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Depressed older patients with the atypical features of interpersonal rejection sensitivity and reversed-vegetative symptoms are similar to younger atypical patients

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Abstract

Objectives—The atypical depression (AD) subtype has rarely been examined in older patients. However, younger AD patients have been characterized as having more severe and chronic symptoms of depression compared with non-AD patients.

Design—Secondary data analysis using ANOVAs and Growth Curve Modeling.

Setting—Clinical Research Center for the Study of Depression in Later Life.

Participants—Depressed older patients (N=248) followed over 2 years.

Method—In a longitudinal study, we examined depression severity and chronicity in patients with major depression with some features of atypical depression, specifically rejection sensitivity and reversed-vegetative symptoms (e.g., hyperphagia, hypersomnia), or leaden paralysis, and compared them to non-AD patients. The Diagnostic Interview Schedule (DIS) was used to assess depressive symptoms and history. Depression severity and chronicity were assessed every three months using the Montgomery Asberg Depression Rating Scale.

Results—The AD symptom group reported more DIS depressive symptoms, more thoughts about wanting to die, earlier age of onset, poorer social support and double the number of lifetime episodes than non-AD patients. Growth curve analyses revealed that, compared with non-AD patients, the AD symptom group had more residual symptoms of depression during the first year of follow-up, but not during the second year.

Conclusion—Characteristics of older patients with features of AD are similar to younger patients. Assessment of atypical symptoms, in particular rejection sensitivity and reversed-vegetative symptoms is essential, and should be considered in treatment plans.

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Keywords

atypical depression; rejection sensitivity; reversed-vegetative symptoms; elderly

Depression is the most common form of emotional suffering in older adults (1) yet atypical depression (AD), a depression subtype that occurs in approximately 25% to 40% of depressed younger patients (2-8) has rarely been examined in older depressed adults. Subtypes of depression among older adults need to be identified so that efficacious treatments for subtypes can be examined and then utilized in the treatment of patients (9). There is a literature demonstrating differential patterns of treatment response among depressed people with vs. without an atypical presentation. Atypicality in general, and, in future work, any associations of atypicality with treatment response in particular, deserves empirical attention in older adults (10).

Converging lines of inquiry support the entity of a subgroup of atypical depressed patients (10) — identifiable in terms of 1) clinical features (e.g., symptom constellation, severe and chronic symptoms, earlier age of onset, and poorer social functioning), 2) pharmacological response (atypically depressed have greater response to monoamine oxidase inhibitors (MAOI)) (10), and 3) neurobiology. For instance, in terms of neurobiological indicators, atypical depression appears to have seemingly opposite effects compared to other presentations of depression. Evidence reviewed by Gold and Chrousos (11) suggests that symptoms of melancholic depression (e.g., insomnia, loss of appetite, lack of responsiveness to the environment) are associated with hypothalamic-pituitary-adrenal (HPA) *hyperactivity*, but symptoms of atypical depression (e.g., lethargy, fatigue, hypersomnia, and hyperphagia) are associated with HPA *hypoactivity* (12). Similarly, Geracioti and colleagues (13) found that among a small sample of depressed patients, the majority of whom had at least one symptom of atypical depression, cerebrospinal fluid corticoctropin-releasing hormone concentrations tended to be lower than among non-depressed volunteers.

Despite considerable research, the characteristics distinguishing AD from other depressive subtypes remain uncertain. Full DSM-IV-TR diagnostic criteria for the atypical specifier for Major Depressive Disorder are characterized by mood reactivity (i.e., mood brightens in response to actual or potential positive events), reversed-vegetative symptoms (e.g., hypersomnia, hyperphagia/weight gain), leaden paralysis (heavy arms or legs) and a distinct, enduring pattern of interpersonal rejection sensitivity (14).

Different portrayals of AD have emerged, and Davidson and Thase (15) assert that no single type of depression can be considered to be ‘atypical.’ Indeed, there has been some controversy regarding the specific symptoms that comprise the diagnosis (see (10, 15-19). Although in the current study we did not have available the full DSM-IV criteria for the atypical depression subtype to consider, we were nonetheless interested in examining depressed patients with features consistent with atypical depression. Specifically, these features included rejection sensitivity (as a core symptom), and in addition reversed vegetative symptoms, and leaden paralysis. In the current study, our AD feature group is consistent with the recent research, described below, that identifies ‘rejection sensitivity’ as the cardinal feature of AD. Importantly, this alternative definition is a ‘possibly constructive hypothesis’ remaining to be tested. In the current study, only one symptom was excluded from the DSM-IV criteria, specifically mood reactivity.

Indeed, there is a body of research arguing against mood reactivity as a criterion for AD (6, 7, 17, 20, 21). Specifically, researchers have argued that mood reactivity does not correlate with the other symptoms of AD, is indicative of lower levels of depression, and is a poor

indicator of AD (6, 20, 21). Thase takes issue with the DSM-IV criteria for AD. The researcher concluded that mood reactivity, the obligatory criterion in the DSM-IV, is neither significantly associated with the other symptomatic criteria nor useful to diagnose AD, and thus should be eliminated (17). However there are researchers who continue to support the use of mood reactivity as a cardinal feature (16).

Parker and Thase (20) put forth a psychological theory of AD. They propose a model differing from the DSM-IV-TR definition of AD. They redefined the disorder as a dimensional nonmelancholic syndrome in which individuals with a personality subtype of "interpersonal rejection sensitivity" are at a greater risk for depression. They also comment that the patients exhibit a variety of dysregulated emotional and self-consolatory responses including eating and sleeping too much (e.g., hyperphagia and hypersomnia). They argue that this reformulated definition of AD may lead to a better understanding and recognition of the disorder within the scope of major depression.

Consistent with Parker and colleagues, several researchers have concluded that interpersonal rejection sensitivity should be considered the core feature of AD (see (6, 17, 22, 23). Frank and Thase (24) describe *rejection sensitivity* as "a crashing drop in subjective well-being following a rebuff or some other setback." It is the most prevalent of the AD symptoms, occurring in 75% to 80% of AD patients (8, 23), and it is the most stable of the DSM-IV diagnostic criteria (8). In support of the criteria for rejection sensitivity, Davidson and colleagues (25) conducted a Post Hoc test of depressed outpatients receiving MAO inhibitors compared to placebo; they found that rejection sensitivity was a significant indicator of treatment response. However, it should be noted that the Davidson study (25) used six self-rated items from the Interpersonal Sensitivity Scale of the SCL-90 (26) to assess rejection sensitivity: (for example, 'your feelings being easily hurt', 'feeling others do not understand you or are unsympathetic', and 'feeling others are unfriendly'). The extent to which these items correspond to the DSM-IV definition of rejection sensitivity (a long-standing pattern of interpersonal rejection not limited to mood disturbances, resulting in significant social or occupational impairment) is not altogether clear.

Another view of atypical symptoms has been put forth by Pollitt (27). AD is also defined, in part, by the presence of "reversed" vegetative symptoms (e.g., hypersomnia, hyperphagia) (18, 24) as well as leaden paralysis (e.g. heavy arms or legs). Studies have found support for a definition of AD based solely on these symptoms (28). Interestingly, one study (29) showed that among AD patients who relapsed, 90% continued to have the reversed vegetative symptoms upon relapse. Another study compared patients diagnosed with the full DSM-IV atypical criteria to those diagnosed with simply the symptoms of hyperphagia and hypersomnia; the characteristics of the patients identified by the two approaches yielded were very similar results (28, 30). However, it should be noted that this was not a stringent test of the differences between the DSM-IV diagnoses of AD to such other definitions of AD —including the definition used in the current study.

The distinctiveness of the DSM-defined AD subtype is supported by several studies that demonstrate biological differences between AD and non-AD patients (2). Gold and Chrousos (11) suggested that symptoms of hyperarousal in depressed patients with vegetative symptoms are associated with hypothalamic-pituitary-adrenal (HPA) activation, whereas others have found that the lethargy, fatigue, hypersomnia, and hyperphagia of individuals with some features of AD produce seemingly opposite effects, namely hypoactivity of the HPA axis (12). In patients with DSM defined AD, hypercortisolism, a physiological marker of typical depression, was found to be absent (13). The differential response to pharmacological interventions lends further support for the distinctiveness of the AD subtype (31). Specifically, studies have found that DSM-IV defined AD patients and

depressed patients with some AD features (31-33) are relatively unresponsive to tricyclic antidepressants but are responsive to monoamine oxidase inhibitors (MAOI) (34, 35). This was particularly the case for depressed patients who scored high on a self-report measure of sensitivity to rejection (25). Third, most, but not all studies of younger DSM defined AD patients have found them to have more severe and chronic symptoms of depression than non AD-patients. AD patients have been characterized as having a preponderance of women, a younger age at onset, greater symptom severity, higher frequency of depressive symptoms, more suicidal thoughts, a longer duration of illness, and poorer social functioning compared to non-AD patients (3, 6, 36, 37).

In a study comparing differences between younger and older adult outpatients with a major depressive episode and bipolar II disorder (38), DSM-IV AD was present in 55.0% of patients under age 60 and in 28.1% age 60 and over. The author pointed out that the high rate of bipolar II patients in the younger group may account for the differences in rates of AD. Indeed, the frequency of DSM-IV AD was found to be more common in bipolar II versus major depression (39). In a community-based survey (40) of older adults (60+) the AD subtype (assessed by the DSM-III) was related to older age, being widowed or divorced, living alone, being frequently alone, having a low number of hobbies and having many long-standing or current social stress factors. In addition, the atypical depressive persons felt that elderly people were not appreciated. These AD features are, for the most part, interpersonal in nature and may be associated with the AD symptom of rejection sensitivity.

Consistent with the literature reviewed above, in the current longitudinal study, we examined older patients with major depression who endorsed the atypical features of rejection sensitivity, and reported at least one symptom of reversed-vegetative symptoms *or* leaden paralysis. However, it is important to note that most of the research on the validity of the AD subtype has been based on the DSM-IV definition of AD and not the definition used in the current study. The fact that the AD features group is based on a less validated definition of the AD subtype should be considered when evaluating the results of the study. Although this revised definition of AD has not been as well-examined as that of the DSM definition, we were nonetheless interested in comparing depressed older adult patients with some AD features to depressed older adult patients with non-AD features.

Thus, we compared the depressed patients with some features of AD to non-AD patients on indices of depression severity and chronicity. The current study makes use of longitudinal data from the Neurocognitive Outcomes of Depression in the Elderly (NCODE) study (41). Consistent with the findings from younger samples, we hypothesized that depressed patients with these AD features would demonstrate more severe indices of depression in comparison with non-AD patients. Consistent with studies of younger sample, we also expected AD patients to have more chronic symptoms of depression, as reflected in the residual symptoms of depression over the two year follow-up period after the index episode. We expect that over time, patients' levels of depressive symptoms would decrease (42) but we hypothesized it will take longer for the AD patients than the typically depressed.

Methods

Participants

This study used previously collected data from the Neurocognitive Outcomes of Depression in the Elderly (NCODE) study (41), an NIMH-supported study at Duke University Medical Center (DUMC) in Durham, North Carolina. Participant recruitment began in 1994 and continues to the present. The NCODE study is approved yearly by the DUMC Institutional Review Board.

Depressed participants (Major depressive disorder)—Non-demented adults over age 60 who presented at the Psychiatry Services unit at Duke University Medical Center or at the Duke General Internal Medicine Clinic and met criteria for a current episode of major depression were recruited into the study. Participants were excluded if they met criteria for another major psychiatric illness (schizophrenia, schizoaffective disorder, bipolar disorder, lifetime alcohol or substance dependence, and dementia). Likewise, participants were excluded if they had neurological illnesses. Patients were not excluded if they also met the criteria for dysthymia as well.

There were 248 inpatients and outpatients who met the criteria for current major depression, and for whom data were obtained at baseline. The patients underwent naturalistic treatment (43). Treatment is based on history of antidepressant use and severity of depression. Never-treated patients were initially prescribed selective serotonin reuptake inhibitors (SSRIs), with augmentation or change if response was not sufficient (43). Patients were not routinely referred for psychotherapy.

Measures

Baseline demographic and depression assessment—Trained interviewers administered the Duke Depression Evaluation Schedule (DDES) a structured interview which is comprised of several validated instruments. The DDES included sections that obtained information from the participant on demographics and social support. The DDES also included the Diagnostic Interview Survey (DIS) (44) which allows for an assessment of DSM–IV current and lifetime Major Depression and history of depression. Items on the DIS paralleled symptom criteria for DSM-IV diagnosis of depression. It fully specified all questions and probes to be used, and it was accompanied by a set of computer programs that made diagnoses on the basis of analysis of symptom scores. The DIS has been used in a set of epidemiological studies sponsored by the National Institute of Mental Health Center for Epidemiological Studies. Its accuracy has been evaluated in a test-retest design (45). It has been found to have good validity and reliability for participants of all ages (46). It is widely used in research in aging populations (see, for example (47-50))

Atypical symptom group—The DIS included items relevant to the assessment of the AD subtype. It is important to note that one study based on the DIS (51) provides evidence for the validity of major depression with atypical features (defined as overeating and oversleeping) as a distinct subtype based on cross-sectional and 1-year prospective data from the Epidemiologic Catchment Area study.

The atypical symptom group in the current study was defined by patients who first met the criteria for current major depression and then had the AD features of rejection sensitivity, and in addition had one or more reversed-vegetative symptoms (i.e., hyperphagia/weight gain, hypersomnia) or leaden paralysis (i.e., heavy arms and legs). The specific questions in the DIS assessing these AD symptoms are described below. Moreover responses were followed up by probes such as asking the respondent to give recent examples, identifying whether or not there was another explanation for the symptom (such as a medical problem, drug or alcohol use, pregnancy, dieting, etc.). If there was another explanation for the symptom the participant was not coded as having met the criteria. Questions were also included that addressed the recency and duration of each symptom.

To meet the criteria for each AD feature described below, the symptom must have lasted two weeks or longer and had occurred in the last month. 1). Hyperpahgia: Have you ever had a period in your life when your eating increased so much you gained as much as 2 pounds a week for several weeks, or gained as much as ten pounds? Have you ever had a period in your life lasting two weeks or longer when your appetite increased so much (when

you weren't dieting) that you felt like you were constantly hungry? 2). Hypersomnia: Have you ever had a period in your life lasting two weeks or longer when you were sleeping too much?; 3). Leaden paralysis: Has there ever been a period of 2 weeks or longer when your arms and legs have felt heavy or leaden? 4). Finally, the interviewer assessed rejection sensitivity. The clinical interviewer was trained to obtain information as to whether the patient had a long-standing pattern of interpersonal rejection (not limited to the current mood disturbance or past episodes) that resulted in significant social or occupational impairment. The initial stem question was as follows: 'Are you unusually sensitive to criticism or rejection?' If the patient responded 'Yes' to the symptom, the interviewer was trained to determine if the symptom met criteria using several probes. For example, in addition to the probes described above, participants were asked: 'give examples of perceived rejection at a time when not depressed', 'describe how the rejection affected you' and 'have you always mostly been this way'. The interviewer also asked questions to determine whether or not there was another explanation for the symptom (such as a medical problem, drug or alcohol use, or limited to only depressive episodes). For most of the depressive symptoms throughout the survey the interviewer rated the patient's response using the following coding scheme: 1. No, 2. Below criteria, 3. Drugs/Alcohol related, 4. Medical explanation, 5. Meets criteria, 7. Refused, 8. Don't know, 9. Skipped.

Baseline assessment of severity of depression—A geriatric psychiatrist completed a Montgomery Asberg Depression Rating Scale (MADRS) (52) to determine severity of depression. The MADRS has been used to assess depression in geriatric populations (53, 54). The MADRS assesses symptoms of depression such as apparent sadness, reported sadness, inner tension, reduced sleep, reduced appetite, concentration difficulties, difficulty in getting started or slowness in initiating and performing everyday activities, inability to feel, pessimistic thoughts, and suicide. Each item had a 6 point response scale. For example, "Apparent sadness" was rated using a six point scale ranging from "No sadness" to "Looks miserable all the time and extremely despondent." All ten items had good toexcellent interrater reliability (55). The intraclass correlation for raters was found to be r=.93, df=162, p<.001.

The MADRS assessment was conducted at baseline and every three months over a two year period. It is important to note that the symptoms comprising the MADRS did not include any atypical symptoms (e.g., hyperphagia, hypersomnia, rejection sensitivity) but did assess vegetative symptoms (e.g., reduced sleep, reduced appetite). The vegetative symptoms are more likely to characterize some of the patients in the non-AD group. Thus the MADRS may under-estimate symptom severity in the AD group. This limitation should be kept in mind.

Clinical assessment of depression—At baseline, a geriatric psychiatrist interviewed each potential participant and completed standardized clinical assessments for major depression, including the MADRS and the Clinical Global Impression scale. Based on the patient's reported symptoms, the results of the DIS, and the clinical interview, the presence or absence of major depression was determined.

Perceived Social Support—At baseline, Perceived Social Support was measured as a continuous variable, with higher levels indicating greater Perceived Social Support. The scale has been well validated and used extensively in epidemiological studies of older adults (56)(57). The measure of perceived support was created from the sum of ten items, for example, items included "you feel that your family and friends listen to you" and "you could count on family or friends in times of trouble." Responses on each item ranged from 1 'Hardly ever' to 3 'Most of the time.' (Cronbach's α = .83).

Perceived Instrumental Support—The ten item Perceived Instrumental Support scale has been well validated and used extensively in epidemiological studies of older adults (56) (57). Examples of items include: "Family helps when you are sick" and "Family shops, runs errands." Responses on each item ranged from 1 'Hardly ever' to 3 'Most of the time.' Higher scores indicate more support. (Cronbach's $\alpha = .74$).

Data analytical plan

First, we describe the patients' demographics and the means and standard deviations of the relevant variables. A series of ANOVAs were performed to examine differences on demographic variables between the AD symptom group and non-AD group. We then compared the AD symptom group and non-AD group on indices of depression severity and chronicity including age of onset, suicidal ideation, lifetime number of episodes, and number of self-reported current depressive symptoms on the DIS. To account for the number of comparisons conducted, we used Bonferroni corrections.

Latent Growth Curve Modeling—Using MADRS scores we constructed a latent growth curve model which included the baseline and eight assessments completed over two years. We expected AD and non-AD patients alike to show a decrease in depression severity over time after the index episode; however, as in younger adults, we expected the decrease in symptoms to be slower for those with AD.

All latent growth curve analyses were conducted using MPlus version 5.2 (58, 59). Standard model fit criteria were used to evaluate the model, with good fit being identified by a non-significant model χ^2, CFI value over .95, and a RMSEA value under .08 (60). AIC values were used to compare different models, with lower AIC scores indicating better fit (61). Missing data were estimated using full–information maximum likelihood (62), which is the standard approach to handling missing data in MPlus. We also estimated these models controlling for variables which may be associated with AD status and depression severity, (e.g., age, gender and education level). In a latent growth curve analysis the changes in a measure over time are used to create latent variables which represent the initial starting value of all participants (the latent intercept), and a latent change variable across participants (the latent slope). The latent slope and intercept are also allowed to correlate. Non-linear or quadratic growth can also be included as a latent variable in this kind of model. Predictors are then regressed onto the latent intercept and slope, allowing for time-invariant predictors to provide more information about where participants started and how they changed over time.

Results

Demographics and descriptive statistics

Demographics are described by AD group status in Table 1. At baseline, there were 248 currently depressed patients, of whom 34.7% were identified as being in the AD-symptom group. That is, among the depressed patients 34.7% had current rejection sensitivity and had one or more reversed-vegetative symptom (e.g., hyperphagia/weight gain, hypersomnia) or leaden paralysis (e.g., heavy arms and legs).

In the sample of the depressed patients there were no gender differences in the percentage of females in the AD (69.8%) and non-AD groups (66.0%). Participants in the AD symptom group were younger than the non-AD patients (67.6(7.1) vs. 70.6(7.4)). There were no differences in race or ethnicity. The sample included Caucasian 86.7%, African-American 8.9%, and other race 4.4%.

Indicators of depression severity at baseline

Indices of depression severity are also summarized in Table 1. Consistent with predictions, the average age of onset of depression was considerably younger for the atypical symptom group (37.7(20.9) years vs. 49.1(19.5) years). Further analyses showed that 29% of the atypical group had their first episode of depression in adolescence, whereas only 8.6% of the non-AD group had their onset in adolescence, ($\chi^2 = 17.7$, $df=1$, $p < .001$).

Symptoms of depression, as measured by number of items endorsed on the DIS (out of 26 items) were calculated. It should be noted that there were multiple probe items for the same symptom. For example, to assess the symptom of sleep disturbance, individuals were asked if he or she 1) had trouble staying asleep, 2) trouble falling asleep, 3) trouble waking up too early, etc. The AD symptom group reported more DIS symptoms of depression, as assessed by the DIS, than the non-AD symptom group (13(2.4) vs. 10.1(2.4)). The AD symptom group reported having thoughts of wanting to die more often than the non-AD symptom group (43.0% vs. 18.5%). (Using Bonferroni corrections all significant p-values were set at $p < .004$).

The number of lifetime depressive episodes among the AD symptom group was double that of the non-AD symptom group (10.3(20.2) vs. 4.5(8.1)). This was, in part, due to the earlier age of onset among the AD patients. Notably, there were no significant differences on severity of baseline depressive symptoms as assessed by the geriatric psychiatrist using the MADRS.

Social support

The AD symptom group reported having less Perceived Social Support than the non-AD group (21.8(4.4) vs. 23.5(3.8)). Also, the AD symptom group experienced their social network as providing less help (e.g., Perceived Instrumental support) than the non-AD group (8.6(2.4) vs. 9.4(2.0)).

Two-Year Trajectory of Depressive Symptoms

Latent growth curve analyses were used to examine differences in the trajectory of symptom severity over two years. Because changes in symptoms do not always follow a linear pattern, we anticipated possible nonlinear effects such that a decrease in symptoms would initially be rapid but would eventually slow down as time increased from the initial index episode. Specifically, there was a high level of depressive symptoms needed to meet diagnostic criteria at baseline and we expected that there might be an initial response to treatment. This response may be reflected in a more rapid initial decay in symptoms, but then residual symptoms would remit more slowly.

In these nonlinear models we explore two possible patterns: 1) piecewise changes (e.g., separate slopes for year one and year two), and 2) curvilinear changes. To estimate the curvilinear effects of change in depressive symptoms, quadratic effects were estimated for each slope examined in the baseline and piecewise models.

In total we examined four latent growth models: 1) a baseline linear model, 2) a baseline model with quadratic effects, 3) a piecewise model with no curvilinear effects, and 4) a piecewise model with curvilinear effects. In each of these models we included AD status as a predictor of slope and intercept, as well as age, sex, and education as covariates for slope and intercept. The relationships between each of these predictors and the intercept and slope are interpreted as a beta weights, as in linear regression analyses. Figure 1 displays the average MADRS score at each assessment broken down by ADversus non-AD group. Table 3 summarizes the goodness-of-fit variables for each model examined.

The final model examined a piecewise model with both linear and quadratic effects for each estimation of growth and provided the best fit to the data of all the models examined (χ^2 = 82.88, *df*=41, CFI=.952, RMSEA=.06, AIC=17845.25). This model indicated that changes in depressive symptoms followed two patterns of change over the two years, with the initial section taking place from assessments 0 through 4 (baseline through year 1), and a second taking place from 5 through 8 (year 2). There also appear to have been quadratic effects for each section of the piecewise model.

In this model education was the only significant predictor of intercept (β= −.32, *df*=41, *p*<.001), with lower levels of education indicating higher baseline levels of depression. In this model the intercept was significantly correlated with the initial linear slope (*r*=−.44, *df*=41, *p*=.01) and the initial quadratic effect (*r*=.36, *df*=41, *p*<.05), indicating that those who started with higher baseline depression scores experienced the fastest decrease of symptoms for the first 5 assessments, and these individuals were also more likely to experience a slight rebound of depressive symptoms between 9 and 12 months.

Although AD status did not significantly predict the model intercept (baseline levels of depression), indicating similar initial levels of depression, AD status did predict the linear slope for the first section of the model (β=.23, *df*=41, *p*=.018), such that those with non-AD improved faster than those with AD features. AD status was also a significant predictor of the quadratic effect for the first portion of the piecewise model (β= −.248, *df*=41, *p*=.019). This indicates that although the non-AD group experienced a faster decrease of depressive symptoms for the first year, this group also experienced a slight rebound of symptoms toward the end of the first year. No variables, including AD status, significantly predicted either the linear slope or quadratic effects of the second portion of the piecewise model, indicating that although the non-AD group experienced a faster decrease of symptoms through the first year, by the final assessment at the end of year 2, there were no significant AD status differences in depressive symptoms. Indeed, as illustrated in Figure 1, both groups had relatively low levels of depressive symptoms during year two.

Discussion

While atypical depression (AD), a depression sub-type (2), comprises approximately 25% to 40% of depressed younger patient samples (2-8), AD has rarely been examined in older adults. It is thus unclear whether AD manifests itself similarly in older and younger depressed patients. Therefore, the goal of the present study was to compare depressed older patients (60+) with some features of AD (e.g., rejection sensitivity, and reversed-vegetative symptoms or leaden paralysis) with depressed older patients without AD features.

Our results were remarkably similar to studies of AD in younger adults in a number of ways. First, our results provide evidence that the prevalence rate of AD is similar in older and younger adults, as 34.7% of the older depressed patients in our sample reported some features of AD, rates similar to those found in younger adults (e.g., 25% to 40% (2-8). The fact that AD remains a prevalent subtype of depression in older adults may be a reflection of the greater symptom severity, greater chronicity and longer duration of illness that have been found in younger AD patients (3, 6, 36, 37). Additionally, as there has been a dearth of research on treatments targeting the AD features of depression, it is possible that younger AD patients are still meeting criteria for AD as they age because treatment has not adequately addressed their symptoms.

Second, similar to studies assessing AD in younger adults, our results provide evidence that the AD symptom group reported a greater number of indices of depression severity and chronicity than the non-AD group. For instance, the average age of onset of depression was

considerably younger for the AD symptom group (37.7(SD=20.0) vs. 49.1(SD=19.5) years). Furthermore, results indicated that 29.1% of the AD symptom group had their depression onset during adolescence compared with only 8.6% in the non-AD group. Depressive episodes in childhood and adolescence, rather than those at older ages, predicted more episodes and longer duration of depression in adult life (63-65) and thus a more chronic course of illness.

With regard to the chronicity of depressive illness, it is noteworthy that the number of lifetime depressive episodes among the older AD group was more than double that of the older non-AD group (10.3(20.2) vs. 4.5(8.1)). In fact, the AD symptom group was found to have many of the characteristics associated with chronicity of depression in elderly depressed patients. This includes more severe initial illness, more previous episodes, and poor social support (66, 67). Thus it is important that practitioners and medical professionals assess for AD features in their depressed patients and consider prescribing efficacious treatments. Additionally, it is important for future research to test whether older adult patients with AD features demonstrate a similar pattern of responsiveness to monoamine oxidase inhibitors compared to younger adult patients with AD (32-35). It is important to note that research supporting the efficacy of MAOIs was conducted on younger depressed patients with either DSM-IV defined AD or on depressed patients with some features of AD; however, studies have not been conducted on the revised definition (featuring rejection sensitivity) examined in the current paper.

Unlike research on younger patients, we found no gender differences. This may reflect age-related changes in the AD prevalence by gender, or it may reflect utilization patterns of older depressed men. Or it may also be a product of the fact that women are more likely to seek treatment for mental health problems (68), so as they age, more women have recovered from their depressive episodes. It would be of interest for future research to investigate factors that might contribute to this lack of gender difference in AD in older adults.

Growth curve analyses demonstrated that the AD group showed a slower course of recovery over the first year than the non-AD group. At the end of the one year period, however, there were no significant differences between the two groups. This established that recovery is at a much more rapid pace for the non-AD group, at least for the first year after the index episode. However, one heartening finding is that both groups appeared to have relatively low rates of depressive symptoms during year 2.

Poor social support is strongly associated with depression in the elderly (69, 70). Downey and colleagues (71) found that individuals with rejection sensitivity misinterpret cues that lead them to perceive rejection and then react in ways that undermine their relationships (72). Moreover, this “rejection sensitivity” has been found to lead to depression after an interpersonal dispute (73). Indeed, we found the AD symptom group had less Perceived Social Support and less Perceived Instrumental Support than non-AD persons. Interpersonal rejection sensitivity may be more than a “symptom” of the AD episode, but may be a trait-like feature (17, 74) that serves as a risk factor for depressive episodes and may account, in part, for the more severe and chronic course of the depressive disorder among those with features of AD; this warrants further investigation. It would be of interest for future research to conduct prospective longitudinal studies focusing on individuals who endorse interpersonal rejection sensitivity but who have not yet experienced a depressive episode with AD features. Such studies would help clarify whether interpersonal rejection sensitivity serves as a risk factor for AD.

Depression is the most frequent cause of emotional suffering in later life and is associated with significant losses in quality of life. Older individuals rarely seek treatment for their

mental health problems (75) and thus the sample examined in the current study is unlikely representative of an older depressed general population. Therefore inferences from the results of the study are limited to the treatment-seeking subset. Moreover, depression among older adults often goes undetected and untreated (76). However, there are efficacious treatments for depression tailored for the needs of elders. While we could find no research specifically focusing on psychotherapy for AD in older adults, in one small sample of geriatric patients with AD, venlafaxine treatment (in an open label trial) was reasonably effective and well tolerated (5).

Because maladaptive cognitions related to interpersonal rejection sensitivity may contribute to the more severe course in AD (77), cognitive behavioral therapy that focuses on maladaptive thoughts may be a useful treatment modality (78). Secondly, because of the centrality of interpersonal difficulties in rejection sensitive individuals, interpersonal therapy (IPT) (79, 80) may be efficacious in the treatment of atypical depressed patients who identify interpersonal disputes as a trigger for depressive episodes. Miller and colleagues (81) found that IPT was an effective treatment with elderly depressed patients.

As in all studies, there are limitations to consider when interpreting the results. Whereas we found the AD symptom group to have more severe and chronic symptoms of depression, the lack of variability in patient demographics (e.g., the sample was primarily Caucasian) may limit the generalizability of the results. Indeed, in an epidemiological population sample (82), those who were defined as being atypically depressed were similar to the non-atypically depressed in severity of depression. This may imply that there may not be any true difference between those with the AD subtype compared to the non-AD subtype in the population in general. Rather, the difference is only observed in those who seek treatment.

An additional limitation is that it is not entirely clear whether AD status is merely a surrogate for depression chronicity and severity. If this were the case one would surely assess for chronicity and severity rather than AD status, and treatment strategies would focus on those who have had a more chronic or severe depression.

This study assumes that the designation of AD is a type of depression that is persistent, yet there is evidence that a significant number of depressed patients with AD are subsequently assessed as having melancholic depression (83). The lack of stability of the diagnosis calls into question conclusions made about the differences of depressed participants with AD features compared to non-AD groups. This is clearly an important area for future investigation.

In the current study, our definition of the AD symptom group was consistent with research that suggested mood reactivity was a poor indicator of AD (6, 7, 17, 20, 21);reversed-vegetative symptoms (and leaden paralysis) could validly be used to identify the AD subtype (28, 30), and a body of research indicating rejection sensitivity should be considered the core feature of AD (6, 17, 22, 23). However, it is quite important to note that the results of the current study do not reflect the findings of depressed individuals who meet the DSM-IV criteria for AD. Rather it is the case that these results are based on a ‘proposed hypothesis for a definition of AD,’ and the results only apply to depressed individuals seeking treatment with the AD features of rejection sensitivity, and reversed-vegetative symptoms or leaden paralysis. Thus our findings can not be said to represent DSM-IV atypical depression, but rather, our findings provide clinicians with a greater understanding of the course of the depressive illness and treatment considerations among elderly adults with the AD symptoms of rejection sensitivity and reversed-vegetative symptoms. Finally, while the validity and reliability of the DIS in the assessment of depression has been shown to be good, there is limited data on the validity and reliability of the AD subtype with rejection sensitivity as a

core characteristic. Moreover, as most of the symptom assessments in the DIS, the assessment of rejection sensitivity relied on just one initial item.

In sum, there are a substantial number of depressed older patients who have the AD features of the rejection sensitivity and reversed-vegetative symptoms. Importantly, older depressed patients with these features are quite similar to AD younger adults, lending further support for the validity of the AD subtype. This older AD symptom group compared to the non-AD group demonstrated more severe and chronic indices of depressive illness and poorer social support. Moreover, reflecting the chronicity of AD, over a one year period we established that the AD group had a slower rate of recovery. Assessment of atypical symptoms, in particular rejection sensitivity and reversed-vegetative symptoms, in depressed older adults is essential, and the presence of these AD features should be considered in developing treatment plans.

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References

1. Blazer, D. Depression in Late Life. CV. Mosby and Company; St. Louis, MO: 1994.
2. Asnis GM, McGinn LK, Sanderson WC. Atypical depression: clinical aspects and noradrenergic function. Am J Psychiatry. 1995; 152(1):31–36. [PubMed: 7802117]
3. Matza LS, Revicki DA, Davidson JR, Stewart JW. Depression With Atypical Features in the National Comorbidity Survey: Classification, Description, and Consequences. Arch Gen Psychiatry. 2003; 60(8):817–826. [PubMed: 12912765]
4. Benazzi F. Prevalence and clinical features of atypical depression in depressed outpatients: a 467-case study. Psychiatry Research. 1999; 86(3):259. [PubMed: 10482345]
5. Steven PR, Marissa M, Dev D, Stuart S, Linda F, Nancy T, Harold S. An open trial of venlafaxine for the treatment of late-life atypical depression. International Journal of Geriatric Psychiatry. 2004; 19(10):989–994. [PubMed: 15449363]
6. Posternak MA, Zimmerman M. Partial Validation of the Atypical Features Subtype of Major Depressive Disorder. Arch Gen Psychiatry. 2002; 59(1):70–76. [PubMed: 11779285]
7. Perugi G, Akiskal HS, Lattanzi L, Cecconi D, Mastrocinque C, Patronelli A, Vignoli S, Bemi E. The high prevalence of “Soft” bipolar (II) features in atypical depression. Comprehensive Psychiatry. 1998; 39(2):63. [PubMed: 9515190]
8. Angst J, Gamma A, Sellaro R, Zhang H, Merikangas K. Toward validation of atypical depression in the community: results of the Zurich cohort study. Journal of Affective Disorders. 2002; 72:125. [PubMed: 12200203]
9. Rapaport MH, Thase ME. Translating the evidence on atypical depression into clinical practice. J Clin Psychiatry. 2007; 68(4):11.
10. Pae CU, Tharwani H, Marks DM, Masand PS, Patkar AA. Atypical depression: a comprehensive review. CNS Drugs. 2009; 23(12):1023–37. [PubMed: 19958040]
11. Gold PW, Chrousos GP. Organization of the stress system and its dysregulation in melancholic and atypical depression: high vs low CRH/NE states. Mol Psychiatry. 2002; 7(3):254–75. [PubMed: 11920153]
12. Chrousos GP, Kino T. Glucocorticoid action networks and complex psychiatric and/or somatic disorders. Stress: The International Journal on the Biology of Stress. 2007; 10(2):213–219.
13. Geracioti TD, Loosen PT, Orth DN. Low cerebrospinal fluid corticotropin-releasing hormone concentrations in eucortisolemic depression. Biol Psychiatry. 1997; 42(3):165–74. [PubMed: 9232208]
14. American Psychiatric Association. DSM-IV: Diagnostic and Statistical Manual of Mental Disorders. 1994.

15. Davidson JR, Thase ME. A history of the concept of atypical depression. J Clin Psychiatry. 2007; 68(2):10–15. [PubMed: 17348762]

16. Quitkin FM, McGrath PJ, Stewart JW, Klein DF. A Reappraisal of Atypical Depression. Am J Psychiatry. 2003; 160(4):798-b–800. [PubMed: 12668381]

17. Thase ME. Atypical Depression: Useful Concept, but it's Time to Revise the DSM-IV Criteria. Neuropsychopharmacology. 2009; 34(13):2633. [PubMed: 19741592]

18. Ohmae S. [The modern concept of atypical depression: four definitions]. Seishin Shinkeigaku Zasshi. 2010; 112(1):3–22. [PubMed: 20184236]

19. Lam RW, Stewart JN. The validity of atypical depression in DSM-IV. Compr Psychiatry. 1996; 37(6):375–83. [PubMed: 8932961]

20. Parker GB, Thase ME. Atypical depression: a valid subtype? J Clin Psychiatry. 2007; 68(Supplement 3):18–22. [PubMed: 17348763]

21. Parker G, Parker K, Mitchell P, Wilhelm K. Atypical depression: Australian and US studies in accord. Curr Opin Psychiatry. 2005; 18(1):1–5. [PubMed: 16639176]

22. Parker G, Roy K, Mitchell P, Wilhelm K, Malhi G, Hadzi-Pavlovic D. Atypical Depression: A Reappraisal. Am J Psychiatry. 2002; 159(9):1470–1479. [PubMed: 12202264]

23. McGrath PJ, Stewart JW, Harrison WM, Ocepek-Welikson K, Rabkin JG, Nunes EN, et al. Predictive value of symptoms of atypical depression for differential drug treatment outcome. J Clin Psychopharmacol. 1992; 3:197–202. [PubMed: 1629387]

24. Frank E, Thase ME. Natural history and preventative treatment of recurrent mood disorders. Annu Rev Med. 1999; 50:453–68. [PubMed: 10073289]

25. Davidson J, Zisook S, Giller E, Helms M. Symptoms of interpersonal sensitivity in depression. Compr Psychiatry. 1989; 30(357-368)

26. Derogatis LR, Rickels K, Rock AF. The SCL-90 and the MMPI: a step in the validation of a new self-report scale. The British Journal of Psychiatry. 1976; 128(3):280–289. [PubMed: 1252693]

27. Politt J. The diagnosis and management of depression. 1965; 604:194–597.

28. Benazzi F. Can only reversed vegetative symptoms define atypical depression? Eur Arch Psychiatry Clin Neurosci. 2002; 252(6):288–93. [PubMed: 12563537]

29. Nierenberg AA, Pava JA, Clancy K, Rosenbaum JF, Fava M. Are neurovegetative symptoms stable in relapsing or recurrent atypical depressive episodes? Biological Psychiatry. 1996; 40(8): 691. [PubMed: 8894060]

30. Benazzi F. Testing atypical depression definitions. Int J Methods Psychiatr Res. 2005; 14(2):82–91. [PubMed: 16175877]

31. Henkel V, Mergl R, Allgaier A-K, Kohnen R, Möller H-J, Hegerl U. Treatment of depression with atypical features: A meta-analytic approach. Psychiatry Research. 2006; 141(1):89. [PubMed: 16321446]

32. Quitkin FM, McGrath PJ, Stewart JW, Harrison W, Tricamo E, Wager SG, Ocepek-Welikson K, Nunes E, Rabkin JG, Klein DF. Atypical Depression, Panic Attacks, and Response to Imipramine and Phenelzine: A Replication. Arch Gen Psychiatry. 1990; 47(10):935–941. [PubMed: 2222132]

33. Liebowitz MR, Quitkin FM, Stewart JW, McGrath PJ, Harrison WM, Markowitz JS, Rabkin JG, Tricamo E, Goetz DM, Klein DF. Antidepressant Specificity in Atypical Depression. Arch Gen Psychiatry. 1988; 45(2):129–137. [PubMed: 3276282]

34. Stewart JW, Tricamo E, McGrath PJ, Quitkin FM. Prophylactic efficacy of phenelzine and imipramine in chronic atypical depression: likelihood of recurrence on discontinuation after 6 months' remission. Am J Psychiatry. 1997; 154(1):31–36. [PubMed: 8988955]

35. Stewart JW, McGrath PJ, Quitkin FM, Klein DF. Atypical depression: current status and relevance to melancholia. Acta Psychiatr Scand Suppl. 2007; 115(supplement 433):58–71. [PubMed: 17280572]

36. Derecho CN, Wetzler S, McGinn LK, Sanderson WC, Asnis GM. Atypical depression among psychiatric inpatients: clinical features and personality traits. Journal of Affective Disorders. 1996; 39(1):55. [PubMed: 8835654]

37. Novick JS, Stewart JW, Wisniewski SR, Cook IA, Manev R, Nierenberg AA, Rosenbaum JF, Shores-Wilson K, Balasubramani GK, Biggs MM, Zisook S, Rush AJ. Clinical and demographic

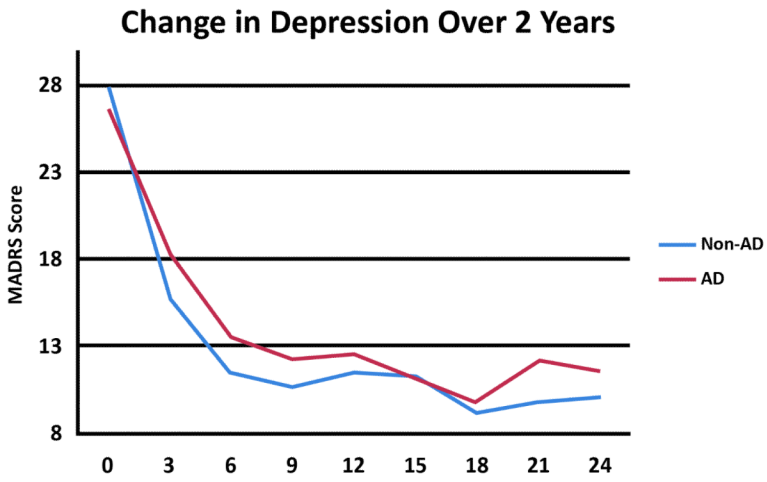
features of atypical depression in outpatients with major depressive disorder: preliminary findings from STAR*D. J Clin Psychiatry. 2005; 66(8):1002–11. [PubMed: 16086615]

38. Benazzi F. Late-Life Atypical Major Depressive Episode: A 358-Case Study In Outpatients. American Journal of Geriatric Psych. 2000; 8(2):117–122.
39. Benazzi F. Testing DSM-IV definition of atypical depression. Ann Clin Psychiatry. 2003; 15(1):9–16. [PubMed: 12839428]
40. Pahkala K, Kivela SL, Laippala P. Social and environmental factors and atypical depression in old age. Z Gerontol. 1990; 23(1):43–9. [PubMed: 2327146]
41. Steffens DC, McQuoid DR, Potter GG. Outcomes of Older Cognitively Impaired Individuals With Current and Past Depression in the NCODE Study. J Geriatr Psychiatry Neurol. 2009; 22(1):52–61. [PubMed: 19196631]
42. Hybels CF, Blazer DG, Steffens DC. Predictors of Partial Remission in Older Patients Treated for Major Depression: The Role of Comorbid Dysthymia. American Journal of Geriatric Psych. 2005; 13(8):713–721.
43. Steffens D, McQuoid DR, Krishnan KR. The Duke Somatic Treatment Algorithm for Geriatric Depression (STAGED) approach. Psychopharmacol Bull. 2002; 36(58-68)
44. Robins L, Helzer J, Croughan J. Diagnostic Interview Schedule: its history, characteristics and validity. Archives of General Psychiatry. 1981; 38:381–389. [PubMed: 6260053]
45. Robins, L.; Helzer, J.; Croughan, J.; Williams, J.; Spitzer, R. NIMH Diagnostic Interview Schedule: Version III. National Institute of Mental Health; Rockville: 1981.
46. Robins LN, Helzer JE, Croughan J, Ratcliff KS. National Institute of Mental Health Diagnostic Interview Schedule: Its History, Characteristics, and Validity. Arch Gen Psychiatry. 1981; 38(4): 381–389. [PubMed: 6260053]
47. Beekman ATF, de Beurs E, van Balkom AJLM, Deeg DJH, van Dyck R, van Tilburg W. Anxiety and Depression in Later Life: Co-Occurrence and Communality of Risk Factors. Am J Psychiatry. 2000; 157(1):89–95. [PubMed: 10618018]
48. Koenig H, Larson D, Hays J, McCullough M, George L, Branch P, Meador K, Kuchibhatla M. Religion and survival of 1010 male veterans hospitalized with medical illness. Journal of Religion and Health. 1998; 37:15–29.
49. Farrell KR, Ganzini L. Misdiagnosing Delirium as Depression in Medically Ill Elderly Patients. Archives of Internal Medicine. 1995; 155:2459–2464. [PubMed: 7503605]
50. Cole MG, McCusker J, Elie M, Dendukuri N, Latimer E, Bel E. Systematic detection and multidisciplinary care of depression in older medical inpatients: a randomized trial. Canadian Medical Association Journal. 2006; 174(1):38–44. [PubMed: 16330624]
51. Horwath E, Johnson J, Weissman MM, Hornig CD. The validity of major depression with atypical features based on a community study. Journal of Affective Disorders. 1992; 26(2):117. [PubMed: 1447429]
52. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. The British Journal of Psychiatry. 1979; 134(4):382–389. [PubMed: 444788]
53. Baudic S, Tzortzis C, Barba GD, Traykov L. Executive Deficits in Elderly Patients With Major Unipolar Depression. J Geriatr Psychiatry Neurol. 2004; 17(4):195–201. [PubMed: 15533990]
54. Parker RD, Elizabeth PF, Hayden BB, Carl FP, David CS. A three-factor analytic model of the MADRS in geriatric depression. International Journal of Geriatric Psychiatry. 2003; 18(1):73–77. [PubMed: 12497559]
55. Williams JBW, Kobak KA. Development and reliability of a structured interview guide for the Montgomery-Asberg Depression Rating Scale (SIGMA). The British Journal of Psychiatry. 2008; 192(1):52–58. [PubMed: 18174510]
56. Landerman R, George LK, Campbell RT, Blazer DG. Alternative models of the stress buffering hypothesis. Am J Community Psychol. 1989; 17:625–641. [PubMed: 2627025]
57. Blazer D, Hughes D. Subjective social support and depressive symptoms: separate phenomena or epiphenomenona. Journal of Psychiatric Research. 1991; 2:191–203. [PubMed: 1779416]
58. Duncan, TE.; Duncan, SC.; Strycker, LA. An Introduction to Latent Variable Growth Curve Modeling: Concepts, Issues, and Applications. Lawrence Erlbaum Associates; New Jersey: 2006.

59. Muthén, L.; Muthén, B. MPlus user's guide. Muthén, L. Muthén, B.; Los Angeles, CA: 2004.
60. Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Structural Equation Modeling. 6(1-55) 199.
61. Burnham KP, Anderson DR. Multimodel inference: Understanding AIC and BIC in model selection. Sociological Methods and Research. 2004; 33:261–304.
62. Anderson TW. Maximum likelihood estimates for a multivariate normal distribution when some observations are missing. Journal of American Statistical Association. 1957; 52:200–203.
63. Kendler KS, Neale MC, Maclean CJ, Health AC, Eaves LJ, Kessler RC. Smoking and major depression: a causal analysis. Arch Gen Psychiatry. 1993; 50:36–43. [PubMed: 8422220]
64. Parker G, Willhelm K, Asghari A. Early onset depression: The relevance of anxiety. Social Psychiatry and Psychiatric Epidemiology. 1997; 32:30–37. [PubMed: 9029985]
65. Piccinelli M, Wilkinson G. Gender differences in depression. British Journal of Psychiatry. 2000; 177:486–492. [PubMed: 11102321]
66. Alexopoulos G, Meyers B, Young R, Kakuma T, Feder M, Einhorn A, Rosendahl E. Recovery in geriatric depression. Archives of General Psychiatry. 1996; 53:305–312. [PubMed: 8634008]
67. Bosworth H, McQuoid D, George L, Steffens D. Time-to-remission from geriatric depression. American Journal of Geriatric Psychiatry. 2002; 10:551–559. [PubMed: 12213689]
68. Sachs-Ericsson N, Ciarlo J. Gender, Social Roles and Mental health: An epidemiological perspective. Sex Roles A Journal of Research. 2000; 43(9/10):339–362.
69. DuPertuis LL, Aldwin CM, Bosse R. Does the source of support matter for different health outcomes? Findings from the Normative Aging Study. Journal of Aging Health. 2001; 13:495–510.
70. Goldberg EL, Van Natta P, Comstock GW. Depressive symptoms, social networks and social support of elderly women. American Journal of Epidemiology. 1985; 121:448–456. [PubMed: 3874542]
71. Downey G, Mougios V, Ayduk O, London BE, Shoda Y. Rejection Sensitivity and the Defensive Motivational System: Insights From the Startle Response to Rejection Cues. Psychological Science. 2004; 15(10):668–673. [PubMed: 15447637]
72. Levy, SR.; Ayduk, O.; Downey, G. Rejection sensitivity: Implications for interpersonal and intergroup processes. Oxford University Press; Oxford: 2001.
73. Ayduk O, Downey G, Kim M. Rejection Sensitivity and Depressive Symptoms in Women. Pers Soc Psychol Bull. 2001; 27(7):868–877.
74. Posternak MA, Zimmerman M. Symptoms of atypical depression. Psychiatry Research. 2001; 104(2):175. [PubMed: 11711170]
75. Goldstrom ID, Burns BJ, Kessler LG, Feuerberg MA, Larson DB, Miller NE, Cromer WJ. Mental health services use by elderly adults in a primary care setting. Journal of Gerontology. 1987; 42:147–153. [PubMed: 3819338]
76. Sachs-Ericsson, N.; Blazer, D. Depression in Late Life: Etiology, Diagnosis and Treatment. Lippincott Williams & Wilkins; Philadelphia: 2006.
77. Levitan RD, Jarrett RB, Schaffer M, McIntire D, Witt-Browder A, Kraft D, Risser RC. Treatment of Atypical Depression With Cognitive Therapy or Phenelzine. Arch Gen Psychiatry. 2000; 57(11):1084. [PubMed: 11074875]
78. Beck A. Cognitive model of depression. Journal of Cognitive Psychotherapy. 1987; 1:2–27.
79. Klerman, GL.; Weissman, MM.; Rounsaville, BJ.; Chevron, ES. Interpersonal Psychotherapy of Depression. Basic Books; New York: 1984.
80. Frank, E.; Frank, N.; Cornes, C. Interpersonal psychotherapy in the treatment of late life depression. In: Klerman, G.; Weissman, M., editors. New Applications of Interpersonal Psychotherapy. American Psychiatric Press; Washington, DC: 1993.
81. Miller MD, Cornes C, Frank E, Ehrenpreis L, Silberman R, Schlernitzauer MA, Tracey B, Richards V, Wolfson L, Zaltman J, Bensasi S, Reynolds CF. III: Interpersonal Psychotherapy for Late-Life Depression: Past, Present, and Future. J. Psychother. Pract. Res. 2001; 10(4):231–238. [PubMed: 11696649]

82. Sullivan PF, Kessler RC, Kendler KS. Latent Class Analysis of Lifetime Depressive Symptoms in the National Comorbidity Survey. Am J Psychiatry. 1998; 155(10):1398–1406. [PubMed: 9766772]

83. Angst J, Gamma A, Benazzi F, Ajdacic V, Rossler W. Melancholia and atypical depression in the Zurich study: epidemiology, clinical characteristics, course, comorbidity and personality. Acta Psychiatr Scand Suppl. 2007; (433):72–84. [PubMed: 17280573]



1. .

Table 1

Demographic characteristics and depression severity indices for the AD symptom group and the Non-AD group

Variable	Atypical Group Mean(SD) or % N=86	Non-Atypical Group Mean(SD) or % N = 162	F^a or χ^2	*p*-value[b]
Age	67.6 (7.1)	70.6 (7.4)	$F = 9.6$	$p < .001$ *
% Female	69.8%	66.0%	$\chi^2 = .247$	$p = .67$
Years of Education	14.9 (2.9)	13.2 (3.0)	$F = 6.0$	$p = .02$
Number of DIS Symptoms	13.0 (2.4)	10.1 (2.4)	$F = 43.4$	$p < .001$ *
Age of Onset	37.7 (20.9)	49.1 (19.5)	$F = 17.8$	$p < .001$ *
Number of Lifetime Episodes	10.3 (20.2)	4.5 (8.1)	Mann-Whiteny $z = 2.95$	$p < .001$ *
% Wanted to Die	43.0%	18.5%	$\chi^2 = 17.1$	$p < .002$ *
Perceived Social Support	21.8 (4.4)	23.5 (3.8)	$F = 9.7$	$p < .001$ *
Perceived Instrumental Support	8.6 (2.4)	9.4 (2.0)	$F = 4.0$	$p < .001$ *
Baseline MADRS	27.6 (8.3)	26.6 (7.3)	$F = 1.3$	$p = .26$

[a] *Df* for F-Tests=1,247, *df* for χ^2 =1

[b] We used a Bonferroni correction for multiple analyses. After Bonferroni correction significance is at p < .004

* Significant after Bonferroni correction

Table 2

Growth Curve Analyses: Tests of Model Fit.

Model	χ^2(df)	CFI	RMSEA	AIC
1. Baseline Linear	539.06(68) *	.46	.17	18247.43
2. Base-Quadratic	289.32(60) *	.74	.12	18013.70
3. Piecewise Linear	360.50(60) *	.65	.14	18084.88
4. Piecewise Quadratic	82.88(41) *	.95	.06	17845.25

* indicates significant at $p<.05$.