects for each type of the maltreatment, SPD and emotional abuse remained significant (β=0.24, p<0.01)	10
Berenbaum et al. (2008) – Study 1 (USA)	Cross-sectional Representative sample from general population	N=1510 (18-95 Mean=44.2 SD=18.1)	52.1%	Telephone interview Physical abuse before 18 (7 items used Sexual abuse, 7 acts Emotional abuse, 2 questions Physical neglect before 12, 8 items Threatening events), adapted from different instruments	46.7% experienced life threatening events	Telephone interview Schizotypal personality disorder – five out of nine subscales SPQ (Raine 1991) (Odd beliefs, Magical thinking, Ideas of reference, Unusual perceptual experiences, Suspiciousness.	-	Individuals who experienced a life- or an injury-threatening event had higher levels of schizotypal symptoms (men: t(684)=4.41, p<0.01; women: t(750)=4.79, p<0.01) Childhood maltreatment independently contributed to the prediction of schizotypal symptoms (for men:β=0.33, p<0.01 and for women:β=0.29, p<0.01)	10
Berenbaum et al. (2008) – Study 2 (USA)	Cross-sectional Representative sample from general population	N=303 (18-89 Mean=43.2 SD=17.6)	53.1%	Physical abuse (modified version of Self-Report of Childhood Abuse Physical) (see Widom and Shepard 1996) Sexual abuse as used in Widom and Morris (1997), Emotional abuse & Physical neglect (relevant portion of Childhood trauma Interview) (Bernstein et al. 1994) + frequency, age of occurrence and perpetrator	-	Schizotypal, Antisocial and Borderline PD (Personality Disorder Interview-IV) (Widiger et al. 1995)	-	Levels of schizotypal symptoms were associated with higher levels of childhood maltreatment more in men than women (z=2.24, p<0.05) Based on regression analysis only emotional abused sign. associated with schizotypal symptoms (β=0.28, p<0.01) For men childhood maltreatment (β=0.46, p<0.01) contributed independently to the prediction of schizotypal symptoms; for women maltreatment (β=0.19, p<0.05) and PTSD (β=0.23, p<0.01) contributed independently	10
Rossler et al. (2007) (Switzerland)	Prospective study Representative sample from general population	N=372 (20/21 follow up at 23,28,30,35 and 41)	-	Life events list according to Holmes/Rahe scale (Holmes and Rahe 1967)	-	SPIKE (Angst et al. 1984); Symptoms Checklist (SCL90-R) (Derogatis 1977) - including paranoid ideation and psychoticism	-	Parental neglect (OR=6.7, 95% CI=2.8-16.3, p<0.001) Conflict among parent(OR=3.5, 95% CI=1.6-7.9, p=0.002); Having been punished more severely than other children (OR=3.1, 95% CI=1.3-7.8, p=0.012) and high 'schizotypal signs'	10

Table 3 Cont'd

Battle et al. (2004) (USA)	Longitudinal study Case-control study	N=517 with PD N=83 with MDD and no PD (18-45)	63.0%	Childhood Experiences Questionnaire – Revised (CEQ-R) (Zanarini et al. 1989) – before age of 18	In SPD only: Caretaker's emotional abuse (N=45, 54%) Caretaker's verbal abuse (N=55, 66%) Caretaker's physical abuse (N=40, 48%) Caretaker's sexual abuse (N=9, 11%) Any neglect (N=71, 85%)	PD assessed by Structured Clinical Interview for DSM-IV Axis I Disorders (Patient Version) (First et al. 1996)	SPD (N=84)	SPD vs MDD Caretaker's emotional abuse ($\chi^2(1)$=15.74, p<0.001) Caretaker's verbal abuse (χ^2 (1)=8.26, p<0.004) Caretaker's physical abuse (χ^2 (1)=8.62, p<0.003) Caretaker's sexual abuse (χ^2 (1)=5.85, p<0.016) Any neglect (χ^2 (1)=9.76, p<0.02)	10
Johnson et al. (1999) (USA) and Johnson et al. (2001) (USA)	Longitudinal study Representative sample from general population	N=793 mothers and their offspring (In 1975 – Mean= 5.6 SD=2.8) (In 1983- Mean= 13.7 SD=2.7) (In 1985- Mean= 16.3 SD=2.8) (In 1991 – Mean= 22.1 SD=2.7)	Offspring 49.2%	Official data on childhood maltreatment obtained from New York State Central registry And self-reports (yes/no responses)	N=31 (4.9%) documented cases exposed to childhood maltreatment N=58 (9.1%) self-reported childhood maltreatment	Diagnostic Interview Schedule for Children (DISC-I) (Costello et al. 1984), Personality Diagnostic Questionnaire (Hyler et al. 1988)	SPD prevalence among individuals with no abuse is 24 (3.4%) vs verbal abuse is 6 (7.7%)	Childhood abuse/neglect and elevated symptom levels of SPD (F(1,637)=26.44, p<0.005) *Adj. offspring age, parental education, parental psychiatric disorder, physical abuse, sexual abuse*	10
Myin-Germeys et al. (2011) (Netherlands)	Cross-sectional Case-control study	Patients (N= 272) (16-55 Mean=28.1 SD=8.2) Controls (N=227) (16-55 Mean=32.3 SD=11.5)	Patients 30.6% Controls 69.7%	Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1997)	High trauma (scoring above 4th quartile) In patients N=155 (57%) In controls N=61 (27%)	Positive and Negative syndrome scale and Structured Interview for Schizotypy-revised (Vollema and Ormel 2000)	-	Healthy comparison group: Trauma/any vs no trauma and positive schizotypy (OR=4.82, 95% CI=2.04-11.39, p<0.001) Abuse and positive schizotypy (OR=5.53, 95% CI=2.15-13.29, p<0.001) Neglect and positive schizotypy (OR=3.67, 95% CI=1.60-8.41, p<0.001)	8
Anglin et al. (2008) (USA)	Longitudinal, Random sample from general population	N=776 (T0 Mean=5 T1 Mean=16.3 T2 Mean=33.1)	49.0%	Maternal separation for at least one month – reported by mothers	-	Self- report schizotypal personality disorder symptom scale (Crawford et al. 2005)	-	Separation before age 5 and average SPD symptoms (b=2.03, SE=1.05, p<0.05)	8

Table 3 Cont'd

Sommer et al. (2010) (The Netherlands)	Cross-sectional Case-control study	N=103 with AVH (9-64, Mean= 44, SD=14) N=60 without AVH (19-60, Mean= 46, SD=15)	With AVH (70.9%) Without AVH (70.0%)	Childhood Trauma Questionnaire (CTQ) (Gregory et al. 2005)	-	Auditory Hallucinations Rating scale Schizotypal Personality (Haddock et al. 1999) Structured Clinical Interview for Personality Disorder (SCID)-II (First et al. 1995)	-	Trauma significantly correlated to total scores on Delusional tendency inventory (*r*=0.3, p=0.002) and Schizotypal personality questionnaire (*r*=0.54, p=0.001) Subjects with AVH reported higher schizotypy than without AVH (t=(162)=12.8, p=0.001)	7
Tyrka et al. (2009) (USA)	Cross-sectional Case-control study	N=105 (18-64 Mean=32.6 SD=12.1)	64.8%	Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1994)	70 (66.7%) considered to have maltreatment moderate to severe level (on one or more categories)	Structured Clinical Interview for DSM-IV I and II (First et al. 1997;First et al. 2002)	-	Emotional abuse/neglect group differed from No abuse for Cluster A PD (and B,C) (U test, df=67,p=0.007) Physical/Sexual differed from control group for clusters A PD (and B,C) (U test, df=73, p=0.001) Two maltreatment groups did not differ from each other for each of the clusters	7
Gibb et al. (2001) (USA)	Cross-sectional Participants based on high and low scores in cognitive-vulnerability to depression project	N=272 (Mean 18.92 SD=1.92)	68.0%	Childhood and adolescent maltreatment – the Life Experiences Questionnaire (Rose et al. 2001) (emotional, physical and sexual abuse)	-	Personality Disorder Examination (Loranger 1988)	-	Paranoid PD dimensional scores were positively related to adolescent physical maltreatment (t(263)=2.93, p<0.001, β=0.33) SPD dimensional scores were related to adolescent emotional maltreatment (t(263) =3.38, p<0.001, β=0.43)	7
Irwin et al. (2001) (Australia)	Cross-sectional Convenience sample - community population	N=116 (18-46 Mean=22.7 SD=7.36)	63.9%	Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1994) – 60 items	-	Schizotypal Personality Questionnaire – Brief (SPQ-B) (Raine and Benishay 1995) Dissociative Experiences Scale (Bernstein and Putnam 1986)	-	'Cognitive perceptual' correlated with physical/emotional abuse (r=0.44, p<0.01), emotional neglect (r=0.36, p<0.001), physical neglect (r=0.34, p<0.001), sexual abuse (r=0.21, p<0.05) 'Interpersonal perceptual' correlated with physical/emotional abuse (r=0.28, p<0.01), emotional neglect (r=0.28, p<0.01), physical neglect (r=0.26, p<0.01) 'Disorganized' correlated with physical/emotional abuse (r=0.39, p<0.001), emotional neglect (r=0.32, p<0.001), physical neglect (r=0.31, p<0.001), sexual abuse (r=0.20, p<0.05)	7

Table 3 Cont'd

Berenbaum (1999) (USA)	Cross-sectional Random community sample	N=458 college students (-)	-	Physical Punishment Scale (Berger et al. 1988) Sexual Abuse Scale (DiTomasso and Routh 1993) Childhood Experiences Questionnaire (Berenbaum and James 1994)	-	Perceptual Aberration (Chapman et al. 1978) Magical Ideation scales (Eckblad and Chapman 1983)	-	Participants who reported a history of childhood maltreatment were 10.5 times more likely to have deviantly high perceptual aberration scores	7
Schürhoff et al. (2009) (France)	Cross-sectional Unaffected first degree relatives of schizophr./ bipolar probands	N=138 unaffected first-degree relatives of schizophr. probands (Mean=54.2 SD=15.4) and bipolar probands (Mean=53.1 SD=15)	Relatives of schizophr. probands 53.7% Relatives of bipolar probands 47.9%	French translated (see Paquette et al. 2004) Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1994)	-	French translated (see Paquette et al. 2004) Self-rating Schizotypal Personality Questionnaire (SPQ) (Raine 1991) -74-item (yes/no)	-	Positive correlation between childhood trauma and scores and schizotypal scores in first-degree relatives of schizophrenic subjects (r=0.27, p<0.004) but not bipolar Especially strong association with positive dimensions and schizotypy in first degree relatives of schizophrenic subjects (r=0.41, p<0.004)	6
Steel et al. (2009) (UK)	Cross-sectional Convenience sample – University students/ staff	N=348 (18-67 Mean=24.9 SD=7.2)	76.0%	The Traumatic Life Events Questionnaire (TLEQ)(Kubany et al. 2000) (Yes/No format)	Sexual harassment (45.3%) Serious accident/illness (44%) Physical abuse (14%) Emotional abuse (21.1%) Child sexual abuse (19.3%)	Schizotypal Personality Scale (Claridge and Broks 1984), 37-forced choice items	-	Physical abuse alone associated with paranoia (OR=5.84, 95% CI=1.5-23.1, p<0.05) And unusual perceptual experiences (OR=6.46, 95% CI= 1.1-36.9, p<0.05) Child sexual abuse with paranoia (OR=4.49, 95% CI= 1.7-12.2, p<0.01) And unusual perceptual experiences (OR=4.00, 95% CI= 1.0-15.2, p<0.05 *All adj. age, gender, anxiety, depression, number of traumas*	6
Berenbaum (2003) (USA)	Cross-sectional Individuals with unusual beliefs	N=75 (18–41 Mean=38.7 SD=3.7)	100%	Posttraumatic diagnostic scale (Foa et al. 1997) Childhood Trauma Questionnaire (Bernstein et al. 1994)(physical/sexual abuse) Self-Report of Childhood Abuse (Widom and Shepard 1996) (physical abuse interview) Sexual abuse (participants asked about 10 specific sexual acts)	-	Schizotypal personality Questionnaire (SPQ) (Raine 1991) 74-item true/false self-report Structure Interview for DSM-IV Personality (SIDP) (Pfohl et al. 1995)	n/a	Schizotypal symptoms associated with all measures of childhood abuse (physical r=0.40, p<0.01), (sexual r=0.32, p<0.01), (emotional r=0.47, p<0.01), (neglect r=0.60, p<0.01) Psychological dysfunction partial mediator between neglect and schizotypal personality – adjusted (r=0.54, p<0.01)	6

Table 3 Cont'd

Raine et al. (2011) (Hong Kong) *also in Fung and Raine (2012)*	Cross-sectional General population study	N=3804 (6-18)	44.1%	Multidimensional Peer Victimization Scale (Mynard and Joseph 2000) (physical victimisation, social manipulation, verbal victimisation)	-	Schizotypal Personality Questionnaire – Child (SPQ-C) (extension of SPQ-B) (Raine and Benishay 1995)	-	Correlation between victimisation and total SPQ (r=0.39, p<0.001), victimisation and SPQ-interpersonal (r=0.29, p<0.001), SPQ-disorganized (r=0.30, p<0.001) and SPQ-cognitive-perceptual (r=0.39, p<0.001)	5
Berry et al. (2007) (UK)	Cross-sectional Convenience sample from general population	N=304 university students (18-53 Median=21)	78.0%	Trauma History Questionnaire Psychosis Attachment Measure (PAM) (Bartholomew and Horowitz 1991) Parental Bonding Instrument (Parker et al. 1979) Attachment history questionnaire (Pottharst 1990)	N=110	Oxford-Liverpool Inventory of Feelings and Experiences scale (O-LIFE) (Mason et al. 1995)	-	Early interpersonal experiences and adult attachment style predicted schizotypy Total distress rating for interpersonal trauma and Unusual experiences (r=0.33, p<0.001), Cognitive disorganisation (r=0.22, not sign.), Introvertive anhedonia (r=0.21, not sign.)	4
Startup et al. (1999) (UK)	Cross-sectional Convenience sample of general population	N= 224 (Mean 39.1 SD=18.5)	64.3%	Two questions (Yes/no format) used in Bryer et al. (1987) study to assess childhood abuse, one question for sexual abuse one for physical	14 (6.2%) reported sexual abuse 37 (16.5%) physical abuse 10 (4.5%) both kinds of abuse	Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE) (Mason et al. 1995) and Dissociative Experience Scale (Carlson and Putman 1993) (for frequency of auditory hallucination)	-	Childhood abuse accounted for a significant proportion (4%) of the variance in the Unusual experiences (R^2 change=0.04, F(3,215)=3.4, p<0.02), no effects on cognitive disorganisation	4
Perkins and Allen (2006) (USA)	Cross-sectional Convenience sample – University students	N=107 (18-53 Mean=23.03 SD=6.61)	66.3%	The Assessing Environments III Questionnaire (Berger and Knutson 1984) – intrafamiliar childhood trauma events Physical Punishment subscale (PP) (see Knutson and Selner 1994)	-	The Tobacyk Revised Paranormal Brief scale (PBS) (Tobacyk 1988)	Mean score 101.8, range=32-153	No abuse vs high abuse and higher precognition (t=-2.05, p=0.045), spiritualism beliefs (t=-4.40, p<0.001), witchcraft (t=-5.80, p=0.015), lower superstition (t=5.70, p<0.001)	3
Merckelbach and Jelicic (2004) (Netherlands)	Cross-sectional Convenience sample - Undergraduate students	N=127 (19-25 Mean=21.7 SD=2.3)	72.4%	Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1994)	-	Dissociative Experiences Questionnaire (DES) (Bernstein and Putnam 1986) – only pathological forms subset Creative Experiences Questionnaire	Mean score on DES=31.4	Fantasy Proneness and self-reported trauma (ρ=0.25, p<0.05) Correlation dissociation and self-reported trauma (ρ=0.44, p<0.05)	2

Table 3 Cont'd

Mynard and Joseph (1997) (UK)	Cross-sectional Convenience sample	N=179 (8-13 Mean=10.7 SD=1.5)	58.1%	6 item Bullying Behaviour Scale 6 item Peer Victimization Scale (Austin and Joseph 1996)	Bullies (11%) Victims (22%) Bully/Victims (18%)	Extraversion Neurotocism, Psychoticism, Lie Scale Junior Eysenck Personality Questionnaire (Eysenck and Eysenck 1975)	-	Positive association between bullying behaviour scale and psychoticism (r=0.31, p<0.01) Bully & victims had the highest score of psychoticism (mean=3.47, not involved mean=2.03, p<0.05)	2

Note: NESARC, National Epidemiologic Survey on Alcohol and Related Conditions. OR, odds ratio. CI, 95% confidence interval. SPD, Schizotypal Personality Disorder. adj., adjusted. PD, Personality Disorder. SPQ, Schizotypal Personality Questionnaire. r, Pearson correlation coefficient. DSM-IV, Diagnostic and Statistical Manual of Mental Disorders 4th ed. ρ, Spearman'r rank correlation coefficient. t, T-test. β, Beta regression coefficient. z, Kruskal Walllis Test. PTSD, Posttraumatic Stress Disorder. MDD, major depression. χ2, Chi-squared test. F, F-ratio. T0, baseline. T1, first measure after T0 (longitudinal study). T2, second measure after T0 (longitudinal study). b, regression coefficient. SE, standard error. AVH, Auditory Verbal Hallucinations. df, degrees of freedom. R, multiple correlation coefficient. U-test, Mann Whitney U test. sign., statistically significant. SPIKE, Semi-structured psychopathological interview.

Twenty-five studies exploring the relationship between childhood trauma and schizotypal traits were examined and all provided support for a positive association, especially the more severe traumatic experiences which predicted schizotypal symptomatology in a dose-response manner (Berenbaum et al. 2008;Myin-Germeys et al. 2011). Overall, the adjusted odds ratio between trauma in general (as defined in each paper) and schizotypy ranged between *2.01* (Afifi et al. 2011) and *4.15* (Lentz et al. 2010) *(adj. for gender, age, marital status and income)*. Examining the association with different trauma types separately, discrepancies between studies were observed. For physical abuse an odds ratio of *5.84* was reported *(and the trait unusual perceptions)* (Steel et al. 2009), however another study found a considerably less robust association between physical abuse and total schizotypy *(OR=1.62)* (Afifi et al. 2011). Likewise, for neglect the odds ratios ranged considerably from *1.35* (Afifi et al. 2011) to *6.7* (Rossler et al. 2007) and for sexual abuse associations ranged between *2.05* (Afifi et al. 2011) and *4.15* (Lentz et al. 2010). Emotional abuse remained associated with schizotypy even after adjusting for different types of trauma with an odds ratio of *1.76* (Afifi et al. 2011). The evidence for a strong association between emotional trauma and schizotypy was confirmed in several other studies (Battle et al. 2004;Berenbaum et al. 2003;Gibb et al. 2001;Lobbestael et al. 2010). Furthermore, the association was not only observed for general schizotypy, but emotional abuse was related to some specific schizotypal traits in a sample of individuals with schizotypal personality disorder (e.g. ideas of reference, excessive social anxiety, a lack of close friends, unusual perceptual experiences and eccentric behaviour or appearance) (Powers et al. 2011). Interestingly, there was emerging evidence that not only victimisation but also being a perpetrator and a victim at the same time *(in this case being a bully and being bullied)* was linked to higher psychoticism (Mynard and Joseph 1997). With extremely limited research in this area however, caution is required when interpreting this result. Nonetheless, bullying is a form of victimisation shown to have

strong ties with schizotypy levels as it was associated with all three schizotypy dimensions (Raine et al. 2011).

In general, childhood trauma was documented to be especially associated with an increased likelihood of experiencing positive schizotypal symptoms with an odds ratio of *4.8* (Myin-Germeys et al. 2011), with abuse showing stronger associations *(OR=5.53)* than neglect *(OR=3.67)* (Myin-Germeys et al. 2011). This is inconsistent with some previous reports, suggesting a slightly stronger association between neglect and schizotypy (Berenbaum et al. 2003;Johnson et al. 1999;Johnson et al. 2000) or reporting no difference in effects of specific trauma types (Tyrka et al. 2009). However, symptom-specific effects of the different trauma types were also shown, implying the possibility of different underlying mechanisms supporting trauma-schizotypy associations, e.g. physical and sexual abuse were associated with paranoia (for physical abuse OR was *5.84* (Steel et al. 2009), for sexual abuse OR equalled *4.49* (Steel et al. 2009)), while physical abuse also predicted unusual perceptions (Powers et al. 2011). A strong association between childhood abuse and positive schizotypal symptomatology was further supported with the levels of precognition, spiritualism, witchcraft, superstition (Perkins and Allen 2006) and unusual beliefs/experiences (Berenbaum et al. 2003;Startup 1999) all more likely to be increased if a participant reported traumatic childhood experiences.

Moreover, the associations between childhood trauma and positive symptoms have been most consistently reported, but negative and disorganized schizotypal dimensions have also been advocated. For example, the association between physical abuse and social anxiety (Powers et al. 2011) and the relationship between bullying and disorganised schizotypy dimension (Raine et al. 2011), including physical/emotional abuse and physical/emotional neglect and disorganised dimensions (Irwin 2001), especially eccentric behaviour (Powers et al. 2011).

2.1.4   Discussion

Childhood trauma and schizotypy association – main findings

Overall, this literature review demonstrated considerable support for the association between general trauma and schizotypy, including the individual effects for all trauma types included in the search. Moreover, the strongest evidence came from the methodologically most robust studies (Afifi et al. 2011;Lobbestael et al. 2010;Rossler et al. 2007). Considering trauma types individually, the results do not allow any firm conclusions to be drawn yet there is some evidence indicating that emotional abuse (Powers et al. 2011) and neglect (Berenbaum et al. 2003) are potentially the strongest predictors of schizotypy symptom load. These types of trauma have also been shown to have deleterious effects on self-efficacy and psychosocial functioning (Berenbaum et al. 2008;Johnson et al. 2001). In stark contrast to that stands another study reporting that physical and sexual abuse were associated with higher levels of paranoia/suspiciousness and unusual perceptual experiences but no such relation was found for emotional abuse (Steel et al. 2009). Even though some authors found symptom-specific effects associated with individual trauma type, other studies reported no differential effect of distinct types of trauma. This has several possible explanations, based around different methodological approaches used in these studies. Beside massive variation in sample sizes and populations studied (e.g. age, gender, general population vs personality disorders sample), the studies used multiple types of trauma measures and varied in inclusion of other trauma-related factors such as severity, frequency and duration *(see methodological limitations in section 2.1.5).* Moreover, the discrepancies in study findings might reflect the high level of comorbidity between PD symptomatology (individual displays symptoms across several PDs) (Lenzenweger et al. 2007), co-morbidity of multiple types of trauma (Dong et al.

2004) with potential synergistic effects (Edwards et al. 2003) and the importance of consideration of multiple interaction factors (in addition to childhood trauma) in the development of schizotypal personality traits.

When considering schizotypy from a more personality-theory position, similar observations have been reported. Evidence shows that childhood abuse is associated with paranormal beliefs and elevated levels of peculiarity (Berenbaum 1999;Berenbaum et al. 2003;Berenbaum et al. 2008;Eckblad and Chapman 1983;Johnson et al. 1999;Johnson et al. 2000;Read and Hammersley 2005) as well as increased levels of the cognitive-perceptual dimension among individuals with schizotypal personality disorder (e.g. ideas of reference, magical thinking, unusual perceptual experiences) (Berenbaum et al. 2008). Not surprisingly, individuals who reported adverse childhood experiences were 4 times more likely to develop a personality disorder (including schizotypal) in early adulthood (Johnson et al. 1999; 2001), with almost all forms of abuse, neglect and household dysfunction showing similar associations (Afifi et al. 2011). Importantly, these associations do not assume causality, but as discussed in later chapters the literature suggests various theoretical models that could account for the childhood trauma-schizotypy association (e.g. traumagenic neurodevelopmental model (Read et al. 2001)). This fits with the theory that personality develops (or is at least partially shaped) from emotion-related experiences in early childhood (Cohen 2008), inferring the possibly detrimental effect that severe traumatic experiences may have on the development of personality traits. Some differences in kinds of personality dysfunction associated with different trauma have also been advocated, where emotional maltreatment was associated with schizotypy in particular, but sexual abuse correlated with more generalised personality dysfunction (Gibb et al. 2001).

Parental separation has been associated with schizotypal personality disorder (Anglin et al. 2008) but no research has yet explored parental separation and schizotypy among the general population. Other studies have linked separation

from parents in childhood and increased risk for personality disorders (Byrne et al. 1990;Kantojarvi et al. 2008), especially if an individual was separated before the age of 5 (Lahti et al. 2012). Although it was hypothesised that parental separation is more related to personality disorder than schizophrenia (Gibbon et al. 2009) this type of trauma stands as a risk factor for a range of adult psychopathologies. Separation from a parent has also shown links to insecure attachments (Woodward et al. 2000) and altered sensitivity to stress via changes in hypothalamic-pituitary-adrenal axis (HPA axis) (Liu et al. 1997).

The role of other factors impacting the childhood trauma-schizotypy association

Nonetheless, some authors failed to find support for any association between particular traumatic experiences and schizotypal symptomatology after adjusting for all trauma types, however some discrepancies can also exist due to different methodological approaches. For example, there was no association reported between emotional abuse and schizotypy (Steel et al. 2009), no association between sexual abuse and schizotypy for both genders (Berenbaum et al. 2008) and no association between physical abuse and schizotypy in women only (Berenbaum et al. 2008). This not only implies the possibility of distinct underlying mechanisms for each trauma type that influence the development and persistence of schizotypal traits, but also stresses the importance of other confounders explaining the childhood trauma-schizotypy association, including age, gender and nature of the trauma itself (age when occurred, frequency, perpetrator etc.). All these factors might help us understand the main clues to the aetiology of psychotic symptoms and psychotic disorders and have been previously documented to play an important role in the childhood abuse and psychosis association: e.g. gender differences *(a trend of association between childhood physical and sexual abuse and psychotic-like experiences for women but not for men (Fisher et al. 2009)),*

including the possibility of different mechanistic pathways leading to psychosis for men and women (Myin-Germeys and Van Os 2007). However, this remains less clear for the childhood abuse and schizotypy association. With the exception of a few studies, the majority of them reported no evidence of moderation by sex (Lentz et al. 2010;Lobbestael et al. 2010;Myin-Germeys et al. 2011). When looking at the effect of age on childhood trauma and schizotypy load association, similar heterogeneity was observed, with one study supporting the positive correlation with age (between neglect and schizotypy for men) (Berenbaum et al. 2008), and another advocating the contrary - a trend of decrease in schizotypy levels with age (Rossler et al. 2007). Due to the very limited research in this area (and methodological restrictions), these findings need to be treated with caution.

On the other hand similar to psychotic-like experiences, some gender-specific underlying mechanisms supporting the childhood trauma – schizotypy association, have been documented. Berenbaum and colleagues (Berenbaum et al. 2008) reported that not only childhood maltreatment but also post-traumatic stress disorder (PTSD) contributed independently to the prediction of schizotypal symptoms, with no PTSD effect found for men. PTSD was documented to play a role of a partial mediator between childhood emotional abuse and some positive schizotypal symptoms in women (e.g. especially unusual perceptual experiences and eccentric behaviours (Powers et al. 2011)). In men however, the association between childhood trauma and schizotypy was significantly moderated by neurodevelopmental disturbance (Berenbaum et al. 2008). Likewise, the differential effects have been reported for abuse and neglect, where neglect was associated with positive and negative schizotypy while childhood abuse (emotional, physical and general) was only associated with the positive schizotypy dimension (Myin-Germeys et al. 2011). It is possible to hypothesize that the difference between abuse and neglect can be explained by the effect that each type of trauma has on

the developing brain as neglect was found to be associated with more severe cognitive and psychosocial deficits (Colvert et al. 2008).

Also, more exploration is required into other adversity-related factors such as age of occurrence, severity and frequency of trauma, multiple victimisation and different perpetrators that have not yet been fully considered. For example, the timing of exposure to adversity has been proposed to play a significant role (traumatic experiences prior the age of 12 (Fisher et al. 2010)) as early trauma was associated with more severe and persistent adult psychopathology (Blaauw et al. 2002). Moreover, the combination effects and multiple traumas have not been fully assessed, yet the research widely supports a dose-response effect of traumatic experiences on schizotypal symptomatology (Myin-Germeys et al. 2011). Some previous studies where the impact of multiple types of trauma were assessed (Liebschutz et al. 2002;Mullen et al. 1996) showed that long-term effects of childhood abuse might not be due to one type of trauma (e.g. sexual abuse) but could result from a combination of other factors such as neglect, family disorganisation etc. (Finkelhor 1986). Also bullying was a form of victimisation seen as a possible consequence of abnormalities in social adjustment (Schreier et al. 2009) and as such not an aetiological factor in itself (Kraemer et al. 2001;Murray and Fearon 1999) but possibly an additional risk factor in the development of psychosis (van Dam et al. 2012). In addition, it was reported that if an individual had one adverse childhood experience the likelihood for having another was 2 to 18 times higher than in those without any adverse childhood experiences (Dong et al. 2004). As the adversities are highly co-morbid (Benjet et al. 2009) it makes it much harder to disentangle the complexity of associations with schizotypal traits and identify the mechanisms supporting these associations.

Distinct pathways underlying childhood trauma-schizotypy association?

Beside some emerging evidence of distinct pathways from childhood abuse to schizotypy, a lot more research is needed to fully understand the differential effects of trauma types and all the possible mechanisms that can influence the levels of schizotypy. Much clearer pathways were observed between traumatic experiences and paranoia and suspiciousness as both correlated with high scores on negative-self and negative-others beliefs scales, as well as with increased depression and anxiety symptoms, all underlying the development of paranoid ideation via intensified feelings of mistrust and suspiciousness (Freeman et al. 2002). Unusual experiences were either seen as an object of intrusive memories of traumatic events (Morrison 2001), or the effect of underlying biological vulnerability caused by early trauma (Garety et al. 2001). In general, two pathways to psychosis were hypothesised – the first endogenous determined mainly by biological factors and the second pathway characterised by predominately environmental influences (Dominguez et al. 2010;Ross et al. 1994). Childhood trauma showed an especially strong association with positive schizotypy (Johns et al. 2004;Myin-Germeys et al. 2011;Shevlin et al. 2011) while negative/disorganised symptoms were mainly determined by biological factors, consistent with the hypothesis that negative/disorganised symptoms are possibly more linked to genetic risk to psychosis (Goldman et al. 2009) and associated with alterations in brain development (Heckers et al. 1999;Rowland et al. 2009). In stark contrast, positive symptomatology is particularly influenced by environmental risk factors, especially childhood trauma (e.g. Janssen et al. 2004;Thompson et al. 2009) but also cannabis exposure (Skinner et al. 2011;Stefanis et al. 2004a), urbanicity (Dominguez et al. 2010) and others *(see section 2.3 for further exploration of possible pathways underlying childhood trauma-schizotypy association)*.

Even though it was initially proposed that childhood traumatic events play a causal role in development of psychosis (Modai et al. 1980;Morrison et al. 2003;Read et al. 2005), the etiology of schizophrenia and nevertheless schizotypal traits showed much more complexity. Genes and environment both interact in various ways (Cannon and Clarke 2005;van Os et al. 2008) to produce the multidimensionality of schizotypal traits, therefore more research work is needed that would help to understand some important clues to aetiology of psychotic symptoms and consequently psychotic disorders.

2.1.5   Methodological issues

Sample characteristics

The studied populations included in papers selected for this review varied massively, with the age of included participants ranging from 6 years to 95, with about half of them incorporating large age ranges *(from 18 to 55 or above (Lobbestael et al. 2010;Myin-Germeys et al. 2011))* and some of them only limited to children and/or adolescent populations (Mynard and Joseph 1997;Raine et al. 2011). With such differences in age ranges among the samples, comparison of findings is extremely difficult, as the period when schizotypal traits were measured might chronologically overlap with or even precede the childhood trauma experiences as measured in another study. Furthermore, certain schizotypal personality traits have been shown to be associated with age e.g. positively correlated with age like introverted anhedonia or negatively correlated like unusual perceptual experiences (Mason and Claridge 2006;Rawlings et al. 2001). Also, especially with younger samples (e.g. children up to the age of 14), it is not possible to fully exclude the possibility that participants might still express schizotypal symptoms in the future as they have not yet passed through the critical period of risk.

In addition, some of the studies only included one type of trauma (e.g. Raine et al. 2011) and did not look for differential effects of different trauma types, whilst others used a much broader definition of childhood trauma and adversity (including any life-threatening event, conflict among parent, early interpersonal experiences, etc.) (Berenbaum et al. 2008;Berry et al. 2007;Rossler et al. 2007). Nevertheless, the effects of different trauma types were compared in the majority of the studies (Afifi et al. 2011;Lobbestael et al. 2010;Powers et al. 2011;Sommer et al. 2010). Besides, the threshold as to what was considered/included as childhood trauma differed between studies. For some it was defined as experiences that occurred at the age of 16 or below (Lentz et al. 2010), but for many studies this was expanded to include traumas that occurred prior to 18 years of age (Afifi et al. 2011;Berenbaum et al. 2008).

Also, one of the major limitations of the reviewed studies was reliance on crude measurements of childhood trauma, like self-report questionnaires or checklist formats completed by an interviewer (e.g. Lentz et al. 2010) which do not allow for any clarification or detailed information about traumatic experiences to be obtained, and also raise the question of under- or over-reporting and recall bias (McFarland and Buehler 1998). Some of the studies only used yes/no responses to assess childhood traumatic events which strips the data of all contextual information (Johnson et al. 1999;Steel et al. 2009) and may result in either over-inclusion or exclusion of certain childhood adversities. As the literature suggests, the frequency and severity of abuse play an important role in the development of schizotypal symptomatology and further psychotic disorders (Fisher et al. 2010;Janssen et al. 2004;Schenkel et al. 2005). As only the most severe trauma was associated with psychotic symptomatology (Bebbington et al. 2011;Schenkel et al. 2005) it is essential to be able to make a distinction between different levels/severity of abuse

to maximise the validity and reliability of the study. Out of the presented studies, only one study (Berenbaum et al. 2008) included full information on age of the occurrence, frequency and perpetrator while a minority of studies used a semi-structured interview to measure childhood trauma (Berenbaum et al. 2008;Lobbestael et al. 2010) or a medical examination or records from social services (Johnson et al. 1999), thus potentially providing better quality data. Despite all the controversy about the reliability of retrospective reporting of traumatic experiences (*further explained in the next section - 2.1.5.1*) there is a lot of encouraging evidence that childhood trauma reports are reasonably reliable and stable over a long period of time even amongst clinically psychotic patients (Dube et al. 2004;Fisher et al. 2011).

Even though the majority of studies included in this review were assessing childhood trauma retrospectively and the possibility of 'reverse causality' could not be completely excluded, the few prospective and longitudinal studies (Battle et al. 2004;Johnson et al. 1999;Johnson et al. 2001) that have been conducted suggest a similar association between childhood trauma and subsequent schizotypy to that demonstrated in cross-sectional retrospective studies (Afifi et al. 2011;Powers et al. 2011). The 'reverse causality' hypothesis suggests that individuals with more psychosis-like experiences or schizotypal traits might be more vulnerable to be exposed to violence and trauma, but the dose-response effect of trauma on schizotypal levels and differential effect of certain trauma types on particular symptoms (e.g. Powers et al. 2011) would be difficult to justify if schizotypy preceded these traumatic experiences.

Study design

Another drawback to data comparison was the major difference in study designs, including studied populations varying from representative community studies to

individuals with personality disorder *(also making it hard to exclude other factors that might come with the disorder itself, possible treatment effects etc.).* Furthermore, the sample sizes varied massively from *75* (Berenbaum et al. 2003) to *34,653* (Afifi et al. 2011), again suggesting a need for caution when comparing their findings. Nonetheless for the majority of studies the sample included between *100* and *1000* participants, coming from either representative general-population studies (Afifi et al. 2011;Berenbaum et al. 2008;Raine et al. 2011) cohort studies (Powers et al. 2011;Sommer et al. 2010), case-control studies (Lobbestael et al. 2010;Myin-Germeys et al. 2011) or convenience samples (Berry et al. 2007;Perkins and Allen 2006;Steel et al. 2009).

Moreover, 90% of the studies were conducted in the USA or Europe and only 10% covered other parts of the world. Therefore, it is difficult to generalise conclusions to other contexts, especially as firstly, childhood trauma has been previously found to have different definitions across the globe (Baumrind 1997;Deater-Deckard and Dodge 1997;Giovanni and Beccerra 1979) and secondly, schizotypal symptomatology *(or some particular traits e.g. magical ideation)* has been argued to be culture dependent (Chavira et al. 2003;Sharpley and Peters 1999).

Definition and assessment of schizotypy

Schizotypy is a multidimensional concept, not only dependent on the country or culture where it is assessed, but also affected by the variations in measures employed to assess its features *(see section 1.1.3).* The heterogeneity of schizotypy measures was also evidenced from this systematic literature review, as some studies assessed schizotypy with standardised tools administered by clinicians (Afifi et al. 2011;Myin-Germeys et al. 2011) but others relied on non-standardised methods and self-reports (Powers et al. 2011;Rossler et al. 2007). To add to the

complexity, in some cases, only a few questions were employed to gauge some specific schizotypal traits. This discrepancy is partially a result of a broader range of schizotypal traits included in the review *(and therefore measures)* not only observed in general population studies (Lentz et al. 2010) but also in clinical cases *(individuals with personality disorders)* (Battle et al. 2004).

Additionally, when looking at trauma-schizotypy relationship it is not possible to fully exclude the potentially confounding effects of other factors suggested in previous research e.g. beside gender, age (Fisher et al. 2009;Fonseca-Pedrero et al. 2008;Fossatti et al. 2003) and ethnicity (Johns et al. 2002;King et al. 2005), also the importance of familial risks (Appels et al. 2004;Siever and Davis 2004;van Winkel et al. 2008) and affective states (Lenzenweger and Loranger 1989) etc. In the presented review, only two studies included the main potential confounders such as psychiatric family history, depression and anxiety (Berenbaum et al. 2008;Steel et al. 2009) while the rest only adjusted for general sociodemographic factors. On the positive side, irrespective of the number of confounders included, the childhood trauma and schizotypy association remained similarly strong.

2.1.5.1   Issues in childhood trauma reporting (retrospective reporting)

There has been a lot of controversy about the validity of retrospective information (Briere and Conte 1993;Della et al. 1990;Williams 1994), especially limitations of child abuse reporting (Beitchman et al. 1991;Briere 1992;Milner 1991) where individuals are asked to describe the experiences based on their recollections.

General forgetting before the age of 5 has been widely cited (Brewin et al. 1993), with no difference in recall after the age of 5 (Widom and Shepard 1996). Especially memories before the age of 3 or 4 are reported to be fragmentary and vulnerable to loss (Fivush 1993;Fivush 1994). Another study even showed a scarcity

of specific events prior to the age of 8 (Nelson 1993). However, some argued that events of personal significance may be remembered even down to the age of 2 (Usher and Neisser 1993), and no recall after the age of 3 is uncommon (Wakefield and Underwager 1992). The notion of childhood amnesia (Brewin et al. 1993;Lewis 1995;Loftus 1993a) has received mixed reviews with some suggesting it is not a true form of amnesia but the lack of autonoetic awareness[9] in early childhood (Howe and Courage 1993;Wheeler et al. 1997), leading to missing personal frame of reference that makes memory autobiographical (Howe and Courage 1993).

Autobiographical memory has been defined as a continuous process of revisions, selections, and re-interpretations (Ross and Conway 1986). However, several studies of autobiographical memory demonstrated that memory can be extremely accurate over a longer period of time (Hudson and Fivush 1991;Wagenaar 1986), especially for significant experiences (Baddeley 1990). Brewer (1986) argued that as memories include a large amount of irrelevant details, they could not reflect solely schematic reconstructions. Episodic autobiographical memory which relates to personal experiences, including general events and episodic, more specific events (Larsen 1992;Piolino et al. 2002;Schacter 1996) is especially sensitive and shows an extreme drop in episodic memories with an encoding at the average age of 4 (Piolino et al. 2002). Even though the memories might be few they still enable the subject to be aware of subjective time, allowing perceived continuum from subjects' past to their future (Wheeler et al. 1997). Similarly, people's memories of the events are found to be more accurate than the timing itself, which also creates a danger that whatever happened after the onset of the illness/disorder is recalled as if it preceded the illness (Chess et al. 1966).

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[9] *Autonoetic awareness is an ability to mentally place ourselves in the past, present and future and as such relates to episodic memory and capability of mental time travel (Metcalfe and Son 2012).*

Nevertheless, there is a general acceptance that memory is at least partially reconstructive with a decline in accuracy and details (Fivush 1993). Beside distortion or loss of information, an individual forgets or redefines behaviour according to his/her later life situation e.g. egocentricity and self-serving attributional biases are just two of the ways that the ego re-evaluates and consequently reconstructs its own past (Greenwald 1980;Ross 1989) while adapting the past emotions and memories to fit with their current situation, beliefs and feelings (Levine 1997). There is also a tendency to distort or reconstruct events to enhance positive affect (Yarrow et al. 1970) *(involvement of amygdala (Phelps and Anderson 1997))*, issues of social desirability pressures (Widom and Shepard 1996), general tendency to seek meaning in memories (Schacter 2001) *(e.g. giving memories an explanatory and evaluative context (Fivush 1995))*, embarrassment or inclination to protect parents/abuser, feelings of deserving the abuse or even a conscious wish to forget the experiences (Della et al. 1990). It is important to note, that loss of content and distortion are relatively independent processes, as distortion occurs during reconstruction after the failure of episodic retrieval also influenced by relevant generic memories (Bahrick et al. 2008). The repression of traumatic events is seen as a form of self-protection (Loftus et al. 1998;Memon and Young 1997;Penfold 1996), but there is a lack of sufficient experimental evidence showing that the person truly is capable of such repression (Loftus et al. 1998;Pope and Hudson 1995). Amnesia as a concept of repression was originally proposed by Freud (1910) which also ties in with memory loss after traumatic experiences *(through dissociation process)* (van der Kolk and Kadish 1987), however the true relationship between the constructs remains unclear (Frankel 1990).

Also, adult memory functioning might be influenced by interaction effects of cognitive development and the childhood trauma itself (Rogers 1995), as trauma may cause a disruption of events encoding or even impairs the development of full

cognitive capacities. Individuals are therefore more susceptible to memory distortions e.g. failure of retrieval, source misattributions, retrospective incomplete memory (Rogers 1995). Abuse-specific amnesia is reported to be high and especially associated with more violent abuse (Herman and Schatzow 1987) that lasted for an extended period of time and had an early onset e.g. abuse that started before 18 and ended before adolescence (Briere and Conte 1993;Herman and Schatzow 1987). Interestingly though, with individuals experiencing more than one childhood abuse the likelihood of forgetting also increases (Feldman-Summers and Pope 1994). Forgetting of such traumatic experiences also depends on contextual factors and who the perpetrator was – if it was a family member *(someone who the subject has a close relationship with)* the subject is more susceptible to forgetting, compared to abuse from strangers (Elliott and Briere 1995;Williams 1994). Specifically for sexual trauma however, the periods of forgetting were documented regardless whether the perpetrator was a relative or nonrelative (Feldman-Summers and Pope 1994).

Mood-congruent recall has been widely cited (Matt et al. 1992;McFarland and Buehler 1998) - *'one always finds mood-biases in recall in autobiographical memories'* (Bower 1987 p.454). Especially anxiety and depression have been seen as having a detrimental effect on memory (Brewin et al. 1993). Similarly, studies showed that some characteristics of the parents (displaying rejection and punishment) were associated with the levels of depression (Lewinsohn and Rosenbaum 1987;Williams 1996). Depressed individuals may also attempt to identify the cause from their past associated with their current state by applying the *'effort after meaning'* (Rogers 1995 p.700), and have the tendency to over-generalize when retrieving autobiographical memories (Williams and Dritschel 1988), similar to those with PTSD (McNally et al. 1994). Depressive symptoms can therefore cause a recall of more unhappy memories and inhibit recall of happy ones,

but such bias is relatively minor (Brewin et al. 1993). In stark contrast, the impact of current depression on stability of measurements of past experiences received no support in some studies (Brewin et al. 1993;Fisher et al. 2011;Gotlib et al. 1988;Parker 1981;Perris et al. 1986). There was also an assumption that depressive people might be more realistic than healthy individuals (Robins et al. 1985) and that emotion enhances the vividness or accuracy of the memory (Reisberg and Heuer 2004).

Also, the recovered memories rely on the reality and source monitoring functions, implying the concerns of suggestibility (Tellegen and Atkinson 1974) and fantasy proneness (Bartholomew et al. 1991). Certain populations like young children, elderly, or abused children suffering from dissociative disorders might be especially prone to such source monitoring errors (Rogers 1995). Dissociative symptoms can not only alter the sense of reality, identity and memory (Putman 1985) but are also associated with psychogenic amnesia (Herman 1992;Holmes 1990).

Even though relying on patient's memories to obtain information has been questioned (Paris 1995), considering the economical and ethical issues of prospective studies, the retrospective method still stands as an adequately reliable technique (Maughan and Rutter 1997), but should be treated with caution. Relative instability and low reliability of young people's reports was indicated for childhood sexual abuse, parental physical punishment (Fergusson et al. 2000;Fry et al. 1996) as well as neglect (Widom and Shepard 1996). There was a negative correlation observed between the age of the first memory and factors such as IQ, language ability, social class, general functioning (Maughan et al. 1995) and gender *(earlier recall for females comparing to males (Nelson 1993))*. Individuals with good functioning tend to forget or minimize early adversity whereas those with poor functioning might exaggerate negative childhood circumstances (Maughan et al.

1995). Also, gender differences in childhood trauma reporting were further supported in another study (Widom and Morris 1997), where women reported significantly more sexual abuse experiences than men *(women 64% where sexual abuse had been documented, men only 16%)*, but can partially reflect the early socialization experiences. Moreover, the recall depends on not only the mood state at the time of encoding but also the length of time since this encoding occurred (Hermann 1994). Besides, different cognitive appraisals play a mediating role as lifetime events influence how something is perceived – is it threatening, harmful, benign? (Lazarus and Launier 1978). Also, people can only recall what they have witnessed themselves and were aware of at the time (*Were they aware of significant financial problems or household discord?)* (Robins et al. 1985). Equally, individuals can have false beliefs (Loftus and Ketcham 1994;Loftus 1993b), including believing the event has occurred but in reality never has (Ceci et al. 1994;Garry et al. 1994). Nonetheless it is more likely that individuals will fail to report than falsely claim abuse (bias to underreporting) (Dill et al. 1991;Fergusson et al. 2000;Widom and Morris 1997). As such, the main concern still lays on underestimation of incidents, as the evidence from well documented cases of abuse suggests that individuals can fail to report even serious abuse and neglect (Hardt and Rutter 2004).

Individuals scoring high on schizotypy might suffer from frequent trauma-related intrusions[10] (Marzillier and Steel 2007) as trauma memories are not contextually processed but based on perceptual information during the traumatic event (Jones and Steel 2012). Consequently, a voluntarily recall of trauma-related experiences is weakened, but at the same time fragments of trauma memories can be re-experienced involuntarily (Holmes et al. 2005) and there is a danger of 'source monitoring' errors (Laroi et al. 2005). Thus, individuals with more prominent

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[10] *Intrusions are uncontrollable psychological response to traumatic experience. They include memories, impulses, images or repetitive thoughts (American Psychiatric Association 1994). If persisted it can lead to PTSD diagnosis, but intrusions are not diagnostically specific and have been linked to other disorders such as psychosis (Morrison et al. 2003).*

schizotypal traits are less likely to distinguish between internally sourced events and those created externally (what they really experienced and what was witnessed/seen only) (Laroi et al. 2005). However, a study comparing low and high schizotypy groups found no significant difference in frequencies of deliberately retrieved memories (Jones and Steel 2012). Furthermore, those with elevated schizotypal scores were also able to retrieve episodic information with less cognitive effort and displayed improved ability to mental travel back in time (Rasmussen and Bernsten 2009;Winfield and Kamboj 2010).

In summary: This systematic literature review provided a substantial support to the association between childhood trauma and schizotypy in a dose-response manner. The associations were particularly strong for childhood trauma (especially emotional abuse) and the positive schizotypy dimension. Many discrepancies are evident with regard to differential effects of trauma types on schizotypy load, with some suggesting similar associations for all trauma types and others reporting trauma-specific outcomes. Methodological limitations of the studies along with the complexity of schizotypy construct per se impede the comparison between them and making any form of conclusions would be premature.

2.2 Childhood trauma in relation to psychotic-like symptoms

The section will present empirical studies exploring the association between childhood abuse and psychotic-like symptoms/experiences (e.g. hallucinations, delusions), using measures specifically designed to assess attenuated psychotic symptomatology. This research area overlaps considerably with schizotypy and cannot be easily distinguished (Bedwell and Donnelly 2005). While schizotypy reflects more chronic, stable symptomatology, attenuated psychotic symptomatology is often associated (not exclusively) with prodromal phases of schizophrenia with more recent onset and escalation in symptom severity (Miller et al. 2003) *(see section 1.1.1)*. Despite theoretical underpinnings separating schizotypy and psychotic-like symptoms, attempts to assess them as a distinct constructs proves more problematic (or possibly arbitrary).

For the purposes of this thesis, schizotypy and psychosis-like symptoms were largely distinguished according to measures proposed to evaluate each of them (*see section 1.1.3.1 for the assessment tools designed to measure schizotypy and section 1.1.3.2 for the measures of attenuated psychotic symptoms)*. The exceptions were the inclusion of tools assessing SPD symptoms based on DSM criteria in the schizotypy section e.g. *Schizotypal Personality Questionnaire (SPQ) (Raine 1991)* and *Schizotypal Personality Scale (STA) (Claridge and Broks 1984)*. SPQ not only incorporates the multidimensionality of schizotypy but the SPQ brief version has also been used to assess dimensional schizotypy in nonclinical populations (Bailey and Swallow 2004;Bedwell et al. 2006;Fonseca-Pedrero et al. 2009b;Mata et al. 2005).

2.2.1   Introduction and search strategy

Literature suggests that childhood trauma is associated with psychotic symptoms in clinical populations (Bebbington et al. 2004;Mueser et al. 1998;Read 1997;Read

and Argyle 1999) but also in ultra-high risk groups (Addington et al. 2013;Bechdolf et al. 2010;Sahin et al. 2013;Thompson et al. 2010) and community populations *(psychotic-like experiences)* (Alemany et al. 2012; 2013;Bendall et al. 2008;Edwards et al. 2003;Janssen et al. 2004;Kelleher et al. 2008;Kessler et al. 2010;Ross and Joshi 1992;Wigman et al. 2011a). Not only the occurrence but also the persistence of psychotic-like experiences has been linked to early victimisation (Mackie et al. 2011) contributing to the risk for developing a full-blown psychotic disorder (De Loore et al. 2007).

Empirical literature search strategy

The search criteria correspond to those adopted for the childhood trauma and schizotypy association *(see section 2.1.2).* The studies that did not meet the schizotypy criteria *(or utilised any of the measures)* but still assessed symptoms that overlap with schizotypal traits were included in this analysis *(see Figure 3).*

The systematic literature review was conducted in the following databases: *PsycInfo* (including *MeSH* Terms), *PubMed*, *EMBASE*, *Web of Science*, where two sets of search terms were used:

1. *trauma** OR *maltreat** OR *abuse* OR *advers** OR *neglect OR bully** OR *victim** OR *parental loss* OR *separat** AND *adolescen** OR *child**
AND
2. *schizoty** OR *psychos** OR *psychotic* OR *illusion* OR *hallucination* OR *delusion* OR *derealisation* OR *depersonalisation* OR *social isolation* OR *hypersensitivity* OR *magical ideation* OR *introversion* OR *referential thinking* OR *suspiciousness* OR *restricted affect*.

The search covered the articles from 1806 to 1st March 2013 and resulted in *17,003* articles in total. The data was transferred into Reference Manager and after duplicates were extracted, *13,050* articles were identified for the title screening. Title screening yielded *801* potentially relevant articles, which were narrowed down to *311* after further abstract screening. Through the full-text screening of the remaining articles we identified *48* studies exploring the relationship between childhood trauma and attenuated psychotic-like symptoms. The reasons for excluding articles at each stage are documented in *Figure 3*.

Inclusion and exclusion criteria

For papers to be included they had to meet the following criteria: (a) an original research paper (including replication studies); (b) written in English language; (c) use a measure of childhood trauma (either emotional, physical or sexual abuse, neglect, bullying, separation from parents or parental loss, or other traumatic experiences) before the age of 18 *(any standardised or non-standardised measure)*; (d) test whether there is an association with psychotic-like symptoms *(any standardised or non-standardised measure)*; (e) use general population/community samples in their studies and not clinical (psychotic) cases; and (f) include complete information on design and measures used in the study to allow completion of a quality assessment tool designed for this analysis (e.g. exclusion of conference abstracts due to limited information).
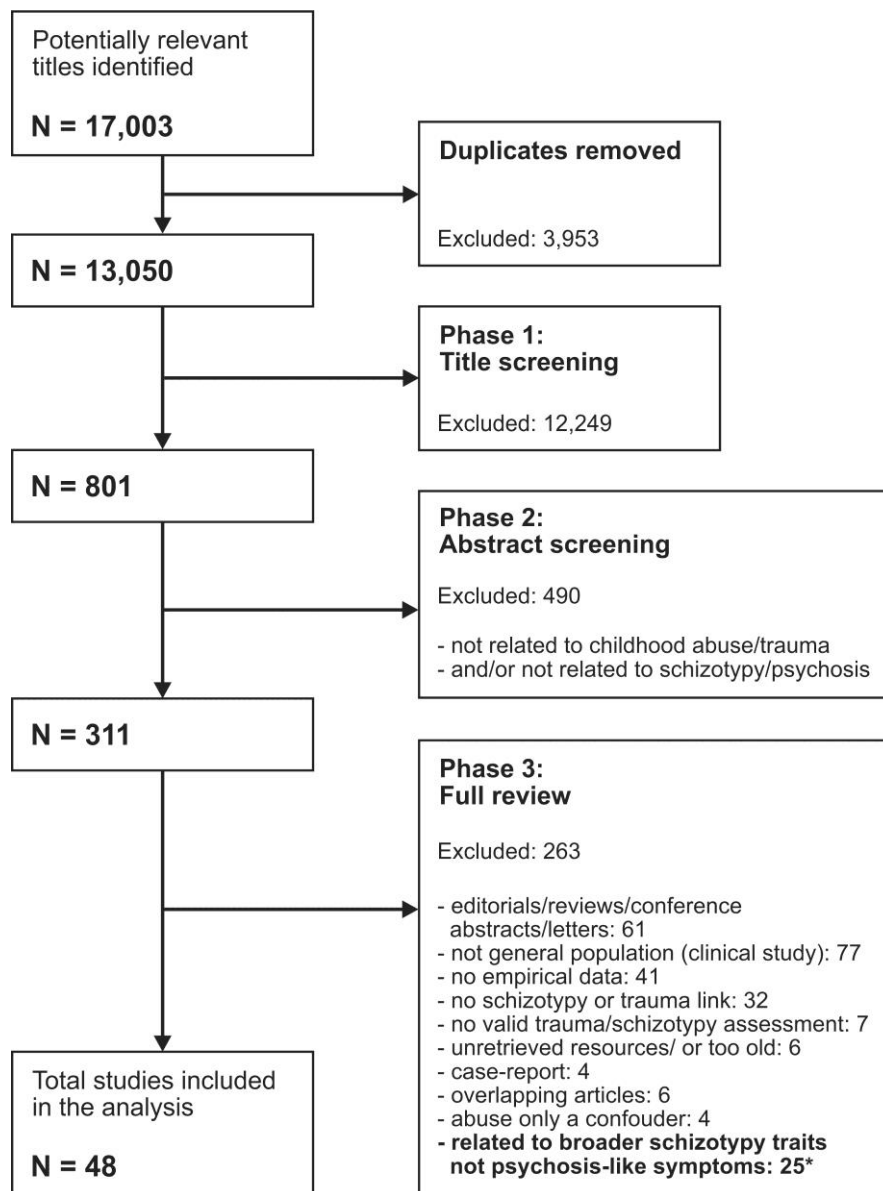
Figure 3: Flowchart of studies included in the literature review.
*The section corresponds to the previous thesis chapter, exploring the association between childhood trauma and schizotypy (Section 2.1)

2.2.2   Results

In the presented table (Table 4), only 35 papers (out of 48) were included with an assigned quality score of 7 points or above out of a maximum 14, to particularly focus on the most methodologically robust studies. The same scoring guide was used as in the preceding section *(see 2.1.2 and Appendix I for full scoring procedure)*. Details of the other papers can be found in Supplementary Table *Suppl.1 (Appendix II).*

Table 4: Summary of studies on childhood trauma in relation to psychosis-like experiences (PSE) (ordered by quality score) (All abbreviations are listed at the bottom of the table)

Authors	Study Design	Sample Recruited (Age)	% female	Measure of Trauma	Number Exposed	Measure of PSE	Number with the Outcome	Measure of Effect	Quality Score
Arseneault et al. (2011) (UK)	Longitudinal study/ Birth cohort study/twins	N=2232 (when 7, then at 10 and at 12)	-	Assessed by interviewing mothers, protocol taken from Lansford et al. (2002)	N=589 children (28%) had been maltreated by an adult or bullied by peers N=70 (3%) experienced both trauma	7 Psychotic symptoms investigated related to Delusions and Hallucinations (see Poulton et al. 2000)	At least one psychotic experience N=125 (5.9%)	Maltreatment or bullying vs none and psychotic symptoms (OR=3.27, 95% CI=2.25-4.76, sign.) Maltreatment and bullying vs none and psychotic symptoms (OR=5.68, 95% CI=3.18-10.14, sign.)	14
Kelleher et al. (2008) (Ireland)	Cross-sectional Representative sample of general population	N=221 (12-15)	-	Question on sexual and physical abuse (part of K-SADS interview) (Kaufman et al. 1996) Exposure to domestic violence - using post-traumatic stress disorder section in K-SADS Bullying (part of K-SADS interview)	-	K-SADS (Kaufman et al. 1996), including hallucinations and delusions	Adolescent with (N=14) and without (N=197) psychotic symptoms Adolescent with (N=10) and without (N=74) psychotic disorder	Childhood physical abuse vs no abuse and psychotic symptoms (OR= 5.06, 95% CI=1.27-27.97, p=0.023) Exposure to domestic violence (OR=10.06, 95% CI=2.20-46.01, p=0.003) History of being a bully (OR= 9.90, 95% CI=2.51-39.05, p=0.001)	13
Scott et al. (2007) (Australia)	Cross-sectional Representative sample from general population	N=10641 (Above 18 years)	-	CIDI (World Health Organization 1993) (10 questions about possible traumatic events in their lifetime)	N=4537 (53.8%) exposed to traumatic event (without PTDS)	CIDI (World Health Organization 1993)	N=478 (4.49%) endorsed one or more delusional experiences	Exposure to any traumatic event vs no event and increased endorsement of delusional experiences (OR=2.03, 95% CI=1.61-2.57, p<0.001) *adj. gender, age, cannabis use, past diagnosis of schizophrenia*	12
Fisher et al. (2013b) (UK)	Prospective study Representative sample from general population	N=6692 sample from ALSPAC (Mean=12.9)	50.9%	For harsh parenting (mothers asked one question) Children interview with Bullying and Friendship Interview Schedule (Wolke et al. 2000)	23.6% exposed to domestic violence prior age 6 4.2% harsh parenting before age 7 10.5% bullied before age 8.5	Psychosis Interview (PLIKSi) (Horwood et al. 2008) derived from DISC-IV (Shaffer et al. 2000)	11.3% probable or definite symptoms ('broad'), 4.7% definite symptoms ('narrow')	Harsh parenting and broadly defined psychotic symptoms (OR=1.02, 95% CI=0.95-1.08, sign.) Domestic violence and broadly defined psychotic symptoms (OR=1.04, 95% CI=0.98-1.10, sign.) Bullying and broadly defined psychotic symptoms (OR=1.13, 95% CI=1.06-1.19, sign.) Harsh parenting and narrowly defined psychotic symptoms (OR=1.01, 95% CI=0.93-1.09, sign.)	11

Table 4 Cont'd

							Domestic violence and narrowly defined psychotic symptoms (OR=1.06, 95% CI=0.97-1.14, sign.) Bullying and narrowly defined psychotic symptoms (OR=1.14, 95% CI=1.03-1.24, sign.) *All adj. sex, ethnicity, birth weight, family psychiatric history, IQ, general family adversity*	
Saha et al. (2011) (Australia)	Cross-sectional Representative sample from general population	N=8773 (Above 16 years)	-	CIDI (see Kessler and Ustun 2004) (10 questions about possible traumatic events in their lifetime)	42% reported one or two trauma exposures 20% reported three or more trauma types 12% five or more traumatic events	CIDI (see Kessler and Ustun 2004)	N=776 (8.4%)	Associations between exposure to trauma vs no trauma and delusional-like experiences (OR=2.64, 95% CI=1.97-3.52, sing.) Dose response relationship between number of types and DLE (χ^2=86.1, df=2, p<0.001)	11
Shevlin et al. (2007c) (USA)	Cross-sectional Representative sample from general population	N=5893 (15-54 Mean=32)	52.0%	Posttraumatic stress disorder module and modified CIDI (World Health Organization 1990) – in childhood	-	Psychosis screening section of CIDI (World Health Organization 1990) - 13 items (psychotic-like symptoms)	Class 1 (1.8%) – paranoia, thought interference, hallucinations Class 2 (6%) – all four hallucinatory items Class 3 (5.9%) – less than class 1 quantitatively	Neglect as a child & Class 1 (OR=2.40, 95% CI =1.16-4.93, p<0.5), Class 2 (OR=1.23, 95% CI=0.70-2.13, p<0.5) & Class 3 (OR=1.52, 95% CI=0.85-2.73, p<0.5) Physically abused as a child & Class 1 (OR=2.27, 95% CI=1.56-4.46, p<0.5) & Class 2 (OR=2.37, 95% CI=1.54-3.65, p<0.5) & Class 3 (OR=1.53, 95% CI=0.91-2.56, p<0.5) Sexually molested & Class 1 (OR=2.68, 95% CI=1.50-4.79, p<0.5) & Class 2 (OR=2.10, 95% CI=1.47-2.99, p<0.5) & Class 3 (OR=1.37, 95% CI=0.90-2.10, p<0.5) *All adj. sex, household composition, income, education years and employment*	11
Spauwen et al. (2006) (Germany)	Prospective study Representative sample from general population	N=2524 (14-24 Mean at T2= 21.7 SD=3.4)	49.0%	CIDI (World Health Organization 1990) (list of 9 groups of specific traumatic events)	At Time 0 trauma reported N=491 (19.5%)	DIA-X/M-CIDI (Wittchen and Pfister 1997) Self-Report Symptom Checklist-90-revised (Derogatis 1977)	N=441 (17.5%) at least one psychotic symptom N=183 (7.3%) reported two or more N= 85 (3.4%) reported three or more	Trauma associated with psychotic symptoms (OR=1.89, 95% CI=1.16-3.08, sign.) *adj. gender,SES,urbanicity,cannabis use,psychosis proneness,DSM-IV mental disorder T0*	11

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Bebbington et al. (2004) (UK)	Cross-sectional Representative sample from general population	N=8580 (16-74)	-	Stressful life events (relationship problems, illness, bereavement, employment, financial crisis, victimisation)	Sexual abuse (3.5%) Bullying (17.4%) Violence in the home (7.0%)	Schedule for Assessment in Neuropsychiat. (SCAN) (World Health Organization 1999)	Psychosis (0.7%)	Sexual abuse and probable psychotic disorder (OR=7.4, 95% CI=3.6-15.2, p<0.001) Bullying and probable psychotic disorder (OR=3.1, 95% CI=1.6-5.9, p<0.001) Violence in the home and probable psychotic disorder (OR=4.7, 95% CI=2.3-9.4, p<0.001) *All adj. level of depression*	11
Janssen et al. (2004) (The Netherlands)	Prospective cohort study Representative sample from general population	N=4045 (18–64 Mean=41.4 SD=11.8)	52.7%	Semi-structured interview on emotional, physical, psychological or sexual abuse before 16 (yes/no) and 6-point scale	N=423 (10.5%) reported childhood abuse	CIDI (Composite International Diagnostic Interview and the Present State Examination) (World Health Organization 1993) Brief Psychiatric Rating Scale (BPRS) (Overall J.E. and Gorham 1962)	Psychosis outcome: BPRS any psychosis N=38 (0.94%) BPRS pathology level N=10 (0.25%) Need-based disorder N=7 (0.17%)	Abuse vs no abuse BPRS any psychosis (OR=2.5, 95% CI=1.1-5.7, p<0.001) BPRS pathology level (OR=9.3, 95% CI=2.0-43.6, p<0.001) Need based disorder (OR=7.3, 95% CI=1.1-49.0, p<0.003) *All adj. age,sex,education,discrimination, ethnicity,urbanicity, marital status,any psychiatric diagnosis, unemployment etc.*	11
Binbay et al. (2012) (Turkey)	Cross-sectional Representative sample from general population	N=4011 (15-64)	58.0%	Adverse life events (dichotomous) between age 0-5 and 6-15	At least one childhood event N=605 (15.1%)	CIDI (World Health Organization 1990)	Subclinical psychotic experience group N=625 Low-impact psychotic symptoms group N=198 High-impact psychotic symptoms N=109	At least once childhood adverse life event and subclinical psychotic experience (OR=1.40, 95% CI=1.10-1.80, p<0.01) At least once childhood adverse life event and low-impact psychotic symptoms (OR=1.80, 95% CI=1.30-2.60, p<0.01) At least once childhood adverse life event and high-impact psychotic symptoms (OR=2.00, 95% CI=1.30-3.20, p<0.01) *adj. age, sex*	10
Murphy et al. (2012) (UK&USA)	Replication Study NCS-R (Cross-sectional, Representative sample from general pop.)	N=2355 (Mean=44.34 SD=17.27)	57.8%	Posttraumatic Stress Disorder module (modified version of CIDI 3.0) (see Kessler 1994) (2 questions on sexual trauma)	6-8% experiences some form of sexual trauma	Psychosis Module CIDI 3.0 (see Kessler 1994)	Range from 0.1% (mind control) to 6.3% (visual hallucinations)	Childhood experience of rape independently contributed to adult psychosis (β=0.15, p<0.05) *adj. age, gender, income, education, pre-trauma psychiatric diagnosis, pre-rape and pre-sexual assault psychotic experiences*	10

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Harley et al. (2010) (Ireland)	Cross-sectional Case-control study	N=211 (12-15)	-	Part of K-SADS (Kaufman et al. 1996) on child abuse (physical and sexual) Post-traumatic disorder section (for domestic violence) & Parents asked the same questions	11.3% (N=24)	Schedule for Affective Disorders and Schizophrenia for School-age Children (K-SADS) (Kaufman et al. 1996) - Psychosis section	N=21 (5%) – psychotic symptoms	Trauma vs no trauma and psychotic symptoms (OR=5.20, 95% CI=1.58-17.13, p=0.007) *if adj. for gender, age, SES, family psychiatric history (OR=6.16, 95% CI=1.65-23.1, sign.)*	10
Freeman and Fowler (2009) (UK)	Cross-sectional Representative sample of general population	N=200 (18-77 Mean=37.5 SD=13.3)	50.0%	Life Stressor Checklist (Wolfe and Kimerling 1997)	At least one traumatic event experience (N=140, 70%) Childhood physical or sexual abuse (N=51, 25.5%) Severe childhood sexual abuse (N=15, 7.5%)	Paranoia and hallucinations – Green et. al. Paranoid Thoughts scale (Part B) (Green et al. 2008) Cardiff Anomalous Perceptions Scale (Bell et al. 2006)	Persecutory ideation (N=85, 42.5%) Verbal hallucinations (N=31, 15.5%) At least one anomalous experience in the clinical psychosis (N=89, 44.5%)	At least one traumatic event was associated with increased risk of endorsing a persecutory thought (OR=2.52, CI=1.36, 4.68, p<0.01) and increased risk of verbal hallucinations (OR=4.75, CI=1.39, 16.29, p=0.001) *adj. age,sex,ethnicity,educational level,SES,intellectual functioning*	10
Schreier et al. (2009) (UK)	Prospective cohort study Community sample	N=6437 (for peer victimisation 8 to 10, psychotic symptoms at age of 12)	-	Bullying and Friendship Interview Schedule (Wolke et al. 2000) Parents/teachers reported on single item included in the strengths and difficulties questionnaire	46.2% reported being victims by one or several students Chronic peer victimisation (13.7%) Severe victimisation (5.2%)	PLIKSi (see Horwood et al. 2008) 7 questions - National Institute of Mental Health DISC-C (Shaffer et al. 2000) 5 items from Schedules for Clinical Assessment in Neuropsychiat. (World Health Organization 1994)	-	Repeated victim was associated with definite psychotic problems (OR=1.94, 95% CI=1.54-2.44, sign.), even stronger with severe victimisation Children with both type of victimisation had higher odds of developing PLIKSi symptoms (OR=4.60, 95% CI=3.24-6.50, sign.)	10
Shevlin et al. (2011) (USA)	Cross-sectional Representative sample from general	N=2353 (Mean=44.35 SD=17.27)	58.0%	Posttraumatic stress disorder module and modified CIDI (Kessler and Ustun	Physical assault (7.7%) Rape (7.9%) Other sexual	The psychosis module of the CIDI (Kessler and Ustun	Visual hallucinations (5.2%) Auditory	Physical assault & visual hallucinations (OR=3.22, 95% CI=146-7.09, p<0.5) Physical assault & auditory hallucinations (OR=4.55, 95% CI=1.69-10.57, p<0.5)	10

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	population			2004)	assault (11.2%)	2004)	hallucinations (3.0%) Both types (2.3%)	Rape & visual hallucinations (OR=3.41, 95% CI=1.72-6.76, p<0.5) Rape & auditory hallucinations (OR=2.97, 95% CI=1.39-6.33, p<0.5)	
Johns et al. (2004) (UK)	Cross-sectional Representative sample from general population	N=8580 (16-74)		Victimisation events (bullying, sexual abuse, etc.)	28.2% victimisation experience	Psychosis Screening Questionnaire (PSQ) (Bebbington and Nayani 1995)	5.5% endorsed one or more items on PSQ	Experience of victimisation and psychotic symptoms (OR=2.0, 95% CI=1.7-2.6, p<0.001) Experience of victimisation and paranoid thoughts (OR=2.6, 95% CI=2.1-3.1, p<0.001) and hallucinatory experiences (OR=2.1, 95% CI=1.57-2.87, p<0.001) *All adj. age, non-white group, urban residence, recent drug use*	10
Alemany et al. (2012) (Spain)	Cross-sectional General population twins	N=230 (115 twin pairs) (Mean=33.8 SD=13.3)	66.1%	Adapted Adverse Childhood Experiences Questionnaire (ACEQ) (Felitti et al. 1998) – 19 items	Mean=2.0 (SD=2.2) 26.3% report at least one adverse childhood event	CAPE (Stefanis et al. 2002)	Mean 'positive' CAPE=25.3, SD=4.0 Mean 'negative' CAPE=22.1, SD=4.8	Childhood adversity associated with positive CAPE (β=0.45, SE=0.16, p=0.008) and negative psychotic experiences on CAPE (β=0.77, SE=0.18, p<0.001) *adj. sex, age*	9
Bartels-Velthuis et al. (2012) (Netherlands)	Prospective Case-control study	N=337 (56% of eligible baseline participants from Bartles-Velthuis et al 2012 study, N=605) (12-13 Mean=13.1, (SD=0.5)	53.0%	Interview questionnaire – 6 items - Traumatic experiences Stressful events designed for TRAILS study (see Bouma et al. 2008) – 36 life events (yes/no format)	-	For AVH positive – Auditory Vocal Hallucination Rating scale (AVHRS) (Jenner and van de Willige 2002) 3-items on delusional ideation developed by Dunedin study (see Poulton et al. 2000)	N=40 (24%) heard voices at baseline/ persistent group N=15 (9%) – only at follow-up/incident group AVH&delusions group (N=37)	Traumatic experiences and both AVH and delusions (OR=2.01, 95% CI=1.63-2.49, p<0.001) Stressful events and both AVH and delusions (OR=1.26, 95% CI=1.16-1.38, p<0.001)	9
Bentall et al. (2012) (UK)	Cross-sectional Representative sample from general population	N=7353 APMS (Above 16 years)	-	Sexual abuse, Physical abuse, Bullying, separation experiences before age 16 - few items (yes/no)	At least one adverse event 25.8%	PSQ (Bebbington and Nayani 1995)	-	Rape and AVHs (OR=6.09, 95% CI=1.38-26.89, p<0.05) Physical abuse and AVHs (OR=3.82, 95% CI=1.01-14.41, p<0.05) Physical abuse and paranoia (OR=5.99, 95% CI=2.39-15.07, p<0.05) *adj. sex, age, ethnicity, education, social class, IQ*	9

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Lataster et al. (2012) (Germany)	Longitudinal General population study	N=3021 (14-24 Mean=18.4 SD=3.4)	48.5%	Munich Interview for the Assessment of Life Events Trauma section of DIA-X/M-CIDI (yes/no answers)	Early adversity (N=605, 35.1%), Serious physical attacks (N=121, 20.0%), Sexual abuse (N=21, 3.5%), Rape (N=11, 1.8%)	CIDI (Wittchen and Pfister 1997) SCL-90-R (Derogatis 1977) Psychoticism and paranoid ideation subscales of the SCL-90R	N=72 (11.9%) reported psychotic symptoms 5% met criteria for psychotic impairment	Early adversity was associated with T3 psychotic symptoms (RR=1.36, 95% CI= 1.02-1.81, p=0.038) Early adversity associated with T3 psychotic impairment (RR=1.68, 95% CI=1.03-2.72, p=0.036) *Unadjusted binomial regression*	9
Wigman et al. (2012b) (Netherlands)	Longitudinal General population study	N=2230 (At T3 N=1862) (10-16 T3 Mean=16.3 SD=0.7)	T3=52.0%	Life events before 11 (moving, hospitalization, sickness or death, parental divorce, victim of violence, bullying, sexual harassment) (see Wigman et al. 2011b)	-	CAPE (Stefanis et al. 2002)	CAPE mean at T3=25.56	Trauma predicted CAPE score in the second quintile (OR=1.09, 95% CI=1.01-1.18, p<0.001) *adj. parental psychopathology*	9
Cutajar et al. (2010) (Australia)	Prospective cohort Case-control study	N=2759 sexually abused (CSA) before 16 N=2677 peers from general population (Mean=10.22 SD=4.4)	CSA population (79.8%)	Records of Police surgeon's office and the Victorian institute of forensic medicine used to identify cases	All cases	Psychiatric information gathered from stateside register	Rate of psychotic disorders in 37 controls (1.4%) and 78 cases (2.8%)	Child sexual abuse (vs no abuse) and psychosis in general (2.8% vs 1.4%, OR=2.1, 95% CI=1.4-3.1, p<0.001) and schizophrenic disorders (1.9% vs 0.7%, OR=2.6, 95% CI=1.6-4.4, p<0.001)	9
Houston et al. (2008) (USA)	Cross-sectional Representative sample of general population	N=5877 (15 –54 Mean=32.0 SD=10.59)	-	Posttraumatic stress disorder module in CIDI (World Health Organization 1990) (rape and sexual molestation under age of 16)	Any sexual trauma under 16 N=543 (9.2%)	CIDI (World Health Organization 1990) to assess lifetime prevalence of non-affective psychosis, Structured Clinical Interview for DSM-III-R	-	Sexual trauma and psychosis (OR=1.80, 95% CI=0.91-3.57, p=0.09) Cannabis by sexual trauma interaction (OR=6.93, 95% CI=1.39-34.63, p=0.02)	9

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De Loore et al. (2007) (Netherlands)	Prospective cohort Community sample	N=1129 (13/14 at T1 Mean=13.4) (15/16 at T2 Mean=15.1)	53.4%	Bullying (one questions, 5- point scale) Sexual trauma (one question Yes/No)	N=50 (4.5%) bullied N=24 (2.1%) sexually abused	4 yes/no questions from Diagnostic Interview Schedule for Children (DISC-C) (Costello et al. 1982)	N=202 (18%) at T1 met criteria for psychotic outcome	Sexual abuse and subclinical psychotic experiences (OR=3.28, 95% CI=1.32-8.18, p<0.05) *adj. age, gender, educational level, psychosis at T0*	9
Shevlin et al. (2007b) (Ireland)	Cross-sectional Representative sample from general population	N=5877 (15-54 Mean=32.02 SD=10.59)	51.9%	Composite International Diagnostic Interview (CIDI) (World Health Organization 1990) – 4 questions	N=234 (4.0%) neglect N=174 (2.9%) raped under 16 N=486 (8.2%) molested under 16	'Beliefs and Experiences' from Composite International Diagnostic Interview (CIDI) (World Health Organization 1990)	One type of hallucinations (N=671, 11.4%), Two types of hall. (N=228, 3.9%), Three types of hall. (N=92, 1.6%)	Neglect (OR=1.65, sign.), rape (OR=2.37, sign.), molestation (OR=1.62, sign.) related to visual hallucinations Rape (OR=1.75, sign.) and molestation (OR=1.93, sign.) related to auditory hallucinations Physical abuse (OR=1.93, sign.), rape (OR=1.75, sign.), molestation (OR=1.85, sign.) related to tactile hallucinations *Unadj.*	9
Lataster et al. (2006) (Netherlands)	Cross-sectional Representative sample from general population	N=1290 (4-18 Mean=14)	50.8%	Bullying (one questions, 5- point scale) Sexual trauma (one question yes/no)	N=74 (5.8%) reported being bullied N=30 (2.4%) reported sexual trauma	Non-clinical psychotic experience - 4 questions (yes/no) from DISC-C (Costello et al. 1982)	N=224 (19.1%) met the criteria for psychosis outcome	Bullying vs no bullying and non-clinical psychotic experience (OR=2.9, 95% CI=1.8-4.9, sign.) Sexual trauma vs no trauma and non-clinical psychotic experiences (OR=3.8, 95% CI=1.8-8.0, sign.) *All adj. age, gender*	9
Bak et al. (2005) (Netherlands)	Longitudinal general population study	N=4045 (16-64 Mean=41.4 SD=11.8)	47.3%	Yes/no answer and six-point scale (emotional, physical, psychological or sexual trauma before the age of 16)	10.5% reported trauma	CIDI (World Health Organization 1993) SCID-I (Spitzer et al. 1992) MACS (Bak et al. 2001) Two items on positive symptom - Brief Psychiatric Rating Scale	35.5% DSM-III-R diagnostic psychiatric disorder	In the 16 observations of an incident psychotic experience in the absence of distress, the baseline rate of early trauma was low (6%), in 21 observations of an incident psychotic experience with distress was higher (43%, OR=10.0, 95% CI=1.04-96.3, p=0.046) *Unadj.*	9

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Whitfield et al. (2005) (USA)	Cross-sectional Representative sample from general population	N=17337 (Mean=57 SD=15.3)	54.0%	Adverse childhood experiences questionnaire, adapted from Conflict Tactics Scale (CTS) (see Straus and Gelles 1990)	64% reported at least one adverse childhood experience category	One yes/no question on ever experiencing hallucinations	Prevalence of hallucination was 2.0%	Emotional abuse vs no abuse and the risk of hallucination (OR=2.3, 95% CI=1.8-3.0, sign.) Physical abuse and the risk of hallucination (OR=1.7, 95% CI=1.4-2.1, sign.) Sexual abuse and the risk of hallucination (OR=1.7, 95% CI=1.4-2.1, sign.) *All adj. age, race, sex, educational attainment*	9
Daalman et al. (2012) (Netherlands)	Cross-sectional Case-control study	N=127 non-psychotic with AVH (Mean=42.41, SD=32.3) N=124 healthy controls (Mean=42.06 SD=14.39)	Healthy controls 60% Non-psychotic individuals with AVH 59%	Childhood Trauma Questionnaire-Short form (CTQ-SF) (Bernstein et al. 2003)	Healthy controls (PA 1.6%, SA 9.7%, EN 16.9%, PN 10.5%, EA 4.8%), Non-psychotic individuals with AVH (PA 14.3%, SA 29.9%, EN 35.4%, PN 22.0%, EA 31.7%)	Psychotic Symptom Rating Scales (PSYRATS) (Haddock et al. 1999) - Auditory Hallucination Rating Scale (AHRS)	N=127	Non-psychotic individuals with AVH vs controls and association with sexual abuse (OR=2.51, p=0.018), emotional abuse (OR=7.3, p<0.001) *adj. gender,age*	8
Kramer et al. (2012) (Belgium)	Prospective and retrospective methods General population female twins	N=508 (18-46 Mean at baseline=27.1 SD=7.4)	100.0%	Childhood Trauma Questionnaire (CTQ) 25-items (Bernstein et al. 2003)	Mean CTQ=1.7, SD=0.6	The Structured Clinical Interview for DSM-IV Axis I disorders (SCID-I) (First et al. 1997) SCL-90-R (Derogatis 1977) CAPE (Stefanis et al. 2002)	N=61 (12.0%) had one or more SCID psychotic symptoms	Childhood adversity associated with psychotic experiences (on SCL-90-R) (β=0.16, p≤0.001), Childhood adversity associated with psychotic experiences (on CAPE) (β=0.13, p≤0.001), Childhood adversity associated with psychotic experiences (on SCID) (β=0.09, p=0.08)	8
Wigman et al. (2012a) (Germany)	Longitudinal study Representative sample from general population	N=3021 (14-24 at T0, T1=1.6 year later, T2=3.5 years after T0, T3=8.4 years after T0)	at T2= 49.1%	N-section of DIA-X/M-CIDI (Wittchen and Pfister 1997) on trauma and PTSD (9 group of traumatic events)	19% at T2	DIA-X/M-CIDI (Wittchen and Pfister 1997) updated CIDI (World Health Organization 1990)	22% present psychotic symptoms at T2	Trauma vs no trauma and disorders of anxiety/depression with psychotic symptoms (OR=2.13, 95% CI=1.55-2.92, p<0.001) compared to trauma and disorders of anxiety/depression without psychosis ($\chi 2$(1)= 5.92, p<0.015) *adj. age, gender, education*	8
Alemany et al. (2013) *see also Alemany et al. (2011)* **(Spain)**	Cross-sectional Convenience sample from general population	N=533 (Mean=22.9 SD=5.4)	54.6%	Childhood Trauma Questionnaire (CTQ) (Bernstein and Fink 1998)	25.5% exposed to at least one childhood abuse event	CAPE (Stefanis et al. 2002)	40.7% often/ nearly always experienced at least one positive PSE, 47.6% for negative PSE	Childhood abuse and positive psychotic experiences (β=0.088, SE=0.044, p=0.047) Childhood abuse and negative psychotic experiences (β=0.107, SE=0.051, p=0.038) *adj. age, gender, schizotypal personality, trait anxiety*	7

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Kennedy et al. (2013) (USA)	Cross sectional Sample of women prisoners	N=159 (18-62 Mean=33.7 SD=9.71)	100%	Childhood Trauma Questionnaire (CTQ) (Bernstein and Fink 1998)	Childhood physical abuse N=83 (53.9%) Childhood sexual abuse N=75 (48.7%) Both types of abuse of the total sample N=57 (37.0%)	MINI (Sheehan et al. 1997)	N=67 (42.0%) report at least one symptom of psychosis lifetime N=34 (21.8%) psychotic symptoms in past 30 days	Those who reported both sexual and physical abuse in their childhood had more psychosis-like experiences (OR=2.38, p=0.05) Total CTQ score sign. predicted psychosis (OR=1.032, p=0.003) *adj. for race*	7
Dominguez el al. (2010) (Germany)	Longitudinal prospective cohort study Representative sample from general population	N=3021 (14-24 Mean=18.3)	49.26%	Munich Interview for the Assessment of Life Events Trauma section of DIA-X/M-CIDI (Wittchen and Pfister 1997) (yes/no answers)	-	CIDI Brief Psychiatric Rating Scale (Overall J.E. and Gorham 1962)	Positive cluster lifetime (N=574, 22.7%) Negative/ Disorganised cluster baseline (N=274, 12.4%)	Associations of trauma with psychopathological symptom clusters T2&T3: Trauma and Positive cluster (OR=1.50, 95% CI=1.29-1.76, p<0.01) Trauma and co-occurrence of positive and Negative/Disorganised clusters (OR=1.67, 95% CI=1.17-2.40, p<0.01)	7
Livingston et al. (1993) (USA)	Cross-sectional Sexually abused children	N=41 (6-15 Mean=10.2 SD=2.3)	43.9%	Children obtained from the list of all children seen at the paediatrics abuse clinic	100% SA N=26 PA N=15	Diagnostic Interview for Children and Adolescents, version (DICA-6R) (Herjanic and Reich 1997)	6 children reported hallucination, Ideas of reference 17 children (41.5%)	Sexual abuse alone and sexual abuse and younger age predicted ideas of reference	7

Note: OR, odds ratio. CI, confidence interval. sign., statistically significant. K-SADS, Schedule for Affective Disorders and Schizophrenina for School-Aged Children. CIDI, Composite International Diagnostic Interview. PTSD, Posttraumatic Stress Disorder. (Un)adj., (un)adjusted. ALSPAC, Avon Longitudinal Study of Parents and Children. DISC-C , Diagnostic Interview Schedule for Children. DISC-IV, Diagnostic Interview Schedule for Children version IV. $\chi 2$, Chi-squared test. df, degrees of freedom. SES, socio-economic status. β, Beta regression coefficient. SE, standard error. PSQ, Psychosis Screening Questionnaire. CAPE, The Community Assessment of Psychic Experiences. AVH, Auditory Verbal Hallucinations. APMS, The Adult Psychiatric Morbidity Survey 2007. T0, baseline. T1, first measure after T0 (longitudinal study). T2, second measure after T0 (longitudinal study). T3, third measure after T0 (longitudinal study). RR, Relative risk. DSM-III-R, Diagnostic and Statistical Manual of Mental Disorders 3th ed- revised. SCL-90-R, Symptom Checklist-90-Revised. DIA-X/M-CIDI, Munich-Composite International Diagnostic Interview. PSE, Psychotic-like experience. NCS-R, National Comorbidity Survey-Replication Study. PVH, Psychiatric Voice Hearers. NPVH, Non-psychiatric Voice Hearers. EA, emotional abuse. PA, physical abuse. SA, sexual abuse. EN, emotional neglect. PN, physical neglect. PLIKSi, Psychosis-like Symptoms Interview. SCID-I, Structured Clinical Interview for DSM-III. MACS, Maastricht Assessment of Coping Strategies. MINI, Mini International Neuropsychiatic Interview.TRAILS, Tracking Adolescents' Individual Life Survey.

Overall, the association between trauma and psychotic-like experiences ranged from an odds ratio of *1.89* (Spauwen et al. 2006) to *5.20* (Harley et al. 2010) (adjusted for gender, age, SES and family psychiatric history). If at least one traumatic event was reported, this not only predicted an increase in psychotic-like symptoms *(for an odds ratio of 2.0)* (Binbay et al. 2012) but also the persistence of such experiences (Mackie et al. 2011).

All types of childhood trauma were found to be associated with delusional experiences *(OR=2.03)* (Scott et al. 2007), including distress, preoccupation and conviction related to delusions (Kilcommons et al. 2008) as well as persecutory thoughts *(OR=2.52, adj.)* (Freeman and Fowler 2009), increased risk of hallucinations *(e.g. verbal OR=4.75, adj. (Freeman and Fowler 2009), auditory verbal hallucinations OR=2.01 (Bartels-Velthuis et al. 2012))* and paranoia *(OR=2.0, adj.)* (Johns et al. 2004), with similar odds ratios reported for emotional and physical abuse (Fisher et al. 2012). For hallucinations, the most significant risk factor was emotional abuse *(OR ranged from 2.0 (Whitfield et al. 2005) to OR=7.3 (Daalman et al. 2012))*, more than physical *(OR=1.7)* or sexual *abuse (OR=1.7)* (Whitfield et al. 2005). Nevertheless, sexual trauma also showed a robust association with subclinical psychotic experiences with an estimated odds ratio of *2.1* (Cutajar et al. 2010) or *3.28* in another study (De Loore et al. 2007; similar also to Lataster et al. 2006) or even *7.4* (Bebbington et al. 2004). Similarly, the association was documented for physical abuse and subclinical psychotic experiences at *OR=2.27* (Shevlin et al. 2007c) or *OR=5.06* (Kelleher et al. 2008). In addition, the evidence supports the association between neglect and psychotic-like experiences *(OR=2.40)* (Shevlin et al. 2007c) and bullying and psychotic-like symptoms *(OR=2.8)* (Mackie et al. 2012), where interestingly being a bully resulted in increased risk of psychotic-like experiences with a high odds ratio of *9.90* (Kelleher et al. 2008).

2.2.3　　　Discussion

Distinct trauma types and specific psychotic-like experiences

A large community study reported a 3.6–fold increase in subclinical positive symptoms in individuals who experienced childhood abuse, with a dose-response effect (Janssen et al. 2004). Specifically, physical, sexual and emotional abuse among individuals at high risk for clinical psychosis were related to positive symptoms, especially grandiosity and suspiciousness (Thompson et al. 2009) with another study reporting associations between childhood trauma and paranoid tendencies (Gracie et al. 2007;Thompson et al. 2009) as well as increased perceptual aberration, which were ten times more common in adults who were maltreated as children (Startup 1999). There is also evidence that individuals with a reported history of childhood abuse showed increased scores on paranormal beliefs and paranormal experiences (Irwin 1992), which can be seen as a result of childhood fantasy *(and traumatic events partially cause heightened fantasy)* (Lawrence et al. 1995).

Furthermore, data shows that other types of childhood victimisation like being a victim of bullying have a significant impact on the development of non-clinical psychotic experiences in young adolescents *(approx. a twofold risk)* (Arseneault et al. 2011;Bebbington et al. 2004;Kelleher et al. 2008;Lataster et al. 2006;Mackie et al. 2011;Schreier et al. 2009), especially chronic and severe victimisation (Schreier et al. 2009). Bullying relates to increased risk of persecutory delusions (O'Moore et al. 1998), visual hallucinations, dissociation, and paranoia in a linear fashion (Campbell and Morrison 2007;Hardy et al. 2005) - growing with the severity, frequency and persistence of this type of trauma (Lataster et al. 2006;Schreier et al. 2009). Even though it can be argued that some children might have predisposed vulnerabilities that put them at increased risk to be a victim of

bullying, the association between childhood victimisation and psychotic symptomatology was significant even when accounting for genetic or psychosocial vulnerabilities (Schreier et al. 2009). Interestingly, the risk of psychotic symptoms increased even more if an individual was bullied and bully/perpetrator at the same time ('bully-victim') (Kelleher et al. 2008). However, it was also hypothesised that it is especially severe traumas that potentially lead to psychosis-like symptomatology, more so than repeated and context-specific types of traumas such as bullying (De Loore et al. 2007)

A recent meta-analysis (Varese et al. 2012b) of the literature on childhood adversities in relation to risk of psychosis that included nonclinical and clinical populations showed a strong overall effect of *OR=2.78 (95% CI=2.34-3.31; for case-control studies OR=2.99, 95% CI=2.12-4.20; for population based cross-sectional studies OR=2.75, 95% CI=2.17-3.47)*. The association observed was significant for all types of trauma included (sexual, emotional and physical abuse, neglect, bullying, separation from a parent) with the exception of parental death (Varese et al. 2012b). There is however some evidence for parental death showing a two-fold increase in risk for developing psychotic disorder (Morgan et al. 2007) and especially loss of mother has been linked to more severe adult psychopathology (Brown et al. 1977;Roy 1985). Although a long-term separation shows an association with increased risk for psychosis (Morgan et al. 2007), this type of trauma might be more an indicator of other associated factors e.g. single parenting, socio-economic status (Wicks et al. 2005).

When looking at associations between childhood trauma and psychotic-like experiences in community samples *(Table 4)*, cumulative dose-response effect of traumas on psychotic-like symptoms was widely documented (Janssen et al. 2004;Whitfield et al. 2005) reflecting the findings for clinical psychosis (Kilcommons and Morrison 2005;Rubino et al. 2009;Schenkel et al. 2005). The effect of multi-victimisation was observed by Kennedy and colleagues (Kennedy et al. 2013) who

found that individuals who experienced more than one type of trauma were approximately twice as likely to report psychotic-like symptoms. Moreover, another study observed that maltreatment or bullying were associated with development of psychotic symptoms with an odds ratio of *3.27*, in case both maltreatment and bullying were present the risk significantly increased to an odds ratio of *5.68* (Arseneault et al. 2011). In contrast to some previous findings, one study did not support the dose-response effect of multi-victimisation (Fisher et al. 2010). In parallel, it was further suggested that the odds for the development of psychotic symptoms increase with the number of adversities in a nonlinear way (the odds do increase but have a declining rate), implying that when a certain number of adversities is reached, the additional effect of another trauma is considerably less powerful (Benjet et al. 2010).

A recent study (Alemany et al. 2012) used a sample of monozygotic twins and found that the childhood trauma and psychosis association does not exist due to genetic confounding. Therefore, these early traumatic experiences might truly stand as a risk factor for psychotic-like symptomatology (Arseneault et al. 2011). It was also hypothesised that the onset of psychotic-like experiences in early adolescence may increase risk for psychotic disorder later in life (Lataster et al. 2006). For example, children who reported hallucinations and delusions at the age of 11 had an increased likelihood by 16-times to develop schizophreniform disorder by the age of 26 than those who did not display these experiences (Poulton et al. 2000).

Childhood trauma and hallucinations and delusions

As previously suggested, childhood trauma has been found to be associated with a predisposition to hallucinations and delusions (e.g. Scott et al. 2007;Shevlin et al. 2007a;Spauwen et al. 2006;Whitfield et al. 2005). The largest study to date

supported the increased risk of hallucinations (1.2 to 2.5-fold) by any of the eight types of childhood trauma measured (Whitfield et al. 2005). Childhood abuse, especially sexual (Kilcommons and Morrison 2005;Ross and Joshi 1992) and emotional abuse (Daalman et al. 2012) as well as neglect are particularly associated with auditory hallucinations (Andrew et al. 2008;Honig et al. 1998;Read et al. 2003;Ross et al. 1994;Shevlin et al. 2007a;Whitfield et al. 2005), as well as beliefs about voices being more malevolent, omnipotent and benevolent (Andrew et al. 2008). Literature also supports a link between childhood trauma and tactile hallucinations (Shevlin et al. 2007a) as well as visual hallucinations (Morrison and Petersen 2003;Ross et al. 1994;Shevlin et al. 2007a), even though these findings have not been consistent (Hammersley et al. 2003). In another study, four types of childhood trauma (physical abuse, neglect, rape and molestation) predicted the increase in visual hallucinations while tactile hallucinations were associated with physical and sexual abuse (Hardy et al. 2005;Shevlin et al. 2007a). Moreover, not only incidence but also persistence and severity of hallucinations were related to early childhood adversity (Bartels-Velthuis et al. 2012). Also, the intrusiveness of these hallucinatory experiences was shown to precede the formation of delusions (Bartels-Velthuis et al. 2012;Escher et al. 2002a). In general, those who reported childhood traumatic events were twice as likely to endorse delusional-like experiences (Read et al. 2005;Saha et al. 2011;Spauwen et al. 2006;van Os et al. 1999), similar for paranoid thoughts, and a five times greater likelihood of verbal hallucinations (Freeman and Fowler 2009). The association was also documented for childhood trauma and Schneiderian symptoms not only in clinical samples (Fink and Golinkoff 1990) but also in the general population studies (Ross and Joshi 1992).

Sexual trauma especially has received a lot of attention in previous studies, with high levels reported in clinical/psychosis samples (Bendall et al. 2008;Briere et

al. 1997;Friedman and Harrison 1984;Honig et al. 1998;Lysakera and LaRocco 2008;Whitfield et al. 2005) and ultra-high-risk groups (Thompson et al. 2010). Childhood sexual abuse, especially involving sexual intercourse, showed a strong association with psychosis in a large general population sample (Bebbington et al. 2011). Similarly, in another study abuse in childhood or adulthood predicted hallucinations and delusions which were associated with all types of trauma, but again the relationship was particularly strong in those who experienced rape (Scott et al. 2007). In addition, children and adolescents sexually abused by more than one perpetrator reported 15 times greater risk for psychosis than those without these experiences (Cutajar et al. 2010). The interaction effect between trauma and gender was also observed, as for males the link between being a victim of sexual abuse and psychotic-like symptoms was not supported (Bebbington et al. 2011), however not all studies found the differential effects of gender in this association (Shevlin et al. 2011). The link between sexual trauma and psychotic symptoms is probably not only reflected in symptom form *e.g. hallucinations (Whitfield et al. 2005), delusions (Calvert et al. 2008)* but also in the symptom content (Hardy et al. 2005;Lysakera and LaRocco 2008). Consistent with these findings, higher rates of subclinical psychotic symptoms of a sexual content were observed in individuals reporting sexual abuse, including hallucinations with sexual content (Hardy et al. 2005) or *'flash-back elements and more symbolic representations'* of traumatic experiences (Ensink 1992 p.126). Also, hallucinations might even reflect some concrete details of the abuse itself (Famularo et al. 1992). Similar relationships with such symptoms and their content (to a lesser degree) can also be observed for physical trauma (Thompson et al. 2010). The effects that sexual abuse has in terms of the symptom content is also evident in command hallucinations, especially hallucinations to harm or kill themselves commonly experienced in those reporting sexual abuse (Andrew et al. 2008;Read et al. 2003).

However, these types of hallucinations might also result from the underlying beliefs in the lack of control over the actions of others (Stueve et al. 1998) or the feeling that others cannot be trusted (Morrison 2001). Again this further highlights that childhood trauma significantly predicts voice characteristics and as such is an important aetiological factor not only in the development of psychotic symptomatology but also in maintaining these symptoms (by emotionally charging subject's beliefs about the voices) (Andrew et al. 2008;Read et al. 2003). Therefore, the subject's beliefs about the voices need to be understood in the context of the nature of the trauma, its meaning for the individual and the extent to which the trauma has remained unresolved (Andrew et al. 2008;Sommer et al. 2010). The emotional content of the hallucinations might even provide the main distinction between clinical psychopathology and more benign hallucinations as voices of the healthy individuals are found to have a predominantly positive emotional content (Honig et al. 1998).

Similarly, in terms of delusional content, it was documented that for individuals with psychosis and a history of childhood abuse, delusions had a sexual nature comprising of sexual grandiose themes such as 'pseudotranssexual' delusions like no longer belonging to one's sex or belonging to both sexes, delusions of sex change (Borras et al. 2007), erotomanic symptoms (Phillips et al. 1996), delusional jealousy (Soyka et al. 1991), false beliefs of marriage or pregnancy or paranoid ideas such as imposed intercourse (Lucas et al. 1962). Evidence from epidemiological studies in community populations indicated that the association between childhood trauma and hallucinations is mainly determined through paranoid perception of the world *(observed through scores on schizotypal and delusional scales),* which subsequently stands as a risk for auditory visual hallucinations (Freeman and Garety 2003;Garety et al. 2001). The increased levels of paranoia/suspiciousness were found in individuals with a history of sexual and

physical abuse, with both types of trauma also showing an association with unusual perceptual experiences and thought disorder (Startup 1999;Steel et al. 2009).

The role of other factors impacting trauma and psychotic-like symptoms association

As previously discussed, negative/disorganised symptoms were found to be associated with alterations in brain development *(therefore a genetic risk)* (Goldman et al. 2009;Heckers et al. 1999;Rowland et al. 2009), which in turn effect the development of positive symptomatology (Dominguez et al. 2010;van Os and Kapur 2009). Consistent with this proposition (Dominguez et al. 2010) it was found that negative/disorganised dimension was associated with younger age, male sex, single marital status and low educational level; positive symptoms on the other hand were particularly influenced by environmental risk factors (childhood trauma, cannabis exposure, urbanicity), suggesting two distinct pathways to psychosis *(biological risk vs environmental influences)* (Ross et al. 1994). There is emerging evidence supporting the gender-specific mechanisms underlying the association between childhood trauma and psychosis-like experiences (Myin-Germeys and Van Os 2007). Males in general show less severe responses after a traumatic experience than females, which can either be explained by exposure to different types of traumas than females, the younger age at the time of exposure, different appraisals of threat or even gender-specific acute psychobiological reactions to trauma (Olff et al. 2007). Furthermore, beside gender-specific pathways, the differential effects of distinct trauma types on psychotic-like symptomatology have been documented. For example, harsh hitting (including maternal hitting, preschool and school hostility) was fully accounted for by depressive symptoms, level of anxiety, external locus of control and low self-esteem while bullying and exposure to domestic violence were only partially mediated by these factors (Fisher et al. 2013b).

In summary: The associations observed were significant for different types of traumatic experiences e.g. sexual abuse, emotional abuse, neglect, physical abuse, bullying, and separation from a parent. Early trauma was linked to an array of psychotic-like experiences such as delusions, hallucinations, paranoia, suspiciousness etc. Many studies observed a dose-response association between trauma and psychotic-like symptoms, in parallel with studies exploring trauma-schizotypy association. Especially severe forms of abuse, lasting for extended period of time showed the most robust association with psychotic-like experiences. There is also some evidence that childhood trauma not only impacts the symptom form but also predicts the symptom content. Moreover, the importance of other factors influencing associations between childhood trauma and psychotic-like experiences was highlighted e.g. the age of individual at the time of trauma occurrence, and gender-specific pathways underlying these associations.

2.3 Possible pathways underlying the childhood trauma – schizotypy association

The section will explore the possible pathways that account for the association between childhood trauma and schizotypy (or psychotic-like symptoms). Different models will be presented and linked to the integrative model – traumagenic neurodevelopmental model. The role of other social *(e.g. recent life events, cannabis use)* and psychological factors *(negative beliefs about others/self, PTSD, dissociation)* as possible mediators/moderators of childhood trauma-schizotypy association will also be explored.

Literature on vulnerability to psychopathology has conceptualised vulnerability as either a stable trait, the endogenous nature of vulnerability or focused on the role of stress in its manifestation (Ingram and Price 2010). Zubin and Spring (1977) advocated the trait-like vulnerability of schizophrenia and placed genetic factors as core determinants of individuals' vulnerability levels. Nevertheless, the stability of vulnerability does not have to imply the influence of genetic factors only (Ingram and Price 2010), as exposure to environmental risks (e.g. childhood trauma) may interact with genetic predispositions, thus shaping the vulnerability factors. *Figure 4* presents factors predictive of psychosis-proneness in a diagrammatic way, with the crude division between biological and environmental influences. This does not necessarily suggest two distinct pathways as there is often interplay between biological and environmental influences that makes it very difficult to disentangle them. Also, early and late childhood are presented separately only for the ease of the presentation but not to imply evidently distinctive separation. Markers of vulnerability in early childhood reflect deficiencies in early cognitive development (Barnett et al. 2012), poor school performance (Sitskoorn et al. 2004) and abnormal premorbid social adjustment (Larsen et al. 2004). Brain deficits and cognitive impairments have been observed in schizophrenia patients through reduction in grey matter volume within the hippocampus and amygdala (Cannon 1998), childhood motor abnormalities (Walker et al. 1994), attention deficits (Cornblatt et al.

1999), and deficits in working memory (Aleman et al. 1999) amongst others. These early cognitive impairments have been linked to specific genes (e.g Cannon et al. 2005) and their manifestation is dependent on maturation processes (Brennan and Walker 2010). Biological or neuropsychological vulnerability markers of genetic liability to psychosis are also conceptualised as 'endophenotypes' and found at increased rates in unaffected relatives of schizophrenia patients compared to the general population (Gottesman and Gould 2003). Late childhood/early adolescence in the 'psychosis-proneness model' is characterised by impairments in social- (Done et al. 1994) or intellectual functioning (Reichenberg et al. 2006) or observed in specific personality traits (implying the importance of individual differences in prediction of schizophrenia spectrum disorders) (Claridge 1997;Meehl 1962). For example, in a ten-year longitudinal study (Kwapil et al. 2013), personality traits such as perceptual aberration, magical ideation, social and physical anhedonia predicted the development of psychotic-like and paranoid symptoms.
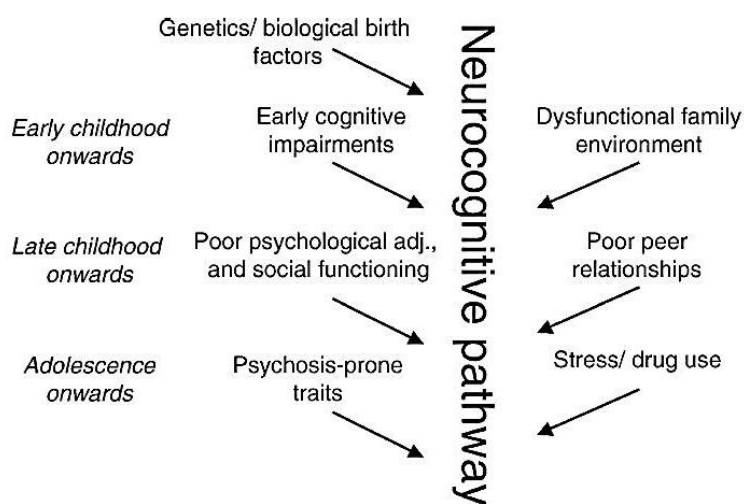
Figure 4: Diagrammatic presentation of factors predicting psychosis-proneness, biological factors on the left-hand side and environmental on the right (taken from Mason and Beavan-Pearson 2005 p.3).

Although defining vulnerability as an endogenous and latent factor suggests that the locus of vulnerability lays within the person (not easily detectable), it is the presence of a stressful/traumatic event that makes identification of vulnerability markers

possible (Ingram and Price 2010). Such environmental influences include social adversity (Wicks et al. 2005), early traumatic experiences (Morgan et al. 2006) or dysfunctional family environment all interacting with genetic predispositions (Tienari et al. 2004). In addition, other factors have been proposed to influence the development of psychosis-like symptomatology such as migration (Morgan et al. 2010), urban upbringing (Krabbendam and van 2005) and cannabis use (Kuepper et al. 2011). Cannabis use was documented as a risk factor for psychosis, as the review of the literature suggests it is likely to precede the schizophrenia-spectrum disorders (Arseneault et al. 2004). However others hypothesised that it might be used as a 'self-medication' to alleviate the negative symptoms (Dixon et al. 1990) or stands just as a proxy of poor premorbid adjustment (Arseneault et al. 2004), which is also associated with psychotic disorders (Cannon et al. 2002).

Even though stress does not represent a core feature of vulnerability, it is suggested that the manifestation of the vulnerability is stress-dependent (Ingram and Price 2010). As a result, vulnerability processes reflect dynamic interactions between genetic predisposition and environmental factors throughout the lifespan (Price and Zwolinski 2010). The early genetic liabilities/neurocognitive impairments are beyond the scope of this thesis, therefore the next chapter focuses primarily on the environmental pathway *(with addition of life events later in individual's life)*, by discussing possible underlying mechanisms of the childhood trauma and schizotypy association.

2.3.1 Hypothesised theoretical models

Several models have been proposed explaining the relationship between childhood adversity and psychosis. Overall, they have focused on either biological underpinnings or psychological processes; however different approaches are

neither completely distinguishable nor mutually exclusive, suggesting the need for integrated models. *Figure 5* gives an overview of the hypothesised sociodevelopmental and neurodevelopmental pathways to psychosis.

Biological models

The literature suggests that childhood trauma may contribute to the biological pathophysiology of psychotic disorders (for review of the literature see Holtzman et al. 2013), via influencing the development of the hypothalamic–pituitary–adrenal (HPA) axis and persistent alterations in neuro-endocrine cells (Walker and Diforio 1997), leading to an acquired vulnerability of enhanced sensitivity to stress (Heim et al. 2000;Read et al. 2001). Therefore, exposure to trauma might lead to alterations in neurobiological mechanisms via changes in release of stress hormones (Garner et al. 2011;Walker et al. 2008). Also, dysregulation of dopaminergic pathways (De Bellis et al. 1999;Depue and Collins 1999) has been advocated to provide a possible link between increased stress sensitivity and development of psychosis. Teicher and colleagues (2003) reported how the early stress leads to several structural and functional neurobiological changes (e.g. underdevelopment of the corpus callosum, amygdala, and hippocampus).

The observation that emotional stress reactivity is observed in individuals with early childhood trauma (Glaser et al. 2006) parallels the evidence that the most damaging effects of trauma are associated with trauma occurrence at a younger age (Fisher et al. 2010). On the other hand, positive symptoms (Garety et al. 2001), including delusions (Freeman et al. 2002) have shown some specific mechanisms in the interaction between vulnerability and stress, suggesting that basic deficiencies in cognitive functioning *(decreased inhibition and deficient self-monitoring skills)* might influence cognitive and perceptual changes, leading to anomalous conscious experiences *(e.g. heightened perception, thoughts experienced as voices, thoughts*

appearing to be broadcast, racing thoughts, unintended actions) (Garety et al. 2001).

Psychological models

There is not only evidence that cumulative stressful events (dose-response association) affect the development of psychotic symptoms (Janssen et al. 2004;Schreier et al. 2009;Shevlin et al. 2007b;Spauwen et al. 2006) but also that interaction between childhood and adulthood victimisation (Briere et al. 1997) increases emotional reactivity, rendering individuals vulnerable to psychotic-like experiences by increasing their emotional reactivity to stressors - the *'affective pathway to psychosis'* (Myin-Germeys et al. 2001). This *'affective pathway'* or *'stress related model'* to psychosis stands in contrast to a *'non-stress pathway'* which reflects more cognitive impairments that present the core genetic vulnerabilities for schizophrenia (Myin-Germeys et al. 2002). Moreover, psychotic symptoms, although different from mood and anxiety are considered to be emotion driven, so it is argued that not the event per se, but the emotions aroused by them increase these symptoms (Docherty et al. 2009). Complementing this suggestion, greater levels of depressive symptoms were documented in nonclinical adolescent populations with auditory and visual hallucinations compared to unaffected controls (Fonseca-Pedrero et al. 2010;Scott et al. 2009a). On the other hand, the association between negative symptomatology and childhood trauma (e.g. Alemany et al. 2012;Myin-Germeys et al. 2011;Rossler et al. 2007) is more difficult to understand. Schurhoff and colleagues (2009) hypothesised that traumatic avoidance, emotional numbing and reduced responsiveness may only resemble negative symptoms but they are actually solely reactions to the traumatic event.

However, in addition to trauma leading to the neurobiological alterations (biological models) it can also predispose to the pathogenic appraisals

(psychological models) which in turn impact the development of psychotic symptoms. Theory suggests that early traumatic experiences may underlie the cognitive vulnerability to psychosis, manifested as negative schemas about the self and others, also fuelling content for psychotic attributions (Lysaker et al. 2005). Thus, after a traumatic event thoughts and beliefs can take a negative turn, leading to a faulty perception of the self and/or others which triggers psychotic symptoms via the interpretation of these intrusions (Dunmore et al. 1997;Dunmore et al. 1999;Kilcommons and Morrison 2005;Morrison 2001;Morrison et al. 2003). As such, negative beliefs about self and others stand as the main mediators of the association between trauma and paranoia (Gracie et al. 2007), also supported by negative emotions, especially anxiety (Freeman and Fowler 2009), mistrust and suspiciousness which further deepen the feelings of threat and paranoia symptoms *('cognitive model of psychosis')* (Freeman et al. 2002). This relationship was also recognised in a large non-clinical sample (Fowler et al. 2006), where paranoid world view was particularly prevalent in those reporting interpersonal trauma (Lovatt et al. 2010). Similarly, in another general population study negative beliefs about self and anxiety levels partially accounted for the association between emotional and physical abuse and paranoia (Fisher et al. 2012). In addition, 'need for care' was not associated with the number of traumatic events but rather influenced by distress and personalizing appraisals following these experiences (Lovatt et al. 2010).

However, the association between early trauma and hallucinations were not explained by these mediators (Freeman and Fowler 2009). Also, according to the cognitive model the unique effects of specific trauma types on psychotic-like experiences would solely depend on how negative schema are formed following the traumatic event. As such, auditory, visual or tactile hallucinations could be predicted by any trauma type, merely depending on the associated negative evaluations (Shevlin et al. 2007a). In contrast, some differential effects of specific trauma types

on hallucinatory experiences were observed e.g. only sexual trauma associated with auditory hallucinations (Ross et al. 1994), no association between neglect and tactile hallucinations (Shevlin et al. 2007b), possibly supporting the dissociative hypothesis (Kilcommons and Morrison 2005) which accounts for the different effect of individual trauma types *(for the role of dissociation see section 2.3.3).*

As shown, the experience of abuse may affect a biological and psychological (Garety et al. 2001) vulnerability for the development of psychotic-like symptoms. Furthermore, exposure to childhood trauma may not only be predisposing individuals to developing negative evaluations of the self and/or world but may also be contributing to appraisals of psychological, perceptual or bodily experiences being outside of their control (Bak et al. 2005). Also, individuals with early traumatic events are more likely to identify their voices as being more powerful and difficult to control (Birchwood et al. 2000). This is not surprising considering the autobiographical nature of cognitive schemas (reflecting subject's past and current experiences) that impact the evaluations of malevolence or benevolence of the voices (Chadwick and Birchwood 1994).

In addition, literature evaluating the relationship between peer victimisation and psychotic-like experiences (e.g. Kelleher et al. 2008;Lataster et al. 2006;Schreier et al. 2009) stress the importance of stability and severity of internalizing and externalizing behaviour which is also associated with increased risk of psychotic symptomatology (Scott et al. 2009b). Additionally, dysfunctional responses or maladaptive coping strategies are more likely to be observed in individuals exposed to childhood adversity (Cohen et al. 1996) possibly resulting in psychotic symptom formation (Bak et al. 2003;Garety et al. 2001). Similarly, early trauma may severely disrupt the ability to trust and form attachments with others (*as pioneered by Bowlby (1969))* impeding social functioning as well as intimate relationships (Liem and Boudewyn 1999), that can also be linked to low self-esteem

(Fleming et al. 1999;Mullen et al. 1996), feelings of guilt or even self-blame (Liem and Boudewyn 1999). However, another plausible explanation is that any link between childhood trauma and dysfunctional responses in relation to psychotic-like symptoms is mediated by personality traits (Bak et al. 2005).

Integrative models

The traumagenic-neurodevelopmental (TN) model (Read et al. 2001) attempted to incorporate these various hypothesised theories linking childhood trauma to psychosis. The model proposes that sufficiently severe trauma can contribute to abnormal neurodevelopmental processes (such as permanent changes in HPA axis, dopamine irregularities, structural brain abnormalities), creating the vulnerability/oversensitivity to stresses later in life. Read et al. (2001) argued that 'stress-diathesis'/'bio-psychosocial' model (Zubin and Spring 1977) lacks in consideration of the complex interactions between biological, social and psychosocial factors. The core of the 'stress-diathesis' model are genetic underpinnings (genetic deficit) and their interaction with environmental risks/stresses that triggers the onset of the disorder. Read et al. (2001) moves beyond the 'oversensitivity to stress' and helps with understanding the cognitive impairments observed in traumatised children, pathways to positive and negative symptoms and the role of dissociative symptoms. A recent literature review reported robust indirect and direct support to the traumagenic-neurodevelopmental model (Read et al. 2014). Also, according to Read and colleagues (2005) a fully integrated model that would explain pathways from early trauma to psychosis combines a TN model and Kapur's (Kapur 2003) proposition of dysregulation of the dopaminergic system. The increase of dopamine release leads to the aberrant assignment of salience to external events and internal representations (causing formation of delusions and

hallucinations – *further explained in the following sections*) and creates lasting vulnerability to later stresses. However, the TN model has its weaknesses – not all traumatised children will develop psychosis and early trauma has been linked to a variety of adult psychopathology *(non-specificity to psychosis),* requiring further research assessing the complex pathways between early trauma and psychosis, also incorporating other mediating or moderating factors.
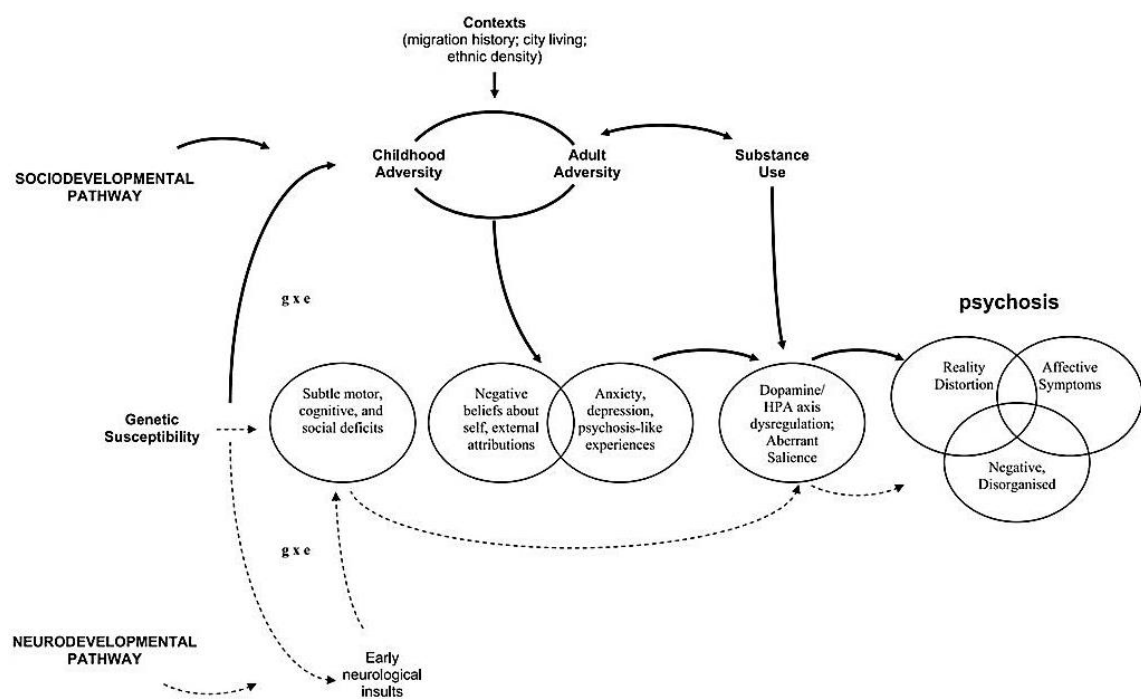
Figure 5: Hypothesised Sociodevelopmental and Neurodevelopmental pathways to psychosis (taken from Morgan et al. 2010 p.661).

2.3.2   The effects of adult traumatic experiences /Life events

There is a lot of evidence highlighting the importance of *environment–environment* correlation (early trauma increases the risk of exposure to adulthood trauma, thus elevating risks for psychotic-like symptoms) and *environment by environment interaction* (exposure to early trauma heightens sensitivity to adult/recent adversity, thus effecting psychotic outcomes) (Collip et al. 2008;Lataster et al. 2012;van Winkel et al. 2008). A recent systematic review of the literature (Beards et al. 2013) indicated overall support of the association between adulthood adversity and increased psychotic and subclinical psychotic experiences, however it stressed the methodological limitations of these studies. Most importantly, a sample of first-episode psychosis patients indicated that childhood trauma and adult disadvantage had synergistic effects in pathways to psychosis (Morgan et al. 2013).

Stressful life events relate to a negative change in individual's stable life pattern (Brown and Harris 1978). These events are not necessarily uncommon and they can either be effected by the individual's own behaviour and actions (for example a divorce) or occur independent of individual's control (for example death of a loved one) (Lazarus and Folkman 1984). Childhood abuse is widely cited in the literature as a risk factor that predisposes to the development of psychosis, psychotic-like symptoms (Arseneault et al. 2011;Bebbington et al. 2004;Hardy et al. 2005;Saha et al. 2011;Shevlin et al. 2007b) as well as high levels of schizotypal traits (Afifi et al. 2011;Berenbaum et al. 2008;Powers et al. 2011;Startup 1999;Steel et al. 2009). However, adverse life events later in life may also trigger traumatic intrusions *(leading to hallucinations etc.)* (Morrison and Petersen 2003). Especially prone to these traumatic intrusions are individuals with higher levels of schizotypy (Steel et al. 2005), also due to poor social and cognitive skills that influence the perception of traumatic events as more unpleasant and difficult to cope with when

compared to individuals with lower schizotypal levels (Bebbington et al. 2004). Additionally, in a general population sample, there was an association observed between traumatic life events and elevated schizotypy dimensions, especially cognitive disorganisation (Kocsis-Bogar et al. 2013). One of the plausible explanations for this association is that life events and daily life hassles in adult life *(re-victimisation)* may re-activate earlier childhood memories and re-traumatize the individual (Honig et al. 1998;Read et al. 2003). Furthermore, individuals with higher positive schizotypy might relive their past experiences with more sensory details associated with a more profound subjective feeling (Winfield and Kamboj 2010). These sensory features especially stimulate the reliving of traumatic experiences from childhood as well as adulthood life events. Beside the memories being strong, they may also be dissociated *(detached from physical or emotional experiences)* from their origin and time perspective is lost (Ehlers and Clark 2000;Steel et al. 2002).

There was also an association reported for negative life events and the development *(an overall weighted OR of 3.19; 95% CI 2.15–4.75) (Beards et al. 2013)* and maintenance of clinical psychosis, as well as increased relapse (Bebbington et al. 1993;Docherty et al. 2009;Lukoff et al. 1984). Likewise, a similar association was found for life events and deterioration of prodromal symptoms in high-risk groups as well as subclinical psychotic symptoms in the general population (Lincoln et al. 2009). Nevertheless, the mechanistic pathways underlying this relationship still remain unclear (Lincoln et al. 2009;Myin-Germeys and Van Os 2007). Early studies stressed the significance of the number of life events in the three months period prior to the onset of psychosis, including the association with independent events in the three week period prior the onset (Brown and Birley 1968;Leff et al. 1983) and the peak of life events one month prior to a psychotic relapse (Ventura et al. 1989). Since then, many studies have failed to show the

same results, maybe also due to methodological limitations (Canton and Fraccon 1985;Chung et al. 1986;Fallon 2008;Jacobs and Myers 1976;Malla et al. 1990). In contrast, the *Camberwell Collaborative Psychosis Study* (Bebbington et al. 1993) reiterated the significance of the excess of severe life events in the six month period prior to the onset of psychosis, but especially in the three months preceding the disorder. Another prospective study described the importance of the last 12 month period prior to relapse occurrence, along with a suggested cumulative effect of these stressful events (Hirsch et al. 1996).

Some authors (Brown et al. 1973;Brown and Harris 1978;van Os et al. 1994) have proposed two distinct ways in which these events relate to the onset of psychosis: a 'formative role', in which events have more fundamental aetiological importance and a 'triggering role' where life events mainly exacerbate the pre-existing genetic vulnerability. The 'formative' role has been associated with more affective psychosis (with a marked effect of the events on the illness) whereas the 'triggering role' has been linked to schizophrenia, also implying that the impact of life events in schizophrenia-spectrum might only have modest effects (van Os et al. 1994). The symptom-specific effects of life events were also documented, with the largest impact on depressive symptoms in individuals with psychosis when compared to positive symptoms like hallucinations and delusions (Norman and Malla 1991;Schwartz and Myers 1977;Ventura et al. 2000). Depressive symptomatology resulted from interpersonal-related stressful events in particular (Pagano et al. 2004). Nevertheless, there is not much evidence for these symptom-specific effects of life events, hence more research is required to make any firm conclusions (Fenton and McGlashan 1994).

In addition, chronic stress might hold the key to the development of psychosis (Zubin and Spring 1977) and contribute to symptom exacerbation (Day et al. 1987;Norman and Malla 1993) or psychosis relapse (Nuechterlein et al. 1994).

There also appears to be a link between the frequency of exposure to independent traumatic events and relapse among schizophrenia patients (Malla et al. 1990). However the most robust evidence supports the casual role of more severe events occurring outside of the individual's control (Brown and Harris 1978;Cullberg 2003;Das et al. 2001;Day et al. 1987;Stueve et al. 1998). Similarly, individuals with schizotypal personality disorder reported more life events, particularly undesirable and independent life events than healthy controls (Tessner et al. 2011). Importantly, it is not just large significant events that influence psychotic symptoms but also minor life events or daily hassles that are associated with symptom exacerbation (Norman and Malla 1994;Norman and Malla 2001), causing more distress to those with schizotypal personality disorders compared to their peers (Tessner et al. 2011). The sensitisation associated with everyday hassles is particularly reflected in increased risk for hallucination and delusions (Myin-Germeys and Van Os 2007). The mechanisms supporting such associations are explained in the *'vulnerability-stress'* model proposed by several authors (Boker et al. 1989;Meehl 1962;Nuechterlein et al. 1994;Zubin and Spring 1977). As explained in the previous section, the model postulates that people have varied degrees of vulnerability to the development of schizophrenia but the manifestation of the symptoms is influenced by the amount of stress individuals encounter. Furthermore, it is not just when the threshold of stressors exceeds genetic vulnerability (Lataster et al. 2010) thus influencing the development of psychosis, but stresses in early life may contribute to the emergence of psychosis by emphasising the vulnerability state (Lardinois et al. 2011;Lataster et al. 2012;Read et al. 2001), as suggested in the TN model. Consequently, with the cumulative effect of stressors in adult life psychosis is triggered (Hirsch et al. 1996;Myin-Germeys et al. 2003a).

Similar to the underlying pathways supporting the childhood trauma and psychosis association, the involvement of the HPA-axis in development of psychotic

symptoms is evidenced in relation to adult life events (Corcoran et al. 2003;Walker and Diforio 1997) – either contributing to dysregulation of physiological stress response (Read et al. 2005) or alternation of the dopamine release mechanisms (Davis et al. 1991;Laruelle 2000). Also, traumatic experiences from childhood can lead to brain changes, altering the stress sensitivity to daily life events later in life (Glaser et al. 2006). The over-activity of the HPA axis has not only been demonstrated in psychotic patients (Cotter and Pariante 2002;Walker and Diforio 1997) but also in ultra-high-risk groups and individuals with schizotypal personality disorder (Garner et al. 2009;Walker and Diforio 1997). Some studies described the impact that adverse life events have on hippocampal volume (Kronmuller et al. 2008;Vythilingam et al. 2002), independent from- but moderated by the disorder e.g. smaller hippocampal volume was observed in traumatised individuals comparing to those without traumatic events but for those who developed PTSD even smaller hippocampal volume was found (Karl et al. 2006;Smith 2005). These findings, however, are not consistently reported in the literature (Cohen et al. 2006).

In terms of psychological pathways (and consistent with the childhood trauma–schizotypy association), cognitive and affective routes have been suggested (Garety et al. 2001). According to this model, a triggering event influences a disruption in cognitive processes and emotional changes/increased reactivity (Myin-Germeys et al. 2003a). Consequently, that leads to a biased conscious appraisal of these experiences by externalising the source of internal experiences, making an anomalous experience psychotic (Baker and Morrison 1998;Bentall et al. 2001). As such, maladaptive cognitive appraisals have been the central focus particularly in theories about hallucination formation (Garety et al. 2001). Following the stressful incident the pre-existing dysfunctional schemas are activated, such as beliefs about perceived danger or uncontrollability of thoughts, leading to negative cognitive biases and associated distress (Garcia-Montes et al. 2006;Laroi and Van der Linden

2005;Morrison et al. 2007). The correlation between metacognitive factors and hallucination proneness was further observed in nonclinical groups (Goldstone et al. 2012;Laroi et al. 2004), where negative beliefs about thoughts (Morrison et al. 2002b) and positive belief about worry (Laroi and Van der Linden 2005) correlated with subjects' predisposition to auditory hallucinations. The strongest mechanistic pathway to hallucinations in nonclinical groups was postulated to be emotional trauma via life hassles (Goldstone et al. 2012), consistent with theories about early trauma affecting sensitisation to later stressors (van Winkel et al. 2008). Negative metacognition however mediated this association, showing that individuals with automatic negative thought processes were especially vulnerable to nonclinical hallucinations (Garcia-Montes et al. 2006;Goldstone et al. 2012).

Some disagreements still remain as to whether individuals with schizophrenia have more stress-prone lifestyles, consequently generating more life events than healthy controls. This can either be the effect of stigmatization, limited social support or lack of coping abilities (Lukoff et al. 1984), or interaction with genetic vulnerability (van Os et al. 1998), past illness or their personality (Bebbington et al. 1993). Also, specific personality traits such as neuroticism and extroversion were observed to heighten exposure and sensitivity to adverse life events (Breslau et al. 1995). Looking at the individuals with schizotypal personality disorder higher rates of crime and legal events are found compared to those with C cluster[11] personality disorders (Pagano et al. 2004). Equally, individuals with schizophrenia are seen as more likely to commit violent acts (Stueve and Link 1997).

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[11] *C Custer PD (anxious or fearful disorders) includes the following types of personality disorders: Avoidant PD, Dependent PD, and Obsessive-Compulsive PD. Schizotypal PD belongs under the Cluster A PD (along with Schizoid and Paranoid personality disorders), which are all characterised by odd or eccentric behaviour. The latest DSM-5 (American Psychiatric Association 2013) included personality disorders as other mental disorders and not on a separate axis as its previous additions.*

In contrast, one study found no difference in the number of events experienced by an ultra-high-risk group compared to healthy controls or first-episode psychosis subjects (Miller et al. 2001), with another study even reporting less life events among schizophrenic patients (Chung et al. 1986;Gureje and Adewunmi 1988;Horan et al. 2005). The main focus in the literature has been on events' appraisals and schizophrenia patients are much more likely to judge negative events as more distressing, reflecting their low self-esteem, diminished controllability (external locus of control) and weakened self-efficiency (Rooske and Birchwood 1998;Wiedl 1992). These subjective appraisals of the events then further conceptualise the stress sensitivity (Taylor and Aspinwall 1996). Also elevated trait 'anhedonia' (Blanchard et al. 2001a) and diminished 'positive affectivity' can influence the appraisal of positive events as less desirable (Horan et al. 2005). Interestingly, a study of psychotic patients one month prior to relapse showed that for individuals with limited cognitive and coping abilities no events were observed, but in those with adequate coping capacities a severe event might have influenced the relapse (Pallanti et al. 1997). A significant increase in life events in the three week period before the onset of psychosis was only evidenced for women, implying that females might have a clearer event-related onset, whereas males tend to have a more insidious onset (van Os et al. 1994).

2.3.3   The role of dissociation and PTSD

The existing literature has proposed two (not necessarily competing) underlying mechanisms that support the childhood abuse and positive symptoms association, one involving posttraumatic stress disorder (PTSD) or dissociative symptomatology and the other relating to negative beliefs about self and others that are formed after childhood abuse *(leading to schemas about the world being a hostile place and the*

individual being weak and vulnerable) (Freeman and Fowler 2009;Kilcommons and Morrison 2005;Morrison et al. 2003). Dissociation is defined as a separation of mental processes (usually normally integrated), consisting of core elements such as depersonalisation *(anomaly of self-awareness)*, derealisation *(experience of unreality of the outside world)* and psychogenic amnesia *(characterized by the presence of retrograde autobiographical memory loss)* (Giesbrecht and Merckelbach 2008). This definition is agreed by many authors but is still in need of refinement (Nijenhuis and van der Hart 2011). Dissociative symptoms are considered to form a continuum between brief, transitory forms commonly observed in everyday life to more severe forms of psychopathology, with overly frequent and intense symptoms, occurring in an inappropriate context (Fischer and Elnitsky 1990;Ross et al. 1991).

Considerable overlap has been evidenced between schizophrenia and dissociative disorders in many phenomenological features (Ellason and Ross 1995;Moskowitz et al. 2008;Ross 2004), even though *ICD-10* (World Health Organisation 1992) and *DSM-V* (American Psychiatric Association 2013) classify the two disorders with completely distinctive symptoms. High levels of dissociation have been found in patients with schizophrenia, especially associated with positive symptomatology like delusions and hallucinations (Honig et al. 1998;Spitzer et al. 1997). Also, Schneiderian symptoms usually associated with schizophrenia are common (or even higher) in dissociative disorder (Ellason and Ross 1997).

Even though dissociation states can be experienced without antecedent trauma (Mayer and Farmer 2003;Merckelbach and Muris 2001), the causal relationship between trauma and dissociation has been suggested (Gershuny and Thayer 1999;Irwin 1998) but not consistently observed (Merckelbach and Muris 2001). Nevertheless, dissociative experiences provide an imperative link between childhood trauma and psychosis (Dorahy et al. 2009;Moskowitz et al. 2008;Offen et al. 2003), with dissociative experiences frequently reported by psychotic patients

(Allen and Coyne 1995;Goren et al. 2012;Merckelbach et al. 2005;Perona-Garcelan et al. 2008;Spitzer et al. 1997) and individuals with high schizotypy levels in clinical (Gleaves and Eberenz 1995;Perona-Garcelan et al. 2010) and nonclinical populations (Bernstein and Putnam 1986;Irwin 1998;Merckelbach et al. 2000;Moskowitz et al. 2005;Pope and Kwapil 2000;Startup 1999). Beside the link between dissociative states and broader psychotic symptoms as well as psychosis-proneness (Pope and Kwapil 2000;Startup 1999), studies indicated some symptom-specific links to dissociation, with the most robust evidence for hallucinations (Kilcommons and Morrison 2005;Perona-Garcelan et al. 2010;Perona-Garcelan et al. 2012). Also, the depersonalisation factor alone was cited to be predictive of hallucinatory experiences (Perona-Garcelan et al. 2012) and dissociation predicted the persistence of such experiences in three year follow-up (Escher et al. 2002b). This evidence led researchers to believe that dissociation processes might mediate the relation between childhood trauma and hallucinatory experiences (Morrison et al. 2003;Moskowitz and Corstens 2007;Varese et al. 2012a), particularly sexual abuse among the types of childhood traumas (Nash et al. 1993;Varese et al. 2012a). Similar evidence comes from other cross-sectional and epidemiological studies looking at the association between sexual abuse and hallucinations (Read et al. 2003;Shevlin et al. 2007a). Individuals who reported childhood trauma displayed more intense hallucinations and delusions when dissociative symptoms were also present (Perona-Garcelan et al. 2012). Nevertheless, one study found that the dissociation process did not act as a mediator between childhood trauma and delusions (Perona-Garcelan et al. 2012). Despite sexual abuse showing the strongest correlation with dissociative symptomatology (Goff et al. 1991;Ross and Keyes 2004), other studies found that dissociation in patients with schizophrenia is associated with childhood physical abuse (Goff et al. 1991), childhood emotional abuse (Holowka et al. 2003) as well as physical neglect (Vogel et al. 2009).

Although factor-analysis of the concepts indicated the strongest association between depersonalisation and schizotypy, the constructs are not easily distinguishable (Watson 2001). Pope and Kwapil (Pope and Kwapil 2000) found a correlation between the *Dissociation Experiences Scale* (Bernstein and Putnam 1986) and measures of schizotypal traits, such as the *Magical Ideation Scale* (Eckblad and Chapman 1983) and *Perceptual Aberration Scale* (Chapman et al. 1978). Overlap was also observed between positive symptoms in schizophrenia and dissociation (Nurcombe et al. 1996;Ross et al. 1994;Ross 2005). However, comparing dissociative and psychotic auditory hallucinations, no studies so far have truly been able to gauge a quantitative or qualitative distinction between the symptoms (Moskowitz and Corstens 2007). When looking at depersonalisation and schizotypy from a phenomenological viewpoint, it can be observed that depersonalisation is linked to self-perception with intact cognition, whereas schizotypy is associated with disturbed cognition as indicated by odd beliefs, magical thinking and ideas of reference (Simeon et al. 2004). These neurocognitive deficits associated with schizotypy have been observed in previous research, including impairments in verbal and working memory, latent inhibition, hemisphere asymmetry etc. (Chen et al. 1997;Dorahy and Green 2008;Lenzenweger and Gold 2000;Matsui et al. 2004;Raine 2006).

The literature suggests few explanations of the dissociation and schizotypy link, the first being that the overlap of the concepts cannot be differentiated (Merckelbach et al. 2000;Watson 2001), the second suggests sharing traumatic etiology (Irwin 2001;Startup 1999) or common cognitive deficits (Giesbrecht et al. 2007), and the third sees both constructs as manifestations of the higher trait 'openness to experiences' (Merckelbach et al. 2000). Although 'openness to experiences' is characterised by fantasy proneness and out-of-body experiences common to both concepts, this theory is not likely to capture the fundamental link

between schizotypy and dissociation (Merckelbach et al. 2000). One study that sought to investigate the overlap between dissociation and schizotypy highlighted that disorganised schizotypy and dissociation are correlated but distinct concepts - dissociation was associated with childhood abuse, increased openness to experiences and increased emotional influence but disorganised schizotypy was not (Cicero and Kerns 2010a). Similarly, positive schizotypy was only associated with one dissociation subscale but not with the other two *(depersonalisation and detachment)*, again confirming separation between the concepts (Cicero and Kerns 2010a). Traumatic etiology underlying schizotypy and dissociation has been a focus in many studies and failed to support the idea that childhood trauma can fully account for the overlap between these concepts (Irwin 2001;Merckelbach and Giesbrecht 2006;Startup 1999). The evidence remained unchanged after taking into account trauma-related distress (posttraumatic intrusions) (Giesbrecht and Merckelbach 2008) that might lead to impaired reality testing (Morrison et al. 2003). In addition, combining childhood trauma with other hypothesised mediators of the dissociation-schizotypy link (e.g. cognitive deficits, fantasy proneness) only explained 58% of the dissociation and schizotypy association (Giesbrecht et al. 2007). Nonetheless, cognitive deficits, including memory and perception, as well as fantasy proneness can also be attributed to genetic factors (Bergeman et al. 1993;Boomsma 1998). Another possible explanation for the association between dissociation and schizotypy comes from studies looking into sleep patterns and dreams (Watson 2001), especially nightmares *(reported as a single best predictor of schizotypy (Claridge et al. 1997))*, characterised by the inability to limit the content of consciousness.

Nonetheless, childhood trauma (especially childhood abuse) has been reported to be the main etiological factor for dissociative disorders, dissociative symptoms and dissociative amnesia in many research studies (Putnam 1989;Ross

et al. 1990;Ross 2004), but not for depersonalisation disorder which is precipitated by general psychopathology instead (Simeon and Abugel 2006). Looking at the schizophrenia-spectrum disorders, the association was found for dissociative symptoms and positive symptoms, with those individuals with mainly negative symptoms scoring significantly less on dissociation scales (Spitzer et al. 1997). This ties in with studies implying that out of the positive schizophrenic features, delusions and hallucinations were especially strongly correlated with dissociation scale scores (Kilcommons and Morrison 2005;Spitzer et al. 1997). In stark contrast stands another study, where highly dissociative schizophrenic individuals displayed more negative symptoms comparing to individuals with low dissociative symptomatology (Ross and Keyes 2004).

The mediating role of PTSD between traumatic experiences and psychosis/ psychosis-like symptoms, including hallucinations and paranoia, has been widely supported (Gaudiano and Zimmerman 2010;Kilcommons and Morrison 2005;McGorry et al. 1991). But, after controlling for trauma severity, only depersonalisation significantly predicted hallucinations (Kilcommons and Morrison 2005). The prevalence of the PTSD in schizophrenia has been estimated between *17%* and *46%* (Gearon et al. 2003), among acutely psychotic people it is on average estimated at *50%* (McGorry et al. 1991;Shaw et al. 2002). Studies emphasized that PTSD can influence psychosis directly, through re-experience of trauma including overarousal and avoidance, or indirectly via re-traumatisation or common outcomes of PTSD (e.g. substance abuse) (Mueser et al. 1998). Moreover, individuals with childhood trauma are also more susceptible to daily stressors (Thakkar and McCanne 2000) and have limited coping resources (Cole and Putnam 1992). Examining this relationship between childhood trauma and psychosis, theorists distinguished the mainly *'biological pathway'* to negative symptoms, and largely *'trauma-induced'* pathway manifested in more positive symptomatology (Ellason and

Ross 1995;Ross et al. 1994;Ross and Joshi 1992). Traumatic experience and consequently PTSD can have a significant effect on the individuals' cognitive schemas, permanently changing beliefs about self and others that are replaced with self-blame and faulty (negative) perception of others (Dunmore et al. 1999;Morrison 2001). Also, social support after a traumatic event has been consistently reported in relation to PTSD symptoms (Brewin et al. 2000;Gold et al. 2000;Kotler et al. 2001;Schumm et al. 2006;Vranceanu et al. 2007), demonstrating that perceived social support among trauma survivors buffered the levels of PTSD and stands as an essential resilience factor in coping with the trauma (Norris and Kaniasty 1996;Wheaton 1985).

2.3.4   The effect of cannabis use

Cannabis is the most widely used illegal substance in Europe (European Monitoring Centre for Drugs and Drug Addiction 2001) with the prevalence of cannabis use in a UK community sample estimated at *72%* (used cannabis at least once) (Barkus et al. 2006). Multiple epidemiological studies report higher odds of cannabis use/misuse in people with psychosis (Green et al. 2005).

Cannabis use was shown to be a risk for development of psychotic symptoms as well as prodromal psychotic states (Arseneault et al. 2002;Fergusson et al. 2003;Large et al. 2011;Linszen et al. 1994;Moore et al. 2007;Semple et al. 2005;Smit et al. 2004;Stefanis et al. 2004a;van Os et al. 2002) in a dose-response fashion (Moore et al. 2007;Stefanis et al. 2004a). The effects of cannabis are observed through increased schizotypy levels in the community population (Barkus and Lewis 2008;Cohen et al. 2011;Dumas et al. 2002;Esterberg et al. 2009;Skosnik et al. 2001;Williams et al. 1996), increased severity of psychotic symptoms in schizophrenia patients (Baigent et al. 1995;Treffert 1978), increased likelihood of

relapse (Linszen et al. 1994) and overall poorer prognosis (Stefanis et al. 2004a;van Os et al. 2002). However, it still remains uncertain whether schizotypal traits predispose individuals to use cannabis or cannabis itself affects schizotypy levels (Degenhardt et al. 2003;van Os et al. 2002). Some authors suggest that those with acquired genetic vulnerability for psychosis might be more prone to psychosis-induced effect of cannabis (van Os et al. 2002;Verdoux et al. 2003a).

Synergistic interaction between childhood trauma and cannabis use was also proposed, with *'more-than-additive'* effects (Cougnard et al. 2007;Harley et al. 2010;Houston et al. 2008). For example, individuals who had been sexually abused as well as started using cannabis before the age of 16 were 12-times more likely to be diagnosed with psychosis (Houston et al. 2008). Moreover, sexual trauma alone without cannabis use significantly increased the risk of psychosis *(OR=2.45)* but the odds ratio almost doubled with cannabis use before *(OR=4.39)* or after *(OR=4.25)* sexual trauma (Shevlin et al. 2009). Longitudinal studies (Cougnard et al. 2007) and community samples (Cougnard et al. 2007;Harley et al. 2010;Houston et al. 2008;Konings et al. 2012) showed similar effects, childhood maltreatment (even non-severe) moderated the association between cannabis and psychotic experiences in a dose dependent fashion (Konings et al. 2012). On the other hand, a nationally representative sample that looked into effects of sexual abuse and cannabis on psychosis found no independent effect for cannabis use (Houston et al. 2008), opposite to some previous reports (Fergusson et al. 2003;van Os et al. 2002). Cannabis use alone was also not the only or sufficient cause for developing psychosis (Arseneault et al. 2002;Degenhardt et al. 2003).

Nonetheless, it is believed that cannabis use influences the positive and negative dimensions of psychosis through subtle alterations in mental states (Stefanis et al. 2004a). Literature stipulates that cannabis powers the repeated stimulation of the endogenous mesolimbic dopaminergic system (Tsapakis et al.

2003). Consequently, *hyperdropaminergic* state can cause the stimulus independent release of dopamine, leading to the aberrant assignment of salience to external events and internal representations. Thus, hallucinations reflect a direct experience of aberrant salience of internal representations, while delusions arrive from cognitive explanations for these experiences (Kapur 2003). Similarly, the interaction between childhood trauma and cannabis use may increase the risk of psychosis via sensitisation of dopamine agents (Kapur 2003), as not only cannabis but also stressful experiences have been linked to increased dopamine release (Soliman et al. 2008;Voruganti et al. 2001). One study suggested that hallucinations were less sensitive than grandiosity to the sensitizing effect of cannabis use (Stefanis et al. 2004a).

The age of first cannabis use significantly predicted psychosis-like symptomatology (Schubart et al. 2011), interestingly more so than lifetime frequency of use (Stefanis et al. 2004a). This also fits with the observation that younger populations (in their developmental stage) are more vulnerable to dopamine sensitisation. This is a result of alterations in cortical development in adolescence and its failure to buffer stress-related releases of dopamine neurons (Laruelle 2000). There was also a trend of early cannabis users *(defined as either below the age of 12 (Schubart et al. 2010), 14 (Konings et al. 2008) or 15 (Stefanis et al. 2004a))* predicting the trajectory for schizotypal traits from the ages of 13 to 35, which remained significant even when pre-existing or co-occurring schizotypal symptoms were considered (Anglin et al. 2012). On the contrary, another study indicated that if psychotic-like symptoms were observed in individuals before the age of 11, cannabis use was no longer associated with psychotic symptoms (Arseneault et al. 2002).

Moreover, it was proposed that not the cannabis use per se relates to schizotypy but those reporting high schizotypy levels are more likely to report

unpleasant after-effects associated with cannabis (Stirling et al. 2008). In another study, there was no association observed between cannabis use and higher schizotypy compared to non-users (Barkus et al. 2006;Stirling et al. 2008), however the frequency (Stirling et al. 2008) and quantity of use (Schubart et al. 2011) among cannabis users proved to be associated not only with higher schizotypy but also strengthen the association with psychotic symptoms and psychotic disorder (Arseneault et al. 2004;Di Forti et al. 2009;Moore et al. 2007). Unexpectedly, another study showed that low levels of recent cannabis use were strongly associated with psychotic-like symptoms than more frequent usage (Hides et al. 2009). This relationship can also be mediated by genetic difference in sensitivity to the psychotomimetic effect of cannabis (Caspi et al. 2005;Henquet et al. 2005a;van Os et al. 2002;Verdoux et al. 2003b). For example, individuals with the *Val/Val* variant of the *COMT* gene, which impacts on dopamine regulation, have been shown to have greater liability to cannabis-induced psychosis (Caspi et al. 2005). Even though it was also suggested that genetic vulnerability can be expressed as a tendency to use more cannabis (Ferdinand et al. 2005), this was not confirmed. Furthermore, exposure to cannabis was shown to be more influenced by environmental than genetic factors (Kendler et al. 2008). Likewise, different sensitivity to *Delta-9-tetrahydrocannabinol* (primary psychoactive ingredient) has not only been linked to genetics (Caspi et al. 2005;Henquet et al. 2006) e.g. *COMT Val158Met* polymorphism (Henquet et al. 2006) but defined by some environmental factors (Harley et al. 2010;Houston et al. 2008).

Also, cannabis contains different cannabinoids with almost the opposite effects. *Delta-9-tetrahydrocannabinol (Delta-9-THC)* was associated with higher levels of unusual experiences (hallucinations and delusions) compared to those using *Delta-9-THC* and *CBD* (cannabidiol) and those using no *CBD,* as *CBD* was observed to have antipsychotic effects (Morgan and Curran 2008). In the same

study those who used cannabis containing *THC* and *CBD* also reported less anhedonia. On the other hand, *Delta-9-THC* substance was not only linked to psychotic symptoms but also anxiety and cognitive deficits in schizophrenia patients and a community population (D'Souza et al. 2005). That said the independent effects of anxiety and depression states did not account for the increased schizotypal symptomatology among early cannabis users (Anglin et al. 2012;Nunn et al. 2001). *Delta-9-THC* was also linked to increased dopaminergic neurotransmission (Murray et al. 2007), which is also associated with psychotic symptoms (Kapur 2003).

Cannabis use was found to be associated with specific schizotypal traits. There was an association found between cannabis use and the positive schizotypy dimension (especially unusual experiences, delusional ideation) (Nunn et al. 2001) but cannabis use attenuated negative symptomatology (e.g. introverted anhedonia) (Nunn et al. 2001), especially alogia in schizophrenia patients (Peralta and Cuesta 1992). Lower introverted anhedonia among cannabis users can be explained in two ways: either substance use itself diminishes the impact of negative symptomatology or individuals with lower levels of anhedonia and social isolation tend to use more cannabis (Nunn et al. 2001;Peralta and Cuesta 1992). When using the *CAPE* measure *(Community Assessment of Psychic Experiences (Stefanis et al. 2002))* however, the correlation was observed between cannabis use and all three dimensions: positive, negative and depressive (Skinner et al. 2011). Another study looking at the relationship between cannabis use and schizotypy in a community population confirmed that cannabis correlated with positive schizotypal traits (ideas of reference, odd beliefs, unusual perceptual experiences, magical thinking and odd or eccentric behaviour) with no gender effect (Dumas et al. 2002). The relationship observed however was influenced by anxiety and depression traits, in line with previous studies that indicated not only a link between schizotypy and anxiety and

depression but also an association between cannabis use and mood states (Lenzenweger and Loranger 1989). After adjusting for the affective states, the only significant correlation was between cannabis and positive schizotypy found in a healthy student population (Dumas et al. 2002). Different hypotheses have been put forward to explain such a relationship: cannabis use has direct pharmacological effects on these traits (dopaminergic hyperactivity) (Ameri 1999;Kapur 2003), or schizotypal traits lead to cannabis use in order to alleviate negative symptoms ('self-medication') (Peralta and Cuesta 1992), or the association coexists without any causality between each other *(underlying vulnerability predisposes individuals to higher psychosis symptoms and vulnerability for cannabis use)* (Schneider et al. 1998). In stark contrast to that, other literature suggests that cannabis users who do develop schizophrenia showed better IQ and better premorbid adjustment, implying that early cannabis use (and not pre-existing vulnerabilities) might cause the early psychosis in those who would otherwise have a good prognosis (Sevy et al. 2001). More recently, however, a study did find lower depressive symptoms in cannabis users (Tosato et al. 2013), confirming the self-medication hypothesis (Henquet et al. 2005a;Kuepper et al. 2011;McGrath et al. 2010).

In summary: Reasoning behind the thesis

(i)   *What we currently know about the relationship between trauma and schizotypy and the underlying pathways*

This systematic literature review gave substantial support to the association between childhood trauma and schizotypy, especially for the positive dimension in a dose-response manner. However, mainly due to methodological limitations, including the inconsistent use of measures and a range of different sampling procedures, the comparison between the studies could be problematic. Even though the most robust effect was observed for emotional abuse, the findings are not

consistent. Also, there are some discrepancies observed with regard to differential effects of trauma types on the schizotypy load, with some reporting similar associations for all types of childhood trauma and others observing no association with specific adversities. However looking overall, all the studies did report an association between at least one type of trauma and schizotypal traits.

(ii)   The deficits in the knowledge

Although there is emerging evidence identifying symptom-specific and exposure-specific underlying mechanisms that support a childhood trauma-schizotypy association, more research is required to fully understand this complex relationship. Not only is it essential to build on methodological limitations of previous studies and go beyond crude self-report measurements by using the highest quality level of assessments, but there is also a need to expand the focus from a single contributor (e.g. psychological, biological) and explore additive and/or sometimes interactive contributors to schizotypal symptomatology. This would help to uncover some valuable clues as to the aetiology of psychotic-like symptoms and accordingly psychotic disorders.

The concerns about the reliability of retrospective measures of childhood abuse cannot be fully eliminated, unless the longitudinal or prospective study design is selected, again not without their own disadvantages. However, retrospective reports have been previously found to be reliable (Fisher et al. 2011;Hardt and Rutter 2004), yet utilizing a more in-depth standardised interview of childhood abuse would further maximise their validity and reliability. The Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1997), often utilised in studies exploring childhood trauma and schizotypy/psychosis-like symptoms, does also not consider factors like subject's age at the occurrence of the trauma, subject's relationship to the perpetrator and severity of abuse. Moreover, it does not consider some other

forms of traumatic experiences or victimisation beside childhood abuse and neglect (like exposure to domestic violence, loss of parent, separation from a parent, and bullying).

Also, there is a long history and complexity of schizotypy measures trying to capture the core of the concept, ranging from one-trait measures (e.g. social anhedonia) to more multifactorial measures. Unfortunately the evidence for the trauma and schizotypy association is mainly derived from self-reported measures (Berenbaum et al. 2003;Steel et al. 2009) and not interview style techniques.

(iii)   The importance of the topic

Focusing on schizotypy would mean the detection of fundamental features of liability to psychosis prior to the illness itself, without the possible interference of factors usually associated with research using clinical samples (e.g. the effects of the illness itself, medication, hospitalisation etc.). Most importantly, it could have substantial implications for clinical assessment and treatment formulation. Childhood trauma has been reported to be a risk factor for an array of psychopathology later in life (Bechdolf et al. 2010). Therefore, better understanding of the risk factors involved in the development of schizotypal traits/psychotic-like symptoms might help with early identification. Many studies stressed the high predictive value of schizotypal symptoms (Kwapil et al. 2013) and early psychotic-like symptoms (Poulton et al. 2000). The most reliable predictor of psychosis among individuals considered at-risk for developing psychosis was the level of schizotypal traits (Mason et al. 2004). Early interventions were documented to have promising outcomes in terms of individual's overall symptoms, social functioning and quality of life (Marshall et al. 2005) along with economic benefits (Falloon et al. 1998). Exploration of the pathways leading to subclinical symptoms would also assist in applying the appropriate interventions to prevent symptoms' progression e.g. target cognitions

and beliefs about self and others as contributors to the development of clinical psychosis (Kuipers et al. 2006) or address the adult re-victimisation (e.g. Desai et al. 2002).

(iv)   Moving forward (building on methodological limitations of previous studies)

- Moving beyond the crude measurement of childhood trauma, the higher quality measures such as Childhood Experience of Care and Abuse – CECA (Bifulco et al. 1994) allows the consideration of contextual factors, like trauma severity, frequency as well as timing of abuse in relation to schizotypal symptom load.

- The use of a direct semi-structured interview to measure traumatic experiences could be one of the strengths of the future research, covering a wider array of traumatic experiences (including parental separation, death of a parent etc.) in relation to schizotypal traits and allowing clarification and more detailed information to be obtained, that can significantly contribute to the value of research findings.

- There is a need for a heterogeneous sample with respect to age, gender and ethnicity, which would allow the exploration of the differences between these groups with regard to childhood trauma and schizotypy associations. Also possibly distinct gender-specific pathways underlying these associations could be explored.

- The Structured Interview for Schizotypy-revised (SIS-R) currently stands as the most comprehensive measure of the broad range of schizotypal symptoms and signs (Kendler et al. 1989). However, it has only been used in one study so far that measured schizotypy traits in relation to childhood

trauma *(CTQ was used to measure childhood trauma)* (Myin-Germeys et al. 2011).

- As it is difficult to think of the development of schizotypal symptoms in terms of a single contributor/factor (e.g. childhood trauma), future research instead needs to focus on genetic and environmental interactions that contribute to the development of schizotypal traits or psychotic disorder and include some possible mediators of this relationship (depression symptoms, negative belief about self and others, cannabis use etc.), as the pathways to schizotypal symptoms might be more complex than previously suggested.

Therefore, the main aims of this thesis are to explore the relationship between different types of childhood trauma/victimisation (sexual, physical, psychological abuse, household discord and peer bullying) and schizotypy load, while considering contextual information about these traumatic experiences and including some of the moderators and mediators underlying these associations.

CHAPTER 3
Methodology

Chapter 3 Methodology

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Main aims of the chapter

- To determine the main objectives and hypotheses of the thesis *(section 3.1)*;

- To present the study's experimental design *(section 3.2)*;

- To describe the data collection procedure and assessment tools used along with the reasoning behind their inclusion *(section 3.3 & 3.4)*.

3.1   *Aims and Hypotheses*

3.1.1 Aims of the thesis

The main aim of this thesis is to explore the relationship between childhood trauma and schizotypy. The subsidiary aims are to look at the underlying mechanisms that support this association. A range of aspects of childhood traumatic experiences are included (e.g. sexual, physical, psychological abuse, household discord and peer bullying) and the age of occurrence, frequency and severity of the traumas will be taken into account. In order to disentangle the complexity of the underlying mechanisms that underlies the childhood trauma - schizotypy relationship (which can also help to understand some important clues to aetiology of psychotic symptoms and therefore psychotic disorders) the research also considered the following confounders and/or moderators/mediators of this relationship:

- *Social:* adult adversity (life events and/or ongoing difficulties, including the characteristics of independence and intrusiveness of events/difficulties), also cannabis use and socio-demographics (ethnicity, employment status);

- *Psychological:* cognitive and affective processes (core negative beliefs about self and others), current depression symptomatology and dissociation; and

- *Genetic:* mental health history (especially psychosis) of first degree family members.

3.1.2 Hypotheses

The following **main hypothesis** will be tested:

1. **Those reporting more childhood trauma will score higher on a schizotypy scale.**

Secondary hypotheses:

2. Schizotypy will be higher in those exposed to both childhood and adulthood traumatic experiences than in those with childhood trauma only.

3. Childhood trauma will lead to development of negative beliefs about self/others and depression which will then increase the schizotypy levels.

4. Individuals with higher familial risk for psychosis and exposure to childhood trauma will display higher schizotypy scores than those without familial risk.

5. Cannabis use will partially account for the association between childhood trauma and schizotypy; cannabis will either mediate the childhood trauma – schizotypy association or interact with childhood trauma to increase the schizotypy levels.

3.2   *Study design*

3.2.1   Experimental design

The participants were recruited as a part of a cross-sectional epidemiological case-sibling-control study conducted in London, United Kingdom. The study overlaps with and forms part of the EU-GEI programme of research (European Network of National Schizophrenia Networks Studying Gene-Environmental Interactions) that is examining gene and environmental interactions in schizophrenia by focusing on first-episode psychosis patients (van Os et al. 2008). EU-GEI was designed *'to identify the interactive genetic, clinical and environmental factors involved in the development, severity and outcome of schizophrenia'* (EU-GEI 2009 p.5). It builds on findings from other epidemiological gene-environmental interaction studies (G x E) (Carter et al. 1999;Spauwen et al. 2006;Tienari et al. 2004;Wahlberg 1997), especially twin and adoption studies (Gottesman and Shields 1976;van Os and Sham 2003), which support evidence of the involvement of environmental factors and genes in the aetiology of schizophrenia. The EU-GEI programme aims to go beyond some of the limitations of previous studies (e.g. convenience cohorts and relying on crude measures), by recruiting clinical samples and representative community control groups and using the highest quality level of assessments (measuring environmental, clinical, genetic and behavioural determinants). Ethical approval for the study was obtained from the South London and Maudsley NHS Foundation Trust ethics committee *(Ethics Reference: 05/Q0706/158).*

Using the case-control design, the aim of the study is to recruit a sample of 300 first-episode psychosis cases and 300 community controls from the same geographical areas as cases (Lambeth and Southwark boroughs in London). Cases are an epidemiologically characterised cohort of all new cases of psychosis, aged

18-35 years, within the south-east London area (covered by SLaM[12] services) with a psychotic disorder as defined by The International Statistical Classification of Diseases (ICD10) F20-29 and F30-33 codes (World Health Organisation 1992), recruited over approximately a three year period (February 2010 to May 2013). Controls are a community-based random sample of healthy volunteers, aged 18-64 and resident in the same geographical area as cases, recruited over approximately a two year period (from February 2011 to May 2013). The scope of this thesis only covers a subsample of controls (N=212, *see Sample size calculation 3.2.2*) recruited under the framework of EU-GEI programme, therefore 'participants' in all future reference imply healthy volunteers without any past or current psychotic disorders.

The population of Lambeth *(population approx. 303,000; Census 2011)* and Southwark *(population approx. 288,000; Census 2011)* boroughs in London is diverse in terms of ethnicity and wealth (Office for National Statistics 2011b;Office for National Statistics 2011c), so the study is able to incorporate the widest range of community controls as possible. The two boroughs have higher deprivation than the English average (e.g. child mortality rate, living in poverty, youth criminality, exclusions from education, family homelessness) (Department of Mental Health 2011a;Department of Mental Health 2011b). However, the areas have a similar proportion of economically active and inactive residents aged 16 and over when compared to the English average (e.g. in Lambeth 81.0% economically active vs 7.6% unemployed) (Office for National Statistics 2011b;Office for National Statistics 2011c).

In the Lambeth area, over a third of residents (37.3%) are from ethnic minority groups, similar to inner London (37.8%). The largest non-white group comprises Black African (11.8%), followed by Black Caribbean (10.1%) individuals.

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[12] *SLaM: South London and Maudsley NHS Foundation Trust. Their mental health services include the Maudsley Hospital, Lambeth Hospital and Bethlem Royal Hospital in London as well as a large number of community-based teams.*

Compared to the inner London average, there are many fewer residents from South Asian backgrounds (Lambeth: 3.6%, inner London average: 10.6%) (Lambeth Council 2012). Similarly, Southwark has 16% of the population belonging to the Black African group, 6% of individuals are Black Caribbean, while South Asians accounted for 1% of the Borough's population (as such ranked as 28th among 33 boroughs of London for the proportion of Asian residents) (Southwark Analytical Hub 2006). The ethnic distribution for the two boroughs and comparison to figures of England as a whole are presented in *Table 5.*

Table 5: Ethnic distribution for England and London Boroughs of Lambeth and Southwark (Census 2011, (Office for National Statistics 2011a)).

Ethnicity	England	Lambeth borough, London	Southwark borough, London
White British	**79.8%**	39.0%	39.7%
White Other	**5.2%**	18.1%	14.6%
Black Caribbean	**1.1%**	9.5%	6.2%
Black African	**1.8%**	11.6%	16.4%
Asian	**7.7%**	6.8%	9.5%
Other	**4.4%**	15.0%	13.6%
Total	**100%**	100%	100%

3.2.2 Sample size calculation

Using the power analysis program *G*Power* (Erdfelder et al. 1996;Faul et al. 2009), the sample size of approximately *212* participants was suggested as providing adequate power for this thesis. The calculations were based on the use of logistic and linear regression analyses consistent with thesis objectives. Basing on an odds ratio of *2.0* (supported by the literature review) and statistical power of approximately *0.90* (aiming between *0.85-0.90*), the indicated sample size was *184 (see Figure 6 to Figure 8).* To allow for any missing data, an additional 15% was

added to this figure, bringing the total to *212*. The calculations were repeated for

linear regression analyses and produced similar results *(see Figure 8).*
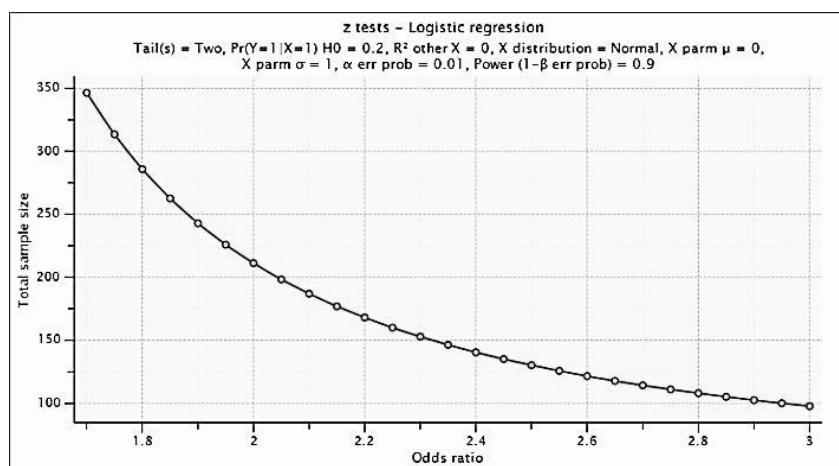
Figure 6: The graph shows the total sample size needed for increase/decrease in odds ratio (at constant power=90%) – for logistic regression analysis.
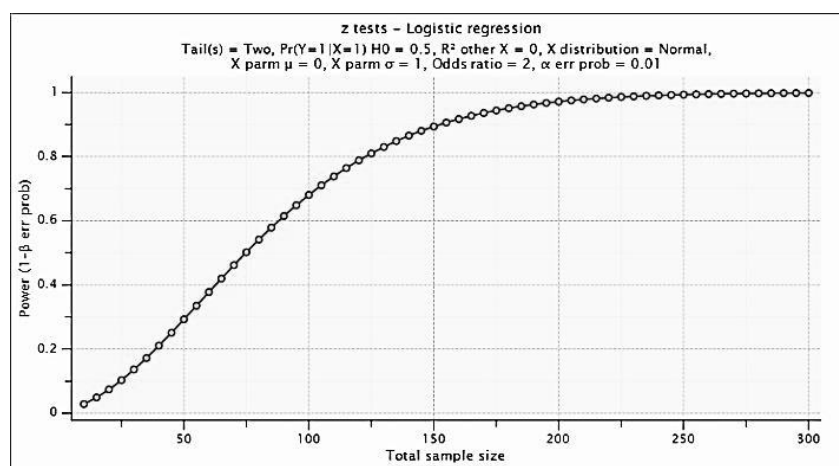
Figure 7: The graph shows the total sample size needed for different statistical power (if OR=2) – for logistic regression analysis
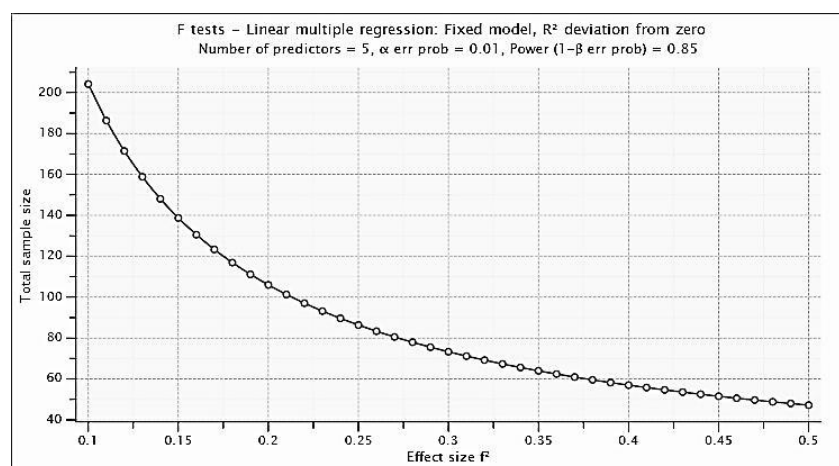
Figure 8: The graph shows the total sample size needed for different effect sizes – for linear regression analysis

3.3   *Sample/Data collection*

Quota sampling method was used to recruit participants. Even though this is a non-random sampling technique, it ensures the sample collected closely resembles the characteristics of the catchment area population. Quota sampling's main advantage lays in oversampling of underrepresented groups, thus enabling detection of group differences or just accounting for socio-demographic variations (Bornstein et al. 2013). However, it does rely on the nonprobability sample within each investigated group (e.g. a non-random sample for each ethnic group), lacking in generalizability estimates of the target population (or of subgroups differences) (Bornstein et al. 2013). The quota sampling technique does have some major positive attributes – it is faster and easier to organise as well as cheaper compared to other sampling methods, however some argue that it can lead to sampling errors (Monette et al. 2013).

Using the quota sampling, the catchment area population was segmented (using Census 2001 data) (Office for National Statistics 2001) to define the proportion of the population in each category (based on age, gender and ethnicity) that was representative of the local population. This was used to set quotas for the number of controls to be recruited from each stratum. The quotas were then modified, to oversample younger controls fitting into the 'Black' ethnic category to better mirror the number of individuals from this category that were likely to present as psychosis cases.

In order to ensure a representative sample was obtained, participants were identified through three main pathways *(see Figure 9 for a diagrammatical presentation of the full recruitment process).* The percentage of participants obtained from each source is presented in the *Table 6.*

(i)   PAF (Postcode Address File) – gives a good representation of private households in Great Britain (Jenkins and Meltzer 1995). Using a publicly available list of all households in the catchment area (obtained from Lambeth

and Southwark Royal mail), a random sample of 1000 addresses was selected. These were split into smaller groups to contact, firstly approached by a letter followed by a household visit at three different times (morning, afternoon, evening). By doing the 'three visits' approach, the likelihood of resident being at home was maximised along with reducing sampling bias (e.g. having more unemployed individuals who are likely to be at home during the day).

(ii) GP services – were contacted through *PCRN* (Primary Care Research Network), which acted as a 'middle man' putting researchers in contact with practices that might be interested in collaboration. The aim was to get an equal split between GP's in Lambeth and Southwark. Once in touch with a practice they randomly selected 400 patients without a history of psychosis (based on diagnostic codes for various illnesses (exclusion criteria) in the search that were provided by the researchers). The list of potential participants was then checked over by a doctor to make sure that those who would not be suitable to contact (e.g. recently had death in the family or terminal illness etc.) were excluded. Each potential participant was then sent a letter which contained the team's contact details, a reply slip (with an option to withdraw) and a free-post envelope. A second letter (with the same content) was re-sent after two weeks to all those who had not yet responded.

(iii) SELCoH (South East London Community Health Service) – the SELCoH study is a cross-sectional population survey of mental and physical health, which was conducted between 2008 and 2010 within the boroughs of Lambeth and Southwark (NIHR Biomedical Research Centre 2008). The team was provided with the names and contact details of SELCoH participants who fitted the inclusion criteria and also expressed an interest to SELCoH about taking part in the study. They were contacted through telephone or email (allowing

three attempts to contact them) before they were excluded and passed back to SELCoH.

(iv)   Other – In addition to the three main recruitment pathways, few participants were recruited via flyers, posters or adverts on online community websites (e.g. gumtree.com), inviting participants from the area to take part in the research study.

Table 6: Characteristics of the recruited sample (gender and ethnicity) according to the three different sources of recruitment (N=212)

	Male N=94				Female N=118				Total N=212 (100%)
	White	*Black Caribbean*	*Black African*	*Other*	*White*	*Black Caribbean*	*Black African*	*Other*	
PAF	7	0	1	0	5	0	1	1	**15 (7.1%)**
GP	30	4	7	5	50	4	6	5	**111(52.4%)**
SELCoH	22	9	5	4	17	9	13	4	**83 (39.1%)**
Other (via flyers, posters)	2	0	0	0	0	1	0	0	**3 (1.4%)**

PAF, Postcode Address File. GP, General Practitioner. SELCoH, South East London Community Health Service

Hubbard and colleagues (Hubbard et al. 2012) looked into possibilities of sampling bias generated by these three recruitment methods (PAF, GP, SELCoH), using data collected from a subsample of 119 participants. The preliminary findings showed that different techniques generated samples with different characteristics - there was a significant difference in gender and a trend towards differences in ethnicity and previous psychological problems - indicating that sampling bias is likely regardless of the method used.
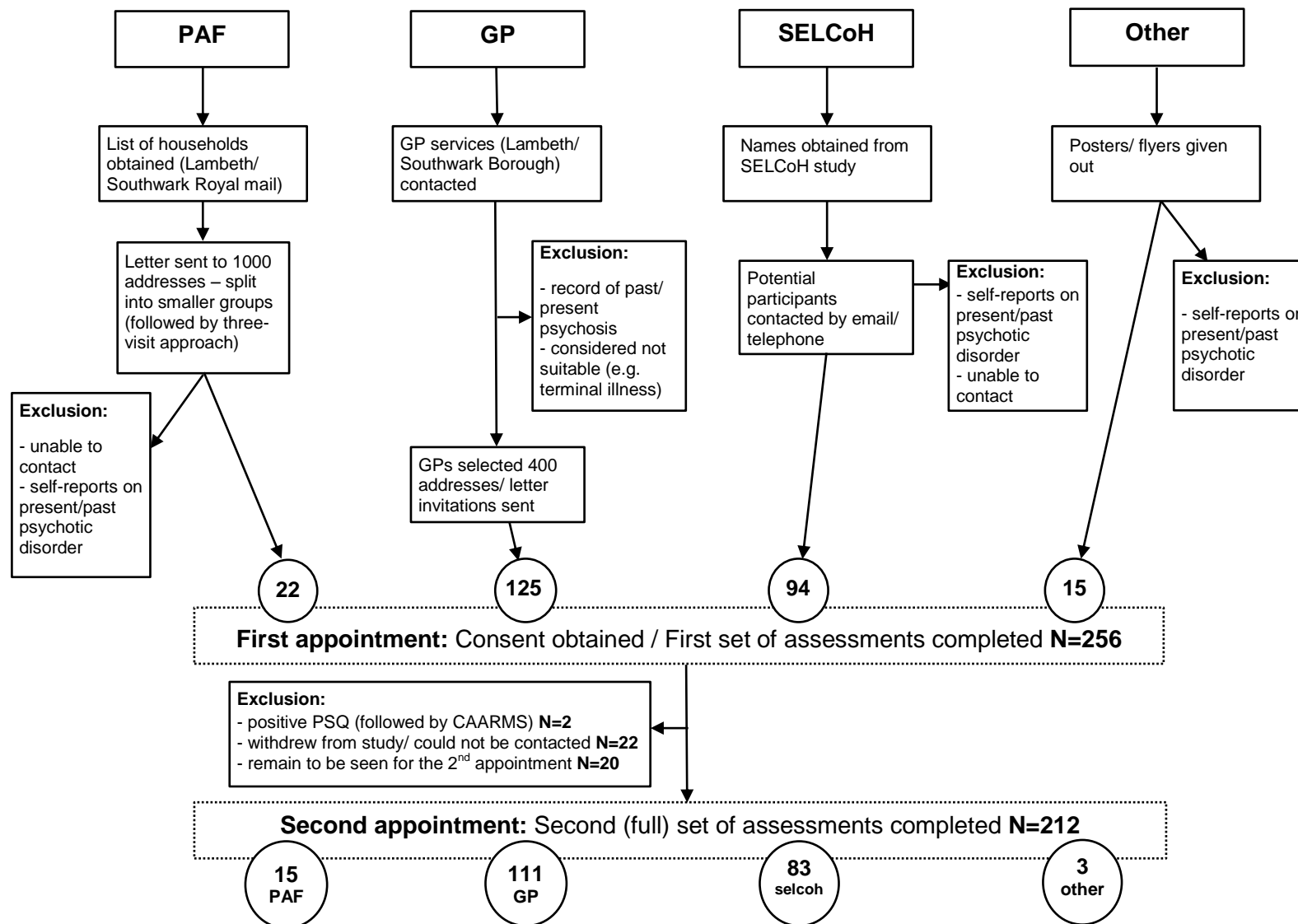
Figure 9: Flowchart presentation of the Recruitment Process

The following criteria were used when identifying suitable participants:

Inclusion criteria:

(i)   aged 18-64;

(ii)   resident within a clearly defined catchment area (Lambeth and Southwark boroughs in London);

(iii)   no evidence of current or past psychosis assessed by the Psychosis Screening Questionnaire (Bebbington and Nayani 1995).

Exclusion criteria:

(i)   under 18 or over 64;

(ii)   not resident within the clearly defined catchment areas;

(iii)   current or past psychotic disorder (screened positive on the Psychosis Screening Questionnaire, Bebbington and Nayani 1995) and were considered to have a psychotic disorder);

(iv)   major language difficulties.

Because of the length of the assessments used in the study, participants were seen on two separate occasions (taking on average 2 hours each). Interviews were conducted by trained researchers (with a background in psychology or psychiatry). At the first appointment an individual was explained the aims of the study, what it involves, potential risks, the voluntary nature of study (including the right to withdraw at any time) as well as confidentiality issues (as per Information Sheet presented, see *Appendix III*) after which their consent was obtained (signed Consent form, see *Appendix III*). Potential participants were screened for any history of psychosis using

Psychosis Screening Questionnaire (PSQ, see *Appendix IV*) (Bebbington and Nayani 1995), which assesses psychotic-like experiences over the past 12 months. The instrument includes six domains covering hypomania, thought insertion, paranoia, strange experiences, hallucinations and a question about any history of treatment for any psychiatric and psychological problems. Each domain has a key screening question, which is followed up by additional questions if the screening question is answered positively. The respondent must answer 'yes' on all questions within the domain to obtain the positive score on that particular symptom category. The PSQ measures positive symptoms that also appear in a standardised in-depth psychiatric interview (Johns et al. 2002) and this measure has been widely used in many community studies (Brugha et al. 2004;Johns et al. 2004;King et al. 2005;Wiles et al. 2006).

The team of researchers, including the lead researcher discussed the instances where psychotic experiences were detected. When more information was required, the decision on eligibility was made using the CAARMS *(The Comprehensive Assessment of At-Risk Mental States)* (Yung et al. 2005) *(only applicable for participants under 35; other instruments would have been used for those over 35, however no positively scoring subject was aged 35 or over)*. The CAARMS instrument is a well-established semi-structured interview allowing researches to differentiate between three different ultra-high-risk for psychosis groups (Yung et al. 2005):

(i)   *Vulnerability group*: Individuals with a family history (first-degree relatives) of a psychotic disorder or schizotypal personality disorder, along with a significant deterioration in mental state and/or functioning;

(ii)   *Attenuated Psychosis group*: Individuals with attenuated psychotic symptoms (reaching sub-threshold psychotic syndrome with regard to severity and/or intensity);

(iii)   *BLIPS group*: Individuals with Brief Limited Intermittent Psychotic Symptoms (BLIPS) - recent history of frank psychotic symptoms, resolving spontaneously within one week (Yung et al. 2005).

If an individual was deemed to meet criteria for any of these groups then they were excluded from the study. This occurred in the case of two participants.

All individuals who completed the first appointment and were considered eligible for the study (no current past/psychosis detected) were invited for the second appointment, usually (but not necessarily) completed by the same researcher. The consent form signed at the first appointment was also applicable to the second appointment. The appointments took place at the Institute of Psychiatry (King's College London) or at participants' homes, depending on their preferences.

3.4   *Main assessment tools*

The EU-GEI study covered an extensive battery of assessments conducted with participants, including diagnostic measurements, psychological questionnaires, neuropsychological testing and biological measurements (beyond the scope of this thesis). This section covers only the assessment tools (including specific items of the measurements where applicable) utilised for the purposes of this thesis (based on the hypotheses). Participants were seen on two separate occasions. The first appointment covered the assessments such as Psychosis Screening Questionnaire, Community Assessment of Psychic Experiences, Sociodemographic schedule, Childhood Experiences of Care and Abuse (and Bullying Questionnaire), Life Events and Difficulties Schedule, Hamilton Depression Scale and The Brief Score Schema Scale. On the second appointment, the assessments covered were Family Interview for Genetic Studies, Cannabis Experiences Questionnaire, Structured Interview for Schizotypy–Revised (SIS-R) and Wechsler Adult Intelligence Scale. These assessment tools are described in detail below.

MRS Sociodemographic Schedule (amended) (Mallett 1997)

This schedule is provided in *Appendix V*. Part 1 of the schedule comprises socio-demographic data such as gender, age, ethnicity, country of birth (and age of migration where applicable) and socio-economic status. Participants are asked to describe their ethnicity on 18 categories adapted from the UK Office of National Statistics (ONS) census. The categories were the following: (i) White British; (ii) White Irish; (iii) White gypsy, traveller; (iv) Other White; (v) Mixed: White and Black Caribbean; (vi) Mixed: White and Black African; (vii) Mixed: White and Asian; (viii) Other Mixed; (ix) Indian; (x) Pakistani; (xi) Bangladeshi; (xii) Chinese; (xiii) Other Asian; (xiv) Black Caribbean; (xv) Black African; (xvi) Black Other; (xvii) Arab; and (xviii) Other. The categories were based on self-identification, as according to the

Office for National Statistics the main principal of ethnic categorisation is a sense of belonging or something that is subjectively meaningful for an individual.

For the ease of the analysis, the categories were combined into 6 main ethnic groups: White British, White Other *(including White Irish and White Other)*, Black Caribbean, Black African, Asian *(including Indian, Pakistani, Bangladeshi, Chinese and Other Asian)* and Other *(including all the rest of the categories)*. The groups were combined to best evaluate the potential differences in schizotypal traits/psychotic-like experiences among ethnic groups according to reports from other studies *(see Section 1.1.4 Socio-demographic characteristics and schizotypy)*. For the same reason, if individuals defined themselves as 'Black British' or had any mixed background (including mixed Black African and Caribbean[13]) they would be categorised under 'Other'.

Part 2 of the schedule comprises questions about the employment status for three different time periods: at the time of the interview, 1 year ago and 5 years ago. Employment categories included: (i) unemployed; (ii) economically inactive (i.e. house person, physical illness/disability, carer, retired); (iii) student; (iv) part-time employed; (v) full-time employed; (vi) self-employed; and (vii) not applicable (rated if participant 17 or below at the time). Participants were also asked about their religious affiliation: (i) none; (ii) Christian; (iii) Jewish; (iv) Muslim; and (v) Other (specify), and the frequency of attending religious services: (i) never; (ii) once or twice a year; (iii) monthly or (iv) weekly.

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[13] *One participant only, not included in the final sample due to incompletion of both appointments*

Wechsler Adult Intelligence Scale (3rd ed.) (WAIS-III) (abbreviated version) (Wechsler 1997)

WAIS-III is one of the most commonly used intelligence measures (Rabin et al. 2005). As the main purpose of its inclusion was to obtain the intelligence quotient (IQ) of the subjects, shortened forms of the WAIS-III were used, consistent with previous literature (e.g. Reid-Arndt et al. 2011), with reported satisfactory validity and reliability (e.g. Jeyakumar et al. 2004). The shortened version consists of the four subtests: (i) Digit symbol substitution/coding (complete); (ii) Arithmetic (only the odd items); (iii) Block design (only the odd items); and (iv) Information (every third item). WAIS-III (abbreviated) took approximately 20 minutes to complete and a copy is provided in *Appendix VI*. A trained researcher used the standardised instructions (Wechsler 1997) to conduct the assessment and calculate the IQ score based on the responses obtained.

3.4.1 Measuring schizotypy: Structured Interview for Schizotypy Revised (SIS-R) (Kendler et al. 1989)

3.4.1.1 Background and description of the SIS-R

A copy of the SIS-R that was used in this thesis is provided in *Appendix VII* and described in detail below.

The Structured Interview for Schizotypy (SIS) was developed following a large controlled family study of schizophrenia in Ireland (Kendler et al. 1989) and built on some limitations from previously available measures of schizotypy (e.g. self-report questionnaires (Kendler et al. 1996b)). Schizotypal symptoms and signs as measured by SIS were found to significantly discriminate the relatives of schizophrenic patients from relatives of controls. This interview version of measuring

schizotypy differed from the previous assessments in the following characteristics (Kendler et al. 1989 p.559):

(i)   *SIS has a built-in contextual assessment of the pathological nature of schizotypal symptoms (e.g. suspiciousness, referential thinking),*

(ii)   *multiple independently scored items and mostly closed response options (adapted from previous instruments) (Chapman and Chapman 1980;Eckblad and Chapman 1983),*

(iii)   *extensive assessment of schizotypal signs in addition to symptoms (e.g. odd behaviour, odd speech) based on observation of the respondent during the interview,*

(iv)   *symptom probes designed to make a participant's positive responses seem nondeviant (e.g. 'Many people sometimes have the feeling when they are in a group that people are looking at them. Do you know that feeling?'),*

(v)   *coverage of relevant symptoms and signs which were not a part of the criteria for schizotypal personality disorder (mirroring Meehl's (1990) view of schizotypal personality as a broader construct, not equivalent to DSM-V definition of SPD).*

SIS-R is a revised version of the original tool incorporating the improved standardised rating procedures (Vollema and Ormel 2000). The measure stands as *'the best candidate for measuring the broad range of (familial) schizotypal symptoms' (p.619)*, as it covers the multidimensionality of the concept and it is able to gauge mild symptomatology (Vollema and Ormel 2000). It also makes a distinction between symptoms (reported by the interviewee) and signs (observed by the interviewer) *(further explained in the next section 3.4.1.2 Composition of SIS-R).*

As the seven-point scale originally created (Kendler et al. 1989) resulted in many arbitrary decisions (due to the subjectivity of the raters), a revised version suggested a four-point rating system to standardise the scoring method (Vollema and Ormel 2000). The frequency and duration associated with schizotypal symptoms and signs are seen as objective variables therefore used to assist in rating symptom severity. For psychotic-like schizotypal symptoms especially (e.g. referential thinking, illusions) another criterion was added, assessing the level of conviction subjects have in their beliefs, again impacting on the symptom severity score (Chapman and Chapman 1980). The stronger the conviction in the (false) belief the respondent holds, the more pathological the idea (and can also develop further into delusional thinking) (Vollema and Ormel 2000). Moreover, the fourth revision of the SIS-R further expands the symptoms' criteria and adds the level of dysfunction associated with schizotypal symptoms. Studies suggest sufficient levels of test-retest reliability, proposing the SIS-R stands as a reliable research instrument for measuring schizotypal features, covering all three dimensions of schizotypy (Vollema and Ormel 2000).

The SIS-R can be subsumed into positive and negative/disorganised schizotypy. Positive schizotypy comprises the following items: referential thinking (part 1: being watched), referential thinking (part 2: being talked about), suspiciousness, magical ideation, illusions, psychotic symptoms and derealisation/depersonalisation (in total containing seven items). Negative schizotypy consists of symptoms such as: social isolation, introversion, hypersensitivity, restricted affect (in total four items). Disorganisation schizotypy encompasses the signs such as: goal directness of thinking, loosening of associations, poverty of speech and oddness (containing in total four items). All the items in the scale are not based on diagnostic criteria and as such reflect the psychosis continuum (dimensional approach to psychosis).

3.4.1.2   Composition of SIS-R

SIS-R consists of 15 sections, 11 of which assess individual schizotypal symptoms/traits and four constitute schizotypal signs (Kendler et al. 1989;Vollema and Ormel 2000) *(SIS-R in Appendix VII, the questions/quotes in this section are direct transcripts from the measure).*

Social Isolation

This section consists of 11 questions. The main part forms 5 closed-option questions that include frequency of contact with friends and family, how close they feel to their friends, frequency of attendance at meetings, social groups or other organisations and religious services. The individual is also asked about how many friends he/she has (where friends are defined as people with whom they have regular contact, including by mail or telephone) and with how many people the individual can share their most personal feelings. The last three questions explore participant's withdrawal from social contacts, which is then field-coded as to what the objective reasons for the isolation are (e.g. individual lived in a remote area without public transport, had a disabling medical condition that prevented him from socialising, has a demanding job). Social isolation relates to the individual's current situation.

Introversion

Introversion and all the rest of the sections aim to explore what sort of a person the individual is generally speaking. If some of their feelings and behaviours have changed over the years, the interviewer enquires about the most apt description of them as a person. The first two questions explore how 'alone' and how 'outgoing' a

respondent is (generally speaking) by providing the following options for each of the two characteristics: 'quite a lot', 'somewhat a lot', 'a little', 'not at all'. The second part of this section consists of 19 yes/no statements, inquiring about an individual's behaviour and feelings associated with different social activities/situations (including anxiety in social situations, self-consciousness etc.).

Hypersensitivity

The first two opening questions to this section address individuals' sensitivity to comments and remarks made about them, including the time they spent thinking about unpleasant remarks before letting them go (by choosing the options provided: 'a week or more', '2 to 3 days', 'one day, 'one hour/few hours'). At the end of the section, the subject is asked about their responses to critical comments, including being considered 'foolish', being 'sensitive', 'touchy' and 'emotionally thin-skinned'. For all four statements given, the decision is made by the participant whether it is 'Certainly true', 'Probably true', 'Probably not true' or 'Certainly not true'.

Referential thinking (PART 1: being watched)

This section starts with a screening question that also *'attempts to make a positive response appear nondeviant'* (Kendler et al. 1989 p.563):

4.1 Many people sometimes have the feeling when they are in a group that people are looking at them.

Do you know that feeling? If yes, how often have you had such a feeling? Would you say 'often', 'sometimes', 'seldom' or 'never'?

Respondents who answer 'never' skip the rest of this section and continue with section 5, otherwise they are asked a series of other questions about their feelings

of being watched, including how many people seem to be watching them and if they feel they are getting special attention. Additionally, they are asked to give an example of one time they remember when they had that feeling and explain their reason(s) as to why they felt they were being looked at. This is a field-coded item - if describing a non-pathological experience ('normal reaction', for example a really tall person having a feeling of being watched) the interviewer jumps to the next section, if scored otherwise, further questions are asked, including where does the feeling usually occur ('only in their neighbourhood', 'only somewhere else' or 'both, in neighbourhood and away from home', 'doesn't apply'), who are the people who seem to be watching them ('both acquaintances and strangers', 'only strangers', 'only acquaintances') and if they think this would happen again ('yes certainly', 'probably would', 'certainly would', 'certainly not'). The last few questions are about the frequency of these feelings ('once a year', 'once a month', 'once a week', 'other') and the level of conviction the subject has about these experiences ('no doubt', 'some doubt', 'a lot of doubt'). The section concludes with one yes/no question whether these experiences affect the individual's daily life.

Referential thinking (PART2: being talked about)

The structure of this section is similar to section 4. It starts with two introductory questions:

5.1. People sometimes get the feeling in public places that people around them are talking about them.

Do you know that feeling? 'Often', 'sometimes', 'seldom' or 'never'?

5.3. Did you ever have the feeling of being laughed at? 'Often', 'sometimes', 'seldom' or 'never'?

If answers to both of these questions are no, then the interviewer continues with section 6. Additional questions are asked for any positive responses. Again, the interviewer inquires about the reasoning for these experiences and a field-code is assigned to an answer (from 'normal reaction' to 'no reason – excessive reaction'). Following that, the respondents are asked if other people are making insinuations about them and again by giving two examples a field-code is given (ranging from 'clearly pathological' to 'clearly normal'). As in section 4, the interviewer asks for the frequency of all these experiences along with the level of conviction. The section concludes with eight closed-option items (with possible responses 'often', 'sometimes', 'rarely', 'never') that summarise the entire section.

Suspiciousness

This section attempts to cover a few key domains of suspiciousness (Kendler et al. 1989 p.563): (i) perceiving of oneself as trusting versus suspicious; (ii) considering other people to be selfish versus altruistic; (iii) distrusting other people; (iv) feeling inappropriately blamed and criticised and (v) needing to be on guard when among other people. The first set of statements has the 'frequency' response option ('often', 'sometimes', 'rarely', 'never'), whereas the second section's response set is a level of (dis)agreement ('certainly agree', 'probably agree', 'probably disagree', 'certainly disagree'). The section ends with a field-coded item that asks whether 'there are people especially determined to frustrate you and make your life difficult', which is followed by the individual's need to take special precautions and a question concerning how the individual gets on with his/her neighbours. An objective reason for suspiciousness is selected by the respondent (either 'nothing has happened in their lives to make it view it this way', 'a little has happened', 'some has happened' or 'a lot has happened') along with a yes/no response concerning whether they think they are a suspicious person.

Restricted affect

This section contains 6 statements on frequency ('often' to 'never') of strong emotions experienced by the respondent. If the individual cannot express the positive emotions more than 50% of the time, the frequency and duration of restricted affect is defined.

Magical Ideation

The first part of this section covers two lists of closed-option statements that include several aspects of magical thinking. Response options for the first set are based on endorsement ('certainly true', 'probably true', 'probably not true', 'certainly not true'), whereas the second response set involves the frequency of these experiences ('often' to 'never'). The second part of the section measures superstitious beliefs (e.g. having ideas that there are things that can bring fortune or misfortune to them, or items that bring them luck). The field-codes are provided to gauge the deviance from subcultural norms. Again, the section concludes with two questions about the frequency of these experiences and the level of conviction about them.

Illusions

This section gathers information about visual and auditory illusions, beginning with the following opening statements:

9.1 People sometimes have the experience of mistaking some object for a human or an animal. (…) Does this ever happen to you? 'Often', 'sometimes', 'seldom' or 'never'?

9.2 Some people sometimes think they heard sounds that are likely not real, such as crackling, a knock, or the sound of a bell? 'Often', 'sometimes', 'seldom' or 'never'?

This is followed by further questions about experiences of hearing voices talking or whispering to them and the feeling of a particular person or force being around them (when they saw no one). Six other items are related to the concept of perceptual aberration and assessed on a 'never' to 'often' scale. This section concludes with three questions about the frequency of the experiences, the level of conviction about them and the effect they have on their lives.

Psychotic phenomena

This section includes 12 questions, assessing different psychotic-like experiences, again aiming to make a positive response appear non-deviant and nonthreatening, for example:

10.2 How often do your thoughts suddenly stop, so that you completely lose train of your thought? 'Often', 'sometimes', 'seldom' or 'never'?

10.8 Sometimes people have the feeling that their thoughts are so real that it is like they were being spoken out loud? Does it ever happen to you? 'Often', 'sometimes', 'seldom' or 'never'?

Positive response to 'thought – insertion' like experiences is followed by further exploration, including the questions about what force or power has put the ideas in their heads, and how out of the ordinary these experiences were? This section concludes with two questions about the frequency of these experiences and the level of conviction.

Derealisation/Depersonalisation

This last section consists of 3 main items, beginning with the opening question:

11.1 Have you ever had the feeling that the surroundings and the people around you were unreal, as if you were looking through some kind of fog? 'Often', 'sometimes', 'seldom' or 'never'?

The other items cover areas such as having 'unreal feeling about yourself' and 'stepping out of your body'. In the case of a positive response to 'stepping out of the body', the interviewer inquires whether this only happened under the influence of medicine or drugs/or when physically ill. If this is the case, the interviewer stops here. Otherwise, further questions explore the frequency and duration of these experiences.

SIS-R Signs / Observation during the interview

The next section is designed to be completed by the interviewer based on the unstructured part of the discussion with the subject.

Goal-directness of speech/thought process

The interviewer assesses any disturbances in the goal-directness during the interview (if respondent answers in a logical and direct manner or wanders off the topic), including verbosity (do these wanderings ultimately come back on track) and vagueness (inability to follow the subject's thought pattern clearly).

Elevated associativity

The interviewer assesses the subject's disturbances in association of thinking, especially if the train of thought could or could not be followed.

Poverty of speech

The interviewer evaluates how many words does the subject need to come up with an answer to the question and how often did the interviewer have to prompt the subject.

Odd/Eccentric behaviour

This dimension is divided into motor behaviour (non-verbal odd behaviour, physical posture, odd tics or other movements), subject's social behaviour (being too familiar, too intrusive, staring, inappropriately enticing, flirty, hostile etc.) and subject's clothing (clothing and grooming appropriate or inadequate).

3.4.1.3   Administration/scoring of SIS-R

The SIS-revised provides clearer definitions of symptoms and signs (Vollema and Ormel 2000) on a four point scale taken from Baron's SSP scale (The Schedule for Schizotypal Personalities) (Baron et al. 1981), therefore reducing the subjectivity of the raters. Also, as previously mentioned, standardised questions were added to assess the 'frequency, duration and conviction', which were shown to assist with symptom severity scoring (Vollema and Ormel 2000).

The ratings were made on a scale, ranging from 'Absent' to 'Severe'. For example: *Social isolation* is rated as *(SIS-R instruction book p 9. (Vollema 2010)):*

  (i)   *Absent (0) 'when the person exhibits none of the most minimal indicators for social withdrawal and no social isolation' (p.9)*; e.g. someone has a wide network of friends, including intimate relationships, talks or sees

them on a regular basis, is an active member of a social group and never withdraws without an objective reason.

(ii)   *Mild (1) 'when there are sometimes mild (incidental, less than once a month, hours at the time) short lived social withdrawal behaviours' (p.9)*; e.g. seeing family or friends less regularly and experienced need to be alone for a few hours, slight hesitation when making new contacts.

(iii)   *Moderate (2)* when *'there are often periods of moderately severe social withdrawal, relatively short in duration (hours rather than days)' (p.9)*; e.g. reduced social activity, someone who shuts himself up in his house for a few days a year, but does not refuse the contacts that are offered like under 'severe' rating.

(iv)   *Severe (3)* rated when *'there are severe forms of social withdrawal (that is, frequent and protracted). This can last for days or even weeks at a time' (p.9)*; e.g. beside being socially inactive, the person refuses offered contacts.

As such, the SIS-R assesses more subtle schizotypal features and not chronic schizotypal personality disorder. For instance, social isolation trait scores would differentiate between milder, clinically not significant forms of isolation but not between levels of severe and chronic social isolation (Vollema and Ormel 2000). Because of that, SIS (and SIS-R) is a research and not a clinical instrument (Kendler et al. 1989) and the scoring system does not have a dichotomised outcome (absent or present), which would facilitate a decision regarding diagnosis. Besides, that would also be misleading as it would imply that we know the true boundaries of schizophrenia (Kendler et al. 1989). Researchers are instead given an option to rely on global scores, or either take the narrow criteria, where only 'severe' scores are used or broader criteria (using 'mild', 'moderate' and 'severe' scores as symptom

'present') (Kendler et al. 1989). The maximum total score for the SIS-R instrument is 60, for positive schizotypy it is 28 and for negative/disorganised schizotypy it is 32. The assessment is completed by a trained researcher and takes approximately 30 minutes.

3.4.1.4   Validity and reliability of SIS-R

Data shows the overall inter-rater reliability of the SIS instrument is good to excellent (Kendler et al. 1989). There are however a few 'problematic' items that are more dependent on the context in which they occur, thus considerably less reliable. SIS showed satisfactory interclass correlations, where the IC coefficient for the seven symptom scales was *0.87 (SD=0.12)*, with only magical thinking scoring lower *(0.75)* (Kendler et al. 1989). Not unexpectedly, reliability in assessments of signs was lower than that of symptoms (ICC equalled *0.69*) (Kendler et al. 1989). Looking at the close-option items only, the ICC was *0.97 (SD=0.07)*, lower than *0.95* only on two items: 'I wonder whether the people I know can really be trusted' *(ICC=0.92)* and 'How often do thoughts come into your mind that feel as if they do not belong there' *(ICC=0.69)* (Kendler et al. 1989). For the four field-code items of the SIS the mean ICC was *0.76*, with only one item scoring below *0.75*: 'Are there people who have gone out of their way to hold you back in life and to make things difficult for you?'. The mean ICC for the 12 global scores (integrated responses of all items in each section) was *0.74* (Kendler et al. 1989). According to Kendler (Kendler et al. 1989), the most useful validation method for SIS provide the studies using the nonpsychotic relatives of schizophrenic patients and control probands as two comparison groups. Three independent studies showed that SIS discriminated between relatives of individuals with schizophrenia and relatives of control groups (validity of the instrument) (Tsuang et al. 2002a).

SIS-revised version showed that the following schizotypal items/traits demonstrated sufficient levels of inter-rater and test-retest reliability (Vollema and Ormel 2000): social isolation, introversion, hypersensitivity, referential thinking, suspiciousness, restricted affect, magical ideation, psychotic symptoms, derealisation/depersonalisation and antisocial behaviour (Vollema and Ormel 2000). Few other items were shown to be more context-dependent and as such more problematic: social anxiety, referential thinking (seeing meanings), illusions, irritability, impulsivity, and dysfunction (personal caretaking and leisure time activities) (Vollema and Ormel 2000). 'Illusion' had a kappa value of *0.39* and even though the items with lower kappa than *0.40* were excluded, SIS-R still kept the assessment of that particular trait as it has been previously considered being the key elements of schizotypy (Meehl 1962;Raine et al. 1994). Similarly, only four signs in SIS-R (goal directedness of thinking, loosening of associations, amount of speech, and oddness) (Vollema and Ormel 2000) were shown to have good levels of inter-rater and test-retest reliability and they are the only ones included in the adapted version of the instrument as used for this thesis.

Some of the main limitations of the SIS-R are the length of the instrument, its life-time framework and force-choice question format (Kendler et al. 1989;Vollema and Ormel 2000). It is also argued that even though SIS includes a broad range of symptoms and signs (Kendler et al. 1989) it still covers a narrow area comparing to more general personality disorder instruments (e.g. Structured Instrument for DSM-III Personality Disorders) (Pfohl et al. 1983). Yet, only that it gives sufficient focus on schizotypy (Kendler et al. 1989). The interview based assessment of schizotypy was also suggested to better assess familial risk factors than self-report measures (Catts et al. 2000), which may be one of the reasons that SIS-R has been used in clinical and genetic-epidemiological investigations (Myin-Germeys et al. 2011;Vollema and Ormel 2000). For the above reasons, the SIS-R was selected as the main measure

of schizotypy in the study that this thesis is based upon. Based on preliminary estimates, the inter-rater reliability in the EU-GEI study was *0.80* (measured in 2011), *0.79* (measured in 2012) and *0.71* (measured in 2013).

3.4.2 Measuring psychosis-like symptomatology: Community Assessment of Psychic Experiences – (CAPE) (Stefanis et al. 2002)

CAPE is a 42-item *(originally 40-item (Hanssen et al. 2003;Stefanis et al. 2002))* self-report assessment of attenuated psychotic experiences in the affective and non-affective domains (Hanssen et al. 2003). It assesses the presence and frequency of lifetime psychotic-like symptoms, including the distress associated with these experiences. Each of the items is rated on a four-point dimensional scale ranging from 1 (never), 2 (sometimes), 3 (often), to 4 (nearly always), followed by a four-point Likert scale measuring distress - 1 (not distressed), 2 (a bit distressed), 3 (quite distressed) and 4 (very distressed). The instrument was originally created based on the 21-item Peters et al. Delusions Inventory (Peters et al. 1999). Few items were omitted due to their ambiguousness (Verdoux et al. 1998) and in addition, 2 items on auditory hallucinations, 8 on depressive and 14 on negative symptoms were added, taken from the Scale for the Assessment of Negative Symptoms and the Subjective Experience of Negative Symptoms (Andreasen 1982;Selten et al. 1993). The measure is, therefore, partly based on diagnostic criteria (assessing deviation from 'normal' experiences), however it adopts the core assumption that attenuated psychotic symptoms can be observed throughout the general population and their clinical value is defined by symptoms' severity and associated distress.

CAPE items can be subsumed under three dimensions consisting of positive (20 items), negative (14 items) and depression symptoms (8 items) (Konings et al.

2012). The evidence for three dimensions of the scale derives from community samples of young men (Stefanis et al. 2002) and female student populations (Verdoux and van Os 2002). The instrument provides the total score (sum of all the items, minimum 42 points to maximum 168) and a total score for each of the three dimensions: positive (min=20 to max=80), negative (min=14 to max=56) and depressive (min=8 to max=32). A copy of the CAPE is provided in *Appendix VIII*.

The three-factor structure has shown discriminative validity across groups of individuals with schizophrenia and other psychotic disorders, mood disorders, anxiety disorders and a non-patient group, with patients with psychotic disorders having the greatest difference in positive psychosis items compared to non-patients *(β= 0.94, 95 % CI: 0.7-1.18)* (Hanssen et al. 2003). Also, the CAPE distress score did not differ significantly among the patient groups, but was significantly higher than in a community population (Hanssen et al. 2003). Interestingly, the CAPE 'psychotic scores' of patients with psychotic disorder compared to the nonclinical group showed some overlap (e.g. range between 1 to 38 for clinical vs. 1 to 28 for nonclinical), suggesting that some nonclinical subjects had more psychotic experiences than schizophrenia patients (although no information is given as to whether the nonclinical group included currently recovered people) (Hanssen et al. 2003).

CAPE was shown also to have a family-specific variation (family-specific variance accounted for *10%* to *40%* of the total variance) and stability over time (Hanssen et al. 2003;Hanssen et al. 2006;Konings et al. 2006). Convergent validity of the CAPE was shown via correlation with the depression scale of the Symptom Checklist-90 (for CAPE depressive subscale), with positive symptoms in the Perceptual Aberration Scale and negative symptoms from the Schizotypal Personality Scale (Stefanis et al. 2002). There was also a good internal consistency of all three subscales (Cronbach's alpha *0.78* to *0.83*) (Brenner et al. 2007).

In contrast to the SIS-R measure that assesses schizotypal experiences/traits, not directly echoing patient experiences (Johns and van Os 2001;Stefanis et al. 2002), CAPE focuses on attenuated forms of psychotic symptoms. It also measures depression (emphasizing that affective symptoms represent an essential part of psychotic disorders), that was excluded from the schizotypy scales (Stefanis et al. 2002). Nevertheless, the literature suggests a moderately strong association between the positive dimension of the CAPE and the positive dimension of the SIS-R $(β=0.52$, $t=8.48$, $p=0.000)$ and the negative dimension of the CAPE and the negative dimension of the SIS-R $(β=0.50$, $t=9.19$, $p=0.000)$ (Konings et al. 2006). The main difference between CAPE and SIS-R is a distinct stand-point with regard to the psychosis continuum model (Konings et al. 2006). Even though they both capture the subclinical expression of psychosis, SIS-R mirrors the fully continuous approach, while CAPE measures symptoms resembling clinically-defined psychotic phenomena (Konings et al. 2006). Therefore, both measures are utilised within this thesis.

3.4.3 Measuring Childhood Trauma: A modified version of the Childhood Experience of Care and Abuse (CECA) 'Interview Version' (Bifulco et al. 1994)

The increased interest in the role of childhood trauma on adult psychopathologies has led to the rise of assessment tools measuring traumatic experiences, especially self-report/questionnaires style measures which are associated with a less accurate recall comparing to interview-style tools (Finkelhor 1986). The CECA instrument was developed incorporating techniques that are likely to increase the effectiveness of the recollections (Bifulco et al. 1994): importance of the recreation of the context surrounding the traumatic event (Tulving and Thompson 1973), continued attention to the topic (Kahneman 1973) and allowing more retrieval attempts (Roediger and

Payne 1982). The interview version of the original Childhood Experience of Care and Abuse measure is designed to assess a wide range of negative childhood experiences prior to the age of 17 (Bifulco et al. 1994;Bifulco et al. 2005). CECA focuses on objective information relating to behaviour rather than subjective experiences/feelings (Bifulco et al. 1994). It also covers a wide range of early traumatic experiences, including household discord, psychological abuse, physical abuse and sexual abuse. The childhood traumatic experiences are rated on a 4-point scale: 0 (none), 1 (some), 2 (moderate), 3 (marked), with the only exception of household discord where the 5-point scale was used: 0 (none), 1 (some), 2 (moderate), 3 (marked), 4 (violence). For a full measure see *Appendix IX.*

One of the main advantages of CECA (Bifulco et al. 1994) measure is a face-to-face interview format. This has been shown to be the best type of measure, as it allows for greater rapport and clarification (Finkelhor 1986). When compared to some other candidates for childhood trauma assessment *(e.g. self-reports such as Childhood Trauma Questionnaire (Bernstein et al. 1997), Childhood Experiences of Violence Questionnaire (Walsh et al. 2008)),* which have an advantage of being easily and quickly administrated*,* CECA gathers more detailed, contextual information about childhood abuse, as well as documenting the age of trauma occurrence, and frequency and severity of abuse. Not only have all these factors been previously shown to have an important role in childhood trauma - psychosis (psychosis-like) relationship (e.g. Fisher et al. 2010), but inclusion of these factors can reduce the subjectivity of trauma interpretations of the interviewees (Bifulco et al. 2002). Another important value of the CECA measure is a standardised 4-point *(to 5-point)* scale *(ranging from mild to severe forms of abuse)* on which the ratings are made by investigators. In this approach, CECA is an advance on some childhood abuse measures adopted in previous studies, where the items are rated as yes/no only *(e.g. Assessing Environments III, (Berger et al. 1988)),* possibly contributing to under-reporting of childhood traumatic experiences (Finkelhor 1986).

Also, using one measure where different types of trauma are assessed *(instead of combination of few measures, each assessing specific trauma type e.g. Physical Punishment Scale (Berger et al. 1988), Sexual abuse scale (DiTomasso and Routh 1993))*, allows for more reliable comparison of effects these distinct trauma types have on schizotypy load.

However, one of the main limitations of CECA instrument is that it only evaluates four types of childhood abuse and does not consider other forms of traumatic experience *(documented in the literature as important predictors of psychosis)*, for example general traumatic events or natural disasters covered by measures such as The Early Trauma Inventory - ETI (Bremner et al. 2000). Although there are several childhood trauma instruments available not many have published reports on their psychometric properties (Bremner et al. 2007). The Early Trauma Inventory has excellent reliability and validity and has an additional value in its inclusion of the impact the trauma has on the individual (Bremner et al. 2000). However, ETI is clinician-administered assessment, therefore much more challenging to be employed as a research instrument. The CECA measure has also been widely validated (e.g. Bifulco et al. 1994; Moran et al. 2002).

3.4.3.1   Composition of the CECA - Interview measure

CECA covers a wide range of early traumatic experiences but consists of four core constructs: household discord, psychological abuse, physical abuse and sexual abuse, thus reflecting key childhood risk factors related to adolescent and adult psychopathology (Lifespan Research Group 2009). The content of the measure originally derived from two studies exploring the development of adult depression in women (Bifulco et al. 1994). The measure was then used on a large representative sample of inner-London mothers and consequently physical abuse and sexual

abuse were added (Moran et al. 2002). Psychological abuse was added to CECA most recently *(and had been a neglected area for a long time, because of problems such its operational definition or under-reporting)*, and has more sadistic and cruel qualities to it compared to other forms of abuse (Moran et al. 2002). The CECA authors conceptualised various forms of psychological abuse *(further defined in section 3.4.3.1)* rather than (more commonly measured) emotional abuse. Although some tried to distinguished between psychological and emotional abuse *(e.g. emotional abuse affects the child's emotional wellbeing and emotional development whereas psychological abuse damages their mental wellbeing and impairs mental development (O'Hagan 1995))*, the constructs are overlapping and in the literature often used interchangeably. The CECA authors do not make the distinction between this two concepts, but prefer the term 'psychological abuse' (Moran et al. 2002). Their definition of this type of abuse however sets a high threshold for inclusion of abusive behaviours *(related to long-term psychopathological outcomes)*, in order to distinguish them from those already assessed in other subscales (Moran et al. 2002).

Despite recognising that different trauma types often overlap *(a clustering of victimisation (Dong et al. 2004))* and especially that psychological abuse correlates highly with other forms of childhood adversities (Moran et al. 2002), it is still important to be able to differentiate them in order to explore the specific effects distinct trauma types have on adult psychopathology. Therefore, CECA would not rate separately the abuse that was only subsidiary to what was considered the main type of abuse, for example if psychological abuse appears as a feature of the same sexual or physical abuse incident is treated as a complex and rated simultaneously (Lifespan Research Group 2009). The support for this approach can also be found in a recent study on typologies of child abuse in a community population using a latent class analysis that revealed distinct abuse typologies despite their frequent co-occurrence (Armour et al. 2013).

The CECA interview version consists of 16 items, all relating to the respondent before the age of 17. It begins with 9 opening questions covering the following topics (Bifulco et al. 1994): (i) family living arrangements; (ii) loss of a biological parent/parents; (iii) separation from a biological parent/parents; (iv) change of schools; (v) exclusion from school; (vi) runaway from home; (vii) institutional care (children's home, fostered); (viii) significant financial problems in the family and (ix) neglected basic needs. These introductory questions are followed by four main sections, concerning (x) household discord and three types of childhood abuse: (xi) familial psychological abuse, (xii) familial physical abuse and (xiii) familial and non-familial sexual abuse. The interview concludes with three questions, two regarding availability of supportive figures (xiv): supportive adult figures & (xv): supportive others their age) and one concerning prolonged feelings of loneliness (xvi).

Living arrangements

The section captures all respondent's family arrangements before the age of 17. The subjects are questioned about the mother and father figure (who brought them up). These options include: no mother/father figure; natural mother/father; step-mother/father; grandmother/father or other (other relatives or adoptive or foster parents). Each living arrangement is noted, including the age at which it started. The arrangements are required to have lasted at least 6 months to be documented.

Death of a parent / Parental separation

If any of the biological parents died before respondents were aged 17 then this is documented along with the age of the respondent at the time of their death. Also, separation from their biological mother and/or father is noted, including the age when separation begun and its duration in months. Separation is for the purposes of

this interview defined as 'not physically living with the parent', apart from separation due to parent's death which is only entered under parent's loss. If separation occurred, the interviewer inquires about the reasons for the separation, which fall under one of the following categories: parental loss; divorce/separation; work; never knew parent; own illness; boarding school; migration or other.

Household discord

This section captures the level of arguments (rowing, violence, including non-personal violence e.g. breaking things) and the tension present in the family as a whole (Bifulco et al. 1994).

It starts with an introductory question *(questions in this section are direct transcriptions from the measure):*

10a. Were there ever frequent arguments or extreme tensions between your parents (or other adults in your household)?

In cases of a positive response to the question, more detailed exploration followed. The interviewer inquires what the family arrangement was at the time, the frequency of these experiences and duration (the respondent's age when it started and ended). Also, the support received at the time is documented (emotional and practical support), including the negative support (e.g. clear statement that the person is to blame or deserved what happened) and official contact (e.g. involvement of social services, police, GP). The questions are repeated for any discord in other family arrangements.

Psychological abuse

The CECA interview (Bifulco et al. 1994) defines psychological abuse as humiliation, degradation, induced shame by comments or actions that degrade and humiliate the

child, terrorising such as invoking fear in a very calculated way, depriving a child of basic needs (beyond neglect), extreme rejection or emotional blackmailing. It only includes severe intentional psychological abuse that is not subsidiary to sexual or physical abuse.

The section begins with four screening question:

11a. Where you ever tormented or treated cruelly by a parent or a member of household?

11b. Did anyone try to frighten you?

11c. Did anyone humiliate you? (e.g. belittle you in front of others, ridicule you)

11d. Did you ever feel that punishments at home were totally unnecessary?

In case of any positive responses to the questions, more detailed exploration followed. The interviewer inquires who the perpetrator was, the frequency of these experiences and the duration (the respondent's age when it started and ended). Also, the support received at the time is documented (emotional and practical support), including negative support (e.g. clear statement that the person is to blame or deserved what happened) and official contact (e.g. involvement of social services, police, GP). If a respondent reports multiple/different experiences of psychological abuse involving different perpetrators, the subsequent questions are repeated.

Physical abuse

The physical abuse section assesses the degree of physical violence to the child from people within the same household (Bifulco et al. 1994). The section begins with three screening questions:

12a. Were you ever hit or slapped on a number of occasions, sufficient to cause harm?

12b. Did parents ever hit you?

12c. Was there ever any violence toward you in the household?

Any positive response is followed by more detailed questions, including the context of abuse such as severity, frequency and perpetrator. Also, the interviewer considers the extent to which the subject was in physical danger, the perpetrator was out of control, the nature of the attack in terms of weapon/object used and type of hitting (duration of each attack/repetition) and likelihood of injury (or actual injury) (Bifulco et al. 1994). This section concludes with questions about emotional and practical support received at the time as well as any official contacts. If a respondent reports multiple experiences of physical abuse involving different perpetrators, the subsequent questions are repeated.

Sexual abuse

Sexual abuse includes most cases of sexual intercourse before puberty as well as sexual approaches by peers if coercion or force was used. Also, it includes incidents where there is physical contact or non-contact verbal sexual solicitations if the perpetrator is a parent, sibling, relative, authority figure or other (e.g. stranger). Willing sexual contact with peers is not rated (Bifulco et al. 1994). This section contains four leading questions, quite similar in their content, but each of them offers a slight variation in the description of unwanted sexual experiences:

13a. Did you ever have any unwanted sexual experiences?

13b. Did anyone ever force or persuade to have sexual intercourse against your wishes?

13c. Did you ever experience any other upsetting sexual experience with a relative or person in authority?

13d. Were you ever in a situation where you were nearly involved in an unwanted sexual experience, but managed to avoid it?

Because of the sensitivity of information as well as other pertinent issues that impact trauma recollections *(see section 2.1.5.1 Issues in childhood trauma reporting)*, four variations of the question could minimize the false negative responses (e.g. words like 'unwanted experiences' might not be identified by those who blame themselves for abuse) (Finkelhor 1984). As in previous sections, a positive response is followed with more detailed questions about the perpetrator, frequency of abuse and the duration. The nature and context of these experiences are also discussed, whether it involved non-intercourse (touching/fondling) or intercourse (vaginal or anal) abuse. Emotional and practical support received at the time is noted along with any official contacts. If a respondent reports multiple experiences of sexual abuse involving different perpetrators, the subsequent questions are repeated.

3.4.3.2   Administration/scoring of the CECA - Interview measure

All researchers involved in the study received training in the CECA measure. At the beginning of the interview it was explained to participants that the following section would touch on some potentially sensitive experiences up to the age of 17. Also, they were reminded that some of the questions might bring up upsetting or painful memories but the interview can be stopped at any time (or the question skipped) if they do not feel comfortable answering. Researchers also reiterated that all the information provided would be treated in the strictest confidence.

In cases where a participant got upset or uncomfortable during the interview they were again reminded there was no need for them to answer and asked if they wanted to stop the assessment. They were also allowed as much time as needed before continuing with the next question. In very few cases (for sexual abuse

questions especially) where participants expressed the preference of writing their experiences down instead of verbally sharing, a clean sheet of paper was provided and some instructions given of what to include: age when happened, frequency etc. Participants were also encouraged to contact the research team or their GPs in cases where adverse reactions to the interview were observed by the researcher. This was especially important as the research shows that disclosure of traumatic experiences in a research environment may result in increased emotional distress (Carlson et al. 2003;Draucker 1999;Newman et al. 1999;Ruzek and Zatzick 2000). Castor-Lewis (1988) looked at participants who experienced incest, and warned that the research environment might be the setting where the trauma is revealed or the childhood experiences have possibly not even been disclosed before taking part in a research study (Ullman 1996). Nevertheless there is a lot of evidence that adverse reactions after participating in a study about victimisation are less common and a great majority of the subjects report positive experiences and beneficial effects associated with taking part (Greenberg and Stone 1992;Henderson and Jorm 1990;Newman and Kaloupek 2004;Ruzek and Zatzick 2000;Walker et al. 1997).

The degree of doubt in the accuracy of subject's recall was rated at the end of the interview based on the following categories (interviewer's subjective impression): (i) No doubt; (ii) Doubt - Recall possibly influenced by symptoms, mental state; (iii) Doubt - Interviewer failed to clarify; (iv) Doubt - Other.

Scoring was completed in pairs, in a short space of time after the interview, using *The CECA - Interview manual scoring guide* (Lifespan Research Group 2009). For more complex experiences of abuse, the decision on ratings was made at the team consensus meetings to avoid interviewers' potential biases, as suggested by Bifulco and colleagues (Bifulco et al. 1994). Also, the 'degree of doubt' was taken

into account when finalising the ratings[14]. The conservative scoring was used to ensure that the severity of childhood experiences was not overvalued, and the false positive responses were minimized. For the analysis, CECA scores (for each of the trauma type) have been dichotomised where *(0) was Trauma absent*, and *(1) Trauma present*. Following established guidelines, trauma was considered *'present'* when the severity rating was *2 and above* ('moderate' or 'marked'; for household discord also including 'violence') *(for full description of the categories/variables used in the analysis see section 4.2.1.2)*.

3.4.3.3 Validity and reliability of CECA - Interview measure

Literature suggests that the use of a single assessment may lead to an under-estimation of the true prevalence of abuse (Fergusson et al. 2000), however retrospective assessment relies heavily on the quality of the measures of such experiences (Hardt and Rutter 2004). The instrument has high reliability, using a sample of 20 interviews assessed by the independent raters showed that on eight out of nine items their agreement reached above *0.78* (weighted kappa) (ranging between *0.63* and *0.92*) (Bifulco et al. 1994). The validity coefficient is satisfactory, assessed using 87 adult pairs of sisters measuring each others' experiences of neglect, physical and sexual abuse, showing the average correlation of *0.60* (weighted kappa) (Bifulco et al. 1997). Another study reported that retrospective self-report measures of sexual abuse have some construct validity (Widom and Morris 1997) and reports of adversity are stable over time (test-retest reliability) (Fisher et al. 2011) (*see section 2.1.5.1 for Issues in childhood trauma reporting*).

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[14] *In the present study, the degree of doubt in the accuracy of subject's recall was rated for 6 participants. Once the doubt was due to participant's poor understanding of English and five times researchers failed to clarify the details with regard to childhood trauma. Therefore, the doubt was taken into account when finalising the ratings, rather than excluding these participants from the analysis.*

Based on preliminary estimates, the inter-rater reliability in the EU-GEI study was

0.81 (measured in 2011), *0.78* (measured in 2012) and *0.88* (measured in 2013).

3.4.4 Measuring bullying: Bullying Questionnaire *(adapted from the Environmental Risk (E-Risk) longitudinal twin study (Arseneault et al. 2006;Shakoor et al. 2011))*

The questionnaire assesses bullying before the age of 17. The definition of bullying

is read to the respondent and includes the following experiences: mean and hurtful

things being said, made fun of, being called mean and hurtful names, being ignored

or excluded from the group of friends, hit, kicked, lies or rumours spread, other

hurtful things. Bullying is not rated for experiences done in a friendly or playful way.

If the respondent did experience bullying, the age of occurrence, duration, type of

bullying (emotional, physical) and support received (including any official contact)

are documented. The final question considers if a participant ever bullied others (for

which only the frequency is noted).

The ratings are then made on a four point scale, ranging from sporadic

teasing (scored 0) to physically hurt or intensive verbal bullying (scored 3). The test-

retest reliability of parent's reports of bullying victimisation was shown to be *0.87

(Arseneault et al. 2006).* The administration and scoring procedure followed the

same rules as CECA-Interview assessment (*see 3.4.3.2 Administration and scoring

of the CECA-Interview measure*). For a copy of the full measure see *Appendix IX*.

3.4.5 Measuring adult adversity: Life Events and Difficulties Schedule (LEDS) (Brown and Harris 1978)

The Life Events and Difficulties Schedule (LEDS) marked a considerable

development in the measurement of life experiences, replacing checklists and self-

report measures with a more comprehensive structured interview-style approach (Brown and Harris 1978). It also pioneered the use of contextualisation and full detailed exploration of life events in the 12 month period of the individual's life. The LEDS has now been used extensively in psychiatric, psychological and social studies, mainly on depression (Brown and Harris 1978). The measure shows a respectable inter-rater reliability, for instance *0.90* for assessing the severity of life events (Brugha and Cragg 1990). Probing questions are used to establish details of what had happened, when and with what impact. Each event is then rated according to a classification system (e.g. work, health, partner relationship etc.), the 'threat or unpleasantness' on a 4-point scale, the focus (on the respondent, joint focus with other person, focus on possession or on another person), dependence/independence from disorder, and finally the level of intrusiveness. Only events with a certain level of severity were previously shown to be associated with the onset of depression, as experiencing a high rate of non-severe events might increase the resilience to becoming depressed (Brown and Harris 1978). Events occurring in the study period often have difficulties developing from them. If the long-term consequences of the events do not clear by the end of 14 days (time period within which ratings in the events section are made), the longer term outcome is recorded – a difficulty. Similarly, difficulties (defined as lasting in excess of 4 weeks without interruption) are rated using the same attributes, apart from severity consisting of seven rather than four point scale (ranging from 'very mild' to 'high marked'. A rating is completed for a long-term threat only. The anchor points (e.g. birthdays, holidays etc.) within the study period were used to help with the accurate dating of events, a technique that has previously been shown to improve recall (Loftus and Marburger 1983). For the purpose of this thesis, only more severe events (scored at 2 – Moderate threat, unpleasantness or 1 – Marked threat, unpleasantness) and more severe difficulties (scored at 3 – High moderate, 2 – Low marked or 1- High marked) were included in the analysis *(for a full description of the*

categories/variables used in the analysis see section 4.2.2.2). A copy of the measure is provided in *Appendix X.*

LEDS was administered as a face-to-face interview and all researchers received training in the measure. The degree of doubt in the accuracy[15] of subject's recall was rated at the end of the interview, based on the interviewer's subjective impression using the same categories as with CECA (see *2.4.3.2*). Similarly, scoring was completed in pairs, in a short space of time after the interview. For more complex cases/experiences, the decision on ratings was made at the team consensus meetings that took place fortnightly. The cases were presented and discussed, and ratings completed in order to maximise the inter-rater reliability.

3.4.6   Measuring cognitive processes (core negative beliefs): Brief Core Schema Scales (BCSS) (Fowler et al. 2006)

This is a 24-item measure concerning beliefs about self and others. Yes/No questions were followed by a four-point rating scale (applicable for positive answers only): (i) 'Believe it slightly', (ii) 'Believe it moderately', (iii) 'Believe it very much', (iv) 'Believe it totally'. The scale assesses the core beliefs about self and others. These can be divided into four dimensions: 'positive self' (six items), 'negative self' (six items), 'positive other' (six items) and 'negative other' (six items). The individual is asked to indicate if the beliefs are held (and to what degree). Evidence suggests good internal consistency across all four subscales of the instrument *(Cronbach's alpha>0.77)* and excellent test–retest reliability (Fowler et al. 2006). The unique advantage of the measure is its evaluation of different aspects of self and others reflecting contemporary schema constructs observed in psychosis patients (Fowler

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[15] *In the present study, the degree of doubt in the accuracy of subject's recall was rated for 3 participants. Once the doubt was due to participant's difficulties remembering details about the life events (due to Asperger syndrome) and twice researchers failed to clarify the details with regard to particular life event. Therefore, the doubt was taken into account when finalising the ratings, rather than excluding these participants from the analysis.*

et al. 2006;Garety et al. 2001). Also, this is the only scale available that assesses the possible combination of negative self-evaluation (e.g. worthless, weak, unloved) along with a negative view of the others (devious, hostile, untrustworthy). As such it captures the individual's appraisals of threat and his/her own vulnerability (going beyond the concept of low self-esteem as measured by other questionnaires (Barrowclough et al. 2003)) in the social context that has been linked to paranoia and psychotic episodes (Fowler et al. 2006). For the analysis, scores on negative beliefs about self and negative beliefs about others were used as continuous variables *(for a full description of the variables used in the analysis see 4.2.5)*. A copy of this measure is provided in *Appendix XI.*

3.4.7 Measuring affective state (depression): Hamilton Rating Depression Scale (HRSD) (Hamilton 1960)

The Hamilton Rating Scale for Depression (HRSD) is a 17-item rating scale rated by a trained researcher on either a 3-point (from 0 to 2) or 5-point (from 0 to 4) Likert scale resulting in total scores between 0 and 50. There are general guidelines provided for making each of the item ratings. Inter-rater reliabilities have been shown to be consistently high with correlations ranging from *0.84 to 0.95* for the total score and internal consistency *0.80* (Steer et al. 1987). Similar results were found in a comprehensive meta-analytic (Trajkovic et al. 2011) review of the reliability of the Hamilton Rating Scale for Depression for the period 1960–2008. Results suggest strong levels of internal consistency, inter-rater and test-retest reliability (mean ranging between *0.87* and *0.94*), however some of the items were observed not to have a satisfactory reliability (e.g. test–retest reliability for items 'loss of weight', 'agitation', 'loss of insight', 'retardation' had pooled means of *0.28, 0.28, 0.41* and *0.49* respectively; weakest inter-rater reliability for 'loss of insight' at *0.27*) (Trajkovic et al. 2011). Two additional items (depersonalisation and obsessive–compulsive

symptoms) were included in this measure to help clinicians/researchers determine the typology of depression but are not calculated in the total HRSD score (Cole et al. 2004). For the analysis, depression score was used in its original form (as a continuous score) *(for a full description of the variables used in the analysis see 4.2.5)*. A copy of this measure is provided in *Appendix XII*.

3.4.8 Measuring genetic/family psychiatric history: Family Interview for Genetic Studies (FIGS) (Maxwell 1996)

The Family Interview for Genetic Studies (FIGS) was developed by main investigators in the NIMH Schizophrenia and Bipolar Disorder Genetics Initiatives (Maxwell 1996). It is a guide for systematically collecting information about relatives in the pedigrees being studied (Maxwell 1996). The FIGS administration proceeds in three steps:

i) A pedigree is drawn including first- and second-degree relatives: the proband's parents, grandparents, siblings, half-siblings (including both of their parents), children and partner. The information gathered includes the date of birth, date of death and its cause (if applicable/known).

ii) General screening questions are asked in reference to all known first degree relatives (parents, siblings, and children) and second degree relatives (half-siblings only). A positive response to any of the questions is followed by additional probes (part iii).

iii) Based on the informant's positive responses to the general screening questions, specific checklists for corresponding symptoms (depression, mania, psychosis, OCD, autism) are completed for each first-degree relative (or half-sibling). For all positive scoring first-degree relatives (or half-siblings) the full face sheet is

completed (based on positive responses to the general screening questions for that particular symptomatology).

Internal consistency of the FIGS as measured by Cronbach's alpha coefficient has been found to be *0.92* for depression, *0.99* for mania; and *0.94* for psychosis (de Villalvilla et al. 2008).

For the analysis, the presence *'1'* or absence *'0'* of familial risk of psychosis included at least one of the first degree relatives (or half-siblings) with a current or previous psychotic episode(s). In addition, the presence *'1'* or absence *'0'* of familial risk for neuropsychiatric disorders was defined as reports of any current or past *psychosis, depression, mania, obsessive-compulsive disorder (OCD) or autism* in a first degree relative (or half-sibling). This broader inclusion of familial risk to neuropsychiatric disorders is supported by studies demonstrating a genetic overlap of schizophrenia with mood disorders, autism (Cross-Disorder Group of the Psychiatric Genomics Consortium 2013) and OCD (Poyurovski et al. 2005) (*for a full description of the categories/variables used in the analysis see section 4.2.3.2).* A copy of the FIGS used in this study is provided in *Appendix XIII*.

3.4.9   Measuring cannabis experiences: Cannabis Experiences Questionnaire (CEQ) (Barkus et al. 2006)

The Cannabis Experiences Questionnaire (CEQ) has been used in several previous studies assessing the association between cannabis and psychosis/psychotic-like experiences (Barkus and Lewis 2008;Di Forti et al. 2009) or schizotypy (Stirling et al. 2008). It is a self-report questionnaire containing 17 items.

For the purpose of this thesis, the following questions of the CEQ were selected for the analysis:

- Have you ever smoked/used cannabis? (Yes/No), How old were you if when you first tried cannabis?, Do you currently use cannabis? (Yes/No)

- How often did/do you use cannabis (highest frequency lifetime)?

  (i) every day, (ii) (more than) once per week, (iii) a few times each month, (iv) a few times each year, (v) only once or twice.

- What type of cannabis do/did you mostly use?

  (i) Hash (cannabis resin/solid), (ii) Imported herbal cannabis, (iii) Home-grown skunk/Sensimilla, (iv) Super skunk, (v) Other (specify).

Subjects were also screened for cannabis dependency – considered positive if they experienced three or more of the following experiences (e.g. 'markedly diminished effect with continued use of the same amount of the substance', 'the characteristic withdrawal syndrome for the substance', 'there is a persistent desire or unsuccessful effort to cut down or control substance use') *(for a full dependence screening list see Appendix XIV).* For ease of analysis, lifetime cannabis use was dichotomised: *'never used cannabis (0)'* and *'have used cannabis (1)'*, regardless of the frequency of use. Frequency of cannabis use was collapsed into two categories: '*no cannabis use', 'use a few times each month*, '*a few times each year'* and '*only used it once/twice'* all collapsed into one category *(0)* and '*everyday use'* or '*(more than) once a week'* into another (1) (*for a full description of the categories/variables used in the analysis see section 4.2.4 – cannabis as a moderator, and 4.2.5 – cannabis as a mediator of trauma-schizotypy association).* A copy of the full measure is provided in *Appendix XIV.*

CHAPTER 4

Recruited sample and Data management

Chapter 4 Recruited Sample and Data management

Main aims of the chapter

- To present the socio-demographic characteristics of the recruited sample *(section 4.1)*;

- To briefly re-capture the measures along with the variables extracted to explore each of the hypotheses *(from section 4.2.1 to 4.2.5)*;

- To describe a detailed analysis procedure along with specific independent and dependent variables (as well as moderators, mediators and con-founders where applicable) for each of the hypotheses *(from section 4.2.1 to 4.2.5)*.

4.1   *Recruited sample*

Using the recruitment procedure described earlier *(see section 3.3)*, the following sample was obtained (recruited between May 2011 and May 2013) *(Table 7):*

Table 7: Ethnic groups (and gender) of the total sample (comparison with ethnic distribution in Lambeth and Southwark Boroughs, Census 2011) (N=256)

Ethnic group	Males N (%)	Females N (%)	Total Sample N (%)	Lambeth* Borough	Southwark** Borough
White - British	52 (46.8)	59 (40.7)	111 (30.5)	39.0%	39.7%
White - Other	11 (9.9)	19 (13.1)	30 (14.4)	18.1%	14.6%
Black Caribbean	17 (15.3)	21 (14.5)	38 (17.6)	9.5%	6.2%
Black African	14 (12.6)	28 (19.3)	42 (16.4)	11.6%	16.4%
Asian	6 (5.4)	5 (3.4)	11 (3.9)	6.8%	9.5%
Other	11 (9.9)	13 (9.0)	24 (17.2)	15.0%	13.6%
Total N (%)	111 (100)	145 (100)	256 (100)	100%	100%

* comparison with the study's total sample, not sign. Chi-Square, p=0.298   ** comparison with the study's total sample, not sign. Chi-Square, p=0.085

Table 7 above demonstrates that there were more female than male participants in the study. The largest ethnic group for both genders was White-British, followed by Black Caribbean and Black African groups. There was no statistically significant difference in terms of ethnic distribution between genders *(Pearson Chi-Square x^2=3.47, df=5, p=0.629).*

For the purpose of this thesis the first 212 participants with completed second appointments were included (and therefore had completed the SIS-R). The characteristics of this subsample are presented in *Table 8*. Out of the total sample recruited (N=256), two participants (0.8%) were excluded due to a present/past psychotic episode, twenty-two (8.6%) withdrew from the study/or could not be contacted after the first appointment and twenty participants still remained to be seen for the second appointment at the point when the data for this thesis were analysed (*see Figure 9 in section 3.3 for the full Recruitment process*).

Table 8: Gender and ethnic distribution of the thesis sample (N=212)

	Males* N (%)	Females* N (%)	Total N (%)
Ethnicity			
White British	47 (50.0)	53 (44.9)	**100 (47.2)**
White Other	8 (8.5)	18 (15.3)	**26 (12.3)**
Black Caribbean	14 (14.9)	14 (11.9)	**28 (13.2)**
Black African	12 (12.8)	20 (16.9)	**32 (15.1)**
Asian	3 (3.2)	5 (4.2)	**8 (3.8)**
Other	10 (10.6)	8 (6.8)	**18 (8.5)**
Total	**94 (100)**	**118 (100)**	**212 (100)**

* Chi-Square X^2=4.27, df=5, p=0.512

The Table above *(Table 8)* shows that the thesis subsample included more female than male participants. For both genders, the largest ethnic group was White-British, followed by Black African and White Other for females, and Black Caribbean and Black African for males. There was no statistically significant difference in relation to the ethnic distribution between genders for the thesis subsample *(Pearson Chi-Square x^2=4.27, df=5, p=0.512)*.

Also, there was no significant difference between those who completed SIS-R measures (N=212) and those who did not (N=44) on age *(t-test t=0.558, df=251, p=0.577)* or gender *(Pearson Chi-Square x^2=0.65, df=1, p=0.419)*. However a difference in ethnicity was approaching significance *(Fisher's Exact Test x^2=9.78, df=5, p=0.052) (see Table 9)* and there was a significant difference in source of recruitment *(Fisher's Exact Test x^2=50.02, df=3, p<0.001) (see Table 10)* between these two groups. The difference in ethnicity however is not attributed to higher 'drop-out/withdrawal' rates in Black Caribbean or Black African groups, but rather reflects the fact that White groups were much easier to recruit with the methods that were utilised *(and 'exclusion' was due to the not yet completed second set of*

assessments – 16 participants among Black Caribbean and Black African at the time of the writing).

Table 9: Ethnicity comparison of those with completed SIS-R ('included') and those without SIS-R ('excluded')

Ethnicity	Included* N (%)	Excluded* N (%)	Total N (%)
White - British	100 (47.2)	11 (25.0)	111 (43.3)
White - Other	26 (12.3)	4 (9.1)	30 (11.7)
Black Caribbean	28 (13.2)	10 (22.7)	38 (14.8)
Black African	32 (15.1)	10 (22.7)	42 (16.4)
Asian	8 (3.8)	3 (6.8)	11 (4.3)
Other	18 (8.5)	6 (13.6)	24 (9.4)
Total	212 (100)	44 (100)	256 (100)

*Fisher's Exact Test X^2=9.78, p=0.052

Table 10: Source of recruitment comparison of those with completed SIS-R ('included') and those without SIS-R ('excluded')

Ethnicity	Included* N (%)	Excluded* N (%)	Total N (%)
PAF	15 (7.1)	7 (15.9)	22 (8.6)
GP	111 (52.3)	14 (31.8)	125 (48.8)
SELCoH	83 (39.1)	11 (25.0)	94 (36.7)
Other	3 (1.4)	12 (27.3)	15 (5.8)
Total	212 (100)	44 (100)	256 (100)

*Fisher's Exact Test X^2=50.02, p<0.001. PAF, Postcode Address File. GP, General Practitioner. SELCoH, South East London Community Health Service

4.1.1 Socio-demographic characteristics of the final sample

For ease of analysis, some of the socio-demographic variables were collapsed into fewer categories. Ethnic groups *(for a full list of categories see Chapter 3 - Methodology)* were combined into 6 main ethnic groups: White British *(1)*, White Other (including White Irish and White Other) *(2)*, Black Caribbean *(3)*, Black African *(4)*, Asian (including Indian, Pakistani, Bangladeshi, Chinese and Other Asian) *(5)* and Other *(6)*. Religious groups were kept as they were originally collected: none *(0)*, Christian *(1)*, Jewish *(2)*, Muslim *(3)* and Other *(4)*. All types of Christianity were gathered under the same category (e.g. Catholicism, Church of

England, Protestantism etc.). With no participants filling the 'Jewish' category, this category was dropped. Original 'employment status' categories were also collapsed into the following: unemployed *(1)*, economically inactive (i.e. house person, physical illness/disability, carer, retired) *(2)*, student *(3)*, and employed (which combined part-time employed, full-time employed and self-employed categories) *(4)*. Age was kept as a continuous variable.

The final sample *(see Table 11)* used for this thesis consists of 94 *(44.3%)* males and 118 *(55.7%)* females, with a mean age of 35 (ranging between 18 and 64). Ethnic distribution was similar for both genders *(x^2=0.061, p=0.804),* nearly half of the sample comprised White British individuals *(47.2%)* and *28.3%* fell in the Black Caribbean and Black African categories. With *59.4%* of participants economically active and *8.5%* unemployed, the sample characteristics are in line with the Census report from the relevant boroughs (Office for National Statistics 2001).

Table 11: Complete socio-demographic characteristics of the thesis sample (N=212) (For socio-demographics of London Southwark and Lambeth populations comparing to Inner London see Table Suppl.2, Appendix XV)

	Males N (%) or M (SD)	Females N (%) or M (SD)	Total N (%) or M (SD)
Gender	94 (44.3)	118 (55.7)	**212 (100)**
Age (mean, SD)	36.0 (13.1)	34.8 (12.8)	**35.3 (12.93)**
Ethnicity			
White British	47 (50.0)	53 (44.9)	**100 (47.2)**
White Other	8 (8.5)	18 (15.3)	**26 (12.3)**
Black Caribbean	14 (14.9)	14 (11.9)	**28 (13.2)**
Black African	12 (12.8)	20 (16.9)	**32 (15.1)**
Asian	3 (3.2)	5 (4.2)	**8 (3.8)**
Other	10 (10.6)	8 (6.8)	**18 (8.5)**
Total	**94 (100)**	**118 (100)**	**212 (100)**
Employment status (1 year prior to the interview)			
Unemployed	7 (38.9)	11 (61.1)	18 (8.5)
Economically inactive (retired, carer etc.)	7 (38.9)	11 (61.1)	18 (8.5)
Student	7 (28.0)	18 (72.0)	25 (11.8)
Part-time employed	8 (32.0)	17 (68.0)	25 (11.8)
Full-time employed	58 (51.8)	54 (48.2)	112 (52.8)
Self-employed	7 (50.0)	7 (50.0)	14 (6.6)
IQ score (mean, SD) (N=204)	108.0 (21.9) (min 56, max 151)	102.6 (17.6) (min 69, max 140)	**104.9 (19.8) (min 56, max 151)**
Religion			
None	45 (46.9)	51 (53.1)	**96 (45.3)**
Christian	39 (40.6)	57 (59.4)	**96 (45.3)**
Muslim	4 (44.4)	5 (55.6)	**9 (4.2)**
Other	6 (54.5)	5 (45.5)	**11 (5.2)**
Total	**94 (100)**	**118 (100)**	**212 (100)**

SD, Standard Deviation. M, Mean. IQ, Intelligence Quotient

4.2   *Data analyses*

4.2.1 Childhood trauma and schizotypy association

4.2.1.1 Outline of measures and variables used for the analysis

To test the main thesis hypothesis (Hypothesis 1, *'Those reporting more childhood trauma will score higher on a schizotypy scale')*, the following data analysis procedure was applied *(Figure 10).*

Measures used	Predictor variable(s)	Outcome variable(s)	Variables controlled for
• SIS-R • CECA & Bullying Quest. • CAPE • Socio-demographics (gender, age, ethnicity, employment status, religion) and IQ	• CECA & Bullying - total trauma score (dichotomised) and dichotomised scores (above severity 2) for psychological, physical, sexual abuse, bullying and household discord • Parental death and separation (dichotomised)	• SIS-R total score (continuous) • SIS-R total score (top 20% and 10% - binary) • SIS-R subscales' scores (positive, negative/ disorganised) • CAPE total • CAPE subscales' scores (positive, negative, depressive)	• Moderator: gender • Adjusted for gender, age, ethnicity, employment status, religion, IQ

Types of analyses

Main:
- **linear regression** for total trauma and SIS-R total as continuous outcome (and each subscale separately)
- **logistic regression** for total trauma and SIS-R total as binary outcome (and each subscale separately)

Subsidiary:
- same as *Main* repeated for CAPE
- descriptive statistics (mean SIS-R & mean CAPE scores; trauma frequencies)
- SIS-R & CAPE scores across gender (t-tests) and ethnicity groups (two-way ANOVA)
- effect of gender on trauma-schizotypy association (linear regression with the interaction term fitted)
- testing the correlation between SIS-R and CAPE (Spearman' rho test)

Figure 10: Overview of the measures used for analysis for Hypothesis 1, including the variables extracted from the measures and types of analyses used

4.2.1.2   Data analysis procedure

Creating variables

The CECA scoring manual (Lifespan Research Group 2009) was used to rate the severities and frequencies of all types of trauma measured. Separation from parents and parental death however only had yes/no responses followed by the age of the participant at the time of the occurrence. Bullying questionnaire scores were gained following the same CECA rating criteria (for severity and frequency), allowing for comparison of the effects between different types of trauma. For the analysis, CECA scores *(household discord, psychological abuse, physical abuse, sexual abuse as well as bullying)* have been dichotomised where *(0) was trauma absent*, and *(1) trauma was present*. Trauma was considered *'present'* (according to established guidelines) when the severity rating was *2 (moderate) or 3 (marked)*. Therefore, a four-point rating scale (or five-point scale for household discord) of all types of trauma measured was collapsed into two categories: *Absent (0)* and *Some (1)* formed the group *'trauma absent'* while *Moderate (2)* and *Marked (3)* were combined into *'trauma present'*. For household discord the 'trauma present' category also included the additional severity rating of violence *(4)*. Total trauma score is a numerical variable and equals the sum of all five types of traumas *(household discord, psychological abuse, physical abuse, sexual abuse and bullying)*. As each trauma type has received a score of *0 (Absent)* or *1 (Present)*, the maximum total trauma score was *5* (each trauma type could only be included once, regardless if one trauma type had more than one severity rating above 2).

For psychological and sexual abuse specifically, new variables were created that combined all levels of this types of abuse, as only *9 (4.3%)* of the subjects scored as moderate or marked severity on psychological trauma (which would be considered 'trauma present' using the aforementioned classification) and *15 (7.2%)* of the

subjects reported sexual abuse as moderate or marked severity. To allow more power for the analysis, these two new variables included all levels of severity *(including 'Some')* but kept the dichotomous form e.g. psychological abuse severity score *Absent (0)* was transformed to *'trauma absent' (0)* while the severity scores *Some (1)*, Moderate (2) and *Marked (3)* were combined into *'trauma present' (1)*. The same procedure was repeated for sexual abuse scores. Previous research has shown that for psychological abuse the inter-rater reliability for moderate and marked severity rating scores combined was *0.68 (weighted kappa)*, but when '*Marked/Moderate'* was combined with '*Some'* it increased to *0.80 (weighted kappa)* (Moran et al. 2002), which also ties in with another study (Bifulco et al. 1994). The parental separation variable was also dichotomised as *0 (no separation)* and *1 (one or more separations longer than 6 months)* in the analysis repeated for both parents. Similarly, death of a parent was dichotomised as *0 (no death)* and *1 (death of one or both parents)*.

The total score on the main schizotypy measure equalled the sum of all 15 global scores (each item/global score ranged from *Absent (0)*, *Mild (1)*, *Moderate (2)* and *Severe (3))*. Therefore, the minimum SIS-R total score was *0* and the maximum was *45*. The positive schizotypy dimension was obtained by summing the following items/global scores *(in total containing seven items)*: referential thinking *(part 1: being watched)*, referential thinking (part 2: being talked about), suspiciousness, magical ideation, illusions, psychotic symptoms and derealisation/depersonalisation. As such, the minimum positive dimension score was *0*, while the maximum was *21*. Negative schizotypy comprised these symptoms: *social isolation, introversion, hypersensitivity, restricted affect (in total four items),* and the disorganisation schizotypy dimension encompassed these signs: *goal directness of thinking, loosening of associations, poverty of speech and oddness (containing in total four items)*. As not enough participants scored positively on the disorganised schizotypy

items *(see Table 17, Chapter 5)* to allow for separate analysis of this dimension, the negative and disorganised dimension have been combined to form negative/disorganised schizotypy, with the total score ranging from *0* to a maximum of *24*. A similar method has been previously adopted (Myin-Germeys et al. 2011). *CAPE* total score equalled the sum of all the items from this scale, ranging from a minimum of *42* points to a maximum of *168* (42 items on a 5-point Likert scale ranging from *Never (1)*, *Sometimes (2)*, *Often (3)* to *Nearly always (4)*). The total score for each of the three dimensions was also obtained: positive dimension *(20 items, min=20 to max=80)*, negative dimension *(14 items, min=14 to max=56)* and depressive dimension *(8 items, min=8 to max=32)*.

Descriptive statistics for SIS-R & CAPE mean scores and scores across gender and ethnicity groups

Descriptive statistics were used to compare the mean and standard deviation for schizotypy scores (for *Total schizotypy* score and *Positive* and *Negative/Disorganised* dimension separately) among gender and ethnicity groups *(see Tables 16 & 18, Chapter 5)*. Similarly, *CAPE* total score and subscores *(positive, negative and depressive dimension)* were described using mean scores and standard deviations among gender and ethnic categories. Schizotypal global scores were also presented individually using the frequency and percentage for both genders. Global scores were collapsed into two categories *'Absent/Mild'* (combining scores of '0' and '1') and *'Moderate/Severe'* (combining scores of '2' and '3') for the clarity of presentation.

Following the previous research findings that schizotypal traits (or sub-dimensions) vary across gender and ethnic groups (*see section 1.1.4 Socio-demographic characteristics and schizotypy*)*, Pearson Chi-square* was used to test for significant differences between genders on each of the schizotypy global

score/item. In cases, where frequencies in any of the categories were less than 5, *Fisher's Exact tests* were run instead *(see Table 17, Chapter 5)*. To check for any significant differences between the schizotypy total mean score (and means on both sub-dimensions) between gender *t-tests* were used. That was repeated for all *CAPE* mean scores. *Two-way ANOVA* was utilised to compare the mean scores between different ethnic categories *(for schizotypy scores and CAPE scores)* followed by the *Bonferroni Post Hoc test (see Table 18, Chapter 5)*.

Testing the main hypothesis

To test the main hypothesis, a linear regression was used *(treating the outcome measure/schizotypy as a continuous variable)* to analyse the relationship between *Childhood Trauma (for Total score and each of the trauma types independently)* and *Schizotypy Total (see Table 19, section 5.1)*. Similarly, linear regression was completed separately for *Positive schizotypy* and *Negative/Disorganised schizotypy* dimension*,* to explore the potentially different effects of trauma types on specific schizotypy dimensions. These analyses were repeated to adjust for potential confounders of these relationships *(gender, age, ethnicity, employment, religion and IQ)*, as identified by the literature review. The same procedure was followed for the exploration of the association between '*death of a parent'/'separation from a parent'* and schizotypy score. Before running linear regressions, four principal assumptions had to be tested that justify the use of linear regression: *(i) Linearity of the relationship, (ii) Independence of the errors (no serial correlation), (iii) Homoscedasticity - rvfplot (constant variance of errors) and (iv) Normality of the error distribution.* Linearity was evident in a *Plot of residuals versus predicted values* (with the points symmetrically distributed around the diagonal line). Normality was checked with *Q-Q plot (quantile-quantile plot).*

As a part of the sensitivity analysis, cut-off points rather than total schizotypy (as a continuous score) were used. The reasoning behind the inclusion of schizotypy as a continuous score and cut-off points allows the exploration of schizotypy from different perspectives *(as presented in section 1.1.5 Schizotypy and the development of psychosis/ The continuum model).* Also, both forms (continuous and dichotomous scores) have been previously used in the literature for schizotypy (as measured by SIS-R) and attenuated psychotic symptoms (as measured by CAPE) (e.g. Alemany et al. 2012;Myin-Germeys et al. 2011;Wigman et al. 2012b). Basing the decision on the distribution of the schizotypy scores, cut off points for approx. *20%* and *10%* of the sample *(exact calculations made using STATA)* were used. To test whether trauma predicted top 20% and top 10% of schizotypy scorers *(including positive and negative/disorganised scores separately)*, *Logistic Regression tests* were carried out, testing for trauma types individually*.* After testing for assumptions of logistic regression, *Hosmer-Lemeshow goodness-of-fit* test was used, which was not significant, meaning the model used was appropriate. As some of the trauma categories did not have enough participants, they were omitted from the table *(see Tables 22 & 23, section 5.1).* The same analyses were repeated when also adjusted for just a few confounders only *(gender, age and ethnicity)*, following the rule that the number of variables included multiplied by *10* cannot exceed the number of the outcome. Also, the association between childhood trauma and psychotic-like experiences (using *CAPE* measure) was evaluated using linear regression and repeated for the Positive and Negative dimensions (to allow the comparison with SIS-R positive and SIS-R negative dimensions) *(see Tables 28 & 29, section 5.1).*

The effects of age at trauma occurrence, gender and testing the dose-response relationship

Also, exploration of the associations between age of trauma occurrence *(defined as 'before the age of 12', and '12 or after') (also see Fisher et al. 2010)* and gender, and *total, positive* and *negative/disorganised* schizotypy were competed using linear regression (with the interaction term fitted – for gender).

To check for the dose-response relationship between trauma severity and frequency and schizotypal symptomatology (as previously evidenced in the literature review), the following new categories were created for each of the trauma types *(household discord, psychological abuse, physical abuse, sexual abuse and bullying)*: 'low severity & low frequency' (1), 'low severity & high frequency' (2), 'high severity & low frequency' (3) and 'high severity & high frequency' (4). The ratings were made using the highest severity score and its associated frequency. The low/high division on severity mirrored *'trauma absent/trauma present'* categories, with any severity above *2 (moderate)* falling under 'high' classification. For the frequency, categories 'never' (0), 'rarely (once or twice)' (1) and 'occasionally (more than twice, less than monthly)' (2) formed 'Low frequency' while 'frequent' (3) and 'very frequent' (4) was collapsed into 'High frequency'. Similarly, the effect of multi-victimisation was evaluated by combining the *high severity* & *high frequency* category from each type of trauma forming the following categories: 'no trauma' (0), '1 type of trauma' (1), '2 or more types of trauma' (2).

Testing the correlation between SIS-R and CAPE

To test the correlation between the positive dimension on *SIS-R* and the positive dimension on *CAPE*, as well as between the negative dimension on *SIS-R* and negative *CAPE* dimension, *Spearman's rho test* was used. Spearman's rho test is a

weaker test than *Pearson product-moment correlation test*, however it was selected as CAPE positive and negative dimensions were not normally distributed *(not meeting the requirements of the Pearson test)*. The positive correlation between the subscales of the instruments would 'permit' to only focus on SIS-R scores to test the rest of the hypotheses (all secondary hypotheses, *see section 3.1.2*).

All data was initially entered into *SPSS 17 (SPSS Inc. 2008)*, and analysed using *STATA 11 (StataCorp. 2009)*.

Following the support obtained for the main hypothesis - *the significant association between childhood trauma and total schizotypy* - the pathways underlying this association were explored by testing the secondary hypotheses (analyses presented in the rest of this chapter). For the rationale behind the inclusion of recent life events, negative beliefs about others/self, depression and cannabis use see thesis section *2.3. (Possible pathways underlying childhood trauma – schizotypy association)*; for the role of genetic risk for psychosis on childhood trauma-schizotypy association refer to thesis section *1.2 (Heritability of schizotypy)*.

4.2.2 Childhood trauma and life events interaction and schizotypy

4.2.2.1 Outline of measures and variables used for the analysis

To test the thesis Hypothesis 2 (*'Schizotypy will be higher in those exposed to both childhood and adulthood traumatic experiences than in those with childhood trauma only'*), the following data analysis procedure was applied *(Figure 11)*.

Measures used	Predictor variable(s)	Outcome variable(s)	Moderator & variables controlled for
• SIS-R • CECA & Bullying Quest. • LEDS • Socio-demographics (gender, age, ethnicity) and IQ	• CECA & Bullying - total trauma score • LEDS (categories: 0 events, 1 event, 2 events, 3 or more events), also events and difficulties combined scores • independent/ intrusive events/difficulties – dichotomised	• SIS-R total score (continuous) • SIS-R subscales' scores (positive, negative/ disorganised)	• Moderator: adulthood trauma (recent life events) • Adjusted for gender, age, ethnicity (2 groups), IQ

Type of analysis

- **linear regression with the interaction term fitted** was used to test the interaction effects between childhood trauma and life events on schizotypy total score
- re-run the analysis for independent and intrusive events only

Figure 11: Overview of the measures used for analysis for Hypothesis 2, including the variables extracted from the measures and type of analysis used

4.2.2.2   Data analysis procedure

Creating variables

Total life events score is a sum of all life events with a score of *2 (Moderate threat/unpleasantness) or 1 (Marked threat/unpleasantness)* for either short-term or long-term threat. By looking at the frequencies of life events *(ranging from 0 to 6)* the scores were grouped into four ordinal values: *'0 events'*, '*1 event'*, *'2 events'*, *'3 or more events'*). The same categories were used for division of total independent events (a sum of all independent events above the score of *2*, either short-term or long-term threat). Total intrusive events scores *(ranged between 0 and 2, with only 3 subjects reporting two intrusive events above the severity of 2)* were then dichotomised (*'no intrusive events' (0)*, '*1 or 2 intrusive events' (1)*). The total number of events and difficulties included all events with the severity of *2* or more, and all difficulties with a severity of *3* or more (including *3 – 'High moderate'*, *2 – 'Low marked'* or 1- *'High marked'*). The scores ranged between *0* and maximum *7*,

with only *6.7%* of subjects reporting *4* or more events/difficulties. To be consistent with previous variables, four groups were created (*'0 events/difficulties'*, *'1 event/difficulty'*, *'2 events/difficulties'*, *'3 or more events/difficulties'*), these were also used for the total scores of *independent* events/difficulties. The total scores of *intrusive* events/difficulties were dichotomised. Schizotypy total was used as a continuous variable, gained by summing up all *15* global schizotypy scores.

Testing Hypothesis 2:

Linear regression (unadjusted and adjusted) was used to measure the main effects of events and difficulties *(and separately for independent events/difficulties and intrusive events/difficulties)* on schizotypal symptomatology (total score).

To assess the interaction effects between childhood trauma and life events on schizotypy total score *(Hypothesis 2)*, both variables were dichotomised and *linear regression* with the interaction term fitted was used. Total trauma scores (a sum of all types of trauma above the severity of *2*: household discord, psychological abuse, physical abuse, sexual abuse and bullying) were collapsed into two categories: *'Trauma present'* (for any scores of 1 or above) and *'Trauma absent'* (scores of 0). Even though the strongest associations were observed between physical and psychological abuse and schizotypy *(see results section for Hypothesis 1)*, the trauma scores were combined and total trauma scores were used in order to increase the statistical power. Also, life events/difficulties total scores *(including independent and intrusive events/difficulties)* were transformed into *'Life events/difficulties present'* (one or more life events or difficulties) and *'No life events/difficulties'* (a score of 0). These analyses were repeated adjusting for gender, age, ethnicity *(two categories only: White/Non-white)* and IQ.

4.2.3   Childhood trauma and familial risk interaction and schizotypy

4.2.3.1   Outline of measures and variables used for the analysis

To test the thesis Hypothesis 4 (*'Individuals with higher familial risk for psychosis and exposure to childhood trauma will display higher schizotypy scores than those without familial risk')*, the following data analysis procedure was applied *(Figure 12).*

Measures used	Predictor variable(s)	Outcome variable(s)	Moderator & variables controlled for
• SIS-R • CECA & Bullying Quest. • FIGS • Socio-demographics (gender, age, ethnicity) and IQ	• CECA & Bullying - total trauma score • Familial risk for psychosis - dichotomised (absence/ presence)	• SIS-R total score (continuous) • SIS-R subscales' scores (positive, negative/ disorganised)	• Familial risk for psychosis • Adjusted for gender, age, ethnicity (2 groups), IQ

Type of analysis

• **linear regression (and interaction term fitted for moderation)** to test the interaction effects between childhood trauma and familial risk for psychosis on schizotypy total score

Figure 12: Overview of the measures used for analysis for Hypothesis 4, including the variables extracted from the measures and type of analysis used

4.2.3.2　　Data analysis procedure

Creating variables

Presence *'1'* or absence *'0'* of familial risk for psychosis included at least one of the first degree relative with a current or previous psychotic episode(s). Looking more broadly, presence *'1'* or absence *'0'* of familial risk for neuropsychiatric disorders was further defined including any current or past *psychosis, depression, mania, obsessive-compulsive disorder (OCD) or autism* again reported for the first degree relatives. Only *6 (2.9%)* of biological parents were reported to have had a psychotic episode, therefore parental illness only was not used in a separate analysis (but combined with any first degree relative).

Testing Hypothesis 4

The main effect of familial risk *(narrow and broad definition separately)* on schizotypy was measured using linear regression.

An interaction term was then added to the linear regression to measure the interaction effects between dichotomised childhood trauma scores and familial risk for psychosis on total schizotypy *(Hypothesis 4)*. The analyses were repeated, controlling for gender, age, ethnicity *(two categories only)* and IQ. As differential effects of trauma types have been previously documented, further analyses were carried out to check for interaction effects with any specific trauma types and familial risks (dichotomised 'present'/'absent' score) using a broad definition only (due to limited numbers of relatives with psychosis only). All levels of severity (some/moderate/marked) were used for psychological and sexual abuse.

4.2.4   Childhood trauma and schizotypy and the effects of cannabis

4.2.4.1   Outline of measures and variables used for the analysis

To test the thesis Hypothesis 5 *('Cannabis use will partially account for the association between childhood adversity and schizotypy; cannabis will either mediate the childhood trauma – schizotypy association or interact with childhood trauma to increase the schizotypy levels')*, a mediation analysis was firstly used *(see section 4.2.5)*. As the analysis showed that cannabis use did not mediate childhood trauma – schizotypy association, the following analysis was completed to test if cannabis was a moderator of this association. The procedure is depicted in *Figure 13*.

Measures used	Predictor variable(s)	Outcome variable(s)	Moderator & variables controlled for
• SIS-R • CECA & Bullying Quest. • Cannabis Experiences Questionnaire • Socio-demographics (gender, age, ethnicity) and IQ	• CECA & Bullying - total trauma score • Cannabis use: dichotomised current and present use • Age of first use, type of cannabis, frequency of use	• SIS-R total score (continuous) • SIS-R subscales' scores (positive, negative/ disorganised)	• Cannabis use • Adjusted for gender, age, ethnicity (2 groups), IQ

Type of analysis

• **linear regression (and interaction term fitted for moderation)** to test the interaction effects between childhood trauma and cannabis use on schizotypy total score

Figure 13: Overview of the measures used for analysis for Hypothesis 5, including the variables extracted from the measures and type of analysis used

4.2.4.2　　Data analysis procedure

Creating variables

For ease of analysis, lifetime cannabis use was dichotomised: *'never used cannabis (0)'* and *'have used cannabis (1)'*, regardless of the frequency of use. Likewise, current use of cannabis was used as a dichotomised score. The age when the subject started using cannabis was split into two categories: *'under 17'* and *'17 and above' (also see Di Forti et al. 2009)*. Using these categories the main effects on schizotypy between the groups were compared, including the possible interaction effects between those who started using cannabis at a younger age (before 17) who also reported childhood trauma. Frequency of cannabis use was collapsed into two categories: '*no cannabis use'*, use '*a few times each month'*, '*a few times each year'* and '*only used it once/twice'* all collapsed into one category *(0)* and '*everyday use'* or *'(more than) once a week' into another (1)*. Type of cannabis was included using the following categories: *'hash' (1), 'imported herbal cannabis' (2), 'home-grown skunk'* or *'super-skunk' (3)* and 'other' (4).

Analysis

Linear regression was run to check the association between lifetime or current cannabis use and schizotypy total score, as well as positive and negative schizotypal dimensions. Interaction effects between childhood trauma (dichotomised score) and cannabis use on schizotypy dimensions was evaluated using linear regression and cannabis use as a possible moderator.

4.2.5   Possible underlying mechanisms supporting childhood trauma – schizotypy association / mediation effects

4.2.5.1   Outline of measures and variables used for the analysis

To test the thesis Hypothesis 3 *('Childhood trauma will lead to development of negative beliefs about self/others and depression which will then increase the schizotypy levels')* and Hypothesis 5 *('Cannabis use will partially account for the association between childhood adversity and schizotypy; cannabis will mediate childhood trauma – schizotypy association')*, a mediation analysis was utilised. In addition to negative beliefs about self/others, depression score and cannabis use, a mediation effect between childhood trauma and schizotypy was also tested (as an exploratory analysis) for recent life events. The procedure is depicted in *Figure 14.*

Measures used	**Predictor variable(s)**	**Outcome variable(s)**	**Moderator & variables controlled for**
• SIS-R • CECA & Bullying Quest. • LEDS • Hamilton's Scale • Brief Core Schema Scale • Cannabis Experiences Questionnaire • Socio-demographics (gender, age, ethnicity) • Genetic risk	• CECA & Bullying - total trauma score • Negative belief about self/others – continuous • Depression score – continuous • Life events – numerical value • Cannabis use (dichotomised)	• SIS-R (binary, top 20% of scorers vs others)	• Cannabis use • Life events • Negative beliefs about self/others • Depression score • Adjusted for socio-demographics (gender, age, ethnicity) and genetic risk for psychosis

Type of analysis

• **KHB tests for mediation** *(for description of the method see 'Analysis' – 4.2.5.2)*

Figure 14: Overview of the measures used for exploratory mediation analyses (Hypotheses 3 & 5), including the variables extracted from the measures and type of analysis used

4.2.5.2   Data analysis procedure

Creating variables

Binary schizotypy score *(top 20% of scorers vs others)* was used as an outcome measure. *'Negative beliefs about others and self'* as well as *'depression score'* were used in their original form (as continuous scores). Also, recent life events were included as a numerical variable, while cannabis use was defined as a dichotomous variable *(yes/no)* for 'lifetime use' of any frequency.

Analysis

KHB (Karlson/Holm/Breen, Karlson et al. 2011) analyses were used to measure the mediation effects between childhood trauma and schizotypy using the following mediators: depression score, negative beliefs about others and negative beliefs about self, recent life events and cannabis use. The KHB technique was developed to examine the decomposition of effects in non-linear probability models (decomposing of the total effect of the variable into a *direct* and *indirect* of spurious part) (Breen et al. 2013;Karlson et al. 2011) *(Figure 15)*. The method compares the full model with a reduced model in which some Z-variables (mediator) are substituted by the residuals of the Z-variables from a regression of the Z-variables on the key variables (X) *(see Breen et al. 2013 for detailed calculations of the model)*. The method also allows for adjustment of confounding variables on the decomposition. In comparison to other decomposition methods, KHB gives unbiased estimates, allows the variable whose effect it decomposes to be discrete or continuous and provides an analytically derived set of readily interpretable statistical tests for many models of the GLM *(Generalized linear model)* framework (Karlson and Holm 2011).
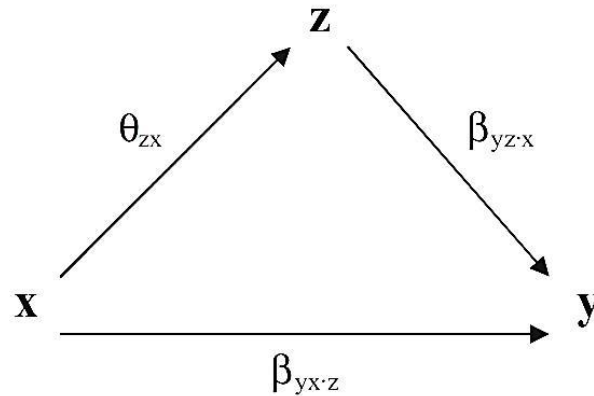
Figure 15: Path decomposition into direct and indirect effects (x=predictor; y=outcome; z=mediator) (Breen et al. 2013 p.168)

Multiple mediation analysis was performed to examine the extent to which childhood trauma-schizotypy associations were mediated by depression score, negative beliefs about others/self, recent life events and cannabis use. The mediation variables were first entered altogether to examine the combined effect on the association between each type of childhood trauma and schizotypy (as a binary outcome). This was followed by examining the impact of each mediation variable on these associations on its own.

Before running these tests, all logistic regressions for total trauma and each of the trauma types had to be significant after adjusting for gender, age and ethnicity. For psychological and sexual abuse all three levels of severity were included *(mild, moderate and severe)*. Mediation analyses were repeated adjusting for gender, age, ethnicity and familial risk for psychosis *('narrow definition'/only first degree relatives with current or past psychotic episodes)*. To explore the possible gender differences in pathways between childhood trauma and schizotypy, the *KHB* analysis procedure was repeated for men and women separately, including the adjusted mediation analyses *(see Suppl. Table 15 & 16, Appendix XVI)*. As for males the *'total trauma'* score *(unadj. OR=1.64, p=0.031, adj. OR=1.36, p=0.213),* *'household discord'* *(unadj. OR=2.55, p=0.052, adj. OR=2.31, p=0.114), 'sexual*

abuse' (unadj. OR=1.30, p=0.723, adj. OR=1.21, p=0.807) and 'bullying' (unadj. OR=1.02, p=0.968, adj. OR=.84, p=0.742) were not significant predictors of top 20% schizotypy scorers these analyses were omitted. Psychological abuse showed a trend towards significance for schizotypy in men *(unadj. OR=3.58, p=0.075, adj. OR=4.54, p=0.060)*, therefore the mediation analyses were completed. Equally, associations for *'household discord' (unadj. OR=2.01, p=0.157, adj. OR=1.70, p=0.318)* and *'psychological abuse' (unadj. OR=4.60, p=0.035, adj. OR=3.65, p=0.121)* in females showed a reasonable association with schizotypy as a binary outcome but failed to reach statistical significance. Therefore, the mediation analyses were not completed for these types of trauma *(see Table Suppl.13, Appendix XVI).*

CHAPTER 5
Results

Chapter 5 Results

Main aims of the chapter

- Present descriptives of the main variables used in the analysis - report on frequencies of trauma types of the sample, including the mean schizotypy scores between gender and ethnicity groups;

- Present the results/findings that would help to disentangle the main questions set out with this thesis *(from section 5.1 to 5.5)*;

- Explore the possible pathways (meditators) of the childhood abuse-schizotypy association *(section 5.5)*.

This chapter will begin with descriptives of the main variables used in the analysis, followed by a presentation of results associated with each of the hypotheses individually.

Frequencies of reported traumatic experiences across gender and ethnicity

59.2% of participants reported at least one childhood traumatic event (*53.7%* when excluding low frequencies) before the age of 17, with the highest frequency reported for household discord *(35.8%)* and bullying *(32.2%)* and the lowest for psychological abuse *(4.3%) (see Table 12)*. Looking at the higher frequencies only, *34.3%* reported one type of trauma and *19.4%* two or more types *(Table 14)*. Some gender differences in types of trauma were observed, as females reported significantly more sexual abuse *(chi^2=6.26, p=0.012)* and males reported more bullying *(chi^2=5.22, p=0.022) (Table 12)*.

Table 12: Frequencies and gender comparison in reported total trauma and all distinct trauma types

	Total		Males		Females	
	Absent N (%)	*Present* N (%)	*Absent* N (% within gender)	*Present* N (% within gender)	*Absent* N (% within gender)	*Present* N (% within gender)
Total trauma (N=206)	84 (40.8)	*122 (59.2)*	37 (40.2)	*55 (59.8)*	47 (41.2)	*67 (58.8)*
Household discord (N=212)	136 (64.1)	*76 (35.8)*	66 (70.2)	*28 (29.8)*	70 (59.3)	*48 (40.7)*
Psychological abuse (N=211)	202 (95.7)	*9 (4.3)*	91 (96.8)	*3 (3.2)*	111 (94.9)	*6 (5.1)*
Physical abuse (N=211)	168 (79.6)	*43 (20.4)*	72 (76.6)	*22 (23.4)*	96 (82.0)	*21 (17.9)*
Sexual abuse (N=208)	193 (92.8)	*15 (7.2)*	90 (97.8)	*2[a] (2.2)*	103 (88.8)	*13[a] (11.2)*
Bullying (N=211)	143 (67.8)	*68 (32.2)*	56 (59.6)	*38[b] (40.4)*	87 (74.4)	*30[b] (25.6)*

[a] *significant gender difference Pearson chi^2(1)=6.26, p=0.012*
[b] *significant gender difference Pearson chi^2(1)=5.22, p=0.022*

Table 13: Frequencies and gender comparison of psychological and sexual trauma (including all levels of severity - some/moderate/marked) (see 4.2.1.2 for full information on the formation of the variables)

(any trauma severity some/moderate/marked)	Total		Males		Females	
	Absent N (%)	*Present* N (%)	*Absent* N (% within gender)	*Present* N (% within gender)	*Absent* N (% within gender)	*Present* N (% within gender)
Psychological abuse (N=211)	*193 (91.5)*	**18 (8.5)**	*85 (90.4)*	**9 (9.6)**	*108 (92.3)*	**9 (7.7)**
Sexual abuse (N=208)	*171 (82.2)*	**37 (17.8)**	*83 (90.2)*	**9 [a] (9.8)**	*88 (75.9)*	**28 [a] (24.1)**

[a] *significant gender difference Pearson chi²(1)=7.23 , p= 0.007*

Table 14: Frequencies for multi-victimisation and between gender comparison

High severity and high frequency only	Total N (%)	Males N (% within gender)	Females N (% within gender)
No abuse/trauma	**93 (46.3)**	*40 (44.0)*	*53 (48.2)*
1 type of trauma	**69 (34.3)**	*33 (36.3)*	*36 (32.7)*
2 or more types	**39 (19.4)**	*18 (19.8)*	*21 (19.1)*
Total	**201 (100)**	*91 (100)*	*110 (100)*

Also, the differences were observed for physical *(Fisher's Exact test p<0.001)* and sexual abuse *(Fisher's Exact test p=0.003)* among ethnicity groups, with the highest percentage of these types of trauma reported by the Black Caribbean group *(53.6% and 37.5%, respectively) (Table 15)*.

Table 15: Frequencies and comparison between ethnic groups in reported total trauma and all distinct trauma types (% within particular ethnic groups)

	White British Trauma present N (%)	*White Other* Trauma present N (%)	*Black Caribbean* Trauma present N (%)	*Black African* Trauma present N (%)	*Asian* Trauma present N (%)	*Other* Trauma present N (%)	*Ethnic differences*
Total trauma	*52 (52.6%)*	*14 (53.8%)*	*21 (77.8%)*	*19 (61.3%)*	*6 (60.0%)*	*10 (66.7%)*	*Pearson chi²=8.82, p= 0.101*
Household discord	*34 (34.0%)*	*8 (30.8%)*	*13 (46.4%)*	*12 (37.5%)*	*5 (50.0%)*	*4 (25.0%)*	*Fisher's Exact test chi²=3.05, p= 0.629*
Psychological abuse (all levels)	*6 (6.0%)*	*4 (15.4%)*	*4 (14.8%)*	*2 (6.2%)*	*1 (10.0%)*	*1 (6.2%)*	*Fisher's Exact test chi²=4.33, p= 0.431*
Physical abuse	*7 (7.0%)*	*4 (15.4%)*	*15 (53.6%)*	*12 (37.5%)*	*2 (20.0%)*	*3 (20.0%)*	***Fisher's Exact test chi²=36.41, p<0.001****

Sexual abuse (all levels)	9 (9.2%)	8 (30.8%)	10 (35.7%)	7 (22.6%)	0	3 (20.0%)	**Fisher's Exact test chi²=16.33, p= 0.003***
Bullying	30 (30.3%)	7 (26.9%)	12 (42.9%)	10 (31.2%)	2 (20.0%)	7 (43.7%)	*Fisher's Exact test chi²=2.52, p=0.626*

*** statistically** *significant at p<0.01*

9 (9.2%)	8 (30.8%)	10 (35.7%)	7 (22.6%)	0	3 (20.0%)	

Schizotypy (SIS-R measure) and attenuated psychotic experiences (CAPE measure) - total scores across gender and ethnicity

The total schizotypy scores ranged between *1* and *23*, with a mean of *9.2 (SD=4.6)*. For the positive schizotypy dimension the mean score was *5.0 (SD=3.1)*, and for the negative/disorganised dimension the mean score was *4.1 (SD=2.3)* with males scoring significantly higher than females *(t=2.305, p=0.022) (Table 16)*. SIS-R total scores were normally distributed, *(Figure 16)*, similar to previous reports using the *Schizotypal Personality Questionnaire (Johns and van Os 2001- data from Stefanis et al 2000)*. The frequencies for each of the schizotypal traits are presented in *Table 17*.
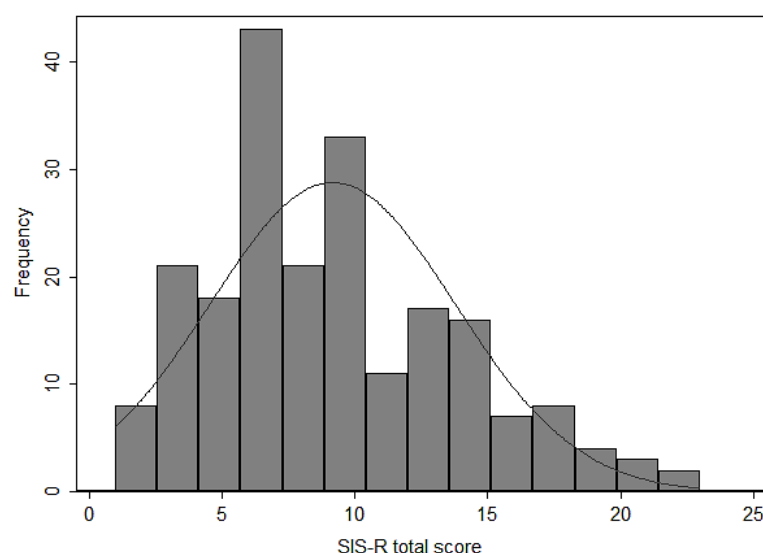
Figure 16: Frequencies of schizotypy total scores (SIS-R) - normal distribution of SIS-R total scores

The *CAPE* total score (attenuated psychotic experiences) ranged between *43* and *103*, with a mean score of *58.9*. There were no gender differences for either *CAPE total*, *CAPE positive* dimension *(mean score 25.1)* or *negative* dimension *(mean score 20.9)*. However, females reported more depressive symptomatology *(mean score for females 13.3, SD=3.44)* compared to men *(mean score for males 12.4,*

SD=2.43, t=-2.139, p=0.034) (Table 16). The frequencies for each of the CAPE items are presented in *Table Suppl.4, Appendix XVI*.

Similarly, differences in schizotypal scores and attenuated psychotic-like experiences were observed between ethnic groups *(Table 18)*. *Black Caribbean* and *Black African* groups scored significantly higher on schizotypal symptomatology (means of *11.8* and *11.0,* respectively) compared to the *White British* group *(mean=7.6, F=5.742, p<0.001)*. Similar results were observed for the positive schizotypy dimension, specifically *(Black Caribbean=6.4*, *Black African=6.3, White British=4.0, F=5.299, p<0.001)*, as well as on positive psychotic-like symptoms *(Black Caribbean=27.0*, *Black African=28.4, White British=23.5, F=8.979, p<0.001, Table 18)*. The group with the significantly higher negative/disorganised schizotypy was Black Caribbean group *(mean=5.4, White British=3.6, F=3.248, p<0.001),* while no differences were observed for negative attenuated psychotic symptoms.

Table 16: Mean schizotypy scores (SIS-R) and attenuated psychotic symptoms scores (CAPE) - between gender comparison

	Total sample Mean (SD) (range min-max)	Males Mean (SD)	Females Mean (SD)	Gender differences (t-test) Significance*
SIS-R Total N=212	**9.17** (4.62) (1-23)	**9.54** (4.81)	**8.87** (4.46)	ns t=-0.195, p=0.296
Positive schizotypy N=212	**5.05** (3.13) (0-14)	**5.0** (3.25)	**5.8** (3.04)	ns t=-1.048, p=0.845
Negative/Disorganised schizotypy N=212	**4.12** (2.31) (0-12)	**4.54** (2.63)	**3.79** (1.99)	t=2.305, p=0.022*
CAPE total N=205	58.89 (9.88) (43-103)	58.97 (8.79)	58.83 (10.73)	ns t=0.097, p=0.922
Positive dimension N=209	25.14 (4.32) (20-46)	25.52 (4.50)	24.77 (4.22)	ns t=-1.122, p=0.263
Negative dimension N=208	20.88 (4.76) (14-37)	21.00 (4.37)	20.76 (5.09)	ns t=-0.210, p=0.834
Depressive dimension N=210	12.91 (3.03) (8-30)	12.45 (2.43)	13.30 (3.44)	t=-2.139, p=0.034*

Note: ns, not significant, p>0.05. SIS-R, Structured Interview for Schizotypy Revised. CAPE, Community Assessment of Psychic Experiences. SD, Standard Deviation

Table 17: Frequencies of schizotypal traits (on SIS-R) in a total sample and between gender comparisons

	Total sample				Males		Females		Gender differences
	Absent N (%)	**Mild** N (%)	**Moderate** N (%)	**Severe** N (%)	**Absent/Mild** (% within gender)	**Moderate/Severe** (% within gender)	**Absent/Mild** (% within gender)	**Moderate/Severe** (% within gender)	*(Pearson Chi-square)*
Social Isolation	95 (44.8%)	74 (34.9%)	36 (17%)	7 (3.3%)	73 (77.7%)	21 (22.3%)	96 (81.4%)	22 (18.6%)	ns
Introversion	31 (16.4%)	104 (49.1%)	66 (31.1%)	11 (5.2%)	53 (56.4%)	41 (43.6%)	82 (69.5%)	36 (30.5%)	X^2=3.887, p=0.049*
Hypersensitivity	47 (22.2%)	77 (36.3%)	65 (30.7%)	23 (10.8%)	57 (60.6%)	37 (39.4%)	67 (56.8%)	51 (43.2%)	ns
Referential thinking 1 (being watched)	84 (39.6%)	97 (45.8%)	30 (14.2%)	1 (0.5%)	79 (84.0%)	15 (16.0%)	102 (86.4%)	16 (13.6%)	ns
Referential thinking 2 (being talked about)	101 (47.6%)	78 (36.8%)	32 (15.1%)	1 (0.5%)	80 (85.1%)	14 (14.9%)	99 (83.9%)	19 (16.1%)	ns
Suspiciousness	33 (15.6%)	116 (54.7%)	54 (25.5%)	9 (4.2%)	69 (73.4%)	25 (26.6%)	80 (67.8%)	38 (32.2%)	ns
Restricted affect	121 (57.1%)	62 (29.2%)	27 (12.7%)	2 (0.9%)	72 (76.6%)	22 (23.4%)	111 (94.1%)	7 (5.9%)	X^2=13.526,p<0.001*
Magical ideation	88 (41.5%)	85 (40.1%)	32 (15.1%)	7 (3.3%)	75 (79.8%)	19 (20.2%)	98 (83.1%)	20 (16.9%)	ns
Illusions	70 (33.0%)	114 (53.8%)	27 (12.7%)	1 (0.5%)	81 (86.2%)	13 (13.8%)	103 (87.3%)	15 (12.7%)	ns
Psychotic phenomena	92 (43.4%)	97 (45.8%)	20 (9.4%)	3 (1.4%)	84 (89.4%)	10 (10.6%)	105 (89.0%)	13 (11.0%)	ns
Derealisation/ Depersonalisation	189 (89.2%)	19 (9.0%)	4 (1.9%)	0	91 (96.8%)	3 (3.2%)	117 (99.2%)	1 (0.8%)	ns (Fisher's Exact test)
Goal-directness of speech/thought process	201 (94.8%)	7 (3.3%)	3 (1.4%)	1 (0.5%)	92 (97.9%)	2 (2.1%)	116 (98.3%)	2 (1.7%)	ns (Fisher's Exact test)
Elevated associativity	205 (96.7%)	5 (2.4%)	2 (0.9%)	0	93 (98.9%)	1 (1.1%)	117 (99.2%	1 (0.8%)	ns (Fisher's Exact test)
Poverty of speech	209 (98.6%)	2 (0.9%)	1 (0.5%)	0	93 (98.9%)	1 (1.1%)	118 (100%)	0	ns (Fisher's Exact test)
Odd/eccentric behaviour	202 (95.3%)	9 (4.2%)	1 (0.5%)	0	93 (98.9%)	1 (1.1%)	118 (100%)	0	ns (Fisher's Exact test)

Note: ns, not significant, p > 0.05

Table 18: Comparison between ethnic groups on mean schizotypy (SIS-R) and attenuated psychotic symptoms (CAPE) scores

	White British	White Other	Black Caribbean	Black African	Asian	Other	Ethnic differences (ANOVA)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Significance*
SIS-R Total	7.63 (3.62)	9.50 (4.63)	11.82[a] (5.07)	10.97[a] (4.56)	9.13 (6.08)	9.94 (5.56)	F=5.742, p<0.001*
Positive schizotypy	4.02 (2.46)	5.35 (3.29)	6.43[a] (3.25)	6.53[a] (3.18)	5.63 (4.27)	5.28 (3.72)	F=5.299, p<0.001*
Negative/Disorganised schizotypy	3.61 (1.94)	4.15 (2.17)	5.39[a] (2.94)	4.44 (2.26)	3.50 (2.07)	4.67 (2.91)	F=3.248, p=0.008*
CAPE total	56.63 (7.67)	63.62[a] (11.14)	59.15 (10.23)	61.93 (12.76)	59.57 (11.15)	58.94 (10.13)	F=2.948 p=0.014*
Positive dimension	23.46 (3.17)	25.23 (2.93)	27.0[a] (4.66)	28.37[a] (9.53)	26.0 (4.67)	25.63 (4.30)	F=8.979, p<0.001*
Negative dimension	20.44 (4.05)	23.08 (5.42)	19.88 (4.84)	21.17 (5.61)	21.14 (4.95)	20.88 (5.60)	F=1.592, p=0.164
Depressive dimension	12.3[b] (2.40)	15.31 (4.60)	12.27[b] (3.18)	12.40[b] (2.75)	12.43 (2.57)	12.44[b] (2.71)	F=4.206, p=0.001*

Note: [a] significantly different from 'White British' after Bonferroni Post Hoc test. [b] significantly different from 'White Other' after Bonferroni Post Hoc test. ns, not significant. SIS-R, Structured Interview for Schizotypy Revised. CAPE, Community Assessment of Psychic Experiences. SD, Standard Deviation

5.1 *Is there support for the Childhood Trauma and Schizotypy association?*

Childhood Trauma and Schizotypy association

There was a linear association observed for total trauma score and schizotypy symptoms *(unadj. β=1.25, p<0.001, see Table 19)*, providing support for the main hypothesis of the thesis *(Hypothesis 1)*. Furthermore, looking at the types of trauma, psychological abuse *(unadj. β=3.68, p=0.001)*, physical abuse *(unadj. β=3.29, p<0.001)*, sexual abuse *(unadj. β=2.49, p=0.003)* and bullying *(unadj. β=1.90, p=0.005)* all predicted schizotypy load *(Table 19)*. For household discord on the other hand no such relationship was observed *(unadj. β=1.11, p=0.094)*. Nevertheless when looking at the highest schizotypy scorers (top 20%), household discord showed an odds ratio of 2.02 *(unadj. p=0.036)*. The strongest support was found for physical abuse *(unadj. OR=5.21, p<0.001)*, similar to psychological abuse *(unadj. OR=4.08, p=0.005, Table 21)*. Moreover, even more robust support was observed between childhood trauma and the top 10% of schizotypy total scores, with the strongest support observed for psychological abuse *(unadj. OR=6.59, p=0.001)* and physical abuse *(unadj. OR=6.18, p<0.001) (Table 21)*.

Table 19: Associations between total trauma (and all distinct trauma types) and total schizotypy

| | SIS-R total score | | | | SIS-R total score | | | |
| | Linear Regression Unadj. | | | | Linear regression Adj.* | | | |
	β coefficient	t-value	p - value	95 % CI	β coefficient	t-value	p - value	95 % CI
Total trauma	*1.25*	*4.17*	*<0.001*	*.66 – 1.84*	*.88*	*2.93*	*0.004*	*.29 - 1.48*
Household discord	*1.11*	*1.68*	*0.094*	*-.19 – 2.41*	*.74*	*1.16*	*0.246*	*-.52 - 2.00*
Psychological abuse	*3.29*	*2.10*	*0.037*	*.21 – 6.37*	*1.72*	*1.09*	*0.278*	*-1.40 - 4.83*
Psychological abuse (all levels of severity)	*3.68*	*3.31*	*0.001*	*1.49 – 5.88*	*2.94*	*2.69*	*0.008*	*.78 - 5.10*
Physical abuse	*3.29*	*4.34*	*<0.001*	*1.80 – 4.79*	*2.11*	*2.61*	*0.010*	*.52 - 3.70*
Sexual abuse	*1.28*	*1.03*	*0.304*	*-1.17 – 3.73*	*1.48*	*1.17*	*0.244*	*-1.02 - 3.40*
Sexual abuse (all levels of severity)	*2.49*	*3.01*	*0.003*	*.86 – 4.11*	*2.46*	*2.98*	*0.003*	*.83 - 4.09*
Bullying	*1.90*	*2.84*	*0.005*	*.58 – 3.21*	*1.48*	*2.26*	*0.025*	*.19 - 2.78*

*Adj. for gender, age, ethnicity, employment status, religion, IQ (Overall model fit, R-values available in Table Suppl.3, Appendix XVI). SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

When adjusting for con-founders (gender, age, ethnicity, employment status, religion and IQ), similar findings were observed, with psychological abuse having the strongest association with schizotypy *(adj. β=2.94, p=0.008)*, followed by sexual abuse *(adj. β=2.46, p=0.003)* and physical abuse *(adj. β=2.11, p=0.010)*. Furthermore, when the associations observed between a particular trauma type and schizotypy were controlled for the other types of trauma, the results remained similar *(e.g. psychological abuse: β=3.33, p=0.024, physical abuse: β=2.17, p=0.012, sexual abuse: β=2.14, p=0.012 and bullying: β=1.54, p=0.032)*. Therefore there was not a particular type of trauma accounting for most of the associations.

Death of a parent and separation from a biological mother showed no association with schizotypy dimensions *(Table 20)*. However, separation from a biological father significantly predicted schizotypy positive *(unadj. β=1.03, p=0.026)* and negative/disorganised dimensions *(unadj. β=1.10, p=0.001, see Table Suppl.7, Appendix XVI)*. After adjusting for age, gender, ethnicity and IQ, a trend was only observed for negative schizotypy *(adj. β=.68, p=0.053)*. The main reason stated for the separation from a father was divorce *(42.6%)*, therefore it might be the divorce itself that can help explain these associations.

Table 20: Association between separation from a parent and parental death experiences and total schizotypy

	SIS-R total score Unadj.			SIS-R total score Adj.*		
	β coefficient	p - value	95 % CI	β coefficient	p - value	95 % CI
Death of a parent(s)	-.18	0.885	-2.59-2.23	-.19	0.873	-2.53 - 2.15
Separation from a biological mother	1.52	0.100	-.29 – 3.33	.81	0.930	-1.75 -1.91
Separation from a biological father	**2.13**	**0.002**	**.81 – 3.45**	-.80	0.244	-.55 - 2.14

*Adj. for gender, age, ethnicity (White, Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

When looking at the two schizotypy dimensions separately, positive schizotypy symptoms were mainly predicted by total trauma *(unadj. OR=1.60, p=0.002)* and especially psychological *(unadj. OR=3.78, p=0.008)* and physical abuse *(unadj. OR=3.38, p=0.001)*, also indicating that the differential effects of trauma types are especially found for the positive dimension (e.g. sexual abuse and bullying showed no association with this sub-dimension) *(Table 22)*. Less clear was specificity of trauma types on negative/disorganised schizotypal dimension *(Table 23)*. Total trauma score *(unadj. OR=1.57, p=0.003)*, along with physical *(unadj. OR=3.24, p=0.001)*, sexual abuse *(unadj. OR=2.65, p=0.011)* and bullying *(unadj. OR=2.39, p=0.008)* increased the negative symptoms (looking at top 20% of scorers), psychological abuse showed a trend toward this association *(unadj. OR=2.63, p=0.055)*, while no effect was seen for household discord. For linear regression scores exploring distinct trauma types and positive and negative/disorganised schizotypy see *Table Suppl.5 & Table Suppl.6, Appendix XVI*.

Table 21: Association between trauma types and top 20% and top 10% of schizotypy scorers

	20 % top scorers SIS-R total Unadj.			20 % top scorers SIS-R total Adj.*			10 % top scorers SIS-R total Unadj.			10 % top scorers SIS-R total Adj.**		
	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI
Total trauma	1.87	<0.001	1.36 – 2.57	1.70	0.002	1.21 - 2.39	2.05	<0.001	1.39 – 3.03	1.83	0.003	1.23 - 2.74
Household discord	2.02	0.036	1.05 – 3.91	2.10	0.044	1.02 - 4.35	2.34	0.052	.99 – 5.53	2.33	0.065	.95 - 5.72
Psychological abuse	4.76	0.024	1.22 – 18.51	4.85	0.039	1.08 - 21.76	4.31	0.050	1.00 – 18.5	(-)	(-)	(-)
Psychological abuse (all levels of severity)	4.08	0.005	1.52 – 10.97	4.31	0.009	1.45 - 12.85	6.59	0.001	2.26 – 19.12	7.62	0.001	2.34 - 24.77
Physical abuse	5.21	<0.001	2.51 – 10.81	3.56	0.003	1.55 - 8.17	6.18	<0.001	2.53 – 15.10	3.94	0.005	1.51 – 10.26
Sexual abuse	1.31	0.660	0.40 - 4.32	1.09	0.885	.30 - 4.00	(-)	(-)	(-)	(-)	(-)	(-)
Sexual abuse (all levels of severity)	2.26	0.039	1.04 – 4.91	2.43	0.047	1.01 - 5.81	3.60	0.007	1.42 – 9.12	3.51	0.016	1.27 – 9.70
Bullying	2.26	0.017	1.16 – 4.40	1.91	0.083	.92 - 3.96	3.45	0.005	1.44 – 8.24	3.30	0.010	1.33 – 8.23

*Adj. for gender, age, ethnicity (White, Black Caribbean/Black African, Other)
** Adj. gender, ethnicity (White, Non-white). CI, Confidence Interval. OR, Odds Ratio. SIS-R, Structured Interview for Schizotypy Revised

Table 22: Association between trauma types and top 20% and top 10% of scorers on positive schizotypy

	20 % top scorers SIS-R Positive Unadj.			20 % top scorers SIS-R Positive Adj.*			10 % top scorers SIS-R Positive Unadj.			10 % top scorers SIS-R Positive Adj.**		
	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI
Total trauma	**1.60**	**0.002**	**1.19 – 2.14**	**1.39**	**0.039**	**1.02 - 1.90**	**2.01**	**<0.001**	**1.37 - 2.94**	**1.82**	**0.003**	**1.23 – 2.72**
Household discord	**1.87**	**0.048**	**1.01 – 3.46**	1.63	0.150	.84 - 3.19	**2.84**	**0.018**	**1.20 – 6.79**	**2.97**	**0.018**	**1.20 – 7.33**
Psychological abuse (all levels of severity)	**3.78**	**0.008**	**1.41 – 10.11**	**3.79**	**0.013**	**1.32 - 10.90**	**3.52**	**0.030**	**1.13 – 10.96**	**3.56**	**0.037**	**1.08 –11.78**
Physical abuse	**3.38**	**0.001**	**1.68 – 6.81**	**2.32**	**0.042**	**1.03 - 5.20**	**4.10**	**0.002**	**1.68 – 9.97**	**2.61**	**0.050**	**1.00 – 6.81**
Sexual abuse (all levels of severity)	1.81	0.120	.86 – 3.83	1.40	0.432	.60 - 3.23	2.11	0.128	.81 – 5.34	2.11	0.160	.74 – 6.00
Bullying	1.75	0.082	.93 – 3.28	1.55	0.216	.77 - 3.09	1.92	0.135	.81 – 4.56	1.70	0.243	.67 – 4.17

*Adj. for gender, age, ethnicity (White, Black Caribbean/Black African, Other)
** Adj. gender, ethnicity (White/Non-white). CI, Confidence Interval. OR, Odds Ratio. SIS-R, Structured Interview for Schizotypy Revised

Table 23: Association between trauma types and top 20% and top 10% of scorers on negative/disorganised schizotypy

	20 % top scorers SIS-R Negative/Disorganised Unadj.			20 % top scorers SIS-R Negative/Disorganised Adj.*			10 % top scorers SIS-R Negative/Disorganised Unadj.			10 % top scorers SIS-R Negative/Disorganised Adj.**		
	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI
Total trauma	**1.57**	**0.003**	**1.16 – 2.12**	**1.50**	**0.013**	**1.09 -2.07**	**2.01**	**<0.001**	**1.37 0 2.94**	**1.60**	**0.029**	**1.05 – 2.43**
Household discord	1.38	0.332	.73 – 2.61	1.48	0.261	.75 - .94	**2.84**	**0.018**	**1.20 – 6.77**	1.78	0.232	.69 – 4.59
Psychological abuse (all levels of severity)	2.63	0.055	0.99 – 7.06	2.67	0.067	.93 - 7.67	**5.05**	**0.002**	**1.83 – 16.71**	**6.09**	**0.003**	**1.81 – 20.44**
Physical abuse	**3.24**	**0.001**	**1.59 – 6.60**	**2.33**	**0.039**	**1.04 - 5.20**	**4.10**	**0.002**	**1.68 – 9.97**	**3.32**	**0.021**	**1.20 –9.21**
Sexual abuse (all levels of severity)	**2.65**	**0.011**	**1.25 – 5.63**	**3.26**	**0.006**	**1.41 - 7.56**	2.01	0.180	.72 – 5.59	2.85	0.076	.90 – 9.02
Bullying	**2.39**	**0.008**	**1.26 – 4.54**	**2.19**	**0.026**	**1.10 - 4.36**	1.93	0.135	.81 – 4.56	1.85	0.198	.73 – 4.70

*Adj. for gender, age, ethnicity (White, Black Caribbean/Black African, Other)
** Adj. gender, ethnicity (White/Non-white). CI, Confidence Interval. OR, Odds Ratio. SIS-R, Structured Interview for Schizotypy Revised

Dose-response association between childhood trauma and schizotypy

A dose-response relationship between a number of types of traumatic events and schizotypal load was also evident, as 2 types of trauma resulted in *β* coefficient of 2.52 *(p=0.009)* which almost doubled for those who reported 3 or more types of trauma *(unadj. β=4.96, p=0.001) (Table 24)*. This trend was observed for positive and negative/disorganised dimension, however when adjusting for confounders, these dose-response associations became less clear. Likewise, considering frequency ratings along with severity, it was found that for a majority of trauma types, high frequency was required along with high severity to significantly impact schizotypy scores (for physical abuse: *unadj. β=4.94, p<0.001*, for psychological abuse: *unadj. β=4.46, p=0.019*, for bullying: *unadj. β=1.91, p=0.012, Table 25)*. On the other hand, even low severity with high frequency in psychological abuse was associated with schizotypy *(unadj. β=4.23, p=0.024)*, and for sexual abuse low severity and low frequency of this type of abuse was associated with schizotypy *(unadj. β=2.77, p=0.009)*. Nevertheless due to the small numbers in each category, this does not allow any firm conclusions to be drawn.

Table 24: Association between number of types of traumatic experiences and total, positive and negative/disorganised schizotypy

High severity and high frequency only	Total SIS-R Unadj.		Total SIS-R Adj.*		SIS-R Positive Unadj.		SIS-R Positive Adj.*		SIS-R Negative Unadj.		SIS-R Negative Adj.*	
	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value
1 type of trauma	.31[a]	0.660	.49	0.485	.50[b]	0.304	.49	0.301	-.18[c]	0.610	-.001	0.987
2 types of trauma	2.52[a]	0.009	2.47	0.007	1.72[b]	0.008	1.58	0.012	.80[c]	0.101	.89	0.062
3 and 4 types of trauma	4.96[a]	0.001	3.15	0.038	2.58[b]	0.012	1.53	0.138	2.37[c]	0.002	1.61	0.041

*Adj. for gender, age, IQ [a] Linear trend test, z=3.45, p<0.001; [b] Linear trend test, z=3.50, p<0.001, [c] Linear trend test, z=2.68, p=0.007
SIS-R, Structured Interview for Schizotypy Revised

Table 25: Association between trauma types (different levels of frequency and severity) and total schizotypy

	House. discord N (%)	β coeff. (p – value)	Psych. abuse N (%)	β coeff. (p – value)	Physical abuse N (%)	β coeff. (p – value)	Sexual abuse N (%)	β coeff. (p- value)	Bullying N (%)	β coeff. (p -value)
No abuse	90 (42.4%)	(-)	193 (91.5%)	(-)	103 (48.8%)	(-)	171 (82.6%)	(-)	87 (42.0%)	(-)
Low severity & low frequency	24 (11.3%)	β=-.21 (0.845)	3 (1.4%)	β=3.13 (0.238)	53 (25.1%)	β=-.07 (0.926)	21 (10.1%)	β=2.77 (0.009)	16 (7.7%)	β=-.44 (0.725)
Low severity & high frequency	18 (8.5%)	β=.5 (0.677)	6 (2.8%)	β=4.23 (0.024)	12 (5.7%)	β=.66 (0.623)	0	(-)	36 (17.4%)	β=.78 (0.386)
High severity & low frequency	8 (3.8%)	β=1.29 (0.452)	3 (1.4%)	β=1.46 (0.581)	16 (7.6%)	β=1.13 (0.335)	10 (4.8%)	β=1.10 (0.459)	5 (2.4%)	β=3.76 (0.075)
High severity & high frequency	72 (34.0%)	β=.79 (0.282)	6 (2.8%)	β=4.46 (0.019)	27 (12.8%)	β=4.94 (<0.001)	5 (2.4%)	β=2.70 (0.193)	63 (30.4%)	β=1.91 (0.012)
Total	212 (100%)	(-)	211 (100%)	(-)	211 (100%)	(-)	207 (100%)	(-)	207 (100%)	(-)

Unadjusted scores

Age of trauma occurrence and association between childhood trauma and

schizotypy

There is some evidence that childhood trauma/abuse that started before the age of 11 shows a stronger association with schizotypy. Also, total trauma score for adversities before the age of 12 was linked to schizotypy traits *(unadj. β=1.67, p=0.011, still trend toward this association after adjusting for confounders)*, especially the positive dimension *(unadj. β=1.44, p=0.002)*. On the contrary, total trauma for adversities at the age of 12 or after had no such effect *(Table 26)*. Equally, psychological abuse that begun before 12 shows a strong relationship with schizotypy total *(adj. β=3.69, p=0.004)*, positive *(adj. β=2.36, p=0.013)* and negative dimensions *(adj. β=1.59, p=0.026)* which failed to reach significance if the abuse started later in life. These results require some caution due to small numbers of psychological abuse in both age groups ('before 12' and '12 or after'). Interestingly, for sexual abuse and bullying the opposite was observed, sexual abuse after the age of 12 was particularly associated with total schizotypy *(adj. β=3.12, p=0.011)*, positive *(adj. β=1.88, p=0.027)* and negative symptoms *(adj. β=1.24, p=0.049)* and bullying that started in adolescence was associated with total schizotypy score *(adj. β=2.35, p=0.017)* and the negative dimension *(adj. β=1.30, p=0.011)*, with less clear associations if trauma started earlier in life *(trend toward significance for positive symptoms only: adj. β=.90, p=0.081)*.

Table 26: Association between age of trauma occurrence and total, positive and negative/disorganised schizotypy dimensions

	SIS-R total Unadj.		SIS-R total Adj.*		SIS-R Positive Unadj.		SIS-R Positive Adj.*		SIS-R Negative/Disorganised Unadj.		SIS-R Negative/Disorganised Adj.*	
	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value
Abuse present Age ≤11												
Total trauma	**1.76**	**0.011**	1.15	0.086	**1.44**	**0.002**	**.96**	**0.034**	.32	0.358	.19	0.585
Household discord (N=56)	1.32	0.073	.86	0.224	**.97**	**0.051**	.57	0.237	.39	0.347	.29	0.431
Psychological abuse (all levels) (N=11)	**4.58**	**0.001**	**3.96**	**0.004**	**2.74**	**0.005**	**2.36**	**0.013**	**1.84**	**0.010**	**1.59**	**0.026**
Physical abuse (N=39)	**3.43**	**<0.001**	**2.39**	**0.005**	**1.97**	**<0.001**	**1.34**	**0.019**	**1.46**	**<0.001**	**1.02**	**0.019**
Sexual abuse (all levels) (N=21)	**2.63**	**0.014**	1.86	0.068	1.08	0.163	.60	0.389	**1.55**	**0.004**	**1.25**	**0.017**
Bullying (N=45)	**1.70**	**0.030**	1.18	0.116	**1.97**	**0.019**	.90	0.081	.46	0.243	.28	0.472
Abuse present Age ≥12												
Total trauma	1.41	0.172	1.46	0.136	.77	0.267	.71	0.283	.64	0.222	.75	0.140
Household discord (N=19)	.75	0.505	.78	0.459	.85	0.265	.70	0.335	-.10	0.866	.08	0.876
Psychological abuse (all levels) (N=7)	2.27	0.193	1.95	0.232	.67	0.570	.40	0.718	1.60	0.071	1.55	0.068
Physical abuse (N=3)	4.50	0.082	2.40	0.345	1.66	0.133	1.29	0.457	1.84	0.162	1.10	0.401
Sexual abuse (all levels) (N=15)	2.23	0.071	**3.12**	**0.011**	**1.64**	**0.052**	**1.88**	**0.027**	.59	0.339	**1.24**	**0.049**
Bullying (N=23)	**2.28**	**0.026**	**2.35**	**0.017**	1.05	0.132	1.06	0.116	**1.23**	**0.018**	**1.30**	**0.011**

*Adj. for gender, age, ethnicity (White, Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised

Gender differences in Childhood Trauma and Schizotypy association

When comparing the association between childhood trauma and schizotypy between genders, the differential effects were observed for sexual abuse (for females *unadj. β=3.61, p<0.001*, for males *unadj. β=.44, p=0.795, the moderating effect of gender not significant, Table 27*), especially evidenced for positive schizotypy symptoms (for females *unadj. β=2.08, p=0.002,* for males *unadj. β=-.61, p=0.592; interaction effect: β=2.69, p=0.036*). However, only *9 (9.8%)* male participants and *28 (24%)* female participants reported sexual abuse at any severity (some/moderate/marked) which requires caution when interpreting the results. Furthermore, looking at physical abuse, the association with total schizotypy load was stronger for males (for females *unadj. β=2.05, p=0.053,* while for males *unadj. β=4.45, p<0.001, the moderating effect of gender not significant*), again mainly affecting the positive dimension (for females *unadj. β=.87, p=0.238,* for males *unadj. β=2.91, p<0.001; interaction effect: β=-2.03, p=0.052*). Also bullying seemed to have a clearer association with schizotypy for females (*unadj. β=3.01, p=0.001,* while for males *unadj. β=.68, p=0.505*), however statistically, the difference was not significant. Gender comparison of association between trauma types and positive and negative/disorganised schizotypy dimensions is attached in *Table Suppl.8 & Table Suppl.9, Appendix XVI*.

Table 27: Gender comparison of association between trauma types and total schizotypy

	Total schizotypy Males		Total schizotypy Females		Childhood Trauma and Gender Interaction effect	
	β coeff.	*p-value*	*β coeff.*	*p-value*	*β coeff.*	*p-value*
Total trauma	*1.15*	*0.019*	*1.33*	*0.001*	*.46*	*0.726*
Household discord	*1.41*	*0.194*	*1.06*	*0.207*	*-.36*	*0.792*
Psychological abuse	*3.45*	*0.040*	*3.84*	*0.013*	*.39*	*0.863*
Physical abuse	*4.45*	*<0.001*	*2.05*	*0.053*	*-2.37*	*0.120*
Sexual abuse	*.44*	*0.795*	*3.61*	*<0.001*	*3.16*	*0.091*
Bullying	*.68*	*0.505*	*3.01*	*0.001*	*2.34*	*0.085*

Unadjusted scores

Childhood Trauma and attenuated/psychotic-like symptoms

In addition to childhood trauma and schizotypy association, the study looked at the relationship between childhood trauma and narrow psychotic-like symptoms (using CAPE). Whilst SIS-R captures general schizotypy/schizotypal personality traits, CAPE focuses on psychosis-like symptomatology by embracing more diagnostic criteria for assessing psychotic symptoms.

Spearman's rho correlation between Positive *SIS-R* and Positive *CAPE* dimension showed moderate correlation *(r=0.608, p<0.001)*, low correlation was found between Negative *SIS-R* and Negative *CAPE (r=0.438, p<0.001)*.

Similar to associations between childhood trauma and schizotypy, all types of trauma impacted on the total psychotic-like symptom score, with the sole exception of household discord which showed an association but failed to reach statistical significance (*unadj. β=2.59, p=0.073, Table 28)*. The most robust associations with attenuated psychotic symptoms were observed for physical (*unadj. β=7.15, p<0.001)*, sexual (*unadj. β=7.12, p=0.009)* and psychological abuse (*unadj. β=6.73, p=0.046)*, all showing similarly strong associations after adjusting for possible confounders. When these associations were adjusted for all trauma types, physical (*unadj. β=6.65, p<0.001)* and sexual abuse (*unadj. β=5.05, p=0.045)* held similar associations with the total CAPE scores, but *β* coefficient for psychological abuse was almost halved (*unadj. β=3.40, p=0.163)*. Also, in parallel with reports using SIS-R, parental loss (*unadj. β=-2.40, p=0.379)* and separation from mother (*unadj. β=1.90, p=0.345)* showed no relationship with psychotic-like symptoms but separation from father did predict the increase in total *CAPE* scores (*unadj. β=4.00, p=0.007)*. The strongest predictor of positive psychotic symptoms was physical abuse (*unadj. β=4.17, p<0.001)*, whilst psychological (*unadj. β=3.04, p=0.009)* and sexual abuse (*unadj. β=3.39, p=0.008)* were the strongest predictors of the negative

dimension *(Table 29).* The associations between distinct trauma types and depressive symptoms of the *CAPE* scale are presented in *Table Suppl.10, Appendix XVI.*

Table 28: Associations between total trauma (and all distinct trauma types) and attenuated/psychotic-like symptoms total (on CAPE measure)

	CAPE total score				CAPE total score			
	Linear Regression Unadjusted				Linear regression Adjusted*			
	β coefficient	t- value	p - value	95 % CI	β coefficient	t- value	p - value	95 % CI
Total trauma	**3.09**	**4.81**	**<0.001**	**1.82 – 4.35**	**2.91**	**4.27**	**<0.001**	**1.57 - 4.26**
Household discord	2.59	1.80	0.073	-.24 – 5.40	2.00	1.35	0.179	-.93 - 4.94
Psychological abuse	**6.73**	**2.01**	**0.046**	**-.12 – 13.34**	6.93	1.95	0.053	-.08 - 13.94
Psychological abuse (all levels of severity)	**6.57**	**2.73**	**0.007**	**1.82 –11 .31**	**6.41**	**2.57**	**0.011**	**1.50 - 11.32**
Physical abuse	**7.15**	**4.31**	**<0.001**	**3.88 – 10.43**	**7.39**	**4.01**	**<0.001**	**3.75 - 11.03**
Sexual abuse	**7.12**	**2.62**	**0.009**	**1.76 – 12.49**	**7.88**	**2.73**	**0.007**	**2.18 – 13.58**
Sexual abuse (all levels of severity)	**5.13**	**2.85**	**0.005**	**1.58 – 8.68**	**2.67**	**2.99**	**0.003**	**1.93 - 9.41**
Bullying	**3.92**	**2.69**	**0.008**	**1.05 – 6.79**	**3.30**	**2.16**	**0.032**	**.28 - 6.33**
Separation from mother	1.90	.95	0.345	-2.06 – 5.87	.94	.43	0.666	-3.36 – 5.24
Separation from father	**4.00**	**2.72**	**0.007**	**1.10 – 6.91**	3.07	2.92	0.056	-.08 – 6.21
Parental death	-2.40	-.88	0.379	-7.76 – 2.96	-2.00	-.71	0.481	-7.60 – 3.59

*Adj. for gender, age, ethnicity (White, Black Caribbean/Black African, Other), IQ. CAPE, Community Assessment of Psychic Experiences. CI, Confidence Interval

Table 29: Associations between total trauma (and all distinct trauma types) and CAPE Positive and CAPE Negative dimensions

	CAPE Positive				CAPE Negative			
	Linear regression Adjusted*				Linear regression Adjusted*			
	β coefficient	t- value	p - value	95 % CI	β coefficient	t- value	p - value	95 % CI
Total trauma	**.89**	**3.19**	**0.002**	**.34 - 1.44**	**1.09**	**3.26**	**0.001**	**.43 - 1.74**
Household discord	**1.41**	**2.40**	**0.017**	**.25 -2.57**	.10	0.14	0.888	-1.31 -1.52
Psychological abuse	.39	0.27	0.787	-2.46 - 3.25	2.54	1.47	0.143	-.86 - 5.94
Psychological abuse (all levels of severity)	.69	0.68	0.498	-1.32 - 2.71	**2.84**	**2.36**	**0.019**	**.47 - 5.23**
Physical abuse	**2.93**	**3.98**	**<0.001**	**1.48 - 4.39**	**2.37**	**2.62**	**0.009**	**.59 - 4.15**
Sexual abuse	.29	0.25	0.804	-1.98 – 2.56	**4.39**	**3.28**	**0.001**	**1.75 – 7.04**
Sexual abuse (all levels of severity)	1.24	1.62	0.106	-.27 - 2.76	**2.57**	**2.84**	**0.005**	**.78 - 4.36**
Bullying	.72	1.17	0.242	-.49 - 1.49	**1.45**	**1.98**	**0.050**	**.00 - 2.90**

Adj. for gender, age, ethnicity (White, Black Caribbean/Black African, Other), IQ (For unadjusted values see Table Suppl.11, Appendix XVI). CAPE, Community Assessment of Psychic Experiences. CI, confidence interval

5.2 Life Events (and interaction with Childhood Trauma) and Schizotypy association

Following previous research findings, the study explored whether recent life events moderated the associations observed between childhood trauma and schizotypy levels.

A life event *(12-month period prior to the interview)* of moderate or marked threat/severity was reported by *53.2%* participants with 30 subjects *(14.1%)* reporting 3 or more events of this severity *(Table 30)*. At least one independent event was documented for *41.4%*, while at least one intrusive event was found for 19 participants *(9.4%)*. Overall, females reported more life events compared to males *(Kruskal-Wallis test=3.59, p=0.058)*.

Table 30: Frequency of recent life events and between gender comparison

	Total events N (%)	Total events Males N (% within gender)*	Total events Females N (% within gender)*	Total independent events N (%)	Total intrusive events (dichotomised score) N (%)
0 events	99 (46.7)	50 (53.2)	49 (51.5)	119 (58.6)	184 (90.6)
1 event	55 (25.9)	25 (26.6)	30 (25.4)	53 (26.1)	19 (9.4)
2 events	28 (13.2)	7 (7.4)	21 (17.8)	16 (7.9)	(-)
3 or more events	30 (14.1)	12 (12.8)	18 (15.2)	15 (7.4)	(-)
Total	212 (100)	94 (100)	118 (100)	208 (100)	203 (100)

* difference between genders on a number of life events Kruskal-Wallis=3.59, p=0.058

Table 31: Frequency of recent life events among individuals with and without childhood trauma

Childhood trauma	Number of recent life events				
	0 events	*1 event*	*2 events*	*3 or more*	*Total*
No trauma	39 (46.4%)	23 (27.4%)	8 (9.5%)	14 (16.7%)	84 (100%)
Trauma present	57 (46.7%)	31 (25.4%)	18 (14.7%)	16 (13.1%)	122 (100%)

The total number of life events was found to significantly predict total schizotypal score (*adj. β=.57, p=0.043, Table 32),* with intrusive events having most robust association with total schizotypy (*adj. β=2.18, p=0.038),* mainly the positive schizotypy dimension (*adj. β=1.32, p=0.069, Table 33)*. Childhood trauma (dichotomised) was associated with total schizotypy (*β=1.40, p=0.027),* but

interaction effects of childhood trauma and intrusive life events further increased this association (for total schizotypy *adj. β=4.20, p=0.045, Table 32*) which was mainly significant for negative/disorganised (*adj. β=2.67, p=0.015, Table 33*) but not positive symptoms (*adj. β=1.53, p=0.284*). Nevertheless, positive symptoms were also predicted by life events (*adj. β=.39, p=0.045*) and the association between intrusive events and positive schizotypy was approaching significance (*adj. β=1.32, p=0.069*).

Recent life difficulties in addition to events resulted in no significant increase in associations with schizotypal load.

Table 32: The interaction effects of the recent life events and difficulties and childhood trauma on total schizotypy score

	Main effects on SIS-R Total Adj.*			Trauma (dichotomised) Interaction effect Adj.**		
	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI
Life events	**.57**	**0.043**	**.02– 1.12**	-.58	0.646	-3.09- 1.22
Life events – **independent**	.46	0.178	-.21–1.14	-.74	0.572	-3.31- 1.83
Life events – **intrusiveness** (dichotomised)	**2.18**	**0.038**	**.12– 4.25**	**4.20**	**0.045**	**.10 - 8.31**
Life events and difficulties	.40	0.118	-.10 - .92	-.49	0.702	-3.00 -2.02
Life events and difficulties – **independent**	.32	0.324	-.32–.95	-.57	0.659	-3.13- 1.98
Life events and difficulties – **intrusiveness** (dichotomised)	1.23	0.226	-.77-3.23	3.31	0.098	-.62 – 7.26

*Adj. for gender, age, ethnicity (White/Non-White), employment status, IQ
**Adj. for gender, age, ethnicity (White/Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table 33: The interaction effects of recent life events and difficulties and childhood trauma on positive and negative/disorganised schizotypy

	Main effects on Positive SIS-R Adj.*			Trauma (dichotomised) Interaction effect Adj.**			Main effects on Negative/Disorganised SIS-R Adj.*			Trauma (dichotomised) Interaction effect Adj.**		
	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI
Life events	**.39**	**0.045**	**.01– .77**	-.58	0.501	-2.28-1.12	.18	0.210	-.10– .47	-.01	0.994	-1.31- 1.30
Life events – **independent**	.41	0.086	-.06–.87	-2.45	0.114	-5.51- .60	.06	0.748	-.29–.41	1.55	0.199	-.82- 3.92
Life events – **intrusiveness** (dichotomised)	1.32	0.069	-.65–2.30	1.53	0.284	-1.28- 4.34	.86	0.110	-.20–1.93	**2.67**	**0.015**	**0.53 - 4.81**
Life events and difficulties	.28	0.121	-.07 - .63	-.54	0.528	-2.24 -1.15	.13	0.337	-.13 - .39	.06	0.932	-1.26 -1.37
Life events and difficulties – **independent**	.31	0.156	-.12–.75	-.77	0.376	-2.50- .95	.04	0.978	-.32–.33	.20	0.769	-1.14- 1.54
Life events and difficulties – **intrusiveness** (dichotomised)	.75	0.284	-.62-2.12	1.28	0.346	-1.39–3.95	.48	0.358	-.55-1.52	2.04	0.053	-.03 – 4.10

*Adj. for gender, age, ethnicity (White/Non-White), employment status, IQ
**Adj. for gender, age, ethnicity (White/Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

5.3 *Familial risk for Psychosis (and interaction with Childhood Trauma) and Schizotypy association*

As literature suggests a strong genetic impact on the development of schizotypal traits, this study also tested the interaction effects between childhood trauma and familial risk for psychosis on schizotypy total score.

Ten participants *(4.9%)* reported having one of their first degree relatives with present or past psychotic disorder *(Table 34)*. When all assessed mental illnesses were considered the number of 'potentially genetically at risk' participants rose to 82 *(40.2%)*. This broader inclusion of familial risk for neuropsychiatric disorders was underpinned by the genetic overlap of schizophrenia with mood disorders observed in several studies (Cross-Disorder Group of the Psychiatric Genomics Consortium 2013).

Table 34: Frequency of familial risk of psychosis ('psychosis/narrow' and 'any mental illness/broad' definition)

Genetic risk	N (%)		Males N (% within gender)		Females N (% within gender)	
	Present	Absent	Present	Absent	Present	Absent
Psychosis (parents only)	6 (2.9)	200 (97.1)	3 (3.3)	89 (96.7)	3 (2.6)	111 (97.4)
Psychosis (any first degree relative)	10 (4.9)	193 (95.1)	7 (7.7)	84 (92.3)	3 (2.7)	109 (97.3)
Any Mental Illness (parents only)	49 (23.7)	158 (76.3)	19 (20.6)	73 (79.3)	30 (26.1)	85 (73.9)
Any Mental Illness (any first degree relative)	82 (40.2)	122 (59.8)	36 (39.6)	55 (60.4)	46 (40.7)	57 (59.3)

The results suggest a strong association between familial risk of psychosis (narrow definition) and schizotypy levels *(adj. β=3.41, p=0.015, Table 35)*. Also, when considering 'risk for neuropsychiatric disorders' as a broader term, association with schizotypal total score was less strong, but still significant *(adj. β=1.40, p=0.027)*. Positive schizotypal symptoms were only associated with the narrow psychosis definition (only first degree relatives with a history of psychosis) *(adj. β=2.10, p=0.031, Table 36)*. In contrast, the negative dimension was associated with familial risks in terms of both narrow *(unadj. β=1.66, p=0.027)* and broad terms

(unadj. β=.70, p=0.033), however after adjusting for potential confounders, only the association with familial risk including any mental illness assessed remained significant *(adj. β=.72, p=0.022)*.

Interaction effects between childhood trauma and familial risk on development of schizotypy were observed for the positive dimension only *(unadj. β=2.01, p=0.031, adj. β=.72, p=0.059)*. When comparing the effects of different trauma types, bullying was the only type of victimisation for which interaction with familial risk for psychosis on schizotypy load approached significance *(adj. β=2.42, p=0.069, Table 37)*. Besides, there was a trend observed for psychological trauma specifically *(interaction with familial risk adj. β=3.07, p=0.057)* and bullying *(interaction with familial risk adj. β=1.73, p=0.060)* with positive schizotypal symptoms. No interaction effects between trauma and familial risk were observed for the negative/disorganised dimension.

Table 35: The interaction effects of familial risk for psychosis and childhood trauma on total schizotypy

Genetic risk	Main effects on SIS-R Total Unadj.			Main effects on SIS-R Total Adj.*			Trauma – dichotomised Interaction effect Unadj.			Trauma – dichotomised Interaction effect Adj.**		
	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI
Psychosis	**4.04**	**0.007**	**1.10 -6.98**	**3.41**	**0.015**	**.66 - 6.16**	2.41	0.512	-4.83 – 6.66	1.58	0.651	-5.30-8.46
any Mental Illness	**1.41**	**0.033**	**.11 – 2.71**	**1.47**	**0.018**	**.26 – 2.69**	2.52	0.074	-.25 – 5.28	1.77	0.181	-.83 – 4.38

*Adj. for gender, age, ethnicity (White, Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence interval

Table 36: The interaction effects of familial risk for psychosis and childhood trauma on positive and negative/disorganised schizotypy

Genetic risk	Main effects on SIS-R Positive Adj.*			Trauma – dichotomised Interaction effect - Adj.*			Main effects on SIS-R Negative/Disorganised Adj.*			Trauma – dichotomised Interaction effect - Adj.*		
	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI
Psychosis	**2.10**	**0.031**	**.19 –4.01**	2.66	0.267	-2.06 -7.38	1.31	0.065	-.08-2.70	-1.08	0.544	-4.59-2.42
any Mental Illness	.76	0.079	-.09 -1.60	.72	0.059	-.07- 3.15	**.72**	**0.022**	**.10 –1.33**	.05	0.940	-1.28-1.38

*Adj. for gender, age, ethnicity (White, Non-White), IQ (For unadjusted values see Table Suppl.12, Appendix XVI). SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table 37: The interaction effects of familial risk for psychosis (including any mental illness of a first degree family member) and childhood trauma types on total schizotypy score

| | Genetic risk present | | | Genetic risk present | | |
| | Interaction effect (on SIS-R Total) Unadj. | | | Interaction effect (on SIS-R Total) Adj.* | | |
	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI
Household discord	0.87	0.526	-1.82 – 3.56	0.34	0.790	-2.20-2.89
Psychological abuse	2.49	0.313	-2.36 – 7.34	2.87	0.213	-1.66 - 7.42
Physical abuse	.28	0.858	-2.79 – 3.34	-.22	0.881	-3.14-2.70
Sexual abuse	-.42	0.806	-3.81 – 2.97	-.80	0.627	-3.81-2.97
Bullying	2.50	0.079	-.29 – 5.27	2.42	0.069	-2.89-5.27

Adj. for gender, age, ethnicity (White, Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

5.4 *Cannabis use (and interaction with Childhood Trauma) and Schizotypy association*

The role of cannabis use (lifetime and current use) as a moderator of the childhood trauma – schizotypy relationship was explored, following several studies implying its associations with either schizotypy levels or psychosis episodes.

A great majority of participants reported using cannabis at some time in their lives *(61.8%, Table 38)*. However, *15.1%* of the users only used/tried cannabis once or twice; weekly or more frequent usage was documented for *37.3%* of males and *21.2%* females. However, no statistically significant difference was observed for the overall frequencies of cannabis use between genders *(Pearson Chi^2=8.40, p=0.136)*. Thirty-five participants *(16.7%)* reported using cannabis in the 12 month period prior to the interview *(Table 39)*. The highest percentage of cannabis users *(N=51, 40.8%)* were using hash (cannabis resin), 37 *(29.6%)* imported herbal cannabis, 31 *(24.8%)* home-grown skunk or super skunk and 6 *(4.8%)* other types of cannabis.

Table 38: Frequency of lifetime cannabis use

	Total N (%)	Males* N (% within gender)	Females* N (% within gender)
No use	81 (38.2)	34 (36.2)	47 (39.8)
Only use it once/twice	32 (15.1)	10 (12.8)	22 (8.5)
A few times each year	21 (9.9)	7 (7.4)	14 (11.9)
A few times each month	18 (8.5)	8 (8.5)	10 (8.5)
(More than) once a week	38 (17.9)	23 (24.5)	15 (12.7)
Every day	22 (10.4)	12 (12.8)	10 (8.5)
Total	212 (100)	94 (100)	118 (100)

* no statistical significant difference between genders, Pearson Chi^2=8.40, p=0.136

Table 39: Frequency of current cannabis use and lifetime dependency

	Total N (%)		Males N (% within gender)		Females N (% within gender)	
	Yes	**No**	**Yes**	**No**	**Yes**	**No**
Current cannabis use	35 (16.7)	175 (83.3)	24* (25.8)	69 (74.2)	11* (9.4)	106 (90.6)
Lifetime dependency	14 (6.6)	198 (93.4)	8 (8.5)	86 (91.5)	6 (5.1)	112 (94.9)

*statistically significant difference, Pearson Chi^2=10.04, p=0.002

Lifetime cannabis use showed no association with total schizotypy levels *(adj. β=.19, p=0.767, Table 42)* and did not predict the positive *(adj. β=.04, p=0.922)* or negative/disorganised dimensions *(adj. β=.14, p=0.657)*. There was however an interaction observed between traumatic experiences in childhood and lifetime cannabis use for positive symptoms *(adj. β=1.77, p=0.046)*, but the association was weak and there was no main effect of cannabis use on positive schizotypal traits *(adj. β=.04, p=0.922)*. Comparing to current cannabis use, no interaction with childhood trauma in prediction of total schizotypy was observed, neither for positive *(for interaction adj. β=.85, p=0.464)* nor negative/disorganised dimensions individually *(for interaction adj. β=-27, p=0.763)*. The results showed that current cannabis use predicted positive schizotypy *(unadj. β=1.78, p=0.038)* but lost significance after adjusting for gender, age, ethnicity and IQ score *(adj. β=1.18, p=0.166)*.

Table 40: Frequency of lifetime cannabis use among those with/without childhood trauma

	Trauma absent* N (%)	Trauma present* N (%)
No cannabis use	39 (46.4)	41 (34.2)
Cannabis use	45 (53.6)	79 (65.8)
Total	84 (100)	120 (100)

statistically not significant difference, Pearson Chi^2 =3.12, p=0.077

Table 41: Types of cannabis among those with/without childhood trauma

	Trauma absent* N (%)	Trauma present* N (%)
Hash	17 (39.5)	32 (41.6)
Imported herbal cannabis	14 (32.6)	23 (29.9)
Home-grown skunk & super-skunk	7 (16.3)	21 (27.3)
Other	5 (11.6)	1 (1.3)
Total	43 (100)	77 (100)

statistically not significant difference, Pearson Chi^2 =7.41, p=0.060

Table 42: The interaction effects of cannabis use and childhood trauma on schizotypy

	Main effects Unadj.			Main effects Adj.*			Trauma – dichotomised Interaction effect Unadj.			Trauma – dichotomised Interaction effect Adj.*		
	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI
Lifetime cannabis use (dichotomous) and **SIS-R Total**	-.23	0.721	-1.53- 1.06	.19	0.767	-1.06-1.43	1.80	0.180	-.84 – 4.44	2.09	0.111	-.49 – 4.67
Lifetime cannabis use and **SIS-R Positive**	-.14	0.748	-1.02 - .74	.04	0.922	-.81 - .89	1.70	0.059	-.06 – 3.57	**1.77**	**0.046**	**.03 – 3.52**
Lifetime cannabis use and **SIS-R Negative/Disorganised**	-.09	0.781	-.74 - .56	.14	0.657	-.49 - .78	.09	0.890	-1.25 – 1.44	.32	0.643	-1.03 – 1.66
Current cannabis use (dichotomous) and **SIS-R Total**	**1.78**	**0.038**	**.90 –3.46**	1.18	0.166	-.49 –2.85	.34	0.848	-3.18 – 3.89	.58	0.734	-2.78 – 3.93
Current cannabis use and **SIS-R Positive**	1.11	0.057	-.03 – 2.25	.72	0.217	-.42- 1.86	.83	0.488	-1.53 – 3.21	.85	0.464	-1.43 – 3.12
Current cannabis use and **SIS-R Negative/Disorganised**	.67	0.122	-.18 -1.52	.46	0.296	-.40 –1.32	-.49	0.591	-2.30 – 1.31	-.27	0.763	-2.01 – 1.48

*Adj. for gender, age, ethnicity (White, Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table 43: The interaction effects of the age of cannabis use and childhood trauma on schizotypy

Early cannabis use (before 17)	Schizotypy score Unadj.			Schizotypy score Adj.*		
	OR	p-value	95% CI	OR	p-value	95% CI
Total schizotypy	2.22	0.075	.92-5.35	2.03	0.195	.79-5.26
Positive schizotypy	**2.28**	**0.049**	**1.00-5.17**	1.75	0.222	.71-4.32
Negative/Disorganised Schizotypy	1.00	0.998	.46-2.18	.77	0.578	.31-1.90

*Adj. for gender, age. OR, Odds Ratio. CI, Confidence Interval

There was a trend observed for early cannabis use *(before the age of 17)* and total schizotypy score *(OR=2.22, p=0.075, Table 43)*, and a positive association found for positive schizotypy scores *(OR=2.28, p=0.049)* compared to individuals who started using cannabis after the age of 17. However, there were no interaction effects observed between childhood trauma and early cannabis use on total schizotypy load *(OR=.86, p=0.910)*. Frequent cannabis also predicted higher schizotypy scores *(OR=2.46, p=0.015)*, especially the positive dimension. The differential effects of different types of cannabis used were also evidenced, as only *'home-grown skunk' and/or 'super-skunk'* had a significant impact on total schizotypy *(unadj. β=2.01, p=0.045, Table 45)* and the positive schizotypy dimension *(unadj. β=1.43 p=0.042)*.

Table 44: The association between the frequency of cannabis use and total and positive and negative/disorganised schizotypy

Frequent cannabis use	Schizotypy total score			Positive Schizotypy			Negative/Disorganised Schizotypy		
	OR	p-value	95% CI	OR	p-value	95% CI	OR	p-value	95% CI
(Unadj.)	**2.46**	**0.015**	**1.17- 4.56**	1.86	0.058	.98- 3.55	1.61	0.161	.82-3.12
(Adj.)	**2.07**	**0.040**	**1.03- 4.15***	1.91	0.058	.98- 3.71*	1.41	0.326	.71-2.78*

*Adj. for gender, age. OR, Odds Ratio. CI, Confidence Interval

Table 45: The comparison between the associations of type of cannabis used and schizotypy

	Schizotypy total score (Unadj).			Positive Schizotypy (Unadj.)			Negative/Disorganised Schizotypy (Unadj.)		
	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI
Imported herbal cannabis[a]	*-1.28*	*0.175*	*-.58-3.14*	*.57*	*0.393*	*-.74-1.87*	*.71*	*0.131*	*-.21- 1.64*
Home-grown skunk & super-skunk[a]	**2.01**	**0.045**	**.05-3.97**	**1.43**	**0.042**	**.05-2.81**	*.58*	*0.246*	*-.40-1.56*
Other[a]	*-.15*	*0.938*	*-3.87-3.57*	*-.23*	*0.859*	*-2.85-2.38*	*.09*	*0.925*	*-1.77-1.95*

[a] *base category: hash (cannabis resin). CI, Confidence Interval*

5.5 *Mediation effects / Pathways from Childhood Trauma to Schizotypy*

The effect of Dissociation on childhood abuse and schizotypy association

With only *5 (2.4%)* respondents scoring on dissociation/depersonalisation item for these experiences in the past 7 days, dissociation was not included in the mediation analyses. Despite this limited number, it was observed that recent dissociation experiences predicted positive schizotypy *(unadj. β=3.23, p=0.022, Table 48).*

Table 46: The frequency of lifetime dissociation symptoms among those with/without childhood trauma (dissociation includes the SIS-R item on lifetime dissociation along with the 'recent dissociation' item)

	Trauma absent* N (%)	Trauma present* N (%)	Total N
Dissociation - absent	77 (91.7)	106 (86.9)	183
Dissociation - present (mild, moderate, severe)	7 (8.3)	16 (13.1)	23
Total	84 (100)	122 (100)	206

*statistically not significant difference, Pearson Chi^2=1.15, p=0.284

Table 47: The frequency of recent (past 7 days) dissociation symptoms among those with/without childhood trauma

	Trauma absent* N (%)	Trauma present* N (%)	Total N
Dissociation - absent	83 (98.8)	118 (96.7)	201
Dissociation - present	1 (1.2)	4 (3.3)	5
Total	84 (100)	122 (100)	206

*statistically not significant difference, Fisher's exact test =.65, p=0.321

Table 48: The association between recent dissociation/derealisation symptoms and total, positive and negative schizotypy

	Dissociation/Derealisation		
	β coeff.	p-value	95% CI
SIS-R Total	3.31	0.114	-.80-7.42
SIS-R Positive	**3.23**	**0.022**	**.46-5.99**
SIS-R Negative	.08	0.940	-1.99-2.15

Unadjusted scores. SIS-R, Structured Interview for Schizotypy Revised.
CI, Confidence Interval

The effect of Cognitive Schemas and Depression score on Schizotypy

Mean 'negative beliefs about others' subscale of the *Brief Core Schema Scales* was 3.67 (SD=5.06), with females scoring slightly higher than males *(mean 3.96 for female, 3.12 for male, difference not significant, Mann-Whitney test=-.96, p=0.335)*. Besides, females also reported slightly more negative beliefs about themselves *(mean for female 1.33, for male 1.18, difference not significant, Mann-Whitney test=-1.14, p=0.253)* and more symptoms of depression *(mean for female 4.33, for male 3.91, Mann-Whitney test=-1.88, p=0.060) (Table 49)*.

The Spearman rho coefficient between depression score and 'negative beliefs about others' was *0.31*, between depression and 'negative self-beliefs' was *0.37* and between 'negative-others' and 'negative-self' beliefs was *0.37*.

Table 49: Mean scores on negative beliefs about self/others and the depression scale

	Mean (SD) (range)	Males Mean (SD) (95% CI)	Females Mean (SD) (95% CI)
Negative beliefs - others	3.67 (5.06) (0 – 24)	3.12 (.50) (2.33-4.31)	3.96 (.48) (3.01-4.90)
Negative beliefs - self	1.26 (2.43) (0 – 19)	1.18 (.27) (.64-1.71)	1.33 (.21) (.92-1.74)
Depression scale	3.91 (4.06) (0 – 24)	3.38 (.38)* (2.63-4.13)	4.33 (.39)* (3.55-5.11)

*approaching **significance** Mann-Whitney test=-1.88, p=0.060. SD, Standard Deviation. CI, Confidence Interval

Table 50: Mean scores on negative beliefs about self/others and the depression scale among those with/without childhood trauma

	Trauma absent Mean SD	Trauma present Mean SD
Negative beliefs - others	2.94 (4.15) [a]	4.29 (5.63) [a]
Negative beliefs - self	.84 (1.67) [b]	1.54 (2.82) [b]
Depression scale	2.71 (2.54) [c]	4.76 (4.66) [c]

[a] Mann-Whitney test=-1.95, p=0.051; b, Mann-Whitney test=-1.83, p=0.067,
[c] Mann-Whitney test=-3.18, p=0.001. SD, Standard Deviation

The results showed a positive association between negative beliefs about others and total schizotypy (adj. *β=.32, p<0.001, Table 51)*. Besides, a stronger association between negative beliefs about self and total schizotypy (adj. *β=.66, p<0.001)* was also observed. Not surprisingly, a similar association was found between depression score and schizotypal traits (adj. *β=.45, p<0.001)*.

Table 51: The effects of the negative beliefs/ depression scores and childhood trauma on schizotypy scores

	Main effects on SIS-R Total			Main effects on SIS-R Total		
	Unadj.			Adj.*		
	β coeff.	p-value	95% CI	β coeff.	p-value	95% CI
Negative beliefs – about others	.39	<0.001	.28 – .50	.32	<0.001	.21- .44
Negative beliefs – about self	.75	<0.001	.51 – .99	.66	<0.001	.43 - .89
Depression score	.47	<0.001	.33 - .61	.45	<0.001	.31 - .59

*Adj. for gender, age, ethnicity (White/Non-White), employment status, IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table 52: The effects of the negative beliefs/ depression scores and childhood trauma on positive and negative/disorganised schizotypy

	Main effects on Positive SIS-R Adj.*			Main effects on Negative/Disorganised SIS-R Adj.*		
	β coefficient	p-value	95% CI	β coefficient	p-value	95% CI
Negative beliefs – about others	.20	<0.001	.12- .28	.12	<0.001	.06- .18
Negative beliefs – about self	.39	<0.001	.23 - .55	.27	<0.001	.15 - .39
Depression score	.28	<0.001	.18 - .37	.17	<0.001	.10 - .25

*Adj. for gender, age, ethnicity (White/Non-White), employment status, IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Mediation effects (from Childhood Trauma to Schizotypy) – of Negative beliefs about Others/Self, Depression score, Cannabis use and recent Life events

The direct and indirect pathways between different types of childhood trauma and schizotypy are presented in *Table 53* along with graphical representations in *Figures 17 to 22*. The table shows the odds ratio (OR), p-values and 95% confidence intervals along with the percentage that stands for the proportion of the total effect of particular trauma type on schizotypy load, that is indirectly explained via all of the mediators or each of the mediators individually.

Looking at the significant mediation effects (using KHB mediation analysis) between trauma and schizotypy, the following was found: household discord and schizotypy was partially mediated by depression score *(indirect effect OR=1.20, p=0.076, explained 25% of the association)*; psychological abuse and schizotypy was partially explained by negative beliefs about self *(indirect effect OR=1.48, p=0.037, explained 26%)* and depression score *(indirect effect OR=1.65, p=0.015, explained 32%)*; physical abuse was mediated by negative beliefs about others *(indirect effect OR=1.51, p=0.008, explained 23%)* and negative beliefs about others *(indirect effect OR=1.35 p=0.029, explained 16%)* as well *as* depression score *(indirect effect OR=1.39, p=0.018, explained 19%)*; mediators of sexual abuse and schizotypy association was negative beliefs about self *(indirect effect OR=1.33, p=0.038, explained 30%)* and depression score *(indirect effect OR=1.27, p=0.063, explained 27%)* and bullying was associated with schizotypal load via depression score *(trend in indirect effect OR=1.20, p=0.084, explained 21%)*.

Cannabis use and life events were not significant mediators between any type of trauma and schizotypy association.

Table 53: Associations between different types of childhood trauma and schizotypy total scores, split into total, direct and indirect effects/pathways via possible mediators (including of all mediators together and each individual mediator on its own)

Type of victimisation	Top 20% of schizotypy Unadj. OR (p-value) CI				Top 20% of schizotypy Adj.* OR (p-value) CI			
	Total effect	*Direct effect*	*Indirect effect*	*% mediated*	*Total effect*	*Direct effect*	*Indirect effect*	*% mediated*
Total trauma								
All mediators	OR=2.01 p<0.001 (1.43-2.86)	OR=1.59 p=0.010 (1.11-2.25)	OR=1.27 p=0.006 (1.07-1.51)	34%	OR=1.83 p=0.002 (1.26-2.66)	OR=1.41 p=0.077 (.96-2.08)	OR=1.29 p=0.009 (1.07-1.57)	43%
Negative beliefs - Others	OR=1.94 p<0.001 (1.40-2.71)	OR=1.71 p=0.001 (1.24-2.38)	OR=1.13 p=0.025 (1.01-1.26)	19%	OR=1.78 p=0.002 (1.23-2.56)	OR=1.60 p=0.010 (1.12-2.30)	OR=1.11 p=0.075 (.99-1.24)	18%
Negative beliefs - Self	OR=2.00 p<0.001 (1.42-2.82)	OR=1.78 p=0.001 (1.28-2.49)	OR=1.12 p=0.028 (1.01-1.25)	17%	OR=1.82 p=0.001 (1.27-2.61)	OR=1.61 p=0.008 (1.13-2.29)	OR=1.13 p=0.037 (1.01-1.27)	20%
Depression score	OR=1.95 p<0.001 (1.39-2.72)	OR=1.69 p=0.002 (1.21-2.36)	OR=1.15 p=0.015 (1.03-1.29)	21%	OR=1.72 p=0.003 (1.21-2.46)	OR=1.46 p=0.039 (1.02-2.09)	OR=1.18 p=0.013 (1.03-1.34)	30%
Cannabis use	OR=1.88 p<0.001 (1.37-2.59)	OR=.99 p<0.001 (1.36-2.59)	OR=.99 p=0.861 (.95-1.04)	0%	OR=1.68 p=0.003 (1.20-2.37)	OR=1.69 p=0.003 (1.19-2.38)	OR=.99 p=0.937 (.95-1.05)	0%

Table 53 Cont'd

Life events	OR=1.87	OR=1.87	OR=1.00	0.3%	OR=1.70	OR=1.67	OR=1.01	2%
	p<0.001	p<0.001	p=0.770		p=0.003	p=0.003	p=0.621	
	(1.36-2.57)	(1.34-2.56)	(.99-1.01)		(1.20-2.40)	(1.19-2.38)	(.97-1.04)	
Household Discord								
All mediators	OR=2.16	OR=1.55	OR=1.39	43%	OR=2.18	OR=1.64	OR=1.33	37%
	p=0.041	p=0.265	p=0.069		p=0.066	p=0.269	p=0.151	
	(1.03-4.50)	(.72-3.35)	(.97-1.98)		(.95-5.01)	(.68-3.92)	(.90-1.97)	
Negative beliefs - Others	OR=2.17	OR=1.83	OR=1.82	22%	OR=2.27	OR=2.04	OR=1.11	13%
	p=0.032	p=0.091	p=0.135		p=0.043	p=0.075	p=0.331	
	(1.07-4.41)	(.91-3.71)	(.95-1.48)		(1.03-5.02)	(.93-4.50)	(.90-1.37)	
Negative beliefs - Self	OR=2.17	OR=1.97	OR=1.10	39%	OR=2.28	OR=2.08	OR=1.10	11%
	p=0.030	p=0.056	p=0.334		p=0.039	p=0.065	p=0.411	
	(1.08-4.36)	(.98-3.94)	(.90-1.34)		(1.04-4.99)	(.95-4.54)	(.88-1.36)	
Depression score	OR=2.10	OR=1.75	OR=1.20	25%	OR=2.05	OR=1.69	OR=1.21	27%
	p=0.036	p=0.115	p=0.076		p=0.074	p=0.194	p=0.106	
	(1.05-4.20)	(.87-3.50)	(.98-1.47)		(.93-4.52)	(.76-3.73)	(.96-1.53)	
Cannabis use	OR=2.05	OR=2.09	OR=.98	0%	OR=2.07	OR=2.10	OR=.99	0%
	p=0.033	p=0.031	p=0.720		p=0.051	p=0.051	p=0.844	
	(1.06-3.96)	(1.07-4.11)	(.86-1.11)		(.99-4.31)	(.99-4.43)	(.86-1.13)	
Life events	OR=2.03	OR=2.05	OR=.99	0%	OR=2.09	OR=2.17	OR=.96	0%
	p=0.036	p=0.033	p=0.654		p=0.049	p=0.039	p=0.388	
	(1.05-3.92)	(1.06-3.97)	(.95-1.03)		(1.00-4.35)	(1.04-4.55)	(.87-1.05)	

Table 53 Cont'd

Psychological abuse								
All mediators	OR=6.82 p=0.002 (2.01-23.20)	OR=3.18 p=0.058 (.93-10.10)	OR=2.15 p=0.064 (.95-4.83)	40%	OR=6.10 p=0.008 (1.62-23.04)	OR=2.89 p=0.114 (.77-10.74)	OR=2.11 p=0.070 (.94-4.76)	41%
Negative beliefs - Others	OR=5.12 p=0.004 (1.71-15.38)	OR=3.85 p=0.015 (1.30-11.36)	OR=1.33 p=0.145 (.90-1.95)	17%	OR=4.97 p=0.010 (1.47-16.78)	OR=3.72 p=0.031 (1.12-12.33)	OR=1.33 p=0.145 (.90-1.97)	18%
Negative beliefs - Self	OR=4.47 p=0.005 (1.56-12.82)	OR=3.02 p=0.040 (1.05-8.69)	OR=1.48 p=0.037 (1.02-2.13)	26%	OR=4.62 p=0.011 (1.42-14.97)	OR=3.11 p=0.057 (.98-10.00)	OR=1.48 p=0.053 (.99-2.21)	26%
Depression score	OR=4.82 p=0.004 (1.64-14.16)	OR=2.92 p=0.048 (1.01-8.49)	OR=1.65 p=0.015 (1.10-2.46)	32%	OR=4.74 p=0.011 (1.42-15.84)	OR=2.76 p=0.096 (.84-9.10)	OR=1.72 p=0.016 (1.11-2.67)	35%
Cannabis use	OR=4.56 p=0.003 (1.65-12.60)	OR=4.56 p=0.003 (1.65-12.62)	OR=.99 p=0.916 (.98-1.02)	0%	OR=4.21 p=0.011 (1.38-12.82)	OR=4.24 p=0.011 (1.39-12.94)	OR=.99 p=0.778 (.94-1.04)	0%
Life events	OR=4.09 p=0.005 (1.52-11.03)	OR=4.19 p=0.005 (1.55-11.34)	OR=.98 p=0.604 (.89-1.07)	0%	OR=4.05 p=0.014 (1.32-12.38)	OR=4.21 p=0.012 (1.37-12.95)	OR=.96 p=0.553 (.84-1.09)	0%
Physical abuse								
All mediators	OR=7.20 p<0.001 (3.08-16.81)	OR=3.74 p=0.002 (1.64-8.55)	OR=1.92 p=0.009 (1.78-3.14)	33%	OR=4.00 p=0.004 (1.55-10.29)	OR=2.06 p=0.135 (.80-5.30)	OR=1.94 p=0.012 (1.15-3.27)	48%

Table 53 Cont'd

Negative beliefs - Others	OR=6.03 p<0.001 (2.73-13.30)	OR=3.99 p<0.001 (1.83-8.68)	OR=1.51 p=0.008 (1.12-2.05)	23%	OR=3.54 p=0.006 (1.44-8.72)	OR=2.38 p=0.057 (.97-5.83)	OR=1.49 p=0.018 (1.07-2.06)	31%
Negative beliefs - Self	OR=6.41 p=0.008 (2.90-14.18)	OR=1.76 p=0.008 (2.20-10.29)	OR=1.35 p=0.029 (1.03-1.76)	16%	OR=3.79 p=0.003 (1.58-9.09)	OR=2.92 p=0.015 (1.23-6.93)	OR=1.30 p=0.078 (.97-1.73)	19%
Depression score	OR=5.89 p=0.008 (2.68-12.90)	OR=4.22 p<0.001 (1.95-9.12)	OR=1.39 p=0.018 (1.06-1.84)	19%	OR=3.46 p=0.005 (1.44-8.29)	OR=2.37 p=0.053 (.99-5.69)	OR=1.46 p=0.021 (1.06-2.01)	30%
Cannabis use	OR=5.25 p<0.001 (2.48-10.67)	OR=5.14 p<0.001 (2.48-10.67)	OR=.99 p=0.934 (.99-1.01)	0%	OR=3.14 p=0.007 (1.37-7.16)	OR=3.13 p=0.007 (1.37-7.15)	OR=1.00 p=0.898 (.98-1.02)	0.1%
Life events	OR=5.24 p<0.001 (2.52-10.91)	OR=5.38 p<0.001 (2.57-11.24)	OR=.97 p=0.520 (.90-1.05)	0%	OR=3.30 p=0.004 (1.46-7.48)	OR=3.40 p=0.003 (1.50-7.73)	OR=.97 p=0.542 (.88-1.07)	0%
Sexual abuse								
All mediators	OR=2.71 p=0.025 (1.13-6.49)	OR=1.83 p=0.181 (.75-4.45)	OR=1.48 p=0.106 (.92-2.37)	39%	OR=2.93 p=0.035 (1.08-7.93)	OR=1.85 p=0.227 (.68-5.05)	OR=1.58 p=0.068 (.97-2.58)	42%
Negative beliefs - Others	OR=2.49 p=0.030 (1.09-5.68)	OR=2.04 p=0.089 (.90-4.64)	OR=1.22 p=0.163 (.92-1.61)	22%	OR=2.48 p=0.060 (.96-6.38)	OR=2.11 p=0.119 (.82-5.42)	OR=1.17 p=0.280 (.88-1.56)	17%

Table 53 Cont'd

Negative beliefs - Self	OR=2.58 p=0.026 (1.12-5.95)	OR=1.94 p=0.118 (.85-4.43)	OR=1.33 p=0.038 (1.02-1.75)	30%	OR=2.86 p=0.026 (1.13-7.23)	OR=2.16 p=0.099 (.86-5.39)	OR=1.32 p=0.063 (.98-1.78)	27%
Depression score	OR=2.38 p=0.036 (1.06-5.36)	OR=1.88 p=0.128 (.83-4.24)	OR=1.27 p=0.063 (.98-1.63)	27%	OR=2.47 p=0.059 (.67-6.32)	OR=201 p=0.144 (.79-5.13)	OR=1.23 p=0.154 (.93-1.62)	23%
Cannabis use	OR=2.23 p=0.042 (1.03-4.84)	OR=2.22 p=0.043 (1.02-4.83)	OR=1.00 p=0.836 (.97-1.04)	0.4%	OR=2.23 p=0.074 (.93-5.36)	OR=2.22 p=0.076 (.92-5.34)	OR=1.00 p=0.826 (.97-1.04)	0.5%
Life events	OR=2.26 p=0.039 (1.04-4.91)	OR=2.26 p=0.046 (1.01-5.03)	OR=1.00 p=0.987 (.82-1.23)	0.3%	OR=2.30 p=0.061 (.96-5.49)	OR=2.08 p=0.109 (.85-5.07)	OR=1.11 p=0.410 (.87-1.41)	12%
Bullying								
All mediators	OR=2.65 p=0.011 (1.25-5.62)	OR=2.19 p=0.042 (1.03-4.66)	OR=1.21 p=0.270 (.86-1.69)	19%	OR=2.35 p=0.041 (1.04-5.35)	OR=1.76 p=0.185 (.76-4.07)	OR=1.33 p=0.167 (.89-2.01)	34%
Negative beliefs - Others	OR=2.55 p=0.011 (1.24-5.24)	OR=2.57 p=0.010 (1.25-5.29)	OR=.99 p=0.929 (.79-1.24)	0%	OR=2.26 p=0.045 (1.01-5.02)	OR=2.39 p=0.033 (1.07-5.32)	OR=.95 p=0.653 (.74-1.20)	0%
Negative beliefs - Self	OR=2.42 p=0.014 (1.19-4.91)	OR=2.09 p=0.039 (1.04-4.22)	OR=1.16 p=0.164 (.94-1.42)	16%	OR=2.24 p=0.041 (1.03-4.84)	OR=1.83 p=0.122 (.85-3.95)	OR=1.22 p=0.098 (.96-1.54)	25%
Depression score	OR=2.41	OR=2.00	OR=1.20	21%	OR=2.08	OR=1.58	OR=1.32	38%

Table 53 Cont'd

	p=0.015	p=0.054	p=0.084		p=0.062	p=0.248	p=0.035	
	(1.18-4.88)	(.99-4.03)	(.97-1.49)		(.96-4.50)	(.73-3.44)	(1.02-1.70)	
Cannabis use	OR=2.29	OR=2.29	OR=.99	0%	OR=2.08	OR=2.08	OR=1.00	0.1%
	p=0.015	p=0.015	p=0.935		p=0.053	p=0.053	p=0.942	
	(1.17-4.46)	(1.17-4.48)	(.95-1.04)		(.99-4.38)	(.99-4.38)	(.97-1.03)	
Life events	OR=2.26	OR=2.24	OR=1.01	1%	OR=2.03	OR=1.92	OR=1.06	8%
	p=0.017	p=0.018	p=0.847		p=0.062	p=0.088	p=0.379	
	(1.16-4.40)	(1.14-4.38)	(.93-1.09)		(.97-4.25)	(.91-4.04)	(.94-1.20)	

*Adj. for gender, age, ethnicity (White, Non-White), familial risk for psychosis. OR, Odds Ratio. CI, Confidence Interval (For the mediators between childhood trauma and schizotypy for females and males separately see Suppl. Table 15 & 16, Appendix XVI)
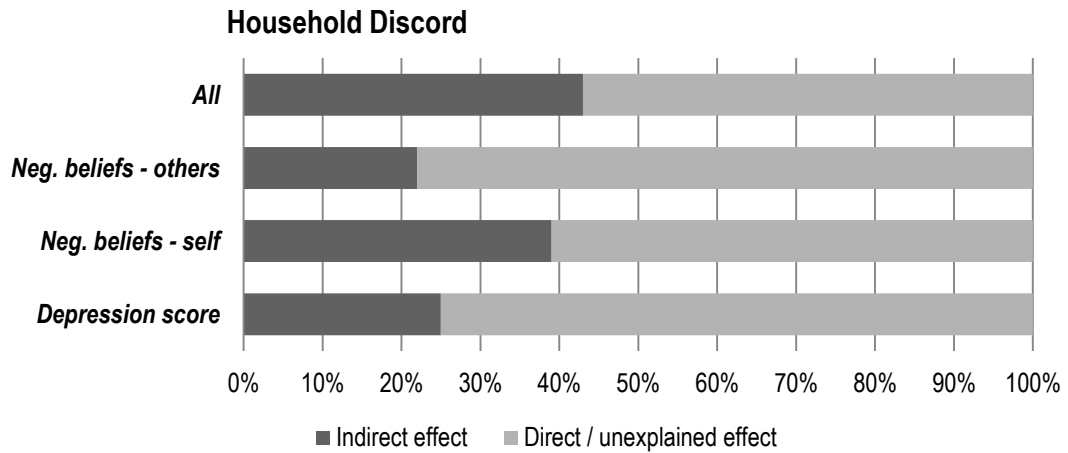
Household Discord

Figure 17: Mediation (in percentage) of the effect of household discord on schizotypy

Psychological Abuse
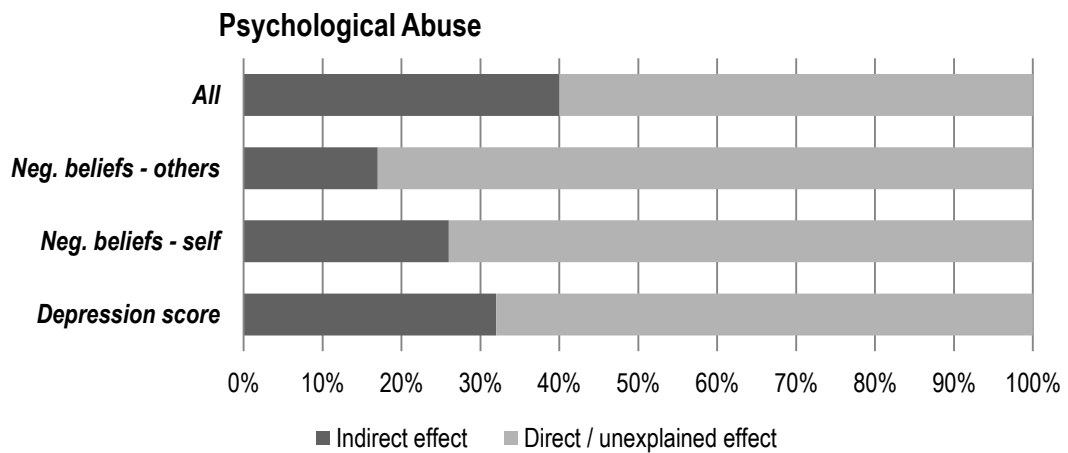
Figure 18: Mediation (in percentage) of the effect of psychological abuse on schizotypy
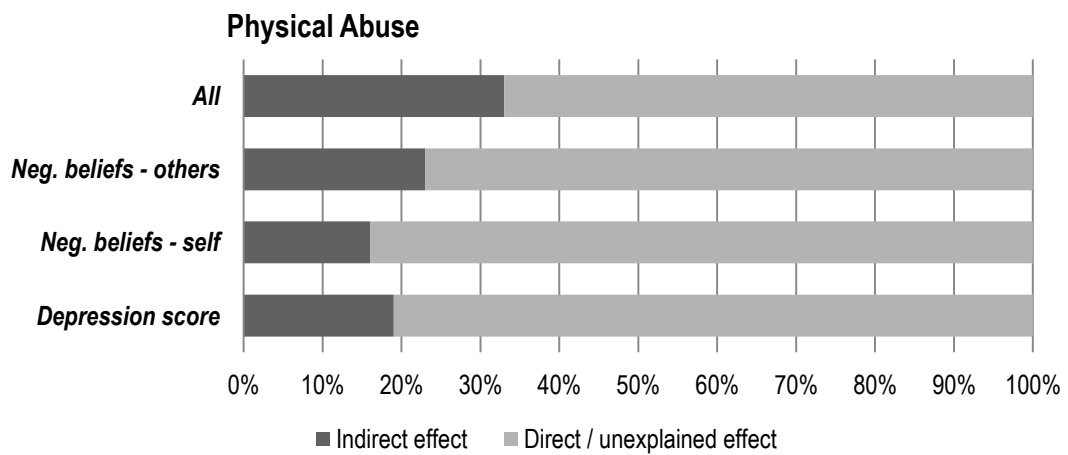
Physical Abuse

Figure 19: Mediation (in percentage) of the effect of physical abuse on schizotypy
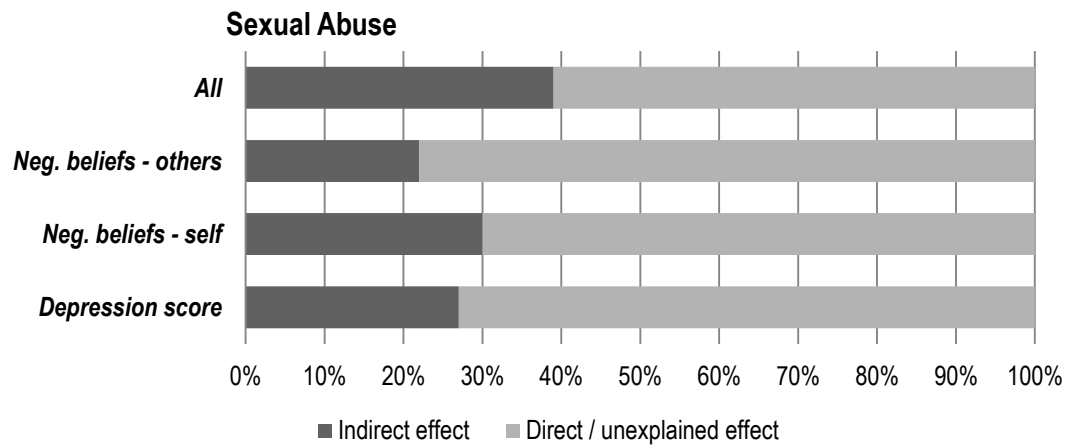
Sexual Abuse

Figure 20: Mediation (in percentage) of the effect of sexual abuse on schizotypy
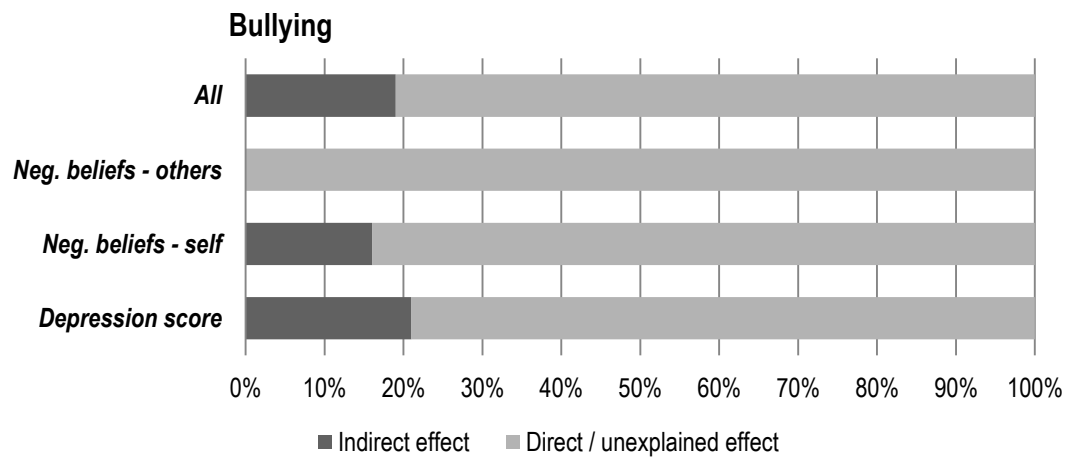
Bullying

Figure 21: Mediation (in percentage) of the effect of bullying on schizotypy
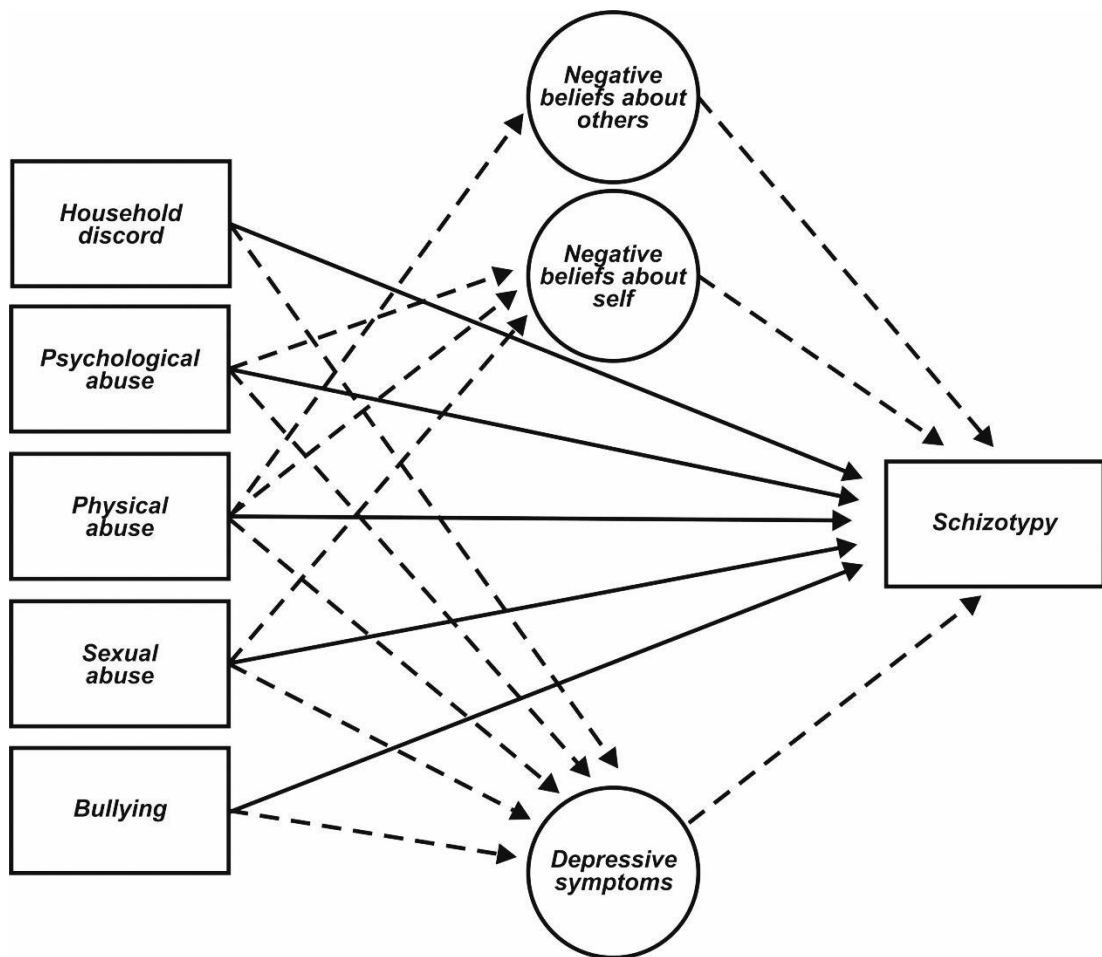
Figure 22: Conceptual path diagram showing significant associations between childhood trauma and schizotypy, direct paths are indicated by solid arrows and indirect paths by dashed arrows.

CHAPTER 6
Discussion

Chapter 6 Discussion

Main aims of the chapter

- To summarise the main findings in relation to the research questions *(section 6.1)*;

- Provide a comprehensive interpretation of the findings and the clinical and research implications *(section 6.2)*;

- Supply an overview of the conclusions in the light of study's strengths and limitations *(section 6.3)*.

6.1 *Overview of the main findings*

The section will present the overview of the main findings in relation to the research questions/hypotheses set out in this thesis. The study findings according to each of the hypotheses and the indication of whether the hypothesis was supported or not are presented in *Table 54*. This will be followed by the interpretation of the findings and link to the existing literature *(section 6.2).*

Hypothesis 1:

The study added to the existing literature by showing the association between childhood trauma and schizotypy in a dose-response fashion. There was a linear relationship observed for psychological abuse, physical abuse, sexual abuse and bullying, which remained significant after adjusting for possible confounders (gender, age, ethnicity, employment, religion, IQ). Household discord was not associated with a linear increase in schizotypy, however it did significantly predict the top *20%* and top *10%* of the schizotypy scorers (approximately 2-fold increase). Among other forms of traumatic experiences (death of a parent, separation from a biological parent) only separation from a biological father showed a trend in association with schizotypal traits, but lost significance after adjusting for confounders. When focusing on the top *20%* and top *10%* of the schizotypy scores it was found that psychological and physical abuse showed the most robust association with schizotypal symptomatology. This was observed for the positive dimension and the negative/disorganised dimension. Specificity of effects of distinct trauma types was mainly evidenced for the positive dimension (no association was found between sexual abuse and bullying and positive schizotypy) with a less clear distinction among trauma types seen for the negative/disorganised dimension. Overall, it was the severe and more chronic forms of traumatic experiences that significantly predicted the total schizotypy load, with the only exception being psychological and sexual abuse. Equally, early trauma especially (occurring before

the age of 12) demonstrated a particularly strong association with schizotypy however for bullying and sexual abuse adolescent victimisation proved more harmful. The gender differences in association between childhood trauma and schizotypy were also observed as sexual abuse predicted schizotypy for females only, whereas physical abuse had much stronger association with schizotypy for males. The association between childhood trauma and schizotypy remained significant after accounting for familial risk for psychosis, therefore childhood trauma appears to stand as a risk factor for developing schizotypal traits regardless of pre-existing genetic vulnerabilities.

Table 54: Main study findings according to each of the hypotheses and the indication of whether the hypothesis was supported or not

Hypotheses	*Main findings*	*Hypothesis supported?*
Those reporting more childhood trauma will score higher on a schizotypy scale.	• *Childhood trauma total score was associated with schizotypy total score - a dose-response relationship* • *All main* types of trauma measured predicted schizotypy, mainly in a linear fashion* • *Associations were observed between childhood trauma and the positive and negative/disorganised schizotypy dimensions* • *The strongest association with schizotypy was evidenced for psychological and physical abuse* • *More severe and chronic abuse/victimisation forms particularly predicted schizotypy*	***Yes***
Schizotypy will be higher in those exposed to both childhood and adulthood traumatic experiences than in those with childhood trauma only.	• *Recent traumatic events were associated with total schizotypy score, after adjusting for confounders these were significant for positive schizotypy only* • *The interaction effects between childhood trauma and life events in relation to schizotypy (total and negative/disorganised dimension) were only observed for intrusive events*	***Partially***
Childhood trauma will lead to development of negative beliefs about self/others and depression which will then increase the schizotypy levels.	• *Childhood trauma was associated with negative beliefs about others/self and depression score* • *Negative belief about others/self and depression score all showed an association with schizotypy* • *All three factors (negative belief about others/self and depression*	***Yes***

Table 54 Cont'd

	score) explained (mediated) a significant proportion of the childhood trauma-schizotypy association	
Individuals with higher familial risk for psychosis and exposure to childhood trauma will display higher schizotypy scores than those without familial risk.	• *Familial risk for psychosis showed a strong association with total schizotypy load, especially the positive dimension* • *An interaction effect was observed between childhood trauma and familial risk for psychosis and positive schizotypy (but not for the total schizotypy or the negative/disorganised schizotypy dimension)*	***Partially***
Cannabis use will partially account for the association between childhood adversity and schizotypy; cannabis use will either mediate the childhood trauma – schizotypy association or interact with childhood trauma to increase the schizotypy levels.	• *Cannabis use did not have a mediating role between childhood trauma and schizotypy* • *A significant interaction effect was observed between childhood trauma and cannabis use and positive schizotypy (but only a weak association and there was no main effect of cannabis use on positive schizotypy)* • *Frequent cannabis use predicted schizotypy, especially home-grown skunk and super-skunk among types of cannabis*	***No***

*household discord, psychological abuse, physical abuse, sexual abuse and bullying, BUT not death of a parent or separation from a biological parent

Hypothesis 2:

Recent life events were found to be significantly associated with schizotypy, with no additional effect of recent life difficulties. Especially the intrusive events (but not independent events) showed the strongest association the with positive schizotypy dimension. An interaction effect between childhood trauma and intrusive life events only further amplified this association but interestingly mainly impacted on the negative/disorganised schizotypy dimension. Life events did not mediate the association between any type of trauma and total schizotypy.

Hypothesis 3:

Individuals who reported childhood trauma experienced significantly more depression symptoms, more negative beliefs about others and more negative beliefs about self (approaching significance) compared to those without traumatic experiences. All three variables (negative beliefs about self/others and depression score) showed an association with total schizotypy score. Mediation analyses revealed that depression stands as a mediator between all types of trauma (household discord, psychological abuse, physical abuse, sexual abuse, bullying) and schizotypal symptomatology. In addition, negative beliefs about self explained a significant proportion of the relationship between psychological, physical and sexual abuse and schizotypy and negative beliefs about others underlied/mediated the physical abuse – schizotypy association.

Hypothesis 4:

Results supported a strong association between familial risk of psychosis and schizotypy, increasing with the narrow definition of familial risk for psychosis (first-degree relatives with present/past episode of psychosis). The effects of proxy

genetic vulnerabilities were significant for the positive and negative/disorganised schizotypy dimension, but especially strong for the positive dimension. Similarly, the interaction effects between childhood abuse and familial risk ('broad definition') on development of schizotypy were observed for the positive but not the negative/disorganised dimension or the total schizotypy score.

Hypothesis 5:

Lifetime cannabis use had no association with total schizotypy levels. In contrast, the trend was observed for current cannabis use and positive schizotypy but lost significance after adjusting for confounders. The interaction observed between childhood trauma and lifetime cannabis use was significant for positive schizotypy (but was only a weak association and there was no main effect of cannabis on positive schizotypy). Lifetime cannabis use was not a significant mediator between any type of trauma measured and total schizotypy score.

6.2 Study findings and link to existing literature

Prevalence of childhood trauma

Overall, the prevalence of childhood trauma in the present study resembles reports from other community samples of the general population. The highest percentage of respondents from the present sample experienced household discord *(35.8%)*, similar to previous findings *(40% reported household dysfunction (Afifi et al. 2011))*. Also, the *NSPCC*[16] report (Radford et al. 2011) on child abuse and neglect in the UK showed that *12%* had been exposed to domestic violence between adults before the age of 11 and *17.5%* between 11 and 17 *(Table 55)*. Furthermore, the report suggested that sexual abuse experienced before the age of 11 was found in *1.2%* and sexual abuse between 11 and 17 in *16.5%* of the sample (Radford et al. 2011), comparable to *17.8%* from the present study when all levels of severity were included. Likewise, a USA study using general population sample reported slightly higher prevalence of sexual abuse *(21.6%)*, but identified a similar percentage of individuals with physical abuse *(20.6% (Edwards et al. 2003), 20.4% in the present sample)*. The present data identified some gender differences in frequencies of specific trauma types e.g. higher rates of sexual abuse for females and a higher percentage of bullying in males which ties in with a USA sample even though looking overall the higher prevalence of trauma was reported *(sexual abuse females - 32%, sexual abuse males - 14%, physical abuse females - 20%, physical males - 22% (Briere and Elliott 2003))*. However, studies worldwide showed that approximately *20%* of women and *5%* to *10%* of men experienced childhood sexual abuse (Butchart et al. 2006), more consistent with the present sample. Measuring the peer victimisation on the other hand has led to more mixed results, with the prevalence varying between *40%* to *80%*, with *15%-20%* suffering more severe and more frequent forms of victimisation (Juvonen and Graham 2001) especially

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[16] *National Society for the Prevention of Cruelty to Children, UK*

common in urban areas (Wang et al. 2009). Mainly because of the problem of its definition, the prevalence of psychological abuse is most difficult to evaluate, most likely leading to an underestimation of this type of trauma. Also, psychological trauma correlates with other forms of maltreatment, especially neglect, antipathy from parents and sexual abuse (Bifulco et al. 2002). Furthermore, emotional abuse and psychological abuse are often used interchangeably. Nonetheless, studies assessing psychological abuse reported the range between *113* to *257* per *1000* children (Fortin and Chamberland 1995). In a highly selected all female sample (adult depressive vulnerability), *16%* experienced psychological abuse at any severity (some, moderate, marked) (Moran et al. 2002) whereas in a control group *4%* of respondents reported this type of trauma (Moran et al. 2002), comparable to the present study.

Table 55: The prevalence of trauma in the present study compared to UK study (NSPCC)

	Household discord		Psychological abuse		Physical abuse		Sexual abuse		Bullying	
	Present (%)		*Present (%)*		*Present (%)*		*Present (%)*		*Present (%)*	
Present Study *(up to 18)*	*35.8*		*4.3*		*20.4*		*7.2*		*32.2*	
UK study (2011)* *(below 11) vs (11-17)*	*(12.0)*	*(17.5)*	*(-)*	*(-)*	*(1.3)*	*(6.9)*	*(1.2)*	*(16.5)*	*(28.0)*	*(59.5)*

*NSPCC, Radford et al. (2011) – household discord included domestic violence only
(Note: the percentages are a brief guidance only, as a range of different trauma definitions and data collection procedures make comparison of data extremely difficult)

The association between childhood trauma (and distinct trauma types) and schizotypy

This study provides robust support to the primary hypothesis that there is an association between childhood trauma and schizotypal traits. This parallels the existing empirical evidence showing the strong association between childhood trauma and total as well as both positive and negative/disorganised schizotypy dimensions (Afifi et al. 2011;Lentz et al. 2010;Lobbestael et al. 2010). Equally, previous studies provided support for different types of childhood abuse (emotional,

physical, sexual) as well as household discord *(in existing schizotypy literature mainly conceptualised as 'violence between parents' (Afifi et al. 2011;Lentz et al. 2010))* and bullying (Mynard and Joseph 1997;Raine et al. 2011) all predicting the total schizotypy scores. Even though the differential effects of specific trauma types (qualitative in addition to quantitative differences (Berenbaum et al. 2008;Myin-Germeys et al. 2011)) have been reported, this support has not been consistent (Afifi et al. 2011). Current findings provided further evidence that all types of childhood trauma measured showed an association with schizotypy, mainly in a linear fashion, with the household discord as the only exception (which showed a nonlinear association and predicted top *20%* and top *10%* of schizotypy scorers). The disproportionate increase of schizotypy when the predictor is household discord does not necessarily imply discontinuity as *"what is discontinuous is the degree of extralinearity associated with the increase at or below the disorder spectrum"* (Binbay et al. 2012 p.1000) (reflecting the quasi-dimensional approach to psychosis). Similar to trauma-schizotypy relationship, strong linear associations were observed between all types of trauma (apart from household discord) and attenuated psychotic symptoms (as measured with CAPE).

Nevertheless, when looking at the strength of these associations, differences between trauma types emerged, with psychological abuse and physical abuse having particularly robust effects on schizotypal symptomatology (approximately *7 times* and *6 times* more likely to predict the highest *10%* on the schizotypy scale, respectively). These findings confirm previous propositions that these types of trauma in particular are the strongest predictors of schizotypy load *(e.g. emotional abuse (Powers et al. 2011), physical abuse (Steel et al. 2009))* but contrary to others which emphasized the highest association with either sexual abuse (Afifi et al. 2011) or neglect (Rossler et al. 2007). The discrepancies can also be attributed to some major differences in the study designs and methodological limitations as fully

described in Chapter 2. However, emotional abuse was previously shown to be associated with most personality disorders (Lobbestael et al. 2010) and especially schizotypal personality disorder (Powers et al. 2011). As the schizotypy construct resembles the symptoms of schizotypal personality disorder, it is not surprisingly that the strongest association in the present study was observed between psychological abuse and schizotypal symptomatology. This finding could have several possible explanations, fragmentarily observed in previously research. As emotional abuse is associated with low self-esteem (Battle et al. 2004), psychological abuse has been linked to feelings of shame, humiliation, defeat and helplessness (Cerezo and Frias 1994;Hoglund and Nicholas 1995;Mullen et al. 1996;Webb et al. 2007). It was also argued that childhood trauma leads to paranormal beliefs in order to alleviate powerlessness and hopelessness (Lawrence et al. 1995) and had been associated with an external locus of control (Irwin 1994) along with spiritualism and witchcraft (Perkins and Allen 2006). In addition, verbal abuse leaves deeper scars and is associated with mistrust and guilt (Ney 1987), also because subjects might identify themselves with the abuser and internalise the abusive statements (Johnson et al. 2001). Equally, childhood physical abuse also damages the self-concept which underpins the feelings of worthlessness (Elliott et al. 2005). That ties in with the outcomes of the present study, where not only negative beliefs about the self but also negative beliefs about others were found to be key mediators of physical abuse and schizotypy association. Another important mediator between all types of trauma included in the study and schizotypy was depression score. Previous studies also showed that powerlessness best predicted anxiety and depression (Canton-Cortes et al. 2012) which predicted general psychological distress (Hazzard et al. 1995), self-blame (Canton-Cortes et al. 2012) and stigmatisation (Feiring et al. 2009), consequently impacting on psychosocial adjustment. Another study (Arseneault et al. 2011) also found that the type of childhood trauma characterised by intention to harm (coming from either a peer or

adult figure) was found to be particularly associated with psychotic symptoms, implying it might be the threat (or appraisal of threat) that charges the psychotic-like symptoms. The present findings added to this suggestion with the observation that intention to harm (most likely underpinned by psychological and physical abuse) was associated with the highest levels of schizotypy. This also helps to explain why a particularly strong association was found for the positive schizotypal dimension, although the effect of childhood trauma was evidenced for positive and negative/disorganised dimensions which mirrors the recent evidence coming from larger general population sample (Myin-Germeys et al. 2011). Besides, all types of trauma measured in the present study apart from household discord showed similar effect on the negative/disorganised dimension, but the differential effects of trauma types were much clearer for a positive schizotypal dimension. This has never been researched before and has (as later discussed) some important clinical implications. While psychological trauma and physical trauma strongly predicted positive schizotypal load, no such association was observed for sexual abuse and bullying. Some have suggested it is not so much the type of trauma itself, but more child maltreatment being severe and prolonged that induces positive symptomatology (when looking at narrow psychosis-like symptoms) (Read et al. 2005;Schreier et al. 2009). In parallel with this proposition, Schreier et al. (2009) found that severe forms of victimisation were especially associated with elevated psychosis-proneness. A dose-response association between childhood trauma and schizotypy has also been confirmed in the present study, consistent with a large body of evidence coming from empirical general population studies (Berenbaum et al. 2008;Fergusson et al. 1996;Janssen et al. 2004;Mullen et al. 1993;Myin-Germeys et al. 2011;Shevlin et al. 2007b;Whitfield et al. 2005) and clinical samples (Kilcommons and Morrison 2005;Rubino et al. 2009;Schenkel et al. 2005).

Also, this provides significant evidence against a 'reverse causality' hypothesis, by which individuals with schizotypal traits are more likely to experience the traumatic events. Furthermore, the dose-response association was not only seen in total schizotypy but observed for both dimensions. Likewise, in general, low severity and frequency of traumatic experiences had no impact on schizotypal load, implying that there are more severe events and particularly more frequent abuses/victimisation that are associated with high schizotypy. In contrast, frequent psychological abuse showed a similar effect regardless of the level of severity. It is worth considering, that psychological abuse includes more repetitive forms of abuse by definition (e.g. humiliation, terrorising), which might have effected this outcome. Sexual abuse on the other hand was the only type of trauma assessed where no severity and frequency threshold was needed to predict the schizotypal symptomatology. All these differences indicate the importance of understanding what effects a specific type of trauma has on an individual, possibly helping us to disentangle the complex pathways underlying the increase in schizotypal traits. For example, sexual abuse of any level of severity and frequency was associated with schizotypal symptoms, for psychological abuse high frequency was needed to induce schizotypy, while for physical abuse and bullying only frequent and severe events were reflected in elevated schizotypy traits. It could be that different mechanisms are underlying each trauma type on the pathway to elevated schizotypy, but some caution is still required due to a limited number of individuals reporting certain trauma types (e.g. psychological abuse).

The effects of multi-victimisation and timing of exposure to trauma

Nevertheless, the effects of multi-victimisation (Varese et al. 2012b) also support the present results, making it even harder to assess all the possible pathways underlying the trauma and schizotypy relationship. Not only has it previously been

documented that different trauma types often overlap *(e.g. one adverse childhood experience increases the likelihood of another for 2-18 times (Dong et al. 2004))* but it could be that a combination of specific types of trauma has an especially detrimental effect. For example, one study showed that a combination of physical neglect, physical abuse and verbal abuse had the strongest impact on children by affecting their hopes and enjoyments (Ney et al. 1994), leaving the children fearful, angry and with a poor self-image. Nevertheless, trauma victims have higher traits such as nervousness, irritability and insecurity (Allen and Lauterbach 2007) but also traits of curiosity and open-mindedness which may increase the risk for re-victimisation (Allen and Lauterbach 2007).

Others have argued for the importance of the timing of exposure to adversity (Fisher et al. 2010) with early trauma predicting more severe and persistent psychopathologies (Blaauw et al. 2002;Offen et al. 2003). As it was previously suggested that early abuse might have an impact on the developing brain (Teicher et al. 2003), it is not surprising that especially traumatic events below the age of 12 in the present study showed an association with schizotypal traits. This could partially be explained with the suggestion that traumatic experiences that start early in life are also more likely to have lasted longer, as there is evidence that the effect on outcomes was only significant if childhood abuse also persists into adolescence (Thornberry et al. 2001). In contrast, the present study found that sexual abuse and bullying showed the opposite effects and predicted schizotypy if the abuse/victimisation occurred after the age of 12. A plausible hypothesis is that late childhood/early adolescence marks a beginning of critical sexual and social developmental stage. Supporting this, sexual abuse in another study was only associated with the risk for developing schizophrenia if the abuse occurred in the *'peripubertal'* and *'postpubertal'* years *(12-16)* and involved penetrative abuse (Cutajar et al. 2010). Moreover, Briere and Elliott (2003) argued that sexual abuse at

a later age along with a number of incidents and multiple abusers predicted psychopathology. Another study stressed that individuals who experienced childhood sexual abuse and adult rape (defined as above the age of 16) were over three times more likely to develop depressive symptoms comparing to those who only experienced either child abuse or adult rape (Schumm et al. 2006). A combination of child and adolescent/adult sexual trauma also increased the likelihood of PTSD symptoms by 17 times (Schumm et al. 2006). Moreover, the effects of the exposure to early trauma have been linked to disruption in early attachment which is associated with HPA axis alterations/dysfunction *(maternal behaviour assists in programming the HPA responses to stress)* (Liu et al. 1997). Early traumatic experiences are further associated with adult attachment style which stands as an independent predictor of positive schizotypy (Berry et al. 2007). Additionally, disruption in early attachment predicts anxiety and depression symptoms along with an inadequate social functioning and interpersonal disturbances (Bowlby 1969) which also explains why separation from a parent (especially mother) before the age of *2* has an impact on emotional and social development (Bowlby 1969).

The results of the present study showed some support to the association between early separation from the father figure and schizotypal traits, but not to separation from the mother figure. However, previous research showed that a parental separation for a year before the age of 16 was associated with increased risk for developing psychosis (Morgan et al. 2007), even though the relationship was less clear if a separation occurred after the age of 2. Moreover, Anglin and colleagues (2008) showed that early separation from the mother figure for at least one month predicted elevated scores on suspiciousness, unusual perceptual experiences, inappropriate affect and odd/eccentric behaviour. As such, parental separation may involve similar mechanisms to those observed in maltreated

children, with separation damaging emotional regulation, leading to inflexible integration of emotions and inducing negative representations of others (Elliott et al. 2005).

The significant association between separation from a father and schizotypy observed in this study could however merely be a reflection of the effects of parental divorce which was the main reason for paternal separation in this sample *(approximately 40%)*. For example, studies showed a trend observed for the early onset of psychosis in those individuals whose parents were divorced (Scherr et al. 2012) and parental marital break-up was found to be a consistent predictor of psychiatric disorder (Kessler et al. 1997), more so than parental loss (Oakley Browne et al. 1995). Even though the separation was previously linked to disruption of early attachments, it is unclear whether the existing association between early separation and schizotypy is more an indication of family disadvantage *(family dysfunction, maybe neglect etc. (Tyrka et al. 2008))*, or socio-economic status *(also lower incomes)* (Agid et al. 1999). In line with the evidence that parental death was not associated with a disturbed family environment like parental separation (Tyrka et al. 2008), also the present study found no support to the association between parental death and schizotypy, which has never been explored before. Similarly, two other studies documented that the psychological effect of a separation was greater than a loss due to death (although still significant) (Agid et al. 1999;Canetti et al. 2000). Also, the meta-analysis of the studies exploring the relation between childhood abuse and psychotic/psychosis-like symptoms provided support for the association for all types of trauma apart from parental death (Varese et al. 2012b). However, parental loss (especially maternal deprivation) not only showed the long-term neurobiological effects on HPA axis observed in animal studies (Ladd et al. 1996) but also predicted depressive symptoms (Lloyd 1980) and anxiety symptoms (Nickerson et al. 2013) but possibly relates to more nonspecific factors of

psychological distress rather than schizotypy. In addition, it was previously documented that the quality of the care that follows the loss (or rather a lack of care or neglect) plays a key role in formation of early attachments (Bowlby 1980) and consequently leads to adult psychopathology (Breier et al. 1988;Harris et al. 1986). Besides, even though there is an association between early experiences with caregivers and adult attachment styles, abuse in childhood might interact with early attachment styles and lead to discontinuity in earlier attachment representation (Waters et al. 2000). Possibly reflecting the association between parental loss and HPA axis or the quality of care that follows the loss, there is a trend for loss of maternal figure to have greater pathogenic significance than loss of a paternal figure (Brown et al. 1977), but the evidence is not consistent and could not be explored in the present study due to limited numbers of participants reporting loss of a maternal figure. Tyrka et al. (2008) however suggested that neither poor parental care nor childhood maltreatment fully explained the effect of separation from a parent on adult psychopathology, but family psychiatric history partially accounted for this association.

The effects of age, gender and ethnicity on childhood trauma-schizotypy association

There was some emerging evidence in the literature suggesting gender-specific effects of childhood trauma on schizotypy/or psychotic-like experiences (Fisher et al. 2009;Myin-Germeys and Van Os 2007). Reflecting this, the present results showed that sexual abuse was a significant predictor of schizotypy load for females only (in particular positive schizotypy), with bullying showing a similar trend. On the other hand, for males the association between physical abuse and schizotypal traits was much clearer than for females. Nonetheless, psychological abuse showed a robust association with schizotypy regardless of gender, consistent with reports on the

relationship between emotional abuse and schizotypy (Berenbaum et al. 2008). Various hypotheses have been put forward to try and explain these differences. Gender specific pathways from trauma to psychotic-like experiences have been reported (Myin-Germeys and Van Os 2007), implying differences in association between schizotypy and neurodevelopmental disturbance between males and females (Berenbaum et al. 2008). Consistent with that, evidence also shows that males score higher on negative schizotypy while women score higher on positive symptoms (Bora and Arabaci 2009;Maric et al. 2003;Raine 1992). The present data supports these findings, however gender differences on positive schizotypal symptomatology did not reach significance, which was also observed in another study (Miller and Burns 1995). Furthermore, gender differences might reflect some broader aspect of social and interpersonal deficits (Miettunen and Jaaskelainen 2010), the interaction between genes and abuse might be gender dependent (Barr et al. 2004), females might appraise the traumatic events as more stressful or tend to internalise difficulties much more than males (McFadyen-Ketchum et al. 1996). Reports consistently show that males have higher scores on externalizing problems, which relates to impulsivity, anger and negative reactions, while girls show less such negative reactions *(either due to internalising which relates to sadness, fear, anxiety or learned gender roles)* (Eisenberg et al. 1994). As such depression and anxiety symptoms might account for the association between childhood trauma and psychotic-like symptoms for females. Also, females are more likely to experience sexual abuse, rape or molestation (Kessler et al. 1995;Sun et al. 2008) and although an all male sample demonstrated that child sexual abuse predicted later psychological adjustment no effect was observed for noncontact forms of sexual abuse (Collings 1995). Finally, males might underreport the experiences of childhood trauma (role of socialization?), but that is highly unlikely and there is no clear evidence as to why this would be the case.

Beside gender differences, the present findings provide evidence of ethnic differences in the childhood abuse and schizotypy association; however results need to be viewed as tentative, due to small numbers and grouping of disparate ethnic subgroups into one. Nevertheless, ethnic groups differed in mean schizotypy scores, with *Black African* and *Black Caribbean* groups scoring significantly higher than *'White'* ethnic groups, especially on positive schizotypal traits, with *Black Caribbeans* also displaying more negative/disorganised traits than other groups. This is in line with some previous studies (King et al. 2005;Sharpley and Peters 1999). Ethnic discrepancies were also reported in the association between childhood abuse and positive symptom severity in those at higher risk for psychotic disorder (Thompson et al. 2009). It was further suggested, that whilst *Black* and other ethnic minorities do not have more traumatic life events comparing to the *White British (also found in the current study for recent life events, but Black and other ethnic minorities did report more childhood traumatic experiences)* they appraise them as more threatening (Gilvarry et al. 1999). Another study even reported that race was an independent predictor of perceptual aberrations in an African American group who scored the highest out of all ethnic categories (Goulding et al. 2009).

A positive correlation between age and schizotypy has also been documented (Fonseca-Pedrero et al. 2008). In contrast, the present study suggests only a slight decrease in schizotypy with age, similar to some previous reports (Goulding et al. 2009), implying that schizotypal traits are developed in adolescence and remain stable throughout adulthood. Literature shows that crucial brain maturation processes take place during adolescence and can either have a direct or indirect effect on personality traits and psychosis-proneness (Gruzelier and Kaiser 1996). While the majority of schizotypal traits show a slow decrease over time, disorganisation for women was only restricted to late adolescence, suggesting that

psychological changes (e.g. emotional disturbances) in adolescence might have a crucial role in psychosis-proneness (Bora and Arabaci 2009). This can also partially explain the gender differences, as the present findings suggest that men experience more disorganised and negative schizotypal symptoms *(consistent with (Fossatti et al. 2003))*. On the other hand, women experienced more positive schizotypal symptoms, consistent with Bora and Arabaci (2009), who found that social anxiety and odd beliefs items were significantly more present among female subjects. As suggested, postpubertal hormones may induce the key organisational effects on the developing brain (possibly interacting with genetic susceptibility for psychopathology) and impact the normal and atypical developmental trajectories (Walker et al. 2004). Furthermore, existing literature advocated that other factors influence psychotic-like symptomatology and some symptom-specific associations have been observed. For example, paranoid thoughts were associated with young age, average IQ and male gender, also neurotic symptoms, victimisation and stressful life events (Johns et al. 2004), while hallucinatory experiences showed an association with female gender, a trend for association with Black ethnic groups (Johns et al. 2002), below-average IQ, neurotic disorder and victimisation (Johns et al. 2004). Despite some emerging evidence that particular religious movements are associated with positive symptomatology compared to nonreligious groups (Day and Peters 1999), very limited research does not allow any firm conclusions to be drawn.

Recent life events as moderators of childhood trauma - schizotypy association

Recent life events have been put forward as important factors in development and persistence of psychotic symptoms (Beards et al. 2013;Myin-Germeys et al. 2003a). Similarly, life events also predicted schizotypy in the present study, however the interaction effects with childhood trauma was only observed for intrusive events

(significant for total schizotypy and negative/disorganised dimension) and not independent events as previously shown (Tessner et al. 2011). These results are consistent with a study using a sample of first-episode psychotic patients which observed the synergistic effects of childhood trauma and recent life events in pathways to psychosis (Morgan et al. 2013). It was previously argued, that stressful events might re-activate pre-existing dysfunctional schemas or emotional changes along with increased reactivity (Myin-Germeys et al. 2003a), leading to maladaptive cognitive appraisals but particular impacting upon positive symptomatology (Garety et al. 2001;Morrison et al. 2007) *(the association was not significant in the present study)*. This not only implies the significance of the characteristics of life events but also the importance of individuals' subjective appraisals of these experiences (Lazarus 1999). Besides, an increase in psychotic experiences in patients after a stressful life event was documented for those with high emotional reactivity and trait anxiety but not in those without these traits (Docherty et al. 2009). Complimenting this evidence and consistent with the current study, it was proposed that intrusive events (Raune et al. 2009) or events occurring outside of the individual's control (Cullberg 2003;Das et al. 2001;Day et al. 1987) were particularly associated with psychotic-like symptoms. However, other studies showed that life events affect depressive symptoms in psychotic patients (Brown and Harris 1978) more so than positive symptomatology (Ventura et al. 2000). Adding to this evidence, the present study supported the interaction between childhood trauma and life events for negative/disorganised dimension but did not observe the same influence for positive schizotypal traits. Some have also argued that stressful life events not only affect the symptom levels in individuals with schizotypal personality disorder (Cohen et al. 2008;Tessner et al. 2011) but also predict decline in psychosocial functioning (Jovev and Jackson 2006).

These findings however do not assist in solving the question of whether individuals scoring higher on schizotypy might have more stress-prone lifestyles thus generating more life events, due to either genetic vulnerabilities (van Os et al. 1998), temperamental traits (Breslau et al. 1995) linked to their personalities (Bebbington et al. 1993) or possibly preceding limited coping abilities, lower socioeconomic status and reduced social support (Lukoff et al. 1984). It was also proposed that superstitions and magical thinking develop as coping mechanisms after a stressful event (Lynn and Rhue 1988), which might explain the associations. Underlying these hypotheses is the evidence that personality traits develop in early adolescents and remain stable throughout adulthood (e.g. stability coefficients in childhood $r=.35$ and $r=.75$ in adulthood) (Ferguson 2010;Hopwood et al. 2013;Roberts and DelVecchio 2000). In addition, it is also the specificity of schizotypal traits (e.g. oddness, suspiciousness) that makes exposure to certain life events more likely.

Negative beliefs and depression as mediators of childhood trauma - schizotypy association

Nevertheless, it was suggested that not the event per se but the emotions elicited by them were associated with psychotic-like symptomatology (Docherty et al. 2009). Traumatic childhood experiences and adulthood events impact on the course of depression and anxiety (Mundt et al. 2000), which also depend on negative beliefs (Beck 2008) possibly activated by early adverse experiences. Reiterating these propositions, current findings show that negative beliefs about others, negative beliefs about the self and depressive symptoms all stand as important mediators of the childhood trauma and schizotypy association. This study added to the growing body of evidence that early trauma stimulates not only physical (via moulding neurodevelopmental abnormalities) but also psychological vulnerabilities (Read et

al. 2001;van Zelst 2008). Importantly, the present findings contributed to the literature by exploring the differential effects of specific trauma types in terms of pathways (mediators) leading to schizotypal load. Whilst all types of trauma included in the study were mediated by depression score, psychological, physical and sexual abuse were also mediated by negative beliefs about self and physical abuse was the only type also significantly mediated by negative beliefs about others.

In parallel to this, previous research also supports the association between all types of trauma and anxiety and depression (Bifulco et al. 1991;Bifulco et al. 1998) in a dose-response fashion (Hovens et al. 2010). However, it was found that it is mainly positive schizotypy that is associated with anxiety and depression, low self-esteem and negative schemas, whereas negative schizotypy shows an association with weakened positive self (Barrantes-Vidal et al. 2013). Besides, studies have outlined the importance of beliefs about self and others mediating the association between childhood abuse and paranoia (Fisher et al. 2012) and anxiety and depression mediating the relation between abuse and psychosis/psychosis-like symptoms (Bebbington et al. 2011;Fisher et al. 2013b). As such, a traumatic event could evoke the negative thoughts and beliefs which are fuelling the faulty perception of self and/or others leading to psychotic-like symptoms via their interpretation of these intrusions (Dunmore et al. 1999;Morrison 2001). Also negative schemas about self and others predicted psychotic phenomena even when the severity of victimisation was controlled for (Campbell and Morrison 2007). However, in contrast to the current findings, another study identified the key role of threat appraisals in response to bullying (Catterson and Hunter 2010), which along with internal blame (Graham et al. 2006;Taylor et al. 2013) predicted depression and anxiety symptoms (Taylor et al. 2013). Nevertheless, not all types of trauma were previously found to be mediated by the same cognitive appraisals and emotional states. The trauma-specific cognitive evaluation has been recognised elsewhere, as

relational victimisation led to negative evaluation of others and self, however physical victimisation provoked a direct retaliation (Taylor et al. 2013), possibly reflecting a subject's attempt to regain their status with peers (Crick and Dodge 1996). However, there is also a likelihood that relational victimisation scars an individual's feelings of belonging, leading to social withdraw and limited opportunities for positive relationships (Taylor et al. 2013).

In the present study, there was also a great percentage of the associations between childhood trauma and schizotypal traits that was left unexplained by the included mediators. This could reflect the 'direct' negative reactions to traumatic experiences observed in elevated schizotypal traits e.g. magical ideation, suspiciousness or an 'unexplained' effect due to residual confounding or mediating factors not included in the study (e.g. external locus of control, anxiety symptoms etc. (Fisher et al. 2013b)). Another theory explaining the mediation variables of the childhood trauma and schizotypy relationship suggests that it is the insecure attachment style *(as shown linked to childhood trauma (Breidenstine et al. 2011)* that stand as a key risk factor for depression (Hankin et al. 2005). Also, a growing body of literature suggests that the relationship between childhood trauma and schizotypal load is partially mediated by PTSD (Powers et al. 2011) or dissociation (Berenbaum et al. 2008). A recent study (Braehler et al. 2013) found that dissociation was associated with emotional abuse in particular in individuals with first-episode psychosis with these being strongest for chronic patients. Also, PTSD cannot only cause a fundamental change in personality (Dunn et al. 2004) but PTSD re-experiencing symptoms were linked to hallucinatory experiences *(a direct link between intrusive memories of trauma and hallucinations)*. However this cannot be the only route (Hardy et al. 2005) and only explains a small percentage of the association between trauma and hallucinations (Gracie et al. 2007), as other mediators included negative schematic beliefs. Importantly, these pathways might

not be mutually exclusive as PTSD symptoms might also further impact these negative schema formations (Gracie et al. 2007). On the other hand, the importance of genes for depression has been advocated to explain the pathways from childhood trauma to psychosis-like symptoms via emotional regulation (Kramer et al. 2012).

The strong familial influence on schizotypy levels

The present evidence suggests a strong association between familial risk for psychosis and schizotypy levels. The association remained significant even when adjusting for childhood trauma, however other factors of the family environment (as a possible reflection of parental psychopathology) that might influence this association were not included e.g. socio-economic status (Whipple and Webster-Stratton 1991), poor parent-child relationship (Fergusson et al. 1996). This results are not surprising as all three schizotypy dimensions have been reported as highly heritable (and the heritability factor is stable throughout early and late adolescence) - at the ages between *11 to 14* estimated to range from *42%-53%* and between the ages of *14* to *16* ranging from *38%* to *57%* (Ericson et al. 2011). Findings of the present study show that positive schizotypal symptoms were only associated with a narrow definition of familial risk for psychosis *(including first-degree relatives with a history of psychosis only)*, while the negative dimension was predicted by familial risk with a narrow or broad conceptualisation, possibly suggesting that negative schizotypal traits are less specific to schizophrenia spectrum disorders. This is in line with other studies showing that the association between childhood trauma and schizotypal load was the strongest among those with genetic vulnerabilities for schizophrenia *(and the increase was observed with the narrow definition of the risk of psychosis)* (Schurhoff et al. 2009;Spauwen et al. 2006). The significance of genetic susceptibilities for schizophrenia spectrum disorders including schizotypy

has received wide support, with relatives of schizophrenia patients showing elevated scores on all three schizotypal dimensions (Appels et al. 2004;Yaralian et al. 2000). Some suggested a heritability of positive symptoms in particular (Battaglia et al. 1999;Kendler et al. 1991), including first-rank psychotic symptoms *(e.g. thought insertion, through broadcasting (Loftus et al. 2000))* with elevated scores observed in pedigrees of individuals with schizophrenia.

The results of the present study showed the significant interaction effects between childhood abuse and familial risk on development of schizotypy for the positive dimension only. When comparing the effects of different trauma types, bullying was the only type of victimisation for which the interaction with familial risk for psychosis on schizotypy load was approaching significance. Besides, there was a trend observed for the interaction effect of genetic risk and psychological trauma and bullying especially on positive schizotypal symptoms. In contrast to some previous reports (Torgersen et al. 2002), no interaction effects between trauma and familial risk were observed for the negative/disorganised dimension. These differences could be explained by some specific genetic and environmental factors that underlie each dimension in addition to the more common aetiology of schizotypal traits (Linney et al. 2003;Reynolds et al. 2000). Also, same genes could have an effect on common phenotype for different mental illnesses which then interact with childhood trauma to induce positive symptoms specifically. Besides, other disorder-specific environmental factors (either harming/risk factors or protective factors) could play an important role. Overall, the genes and environment interaction in the development of psychotic-like symptoms led researchers to explore the mechanisms supporting this interaction, producing three hypotheses (Carter et al. 2002). Firstly, genes are not necessary for the development of schizophrenia but contribute to the risk additively. Looking at a sample of monozygotic twins, childhood adversity was associated with psychotic experiences

(positive and negative dimension) however the association was not due to genetic confounding (Alemany et al. 2012). Additional support for this proposition are studies where no interaction between trauma and genetic liability was found (Arseneault et al. 2011;Wigman et al. 2012b). The second hypothesis advocates that genetic vulnerability is associated with sensitivity to the environment therefore traumatic experiences increase the risk of developing psychosis. For example, distress associated with positive and negative subclinical symptoms was shown to be moderately influenced by genetic factors (Jacobs et al. 2005). The third hypothesis suggests that genetically vulnerable individuals are more prone to produce this negative environment, as heritable personality traits can influence the exposure to stressors (Kendler et al. 1993b). Individuals with schizotypal personality disorder were also shown to have lower levels of positive events (Pagano et al. 2004). However with regard to early trauma, this relates to the question of whether childhood trauma is more common in families with genetic risk for schizotypal symptoms.

Current evidence demonstrate the importance of familial influences as gauged by the broader concept of parental psychopathology and the narrow definition of familial risk of psychosis. General parental psychopathology has been found to predict psychotic-like experiences *(age 15 to 16)*, whereas psychotic parental psychopathology predicted the persistence of these experiences *(from 10 to 16)* (Wigman et al. 2012b); and persistence of psychotic-like symptoms predicts psychotic disorders (Dominguez et al. 2011). It is not only a genetic risk (Lataster et al. 2009), but also childhood trauma that was found to be associated with persistence of psychotic-like symptoms (Spauwen et al. 2006). Therefore it is essential to explore the multiple ways in which genes and environment interact. Although the moderate stability of schizotypal traits between early and middle adolescence can be partially explained by genetic influences, new genetic and

environmental factors affected schizotypy in middle adolescence (Ericson et al. 2011). That not only implies a change in schizotypal traits during adolescence but also adds to the complexity of genetic and environmental interactions. According to the hypothesis set out within this thesis, it is important to stress, that even though some individuals had pre-existing familial risks for psychosis when the association between childhood abuse and schizotypy was adjusted for these familial risks, the association stayed similarly strong *(see Table Suppl.14, Appendix XVI)*. Therefore early abuse is a risk for higher schizotypy load with or without proxy genetic vulnerabilities (as indexed by family psychiatric history).

The effect of cannabis use on positive schizotypy

Genes associated with the development of psychotic symptoms can also be expressed as sensitivity to the effect of cannabis (GROUP 2011;Veling et al. 2008) *(e.g. COMT gene (Caspi et al. 2005))*. Lifetime cannabis use according to this study showed no association with total schizotypy levels, consistent with some previous reports (Degenhardt et al. 2003;Houston et al. 2008). An association was observed between current cannabis use and schizotypy, which lost significance after adjusting for confounders. Similarly, starting to use cannabis before the age of 17 was associated with positive schizotypy, but again lost significance after adjusting for confounders. The existing literature shows that cannabis use was associated with elevated positive schizotypal traits *(e.g. ideas of reference, odd beliefs, magical thinking and odd or eccentric behaviour (Dumas et al. 2002))* but also attenuated negative symptomatology (Nunn et al. 2001). Also in other community samples cannabis use was associated with mainly positive and disorganised schizotypy dimension but not with negative symptom load (Cohen et al. 2011;Williams et al. 1996).

Previous studies hypothesised that either cannabis use alleviates negative symptoms *(according to the 'self-medication' hypothesis) (Peralta and Cuesta 1992)* or those individuals with less negative schizotypal traits are more prone to use cannabis (Nunn et al. 2001). The synergistic effects between early trauma and cannabis use have also been widely cited (Cougnard et al. 2007;Harley et al. 2010;Houston et al. 2008) but were not observed in the present study. Interestingly, even non-severe maltreatment has been shown to interact with cannabis to predict psychosis risk (Konings et al. 2012). The suggestion that early cannabis use in particular predicts schizotypy (Konings et al. 2008;Schubart et al. 2010) because of the impact it has on the developing brain (especially at periods of brain maturation (Trezza et al. 2008)) was however confirmed in the current study. But, this could also be attributed to the fact that a majority *(54%)* of the present sample reported using cannabis before the age of 17, with only *8.5%* starting after the age of 22. Also, frequency of cannabis use did predict higher schizotypy, particularly the positive dimension, replicating data from other community studies (Skinner et al. 2011;Stirling et al. 2008). For example, current users showed elevated levels of schizotypy scores compared to past cannabis users (Skosnik et al. 2001), which possibly reflects the prolonged use of cannabis. Even though it could be argued that intoxicated individuals are more prone to report schizotypal traits, this is not very likely (Williams et al. 1996).

Furthermore, cannabis use can either have direct pharmacological effects on schizotypal traits (Ameri 1999;Kapur 2003), via impacting dopaminergic release sensitivity (Voruganti et al. 2001) or indirectly by influencing depression and/or anxiety states (Skinner et al. 2011). Besides, social anxiety has been reported to moderate the relationship between schizotypy and frequency of cannabis use (Najolia et al. 2012). Even though according to the present results cannabis had no link to depression scores as measured by the *Hamilton Depression Scale* (in parallel

with Nunn et al. 2001), it was associated with the depressive dimension of the *CAPE* (also found in Skinner et al. 2011). Also, Delta-9-THC *(especially linked to psychotic symptoms comparing to CBD, which has antipsychotic effects (Morgan and Curran 2008))* is not only associated with psychotic symptomatology but also with anxiety and cognitive deficits among healthy participants. Consistent with these reports, home-grown skunk and super-skunk among all types of cannabis demonstrated particularly strong effects on total schizotypy in the current sample.

Even though some have argued that cannabis use is an independent risk factor for developing psychosis (Arseneault et al. 2002;van Os et al. 2002) it is unclear whether individuals with pre-existing genetic vulnerabilities for developing psychosis are prone to cannabis use (Ferdinand et al. 2005) or maybe more susceptible to the effects of cannabis (van Os et al. 2002). Maybe schizotypal traits precede the cannabis use, as early psychotic experiences have been previously linked to cannabis use as a form of self-medication (Henquet et al. 2005a;Kuepper et al. 2011). Besides, it was found that the interaction between childhood abuse and cannabis had an effect on psychotic symptomatology (Houston et al. 2008;Murphy et al. 2012;Shevlin et al. 2009) but cannabis use was not an independent risk factor for schizotypal traits after adjustment for early trauma. This possibly goes against the hypothesis that the association between cannabis use and schizotypy coexists without any causality and that underlying vulnerability predisposes individuals to higher psychosis symptoms and vulnerability for cannabis use (Schneider et al. 1998). Furthermore, especially the association between more frequent cannabis use and schizotypy as well as the effect of only particular types of cannabis (e.g. super skunk) on positive symptoms would be difficult to interpret if the association between schizotypy and cannabis use is fully accounted for by genetic predispositions. On the other hand, cross-sensitisation effects between childhood trauma and cannabis use were observed for positive schizotypy load and childhood trauma was found to

be moderated by the specific effects of cannabis (Harley et al. 2010), possibly by influencing the dopamine release in the brain (Collip et al. 2008). Individuals with high schizotypal traits reported more unpleasant after-effects of cannabis use compared to those without such traits (Barkus et al. 2006;Stirling et al. 2008), again possibly implying the influence of genetic vulnerabilities. Childhood trauma did not predict the use of cannabis in the present study (there was no association between childhood trauma and cannabis use), which reflects *environment by environment interaction* (exposure to early trauma heightens sensitivity to cannabis, thus effecting schizotypy levels) and not environment – environment correlation (Harley et al. 2010;Konings et al. 2012).

6.3   *Study limitations*

The results need to be considered in the light of the study's strengths and limitations.

This study built on some of the methodological limitations of previous reports exploring the relationship between childhood trauma and schizotypy (e.g. crude measurements, non-heterogeneous sample) by employing more comprehensive interview measures of exposure and outcome, utilising an epidemiological sample of community controls and exploring additive and/or interactive contributors to schizotypal symptomatology. The data was collected using a relatively large community population study, contributing to the heterogeneous sample with respect to gender, ethnicity and age, allowing the evaluation of the trauma-schizotypy association when all these factors were taken into account. Additionally, the detailed interview of childhood experience *(CECA)* not only maximised the validity and reliability of the retrospective reporting of childhood trauma but assured the consideration of contextual factors (e.g. trauma severity, frequency, age when abused occurred). In addition to the inclusion of the types of trauma measured by the instrument (household discord psychological abuse, physical abuse, sexual abuse) also bullying, parental death and parental separation were considered, filling the gap in the existing literature. Likewise, *SIS-R* was utilised as it is an extensive assessment of schizotypal symptoms and signs, covering all three dimensions of schizotypy and able to identify milder forms of schizotypal symptomatology. Besides echoing the multidimensionality of the schizotypy concept, the study incorporated the two distinct standpoints of the construct manifestation: fully-dimensional approach/schizotypal personality traits using SIS-R and assessment of attenuated psychosis-like symptoms using CAPE.

Also, this is the first study looking at the pathways (cognitive schemas, affective state) underlying the trauma – schizotypy association which ties in with studies assessing the mechanisms underlying the early trauma and psychosis

association. Therefore, the present findings contribute to the 'psychosis as a continuum' hypothesis. Another significant advantage of this research is the exploration of additive and/or interactive contributors to schizotypal symptomatology (e.g. genetic risk, cannabis use, life events). Therefore, the current study integrates a comprehensive exploration of distinct types of childhood trauma and their association with schizotypy whilst at the same time expanding the focus from a single predictor of this relationship by adding other social, psychological and genetic factors to the model.

However, the sample was obtained using quota sampling, which is a non-random technique and even though the participants recruited are representative of the local population, there is a question of generalizability of the findings to other populations. In order to include the quotas that would represent Southwark and Lambeth boroughs in London, oversampling of certain ethnic groups was needed, again possibly limiting generalizability of results to other cultures/countries. Similarly, the population of these boroughs is unusual and diverse in terms of ethnicity and wealth *(see section 3.2.1)* and not representative of England. Also, no information was collected from individuals who did not want to take part in the research, opening up the possibility of 'self-selection', where subjects refused to take part in the research study because of certain characteristics – e.g. exclusion of shy participants, more suspicious, those with more family history of psychosis, considering they were notified what the aims of the study were and maybe they were not willing to take part after identifying themselves with certain aspects of the research. In addition, the reasonably small sample size limited some statistical analyses (especially mediation analyses) because of the small numbers reporting particular types of childhood abuse (e.g. psychological abuse) as well as the small number of individuals with familial risk for psychosis (using the 'narrow definition'). Moreover, reported gender differences (although consistent with previous findings

(Morgan and Fisher 2007)) and ethnic differences require some caution when interpreting the findings as there were small numbers in each group within the trauma types, also resulting in more arbitrary combining of a few ethnic groups into less categories. Nevertheless, sample size was calculated using a sample size calculation (although only specific to the main thesis hypothesis) *(see section 3.2.2)* and results are in line with outcomes of larger population-based studies (Afifi et al. 2011;Berenbaum et al. 2008;Johnson et al. 1999).

It is also important to consider some of the limitations of the assessment tools utilised in the study. Even though the *CECA* interview (Bifulco et al. 1994) has a lot of advantages *(as discussed in section 3.4.3)* and the face-to-face interview provides an opportunity to gather contextual information and clarify any ambiguities that might lead to misinterpretations, it is still a retrospective measure and no additional information is collected about the accuracy of the data. Therefore, the conclusions are based on reported abuse and it is not possible to certainly say that the abuse/trauma has really occurred. There is also a concern that researcher-defined maltreatment based on certain behaviour/characteristics might result in overestimation of the abuse (Silvern et al. 2000), however the evidence shows that the underestimation of childhood abuse is much more likely than the overestimation (Dill et al. 1991). Retrospective reporting of childhood trauma has caused a lot of controversy and has (beside the underreporting) raised the concerns of other factors influencing a recall e.g. amnesia (Fivush 1993) mood congruent recall bias (Matt et al. 1992), and the reconstructive nature of memory (Schacter 2001). Nevertheless, retrospective reports have been documented to be reliable over time even for a psychotic population (Fisher et al. 2011). Also, a comparison study of prospective and retrospective measures of childhood maltreatment showed that both types of assessment showed an equal strength of association between trauma and adult psychopathology (Scott et al. 2012). Interview-based research is also shown to

minimize the bias in reporting of maltreatment due to depression (Brewin et al. 1993).

Besides, a problem of definitions of the particular trauma types has also been stressed, which can potentially have a significant impact on the study outcomes. Particularly, psychological abuse is seen as problematic not only in its definition but there is also a danger that this particular trauma type is overlooked by other more tangible forms of abuse (physical abuse and sexual abuse) (Moran et al. 2002). By using the semi-structured interview however which allows for clarification of abuse and possibly extension of already existing categories of psychological trauma, these concerns were minimised. Consensus meetings and ratings completed in pairs also contributed to the reliability of the results. Furthermore, a conservative cut-off point *(a score of at least 2 – moderate severity)* was used to make sure only the most severe trauma was included. Although this can lead to a minor underestimation, a study including different levels of sexual abuse in the analysis found that non-severe forms of this type of abuse (e.g. touching, inappropriate talk) was not associated with psychosis (Bebbington et al. 2011). Besides, the prevalence of early abuse is consistent with the previous UK reports using larger community samples (May-Chahal and Cawson 2005;Radford et al. 2013). Another concern was the age of trauma occurrence which was only based on an estimation, thus timing of abuse could be unreliable (Hardt and Rutter 2004), but the data has been collapsed into two categories only *(under 12, 12 and above)* to improve reliability.

The clustering of the victimisation (*one adverse childhood experience increases the likelihood of another)* has been widely cited (Dong et al. 2004;Kessler et al. 2010;Ney et al. 1994), implying the possibility that the differences in trauma types are due to different clusterings and not purely a distinct effect of certain traumas. Childhood trauma could also only be a proxy for other factors e.g. poor

parenting, low socioeconomic status, family dysfunction etc. that have all been associated with adult psychopathology (Benjet et al. 2010). Moreover, additional risks like parental substance abuse in the family, criminal activity in the family can co-occur with childhood abuse (Felitti et al. 1998), and not accounting for these factors can lead to a wrong attribution of trauma impact *(making it difficult to disentangle a true impact of trauma without other environmental factors)*. Also, poorer parental care and parental overprotection was associated with individuals with schizophrenia (Willinger et al. 2002) and these parental rearing styles can also be an effect of earlier exposure to trauma (Janssen et al. 2005).

Besides, the present study did not include neglect as a separate trauma type which has been documented to predict schizotypy load (Johnson et al. 2000;Myin-Germeys et al. 2011). Studies worldwide even showed that neglect might have the largest prevalence out of all maltreatment types (Butchart et al. 2006), however some reported no relationship between childhood neglect and psychotic-like experiences (Alemany et al. 2011;Fisher et al. 2010). Also cyberbullying was not included as a separate type of victimisation and there are similar effects observed on psychosocial adjustment comparing to traditional peer bullying (Dempsey et al. 2009). Cyberbullying is a latent form of peer bullying (Dempsey et al. 2009) but not necessarily a rare one *(approximately 30% of young people reported being a victim of cyber bullying, 20% reporting being threatened online (Patchin and Hinduja 2006))*.

Even though the effects of multi-victimisation have been measured (and a dose-response of trauma on schizotypy observed), previous reports also found the most detrimental effects of specific trauma combinations (Ney et al. 1994), which were not considered in our study. Similarly, despite considering the frequencies of traumatic events in a separate analysis, for the main effects between trauma and schizotypy and mediation effects the inclusion was only based on the severity of the

trauma regardless of the frequency, possibly contributing to the over-inclusion of the traumatic events. Nevertheless, the importance of including the single incidence of psychological abuse in addition to more chronic abuse was previously highlighted as they might be an important part of more continuous abuse pattern and *'might constitute the tip the iceberg in relation to ongoing interaction'* (Moran et al. 2002 p.235). As mentioned, trauma type is usually not a one-dimensional phenomenon. A recent study on typologies of child abuse in a community population that used a latent class analysis revealed four distinct abuse typologies: a non-abused group, a psychologically maltreated group, a sexually abused group and a group experiencing multiple abuse types (Armour et al. 2013), while physical abuse typology was not found and maybe just co-occur with other forms of abuse (Armour et al. 2013).

Although using a face-to-face interview for measuring schizotypy was a significant advantage of this study and SIS-R assesses signs/disorganised dimension (e.g. odd behaviour, thought disturbances) that cannot be done using traditional questionnaires, the measure is still based on self-report, possibly 'allowing' the misinterpretations and underreporting of certain traits. Also, the CAPE instrument is a self-report measure raising similar concerns (Kendler et al. 1996a), however evidence shows there is a correlation between self-reports using CAPE and clinically assessed psychotic experiences (Konings et al. 2006).

Also, since not many respondents scored positively on disorganised schizotypal traits, the dimension was combined with the negative schizotypy dimension and used for all the corresponding analyses. Although disorganised schizotypal traits were shown to be less stable than positive and negative schizotypy dimension (Bergman et al. 2000) raising concerns about combining it with negative schizotypy, a similar structure has been used in previous studies (Myin-Germeys et al. 2011). The basis for this approach is the traditional two-dimensional view of

schizotypy (Kendler et al. 1991;Siever and Gunderson 1983), but it was the three factorial structure of schizotypy that has received the most support (Kendler and Hewitt 1992;Venables and Bailes 1994;Vollema and Vandenbosch 1995). Also, some found no association between disorganised schizotypy and childhood trauma (Hammersley et al. 2003), possibly resulting in underestimation of the effect the trauma has on negative schizotypy in this study, when combining the dimension with disorganised traits. Each of the traits might also reflect different aetiologies that have been missed with the adopted methodology and it could be argued that the grouping of schizotypal traits into two dimensions (positive and negative/disorganised) is over-simplistic. Another study also observed different clusters in individuals scoring high on the schizotypy scale *(mainly positive symptoms cluster, mainly negative symptoms cluster and two clusters with mixed symptoms of different severity levels)* (Suhr and Spitznagel 2001) adding to the evidence of the complexity of the construct. This is important as each symptom pattern might have unique behaviour and cognitive deficits, have different familial risk factors and a different relationship to schizophrenia (Clementz et al. 1991;Kendler et al. 1996b;Squires-Wheeler et al. 1997). Additionally, there is a possible overlap of positive and negative dimensions – e.g. paranoia reflects paranoid ideation as observed for positive schizotypy but also withdrawal from social relationships – a trait of negative schizotypy (Cicero and Kerns 2010b). Similarly, social isolation can either be a result of low self-esteem or even rejection in those with primarily positive schizotypal symptoms or there is a lack of motivation for social interaction in those with heightened negative schizotypy (Barrantes-Vidal et al. 2013).

The study utilised statistical techniques that imposed artificial boundaries on dimensional/schizotypy scale by looking at the top 20% and top 10% of schizotypy scorers, consequently reducing the statistical power. Moreover, the main effects between trauma types and childhood abuse were analysed using linear regression

and whilst it is argued that some environmental risk factors impacted the position on psychotic spectrum linearly (like childhood abuse) other factors for example proxy genetic risk or cannabis impacted the position in a positive extra-linear fashion (Binbay et al. 2012).

It is also worth pointing out, that besides the evidence that psychotic-like symptoms and schizotypy traits are convergent constructs (Claridge et al. 1996;Stefanis et al. 2002) the interchangeable usage of terms such as psychosis-proneness, schizotypal personality, psychotic-like experiences might not be entirely justifiable. The exact relationship between these terms still has not been truly unravelled and the true discriminate and construct validity of schizotypy in relation to psychosis is yet to be explored. What is important however is that high-risk mental states also include individuals with attenuated psychotic symptoms and those with a family history of a psychotic disorder or schizotypal personality disorder (along with a significant deterioration in functioning), placing schizotypy levels as important predictors of transition to clinical disorder.

Another limitation of the study was the usage of the FIGS interview to measure genetic risks, as parental psychopathology stands as a proxy for genetic liability thus is it not a very sensitive method. Genetic risk needs a more comprehensive assessment, further exploring specific genes *(going beyond the scope of this thesis)* but could on the other hand lead to false-positives considering the vast amount of genetic variations that can be used in the G x E model (van Os et al. 2008). Using parental psychopathology as a measure for genetic risks is nonspecific but well validated and includes complete genetic load (including gene-gene interactions), widely used for G x E modelling. Also, family history of psychosis not only reflects the proxy genetic risk but possibly fragments of an individual's early environment (van Os et al. 2008).

The retrospective design was also applied to recent life events, therefore a recall bias cannot be completely avoided. Research shows that individuals might unintentionally apply causality or bias in their response that would help them understand what has happened to them (Phillips et al. 2007). However, to reduce this possibility, the event was discriminated as either independent or dependent, to also allow the exploration of the relations between stressful events and schizotypy/psychosis-proneness. The inclusion of life events was limited to severe events/difficulties without consideration of daily hassles. Not only are daily hassles associated with axis II disorders *(personality disorders according to previous versions of DSM)* (Malla et al. 1990) but the frequency of daily hassles in adolescents at high risk for psychotic disorder was shown to predict an increase in positive prodromal symptoms one year later (Tessner et al. 2011). Besides, one of the strongest pathways to subclinical hallucinations appeared to be from emotional trauma via life hassles (Goldstone et al. 2012). Also, the present study made no distinction between the types of the life events (e.g. health, work) and there is evidence that individuals with SPD report a higher frequency of health-related problems (e.g. physical illness, death of a family member) (Tessner et al. 2011) as well as higher incidence of crime and legal events (Pagano et al. 2004).

Furthermore, self-report assessment of the lifetime cannabis use raises concerns about underreporting, however *61.8%* of the sample did report using cannabis at least once in their lives, fitting well with previous reports. The prevalence of cannabis use in a UK community sample was estimated at *72%* (used cannabis at least once) (Barkus et al. 2006) while a USA and Canada sample reported that prevalence ranged between *46%* and *70%* among late adolescents (Adlaf et al. 2005). As the usage of frequency categories was arbitrary (e.g. 'more than once a week', 'few times each month') and only based on recall, the frequencies were collapsed into two groups only (high/frequent use and

no/occasional use). The analysis however only considered the highest frequency of lifetime cannabis use but did not account for duration of the use, previously suggested to have an effect on psychosis-like symptomatology (McGrath et al. 2010).

It is important to stress that the associations found between childhood trauma and schizotypy do not necessarily imply causality. Causality is compromised by the assumption that childhood trauma preceded the development of schizotypy traits, but is there evidence otherwise? Are those with schizotypy more vulnerable to experience trauma? The reverse causality question is especially relevant for bullying, as children might experience more bullying because of their personality traits – odd behaviour, unusual ideas, social isolation (Bendall et al. 2008;Lataster et al. 2006;Schreier et al. 2009). The SIS-R gives no definite information about the timeframe of schizotypal traits, making it impossible (in such retrospective study design) to determine that trauma preceded the development of schizotypal traits (childhood trauma generally includes events by the age of 18 and schizotypal traits are developed in early/late adolescence).

Another explanation would be that individuals with pre-existing vulnerabilities to psychosis are more likely to report traumatic experiences regardless of the actual experience, however controlling for psychosis vulnerability indicated this is unlikely (Spauwen et al. 2006). Likewise, there is a question of whether cannabis use leads to the development of psychotic experiences or psychotic experiences result in cannabis use (self-mediation hypothesis, however not likely to explain the entire association). On the other hand, the few prospective studies that have looked at the relationship between trauma and schizotypy show a similar early trauma and schizotypy association (Battle et al. 2004;Johnson et al. 1999;Rossler et al. 2007). Furthermore, the present study contributed to the widely cited evidence of a dose-response relationship (Berenbaum et al. 2008;Myin-Germeys et al. 2011). Distinct

trauma types also showed some unique pathways to schizotypal traits, which would be difficult to explain if schizotypy preceded the early adversity. Research also observed higher rates of psychotic symptoms with sexual content in those reporting sexual abuse with a similar association observed for physical abuse (Thompson et al. 2010). Sexual delusions have also been linked to incest (Read and Argyle 1999), however the evidence is not consistent (Hardy et al. 2005).

Another methodological shortcoming arises from the assessment of the mediation effects. A cross-sectional study design does not permit 'true' mediation analyses, therefore the findings must be seen as preliminary and any claims about the mediation are weak. Also, negative beliefs about self and others were assessed without a particular timeframe while the depression scale was limited to the past 7 days. As five possible mediators were put in the total model along with adjustment for confounding factors the numbers in each of the groups were small, therefore future research if conducted should ensure a large enough sample to include all these mediators. Numbers were especially small for psychological abuse and sexual abuse (especially limited in men who experienced sexual abuse, which lowered the statistical power). Maybe that explains why some types of trauma were not associated with schizotypy in males or females, as previously found in the literature. There was also not much room for comparing severity levels of different types of traumas between genders.

An additional limitation of the current study was the lack of PTSD and anxiety measures. Literature suggests that schizotypal personality disorder shows high rates of comorbidity with mood and anxiety disorders (e.g. Pulay et al. 2009). For example, prevalences of lifetime mood and anxiety disorders among respondents with schizotypal personality disorder were *67.5%* and *67.6%*, respectively (Pulay et al. 2009). Moreover, high anxiety levels (in addition to depression levels) were observed in individuals who also reported high physical and social anhedonia

(considered key features of schizotypy) (Rey et al. 2009). There is also a strong comorbidity of schizotypal personality disorder and PTSD (Pulay et al. 2009). The mediating role of PTSD between traumatic experiences and psychosis (especially positive dimension) has received a wide support (Gaudiano and Zimmerman 2010;Kilcommons and Morrison 2005;McGorry et al. 1991). PTSD was reported to have a significant effect on the individuals' cognitive schemas, permanently changing beliefs about self and others (Dunmore et al. 1999;Morrison 2001). Moreover, psychotic symptoms are considered to be emotion-driven, implying that it is not the event per se, but the emotions (including anxiety, depression) aroused by them that increase these symptoms (Docherty et al. 2009).

Beside reports on comorbidity between these constructs, there is also considerable overlap between schizophrenia-spectrum disorder features and symptoms of depression (e.g. anhedonia, social withdrawal), anxiety (e.g. concentration difficulties, worry) (Lewandowski et al. 2006) and PTSD symptomatology (psychotic-like symptoms, Hardy et al. 2005). But, PTSD, anxiety and depression are especially associated with positive schizotypy dimension *(possibly affecting the associations observed between childhood trauma and positive schizotypy in the current study)*, with less clear relationship observed for negative dimension (Lysaker et al. 1995). For example, Lewandowski and colleagues (2005) showed how specific positive schizotypal traits such as schizophrenia-like perceptual experiences and magical ideation significantly correlated with depression *(r=0.36, r=0.27, respectively)* and anxiety symptoms *(r=0.34, r=0.36, respectively)*. Another study reported that positive schizotypy was associated with measures of anxious apprehension, anxiety arousal, trait anxiety and depression, whereas negative schizotypy was associated with depression only (Mohanty et al. 2008). Moreover, Morrison and Cohen (2014) showed that ideas of reference, for example, are not always paranoid in nature but might display socially

anxious themes, again implying that anxiety significantly affects positive schizotypal traits. Despite the evidence that schizotypy, depression and anxiety symptoms are related to each other, the precise nature of the relationship between them remains unclear.

Anxiety and stress levels were postulated to have a key role in biological model of psychosis with dysregulation of dopaminergic pathways (De Bellis et al. 1999;Depue and Collins 1999) providing a possible link between increased stress sensitivity and development of psychosis. Similarly, the sociodevelopmental - cognitive model (Howes & Murray, 2014) incorporated various hypothesised pathways linking childhood trauma to psychosis. The model proposed that early stress leads to dopamine dysregulation, causing the aberrant assignment of salience to stimuli. It is then the cognitive interpretations of these stimuli which consequently result in psychotic symptoms but also more psychosocial stress (increased anxiety levels), therefore creating a vicious cycle. Nevertheless, anxiety (and depressive) symptoms may accompany, sustain or exacerbate positive schizotypal traits (Debbane et al. 2009).

On the other hand, a study that performed the confirmatory factor analysis indicated that the best fitting model consisted of two separate schizotypy factors (positive and negative) and a third negative affect factor with loadings of anxiety and depression symptoms (Lewandowski et al. 2006). In addition, despite the evidence of anxiety having a key role in the development of psychosis-like symptoms, one study suggested that it is actually the metacognitive beliefs which influence independently on expression of both anxiety and positive symptoms (Debbane et al. 2012). This study offers more support to the trauma-schizotypy associations observed in the present study which did explore the mediating role of metacognitive beliefs. However, findings also confirmed reciprocal relationship between anxiety and positive schizotypy (Debbane et al. 2012). Nevertheless, it is important to note,

that consideration of cognitive schemas and anxiety are by no means exhaustive of significant factors impacting the expression of schizotypy levels.

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The study was necessarily selective in inclusion of the factors/variables leaving out other potential moderators or mediators of trauma and schizotypy association e.g. dissociation (Dorahy et al. 2009), social deprivation (Morgan et al. 2009), insecure early attachments (Berry et al. 2007), and protective/resilient factors associated with subsequent adjustment (Finkelhor et al. 1990). Resilience factors are essential for the individual's understating of traumatic events and dealing with them, but can also reflect genetic variations and interaction with developmental experience (Cicchetti and Rogosch 2012). Especially perceived social support (perception that the support will be there if needed) was observed to have a significant effect (Norris and Kaniasty 1996), particularly for females (Brewin et al. 2000). Social support also mediated the relationship between childhood trauma and

PTSD (Vranceanu et al. 2007). Moreover, social adversity (low socioeconomic status, single-parent household, unemployment) is associated with elevated stress and limited resources to deal with it (Wicks et al. 2005). However, a longitudinal study showed that there is little evidence that marital status, educational attainment, weekly income or social class is associated with self-reported psychosis symptoms (Wiles et al. 2006). Despite that, unemployment was the only variable of social adversity included in the present study which is a vague and incomplete measurement.

Existing literature suggests that childhood abuse predicts 6 out of 11 personality disorders (e.g. borderline, paranoid, dependent) (Gibb et al. 2001), so maltreatment might not have specific effects for schizotypal traits. One study advocated that all forms of childhood maltreatment are more associated with borderline PD than any other types of personality disorders (Battle et al. 2004). There is also a strong comorbidity of schizotypal personality disorder with anxiety disorders, PTSD (Pulay et al. 2009) and cluster B personality disorders (antisocial, borderline, narcissistic PD) even more so than with other cluster A personality disorders (Lentz et al. 2010).

All these complex associations make it especially difficult to truly unravel all the underlying pathways that support the trauma and schizotypy association. Moreover, even if two individuals develop the same form of psychopathology, they might have different underlying dynamics between genetic vulnerabilities and environmental factors on the pathway to the disorder (Pickles and Hill 2006).

CHAPTER 7
Final remarks

Chapter 7 Final remarks

Main aims of the chapter

- Brief summary of the main study findings - a diagrammatic presentation (*section 7.1, Figure 23);*

- Outline the clinical implications of the study/ going beyond the research into clinical practice, prevention and treatment *(section 7.2)*;

- Propositions for the future research *(section 7.3).*

7.1 *Brief summary of the findings (Integrated model)*

The integrated model showing pathways between childhood trauma and schizotypy according to findings of the present study is depicted in *Figure 23*. Schizotypy has a strong (proxy) genetic component (especially for positive schizotypy); however childhood trauma (as one of the early environmental influences) also shows a robust association with total schizotypy levels (and both sub-dimensions) regardless of the genetic underpinnings. Beside the type of childhood trauma (with the strongest effects of psychological and physical abuse), the other factors influencing the association between early trauma and schizotypy are the frequency and severity of the abuse along with multi-victimisation, the age of trauma occurrence and individual's gender. Recent life events (especially intrusive events) can interact with these early traumatic experiences to further influence the levels of schizotypal traits (especially negative/disorganised schizotypal traits, which are possibly less specific to schizophrenia spectrum disorder). Also, frequent cannabis use (also dependent on the type of cannabis) can have a significant effect on positive schizotypal dimension, especially if the subject started using it before the age of 17. The findings also emphasised that childhood trauma (with variations among trauma types) led to negative cognitive schemas (negative beliefs about self and others) and depression that explained a significant percentage of the trauma-schizotypy link. The present results tie in with the findings from studies exploring aetiologies of psychosis (including the role of childhood trauma) as shown in *Figure 5 - Hypothesised sociodevelopmental and neurodevelopmental pathways to psychosis, section 2.3.1 Hypothesised theoretical models*. Therefore, on an aetiological basis, these results provide an additional support to the 'psychosis continuum' theory, implying that there is a gradual transition from schizotypal traits or subclinical psychotic experiences to clinically relevant psychotic disorders.
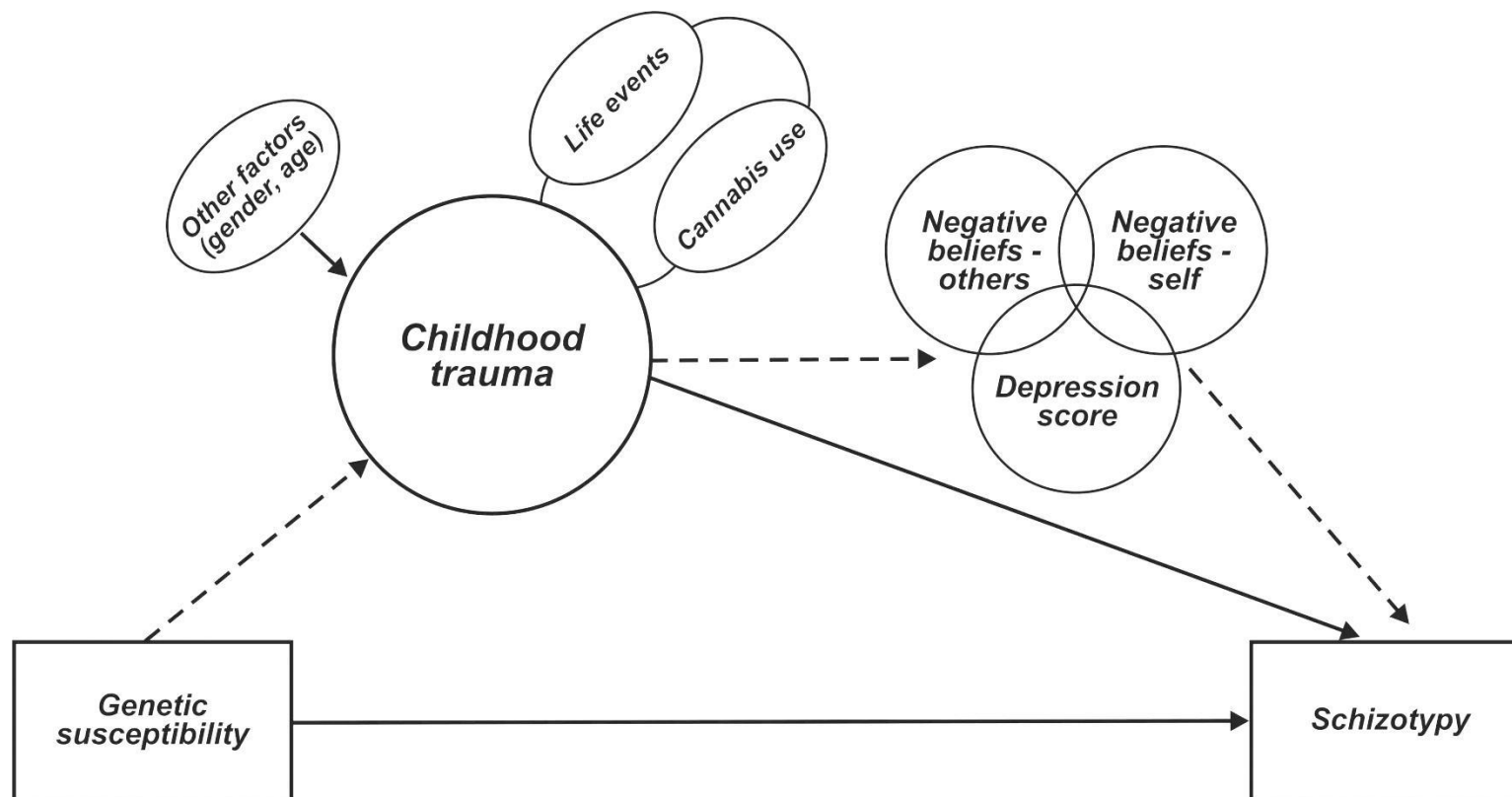
Figure 23: Integrated model - hypothesised diagram showing pathways between childhood trauma and schizotypy according to the present findings; direct paths are indicated by solid arrows and indirect paths by dashed arrows.

7.2   *Clinical Implications*

The study carries some important implications for clinical practice. Firstly, the results complement a large body of evidence that causes and pathways to full blown psychosis can be studied before the onset of clinically significant symptoms (contributing to the dimensional hypothesis of psychosis). Childhood trauma (e.g. Bendall et al. 2008;Morgan and Fisher 2007), life events (e.g. Beards et al. 2013) and cannabis use (e.g. Semple et al. 2005) have all been shown to have a significant aetiological role in the development of psychotic or psychotic-like symptoms in clinical samples (in parallel with current findings for schizotypy). Also, depression (Fisher et al. 2013b) and negative beliefs about self/others (Bentall et al. 2001;Garety et al. 2001;Morrison 2001) have been previously documented to partially account for the association between childhood trauma and psychotic symptoms. Therefore, schizotypy provides a crucial framework for identifying the risk factors for schizophrenia spectrum disorder at subclinical levels. Furthermore, schizotypy allows the exploration of the nonclinical schizophrenic psychopathology without the effects of comorbid factors such as the effects of medication, hospitalisation or stigma. There is evidence that risk indicators such as schizotypal personality, attenuated psychosis-like symptoms or family history of schizophrenia spectrum disorders can predict clinical psychosis (Miller et al. 2001;Olin and Mednick 1996;Yung et al. 1998;Yung and McGorry 1996), making early identification and intervention particularly important.

Nevertheless the early recognition and treatment of psychosis can be elusive (Yung et al. 1996), especially as the relationship between the subclinical symptoms and clinical manifestations still needs to be fully understood. Some of the other concerns with early intervention include false-positive diagnoses (Simon et al. 2013;Thompson et al. 2011;Yung et al. 2010;Yung and McGorry 1996), unnecessary short- or long-term side effects of the treatments (Schaffner and McGorry 2001), early stigmatisation (Heinssen et al. 2001;Yang et al. 2010) and low

predictive power of the symptoms (Yung et al. 2008). Also, non-specificity of the psychotic symptoms in predicting adult schizophrenia has been reported (Fusar-Poli et al. 2013;Gottesman and Erlenmeyer-Kimling 2001;Schaffner and McGorry 2001;Werbeloff et al. 2012), implying a lack of clinical value. Similarly, schizotypal traits were not only observed in relatives of schizophrenia patients but also relatives of schizoaffective, affective and atypical psychotic patients (Mata et al. 2003;Squires-Wheeler et al. 1989). Also, there is a lot of evidence that many psychotic-like symptoms are transient and clinically insignificant, especially in children and adolescents (e.g. Hanssen et al. 2005;Simon et al. 2009).

Nonetheless, many advocated the high predictive values of early psychotic-like symptoms (Poulton et al. 2000). A recent report on a 10-year longitudinal study showed that positive and negative schizotypy predicted the development of schizophrenia-spectrum disorders along with impaired functioning (Kwapil et al. 2013). Also, the most reliable predictor of transition to psychosis in a group of individuals displaying at-risk mental states was the level of schizotypal traits (Mason et al. 2004). Using a sample of individuals identified as at-risk for developing psychosis, the rate of transition to psychosis was 41% within the first year and 50% in a two-year follow up (McGorry et al. 2001;Thompson et al. 2001). Not only was it suggested that the main neuropsychological and clinical problems in the schizophrenia patients pedigrees can be at least partially reversible but that the nonclinical manifestations of 'schizotaxia' can be prevented before transition to full blown psychotic disorder (Tsuang et al. 2002b). In general, studies supported that longer periods of untreated symptoms are associated with worse outcome in terms of symptomatology, overall functioning and quality of life (Marshall et al. 2005;Perkins et al. 2005), inferring that early psychosis is a critical period (Birchwood et al. 1997). All this evidence stresses the importance of early identification to either prevent or delay transition to clinical psychosis or treat current

symptoms (Killackey and Yung 2007) and has been previously reported to have promising results.

The intervention strategies proposed involved either a combination of low doses of antipsychotic medication and psychosocial interventions (McGorry et al. 2002;Schaffner and McGorry 2001) or only psychosocial interventions (e.g. cognitive-behavioural therapy (CBT) (Bechdolf et al. 2012) or adapted CBT especially targeting cognitive biases (van der Gaag et al. 2012)) with less damaging side effects (Morrison et al. 2004). However, in a later study Morrison and colleagues (Morrison et al. 2012) found no significant difference between groups (one received cognitive therapy one did not) on reduction of transition to psychosis and reduced severity of psychotic symptoms in at-risk individuals (also in Addington et al. 2011). Addington et al. (2003) argued that even though some individuals might never transition to clinical psychosis, they display symptoms often associated with distress and impaired functioning and therefore intervention techniques can still be beneficial (Schaffner and McGorry 2001). Also CBT has proved successful for treating positive symptoms but also co-morbid mood related disorders/symptoms such as depression and anxiety (Morrison et al. 2004). Furthermore, the use of CBT has been widely advocated as it targets cognitions and beliefs about self and others that are particularly important contributors to the development of clinical psychosis (Kuipers et al. 2006). Antipsychotic medication on the other hand was argued to either ameliorate the pathophysiologic processes causing psychosis (therefore decreasing or delaying onset) (Lieberman et al. 1997;McGlashan et al. 2006;McGorry et al. 2002) or supresses the symptoms (Woods et al. 2003) but not a possible progression of the illness (Hegarty et al. 1994). This is not to suggest that universal and selective interventions should be in place, as it is not feasible (and considering the low incidence rate of psychotic illness also ethnically questionable (McGorry et al. 2002)), but the prevention of transition to full blown psychosis has

been shown to be a valuable intervention method (Yung et al. 2007) and also carries financial benefits (Falloon et al. 1998). Importantly, schizotypal traits must not be considered in isolation, but in combination with other background information such as traumatic life events, social deficits, decrease in functioning, coping skills, social support and other resilience factors (Yung and McGorry 1997). Mostly authors agree on the importance of continuous therapeutic support and follow-ups to minimise the transition rates (McGlashan et al. 2001). Moreover, an integrated treatment (assertive community treatment model) was shown to reduce family burden of illness (Jeppesen et al. 2005), which can further improve the family communication among individuals at high risk for psychosis and consequently even impact the course of the early psychosis (O'Brien et al. 2014) (e.g. protective factors of family environment (Gonzalez-Pinto et al. 2011)).

Childhood abuse/trauma has a significant effect on an array of adult psychopathology like depression (e.g. Bifulco et al. 1991), anxiety (e.g. Kessler et al. 1997), eating disorders (e.g. Rorty et al. 1994), dissociation (e.g. Martinez-Taboas et al. 2006), substance misuse (e.g. Kendler et al. 2000), PTSD (e.g. Duncan et al. 1996), suicidal ideation (e.g. Blaauw et al. 2002), personality disorders (e.g. Afifi et al. 2011) and especially psychotic-like symptoms and psychotic disorders (e.g. Morgan and Fisher 2007;Read et al. 2005). Consequently, since the 1980s a large body of research has stressed the importance of routine enquiry about the trauma histories within the mental health services (Jacobson and Richardson 1987;Larkin and Read 2008;Read and Fraser 1998). It is essential to ensure that any child protection policies and procedures are followed as well as to have programs in place designed to reduce the prevalence of traumas. This is also important as psychotic patients who reported childhood abuse have also experienced earlier hospital admission, higher symptom severity, longer hospitalisations, more relapse episodes and more suicide ideations (Goff et al. 1991;Mullen et al. 1993;Read 1998;Ucok and

Bikmaz 2007). Therefore, it is essential for practitioners to monitor and protect children following childhood abuse as effective treatment requires an understanding of what is behind the individual's presentation. Whilst clinical literature widely recognises the impact of early trauma on adult psychopathology, more milder range of symptoms often go undetected and clinicians do not inquire about the childhood adversities (Young et al. 2001). As personality traits are partially shaped by childhood emotion-related experiences (Cohen 2008), it is important to consider the damaging effect that the trauma has on individual's personality (e.g. schizotypal traits). Therapies for trauma survivors and treatment for other symptoms evidenced to be good intervention techniques include CBT, psychodynamic therapies and integrative approaches (e.g. Kirshner 2013;Martindale et al. 2000;Morrison 2009). The present study also emphasized the importance of focusing on psychological and physical abuse as these types of trauma showed the most detrimental effects. As the main focus is ameliorating the psychological impact of childhood traumas, CBT has been suggested to assist individuals in understanding the beliefs, emotional states and thoughts that can lead to a reduction in depression, anxiety and PTSD symptoms (Larkin and Read 2008). Furthermore, it is essential to understand the negative appraisals and threat appraisals associated with specific trauma types and target those negative evaluations (negative beliefs about self/others) possibly preventing the onset of psychotic symptoms. Trauma-specific interventions involve restructuring of cognitive appraisals of trauma and consequently restriction of exposure to trauma triggers, preventing the secondary adversities (Kaufman and Henrich 2000). Furthermore, the present study identified the significance of trauma at an early age, indicating that ongoing clinical support is required for individuals exposed to early trauma to alleviate increased risk for developing schizotypal or psychotic-like symptoms. The interventions do not necessarily have to focus directly on the abuse but can provide more indirect support for individual's transition during these critical developmental stages (help

with adaptation, new social roles) (Kaufman and Henrich 2000). It is possible to speculate that attempts to reduce trauma and victimisation among children can moderate the risk of developing psychotic-like experiences and in turn psychotic disorders.

Another concern that needs addressing is the adult re-victimisation that is common among victims of childhood abuse (e.g. Desai et al. 2002). Also, some life events cannot be prevented (neither the intrusiveness of events especially linked to increased schizotypy) but by influencing the individual's understanding of the events (including victimisation) and preventing negative beliefs which can be modified with appropriate therapeutic approaches the negative schemas can be ameliorated. Also treatments tailored for those who experienced childhood trauma are required to help reduce sensitivity to stress of life events/or daily hassles, along with reduced overgeneralising thoughts and reduced external attributions, which further impacts individual's threat appraisals, perceived locus of control and associated feelings of defeat, humiliation etc. (e.g. Garety et al. 2001).

Another suggestion for the clinical practice arises from the support found for an association between cannabis use and (positive) schizotypy. The question still remains as to whether cannabis exacerbates symptoms or is being used as a form of self-medication after a traumatic event. Irrespective of this, cannabis users need to be assessed for history of childhood trauma and individuals with childhood trauma need to be asked about their cannabis use. This applies to clinical settings and the general population. The present study showed that type of cannabis used and frequency of its use can affect schizotypal load. It is therefore important to work on reduction of cannabis use among those with traumatic experiences to alleviate positive schizotypal symptoms or psychotic-like symptoms.

The study's findings can be a valuable help in conceptualising treatment plans, yet more research is needed to understand the true nature and time-course in which psychotic symptoms are developed (Lieberman et al. 2001). Also, future research needs to deal with the problems of heterogeneity of early interventions, as individuals considered at-risk are clinically diverse (Schaffner and McGorry 2001). Nevertheless, exploration of the early vulnerability factors for psychosis and consequently early interventions challenge the historical view of therapeutic pessimism in schizophrenia (Lieberman et al. 2001).

7.3   *Future directions*

If replicated, the study would need a larger sample, especially to assure enough cases in more infrequent types of childhood abuse (e.g. psychological abuse, sexual abuse in men), thus maximising the statistical power. Although a larger scale prospective study would provide the most robust evidence on mediators between childhood trauma and schizotypy and help to truly understand the long-term trajectory of the vulnerability factors (Ingram and Gallagher 2010) it carries a lot of limitations (ethical and practical). If a cross-sectional design is used, it would be essential to have a more structured timeframe for each of the assessments (especially depression, negative beliefs) to be able to more successfully position these variables on the timeline, although the overlaps between the concepts cannot be completely avoided (e.g. childhood trauma generally includes events by the age of 18, schizotypal traits are developed in early/late adolescence). The assessment of familial risks for psychosis ('narrow definition') also requires to be repeated as findings of the present study showed a strong association between familial risk and schizotypy; but the very restricted numbers reduced the possibilities for more statistical analyses (e.g. gender differences, different pathways to schizotypy with/without family risk).

Besides, to further explore the underlying pathways to the development of schizotypal traits, it is important to explore how childhood trauma impacts on various neurocognitive biomarkers (e.g. brain alterations, HPA axis disturbances) in individuals with high schizotypy scores (Diwadkar et al. 2006) which is widely cited the in schizophrenia literature. Also, the focus should be expanded to genome-wide association or epigenetic techniques to fully explore the underlying pathways between childhood trauma and schizotypy, including pathophysiology like dopamine functioning, neuroendocrine responses to stress etc. In addition, the impact of prenatal and postnatal environmental factors needs to be accounted for. For example, small placenta weight, low birth weight predicted more pronounced

schizotypal traits (Lahti et al. 2009) which ties with findings in individuals with schizophrenia (Jones et al. 1998). The importance of the early environment in the expression of disorder should not be underestimated as 85% of individuals with schizophrenia have no genetic risks for the disorder (Wahlberg and Wynne 2001). Clinical practice would benefit from research into other forms of childhood victimisations/adversities not included in the present study for example cyberbullying, witnessing of traumatic event etc. Also, the combination of these risk factors needs to be assessed and separate etiological pathways considered. Furthermore, a cross-cultural validation of the associations supported by the current study is needed to generalise the findings to other populations/countries, as the differences have been reported, either because of culture-specific definitions or other culture-dependant factors.

In addition to the need for a broader inclusion of traumatic events/early adversities, also negative beliefs were only considered as two separate concepts (negative belief about others/self). However, there is support for different forms of appraisals of negative beliefs. One study for example proposed six types of distinct threat appraisal that can act as mediators between exposure to early trauma (violence) and adjustment problems: harm to others, physical harm to self, negative evaluation by others, negative self-evaluation/self-blame, material loss and loss of relationships (Kliewer and Sullivan 2008).

There is still a lingering question as to what influences the transition from high schizotypy to full-blown psychosis and whether the symptoms are linearly becoming more severe? More likely the answer lays in the complex interactions of genetic and environmental factors. Unaffected individuals also include those at high risk for developing psychosis and identifying schizotypal traits in these groups could also provide insight into behavioural, clinical and physiological processes underlying the schizophrenia spectrum liability.

In conclusion, It is important to stress that vulnerability research presents the future of psychopathology research as it brings us closer to understanding the underlying causes of psychopathology, helps in prevention of onset of psychopathology and help conceptualise intervention techniques (Ingram and Price 2010).

Final Conclusion

This study complements previous research supporting the strong association between childhood trauma and schizotypy. Building on methodological limitations of previous reports it provides further evidence to the effect of different types of childhood trauma on schizotypal symptomatology. The strongest association with schizotypy was found for psychological and physical abuse. It was also more severe and chronic abuse/victimisation forms that particularly predicted schizotypy, in a dose-response fashion. The findings also demonstrated some differential effects of specific types of trauma on the positive and negative/schizotypal dimensions and revealed the possibility of differences in pathways leading to schizotypal traits. The comprehensive model including some social, psychological and proxy genetic factors extended the understanding of complexity and interaction of the risk factors (e.g. life events, cannabis use) underpinning schizotypal symptomatology. This study has an important value for clinical practice but the findings should be considered in the light of its limitations.

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Appendices

Appendix I: *Scoring Guide for the Quality* assessment of the empirical research papers

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Appendix XVI: *Additional Result Tables*:

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Scoring Guide

A) SELECTION BIAS

(Q1) Are the individuals selected to participate in the study likely to be representative of the target population?

Score of 2	In general population studies, the entire sample was randomly selected. In case-control/cohort studies, the sample was made up of cases (either schizotypal traits/psychotic-like experiences assessed as present OR documented evidence of childhood adversity) and randomly sampled controls (either no evidence of schizotypal traits/psychotic-like experiences OR no documented evidence of childhood adversity).
Score of 1	The sample was made up of either cases only or randomly sampled controls, or there were no control subjects.
Score of 0	There was a non-random selection process or the sampling method was not reported.

(Q2) What percentage of selected individuals agreed to participate?

Score of 2	70-100% of participants.
Score of 1	50-69% of participants.
Score of 0	Less than 50% of participants, or not reported or not applicable.

(Q3) What is the sample size?

Score of 2	At least 100 subjects in case and control groups (in general population samples, the same rule applies for those with schizotypal traits/psychotic-like experiences and those without); and/or evidence of a sample size calculation of adequate statistical power.
Score of 1	At least 50 subjects in each group.
Score of 0	Less than 50 subjects in each group.

B) MEASUREMENT OF EXPOSURE- CHILDHOOD TRAUMA

(Q4) What was the quality of the childhood trauma measurement tool?

Score of 2	Semi-structured interview measure or documented evidence (forensic report/medical examination/social services' records).
Score of 1	Checklist measure, administered as an interview.
Score of 0	Self-report checklist.

(Q5) Did the measure assess different types of trauma?

Score of 2	There was an assessment of different types of trauma and they were analysed separately.
Score of 1	There was an assessment of different types of trauma but they were not explored separately in the analysis.
Score of 0	No distinction was made between different types of trauma, or not reported.

C) MEASUREMENT OF OUTCOME- SCHIZOTYPY/ PSYCHOTIC-LIKE EXPERIENCES

(Q6) How was schizotypy measured?

Score of 2	Structured assessment by clinician
Score of 1	Structured assessment by trained research worker or self-report standardised measure for schizotypy/`psychotic-like experiences.
Score of 0	Brief self-report checklist/1-2 items only or unstandardised measure.

D) CONFOUNDING

(Q7) Was there an assessment of confounding and adjustment for it in the analysis?

Score of 2	Potential confounders were measured and adjusted for in the analysis (e.g. basic demographic information and other risk factors- such as genetic risk, substance use, depression/anxiety levels).
Score of 1	Adjustment for basic demographics.
Score of 0	No adjustment for confounders or not reported.

Table Suppl.1: *Continued Summary of studies on childhood adversity in relation to psychosis-like experiences (ordered by quality score)*
(All abbreviations are listed at the bottom of the table)

Authors	Study Design	Sample Recruited (Age)	% female	Measure of Trauma	Number Exposed	Measure of Schizotypy	Number with the Outcome	Measure of Effect	Quality Score
Gromann et al. (2013) (The Netherlands)	Cross-sectional Community sample	N=724 (10-14 Mean=11.9, SD=0.76)	48.3%	The Bullying Role Nomination Procedure (BRNP) (Goossens et al. 2006) 2 questions from Revised Olweus Bullying Questionnaire (Olweus 1996b)	79 (10.9%) self-reported victims 33 (4.6%) peer-reported victims 37 (5.1%) self-and peer-reported victims	4 yes/no questions to assess nonclinical psychotic experiences from Diagnostic Interview Schedule for Children (DISC-C) (Shaffer et al. 2000)	421 (58.1%) at least one psychotic-like experience 200 (27.5%) at least two PSE 68 (9.4%) at least three PSE	Self-reported victims had more PSE than non-victims (F=11.14, p<0.001) Direct relational victimization predicted psychosis scores (β=0.08, t=1.98, p=0.05) Indirect relational victimization: (β=0.16, t=3.53, p=0.001) Physical victimization: (β=0.12, t=3.11, p=0.005) *adj. gender, age*	6
Mackie et al.(2012) (UK)	Longitudinal and prospective measures General adolescent population sample	N=1098 (Mean=13.6)	39.1%	Two questions from Revised Olweus Bully/Victim Questionnaire (Olweus 1996b)	Bullied in low trajectory class of psychosis N=257 (31.6%) In elevated trajectory class N=22 (50%) In increasing trajectory class N=39 (49.4%)	9 questions (q) assessing hallucinatory experiences and delusional beliefs 5 q adapted from Diagnostic Interview Schedule (Costello et al. 1982) 4 q taken from another study (Laurens et al. 2007a)	An increasing class N=79 (8.4%) An elevated class N=44 (4.7%)	Those who experienced bullying once or twice a month report elevated psychotic experiences (OR=2.73, 95% CI=1.25-4.52, p<0.01) Those who experienced bullying three or more times a month a month report increasing psychotic experiences (OR=3.43, 95% CI=1.82-6.46, p<0.001) *adj. basic demographics, depression, cigarette, alcohol, previous psychotic experiences at T1*	6
Mackie et al.(2011) (UK)	Prospective cohort study Longitudinal study	N=409 who scored above the mean on four personality risk factors in a larger study (Mean=14 years 7 months)	In persistent psychotic-like experience group only =58.3%	Peer victimization Revised Olweus Bully/Victim questionnaire (Olweus 1996b)	-	9 q assessing hallucinatory experiences and delusional beliefs adapted from Diagnostic Interview Schedule (Costello et al. 1982), see Laurens et al. 2007a	-	Victimization increased the risk of persistent psychotic-like experiences (OR=2.8, 95% CI=1.2-6.8) *adj. all demographics, SUPRS measure*	6
Gracie et al. (2007) (UK)	Cross-sectional Random community sample	N=228 (Mean= 28.9, SD=8.7)	70.6%	The Traumatic Life Events Questionnaire (TLEQ) (Kubany et al. 2000) 2 items from CTQ	At least one traumatic event experienced by N=202 (88.6%)	The Self-Report Scale Post-traumatic Stress Disorder (SRS-PTSD) (Carlier et al. 1998)	Paranoia scale (Mean =41.7, SD=14.9) LSHS	*Trauma groups showed significantly higher levels of paranoia:* Childhood trauma (t=-5.2, df=159, p=0.001) Sexual abuse/assault (t=-5.58, df=127, p=0.001)	6

Table S1 Cont'd

				(Bernstein and Fink 1998) (emotional abuse and neglect and physical neglect)		The Paranoia Scale (Fenigstein and Venable 1992) The Launay Slade Hallucination scale (LSHS) (Launay and Slade 1981) The Structured Interview for Assessing Perceptual Anomalities (SIAPA) (Bunney et al. 1999)	(Mean=2.7, SD=2.2)	Physical assault (t=-5.74, df=124.43, p=0.001) Sexual assault associated with higher levels of perceptual anomalies (t=-2.5, df=127, p=0.01)	
Sun et al. (2008) (China)	Cross-sectional Random community sample	N=1307 (18-25 Mean=19.8, SD=1.20)	53.6%	Modified questionnaire on child sexual abuse (CSA) (see Fergusson et al. 1996) (non-physical and physical contact)	22% in female sample experienced physical or/and non-physical contact before 18, 11.4% physical CSA, 14.7% in male sample experienced physical or/and non-physical contact	Symptom Check-List-90 (SCL-90) (Derogatis 1977)	-	CSA (physical contact) vs national youth group on paranoid ideation (t=5.65, p<0.001) CSA (physical contact) vs national youth group on psychoticism (t=5.73, p<0.001) CSA (non-physical contact) vs national youth group on psychoticism (t=2.49, p<0.05)	5
Campbell and Morrison (2007) (UK)	Cross-sectional Random community sample	N=373 14-16 (Mean=14.8, SD=7)	56.2%	Bully/Victim Questionnaire (BVQ) (Olweus 1989, unpublished)	92 (25%) experienced bullying at least once or twice 25 (6.6%) reported more frequent experiences of bullying	Paranoia Scale (Fenigstein and Venable 1992) Revised Launay-Slade Hallucination Scale (Launay and Slade 1981) - auditory subscale	-	Bullying significantly associated with hallucinations (F(1,369)=21.736, p<0.005) and paranoia (F(1,366)=20.755, p<0.005)	5
Fisher et al. (2012) (UK)	Cross-sectional Convenience sample	N=212 (Mean=27, SD=8.4)	65.4%	Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 2003) prior 17	-	Psychosis Screening Questionnaire (PSQ) (Bebbington and Nayani 1995) – paranoia questions	Physical neglect (N=58, 27.1%) Emotional abuse (N=55, 25.7%) Physical abuse (N=45, 21.0%) Emotional neglect (N=40, 18.7%)	Emotional abuse and adult paranoia (OR=1.32, 95% CI=1.09-1.59, sign.) Physical abuse and adult paranoia (OR=1.29, 95% CI=1.07-1.55, sign.) *adj. for gender, age at interview, ethnicity, family psychiatric history*	4
Goldstone et al. (2012) (Australia)	Cross-sectional Case-control study	N=113 non-clinical sample N=100 psychosis patients	Non-clinical sample 59% Clinical sample 46%	Early Trauma Inventory (ETI) (Bremner et al. 2000)	Non-clinical sample: Physical trauma mean=1.63 (SD=1.54) Emotional trauma mean=1.38 (SD=1.38) Sexual trauma (10%)	Launay Slade Hallucinations Scale-Revised (LSHS-R) (Laroi et al. 2004)	LSHS-R for non-clinical sample (hallucinations M=12.71 (SD=10.86) Auditory hallucinations M=1.24	Non-clinical sample only: Emotional trauma and hallucinations (r=0.32, p<0.01) Physical trauma and hallucinations (r=0.19, p<0.05) Emotional trauma and auditory hallucinations (r=0.30, p<0.01) Physical trauma and hallucinations (r= 0.20, p<0.05)	4

							(SD=1.76)		
Haj-Yahi and Tamish (2001) (Israel)	Cross-sectional Convenience sample	N=652 (18-37 Mean=20.64, SD=2.28)	60%	Finkelhor's (1979) scale for measuring sexual abuse (yes/no)	5.7% sexually abused by a family member before 12 11.6% sexually abused by a relative before 12 13.2% sexually abuse once by stranger before 12	Brief Symptoms Inventory (BSI) (Derogatis and Melisaratos 1983)	-	*Abuse by immediate family member:* Nonabused vs abused under age of 12 and psychoticism (t=-4.344, p<0.001) Nonabused vs abused between 12-16 and psychoticism (t=-2.700, p<0.01) Nonabused vs abused over 16 and psychoticism (t=-3.002, p<0.01) Nonabused vs abused under age of 12 and paranoid ideation (t=-4.300, p<0.001) Nonabused vs abused between 12-16 and paranoid ideation (t=-3.733, p<0.001) Nonabused vs abused over 16 and paranoid ideation (t=-3.674, p<0.001)	4
Ross and Joshi (1992) (Canada)	Cross-sectional Random sample from general population	N=502 adults population drawn from another study (Mean=42.5, SD 16.5)	63.3%	Dissociative Disorder Interview Schedule (DDIS) (Ross et al. 1989) – on physical abuse and sexual abuse	20.9% experienced one or more Schnederian symptom(s)	Dissociative Disorder Interview Schedule (DDIS) (Ross et al. 1989)	79.1% reported no Schneiderian symptoms 4.5% reported four or more	Childhood trauma features differentiate those with three or more schneiderian symptoms from those with none: For PA (x2(1)=11.615, p<0.001) For SA (x2(1)=18.780, p<0.001) PA and SA (x2(1)=42.439, p<0.001) Sexual symptom content was seven times more likely to be found (14%) in the CSA and CPA groups than in non-abused group (2%) (x2(1)=6.04, p<0.02)	4
Kilcommons et al. (2008) (UK)	Cross-sectional Case-control group	N=40 sexually abused group (17-54 Mean=28.72, SD=10.53) Control group (17-55 Mean=22.03, SD=7.92)	SA group 87.5% Control group 87.5%	The Davidson Trauma Scale (DTS) (Davidson 1996) The Sexual Events Questionnaire (SEQ2) (Calam and Slade 1989)	-	21-item Peters et al. Delusional Inventory (PDI-21) (Peters et al. 1999) The Revised Hallucination Scale (RHS) (Launay and Slade 1981) Auditory Hallucination subscale (AHRS) and Visual Hallucination Scale (VHRS) from PSYRATS interview (Haddock et al. 1999) The Dissociative Experience Scale (DES) (Bernstein and Putnam 1986)	100% in the SA group experienced delusional ideation to some extent 100% of SA endorsed at least 4 items on the RHS measure	SA scored higher on all measures of psychotic-like experiences compared to control group: RHS vividness (F=8.20, p=0.005) RHS auditory (F=6.77, p=0.011) RHS visual (F=20.14, p=0.001) PDI total (F=20.92, p=0.001) PDI distress (F=24.68, p=0.001) PDI preoccupation (F=17.52, p=0.001) PDI conviction (F=26.09, p=0.001) Total number of SA associated with AHRS (r=0.36, p<0.05), VHRS total (r=0.50, p<0.05), vivid imagery and daydreaming (r=0.65, p<0.05), visual hallucinations (r=0.58, p<0.05), PDI total (r=0.53, p<0.05), PDI preoccupation (r=0.59, p<0.05), PDI distress (r=0.51, p<0.05), PDI distress (r=0.51, p<0.05), PDI conviction (r=0.58, p<0.05) *adj. age*	3
Collings (1995) (South Africa)	Cross-sectional Convenience	N= 284 men (17-36,	0%	Childhood sexual victimization survey before	-	Brief Symptom Inventory (BSI) (Derogatis and	Non-contact abuse N=56	Control group vs 'contact abuse' group on paranoid ideation (F=16.01, p<0.001)	

	sample	Mean=19.7)		18		Melisaratos 1983)	(20%) Contact abuse N=26 (9%)	Control group vs 'contact abuse' group on psychoticism (F=8.47, p<0.01)	3
Konings et al. (2012) Replication study (Greece & Netherlands)	Replication study Greek National Perinatal study prospective cohort study - *see Stefanis et al. 2004a* and NEMESIS prospective study in general population - *see Cougnard et al. 2007*	(Greek National Perinatal study N=1636) (NEMESIS N=4842 Greek National Perinatal study - at birth, at age 7 and at age 19, range 18-64 T0=41.2, SD=11.9)	Greek National Perinatal study: 55% NEMESIS: 53%	Greek National Perinatal study: Physical punishment (one question) at T1 NEMESIS: semi-structured interview on emotional, physical, sexual abuse before 16 (4 questions) – (see Janssen et al. 2004) at T0	Greek National Perinatal study: sometimes (58%), often (12%) NEMESIS: 8.5% moderate to severe maltreatment	Greek National Perinatal study: CAPE (Konings et al. 2006;Stefanis et al. 2002) at T2&T3 NEMESIS: CIDI (World Health Organization 1993) at T1&T2	-	Greek National Perinatal study: exposure to T1 childhood maltreatment positively associated with T2 psychosis outcome (β=0.11, CI 0.03-0.18, p=0.006), dose-response relationship NEMESIS: childhood maltreatment positively associated with psychosis outcome at T1/T2 (OR=1.96, 95% CI=1.73-2.20, p<0.001)	n/a (replication of two studies)

Note: PSE, Psychotic-like experiences. F, F-ratio. β, Beta regression coefficient. t, T-test. OR, odds ratio. CI, Confidence Interval. adj., adjusted. SUPRS, Substance Use Risk Personality Scale. CTQ, Childhood Trauma Questionnaire. df, degrees of freedom. r, Pearson correlation coefficient. (C)PA, Physical abuse, (C)SA, Sexual abuse, RHS, The Revised Hallucination Scale. PDI, Peters et al Delusions Inventory. NEMESIS, The Netherlands Mental Health Survey and Incidence Study. CIDI, Composite International Diagnostic Interview. T0, baseline. T1, first measure after T0 (longitudinal study). T2, second measure after T0 (longitudinal study). b, regression coefficient. PSYRATS interview, Psychotic Symptom Rating Scales.

Participant Information Sheet and Consent Form

Institute of Psychiatry

at The Maudsley

Division of Psychosis Studies,

Section of General Psychiatry

Internal Box PO 63
De Crespigny Park
Denmark Hill
London SE5 8AF
Tel   + 44 (0) 0207 848 0100
Fax + 44 (0) 0207 7019044
Email: r.murray@iop.kcl.ac.uk

KING'S College LONDON

University of London
www.kcl.ac.uk

Information and Consent Form

TITLE OF PROJECT: GENETICS AND PSYCHIATRIC ILLNESS (GAP)

You have been asked to take part in a study being conducted in the South London and Maudsley NHS Trust. Before you decide whether to enter the study, it is important that you understand why the research is being done and what it will involve. Please take time to read the following information and ask any questions if something is not clear or you wish to know more.

What are the aims of the study?

In our research project we are interested in identifying what the main risk factors that predispose to psychosis are. In particular, we want to know whether there are any genes that increase the risk of developing a psychotic disorder, either alone or by interacting with environmental factors such as stress, cannabis, and infections. Part of the reason why some people become ill may lay in genetic differences between people, in the same way that we are different in the colour of our eyes, hair etc. To achieve this, we will compare the genetic make-up of people with a diagnosis of psychosis with the make-up of people with similar characteristics but no history of mental health problems.

We also aim to establish whether some genes might influence the course of the illness and response to medication. Some patients experience an improvement of their psychiatric symptoms when they are treated with medications, whereas others do not do so well and/or experience severe side-effects. Therefore we aim to look at how genes can influence individual differences in response to drug treatment so that we may be able to choose better drugs for each person.

In conclusion, the type of genetic analysis that we carry out is only for research purposes and does not at present produce clinically relevant results.

Why are we asking for your help?

You have been invited to take part in this study because of the nature of the symptoms that you appear to have been experiencing. During the course of the study approximately 1000 people who have had symptoms like yours will be asked to take part.

What will we ask of you if you take part in the study?

For this project we will ask from you a small sample of blood, about 20 mls (a few tablespoons full) or cheek swab and saliva samples for metabolic and genetic analysis. We may also use your blood and saliva sample to:

1) Measure the level of hormones and proteins contained in the blood serum and in the saliva.
2) Look at the expression of some genes of interest in the white cells contained in the blood.

A medically trained researcher will take the blood sample using disposable sterile equipment. It will only take few minutes as for any routine blood sample. If you are unable or unwilling to give a blood sample it is also possible to perform genetic analysis from cheek swab samples, a simple procedure that (we can show you the kit and illustrate the procedure) collects dead cells present in your saliva and in your mouth. From the cheek swab sample we cannot measure level of medication or look at expression of genes, we can only extract a small amount of DNA. Therefore we prefer to ask for a blood sample to guarantee a better quality of our results and make the most out of your generous help.

A researcher will demonstrate how to collect the saliva sample and will provide you with the tubes required. The level of some proteins contained in the saliva can give us an indication of differences in the level of stress experienced by healthy volunteers and people suffering from mental illnesses.

We will also ask for some of your time to collect clinical and socio-demographic information using standardised research instruments: diagnostic interview, symptoms rating scale, socio-demographic interview and neuropsychological tests.

If you have already taken part in other research projects at the Institute of Psychiatry, London that involved some of the assessment we are interested in, we will not ask you to undergo them again but we request your permission to use the existing data.

Some people within the study will be invited to undergo an MRI scan of the head and of another region of the body (the adrenal gland, a small gland above the kidney).They will be presented with separate information and consent forms for this procedure.

The sample collection and the clinical assessment will require approximately 3 hours of your time. Moreover we would like to contact you again for follow up (up to 24 months) to repeat the above assessments to investigate changes over time. We will also reimburse any travel expense related to your participation into the study.

We will also ask for your consent to contact your GP, mother (or father) and a sibling. This is 1) to collect information from your GP records and mother about events that may have occurred very early in your life, such as complications during pregnancy and neonatal infections, 2) to conduct some of the same assessments with your sibling that we have conducted with you, and 3) to ask your sibling similar questions that we have asked you about the environment in which you both grew up and experiences you may have had in childhood. We will only contact your GP and/or relative(s) with your explicit consent and we will not disclose any information we have collected from you to them. If you agree for us to contact your mother (or father) and/or a sibling, we will only proceed to interview them if they provide consent.

What are the risks?

The risks involved are those of ordinary blood tests such as small pain and occasionally a small bruise around the area from where the sample has been taken. There is no risk involved in the collection of saliva.

Is Confidentiality guaranteed?

All personal information about you is regarded as strictly confidential; only researchers belonging to the study team, and not external collaborators, know which sample belongs to whom. All the information about you will be coded; you will not be identifiable in any research outcome.

1) The blood samples first and the DNA samples after extraction will be stored in the Institute of Psychiatry secured laboratory for 5 years.
2) The samples will be coded using bar codes (numbers and letters not referring to your name or date of birth) that will be entered on a secure computerized data base.
3) The clinical information collected on the sample will be securely held in the Institute of Psychiatry building.
4) Nothing that you have told us will be mentioned to any relative you might give us permission to contact.

The access to the samples and the related information will be restricted to the researchers involved in the study. In case of commercial collaborations only the coded data will be shared, therefore no researcher external to the study team will ever have access to personal data concerning participants.

Any future work will pursue aims related to the topic of this project and any extension of the project beyond 5 years, will be subject to review by a research ethics committee. You are free to withdraw from this study at any point without giving a reason by contacting the researcher whose details are at bottom of the consent form. Withdrawal will not affect any of the care and treatment you receive.

What are the benefits for you of taking part?

This is a research project, looking at comparing a group of healthy volunteers with people experiencing their first psychotic episode. As mentioned before, this study will not produce individual test results for any of the data collected. Therefore we cannot offer direct benefits for you. We will be able to provide all participants with a general summary of our research, when the project is complete, through a project newsletter. Our research study is also described on the Institute of Psychiatry general website (www.iop.kcl.ac.uk), under the Division of Psychological Medicine, Department of General Psychiatry.

Who is funding this project?

This study is funded by the The Maudsley Charitable Fund, the Department of Health, the Wellcome Trust and the European Union. Thank you very much for your time and once again please ask for more information on both the project and/or your illness/symptoms if it is still unclear.

Contact details for research team:

Dr Marta Di Forti
Institute of Psychiatry
Tel 020 7848 5352
e-mail: marta.diforti@kcl.ac.uk

CONSENT FORM

If you have come to the decision to enter the study after carefully considering the information provided, please read and sign this form.

TITLE OF PROJECT: GENETICS AND PSYCHIATRIC ILLNESS (GAP)
Researcher: Dr Marta Di Forti, Institute of Psychiatry

		Yes	No
1)	I have read the information sheet and I have been given a copy. I was given the opportunity to ask questions. I understand why the research is being done and the risks involved.	☐₁	☐₂
2)	I agree to give a sample of blood/cheek swab and saliva samples for research in the above project. I understand how the sample will be collected, that giving the sample is voluntary and that I am free to withdraw at any time without giving a reason, and without my medical treatment or legal rights being affected. I understand that I will be contacted in the future to repeat part of the assessment.	☐₁	☐₂
3)	I understand that research using the sample I give will involve genetic analysis aimed at understanding the role of genes in disease and response to drugs, that the data produced are for research rather than clinical purposes, and that these results will have no implications for me personally.	☐₁	☐₂
4)	I understand I will not receive any 'test' results from this study, because the assessment I will undergo, does not produce clinically relevant information but just research data. The project newsletter will describe the general importance of any research results obtained.	☐₁	☐₂
5)	I give permission for my previous research records to be looked at, and information from them to be analysed in strict confidence by responsible professional staff from the research team. Researchers external to the study team, collaborating in the project (including commercial collaborations) will only access my coded data.	☐₁	☐₂
6)	I agree that the samples I have given and the information gathered about me can be examined and stored (for 5 years) at the Institute of Psychiatry. I understand that future research may be performed by researchers other than those who conducted the first project, including researchers from commercial organisations. To guarantee confidentiality, I agree that researchers external to the study team, including those from commercial collaborators, will only have access to coded data and not to personal details. Any future research will only pursue aims related to the topic of this project, and any extension of the project beyond 5 years, will be subjected to review by a research ethics committee.	☐₁	☐₂
7)	I consent to the input of coded data obtained from my blood sample and from the information gathered about me into a computer, to be used for statistical analysis and research. I understand I have the right to request, via the study co-ordinator, to review data concerning me, and to have such data modified if inaccurate, or deleted.	☐₁	☐₂
8)	I understand I will not benefit financially if this research leads to the development of a new treatment or medical test but my travel expenses will be reimbursed.	☐₁	☐₂

9) I give permission for my GP records to be looked at. ☐₁ ☐₂

10) I agree to my mother being approached to participate in this study. ☐₁ ☐₂

Contact details:

Name ....

Address ....

....

Phone Number ....

11) I agree to a sibling being approached to participate in this study. ☐₁ ☐₂

Contact details:

Name ....

Address ....

....

Phone Number ....

....
Name of Subject   Date   Signature

....
Name of Researcher   Date   Signature

Would you like to be sent further information about the project in our newsletter?

Yes No
☐₁ ☐₂

Contact details for research team:

Dr Marta Di Forti
Institute of Psychiatry
Tel 020 7848 5352
e-mail: marta.diforti@kcl.ac.uk

Psychosis Screening Questionnaire (PSQ)

PSYCHOSIS SCREENING QUESTIOINNAIRE

Subject number: 2EU02. |__|__|__|__| **Date of Birth** |__|__|-|__|__|-|**1**|**9**|__|__|

Time interval: Lifetime

Interviewer: ....   **Date** |__|__|-|__|__|-|**2**|**0**|__|__|

Code: No = 0   Unsure = 1   Yes = 2

In this survey we have to ask about a whole range of experiences. Some of these experiences are quite rare.
However, I would be very much obliged if you would bear with us and answer the questions I am going to ask you now.

Q1.   Over the past year, have there been times when you felt very happy indeed without a break for days on end?

(a)   Was there an obvious reason for this?

(b)   Did your relatives or friends think it was strange or complain about it?

Q2.   Over the past year, have you ever felt that your thoughts were directly interfered with or controlled by some outside force or person?

(a)   Did this come about in a way that many people would find hard to believe, for instance through telepathy?

Q3.   Over the past year, have there been times when you felt that people were against you?

(a)   Have there been times when you felt that people were deliberately acting to harm you or your interests?

(b)   Have there been times when you felt that a group of people was plotting to cause you serious harm or injury?

Q4.   Over the past year have there been times when you felt that something strange was going on?

(a)   Did you feel it was so strange that people would find it very hard to believe?

Q5.   Over the past year, have there been times when you heard or saw things that other people couldn't

(a)   Did you at any time hear voices saying quite a few words or sentences when there was no-one around that might account for it?

Q6.   Have you ever received treatment for any psychiatric or psychological problem?

………..

MRC Sociodemographic Schedule

MRC SOCIODEMOGRAPHIC SCHEDULE (Amended) Part 1

EU GEI

STUDIE: EU GEI	Date of Birth
Subject number: |__| EU |__|__| - |__|__|__|__|__|	|__|__|-|__|__|-| **1** | **9** |__|__|
Time interval: Present	Period – Replicat |**0**|__|-|**0**|__|
Interviewer: ………………………………..	Date |__|__|-|__|__|-| **2** | **0** |__|__|

1. **Gender**   O1 Male   O2 Female

2. **Age**   \|__\|__\|

3. **Ethnicity All Sites:**

   O1 White   O2 Black   O3 Mixed   O4 Asian   O5 North African   O6 Other

4. **Ethnicity Site Specific (UK)**

O11 White British	O12 White Irish	O13 White gypsy, traveller	O14 Other White
O15 Mixed (w, bc)	O16 Mixed (w, ba)	O17 Mixed (w, as)	O18 Other Mixed
O19 Indian	O20 Pakistani	O21 Bangladeshi	O22 Chinese
O23 Other Asian	O24 Black Caribbean	O25 Black African	O26 Other Black
O27 Arab	O28 Other, specify:_____		

5. **Place of Birth**

O1 Austria	O2 Belgium	O3 France	O4 Germany
O5 Ireland	O6 Italy	O7 Spain	O8 Suisse
O9 The Netherlands	O10 Turkey	O11 United Kingdom	O12 Brazil
O13 Australia	O14 other, specify: _____		

6. **Age of migration (if applicable)** |__|__|

7. **Father's place of birth**

O1 Austria   O2 Belgium   O3 France   O4 Germany

O5 Ireland   O6 Italy   O7 Spain   O8 Suisse

O9 The Netherlands   O10 Turkey   O11 United Kingdom   O12 Brazil

O13 Australia   O14 other, specify: _____

8. **Mother's place of birth**

O1 Austria   O2 Belgium   O3 France   O4 Germany

O5 Ireland   O6 Italy   O7 Spain   O8 Suisse

O9 The Netherlands   O10 Turkey   O11 United Kingdom   O12 Brazil

O13 Australia   O14 other, specify: _____

9. **First language:**

O1 English   O2 German   O3 French   O4 Dutch

O5 Spanish   O6 Turkish   O7 Italian   O8 Kurdish

O9 Portuguese   O9 other, specify:_____

10. **Ever employed**   O0 No   O1 Yes

11. Social class (Subject)

(provide descriptions only)	Current	Main
a. **Job Title**	_____	_____
b. **What do (did) you mainly do?**	_____	_____
c. **What does/did organization make?**	_____	_____

d. **Social class subject current:**

O_1 Higher grade Professional O_2 Lower grade Professional

O_3 Intermediate occupations O_4 Small Employer and self employed occupations

O_5 Self employed occupations O_6 Lower supervisory and lower technician occupations

O_7 Lower services, sales and clerical occupations O_8 Lower technical occupations

O_9 Routine Occupations O_{10} Never worked and long-term unemployed

d. **Social class subject main**

O1 Higher grade Professional   O2 Lower grade Professional

O3 Intermediate occupations   O4 Small Employer and self employed occupations

O5 Self employed occupations   O6Lower supervisory and lower technician occupations

O7 Lower services, sales and clerical occupations   O8 Lower technical occupations

O9 Routine Occupations   O10 Never worked and long-term unemployed

12. Social class Father (other_____)

(provide descriptions only)	At birth of participant	Main
a. **Job Title**	_____	_____
b. **What do (did) you mainly do?**	_____	_____
c. **What does/did organization make?**	_____	_____

d. **Social class father at birth of participant**

- O_1 Higher grade Professional
- O_2 Lower grade Professional
- O_3 Intermediate occupations
- O_4 Small Employer and self employed occupations
- O_5 Self employed occupations
- O_6 Lower supervisory and lower technician occupations
- O_7 Lower services, sales and clerical occupations
- O_8 Lower technical occupations
- O_9 Routine Occupations
- O_{10} Never worked and long-term unemployed

d. **Social class father main:**

- O1 Higher grade Professional
- O2 Lower grade Professional
- O3 Intermediate occupations
- O4 Small Employer and self employed occupations
- O5 Self employed occupations
- O6 Lower supervisory and lower technician occupations
- O7 Lower services, sales and clerical occupations
- O8 Lower technical occupations
- O9 Routine Occupations
- O10 Never worked and long-term unemployed

13. Mother's age at birth |__|__|

14. Father's age at birth |__|__|

15. Number of brothers and sisters |__|__|

16. Do you consider yourself to have (or ever have had) a hearing impairment? O_0 No O_1 Yes

17. Was the onset of the hearing impairment before the age of 18 years? O_0 No O_1 Yes

18. Where have you lived during your life, starting with the place you were born?

No.	Country	City/Town	Street/Postcode	Age				Change of School	
				From		To			
1.								O0 No	O1 Yes
2.								O0 No	O1 Yes
3.								O0 No	O1 Yes
4.								O0 No	O1 Yes
5.								O0 No	O1 Yes
6.								O0 No	O1 Yes
7.								O0 No	O1 Yes
8.								O0 No	O1 Yes
9.								O0 No	O1 Yes
10.								O0 No	O1 Yes
11.								O0 No	O1 Yes
12.								O0 No	O1 Yes
13.								O0 No	O1 Yes
14.								O0 No	O1 Yes
15.								O0 No	O1 Yes

Current

No.	Country	City/Town	Street/Postcode	Age			
				From		To	
1.							

19. Are you registered with a GP? O0 No O1 Yes

If YES, provide name and address of GP

....

....

MRC SOCIODEMOGRAPHIC SCHEDULE (Amended) Part 2

EUGEI

1. **Since leaving your parents' home, have you lived with others?** O_0 No O_1 Yes

2. **Who do you live with…?(For b-d: Rate if age 17 or older at time; otherwise N/A not applicable)**

	Alone	Alone, with children	Partner, Spouse	Partner, Spouse, with children	Parents	Other family	Friends	Other: specify (e.g. hostel, halls of residence)	N/A
a) Now	O_1	O_2	O_3	O_4	O_5	O_6	O_7	O_8_____	O_9
a) 1 yr ago	O_1	O_2	O_3	O_4	O_5	O_6	O_7	O_8_____	O_9
b) 5 yrs ago	O_1	O_2	O_3	O_4	O_5	O_6	O_7	O_8_____	O_9

3. **Do you own/ rent your home…? (For b-d: Rate if age 17 or older at time; otherwise N/A not applicable)**

	Privately owned (self)	Privately owned (family)	Rented (Private)	Rented (government)	Other, specify:	N/A
a) Now	O_1	O_2	O_3	O_4	O_5_____	O_6
b) 1 yr ago	O_1	O_2	O_3	O_4	O_5_____	O_6
c) 5 yrs ago	O_1	O_2	O_3	O_4	O_5_____	O_6

4. **Overcrowding**

		a) Now	b) 1 yr ago	c) 5 yrs ago
i.	How many persons live(d) with you?	☐☐	☐☐	☐☐
ii.	How many rooms in your home (exclude kitchen and bathrooms)	☐☐	☐☐	☐☐

5. **Have you ever had a long-term relationship (one year or more)** O_0 No O_1 Yes

6. **How many children do you have?** |__|__|

7. **What is your relationship status …? (For b-d: Rate if age 17 or older at time; otherwise N/A not applicable)**

	Single	Married, living with someone	In a steady relationship	Divorced, separated	Widowed	N/A
a) Now	O1	O2	O3	O4	O5	O6
b) 1 yr ago	O1	O2	O3	O4	O5	O6
c) 5 yrs ago	O1	O2	O3	O4	O5	O6

8. **What is the highest level of education you have achieved?**

O1	School, no qualifications	(to end of compulsory education; passed no exams, tests, etc.)
O2	School, with qualifications	(to end of compulsory education; passed one or more exams, tests, etc.)
O3	Tertiary, Further	(first level of non-compulsory education; e.g. A-levels, Baccalaureate)
O4	Vocational	(job related education, e.g. teacher training, plumber, electrician, etc.)
O5	Higher (undergraduate)	(University; first degree)
O6	Higher (postgraduate)	(University: higher than first degree level, e.g. Masters, PhD)

9. **How many years have you been in education?** (from beginning of compulsory education) |__|__|

10. **Are you employed (paid work) …? (For b-d: Rate if age 17 or older at time; otherwise N/A not applicable)**

	Unemployed	Economically inactive (i.e. house person, physical illness/disability, career, retired)	Student	Part-time employee	Full-time employee	Self-employed	N/A
a) Now	O1	O2	O3	O4	O5	O6	O7
b) 1 yr ago	O1	O2	O3	O4	O5	O6	O7
c) 5 yrs ago	O1	O2	O3	O4	O5	O6	O7

11. What is your monthly income …? (For b-d: Rate if age 17 or older at time; otherwise N/A not applicable)

		a) Now		b) 1 yr ago		c) 5 yrs ago		
i.	**Nett monthly income Ł**	_____		_____		_____		
ii.	**Income below median**	O0 No	O1 Yes	O0 No O1 Yes O3 N/A		O0 No O1 Yes O3 N/A		
iii.	**Income below official poverty line**	O0 No	O1 Yes	O0 No O1 Yes O3 N/A		O0 No O1 Yes O3 N/A		
iv.	**Receipt of welfare benefits**	O0 No	O1 Yes	O0 No O1 Yes O3 N/A		O0 No O1 Yes O3 N/A		
	If YES, specify	_____		_____		_____		

12. What is your religious affiliation?

O0 None   O1 Christian   O2 Jewish

O3 Muslim   O4 Other, specify _____

13. How often do you attend religious services?

O0 Never   O1 Once or twice a year   O2 Monthly   O3 weekly

For first-generation migrants only

14. Where, on a scale from 1 to 10, do you rate your fluency in the majority language? |__|__|
    (1=not fluent at all, 10= very fluent)

Wechsler Adult Intelligence Scale (3rd ed.) (WAIS-III, abbreviated) – Scoring Sheet

EU GEI shortened WAIS-III Answer Sheet

EU:GEI

STUDIE: EU GEI	Date of Birth
Subject number: |__|EU| |__|-| | | | | |	|__|__|-|__|__|-| **1** | **9** |__| |
Time interval: present	Period – Replicat |**0**|__|-|**0**|__|
Interviewer :	Date |__|__|-|__|__|-|**2**|**0**|__|__|

1. Digital Symbol Coding

Time Limit	120 seconds
Completion Time	
Total Raw Score	(Maximum =133)

2. Arithmetic

	Problem	Time Limit (seconds)	Completion Time in Seconds	Correct Response	Response	Score (0 or 1)
	1	15		3		
	2	15		7		
	3	15		5		
	4	15		2		
Start→	5	15		£9.00		
	7	30		5		
	9	30		8		
	11	30		£10.50		
	13	60		£186.00		
	15	60		£600.00		
	17	60		£51.00		
	19	60		1 of 4 or 5 of 20		0 1 $^{(11\text{-}60s)}$ 2 $^{(1\text{-}10s)}$
					Total Raw Score	

3. Block Design

	Correct Design	Time Limit	Incorrect Design			Completion Time in Seconds	Correct Design		Score (Circle the appropriate score for each design.)			
1.		30"	Trial 1	Trial 2			Y	N	0	Trial 2 1	Trial 1 2	
2.		30"	Trial 1	Trial 2			Y	N	0	Trial 2 1	Trial 1 2	
3.		30"	Trial 1	Trial 2			Y	N	0	Trial 2 1	Trial 1 2	
4.		30"	Trial 1	Trial 2			Y	N	0	Trial 2 1	Trial 1 2	
5.		60"	Trial 1	Trial 2			Y	N	0	Trial 2 1	Trial 1 2	
7.		60"					Y	N	0	16"–60" 4	11"–15" 5	6"–10" 6 1"–5" 7
9.		60"					Y	N	0	21"–60" 4	16"–20" 5	11"–15" 6 1"–10" 7
11.		120"					Y	N	0	66"–120" 4	46"–65" 5	31"–45" 6 1"–30" 7
13.		120"					Y	N	0	76"–120" 4	56"–75" 5	41"–55" 6 1"–40" 7

START →

Total Raw Score [ ]

WAIS-III Answer Sheet, English, EU GEI, September 2010, page 2

466 |

4. Information

Item	Question	Response	Score (0 or 1)
1	Saturday		
2	Age		
3	Ball		
4	Months		
6	Sunrise		
9	Brazil		
12	Cleopatra		
15	Olympics		
18	Sistine Chapel		
21	Water		
24	Continents		
27	Speed of Light		
		Total Raw Score	

Start→ (points to item 6 row)

Structured Interview for Schizotypy-Revised (SIS-R)

SIS-R

EUGEI

STUDIE: EU GEI	Date of birth
Subject Number: |_| EU |_|_| - |_|_|_|_|	|_|_|-|_|_|-|1|9|_|_|

Time interval: last month	Period – Replicat |0|_|-|0|_|
Interviewer:	Date |_|_|-|_|_|-|2|0|_|_|

Instructions:

Read all possible responses aloud to the subject. Score conservatively: in case of doubt between 2 categories, score the lowest category.

1. SOCIAL ISOLATION

1.1 How many friends do you have? *(If none go to 1.4)* \|_\|_\|_\|

By friends I mean people with whom you have regular contact, including by mail or telephone.

1.2 How often do you have contact with your friends? *(if never go to 1.4)*
How often do you see, speak or write to them?

O1 Every day O4 once a month
O2 two or three times per week O5 less than once a month
O3 once a week O6 never

1.3 How close do you feel to your friends?

O1 Very close O3 a little close
O2 somewhat close O4 Not at all close

1.4 How often do you have contact with your family/members of your family with whom you don't live? How often do you see, speak or write to them?

O1 Every day O4 once a month
O2 two or three times per week O5 less than once a month
O3 once a week O6 never

1.5 How often do you take part in meetings, clubs, social groups or other organizations? *(excluding church meetings)*

O1 Every day O4 once a month
O2 two or three times per week O5 less than once a month
O3 once a week O6 never

SIS-R, English, EU GEI, 4-D, Sept. 2010, page 1

1.6   How often do you attend church services?

O1 Every day   O4 once a month
O2 two or three times per week O5 less than once a month
O3 once a week   O6 never

1.7   Is there someone with whom you have an intimate relationship and with whom you can share your most personal feelings? *If the subject is married*: "This can refer to your partner."
If no, go to 1.9

O1 Yes   O5 no

1.8   With how many people do you have such a personal relationship? |_|_|

1.9   Have you recently been to visit people?   O0 No   O1 Yes

1.10   What do you do if people *(friends/acquaintances/family)* come to your door unexpectedly?

....

1.11   Do you sometimes withdraw yourself from social contacts? How often? How long does that last?

....

1.12   OBJECTIVE REASONS FOR SOCIAL ISOLATION
(ex. Illness, physical handicap, most friends are dead, live in an isolated area without public transport, demanding job)
Does your physical health make it difficult for you to visit people? And/or
Does your situation or lack of transport make it difficult for you to visit people?

O1 No objective reason
O3 Some objective reason that cannot explain everything
O5 Clear reasons that likely can explain everything

1.13   GLOBAL ASSESSMENT OF SOCIAL ISOLATION

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

2. INTROVERSION

In the rest of this interview, I will ask you about what sort of person you are, GENERALLY SPEAKING. For some of the questions, your feelings could have changed over the years. In that case, I would appreciate the answer that is the most apt description of you as a person.

People differ in the degree to which or how much they want to be alone or to be with other people. That is: some people are more self-directed and some are more directed to others.

2.19 Do you regard yourself in general as someone who is alone quite often, alone somewhat, a little alone is or who is not at all alone?

O1 Quite a lot   O5 A little
O3 Somewhat   O7 Not at all

2.20 Do you regard yourself in general as quite, somewhat, a little, or not at all outgoing?

O1 Very   O3 Somewhat   O5 A little   O7 Not at all

2.21 Would you answer the following questions with either "yes" or "no" (considering yourself in general).

		Yes	No
a.	Are you a chatty person?	O1	O0
b.	Are you somewhat lively?	O1	O0
c.	Do you usually take the initiative in making new contacts?	O1	O0
d.	Do you enjoy working with others?	O1	O0
e.	Do you tend to remain in the background at social events?	O1	O0
f.	Do you like to get out and about among people?	O1	O0
g.	Do you like having a lot of action and excitement around you?	O1	O0
h.	Are you usually calm in the company of other people?	O1	O0
i.	Can you get a party going?	O1	O0
j.	Do you enjoy meeting new people?	O1	O0

2.22 Would you answer the following questions with either "yes" or "no".

		Yes	No
a.	If things are going quite well with a good friend then I feel that I am also doing well.	O1	O0
b.	If something is bothering me, I like to talk about it with other people.	O1	O0
c.	I like hobbies and uses of my free time in which I do not need to involve other people.	O1	O0
d.	As a rule, I enjoy being by myself, thinking and daydreaming.	O1	O0
e.	I am much too independent to get truly involved with other people.	O1	O0
f.	People who try to get to know me better mostly give up after a little while.	O1	O0
g.	I could be quite happy sitting in a cabin in the wood or the mountains.	O1	O0
h.	If it were up to me, I would rather be with others than alone.	O1	O0
i.	I feel quite close to my friends.	O1	O0

2.23 GLOBAL ASSESSMENT OF INTROVERSION

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

3 HYPERSENSITIVITY

3.1 How sensitive are you in general, to comments and remarks made about you?

O1 Very   O3 Somewhat   O5 A Little   O7 Not at all

3.2 If someone should make an unpleasant remark about you that you did not deserve, how long would you think about it before letting go of it?

O1 A week or more   O2 2 to 3 days   O3 One day   O4 One hour/ a few hours

How often does that happen (that feeling)?

(1=once a year, 2= once a month, 3=every week)

3.3 I have a short list of statements here. *Could you indicate, for each one separately whether it is:* Certainly true, probably true, probably not true, absolutely not true

		Certainly true	Probably true	Probably not true	Absolutely not true
a.	I avoid doing things because I am afraid I will make myself look like a fool.	O1	O2	O5	O7
b.	I am quick to take offense	O1	O2	O5	O7
c.	Emotionally, I have something of a thin skin.	O1	O2	O5	O7
d.	I come across to other people as a little dense.	O1	O2	O5	O7
e.	Any kind of criticism puts me off my stride.	O1	O2	O5	O7

3.4   GLOBAL ASSESSMENT OF HYPERSENSITIVITY

    O0 Absent   O1 Mild   O2 Moderate   O3 Severe

4 REFERENTIAL THINKING (PART I being watched)
   Many people sometimes have the feeling when they are in a group that people are
   looking at them.

4.1   Do you know that feeling? If yes, how often have you had such a feeling? *(if never go
       to 5.1)*.
       O1 Often   O3 Sometimes   O5 Seldom   O7 Never

4.2   When this happens do you have the feeling that a lot of people are looking at you, or
       only a few people or just one person?

       O1 A lot of people   O2 A few   O5 One

4.3   When this happens (the feeling of being watched), do you then have the feeling that
       you are getting special attention?

       O1 Yes   O3 Possibly   O5 No

4.4   Could you give me an example of one time that you can remember when you had the
       feeling that you were being watched by other people? Why were they looking at
       you?
       ....
       ....

4.5   Why do you think you were being watched? *(if reasons are obvious go to 5.1)*

       O1 There was no real reason for it.
       O3There was not much of a reason for it (over-reaction)
       O5 There was a good reason for it, but it was an over-reaction
       O7 There were obvious reasons for it (normal reaction)

4.6   Where does the feeling of being watched occur?

       O1 Both in my neighbourhood and away from home
       O3 Only when I am somewhere else
       O5 Only in my neighbourhood
       O7 Doesn't apply, I never go far from home

4.7   Are the people who seem to be watching you acquaintances, strangers, or both?

O1 Both acquaintances and strangers   O3 Only strangers   O5 Only acquaintances

4.8   If tomorrow you would go to some public place, do you think it would happen to you again?

O1 yes certainly   O3 probably would   O5 probably not   O7 certainly not

4.9   How often do these sorts of ideas (4.1 – 4.8) happen?

O1 Once a year   O2 once a month   O3 once a week   O4 other

4.10   When they occur, do you doubt they are true?

O1 No doubt   O2 Some doubt   O3 A lot of doubt

4.11   Do they affect your daily life?   O0 No   O1 Yes

4.12   GLOBAL ASSESSMENT OF REFERENTIAL THINKING

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

5. REFERENTIAL THINKING (PART III being talked about)
People sometimes get the feeling in public places that people around them are talking about them.

5.1   Do you know that feeling? *(if no go to 5.3)*   O0 No   O1 Yes

5.2   How often do you have such a feeling?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

5.3   Did you ever have the feeling in public places of being laughed at?*

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

*If at 5.1 no is the response and at 5.3 never, then go to 5.6.
If 5.1, 5.3 or both had positive responses, then continue on*

5.4 Do they laugh of talk about you more than about others?

O1 Certainly   O3 Possibly   O5 No

5.5 Why do you think they are laughing or talking about you?

O1 They have no reason to do this
O3 Little reason, excessive reaction
O5 Some reason, but over-reaction
O7 Had quite good reasons, describes normal reaction

5.6 When you are in a public place, how often do you have the feeling that other people are making insinuations about you? *(if never go to 5.8)*

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

5.7 Could you give two examples of this?

....
....

Field code:
O1 Clearly pathological   O3 Probably pathological
O5 Probably normal   O7 Clearly normal

5.8 Does it sometimes seem that people around you are speaking in some kind of code that sounds like they are talking normally but are actually making nasty remarks about you? *(if No, go to 5.10)*

O5 No O1 Yes

5.9 How often does this sort of double talk happen around you?

O1 Often   O3 Sometimes   O5 Seldom

5.10 How often do these ideas (5.1 -5.9) occur?

O1 Once a year   O2 once a month   O3 once a week   O4 other

5.11 When they happen, do you ever doubt if they are true?

O1 No doubt   O2 Some doubt   O3 A lot of doubt

5.12 Do they affect your daily life?
....
....

5.13   Now I will ask you some questions that summarize the experiences we were just discussing. These are a feeling that some people have when they are in public places, such as a restaurant, out shopping, at a party, or just walking down the street. This is about the feeling of being watched as well as the feeling that people are talking about you.

		Often	Sometimes	Seldom	Never
a.	When I am around people, it seems like they know who I am, even if they had never met me. If yes, what is the reason for this?	O1	O2	O5	O7
b.	When I am around people, I feel as if they are looking at me.	O1	O2	O5	O7
c.	When I am around people, I have the feeling that they are talking about me.	O1	O2	O5	O7
d.	When I am around people, I feel that people are staring at me.	O1	O2	O5	O7
e.	When I am around people, I have the feeling that people do not like me because of the way they are looking at me.	O1	O2	O5	O7
f.	When I am around people, I have the feeling that people are making a fool of me.	O1	O2	O5	O7
g.	When I am around people, I have the feeling that they are saying nasty things about me.	O1	O2	O5	O7

5.14   GLOBAL ASSESSMENT OF REFERENTIAL THINKING (seeing meanings)

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

6. SUSPICIOUSNESS

Remember that this part of the interview asks what kind of person you are generally. Answer what is most typical for you as a person.

6.1   Some people tend to trust others quickly while others are less inclined to do so. How do you see yourself?

O1 Quick to trusting   O3 Moderately quick   O5 Slow to trust   O7 Not at all

6.2   People vary in their view of other people and how much they can be trusted. Here are two statements. :

1. Most people cannot be trusted. If they get the chance, they will misuse you.
2. Most people can, in principle, be trusted. If they get the chance, they do their best to help their fellows.

In which of these do you belief most?
O1 The first   O3 Both   O5 The second

6.3   Here is a list of feelings that people could have. Please indicate how often you have had each sort of feeling.

		Often	Sometimes	Seldom	Never
a.	I feel that I cannot really trust the people I know.	O1	O2	O5	O7
b.	I feel that people are more critical of me than I deserve.	O1	O2	O5	O7
c.	I feel that when I am out among people, I have to be on my guard.	O1	O2	O5	O7
d.	I feel that people blame me for things that are not my responsibility.	O1	O2	O5	O7

6.4   How often does that happen?

O1 Once a year   O2 once a month   O3 once a week   O4 other

6.5   Could you tell me for each of the following statements whether you: certainly agree, probably agree, probably disagree, certainly disagree.

		Certainly agree	Probably agree	Probably disagree	Certainly disagree
a.	It is probably safest never to trust someone.	O1	O3	O5	O7
b.	If I trust in people too much, sooner or later they still will let me down.	O1	O3	O5	O7
c.	If I do not take care, people will misuse me.	O1	O3	O5	O7
d.	People seem to lie to me often.	O1	O3	O5	O7
e.	If you have told someone a secret, sooner or later they will use the information you gave them to hurt you.	O1	O3	O5	O7
f.	I can stay resentful for some time.	O1	O3	O5	O7
g.	I have the feeling that I am the target of some sort of conspiracy.	O1	O3	O5	O7

6.6   Are there people who are especially determined to frustrate you and make your life difficult?   (if no, go to 6.8)

O1 Yes   O5No

6.6A   What makes you think that? How do they work against you?

....

Field code:

O1 Clearly pathological   O3 Probably pathological
O5 Probably normal   O7 Clearly normal

6.7 Do you have the feeling that you must take special precautions to protect yourself from others? *(if no, got 6.8)*

6.7A O1 Yes O5No

What sort of precautions do you take?

....

O1 None O3 A little O5 Some O7 A Lot

6.8 How do you get on with your neighbours? Have you had disagreements with them? Have they made a special point to create problems for you? Why did they do this?

O1 There are great problems that cannot be accounted for.
O3 There are problems, not easily accounted for.
O5 There are problems that appear justified.
O7 There are no problems.

6.9 Objective reasons for suspiciousness. You say: "..." Has something happened in your life that you view it this way?

O1 Nothing O2 A little O5 Some O7 A Lot

6.10 Do you think you are a suspicious person? O0 No O1 Yes

6.11 GLOBAL ASSESSMENT OF SUSPICIOUSNESS

O0 Absent O1 Mild O2 Moderate O3 Severe

7. RESTRICTED AFFECT

7.1 Can you tell me whether the following statements apply to you?

	Often	Sometimes	Seldom	Never
a. I like to hug people who are close to me.	O1	O2	O5	O7
b. If I am happy, I show it.	O1	O2	O5	O7
c. I can show my true feelings.	O1	O2	O5	O7
d. I am affected emotionally by such things as music or the beauty of nature.	O1	O2	O5	O7
e. I am sentimental.	O1	O2	O5	O7
f. If I like people, I let them know.	O1	O2	O5	O7

7.2 Could you say how often approximately per month that you have positive feelings? How often in these cases, are you not able to express positive feeling? Is that...

O1 More than 50%
O2 Cannot be expressed less than 50%
O3 Nearly never express them

7.3 **If restricted affect is present**   O1 Present   O2 Absent

You have said that you cannot (properly) express certain (positive) feelings. Are these feelings present, but you cannot express them, or are these feelings merely absent?
....

7.4 **If restricted affect is present: How often does this occur?**

O1 Every day   O2 Two or three times per week   O3 Once a week
O4 Once a month   O5 Less than once a month

For how long does this last: |__|__| days

7.5 **GLOBAL ASSESSMENT OF RESTRICTED AFFECT**

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

8   MAGICAL IDEATION

8.1 Could you tell me whether these statements apply to you?

		Certainly true	Probably true	Probably not true	Absolutely not true
a.	I think that I could read other people's thoughts, if I wanted to.	O1	O3	O5	O7
b.	Horoscopes work out so often that it could not be purely chance.	O1	O3	O5	O7
c.	Numbers such as 13 and 7 have a special meaning for me.	O1	O3	O5	O7
d.	Sometimes, I can predict the future.	O1	O3	O5	O7
e.	Good luck mascots protect me from evil.	O1	O3	O5	O7
f.	I have noticed that if I just think about something enough it will happen.	O1	O3	O5	O7
g.	I have the feeling that the spirits of the dead can influence the living.	O1	O3	O5	O7
h.	I believe in black magic.	O1	O3	O5	O7
i.	Accidents can be caused by mysterious forces.	O1	O3	O5	O7

8.2 Here is another list of statements. I would like to know how often you have had experiences like these.

		Often	Sometimes	Seldom	Never
a.	I can communicate with other people when they are not nearby.	O1	O2	O5	O7
b.	I can sense when something terrible will happen to people standing near me.	O1	O2	O5	O7
c.	I feel there is an evil spirit around me.	O1	O2	O5	O7
d.	My dreams work out.	O1	O2	O5	O7
e.	I have the feeling that other people can read my thoughts.	O1	O2	O5	O7

8.3 Many people think that there are things that can bring fortune or misfortune, such as seeing a black cat or Friday the 13th. Do you have similar ideas? *(if no, got 8.5).* *

O1 Yes　　O5 No

8.4 What are these ideas?

....

8.5 Many people do certain things to keep bad luck away or to bring them luck, such as holding a lucky charm or having a horseshoe. Do you have these sorts of things? *(score superstition only with a yes).* *

O1 Yes　　O5 No

8.6 What do you do to prevent bad luck?

....

* *If the answer to both 8.3 and 8.5 is NO, go to 8.9*

* *If 8.3 or 8.5 is answered YES, continue*

8.7 Do members of your family and friends think the same way about these things?

O0 No　　O1 Yes

8.8 Field code: deviation of the superstition from subcultural norms.

O1 Very divergent　　　　O3 Moderately divergent
O5 Mildly divergent　　　　O7 Not divergent

8.9 How often do these ideas (8.1-8.8) occur?

O1 Once a year　　O2 once a month　　O3 once a week　　O4 other

8.10 Did you doubt their accuracy when they occurred, or were you convinced by them?

O1 No doubt   O2 Some doubt   O3 a lot of doubt

8.11 Do they affect your daily life? O5 No   O1 Yes

....

8.12 GLOBAL ASSESSMENT OF MAGICAL IDEATION

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

9 ILLUSIONS

People sometimes have the experience of mistaking some object for a human or an animal. For example, when walking in the dark you could mistake a tree trunk for a person, or a stick on the ground for a snake. Or when driving at twilight or dusk and you glimpse a lamppost out of the corner of your eye, you could think it is a man standing by the road.

9.1 Does that ever happen to you?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

9.2 Some people sometimes think that they have heard sounds that are likely not real, such as crackling, a knock, or the sound of a bell. How often have you experienced that?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

9.3 How often has it happened that you thought you heard your name called out, but then realized that you imagined it?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

9.4 When it is very quiet, some people sometimes have the experience of hearing voices whispering to them or talking when no one is nearby. Does that ever happen to you? *(if no, got to 9.6)*

O1 Yes   O5 No

9.5 How often has this happened?

O1 Often   O3 Sometimes   O5 Seldom

9.6 Have you ever had the feeling that a particular person or force was near you when you saw no one? What kind of person, or force was that?

O1 Yes, a dead family member or close friend
O3 Yes, a religious experience
O5 Yes, some other experience.
O7 No

9.7   How often do you have that feeling?

O1 Often   O3 Sometimes   O5 Seldom

9.8   Here is a list of experiences, which people can sometimes have. How often do you have had such experiences?

		Often	Sometimes	Seldom	Never
a.	My hearing is so sensitive that ordinary sounds are nearly painful.	O1	O2	O5	O7
b.	People whom I know well look like strangers	O1	O2	O5	O7
c.	Ordinary colours are too harsh.	O1	O2	O5	O7
d.	Ordinary things appear to be unusually large or small.	O1	O2	O5	O7
e.	Well-known objects had an odd appearance.	O1	O2	O5	O7
f.	My eyesight is so sensitive that ordinary light hurts my eyes.	O1	O2	O5	O7

9.9   How often do these experiences (9.1- 9.8) occur?

O1 Once a year   O2 once a month   O3 once a week   O4 other

9.9A   How certain were you that they were accurate at the time they occurred?

O1 No doubt   O2 Some doubt   O3 A lot doubt

9.10   Do these experiences have an effect on your daily life?   O0 No   O1 Yes

9.11   GLOBAL ASSESSMENT OF ILLUSIONS

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

10 PSYCHOTIC PHENOMENA
    Note if present; ask follow-up questions (i.e. what was it and how often did this occur)

10.1   How often do your thoughts become tangled up?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

10.2   How often do your thoughts suddenly stop, so that you completely lose train of your thought?   *(if never, go to 10.4)*

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

10.3   Have you ever had the feeling that some external power or some force or other has stopped your thoughts or even taken them out of your head?

O1 Yes, it takes these thoughts from my mind
O3 Yes, it only stops these thoughts
O5 No

10.4   Does it ever happen that some ideas suddenly appear in your head that you have the feeling do not belong there? If yes, how often? *(if no, go to 10.8)*

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

10.5   How often do ideas suddenly appear in your head that you have the feeling that some external power or some force or other put them there? *(If never, go to 10.8)*.

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

10.6   Which power of force puts ideas in your head?

O1 God
O3 The devil
O5 Close family member or friend
O7 Some other, e.g. ....

10.7   How do you find these?

O1 Very out of the ordinary
O3 Moderately out of the ordinary
O5 Mildly out of the ordinary
O7 Not out of the ordinary

10.8   Sometimes, people have the feeling that their thoughts are so real that it is as if they were being spoken out loud? Does that ever happen to you? *(if no, go to 10.12)*

O1 Yes   O5 No

10.9   How often has that happened to you?

O1 Often   O3 Sometimes   O5 Seldom

10.10   Do you also have the feeling that other people can hear them?

O1 Yes   O2 Perhaps   O3 No

10.11   If present, how often does this experience (10.1 - 10.10) occur?

O1 Once a year   O2 once a month   O3 once a week   O4 other

10.12 How certain are you of the accuracy of these ideas/experiences?

O1 No doubt   O2 Some doubt   O3 A lot doubt

10.13 GLOBAL ASSESSMENT OF PSYCHOTIC SYMPTOMS

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

11 DEREALIZATION/DEPERSONALIZATION

11.1 Have you ever had the feeling that the surroundings and the people around you were unreal, as if you were looking through a kind of fog?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

11.2 Have you ever had an unreal feeling about yourself, as if you were not actually alive? As if you were a robot or a machine?

O1 Often   O2 Sometimes   O5 Seldom   O7 Never

11.3 Have you ever had the feeling that you had stepped out of your body?
(if no, go to 11.6)

O1 Yes   O5 No

11.4 How often do you have the feeling that you had stepped out of your body?

O1 Often   O2 Sometimes   O5 Seldom

11.5 Did this happen only when you were physically ill, or had used some medicine or drug?

O1 Yes   O5 No

11.6 If present, how often do these experiences (11.1 – 11.3) occur?

O1 Once a year   O2 once a month   O3 once a week   O4 other

How long did it last? _____

11.7 GLOBAL ASSESSMENT OF DEREALIZATION/DEPERSONALIZATION

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

<u>OBSERVATION DURING THE INTERVIEW</u>
NOTE: Remember that this section (12-15) must be scored based on the unstructured part of the discussions with the subject.

12 <u>GOAL-DIRECTNESS OF SPEECH/THOUGHT PROCESS</u>

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

Does the subject maintain his/her attention on the topic of the questions and does he/she answer in a direct and logical manner? Or does he/she wander off topic during the conversation? If yes, how often and how far does he/she stray from the topic at hand? Assess verbosity (that these wanderings ultimately come back on track) and vagueness (an inability to follow the subject's thought pattern clearly).

13 <u>ELEVATED ASSOCIATIVITY</u>

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

Organization of associations:
Did the subject's associations have some logical meaning for you as interviewer? Could you follow the train of thought? With many verbose people, the interviewer can nonetheless follow a wandering train of thought. This is much more difficult among others.

14 <u>POVERTY OF SPEECH</u>

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

How many words does he/she need to come up with in answer to the questions? How often do you need to prompt the subject?

15 <u>ODD/ECCENTRIC BEHAVIOR</u>

15.1 Motor system (posture, gait, movements)

O1 The motor system was clearly odd
O3 The motor system was moderately odd
O5 The motor system was mildly odd
O7 No signs of odd motor system

Was the subject's non-verbal behaviour odd or eccentric? Had he/she an anomalous physical posture? Had he/she any odd tics or other odd motor movements?

15.2 **Adequacy of subject's social behaviour**

O1 Social behaviour clearly odd
O3 Social behaviour moderately odd
O5 Social behaviour mildly odd
O7 No indicators of odd social behaviour.

Was the subject's social behaviour socially inadequate in one way or another? Was the subject too familiar, that is, too intrusive, staring, inappropriately enticing, flirty or hostile? Could you understand the subject's social hints or was something missing? Consider here also whether he/she talked to him/herself and whether he/she made inappropriate jokes.

15.3 **Clothing – is the subject adequately clothed?**

O1 Very Poor	Clothing and grooming very inadequate
O2 Poor	Clothing and grooming moderately inadequate
O3 Reasonable	Clothing and grooming somewhat inadequate
O4 Reasonable to good	Clothing and grooming adequate
O5 Good	Clothing and grooming very adequate

Is the subject tidy or clean? Keep in mind the subject's work, a farmer dresses differently than does an administrative clerk.

15.4 **GLOBAL ASSESSMENT OF ODDNESS**

O0 Absent   O1 Mild   O2 Moderate   O3 Severe

Community Assessment of Psychic Experiences (CAPE)

CAPE

EUGEI

STUDIE: EU GEI	Date of Birth
Subject number: |__| EU |__|__| - |__|__|__|__|	|__|__|-|__|__|-**1**|**9**|__|__|
Time interval: lifetime	Period – Replicat |**0**|__|-|**0**|__|
Interviewer: ……………………………………..	Date |__|__|-|__|__|-**2**|**0**|__|__|

Explanatory remarks on the questionnaire:

The CAPE is developed to measure certain feelings, thoughts and mental experiences. We assume that these feelings, thoughts and mental experiences are much more common in the general population than previously presumed, and that most people did experience similar feelings, thoughts and/or experiences somewhere during their lives.

The next pages are divided in Column A and B. In **Column A** you can indicate **how often** you experienced a certain feeling, thought or mental experience **during your life**. Please put a tick against the most applicable.

There are no right or wrong answers.

In case there where periods where you suffered more from certain feelings, thoughts or mental experiences, please answer the questions about the worst period during your life.

If you ticked "never", please go to the next question.

If you ticked "sometimes", "often" or "nearly always" please indicate in Column B how distressed you are by this experience at the corresponding question.

During your lifetime, how often did you experience a certain feeling, thought or mental experience?

Column A

Please indicate how distressed you were:

Column B

	Never	Sometimes	Often	Nearly always	Not dis-stressed	A bit dis-stressed	Quite dis-stressed	Very dis-stressed
1. Do you ever feel sad?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
2. Do you ever feel as if people seem to drop hints about you or say things with a double meaning?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
3. Do you ever feel that you are not a very animated person?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
4. Do you ever feel that you are not much of a talker when you are conversing with other people?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
5. Do you ever feel as if things in magazines or on TV were written especially for you?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
6. Do you ever feel as if some people are not what they seem to be?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
7. Do you ever feel as if you are being persecuted in some way?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
8. Do you ever feel that you experience few or no emotions at important events?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
9. Do you ever feel pessimistic about everything?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
10. Do you ever feel as if there is a conspiracy against you?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
11. Do you ever feel as if you are destined to be someone very important?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
12. Do you ever feel as if there is no future for you?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
13. Do you ever feel that you are a very special or unusual person?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
14. Do you ever feel as if you do not want to live anymore?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
15. Do you ever think that people can communicate telepathically?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
16. Do you ever feel that you have no interest to be with other people?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
17. Do you ever feel as if electrical devices such as computers can influence the way you think?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
18. Do you ever feel that you are lacking in motivation to do things?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
19. Do you ever cry about nothing?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
20. Do you belief in the power of witchcraft, voodoo or the occult?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4

During your lifetime, how often did you experience a certain feeling, thought or mental experience? **Please indicate how distressed you were:**

	Column A				Column B			
	Never	Sometimes	Often	Nearly always	Not dis-stressed	A bit dis-stressed	Quite dis-stressed	Very dis-stressed
21. Do you ever feel that you are lacking in energy?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
22. Do you ever feel that people look at you oddly because of your appearance?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
23. Do you ever feel that your mind is empty?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
24. Do you ever feel as if the thoughts in your head are being taken away from you?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
25. Do you ever feel that you are spending all your days doing nothing?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
26. Do you ever feel as if the thoughts in your head are not your own?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
27. Do you ever feel that your feelings are lacking in intensity?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
28. Have your thoughts ever been so vivid that you were worried other people would hear them?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
29. Do you ever feel that you are lacking in spontaneity?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
30. Do you ever hear your own thoughts being echoed back to you?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
31. Do you ever feel as if you are under the control of some force or power other than yourself?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
32. Do you ever feel that your emotions are blunted?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
33. Do you ever hear voices when you are alone?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
34. Do you ever hear voices talking to each other when you are alone?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
35. Do you ever feel that you are neglecting your appearance or personal hygiene?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
36. Do you ever feel that you can never get things done?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
37. Do you ever feel that you have only few hobbies or interests?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
38. Do you ever feel guilty?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
39. Do you ever feel like a failure?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
40. Do you ever feel tense?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
41. Do you ever feel as if a double has taken the place of a family member, friend or acquaintance?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4
42. Do you ever see objects, people or animals that other people cannot see?	O_1	O_2	O_3	O_4	O_1	O_2	O_3	O_4

APPENDIX IX

Childhood Experience of Care and Abuse (CECA) 'Interview Version'

CHILDHOOD EXPERIENCES OF CARE AND ABUSE INTERVIEW

EU GEI

I would now like to ask you some questions about your childhood and adolescence. We are interested in different experiences you may have had before you were 17 years of age. Some of the experiences I want to ask about may bring back upsetting or painful memories, so if at any time you do not wish to answer a question please say so. Of course, all information you provide will be treated in the strictest confidence. All questions in **bold** should be asked and additional probes used as appropriate.

I would like to begin by asking:

1. **Who were your main parent figures (or carers) before you were 17?** (i.e., who brought you up)

   Are your parents still alive?

   If **no**

2. **Did one or both of your parents die before you were 17?**

3. **Were you ever separated from a parent (longer than 6 months) before you were 17?**

   If **yes**   Which parent?

   How old were you at first separation?

   How long were you separated?

   What was the main reason for separation? **Probe for** parental illness, divorce (parents separated), work, never knew parent, own illness, boarding school, migration, other

4. **Did you ever change schools?** (other than change from primary to secondary)

5. **Were you ever expelled from school?**

6. **Did you ever run away from home?** (i.e., stayed away for more than two nights)

7. **Were you ever taken into care?** (i.e., children's home, fostered)

8. **Were there ever times when your family was significantly short of money?**

9. **Were your basic needs ever neglected?** (i.e., for food, clean clothing, etc.)

HOUSEHOLD DISCORD

10. Were there ever frequent arguments or extreme tensions between your parents?

If **YES** I would like to ask you a little more about this. To begin with:

Discord

Can you tell me how well generally your parents got on together? (nb clarify family arrangement being considered**)**

Do you think they were close? Did you ever see them show each other affection?

How often would they argue?

What was it like?

Would there be raised voices?

Was there any violence or throwing things?

Were the arguments in front of you?

Did you ever get involved?

Did your brothers and sisters?

Was there a lot of tension in the home?

Any periods when they stopped talking to each other?

Violence

Was there any violence in the home?

If **yes**   Who was involved?

What would happen?

Were there ever any injuries as a result?

Any hospital treatment?

Were police or anyone else in authority ever called?

Age, Duration

How old were you when you first became aware of the arguments, tensions, violence?

How long did it go on for?

How many times did it happen?

When did it end? Why did it end at that time?

Support

Did you tell anyone about it?

If **yes**   **When did you first tell someone?**

Were they helpful?

Were they sympathetic?

Was anyone particularly helpful? Who?

What did they do or say?

If applicable, ask about other family arrangements. If **yes** to 'ever frequent arguments or extreme tensions' for another family arrangement, then, repeat questions.

PSYCHOLOGICAL ABUSE

11. Were you ever tormented or treated cruelly by a parent or a member of household?

Did anyone try to frighten you?

Did anyone humiliate you? (e.g., belittle you in front of others, ridicule you)

Did you ever feel that punishments at home were totally unnecessary?

If **yes**   I would like to ask you a little more about this. To begin with:

Perpetrator   **Who was it that did that?** What did they do?

Frequency   **How often would that happen?**

Age, Duration   **When did it start?**
                         When did it end?

Support   **Did you tell anyone about it?**

          If **yes**   **When did you first tell someone?**
                       Were they helpful?
                       Were they sympathetic?
                       Was anyone particularly helpful? Who?
                       What did they do or say?

Did anyone else in the household treat you like this? If **yes,** repeat questions.

PHYSICAL ABUSE

12. Were you ever hit or slapped on a number of occasions, sufficient to cause harm?

   Did parents ever hit you?

   Was there ever any violence toward you in household?

   If **yes**   I would like to ask you a little more about these experiences. To begin with, can I ask:

Perpetrator	**Who hit you?**
	Did your parents hit you?
	Did anyone else in the household? Who?
	If **yes** **Was that slapping, or was it worse than that?**
	Did … … … ever hit you with anything, like a stick or a belt?
	If with hand, was it a slap or punch?
	Was it ever the head or face?
	Were you ever badly beaten? What happened?
	Were you ever hurt?
	Did you have bruises, cuts?
	Did you ever need hospital treatment or medical attention?
	What was the worst occasion?
	What happened?
	How many times was it like that?
	Was it very frightening?
	Was there anything about it that made you particularly frightened?
Age, Duration	**When did the violence start?**
	How often did it happen?
	How long did it go on for?
	When did it end? Why did it end at that time?
Support	**Did you tell anyone about it?**
	If **yes** **When did you first tell someone?**
	Were they helpful?
	Were they sympathetic?
	Was anyone particularly helpful? Who?
	What did they do or say?

Official Contact Did you see a doctor, nurse or social worker at that time?   What about the police?

Did anyone else in the household hit you? If **yes,** repeat questions.

SEXUAL ABUSE

13. **Did you ever have any unwanted sexual experiences?**

    Did anyone ever force or persuade you to have sexual intercourse against your wishes?

    Did you ever experience any other upsetting sexual experiences with a relative or person in authority?

    Were you ever in a situation where you were nearly involved in an unwanted sexual experience, but avoided it?

    If **yes**   I would like to ask you a little more about these experiences. To begin with, can I ask:

Perpetrator	**Who was involved?**
	Was … … … living in the same house as you when this happened?
	Can you tell me what happened?
Age, Duration	**How old were you when it first happened?**
	How long did it go on for?
	How many times did it happen?
	When did it end?
	Why did it end then?
Physical Contact	**Did it involve touching?**
	If **yes** **Where did … … … touch you?**
	Was it between your legs?
	Did you have to touch him (her)? Where?
	Did it involve masturbation?
	Was that to him (her) or you?
	If appropriate: Did it involve anything else, like assault with an implement?
	Did he (she) have sexual intercourse with you?
	If **no** **Did he (she) ask you to have sex with him (her)?**
	What did he (she) say?
	Was he (she) very persistent?
	How did you avoid him (her) touching you?
Support	**Did you tell anyone about it?**
	If **yes** **When did you first tell someone?**
	Were they helpful?
	Were they sympathetic?
	Was anyone particularly helpful? Who?
	What did they do or say?
Official Contact	**Did you see a doctor, nurse or social worker at that time?**
	What about the police?

To end with, I would like to ask three final questions. Before age 17 …

14. Were there adults you could go to with problems or to discuss feelings?

15. Were there others your age you could go to with problems or to discuss feelings?

16. Did you ever feel lonely for a significant period (i.e., 6 months or more)?

BULLYING

I am now going to ask you a few questions about teasing and bullying you may have experienced before 17 years of age. By the terms teasing and bullying we mean when people of a similar age to you:

Said mean and hurtful things or made fun of you or called you mean and hurtful names; Completely ignored or excluded you from their group of friends or left you out of things on purpose; Hit, kicked or shoved you, or locked you in a room; Told lies or spread rumours about you; Other hurtful things. (nb We don't call it teasing or bullying when it is done in a friendly or playful way.)

Did you have any such experiences before the age of 17?

If yes   Who by?

Can you tell me what happened? How often did it happen? Were you hurt at all?

When did it start? How old were you?

When did it stop?

Did you tell anyone about it?

If yes   **When did you first tell someone?**

Were they helpful?

Were they sympathetic?

Was anyone particularly helpful? Who?

What did they do or say?

Were there any other times that it happened?

If yes   Repeat above probes

What about the other way round? Did you ever find yourself teasing or bullying anybody?

NOTES

APPENDIX X

Life Events and Difficulties Schedule (LEDS)

LIST OF THREATENING EXPERIENCES

Time interval: 12 months pre-interview

Date of onset |__|__|-|__|__|-|__|__|__|__|

Interviewer: .... **Date** |__|__|-|__|__|-|**2**|**0**|__|__|

Each section refers to a particular group of life events or difficulties category but in the course of the interview a question may throw up information about another life event or difficulty and this should also be probed for carefully. Each section begins with obligatory opening questions (**in bold**) followed by probes that should only be used where a positive response has followed from the opening question. A brief description of any event or difficulty should be recorded.

nb Make full notes in the space provided (adding extra sheets if necessary) and complete ratings and consensus as soon as possible.

Notes

1. All questions relate to the 12-month period before interview.

2. For all difficulties, establish when they started and whether the level has changed.

3. FOR EVERY EVENT OR DIFFICULTY REMEMBER: Always establish the dates.

4. FOR EVERY EVENT OR DIFFICULTY REMEMBER: Probe for when problem was solved.

Introduction

Now, I would like to ask about the 12 month period before today. I would like to ask questions about things that may have happened to you, or to people close to you during that 12 month period.

Time Period _____ (12 months pre-interview) to _____ date of interview

The questions I ask will relate to your partner, children, siblings, parents, other members of household, and very close friends. So, before I ask about things that may have happened, can I begin by asking …

Friends, Confidants

- Is there anyone, either family or friends, that you feel very close to? Anyone else? (Note main confidants)

- If you had a problem of some sort, who would be the first person you would want to discuss it with?

- Who else can you confide in about personal things or worries?

- Are your answers the same for the 12 month period prior to onset, or have things changed since then?

Support

For each event or difficulty of probable moderate or marked threat, probe for support (if any) received …

Did you tell anyone about it?

If **yes** **When did you first tell someone?**

Were they helpful? Were they sympathetic?

Was anyone particularly helpful? Who?

What did they do or say?

I would like begin by asking you some questions about health (all of which relate to the 12 months before today) …

1) HEALTH, ACCIDENTS, DEATHS

Have you or anyone else been ill?

If **yes**   How serious was it? An emergency? Has anyone been off work because of it?

Have you or anyone else been admitted to or left hospital?

If **yes**   For what sort of illness or injury?

If it is a long term problem, **ASK**: is the problem still ongoing? (i.e., to point of onset for cases)

Have there been any other health problems at all that you might have overlooked?

[For example, have there been any surgical operations in the last 12 months? Have you had any bad news about an illness that has been going on for some time? Are there any long term health problems affecting anyone close?]

Have you or anyone else had an accident? (either a car accident, as a pedestrian, or at home)

If **yes**   What happened? How serious was it?

Has anyone close to you died? (Prompt for family, members of household, close friends)

If **yes**   Was it unexpected? Were you involved at all? Were you present?

Has anyone attempted suicide?

Has anyone else died or nearly died?

Can I now ask you some questions about relationships?

2) RELATIONSHIPS, INTERACTION CHANGES

If in a long-term relationship (6 months or more) or living with partner …

How well would you say that you and your partner get on in general?

Would you say there are any problems in your relationship?

How often do you and he/she argue or have rows? Have there been any other problems to do with money, work, relatives such as in laws or any sexual problems?

Have either of you ever considered a separation or divorce?

Have you been separated for any length of time?

If **yes**   For how long were you apart?

- 3 -

For all ...

Have any relationships ended?

If yes   Why did that happen when it did?

Have you lost contact with anyone who used to be close?

Is there anyone (else) whom you see much less of than you used to?

Have you had any other crises in the family or involving close friends? (e.g., major argument)

For all interaction change events, **probe:** Temporary? How long away? How often seen before the change? How much did you do together? How often do you see each other now? Distance, Telephone contact; How did you get along? How about now? Preparation? Evidence of rejection, guilt

3) BIRTHS, CHILDREN

Have you or anyone in the family or any close friends been expecting a baby or had a baby?

If yes   Was it planned? Did the birth go smoothly?

Have your children had any problems at school (e.g., truancy) or have they been a problem at home?

Do you worry about their friends?

Any other problems with your children?

I'd now like to move on to ask about work and/or studies ...

4) WORK, EDUCATION

If subject currently working ...

Do you enjoy your job?

Has anything notable happened at work?

Have you been off work at all or put into a new job or changed jobs?

How do you get on with your work colleagues? Have you had any trouble or difficulties with them?

Have you been out of work at all?

If yes   How long were you out of work? Were you looking for a job or did you prefer to stay at home? How did your previous job come to an end?

If subject not currently working ...

How did you last job come to an end? (Probe: redundancy, sacked, left for other reason)

If subject currently studying (or been studying in 12-month period) ...

Have you any problems at school or college? (Probe: with course work, fellow students, teachers)

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5) FINANCES, HOUSING

Have you had any money worries during …?

If yes Have you gone without things you really need? Have you had problems paying bills, the rent or mortgage? Have you had any debt problems? Have you tried to borrow from anybody?

Have you had any problems with your housing or neighbours?

Have you had any changes regarding housing or neighbours?

Any other housing problems?

Possible **probes**: Why did you move? What happened? Decision to move? Were there any difficulties? Have there been any difficulties since? Expense; Consequences; Did you feel cut off? (friends, babysitters, etc.); New friends; Impact on jobs; Any problem re: house/neighbours, etc.

6) CRISES, VICTIMISATION, LEGAL

Has there been any crises or emergencies of any kind?

Has anything valuable been stolen or lost or has your home been broken into in the last six months?

Has anyone been attacked or assaulted?

Have you witnessed an assault or other traumatic event?

Have you or anyone in your family had any contact with the police or lawyers or court, at all?

If yes Nature of offence; First time done it; First time in court; Other convictions; Verdict and sentence; Financial implications; What have other people said; What have they said at work; Driving affected (if licence lost, etc.); Implications re: other people involved; Were you afraid they would try to get their own back?

Sometimes people find they undergo difficulties connected with living in the UK, such as problems with visas, immigration authorities, or perhaps discrimination against you by others.

Have you experienced anything like this in this period?

A couple of final questions …

Sometimes people learn unexpected things about others close to them such as discovering their friend has been stealing, or their partner has been seeing someone else. **Has anything like this happened to you?** (e.g., something that changes your idea of the person's character?)

Anything else like that?

Any other event (positive or negative) that we've not discussed? Any other ongoing problems or difficulties?

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The Brief Core Schema Scales (BCSS)

The Brief Core Scheme Scales

EUGEI

STUDIE: EU GEI	Date of Birth
Subject number: |__| **EU** |__|__| - |__|__|__|__|__|	|__|__|-|__|__|-|**1**|**9**|__|__|
Time interval: Present	Period – Replicat |**0**|__|-|**0**|__|
Interviewer: ……………………………	Date |__|__|-|__|__|-|**2**|**0**|__|__|

This questionnaire lists beliefs that people can hold about themselves and other people. Please indicate whether you hold each belief (NO or YES). If you hold the belief then please indicate how strongly you hold it by circling a number (1–4). Try to judge the beliefs on how you have generally, over time, viewed yourself and others. Do not spend too long on each belief. There are no right or wrong answers and the first response to each belief is often the most accurate.

MYSELF	NO	YES	Believe it slightly	Believe it moderately	Believe it very much	Believe it totally
1. I am unloved	O_0	O_1	O_1	O_2	O_3	O_4
2. I am worthless	O_0	O_1	O_1	O_2	O_3	O_4
3. I am weak	O_0	O_1	O_1	O_2	O_3	O_4
4. I am vulnerable	O_0	O_1	O_1	O_2	O_3	O_4
5. I am bad	O_0	O_1	O_1	O_2	O_3	O_4
6. I am a failure	O_0	O_1	O_1	O_2	O_3	O_4
7. I am respected	O_0	O_1	O_1	O_2	O_3	O_4
8. I am valuable	O_0	O_1	O_1	O_2	O_3	O_4
9. I am talented	O_0	O_1	O_1	O_2	O_3	O_4
10. I am successful	O_0	O_1	O_1	O_2	O_3	O_4
11. I am good	O_0	O_1	O_1	O_2	O_3	O_4
12. I am interesting	O_0	O_1	O_1	O_2	O_3	O_4

OTHER PEOPLE	NO	YES	Believe it slightly	Believe it moderately	Believe it very much	Believe it totally
1. Other people are hostile	O_0	O_1	O_1	O_2	O_3	O_4
2. Other people are harsh	O_0	O_1	O_1	O_2	O_3	O_4
3. Other people are unforgiving	O_0	O_1	O_1	O_2	O_3	O_4
4. Other people are bad	O_0	O_1	O_1	O_2	O_3	O_4
5. Other people are devious	O_0	O_1	O_1	O_2	O_3	O_4
6. Other people are nasty	O_0	O_1	O_1	O_2	O_3	O_4
7. Other people are fair	O_0	O_1	O_1	O_2	O_3	O_4
8. Other people are good	O_0	O_1	O_1	O_2	O_3	O_4
9. Other people are trustworthy	O_0	O_1	O_1	O_2	O_3	O_4
10. Other people are accepting	O_0	O_1	O_1	O_2	O_3	O_4
11. Other people are supportive	O_0	O_1	O_1	O_2	O_3	O_4
12. Other people are truthful	O_0	O_1	O_1	O_2	O_3	O_4

Hamilton Rating Scale for Depression (HRSD)

Hamilton Depression Rating Scale

Subject number: 2EU02. |__|__|__|__| Date of Birth |__|__|-|__|__|-|**1**|**9**|__|__|

Time interval: Past 7 days

Interviewer: ....   Date |__|__|-|__|__|-|**2**|**0**|__|__|

Who should conduct this interview A researcher trained in the SCAN.

Interviewer: The first question for each item should be asked exactly as written. Often this question will elicit enough information about the severity and frequency of a symptom for you to rate the item with confidence. Follow-up questions are provided, however, for use when further exploration or additional clarification of symptoms is necessary. The specified questions should be asked until you have enough information to rate the item confidently. In some cases, you may also have to add your own follow-up questions to obtain necessary information.

Notes:

- *Time period:* Although the interview questions indicate that the ratings should be based on the patient's condition in the past week, some investigators using this instrument as a change measure may wish to base their ratings on the previous two to three days. If so, the questions may be preceded by "In the last couple of days..."

- *Loss of weight item:* It is recommended that this item be rated positively whenever the patient has lost weight relative to their baseline weight (i.e. before their current episode of depression), provided that they have not begun to gain back lost weight. Once the patient has begun to gain weight, however, even if they are still below their baseline, they should no longer be rated positively on this item.

- *Referent of 'usual' or 'normal' condition:* Several of the interview questions refer to the patient's usual or normal functioning. In some cases, such as when the patient has Dysthymia or Seasonal Affective Disorder, the referent should be to the last time they felt OK (i.e. not depressed or high) for at least two weeks.

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Overview: I'd like to ask you some questions about the past week.

- How have you been feeling since last (DAY OF WEEK)?
- IF OUTPATIENT: Have you been working? IF NOT: Why not?

1. Depressed Mood (sadness, hopeless, worthless)   SCORE: _____

- What's your mood been like this past week?

  - Have you been feeling down or depressed?
  - Sad? Hopeless?
  - In the last week, how often have you felt (OWN EQUIVALENT)? Every day? All day?
  - Have you been crying at all?

0	Absent
1	Indicated only on questioning
2	Spontaneously reported verbally
3	Communicated non-verbally (i.e., facial expression, posture, voice, tendency to weep)
4	Spontaneous verbal and non-verbal communication

nb IF SCORED 1-4 ABOVE, ASK: How long have you been feeling this way?

Notes

2. Work and Activities   SCORE: _____

- How have you been spending your time this past week (when not at work)?
  - Have you felt interested in doing (THOSE THINGS) or do you feel you have to push yourself to do them?
  - Have you stopped doing anything you used to do? IF YES: Why?
  - Is there anything you look forward to?
  - (AT FOLLOW-UP: has your interest been back to normal?)

0	No difficulty
1	Thoughts and feelings of incapacity, fatigue, or weakness related to activities, work or hobbies
2	Loss of interest in activity, hobbies or work – by direct report of the patient or indirect in listlessness, indecision and vacillation (feels he has to push self to work or activities)
3	Decrease in actual time spent in activities or decrease in productivity. In hospital, patient spends less than 3 hours/day in activities (hospital job or hobbies) exclusive of ward chores
4	Stopped working because of present illness. In hospital, no activities except ward chores, or fails to perform ward chores unassisted

Notes

3. Genital Symptoms (e.g., loss of libido, menstrual)   SCORE: _____

- How has your interest in sex been this week? (I'm not asking about performance, but your interest in sex – how much you think about it.)
- Has there been any change in your interest in sex (from when you were not depressed)?
- Is it something you've thought much about? IF NO: Is that unusual for you?

0	Absent
1	Mild
2	Severe

Notes

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4. Somatic Symptoms - Gastrointestinal

SCORE: _____

- How has your appetite been this past week? (What about compared to your usual appetite?)
- Have you had to force yourself to eat?
- Have other people had to urge you to eat?

0	None
1	Loss of appetite but eating without encouragement
2	Difficulty eating without urging

Notes

5. Loss of Weight

SCORE: _____

- Have you lost any weight since this (DEPRESSION) began?
  - o IF YES: How much?
  - o IF NOT SURE: Do you think your clothes are any looser on you?

0	No weight loss
1	Probable weight loss associated with depression
2	Definite (according to subject) weight loss
3	Not assessed

Notes

6. Insomnia: Early

SCORE: _____

- How have you been sleeping over the last week?
- Have you had any trouble falling asleep at the beginning of the night? (Right after you go to bed, how long is it taking you to fall asleep?)
- How many nights this week have you had trouble falling asleep?

0	No difficulty falling asleep
1	Complains of occasional difficulty falling asleep (i.e., more than 1-2 hours)
2	Complains of nightly difficulty falling asleep

Notes

7. Insomnia: Middle

SCORE: _____

- During the past week, have you been waking up in the middle of the night?
  - • IF YES: Do you get out of bed? What do you do? (Only to go to the bathroom?)
  - • When you get back in bed, are you able to fall right back asleep?
  - • Have you felt your sleeping has been restless or disturbed some nights?

0	No difficulty
1	Complains of being restless and disturbed during the night
2	Waking during the night (any getting out of bed, except to use toilet)

Notes

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8. Insomnia: Late   SCORE: _____

- What time have you been waking up in the morning
  for the last time, this past week?
    - IF EARLY: Is that with an alarm clock, or do you just
      wake up yourself?
    - What time do you usually wake up (that is, before
      you got depressed)?

0	No difficulty
1	Waking in early hours of morning, but goes back to sleep
2	Unable to fall asleep again if gets out of bed

Notes

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9. Somatic Symptoms: General   SCORE: _____

- How has your energy been this past week?
    - Have you been tired all the time?
    - This week, have you had any backaches,
      headaches, or muscle aches?
    - This week, have you felt any heaviness in your
      limbs, back or head?

0	None
1	Heaviness in limbs, back or head. Backache, headache, muscle aches, loss of energy and fatigability
2	Any clear cut symptom

Notes

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10. Feelings of Guilt   SCORE: _____

- Have you been especially critical of yourself this past
  week, feeling that you've done things wrong, let
  people down?
    - IF YES: What have your thoughts been?
    - Have you been feeling guilty about anything
      you've done or not done?
    - Have you thought that you've brought (THIS
      DEPRESSION) on yourself in some way?
    - Do you feel you're being punished by being sick?

0	Absent
1	Self-reproach; feels has let people down
2	Ideas of guilt or rumination over past errors or sinful deeds
3	Present illness is a punishment. Delusions of guilt
4	Hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

Notes

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11. Suicide   SCORE: _____

- This past week, have you had any thoughts that life is
  not worth living, or that you'd be better off dead?
  What about having thoughts of hurting or even killing
  yourself?
    - IF YES: What have you thought about?
    - Have you actually done anything to hurt yourself?

0	Absent
1	Feels life is not worth living
2	Wishes was dead or any thoughts of possible death to self
3	Suicidal idea or gesture
4	attempts at suicide

Notes

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12. Anxiety: Psychic

SCORE: _____

- Have you been feeling especially tense or irritable this past week?
- Have you been worrying about unimportant things, things you wouldn't ordinarily worry about? IF YES: Like what, for example?

0	No difficulty
1	Subjective tension and irritability
2	Worrying about minor matters
3	Apprehensive attitude apparent in face or speech
4	Fears expressed without questionning

Notes

13. Anxiety: Somatic

SCORE: _____

In this past week, have you had any of these physical symptoms? (READ LIST, PAUSING AFTER EACH FOR REPLY).

(Physiological concomitants of anxiety, such as GI (dry mouth, gas, indigestion, diarrhea, cramps, belching); C-V (heart palpitations, headaches); Respiratory (hyperventilating, sighing); Having to urinate frequently; Sweating).

- How much have these things been bothering you this past week? (How bad have they gotten? How much of the time, or how often, have you had them?)

0	Absent
1	Mild
2	Moderate
3	Severe
4	Incapacitating

nb Don't rate if clearly due to medication (E.g. dry mouth and imipramine)

Notes

14. Hypochondriasis

SCORE: _____

- In the last week, how much have your thoughts been focussed on your physical health or how your body is working (compared to your normal thinking)?
  - Do you complain much about how you feel physically?
  - Have you been asking for help for things you could really do yourself? IF YES: Like what, for example? How often has that happened?

0	Not present
1	Self-absorption (bodily)
2	Preoccupation with health
3	Frequent complaints, requests for help, etc.
4	Hypochondriacal delusions

Notes

15. Insight (rating based on observation)

SCORE: _____

0	Acknowledges being depression or ill OR not currently depressed
1	Acknowledges illness but attributes cause to bad food, climate, overwork, virus, need for rest, etc.
2	Denies being ill at all

Notes

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16. Retardation (rating based on observation)   SCORE: _____

(Slowness of thought and speech; impaired ability to
concentrate; decreased motor activity)

0	Normal speech and thought
1	Slight retardation at interview
2	Obvious retardation at interview
3	Interview difficult
4	Complete stupor

Notes

17. Agitation (Rating based on observation)   SCORE_____

0	– none
1	– fidgetiness
2	– playing with hands, hair etc
3	– moving about, can't sit still
4	– hand-wringing, nail biting, hair-pulling, biting of lips

TOTAL 17-ITEM DEPRESSION SCORE: _____

19. Depersonalisation and Derealisation (such as feelings of unreality and nihilistic ideas)

SCORE_____

In this past week, have you ever suddenly had the feeling that everything is unreal, or you're in a dream, or cut off from other people in some strange way? Any spacey feelings?

- IF YES: How bad has that been? How often this week has that happened?

0	– absent
1	– mild
2	– moderate
3	– severe
4	– incapacitating

19. Obsessional and Compulsive Symptoms   SCORE_____

In this past week, have there been things you've had to do over and over again, like checking the locks on the doors several times?

- IF YES: Can you give me an example?

Have you had any thoughts that don't make sense to you, but that keep running over and over in your mind?

- IF YES: Can you give me an example?

0	– absent
1	– mild
2	– severe

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APPENDIX XIII

Family Interview for Genetic Studies (FIGS)

FIGS Pedigree Data form

EU·GEI

STUDIE: EU GEI	Date of Birth
EU-GEI ID number*: |__|EU|__|__|-|__|__|__|__|__|	|__|__|-|__|__|-|**1**|**9**|__|__|
Time interval: lifetime	Period – Replicat |**0**|__|-|**0**|__|
Interviewer: ……………………………………	Date |__|__|-|__|__|-|**2**|**0**|__|__|

Dates of births (- and deaths) of:

-First degree family members: parents - (Half) siblings - children /and partner

From the angle of: ....(name patent)

Please note at 'information', if known,

- Part of multiple births (twin, triplet, etc.)
- Cause of death (if known, and if applicable)
- Mental disorders (e.g. depression, mania, psychosis, etc.)
- Alcohol- or substance abuse

FIGS ID	Surname and first name	Date of birth	Date of death	Information

Family Interview for Genetic Studies   **FIGS Pedigree**

Subject number: |__|EU|__|__|-|__|__|__|__|__|

Date |__|__|-|__|__|-|**2**|**0**|__|__|

Interviewer: ………………………………
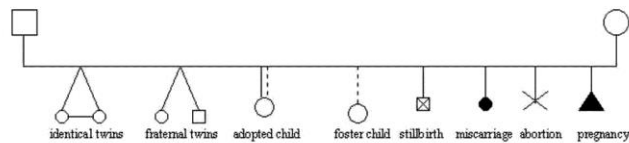
Please mark the core patient
With double lines, see for example:

identical twins   fraternal twins   adopted child   foster child   stillbirth   miscarriage   abortion   pregnancy
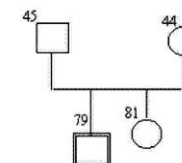
FIGS Pedigree, English, EU GEI, September 2010

FIGS Relative form

EUGEI

1. Pedigree ID number of relative

2. Is this relative the same person as the pedigree proband?*   O0 No   O1 Yes

3. Family history method or Family interview method   O1 Family history method
   O2 Family Interview method

4. Pedigree ID number of father of relative

5. Father mentioned at question 4 is biological father   O0 No   O1 Yes

6. Pedigree ID number of mother of relative

7. Mother mentioned at question 6 is biological mother   O0 No   O1 Yes

8. Birth year of relative

9. Gender of relative   O1 Man   O2 Woman

10. EU-GEI ID of relative if applicable

11. Part of multiple birth   O0 No   O1 Yes monozygotic
    O2 Yes dizygotic
    O3 Yes but unknown

12. Still alive?   O0 No   O1 Yes

13. Year of death

14. Cause of death   O1 Natural cause
    O2 Suicide
    O3 Unnatural

15. Ever had a depression (if no go to question 23)   O0 No   O1 Yes

16. Treatment for depression   O0 No   O1 GP
    O2 MHS/Social Worker
    O3 Other
    O4 Unknown

17. Treatment for depression setting   O0 None   O1 Inpatient
    O2 Outpatient
    O3 Medication only
    O4 Unknown

18. Age at onset of depression   ☐☐ Years

19. Number of episodes of depression   ☐☐

Please use EU-GEI number of the pedigree PROBAND: **when the pedigree contains more than one patient participating in EU-GEI the proband is the YOUNGEST patient; when control pedigrees contain more than one person participating in EU-GEI the YOUNGEST control is the proband. Please note healthy siblings of patients cannot be marked as proband.**

20. Duration of longest depression episode   ☐☐ Month(s)

21. Impairment by depression

O_1 Mild
O_2 Moderate
O_3 Severe
O_4 Unknown

22. Reliability of depression information

O_1 Good
O_2 Fair
O_3 Poor

23. Ever had a manic episode (if no go to question 31) O_0 No O_1 Yes

24. Treatment for mania O_0 No

O_1 GP
O_2 MHS/Social Worker
O_3 Other
O_4 Unknown

25. Treatment for mania setting O_0 None

O_1 Inpatient
O_2 Outpatient
O_3 Medication only
O_4 Unknown

26. Age at onset of mania   ☐☐ Years

27. Number of episodes of mania   ☐☐

28. Duration of longest manic episode   ☐☐ Month(s)

29. Impairment by mania

O_1 Mild
O_2 Moderate
O_3 Severe
O_4 Unknown

30. Reliability of mania information

O_1 Good
O_2 Fair
O_3 Poor

31. Ever had a psychotic episode (if no go to question 39) O_0 No O_1 Yes

32. Treatment for psychosis O_0 No

O_1 GP
O_2 MHS/Social Worker
O_3 Other
O_4 Unknown

33. Treatment for psychosis setting O_0 None

O_1 Inpatient
O_2 Outpatient
O_3 Medication only
O_4 Unknown

34. Age at onset of psychosis   ☐☐ Years

35. Number of episodes of psychosis   ☐☐

36. Duration of longest psychotic episode   ☐☐ Month(s)

37. Impairment by psychosis

O_1 Mild
O_2 Moderate
O_3 Severe
O_4 Unknown

38. Reliability of psychosis information

O_1 Good
O_2 Fair
O_3 Poor

39. Ever had an OCD episode (if no go to question 47)	O0 No	O1 Yes
40. Treatment for OCD	O0 No	O1 GP O2 MHS/Social Worker O3 Other O4 Unknown
41. Treatment for OCD setting	O0 None	O1 Inpatient O2 Outpatient O3 Medication only O4 Unknown
42. Age at onset of OCD		⬚⬚ Years
43. Number of episodes of OCD		⬚⬚
44. Duration of longest OCD episode		⬚⬚⬚ Month(s)
45. Impairment by OCD		O_1 Mild O_2 Moderate O_3 Severe O_4 Unknown
46. Reliability of OCD information		O_1 Good O_2 Fair
47. Ever diagnosed with autism (if no you can stop here)	O_0 No	O_1 Yes
48. Treatment for autism	O_0 No	O_1 GP O_2 MHS/Social Worker O_3 Other O_4 Unknown
49. Treatment for autism setting	O_0 None	O_1 Inpatient O_2 Outpatient O_3 Medication only O_4 Unknown
50. Age at diagnosis of autism		⬚⬚ Years
51. Impairment by autism		O_1 Mild O_2 Moderate O_3 Severe O_4 Unknown
52. Reliability of autism information		O_1 Good O_2 Fair O_3 Poor

SCREENINGSQUESTIONS (for the illness history of the relative)

The following questions only refer to relatives who were not adopted.

Did anyone:
1. Have problems with their nerves or emotions? Take medicine or see a doctor, psychiatrist or psychologist for it? Take lithium? **(Depression?)**
2. Feel very low for a couple of weeks or more, or have a diagnosis of depression? **(Depression)**
3. Attempt or complete suicide? **(Depression? Psychosis?)**
4. Seem overexcited (or manic) day and night, or have a diagnosis of mania or bipolar disorder? **(Mania?)**
5. Have visions, hear voices, or have beliefs that seem strange or unreal? **(Psychosis?)**
6. Have unusual or bizarre behaviour, or have a diagnosis of schizophrenia or other psychotic disorder? **(Psychosis?)**
7. Have trouble with the police, with completing school, or with keeping a job? **(Psychosis?)**
8. Was anyone hospitalized for psychiatric problems? **(Psychosis, Depression/ Mania)**
9. Have obsessive/ recurring thoughts and/or compulsions? **(OCD?)**
10. Have difficulty with social contact, communication and adaptation to new situations during childhood and adolescence? Ever been diagnosed with autism or an autism-related neurodevelopmental disorder **(Autism?)**

For each of these given a positive response in the General Screening, complete the symptom questions below for any suspected: Depression/Mania, Psychosis, and OCD.

DEPRESSION CHECKLIST

During depression...	YES	NO
1 a) was he/she depressed most of the day, nearly every day for as a week long as or more?		
1 b) did he/she lose interest in things or become unable to enjoy most things, for as long as a week?		
1 c) did he/she have a change in appetite or weight without trying to?		
1 d) did he/she have a change in sleep patterns (either too much or too little)?		
1 e) did he/she become unable to work, go to school, or take care of household responsibilities?		
1 f) did he/she move or speak more slowly than usual?		
1 g) did he/she pace or wring his/her hands		
1 h) did he/she have less energy or feel tired out?		
1 i) did he/she feel guilty, worthless or blame himself/herself?		
1 j) did he/she have trouble concentrating or making decisions?		
1 k) did he/she talk of death or suicide? Or try suicide?		
1 l) did he/she have visions, or hear voices, or have beliefs or behaviour that seem strange or unusual, at the same time as (symptoms above)?		

(**If Yes**, complete a Psychosis Checklist after this one.)

MANIA CHECKLIST

For most of the time day and night over several days, did he/she (more than usual)....	YES	NO
1 a) seem too happy/high/excited?		
1 b) become so excited or agitated it was impossible to converse with him/her?		
1 c) act very irritable or angry?		
1 d) need less sleep without feeling tired		
1 e) show poor judgement (e.g., spending sprees, sexual indiscretions?)		
1 f) behave in such a way as to cause difficulty for those around him/ her obnoxious/ manipulative)?		
1 g) feel that he/she had special gifts or powers		
1 h) become more talkative than usual?		
1 i) jump from one idea to another?		
1 j) become easily distracted?		
1 k) get involved in too many activities at work or school?		
1 l) have visions? Hear voices? have beliefs or behaviour that seem strange or unusual? at the same time as (above symptoms)?		

PSYCHOSIS CHECKLIST

Code for a single episode (best recalled, worst episode if possible). What were his/her unusual beliefs or experiences?

Did he/she ever:	YES	NO
1 a) believe people were following him/her, or that someone was trying to hurt or poison him/her?		
1 b) believe someone was reading his/her mind?		
1 c) believe he/she was under the control of some outside person or power or force?		
1 d) believe his/her thoughts were broadcast, or that an outside force took away his/ her thoughts or put thoughts into his/her head?		
1 e) have any other strange or unusual beliefs?		
1 f) see things that were not really there?		
1 g) hear voices or other sounds that were not real?		
1 h) speak in a way that was difficult to make sense of?		
1 i) seem to be physically stuck in one position, or move around excitedly without any purpose		
1 j) appear to have no emotions, or inappropriate emotions?		
2) How long did the <u>longest</u> of these experiences last?		

> **INTERVIEWER: If subject did NOT have any episode of Major Depression or Mania skip to OCD**

	YES	NO
3) When any (SX above) happened, did he/she also have the mood disturbance we discussed before, <u>at the same time?</u>		

IF NO SKIP TO OCD

OBSESSIVE COMPULSIVE DISORDER CHECKLIST

OBSESSIVE THOUGHTS are unwelcome and distressing ideas, thoughts, images or impulses that repeatedly enter your mind. They may seem to occur against the will of the person. They may be repugnant to the person, the person may recognize them as senseless.

Obsessive thoughts	YES	NO
1) Obsessions longer than 1 hours/day up to 8 hours/day		
2) Obsessions longer than 8 hours/ day		

COMPULSIONS are behaviours or acts that the person feels driven to perform although you he or she may recognize them as senseless or excessive. At times, the person may try to resist doing them but this may prove difficult. The person may experience anxiety that does not diminish until the behaviour is completed.

Compulsions	YES	NO
1 f) Compulsions longer than 1 hours/day up to 8 hours/day		
1 g) Compulsions longer than 8 hours/ day		

AUTISM CHECKLIST

	YES	NO
1 a) Does he/she like to engage in social interactions?		
1 b) Is he/she able to develop and maintain peer relations?		
1 c) Did he/she show any lack or delay in the development of spoken language?		
1 d) Does he/she use normal non-verbal behaviour during social interaction (eye contact, facial expression, body language)?		
1 e) Is he/she able to show empathy and understand emotions of other people?		
1 f) Does he/she show preoccupation with one or more stereotyped and restricted patterns of interest?		
1 g) Does he/she adhere inflexibly to specific, non-functional routines or rituals?		
1 h) Did these problems persist into adulthood [NB persistence into adulthood required for diagnosis]?		

The Cannabis Experiences Questionnaire (CEQ)

Cannabis Experiences Questionnaire   **EU:GEI**

STUDIE: EU GEI	Date of Birth
Subject number: |__| EU |__|__| - |__|__|__|__|	|__|__|-|__|__|-**1|9**|__|
Time interval:	Period – Replicat **|0|**__|-**|0**|__|
Interviewer:	Date |__|__|-|__|__|-**2|0**|__|__|

Instructions to researcher: *Please tick boxes as appropriate to indicate patient's responses. Please be reminded that some questions allow for more than one response.*

15.1 Have you ever smoked/used cannabis?   O1 Yes   O0 No

If answer is **NO,** *go to 15.17*

15.2 How old were you when you first tried cannabis? ....   ☐☐ years

15.3 Why did you first try cannabis? *(You can tick more than one box)*:

a)   My friends were using it.   O1 Yes   O0 No
b)   My family members were using it   O1 Yes   O0 No
c)   To feel better (to get relief from either physical or psychological discomfort)   O1 Yes   O0 No
d)   Other *(please explain) (not for data entry)*   O1 Yes   O0 No

_____

Instructions to researcher: *Please consider as current smokers all participants who report usually using/ smoking cannabis (incl. patients who have not smoked while inpatient/in prison and patients who report occasional use even if it is once every couple of years etc).*

15.4 Do you currently use cannabis?   O1 Yes   O0 No

If **Yes**, please answer *b*, if **No,** *go to 15.7*

b. If **YES**, why did you continue to use cannabis? *(You can tick more than one box)*:

a) I like the effect, it gives me a buzz   O1 Yes   O0 No
b) It makes me feel relaxed   O1 Yes   O0 No
c) It makes me feel less nervous and anxious   O1 Yes   O0 No
d) It makes me feel more sociable   O1 Yes   O0 No
e) Other *(please explain)*   O1 Yes   O0 No

_____

15.5 Would you like to stop using cannabis one day?   O1 Yes   O0 No

b. If yes, please explain *(not for data entry)*:

_____

15.6 Does/did cannabis affect your health in any way   O1 Yes   O0 No

b. If yes, please explain *(not for data entry)*:

_____

15.7 If you are not a current user, how long ago did you stop smoking cannabis?   [ ][ ] months

b. Why did you stop? please explain *(not for data entry)*:

_____

15.8 How do/did you mostly use cannabis?

    O1 I smoke/smoked it in a joint with tobacco

    O2 I smoke/smoked it in a joint without tobacco

    O3 I smoke/smoked it using a bong

    O4 I eat/ate or drink/drank it

    O5 Other (please explain): _____

15.9 How often do/did you use cannabis?

    O1 Every day

    O2 (More than) once a week

    O3 A few times each month

    O4 A few times each year

    O5 Only once or twice

15.10 When do/did you mostly use cannabis?

    O1 During the day

    O2 During the evening

    O3 During the day and evening

    O4 At weekends

    O5 During weekends and weekdays

15.11 Do you/did you mostly use cannabis:

    O1 Socially (with friends)

    O2 On my own

15.12 On average how much money per week do/did you usually spend on cannabis?

O1 Less than £2.50   (< €2.75)

O2 £2.50 - £5   (€2.75 - €5.50)

O3 £6 - £10   (€6.50 - €11)

O4 £11 - £15   (€12 – €16.50)

O5 £16 - £20   (€17.50 - €22)

O6 Above £20   (above €22)

15.13 What type of cannabis do/did you mostly use?

O1 Hash (cannabis resin/solid)

O2 Imported herbal cannabis

O3 Home-grown skunk/ Sensimilla

O4 Super skunk

O5 Other *(please state)*: _____

15.14. Why did you choose the above type? _____

15.15. How often have you had these experiences while smoking cannabis?

Please rate whether it was a good, bad or neutral experience. If rarely or never, ignore rating (good, bad, neutral) and go to next item.

	Rarely or never	From time to time	Sometimes	More often than not	Almost always	Good	Bad	Neutral
a) Fearful	O0	O1	O2	O3	O4	O1	O2	O0
b) Feel like going crazy/mad	O0	O1	O2	O3	O4	O1	O2	O0
c) Nervy	O0	O1	O2	O3	O4	O1	O2	O0
d) Suspicious	O0	O1	O2	O3	O4	O1	O2	O0
e) Feeling happy	O0	O1	O2	O3	O4	O1	O2	O0
f) Full of plans/ideas	O0	O1	O2	O3	O4	O1	O2	O0
g) Hearing voices	O0	O1	O2	O3	O4	O1	O2	O0
h) Able to understand the world better	O0	O1	O2	O3	O4	O1	O2	O0
i) Seeing visions	O0	O1	O2	O3	O4	O1	O2	O0

15.16 Life Time Cannabis History questionnaire

Instructions to researcher: *Please hand this section over to participant for completion. Explain to participant how to complete this part by using (a) as an example: If you were smoking cannabis when you were 15, were smoking 2-3 joints per day on average, you usually smoked hash and you only smoked by yourself.*

a) AGE RANGE: 0-11

i.	Did you use cannabis between the ages of 0 and 11?	O1 Yes O0 No
ii.	Frequency	O1 Every day
		O2 More than once a week
		O3 About once a week
		O4 About once/twice a month
		O5 A few times a year
		O6 About once a year
		O7 I have only used cannabis once or twice
iii.	Quantity *(average per day)*	O1 1 Joint
		O2 2 or 3 Joints
		O3 4 or more Joints
iv.	Mostly shared	O1 Yes O0 No
v.	Type	O1 Hash (cannabis resin/solid)
		O2 Imported Herbal cannabis
		O3 Home-grown skunk/Sensimilla
		O4 Super skunk
		O5 Other (please state):_____
vi.	Setting of use	O1 Socially (with friends)
		O2 On my own
		O3 Both

b) AGE RANGE: 12-16

i.	Did you use cannabis between the ages of 12 and 16?	O1 Yes O0 No
ii.	Frequency	O1 Every day
		O2 More than once a week
		O3 About once a week
		O4 About once/twice a month
		O5 A few times a year
		O6 About once a year
		O7 I have only used cannabis once or twice

iii.	Quantity *(average per day)*	O1 1 Joint
		O2 2 or 3 Joints
		O3 4 or more Joints

| iv. | Mostly shared | O1 Yes O0 No |

v.	Type	O1 Hash (cannabis resin/solid)
		O2 Imported Herbal cannabis
		O3 Home-grown skunk/Sensimilla
		O4 Super skunk
		O5 Other (please state):_____

vi.	Setting of use	O1 Socially (with friends)
		O2 On my own
		O3 Both

c) AGE RANGE: ABOVE THE AGE OF 17

| i. | Did you use cannabis from the age of 17 onwards? | O1 Yes O0 No |

ii.	Frequency	O1 Every day
		O2 More than once a week
		O3 About once a week
		O4 About once/twice a month
		O5 A few times a year
		O6 About once a year
		O7 I have only used cannabis once or twice

iii.	Quantity *(average per day)*	O1 1 Joint
		O2 2 or 3 Joints
		O3 4 or more Joints

| iv. | Mostly shared | O1 Yes O0 No |

v.	Type	O1 Hash (cannabis resin/solid)
		O2 Imported Herbal cannabis
		O3 Home-grown skunk/Sensimilla
		O4 Super skunk
		O5 Other (please state):_____

vi.	Setting of use	O1 Socially (with friends)
		O2 On my own
		O3 Both

Cannabis Experiences Questionnaire, English, EU GEI, 4-D, Sept 2010, Page 5

d) If your pattern of cannabis use has changed overtime, please state why? (not for data entry)

_____

e) Dependence screening for cannabis

Have you ever experienced 3 or more of the following characteristics?

	Lifetime		Last 12 months	
1. Tolerance, as defined by either of the following:				
a. A need for markedly increased amounts of the substance to achieve intoxication or desired affect.	O1 Yes	O0 No	O1 Yes	O0 No
b. Markedly diminished effect with continued use of the same amount of the substance	O1 Yes	O0 No	O1 Yes	O0 No
2. Withdrawal, as manifested by either of the following:				
a. The characteristic withdrawal syndrome for the substance	O1 Yes	O0 No	O1 Yes	O0 No
b. The same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms	O1 Yes	O0 No	O1 Yes	O0 No
3. The substance is often taken in larger amounts or over a longer period than was intended	O1 Yes	O0 No	O1 Yes	O0 No
4. There is a persistent desire or unsuccessful efforts to cut down or control substance use	O1 Yes	O0 No	O1 Yes	O0 No
5. A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects	O1 Yes	O0 No	O1 Yes	O0 No
6. Important social, occupational, or recreational activities are given up or reduced because of substance use	O1 Yes	O0 No	O1 Yes	O0 No
7. The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance	O1 Yes	O0 No	O1 Yes	O0 No

15.17 Instructions to researcher: Please ask for each drug: *Did you ever use?* If YES, please continue the questions concerning current and past use. *"Please also assess alcohol and drugs when applicable, (see separate Alcohol& Nicotine sheet)"*

^a **Dependence screening for any drugs:**

1. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of the substance to achieve intoxication or desired affect.
    b. Markedly diminished effect with continued use of the same amount of the substance
2. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for the substance
    b. The same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms
3. The substance is often taken in larger amounts or over a longer period than was intended
4. There is a persistent desire or unsuccessful efforts to cut down or control substance use
5. A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects
6. Important social, occupational, or recreational activities are given up or reduced because of substance use
7. The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance

^b **Definition 'Abuse'**

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one or more) of the following, occurring within a 12-month period:

1. Recurrent substance use resulting in a failure to fulfil major role obligations at work, school, or home
2. Recurrent substance use in situations in which it is physically hazardous
3. Recurrent substance-related legal problems
4. Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance

B. The symptoms

The symptoms have never met the criteria for Substance Dependence for this class of substance

Table Suppl.2: *Socio-demographics of Southwark and Lambeth Boroughs in London and comparison to Inner London (Office for National Statistics, Census 2011).*

	Lambeth Borough, London	Southwark Borough, London	Inner London
Sex	*(%)*	*(%)*	*(%)*
Males	49.8	49.5	49.8
Females	50.2	50.5	50.2
Age			
Under 18	19.9	20.5	20.4
18-24	10.6	11.9	11.3
25-44	43.0	49.7	41.1
45+	26.5	17.9	27.2
Religion			
Christian	53.1	52.5	45.4
Muslim	7.1	8.5	14.4
Other religion	11.8	12.3	16.2
No religion/Atheist	28.0	26.7	23.9
Employment status			
Unemployed	6.0	6.0	5.6
Economically inactive	23.0	27.1	27.9
Student	3.8	4.9	4.3
Part-time employed	9.5	9.9	9.4
Full-time employed	46.5	42.2	41.1
Self-employed	11.3	10.0	11.6

APPENDIX XVI

Additional Result Tables

Table Suppl.3: *Overall Model fit: Associations between total trauma (and all distinct trauma types) and total schizotypy*

	SIS-R total score Linear regression Adj.*		
	R-squared	*Prob>F*	*Adj. R-squared*
Total trauma	**0.234**	**<0.001**	**0.171**
Household discord	**0.208**	**<0.001**	**0.146**
Psychological abuse	**0.199**	**<0.001**	**0.135**
Psychological abuse *(all levels of severity)*	**0.227**	**<0.001**	**0.165**
Physical abuse	**0.219**	**<0.001**	**0.157**
Sexual abuse	**0.203**	**<0.001**	**0.138**
Sexual abuse *(all levels of severity)*	**0.241**	**<0.001**	**0.175**
Bullying	**0.216**	**<0.001**	**0.153**

Adj. for gender, age, ethnicity, employment status, religion, IQ. SIS-R, Structured Interview for Schizotypy Revised.

Table Suppl.4: *Frequencies for Individual CAPE items (psychotic-like symptoms)*

	Total sample CAPE			
	Never N (%)	*Sometimes N (%)*	*Often N (%)*	*Nearly Always N (%)*
Positive symptoms				
Q2: Double meaning	*105 (49.8%)*	*96 (45.0%)*	*10 (4.7%)*	*0*
Q5: Message from TV	*173 (82.0%)*	*37 (17.5%)*	*1 (0.5%)*	*0*
Q6: False appearance	*50 (23.7%)*	*136 (64.4%)*	*22 (10.4%)*	*3 (1.4%)*
Q7: Being persecuted	*164 (77.7%)*	*42 (19.9%)*	*3 (1.4%)*	*2 (0.9%)*
Q10: Conspiracy	*190 (90.0%)*	*20 (9.5%)*	*1 (0.5%)*	*0*
Q11: Being important	*119 (56.4%)*	*66 (31.3%)*	*14 (6.6%)*	*12 (5.7%)*
Q13: Being special	*104 (49.5%)*	*81 (38.6%)*	*13 (6.2%)*	*12 (5.7%)*
Q15: Telepathy	*153 (72.5%)*	*46 (21.8%)*	*6 (2.8%)*	*6 (2.8%)*
Q17: Influenced by devices	*92 (43.4%)*	*97 (45.8%)*	*20 (9.4%)*	*3 (1.4%)*
Q20: Voodoo	*148 (70.1%)*	*51 (24.2%)*	*7 (3.3%)*	*5 (2.4%)*
Q22: Odd looks	*132 (62.6%)*	*73 (34.6%)*	*5 (2.4%)*	*1 (0.5%)*
Q24: Thought withdrawal	*206 (97.6%)*	*3 (1.4%)*	*0*	*2 (0.9%)*
Q26: Thought insertion	*199 (94.3%)*	*11 (5.3%)*	*1 (0.5%)*	*0*
Q28: Thought broadcasting	*186 (88.6%)*	*23 (10.9%)*	*1 (0.5%)*	*0*
Q30: Thought echo	*193 (91.5%)*	*18 (8.5%)*	*0*	*0*
Q31: External control	*199 (94.3%)*	*10 (4.7%)*	*1 (0.5%)*	*1 (0.5%)*
Q33: Verbal hallucinations	*205 (97.2%)*	*6 (2.8%)*	*0*	*0*

Q34: Voices conversing	209 (99.5%)	2	(0.9%)	0		0	
Q41: Capgras	205 (97.2%)	5	(2.4%)	1	(0.5%)	0	
Q42: Visual hallucinations	204 (96.7%)	7	(3.3%)	0		0	
Negative symptoms							
Q3: Lack of enthusiasm	129 (61.1%)	65 (30.8%)		13 (6.2%)		4	(1.9%)
Q4: Not talkative	99 (47.1%)	88 (41.9%)		17 (8.1%)		6	(2.9%)
Q8: No emotion	131 (62.1%)	66 (31.3%)		13 (6.2%)		1	(0.5%)
Q16: No interest in others	118 (55.9%)	88 (41.7%)		5	(2.4%)	0	
Q18: Lack motivation	58 (27.5%)	129 (61.1%)		22 (10.4%)		2	(0.9%)
Q21: No energy	37 (17.5%)	144 (68.2%)		28 (13.3%)		2	(0.9%)
Q23: Empty mind	162 (77.1%)	47 (22.4%)		1	(0.5%)	0	
Q25: Lack of activity	144 (68.2%)	62 (29.4%)		4	(1.9%)	1	(0.5%)
Q27: Blunted feelings	158 (75.2%)	46 (21.9%		6	(2.9%)	0	
Q29: Lack of spontaneity	122 (57.8%)	79 (37.4%)		8	(3.8%)	2	(0.9%)
Q32: Blunted emotions	158 (74.8%)	51 (24.3%)		2	(0.9%)	0	
Q35: Lack of hygiene	135 (72.5%)	54 (25.6%)		2	(0.9%)	2	(0.9%)
Q36: Unable to terminate	103 (48.8%)	91 (43.1%)		14	(6.6%)	3	(1.4%)
Q37: Lack of hobby	125 (59.2%)	72 (34.1%)		9	(4.3%)	5	(2.4%)
Depression symptoms							
Q1: Sad	6 (2.8%)	185 (87.7%)		17	(8.1%)	3	(1.4%)
Q9: Pessimism	119 (56.4%)	82 (38.9%)		6	(2.8%)	4	(1.9%)
Q12: No future	160 (75.8%)	45 (21.3%)		5	(2.4%)	1	(0.5%)
Q14: Not worth living	176 (83.4%)	34 (16.1%)		0		1	(0.5%)
Q19: Frequently cry	147 (69.7%)	58 (27.5%)		5	(2.4%)	1	(0.5%)
Q38: Guilty	44 (20.8%)	142 (67.3%)		17	(8.1%)	8	(3.8%)
Q39: Failure	105 (50.0%)	88 (41.9%)		14	(6.7%)	3	(1.4%)
Q40: Feeling tense	39 (18.5%)	136 (64.4%)		34 (16.1%)		2	(0.9%)

CAPE, Community Assessment of Psychic Experiences.

Table Suppl.5: *Associations between total trauma (and all distinct trauma types) and positive schizotypy*

| | SIS-R Positive | | | | SIS-R Positive | | | |
| | Linear Regression Unadj. | | | | Linear regression Adj.* | | | |
	β coefficient	t- value	p - value	95 % CI	β coefficient	t- value	p - value	95 % CI
Total trauma	**.78**	**3.86**	**<0.001**	**.38 – 1.18**	**.52**	**2.52**	**0.012**	**.11 - .93**
Household discord	**.89**	**2.00**	**0.047**	**.01 – 1.77**	.50	1.15	0.251	-.36 - 1.36
Psychological abuse	**2.26**	**2.13**	**0.034**	**.17 – 4.34**	1.13	1.05	0.293	-.99 - 3.25
Psychological abuse (all levels of severity)	**1.94**	**2.54**	**0.012**	**-.44 – 3.44**	1.46	1.95	0.053	-.02 - 2.94
Physical abuse	**1.86**	**3.57**	**<0.001**	**.83 – 2.89**	**1.10**	**1.98**	**0.049**	**.01 - 2.19**
Sexual abuse	.15	0.18	0.856	-1.51 - 1.81	.09	0.11	0.915	-1.62 - 1.80
Sexual abuse (all levels of severity)	**1.31**	**2.32**	**0.021**	**.20 – 2.42**	**1.19**	**2.10**	**0.037**	**.07 - 2.31**
Bullying	**1.17**	**2.59**	**0.010**	**.28 – 2.07**	**.90**	**2.01**	**0.046**	**.01 - 1.78**

*Adj. for gender, age, ethnicity, employment status, religion, IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table Suppl.6: *Associations between total trauma (and all distinct trauma types) and negative/disorganised schizotypy*

	SIS-R Negative/Disorganised Linear Regression Unadj.				SIS-R Negative/Disorganised Linear regression Adj.*			
	β coefficient	t- value	p - value	95 % CI	β coefficient	t- value	p - value	95 % CI
Total trauma	**.46**	**3.03**	**0.003**	**.16 - .77**	**.36**	**2.30**	**0.023**	**.05 - .68**
Household discord	.22	0.66	0.511	-.44 - -.87	.24	0.73	0.468	.41 - .90
Psychological abuse	1.03	1.30	0.194	-.53 – 2.59	.58	0.71	0.478	-1.04 - 2.21
Psychological abuse (all levels of severity)	**1.75**	**3.11**	**0.002**	**.64 – 2.85**	**1.48**	**2.59**	**0.010**	**.35 - 2.60**
Physical abuse	**1.43**	**3.72**	**<0.001**	**.67 – 2.19**	**1.01**	**2.40**	**0.017**	**.18 - 1.84**
Sexual abuse	1.13	1.82	0.070	-.90 – 2.35	**1.39**	**2.13**	**0.035**	**.10 - 2.68**
Sexual abuse (all levels of severity)	**1.18**	**2.84**	**0.005**	**.36 – 1.99**	1.27	2.96	0.003	.42 - 2.12
Bullying	**.72**	**2.13**	**0.035**	**.53 – 1.29**	.59	1.71	0.089	-.09 - 1.26

*Adj. for gender, age, ethnicity, employment status, religion, IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table Suppl.7: *Association between separation from a parent and parental death experiences and positive and negative/disorganised schizotypy*

	SIS-R Positive Unadj.			SIS-R Positive Adj.*			SIS-R Negative/Disorganised Unadj.			SIS-R Negative/Disorganised Adj.*		
	β coeff.	p - value	95 % CI	β coeff	p - value	95 % CI	β coeff.	p - value	95 % CI	β coeff.	p - value	95 % CI
Death of a parent(s)	-.23	0.785	-1.86 –1.41	-.19	0.817	-1.79 - 1.42	.05	0.936	-1.7 - 1.27	-.01	0.997	-1.20 - 1.20
Separation from mother	.70	.261	-.53 - 1.94	-1.67	.792	-1.42 -1.08	.82	0.078	-.09 - 1.73	.25	0.603	-.69 - 1.19
Separation from father	*1.03*	*0.026*	*.13 – 1.94*	.11	.805	-.81 - 1.04	*1.10*	*0.001*	*.43 – 1.76*	.68	0.053	-.01 - 1.37

*Adj. for gender, age, ethnicity (White, Non-White), IQ. SIS-R, Structured Interview for Schizotypy Revised. CI, Confidence Interval

Table Suppl.8: Gender comparison of association between trauma types and positive schizotypy

	Positive schizotypy Males		Positive schizotypy Females		Childhood Trauma and Gender Interaction effect	
	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value
Total trauma	**.86**	**0.009**	**.72**	**0.007**	-.07	0.936
Household discord	1.27	0.082	.63	0.272	-.64	0.483
Psychological abuse	1.72	0.131	**2.17**	**0.040**	.45	0.771
Physical abuse	**2.91**	**<0.001**	.87	0.238	-2.03	0.052
Sexual abuse	-.61	0.592	**2.08**	**0.002**	**2.69**	**0.036**
Bullying	.48	0.479	**1.98**	**0.002**	1.49	0.104

Unadjusted scores

Table Suppl.9: Gender comparison of association between trauma types and negative/disorganised schizotypy

	Negative/Disorganised schizotypy Males		Negative/Disorganised Females		Childhood Trauma and Gender Interaction effect	
	β coeff.	p-value	β coeff.	p-value	β coeff.	p-value
Total trauma	.29	0.285	**.61**	**0.001**	1.04	0.300
Household discord	.14	0.811	.43	0.255	.28	0.674
Psychological abuse	1.73	0.059	**1.67**	**0.015**	-.06	0.958
Physical abuse	**1.55**	**0.015**	**1.21**	**0.011**	-.34	0.661
Sexual abuse	1.06	0.253	**1.53**	**<0.001**	.47	0.611
Bullying	.19	0.728	**1.04**	**0.014**	.84	0.217

Unadjusted scores

Table Suppl.10: *Associations between total trauma (and all distinct trauma types) and CAPE – Depressive subscale*

| | CAPE Depressive subscale | | | | CAPE Depressive | | | |
| | Linear Regression Unadjusted | | | | Linear regression Adjusted* | | | |
	β coefficient	t- value	p - value	95 % CI	β coefficient	t- value	p - value	95 % CI
Total trauma	.79	3.99	0.001	.40 – 1.19	.97	4.84	<0.001	.58 - 1.37
Household discord	.64	1.48	0.144	-.23 – 1.49	.57	1.29	0.198	-.30 -1.45
Psychological abuse	3.23	3.19	0.002	1.23 – 5.23	3.98	3.80	<0.001	1.92 - 6.05
Psychological abuse (all levels of severity)	2.47	3.38	0.001	1.03 – 3.91	2.87	2.88	<0.001	1.41 - 4.32
Physical abuse	1.02	1.96	0.051	-.00 – 2.04	1.82	3.28	0.001	.73 - 2.92
Sexual abuse	2.61	3.15	0.002	3.97 – 4.23	3.00	3.51	0.001	1.31 – 4.69
Sexual abuse (all levels of severity)	1.50	2.72	0.007	.41 – 2.58	1.80	3.19	0.002	.68 - 2.91
Bullying	1.01	2.27	0.024	.13 – 1.88	1.29	2.86	0.005	.40 - 2.18

*Adj. for gender, age, ethnicity (White, Black Caribbean/Black African, Other), IQ. CAPE, Community Assessment of Psychic Experiences.CI, Confidence Interval

Table Suppl.11: *Associations between total trauma (and all distinct trauma types) and CAPE Positive and CAPE Negative (Unadjusted values)*

| | CAPE Positive | | | | CAPE Negative | | | |
| | Linear Regression Unadjusted | | | | Linear Regression Unadjusted | | | |
	β coefficient	t- value	p - value	95 % CI	β coefficient	t- value	p - value	95 % CI
Total trauma	1.27	4.53	<0.001	.72 – 1.82	1.03	3.29	0.001	.41 – 1.66
Household discord	1.65	2.68	0.008	.44 – 2.56	.35	0.51	0.607	-1.00 – 1.71
Psychological abuse	.89	0.60	0.546	-2.02 – 3.81	2.55	1.58	0.116	-.64 – 5.75
Psychological abuse (all levels of severity)	.99	0.93	0.352	-1.11 – 3.10	3.04	2.69	0.009	-.75 – 5.33
Physical abuse	4.17	6.04	<0.001	2.81 – 5.54	1.61	1.98	0.049	.01 – 3.21
Sexual abuse	.95	0.81	0.417	-1.35 – 3.24	3.39	2.69	0.008	.90 – 5.88
Sexual abuse (all levels of severity)	1.57	2.00	0.047	.02 – 3.11	1.99	2.32	0.021	.30 – 2.68
Bullying	1.30	2.04	0.042	.04 – 2.55	1.73	2.48	0.014	.35 – 3.11

CAPE, Community Assessment of Psychic Experiences.CI, Confidence Interval

Table Suppl.12: *For interaction effects of genetic risk and trauma types and positive & negative/disorganised schizotypy*

Genetic risk	Main effects on SIS-R Negative Unadj.			Trauma – dichotomised Interaction effect - Unadj.			Main effects on SIS-R Positive Unadj.			Trauma – dichotomised Interaction effect - Unadj		
	β coeff.	p - value	95 % CI	β coeff.	p - value	95 % CI	β coeff.	p - value	95 % CI	β coeff.	p - value	95 % CI
Psychosis	**1.66**	**0.027**	**.19 – 3.13**	-.11	0.952	-3.77 – 3.55	**2.38**	**0.020**	**.38 – 4.37**	2.52	0.308	-2.35 – 7.40
any Mental Illness	**.70**	**0.033**	**.06 – 1.35**	.47	0.509	-.93 – 1.87	.71	0.155	-.17 –1.59	**2.01**	**0.031**	**.19 – 3.90**

SIS-R, Structured Interview for Schizotypy Revised.CI, Confidence Interval

Table Suppl.13: *Logistic regressions for total trauma and each of the trauma types among genders*

	Males 20 % top scorers SIS-R Unadj.			Males 20 % top scorers SIS-R Adj.*			Females 20 % top scorers SIS-R Unadj.			Females 20 % top scorers SIS-R Adj.*		
	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI
Total trauma	**1.64**	**0.031**	**1.05 – 2.56**	1.36	0.213	.84 -2.21	**2.25**	**0.001**	**1.40 - 3.61**	**2.00**	**0.006**	**1.22 – 3.29**
Household discord	2.55	0.052	.99 – 6.55	2.31	0.114	.82 - 6.54	2.01	0.157	.76 – 5.32	1.70	0.318	.60 – 4.87
Psychological abuse (all levels of severity)	3.58	0.075	0.88 – 14.54	4.54	0.060	.94 - 21.56	**4.60**	**0.035**	**1.11 – 18.99**	3.65	0.121	.71 – 18.77
Physical abuse	**5.98**	**0.001**	**2.13 – 16.78**	**3.59**	**0.029**	**1.14 - 11.27**	**4.31**	**0.007**	**1.48 – 12.54**	**3.84**	**0.038**	**1.07 –13.77**
Sexual abuse (all levels of severity)	1.30	0.723	.30 – 5.65	1.21	0.807	.26 - 5.75	**4.33**	**0.005**	**1.57 – 11.96**	**3.29**	**0.035**	**1.09– 9.92**
Bullying	1.02	0.968	.41 – 2.53	.84	0.742	.30 - 2.33	**5.02**	**0.002**	**1.82 – 13.83**	**5.43**	**0.003**	**1.78 – 16.55**

* age, ethnicity (White, Non-white), IQ. SIS-R, Structured Interview for Schizotypy Revised.OR, Odds Ratio, CI, Confidence Interval

Table Suppl.14: Association between trauma types and top 20% and top 10% of schizotypy scorers (adjusted for genetic risk for psychosis)

	20 % top scorers SIS-R total Unadj.			20 % top scorers SIS-R total Adj.*			10 % top scorers SIS-R total Unadj.			10 % top scorers SIS-R total Adj.**		
	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI	OR	p - value	95 % CI
Total trauma	1.87	<0.001	1.36 – 2.57	1.81	0.001	1.29 - 2.53	2.05	<0.001	1.39 – 3.03	1.92	0.002	1.28 - 2.88
Household discord	2.02	0.036	1.05 – 3.91	2.09	0.036	1.05 - 4.18	2.34	0.052	.99 – 5.53	2.25	0.067	.94 - 5.36
Psychological abuse (all levels of severity)	4.08	0.005	1.52 – 10.97	4.16	0.007	1.48 - 11.71	6.59	0.001	2.26 – 19.12	8.06	<0.001	2.57 - 25.24
Physical abuse	5.21	<0.001	2.51 – 10.81	4.92	<0.001	2.29 - 10.57	6.18	<0.001	2.53 – 15.10	5.40	<0.001	2.18 – 13.33
Sexual abuse (all levels of severity)	2.26	0.039	1.04 – 4.91	2.54	0.031	1.09 - 5.93	3.60	0.007	1.42 – 9.12	3.06	0.022	1.18 – 7.95
Bullying	2.26	0.017	1.16 – 4.40	1.69	0.152	.82 - 3.49	3.45	0.005	1.44 – 8.24	2.93	0.020	1.18 – 7.26

*Adj. for gender, age, genetic risk for psychosis ('broad definition')
** Adj. genetic risk for psychosis ('broad definition'). SIS-R, Structured Interview for Schizotypy Revised.OR, Odds Ratio, CI, Confidence Interval

Table Suppl.15: *Associations between different types of childhood trauma and schizotypy total scores, split into total, direct and indirect effects/pathways via possible mediators - for males*

Males	Top 20% of schizotypy Unadj.				Top 20% of schizotypy Adj.*			
	Total effect	**Direct effect**	**Indirect effect**	**% mediated**	**Total effect**	**Direct effect**	**Indirect effect**	**% mediated**
Psychological abuse	OR, p-value, 95%CI							
All mediators	OR=4.47 p=0.066 (.91-22.05)	OR=2.19 p=0.336 (.44-10.85)	OR=2.04 p=0.128 (.81-5.11)	48%	OR=4.71 p=0.097 (.76-29.34)	OR=2.56 p=0.315 (.41-16.01)	OR=1.84 p=0.144 (.81-4.16)	39%
Negative beliefs - Others	OR=4.16 p=0.063 (.92-18.71)	OR=3.50 p=0.099 (.79-15.55)	OR=1.19 p=0.478 (.74-1.91)	12%	OR=4.63 p=0.074 (.81-24.94)	OR=4.04 p=0.101 (.79-21.41)	OR=1.15 p=0.549 (.73-1.80)	9%
Negative beliefs - Self	OR=3.83 p=0.073 (.88-16.72)	OR=2.93 p=0.154 (.67-12.79)	OR=1.31 p=0.281 (.80-2.134)	20%	OR=4.25 p=0.084 (.82-21.91)	OR=3.36 p=0.144 (.66-17.05)	OR=1.26 p=0.356 (.77-2.09)	16%
Depression score	OR=4.13 p=0.068 (.90-18.90)	OR=2.22 p=0.307 (.48-10.30)	OR=1.86 p=0.075 (.94-3.67)	44%	OR=4.50 p=0.077 (.85-23.81)	OR=2.56 p=0.268 (.48-13.54)	OR=1.76 p=0.100 (.90-3.44)	37%
Cannabis use	OR=3.52 p=0.078 (.87-14.32)	OR=3.55 p=0.077 (.87-14.53)	OR=.99 p=0.887 (.88-1.12)	0%	OR=3.74 p=0.091 (.81-17.27)	OR=3.77 p=0.089 (.81-17.48)	OR=.99 p=0.874 (.89-1.10)	0%
Life events	OR=3.63 p=0.074 (.88-14.94)	OR=3.58 p=0.077 (.87-14.73)	OR=1.01 p=0.847 (.87-1.16)	1%	OR=3.95 p=0.084 (.83-18.86)	OR=3.90 p=0.087 (.82-18.52)	OR=1.01 p=0.853 (.88-1.17)	1%
Physical abuse								
All mediators	OR=7.62 p=0.001 (2.38-24.39)	OR=3.69 p=0.025 (1.18-11.58)	OR=2.06 p=0.042 (1.03-4.14)	36%	OR=4.45 p=0.021 (1.25-15.92)	OR=2.22 p=0.215 (.63-7.84)	OR=2.00 p=0.041 (1.03-3.92)	47%
Negative beliefs - Others	OR=6.67 p=0.001 (2.29-21.22)	OR=4.95 p=0.004 (1.68-14.56)	OR=1.41 p=0.100 (.94-2.12)	18%	OR=4.18 p=0.021 (1.24-14.15)	OR=3.11 p=0.063 (.94-10.28)	OR=1.34 p=0.149 (.90-2.02)	21%
Negative beliefs - Self	OR=7.06 p=0.001 (2.32-21.51)	OR=5.02 p=0.003 (1.73-14.60)	OR=1.40 p=0.143 (.89-2.21)	17%	OR=4.26 p=0.017 (1.29-14.00)	OR=3.07 p=0.059 (.96-9.87)	OR=1.38 p=0.178 (.86-2.23)	22%
Depression score	OR=7.24 p=0.001 (2.33-22.47)	OR=4.39 p=0.009 (1.45-13.28)	OR=1.65 p=0.050 (1.00-2.72)	25%	OR=4.55 p=0.016 (1.32-15.63)	OR=2.60 p=0.121 (.78-8.70)	OR=1.75 p=0.056 (.98-3.12)	37%
Cannabis use	OR=6.03 p=0.001 (2.12-17.14)	OR=7.03 p=0.001 (2.30-21.52)	OR=.86 p=0.369 (.61-1.20)	0%	OR=3.58 p=0.028 (1.14-11.20)	OR=4.26 p=0.021 (1.24-14.57)	OR=.84 p=0.420 (.55-1.28)	0%
Life events	OR=6.14 p=0.001 (2.16-17.43)	OR=6.21 p=0.001 (2.18-17.68)	OR=.99 p=0.843 (.88-1.11)	0%	OR=3.76 p=0.022 (1.21-11.71)	OR=3.86 p=0.020 (1.23-12.06)	OR=.97 p=0.712 (.85-1.11)	0%

*Adj. for age, ethnicity (White, Non-White), genetic risk for psychosis. OR, Odds Ratio. CI, Confidence Interval

Table Suppl.16: *Associations between different types of childhood trauma and schizotypy total scores, split into total, direct and indirect effects/pathways via possible mediators - for females*

Females	Top 20% of schizotypy Unadj.				Top 20% of schizotypy Adj.*			
	Total effect	**Direct effect**	**Indirect effect**	**% mediated**	**Total effect**	**Direct effect**	**Indirect effect**	**% mediated**
Total trauma	OR, p-value, 95%CI							
All mediators	OR=2.78 p<0.001 (1.59-4.88)	OR=2.18 p=0.010 (1.20-3.93)	OR=1.28 p=0.138 (.92-1.77)	24%	OR=2.56 p=0.002 (1.42-4.64)	OR=2.06 p=0.030 (1.07-3.96)	OR=1.24 p=0.231 (.89-1.78)	23%
Negative beliefs - Others	OR=2.57 p<0.001 (1.52-4.33)	OR=1.21 p=0.002 (1.33-3.66)	OR=1.16 p=0.103 (.97-1.40)	16%	OR=2.31 p=0.003 (1.34-3.40)	OR=12.09 p=0.007 (1.22-3.57)	OR=1.10 p=0.267 (.92-1.32)	12%
Negative beliefs - Self	OR=2.49 p=0.001 (1.48-4.18)	OR=2.28 p=0.001 (1.37-3.80)	OR=1.09 p=0.221 (.95-1.25)	9%	OR=2.30 p=0.003 (1.33-3.98)	OR=2.07 p=0.008 (1.21-3.54)	OR=1.11 p=0.197 (.95-1.31)	13%
Depression score	OR=2.32 p=0.001 (1.41-3.81)	OR=2.00 p=0.007 (1.21-3.29)	OR=1.16 p=0.102 (.97-1.38)	18%	OR=2.08 p=0.005 (1.24-3.49)	OR=1.76 p=0.038 (1.03-3.00)	OR=1.18 p=0.100 (.97-1.45)	23%
Cannabis use	OR=2.25 p=0.001 (1.40-3.62)	OR=2.34 p=0.001 (1.38-1.30)	OR=.96 p=0.797 (.71-1.30)	0%	OR=2.07 p=0.005 (1.25-3.45)	OR=2.13 p=0.008 (1.22-3.71)	OR=.97 p=0.871 (.71-1.33)	0%
Life events	OR=2.25 p=0.001 (1.40-3.61)	OR=2.25 p=0.001 (1.40-3.61)	OR=1.00 p=0.998 (1.00-1.00)	0%	OR=2.10 p=0.004 (1.26-3.48)	OR=2.08 p=0.004 (1.26-3.46)	OR=1.01 p=0.826 (.95-1.06)	1%
Physical abuse								
All mediators	OR=7.27 p=0.004 (1.85-28.33)	OR=2.88 p=0.119 (.76-10.85)	OR=2.52 p=0.054 (.99-6.42)	47%	OR=4.67 p=0.046 (1.03-21.23)	OR=2.26 p=0.301 (.48-10.58)	OR=2.07 p=0.113 (.84-5.08)	47%
Negative beliefs - Others	OR=5.15 p=0.007 (1.56-16.98)	OR=2.82 p=0.086 (.86-9.20)	OR=1.82 p=0.025 (1.08-3.09)	37%	OR=3.09 p=0.108 (.78-12.28)	OR=1.89 p=0.370 (.47-7.64)	OR=1.63 p=0.070 (.96-2.78)	43%
Negative beliefs - Self	OR=5.41 p=0.005 (1.66-17.67)	OR=4.16 p=0.017 (1.30-13.33)	OR=1.30 p=0.166 (.90-1.89)	16%	OR=3.95 p=0.046 (1.03-15.19)	OR=3.34 p=0.077 (.88-12.69)	OR=1.18 p=0.457 (.76-1.84)	12%
Depression score	OR=4.58 p=0.010 (1.44-14.56)	OR=3.38 p=0.038 (1.07-10.66)	OR=1.35 p=0.109 (.93-1.97)	20%	OR=3.18 p=0.082 (.86-11.69)	OR=2.37 p=0.189 (.64-8.79)	OR=1.34 p=0.152 (.90-2.01)	25%
Cannabis use	OR=4.69 p=0.005 (1.58-13.93)	OR=4.13 p=0.012 (1.37-12.44)	OR=1.13 p=0.369 (.86-1.50)	8%	OR=3.20 p=0.070 (.91-11.32)	OR=2.86 p=0.107 (1.80-10.26)	OR=1.12 p=0.436 (.84-1.49)	10%
Life events	OR=4.31 p=0.007 (1.48-12.56)	OR=4.40 p=0.007 (1.49-12.98)	OR=.98 p=0.802 (.84-1.14)	0%	OR=3.18 p=0.071 (.90-11.16)	OR=3.35 p=0.061 (.94-11.88)	OR=.95 p=0.578 (.79-1.14)	0%

Sexual abuse								
All mediators	OR=7.90 p=0.002 (2.14-29.13)	OR=5.67 p=0.009 (1.53-20.31)	OR=1.42 p=0.356 (.67-2.99)	17%	OR=5.62 p=0.012 (1.46-21.70)	OR=3.70 p=0.069 (1.46-21.70)	OR=1.52 p=0.299 (.69-3.33)	24%
Negative beliefs - Others	OR=5.88 p=0.003 (1.85-18.64)	OR=4.62 p=0.008 (1.49-14.29)	OR=1.27 p=0.269 (.83-1.95)	14%	OR=4.14 p=0.022 (1.23-13.96)	OR=3.53 p=0.039 (1.06-11.72)	OR=1.17 p=0.442 (.78-1.77)	11%
Negative beliefs - Self	OR=5.60 p=0.003 (1.81-17.30)	OR=4.58 p=0.007 (1.52-13.80)	OR=1.22 p=0.251 (1.87-1.73)	11%	OR=4.43 p=0.013 (1.37-14.35)	OR=3.61 p=0.030 (1.13-11.50)	OR=1.23 p=0.295 (.84-1.80)	14%
Depression score	OR=4.88 p=0.004 (1.67-14.27)	OR=3.94 p=0.011 (1.36-11.36)	OR=1.24 p=0.170 (.91-1.68)	13%	OR=3.70 p=0.026 (1.17-11.71)	OR=3.00 p=0.058 (.96-9.37)	OR=1.23 p=0.222 (.88-1.73)	16%
Cannabis use	OR=4.27 p=0.005 (1.54-11.86)	OR=3.74 p=0.014 (1.31-10.67)	OR=1.14 p=0.397 (.84-1.55)	9%	OR=3.30 p=0.032 (1.11-9.84)	OR=2.99 p=0.054 (.98-9.13)	OR=1.10 p=0.485 (.84-1.45)	8%
Life events	OR=4.36 p=0.005 (1.57-12.07)	OR=4.77 p=0.004 (1.65-13.76)	OR=.91 p=0.510 (.70-1.19)	0%	OR=3.36 p=0.028 (1.14-9.93)	OR=3.13 p=0.047 (1.01-9.68)	OR=1.07 p=0.716 (.74-1.56)	6%
Bullying								
All mediators	OR=9.92 p=0.001 (2.61-37.70)	OR=10.73 p=0.001 (2.48-46.33)	OR=.92 p=0.839 (.43-1.96)	0%	OR=10.40 p=0.001 (2.48-43.52)	OR=10.69 p=0.003 (2.21-51.73)	OR=.97 p=0.950 (.41-2.31)	0%
Negative beliefs - Others	OR=8.69 p=0.001 (2.50-30.19)	OR=9.40 p<0.001 (2.67-33.14)	OR=.92 p=0.754 (.57-1.51)	0%	OR=9.78 p=0.001 (2.53-37.88)	OR=12.35 p<0.001 (3.04-50.20)	OR=.79 p=0.392 (.46-1.35)	0%
Negative beliefs - Self	OR=6.61 p=0.001 (2.11-20.74)	OR=6.33 p=0.001 (2.03-19.72)	OR=1.04 p=0.803 (.74-1.47)	2%	OR=6.96 p=0.002 (2.08-23.35)	OR=6.20 p=0.003 (1.87-20.52)	OR=1.12 p=0.546 (.77-1.63)	6%
Depression score	OR=5.41 p=0.002 (1.86-15.75)	OR=4.02 p=0.010 (1.39-11.59)	OR=1.35 p=0.089 (.95-1.90)	18%	OR=5.71 p=0.003 (1.83-17.85)	OR=4.04 p=0.018 (1.27-12.85)	OR=1.41 p=0.084 (.95-2.09)	20%
Cannabis use	OR=4.96 p=0.002 (1.80-13.69)	OR=4.62 p=0.006 (1.54-13.80)	OR=1.07 p=0.476 (.96-1.66)	4%	OR=5.42 p=0.003 (1.78-16.52)	OR=5.25 p=0.006 (1.62-16.94)	OR=1.03 p=0.867 (.69-1.54)	2%
Life events	OR=5.04 p=0.002 (1.82-13.93)	OR=5.37 p=0.002 (1.89-15.22)	OR=.94 p=0.570 (.76-1.16)	0%	OR=5.54 p=0.003 (1.82-16.88)	OR=5.22 p=0.004 (1.69-16.14)	OR=1.06 p=0.657 (.82-1.37)	3%

*Adj. for age, ethnicity (White, Non-White), genetic risk for psychosis. OR, Odds Ratio. CI, Confidence Interval