

Earlier stress exposure and subsequent major depression in aging women

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Objective: Despite evidence that stress exposure earlier in the life course may have long-term consequences for psychopathology, most models of vulnerability for late life depression are limited to current stressors or to retrospective reports of stress history. This study estimates the influences of earlier stressors assessed longitudinally on subsequent major depressive disorder (MDD) in women at average age 60 (range 50–75).

Method: MDD, negative life events (NLE), and marital stress were assessed multiple times in a community-based sample of 565 women followed for three decades. Adverse events experienced in childhood also were assessed prior to outcome.

Results: Greater childhood adversity, earlier high levels of NLE and marital stress, and a more rapid increase in marital stress over time elevated the odds of MDD at average age 60 independent of all stressors and other salient risk factors. Childhood adversity was mediated in part by intervening risks. Prior depression, earlier poor health status, a more rapid deterioration in health with age, and current disability owing to physical problems also were related independently to later MDD.

Conclusions: These findings support the enduring effects of earlier stress burden on MDD in women into old age and, in light of the increasing proportion of older women in the population, have important clinical implications for identification and treatment of those at risk for depression. Findings also underscore the need to develop resources to counteract or buffer similar stress exposure in younger generations of women. Copyright © 2009 John Wiley & Sons, Ltd.

Key words: late life depression; early stressors; women

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Introduction

Most empirical models of vulnerability for late life depression include recent stressful events among the key risks (Blazer and Hybels, 2005). Nonetheless, despite evidence that early negative experiences have long-term psychological consequences (Tenant, 1988; Spertus *et al.*, 2003), few studies have examined the continued influence of previous stress exposure on depression in elders. Indeed, a recent meta-analysis linking negative life events (NLE) with depression in individuals over age 64 reported that only one out of 25 studies assessed history of NLE (Kraaij *et al.*, 2002). In that study, current depressed mood in community-

dwelling older persons was related to retrospective accounts of disadvantaged circumstances and abuse in childhood and interpersonal relationship stress and NLE in adulthood (Kraaij and Wilde, 2001).

Cross-sectional research on depressogenic impact of earlier stressors is subject to potential bias owing to selective recall of NLE by depressed *versus* nondepressed persons, and may result in unwarranted support for said causal effects. A small number of longitudinal studies implicate previous stressors as risks for increased depressive symptoms in older persons. However, these typically are two wave studies in which symptom change is measured over relatively short intervals and stressors are assessed at outcome.

For example, increased depressive symptoms since baseline were related to more NLE reported at 3-year follow-up by a population-based sample of elders (Fiske *et al.*, 2003), and to paternal death and stressful marital relationships reported at 5-year follow-up by an epidemiologic sample of older women (Kivelä *et al.*, 1996, 1998). It remains unclear, however, whether stressful circumstances assessed at earlier life stages increase risk for later depression, especially clinically significant major depressive disorder (MDD).

Investigation of continuing effects of earlier stress burden on late life depression is best accomplished with longitudinal data; however, such data generally are not available. Moreover, women are at elevated risk for depression throughout the lifespan (Barry *et al.*, 2008), especially in response to prolonged interpersonal stress (Hammen, 2003a); yet to date, stress-related research on elders has focused nearly exclusively on acute NLE. To address those gaps, the current study employs longitudinal data from a community sample of 565 women assessed over three decades to examine whether MDD at average age 60 (hereafter referred to as late life MDD) is associated with childhood adversity, and both NLE and chronic marital stress over adulthood. Other salient risks, including prior depression, earlier poor health, deteriorating health with age, and current disability, are examined in the analyses. Based on the evidence cited above, we hypothesize that greater childhood adversity and higher levels or increasing trajectories of NLE and marital stress earlier in adulthood will elevate vulnerability to late life MDD independent of each other and of other salient risks. We also posit that the relation between childhood adversity and late life MDD will be mediated in part by intervening prior depression or earlier poor health or both.

Method

Sample

The 565 women in this study are from a cohort of 758 mothers randomly selected and interviewed for a child behavior study in 1975 (T_1) based on residence in one of two upstate New York counties and having a child aged one to 10 at home. Follow-up interviews were conducted in 1983, 1986, 1992, and 2003 (T_2 – T_5). These women are primarily white (92%; 7% black, and 1% other); span the full range of socioeconomic statuses; and, at sampling, were demographically representative of mothers of same-aged children in the northeastern region of the United States (Cohen

and Cohen, 1996). Over 90 per cent of this sample participated in at least three of the T_1 – T_4 assessments. At T_5 , 609 of the 685 surviving women (89%) were re-interviewed; those not re-interviewed refused (31) or were ill (17), unavailable (16), or not located (12). Compared to women interviewed at T_5 , those not re-interviewed did not differ significantly with regard to age, lifetime depression, childhood adversity, or NLE or marital stress in earlier waves, but did report poorer health at T_1 – T_4 (e.g., 4.06 vs. 4.24 (raw scores) at T_2 , two-tailed $t = 2.55$, $df = 1, 684$, $p = 0.011$).

Analyses are limited to the 565 women between ages 50–75 at T_5 .

Procedure

Women were interviewed at home by trained experienced lay interviewers. Written informed consent was obtained after the study was described. Data are protected by a Certificate of Confidentiality issued by the National Institutes of Health.

Depression

Prior depression was assessed at T_4 with items adapted from the New York High Risk Study Family History Interview (Moldin *et al.*, 1987). Diagnostic criteria were depressed mood or loss of interest/lack of enjoyment in all things most of the time for 1 month not attributable to bereavement or alcohol/drug use; four additional symptoms (trouble sleeping, sleeping more, tired easily or less energy than usual, change in appetite/weight, difficulty concentrating or paying attention, agitated or fidgety, felt guilty or to blame for things, persistent suicide thoughts or attempt); and impaired role function. Prior depression in these women has been associated with trait neuroticism (Sneed *et al.*, 2007) and offspring psychiatric symptoms (Johnson *et al.*, 2001). At T_5 , the women responded to interview items covering DSM-IV diagnostic criteria for major depressive disorder (MDD), including depressed mood or loss of interest/enjoyment in everything most of the day nearly every day for 2 weeks in the past year not attributable to bereavement, medication, or alcohol use, and four additional symptoms (change in appetite or loss/gain in weight without trying, trouble falling/staying asleep, trouble waking, tired or fatigued/less energy than usual, restless or agitated, difficulty paying attention or concentrating, feeling guilty or to blame for things; persistent thoughts of death/suicide or suicide attempt). Impaired role function during the depressed

state was required. This measure of MDD has been associated significantly with earlier depressive symptoms and obesity in these women (Kasen *et al.*, 2008).

Stress exposure

Three stress measures are included:

- (1) Childhood adversity is a count of negative events occurring before age 16 reported at T_4 , including parental/other close family death, maltreatment, serious illness or disability, serious accident or injury, criminal/violence victimization or witnessing, parental separation/divorce, severe financial hardship, and more than three major residential relocations.
- (2) Negative life events (NLE) were assessed for the past year at T_1 and for the period since each previous interview thereafter. NLE are similar to those covered in other studies (Kendler *et al.*, 2002) and include loss events (death of husband/partner, offspring, parent, other close person); serious medical illness or accident/injury (self or close others); divorce/separation; other marital/family events (e.g., self/partner job loss, criminal involvement or alcohol/drug use by husband/partner or offspring; offspring divorced); serious financial/legal problems; and life-threatening events (e.g., serious fire or other fateful event, physical assault/other victimization, witnessed violence/criminal acts).
- (3) Marital relationship was assessed at T_2 – T_5 with 6 items from the Locke–Wallace Marital-Adjustment and Marital-Prediction Tests (Locke and Wallace, 1959) covering frequency of negative (arguing, name-calling/yelling, rough stuff) and positive (help each other in troubled times, affectionate with each other, spend time together) interactions with husband, ex-husband or current partner relationship, rated from 0 (never or hardly ever) to 4 (always or almost always). Positive and negative interactions both affect relationship quality (Kendler *et al.*, 2002); thus, positive items were reverse scored and combined with negative items to create a marital stress scale. Mean (SD) raw scores are 6.2 (3.4), 6.3 (3.6), 6.4 (3.6), and 4.9 (2.3) across T_2 – T_5 ; Cronbach's α range from 0.80–0.78, indicating satisfactory internal consistency.

Health

Health status was self-rated at T_1 – T_5 with one item, reverse scored from 1 (excellent) to 5 (very poor) so that higher scores reflect poorer health. Mean (SD) raw

scores at T_1 – T_5 are 1.67 (0.68), 1.74 (0.73), 1.75 (0.73), 1.83 (0.75), and 2.56 (1.06), respectively. Self-rated health has been related to physical symptoms in these women (Chen *et al.*, 2007).

Current disability

Disability was assessed at T_5 with the 4-item Role Limitations (due to physical problems) subscale of the Medical Outcomes Survey (SF-36, Ware, Jr. and Sherbourne, 1992). Each item (e.g., had difficulty performing your work or other activities) was scored 2 (yes) or 1 (no), so that higher scores indicate greater disability.

Data analysis

As NLE, marital stress, and health status may vary with age and over time, all data points were used to estimate earlier risk level fixed at a constant age (intercept) and annual linear change in risk (slope or trajectory). To obtain estimates, repeated assessments of NLE, marital stress, and health were employed as dependent variables in individual growth models (Chen and Cohen, 2006): For every woman, each year in the assessed period was measured as a deviation from the year at which she was age 42. Analyses were conducted with SAS PROC MIXED (Littell *et al.*, 1996), which generated empirical Bayes (EB) estimates of intercept and trajectory of NLE, marital stress, and health for each woman. EB estimates are based on maximum likelihood estimations, which tend to be more reliable than estimations based on ordinary least squares (Hedeker, 2004). This method has been used by others with repeated risk measures for later pathologic outcomes to exploit the benefits of having multiple risk assessments over time (McArdle *et al.*, 2005; Perrin *et al.*, 2007).

Logistic regression analysis, conducted with SPSS 15.0 (SPSS, Inc., 2006), estimated effects of childhood adversity, earlier NLE and marital stress (at age 42), and NLE and marital trajectories on late life MDD, independent of prior depression, earlier poor health (at age 42), health trajectory, and current disability. All scaled covariates were standardized for the analyses; raw mean (SD) scores are shown in Table 1. Interactions between covariates also were tested.

Conditional missing data

Marital stress values, which are conditional on role occupancy, were missing during periods without or

Table 1 Sample characteristics: mean and standard deviation (sd) or number and per cent (%) on study variables

Sample characteristics	Mean (SD)	Number (%)
Age at T_1	31.6 (5.8)	
Age at T_2	39.7 (5.8)	
Age at T_3	42.3 (5.8)	
Age at T_4	48.5 (5.8)	
Age at T_5	60.0 (5.7)	
Childhood adversity before age 16	1.00 (1.08)	
Adult NLE estimated at age 42 ^a	3.32 (1.54)	
Annual change in NLE ^a	0.27 (0.15)	
Adult marital stress estimated at age 42	6.01 (2.34)	
Annual change in marital stress	-0.035 (0.02)	
Adult health status (reversed) estimated at age 42	1.78 (0.54)	
Annual change in health status (reversed) ^b	0.04 (0.02)	
Current disability due to physical problems	4.98 (1.46)	
Prior depressive episode		68 (12.0)
Major depressive disorder at mean age 60 (T_5)		47 (8.3)

^aNLE (negative life events) scored cumulatively.

^bIncrease indicative of deteriorating health due to reversed scoring.

not having contact with a spouse, ex-spouse, or partner: 137, 68, and 27 women were missing marital stress data at one, two, and three points in time, respectively. Growth models for repeated measures (described above) were used to extrapolate from actual values at observed waves to replace missing values, based upon the assumption of a linear trend in our longitudinal model.

Multiple imputation methods were applied for the 10 women with missing marital data at all four data points to reduce information loss (Schafer, 1997; Allison, 2001; Little and Rubin, 2002): Age, NLE and health scores (from all women), and marital stress scores (from the 555 women with these data) were used to impute missing values, which were replaced by random draws from a distribution of plausible values generated by SAS PROC MI (SAS Institute, 2007).

Results

Sample characteristics

Sample characteristics pertaining to study variables are shown in Table 1. Mean age was 31.6, 39.7, 42.3, 48.5, and 60.0 at T_1 – T_5 . On average, women reported one childhood adversity. At age 42, mean number of NLE accumulated was 3.3 with a 0.27 unit annual increase; mean marital stress was 6.01, with a 0.035 unit annual decline; and mean health status was 1.78, with a 0.021 unit annual decline, the latter reflected by increasing score values owing to reversed scoring of health. Sixty-eight (12.0%) women met diagnostic criteria for prior depression; 47 (8.3%) met criteria for late life MDD.

Relations among previous stressors

Correlations between study covariates are shown in Table 2. The largest ($r=0.71$) indicates that earlier high NLE (at age 42) is related to a more rapidly increasing NLE trajectory. Other significant correlations between stressors indicate that greater childhood adversity is related to earlier high NLE ($r=0.10$) and a more rapidly increasing NLE trajectory ($r=0.09$); that earlier high NLE also is related to earlier high marital stress ($r=0.17$) and slower decline in marital stress ($r=-0.12$); but that earlier high marital stress is related to more rapid *decline* in marital stress ($r=-0.36$).

Main effects

Odds ratios compare women at high risk for late life MDD (mean > 1 SD) to those at low risk (mean < 1 SD); associations were examined first at a bivariate level (not tabled): Odds increased for women with greater childhood adversity (OR = 1.87, 95% Confidence Interval (CI) = 1.45–2.41), earlier high NLE (OR = 2.69, CI = 2.07–3.51), a more rapidly increasing NLE trajectory (OR = 1.88, CI = 1.44–2.46), earlier high marital stress (OR = 2.42, CI = 1.80–3.25) and a more rapidly increasing marital stress trajectory (OR = 2.31, CI = 1.69–3.17). Odds also were elevated for younger women (within the 50–75 age range) (OR = 0.84, CI = 0.79–0.89), and for women with prior depression (OR = 16.91, CI = 8.60–33.25), earlier poor health (OR = 3.20, CI = 2.37–4.31), more rapidly

Table 2 Correlation coefficients among study covariates

	2	3	4	5	6	7	8	9	10
(1) Age at outcome (T_5)	0.02	-0.15*	-0.17*	-0.04	0.03	-0.08	-0.29*	-0.04	0.10*
(2) Childhood adversity		0.10*	0.09*	0.03	0.02	0.15*	0.15*	0.08	0.12*
(3) Adult NLE estimated at age 42 ^a			0.71*	0.17*	-0.12*	0.16*	0.10*	0.02	0.06
(4) Annual change in NLE ^a				0.08	-0.04	0.12*	0.11*	0.04	0.10*
(5) Adult marital stress estimated at age 42					-0.36*	0.14*	0.15*	0.09*	0.06
(6) Annual change in marital stress						-0.03	-0.02	0.06	-0.01
(7) Prior MDD episode							0.09*	-0.02	0.02
(8) Adult health status (reversed) estimated at age 42								0.31*	0.34*
(9) Annual change in (reversed) health status									0.39*
(10) Current physical disability									

^aNLE (negative life events) scored cumulatively.

* $p < 0.05$.

deteriorating health (OR = 1.95, CI = 1.50–2.55), and greater current disability (OR = 3.03, CI = 2.34–3.94).

Each earlier stressor except an increasing NLE trajectory was associated independently with late life MDD when examined simultaneously (controlling age), with minimal change in bivariate odds cited above (Model 1, Table 3). Analyses not shown here revealed that the reduced effect of NLE trajectory was attributable to the addition of earlier adult NLE into the equation, owing in part to their high correlation ($r = 0.71$, Table 2) and to the cumulative scoring of NLE; thus, NLE trajectory was not considered further. Albeit reduced, odds for late life MDD remained significantly elevated with greater childhood adversity (OR = 1.45), earlier high NLE (OR = 2.09) or marital stress (OR = 1.58), and more rapidly increasing marital stress (OR = 2.19), independent of age, prior depres-

sion, earlier poor health, and more rapidly deteriorating health, all of which also continued to significantly elevate odds for late life MDD (Model 2, Table 3). Those effects were virtually unchanged after accounting for current disability, which was significantly associated with MDD (OR = 2.00) independent of earlier risks (Model 3, Table 3).

Mediation effect

The significant relation between childhood adversity and late life MDD in Model 1 was attenuated in Model 2 after consideration of prior depression and earlier poor health (Table 3). Other criteria for a mediator model outlined by Baron and Kenny (1986) were met for prior depression and earlier poor health (but not

Table 3 Odds of late-life MDD associated with earlier stress exposure, adult health, prior depression, and current disability in 565 women

Risk factors for MDD	Model 1		Model 2		Model 3	
	OR	95%CI	OR	95%CI	OR	95%CI
Age at outcome (T_5)	0.86**	0.81–0.92	0.92*	0.86–0.98	0.90*	0.85–0.97
Previous stress exposure scales ^a						
Childhood adversity	1.81**	1.38–2.37	1.45*	1.08–1.94	1.34	0.99–1.81
Adult NLE estimated at age 42 ^b	2.82**	1.92–4.16	2.09**	1.55–2.81	2.07**	1.53–2.81
Annual change in NLE ^b	0.77	0.52–1.13	—	—	—	—
Adult marital stress estimated at age 42	2.02*	1.47–2.77	1.58*	1.14–2.19	1.57*	1.13–2.18
Annual change in marital stress	2.58**	1.86–3.57	2.19**	1.57–3.07	2.16**	1.54–3.04
Prior MDD episode			9.24**	4.36–19.62	9.22**	4.52–20.91
Health status (reversed) scales ^a						
Adult health status estimated at age 42			2.26**	1.63–3.13	1.81*	1.28–2.56
Annual change in health status			2.01**	1.52–2.65	1.65*	1.23–2.20
Current physical disability ^a					2.00**	1.44–2.75

Note: Odds ratios compare women at high risk (mean above +1 SD) to women at low risk (mean below -1 SD).

^aScales standardized.

^bNLE (negative life events) scored cumulatively.

* $p < 0.05$.

** $p < 0.01$.

deteriorating health): Childhood adversity was correlated significantly with prior depression and earlier poor health (Table 2), and each significantly elevated the odds for late life MDD (Table 3). Table 4 shows increased odds for late life MDD with greater childhood adversity first at a bivariate level (OR = 1.87); then independent of prior depression (OR = 1.59), earlier poor health (OR = 1.58) and both prior depression and earlier poor health (OR = 1.39), indicating their additive effects.

Interaction effects

Covariate interactions were tested but were not significant beyond chance levels.

Discussion

We estimated long-term consequences of stressors for late life MDD in community-dwelling women followed longitudinally over three decades: Greater childhood adversity, earlier high NLE and marital stress, and an increasing trajectory of marital stress with age all elevated odds for subsequent MDD. These findings are compatible with retrospective reports that previous stress exposure elevates risk for depressive syndrome in older persons (Kraaij *et al.*, 2002). Our research also extends earlier work by estimating influences of multiple stressors assessed longitudinally over a lengthy period on MDD in aging women independent of other salient risks.

Compatible with reports on population-based samples that older individuals have the lowest

prevalence of major depression (Regier *et al.*, 1993), older women in this sample were at decreased odds for MDD. Consistent with research on recurrent depressive episodes in women (Hammen, 2003b), prior depression increased the odds of later MDD. Both earlier poor health and more rapidly deteriorating health with age also elevated the odds of late life MDD, supporting the link between increasing medical burden and depression in older persons (Alexopoulos *et al.*, 2002). For example, sustained elevated levels of proinflammatory cytokines (Thomas *et al.*, 2005) and age-related structural brain changes owing to vascular risk factors and ischemic lesions (Alexopoulos *et al.*, 1997b; Krishnan *et al.*, 2004) have been implicated in emergence of late life depression. In fact, vascular depression has been proposed as a distinct diagnostic subtype of late life depression (Alexopoulos *et al.*, 1997a; Steffens and Krishnan, 1998), and recent evidence supports its internal validity (Sneed *et al.*, 2008). Feeling poorly earlier in adulthood may portend biologic risk for late life depressive onset. In addition, consistent with others' findings that disability and late life depression are highly related (Schillerstrom *et al.*, 2008), odds of MDD increased with current disability independent of earlier risks; these data, however, do not permit a directional interpretation of this finding.

Partial attenuation of the childhood adversity–late life MDD association was indicated owing to intervening depression and earlier poor health. Early traumatic experiences may result in a broad range of adult behavioral, emotional, and physical problems that elevate vulnerability for late life depression. Others have found that intervening depression plays a critical mediating role between such experiences and later adult depression (Kessler and Magee, 1993). The finding here reinforces the perspective that childhood adversities may have enduring effects by establishing less desirable pathways earlier in life (Wheaton and Gotlib, 1997).

Earlier high NLE doubled the odds of late life MDD independently of other salient risks, bolstering reports based on differential recall of previous stressors by depressed *versus* non-depressed elders. Moreover, notwithstanding expectations that proximal risks are more lethal than distal risks, this finding also corroborates the notion that high stress exposure has long-term consequences for depression, and suggests that stress burden may be cumulative over the lifespan (Turner *et al.*, 1995).

An increasing trajectory of marital stress and, to a lesser extent, earlier high marital stress, both significantly raised the odds for late life MDD. This finding is

Table 4 Mediation of childhood adversity effects by intervening risks prior depression and poor health status earlier in adulthood

Mediator models	OR	95%CI
(1) Childhood adversity ^a	1.87**	1.45–2.41
(2) Childhood adversity ^b	1.59*	1.23–2.06
(3) Childhood adversity ^c	1.58*	1.20–2.07
(4) Childhood adversity ^d	1.39*	1.05–1.82

Note: Odds ratios compare those at high risk (mean above +1 SD) to those at low risk (mean below –1 SD). All scales used in the analyses standardized.

^aBivariate odds.

^bOdds independent of prior depression.

^cOdds independent of adult health status (reversed) estimated at age 42.

^dOdds independent of both prior depression and adult health status (reversed) estimated at age 42.

* $p < 0.05$.

** $p < 0.01$.

Key Points

- Earlier stress assessed longitudinally elevated the risk for major depressive disorder in a community-based sample of older women independent of other salient risks.
- Greater adversity in childhood, higher levels of negative life events and marital stress earlier in adulthood, and an increasing trajectory of marital stress with age, all were associated independently with later major depressive disorder.
- The association between adversities in childhood and later major depressive disorder was mediated in part by an intervening depressive episode and poor health earlier in adulthood.

compatible with reports based on age group differences that marital relationships assume more importance for psychological well-being in late life than the middle adult years (Cartensen *et al.*, 1995; Mancini and Bonanno, 2006). Indeed, others have shown that an increasing trajectory of marital stress is more debilitating to health in older women than younger women (Umberson *et al.*, 2006). Younger women in high stress marriages also are more likely to divorce, reducing stress, which may explain the inverse correlation found here between earlier high marital stress and more rapidly declining marital stress (Table 2). Moreover, divorce may have led to successful remarriage among some women, resulting in enhanced relationship quality. Our finding, based on longitudinal data, suggests that marital/partner relationship quality is related to mental health in adult women, but becomes increasingly more important as women grow older, perhaps owing to the increasing vulnerabilities inherent in aging.

Conclusion

Findings here support enduring effects of earlier stress burden on MDD among aging women. Stressors examined emphasized loss, problems involving family, and interpersonal stress with spouse/partner rather than, for example, negative workplace experiences. Among women in maternal and marital/partner roles, however, family-related stressors may be the most depression-provoking. Late life depression increases risk for medical illness and poor prognosis, and physical and cognitive functional decline; accordingly, these findings have important implications for

identification and treatment of aging women with elevated vulnerability. In addition, findings also bear on younger generations of women: Development of resources and prevention efforts that counteract or buffer stressful experiences common to women in family roles would not only be advantageous for their mental health but also would benefit their families and society as a whole.

Findings should be interpreted in light of study limitations. These women all are mothers and primarily white; thus, findings cannot be generalized to women without children or in other racial groups. Nonetheless, this sample was selected to be demographically representative of mothers with same-age offspring living in the northeastern United States at time of sampling, a substantial population for whom results may be applicable. Childhood adversity and NLE were recalled over lengthy intervals between waves, increasing potential for recall bias. Although partner support was assessed in the marital stress measure, support from a wider social network also may influence late life depression (Kubzansky *et al.*, 2000). Self-rated health served as a proxy for biologic pathways implicated in late life depression. Albeit a shortcoming, self-rated health may be a plausible measure owing to its associations with subsequent mortality (Idler and Benyamini, 1997) and chronic illness (Shadbolt, 1997), and with biomarker indicators of age-related disease pathology (Lekander *et al.*, 2004; Jylha *et al.*, 2006). Because MDD was assessed by structured interview administered by trained lay interviewers, elements of clinical judgment and flexible questioning in clinical interviews, considered by some to be the standard by which other diagnostic instruments should be measured (e.g., Spitzer, 1983), were missing. Structured interviews may be more suitable for population-based samples, however, owing to increased feasibility; higher prevalence of borderline cases (for which clinician agreement declines); and evidence that clinical interviewing may elicit evasion in community participants (Wittchen *et al.*, 1999). Because MDD was not assessed between T_4 and T_5 , an 11-year interval, we may have missed prior depressive episodes. Antidepressant use may reduce depressive syndrome in elders (Nelson *et al.*, 2008) but was not examined; thus, analyses did not account for that potential effect.

Conflict of Interest

The authors have no potential conflicts of interest to be disclosed.

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REFERENCES

- Allison PD. 2001. *Missing Data*. Sage Publications: Thousand Oaks, CA.
- Alexopoulos GS, Buckwalter K, Olin J, et al. 2002. Comorbidity of late life depression: an opportunity for research on mechanisms and treatment. *Biol Psychiatry* **52**: 543–558.
- Alexopoulos GS, Meyers BS, Young RC, et al. 1997a. 'Vascular depression' hypothesis. *Arch Gen Psychiatry* **54**: 915–922.
- Alexopoulos GS, Meyers BS, Young RC, et al. 1997b. Clinically defined vascular depression. *Am J Psychiatry* **154**: 562–565.
- Baron RM, Kenny DA. 1986. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* **51**: 1173–1182.
- Barry LC, Allore HG, Guo Z, Bruce ML, Gill TM. 2008. Higher burden of depression among older women. *Arch Gen Psychiatry* **65**: 172–178.
- Blazer DG, Hybels CF. 2005. Origins of depression in later life. *Psychol Med* **35**: 1241–1252.
- Cartensen LL, Gottman JM, Levenson RW. 1995. Emotional behavior in long-term marriage. *Psychol Aging* **10**: 140–149.
- Chen H, Cohen P. 2006. Using individual growth model to analyze the change in quality of life from adolescence to adulthood. *Health Qual Life Outcomes* **4**: 10. DOI: 10.1186/1477-7525-4-10. Available at <http://www.hqlo.com/content/4/1/10>
- Chen H, Cohen P, Kasen S. 2007. Cohort differences in self-rated health: evidence from a three-decade, community-based, longitudinal study of women. *Am J Epidemiol* **166**: 439–446.
- Cohen P, Cohen J. 1996. *Life Values and Adolescent Mental Health*. Lawrence Erlbaum Associates, Inc: Mahwah, NJ.
- Fiske A, Gatz M, Pederson NL. 2003. Depressive symptoms and aging: the effects of illness and non-health-related events. *J Gerontol Psychol Sci* **58B**: P320–P328.
- Hammen C. 2003a. Interpersonal stress and depression in women. *J Affect Disord* **74**: 49–57.
- Hammen C. 2003b. Social stress and women's risk for recurrent depression. *Arch Womens Ment Health* **6**: 9–13.
- Hedeker D. 2004. An introduction to growth modeling. In *Quantitative Methodology for the Social Sciences*, Kaplan D (ed). Sage Publications: Thousand Oaks, CA; 215–234.
- Idler EL, Benyamini Y. 1997. Self-rated health and mortality: a review of twenty-seven community studies. *J Health Soc Behav* **38**: 21–37.
- Johnson JG, Cohen P, Kasen S, Smailes EM, Brook JS. 2001. Associations of maladaptive parental behavior with psychiatric disorder among parents and their offspring. *Arch Gen Psychiatry* **58**: 453–460.
- Jylha M, Volpato S, Guralnik JM. 2006. Self-rated health showed a graded association with frequently used biomarkers in a large population sample. *J Clin Epidemiol* **59**: 465–471.
- Kasen S, Cohen P, Chen H, Must A. 2008. Obesity and psychopathology in women: a three decade prospective study. *Int J Obesity* **32**: 558–566.
- Kendler KS, Gardner CO, Prescott CA. 2002. Toward a comprehensive developmental model for major depression in women. *Am J Psychiatry* **159**: 1133–1145.
- Kessler RC, Magee WJ. 1993. Childhood adversities and adult depression: basic patterns of association in a U.S. National Survey. *Psychol Med* **23**: 679–690.
- Kivelä SL, Königä-Saviaro P, Laippala P, Pahkala K, Kesti E. 1996. Social and psychosocial factors predicting depression in old age: a longitudinal study. *Int Psychogeriatr* **8**: 635–644.
- Kivelä SL, Luukinen H, Koski K, Viramo P, Pahkala K. 1998. Early loss of mother or father predicts depression in old age. *Int J Geriatr Psychiatry* **13**: 527–530.
- Kraaij V, Arensman E, Spinhoven P. 2002. Negative life events and depression in elderly persons: a meta-analysis. *J Gerontol Psychol Sci* **57B**: P87–P94.
- Kraaij V, de Wilde EJ. 2001. Negative life events and depressive symptoms in the elderly: a life span perspective. *Aging Ment Health* **5**: 84–91.
- Krishnan KR, Taylor WD, McQuoid DR, et al. 2004. Clinical characteristics of magnetic resonance imaging-defined subcortical ischemic depression. *Biol Psychiatry* **55**: 390–397.
- Kubzansky LD, Berkman LF, Seeman TE. 2000. Social conditions and distress in elderly persons: findings from the MacArthur Studies of Successful Aging. *J Gerontol Psychol Sci* **55B**: P238–P246.
- Lekander M, Elofsson S, Neve IM, et al. 2004. Self-rated health is related to levels of circulating cytokines. *Psychosom Med* **66**: 559–563.
- Littell RC, Milliken GA, Stroup WW, Wolfinger RD. 1996. *SAS System for Fixed Models*. SAS Institute, Inc: Cary, NC.
- Little R, Rubin D. 2002. *Statistical Analysis with Missing Data*, 2nd edn. Wiley: New York.
- Locke HK, Wallace JM. 1959. Short marital adjustment and prediction tests: their reliability and validity. *Marriage Fam Living* **21**: 251–255.
- Mancini AD, Bonanno GA. 2006. Marital closeness, functional disability, and adjustment in late life. *Psychol Aging* **21**: 600–610.
- McArdle JJ, Small BJ, Bäckman L, Fratiglioni L. 2005. Longitudinal models of growth and survival applied to the early detection of Alzheimer's disease. *J Geriatr Psychiatry Neurol* **18**: 234–241.
- Moldin SO, Gottesman II, Erlenmeyer-Kimling L. 1987. Psychometric validation of psychiatric diagnoses in the New York High-Risk Study. *Psychiatry Res* **22**: 159–177.
- Nelson CJ, Delucchi K, Schneider LS. 2008. Efficacy of second generation antidepressants in late-life depression: a meta-analysis of the evidence. *Am J Geriatr Psychiatry* **16**: 558–567.
- Perrin MA, Chen H, Sandberg DE, Malaspina D, Brown AS. 2007. Growth trajectory during early life and risk of adult schizophrenia. *Br J Psychiatry* **191**: 512–520.
- Regier DA, Farmer ME, Rae DS, et al. 1993. One-month prevalence of mental disorders in the United States and sociodemographic characteristics: the epidemiologic catchment area study. *Acta Psychiatr Scand* **88**: 35–47.
- SAS Institute, Inc. 2007. *Statistical Analysis System, Version 9.1*, SAS Institute, Cary, NC.
- Schafer JL. 1997. *Analysis of Incomplete Multivariate Data*. Chapman & Hall: New York.
- Schillerstrom JE, Royall DR, Palmer RF. 2008. Depression, disability and intermediate pathways: a review of longitudinal studies in elders. *J Geriatr Psychiatry Neurol* **21**: 183–197.
- Shadbolt B. 1997. Some correlates of self-rated health for Australian women. *Am J Public Health* **87**: 951–956.
- Sneed JR, Kasen S, Cohen P. 2007. Early life risk factors of late-onset depression. *Int J Geriatr Psychiatry* **22**: 663–667.
- Sneed JR, Rindskopf D, Steffens DC, et al. 2008. The vascular depression subtype: evidence of internal validity. *Biol Psychiatry* **64**: 491–497.
- Sperthus IL, Yehuda R, Wong CM, Halligan S, Seremetis SV. 2003. Childhood emotional abuse and neglect as predictors of psychological and physical symptoms in women presenting to a primary care practice. *Child Abuse Negl* **27**: 1247–1258.
- Spitzer RL. 1983. Psychiatric diagnosis: are clinicians still necessary? *Compr Psychiatry* **24**: 399–411.
- SPSS, Inc. 2006. *SPSS 15.0 Basic User's Guide*. Prentice-Hall: Chicago, IL.
- Steffens DC, Krishnan KR. 1998. Structural neuroimaging and mood disorders: recent findings, implications for classification, and future directions. *Biol Psychiatry* **43**: 705–712.
- Tenant C. 1988. Parental loss in childhood: its effect in adult life. *Arch Gen Psychiatry* **45**: 1045–1050.
- Thomas AJ, Davis S, Morris C, et al. 2005. Increase in interleukin-1 β in late-life depression. *Am J Psychiatry* **162**: 175–177.
- Turner RJ, Wheaton B, Lloyd DA. 1995. The epidemiology of social stress. *Am Soc Rev* **60**: 104–125.

- Umberson D, Williams K, Powers DA, Lui H, Needham B. 2006. You make me sick: marital quality and life over the life course. *J Health Soc Behav* **47**: 1–16.
- Ware JE Jr, Sherbourne CD. 1992. The MOS 36-Item Short-Form-Health Survey (SF-36): I. Conceptual framework and item selection. *Med Care* **30**: 473–483.
- Wheaton B, Gotlib IH. 1997. Trajectories and turning points over the life course: concepts and themes. In *Stress and Adversity over the Life Course: Trajectories and Turning Point*, Gotlib IH, Wheaton B (eds). Cambridge University Press: Cambridge, UK; 1–25.
- Wittchen H-U, Üstün TB, Kessler RC. 1999. Diagnosing mental disorders in the community: a difference that matters? *Psychol Med* **29**: 1021–1027.