AGING AND LATE-LIFE DEPRESSION*

Zheng Wu, PhD
Department of Sociology, University of Victoria, Victoria, BC V8W 3P5
Email: zhengwu@uvic.ca

Christoph M. Schimmele, M.A.
Department of Sociology, University of Victoria, Victoria, BC V8W 3P5
Tel. 250-588-3242 Email: chrissch@uvic.ca

Neena L. Chappell, PhD
Department of Sociology and Centre on Aging, University of Victoria, Victoria, BC V8W 3P5
Email: nlc@uvic.ca

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ABSTRACT

Our objective is to estimate age-related changes in depressive symptomatology among senior-age persons, while addressing important methodological limitations within previous studies. The empirical analysis used 3 waves of longitudinal data (1991, 1996, 2001) and cross-sectional data from a nationally representative, age- and sex-stratified sample of 10,263 Canadian aged ≥65. We used generalized linear mixed model (GLMM) techniques to estimate age-related changes in level of depressive symptoms, the risk of major depressive disorder (MDD), and the risk of subsyndromal depression (SSD). There is an age-related increase in depressive symptoms in late-life, but it occurs through indirect mechanisms and is not an effect of age per se. The relationship between age and the risk of MDD is significant and non-linear (U-shaped), after adjusting for selected covariates. There is no significant age effect on the risk of SSD for seniors. There are two important conclusions. First, physiological and mental declines mediate the age-depression relationship, making late-life depressive symptomatology a largely co-morbid condition. Second, the age-depression relationship for the risk of MDD is non-linear, and persists after introducing controls for physiological and mental decline. Our findings illustrate that the risk of MDD decreases between ages 65-79 and increases around ages 80-83.

AGING AND LATE-LIFE DEPRESSION

There is a well-established linkage between age or life-stage and depressive symptomatology (Jorm 2000; Karel, 1997; Kessler et al. 1992; Mirowsky and Ross 1992). In Canada, Patten et al. (2006) observe that the 12-month prevalence of major (or clinical) depression is 5% for ages 15-25, 4.5% for ages 26-45, 3.7% for ages 46-64, and 1.9% for ages 65 and older. Hence, Patten et al. demonstrate that the prevalence of depression appears to decline as a person ages, which contradicts mainstream presumptions that old-age is a depressing life-stage. What remains indefinite, however, is whether this aggregate finding represents a low prevalence of depression for seniors of all ages or if it is attributable largely to young-old seniors. The young-old often manage to ward-off sources of depressive symptoms (stressors) through social support and other health-fostering assets (Pearlin and Skaff, 1995). But these protective resources cannot postpone age-related losses forever, considering that the biological potential for sustaining robust health is not unlimited, and significant declines in well-being are not uncommon experiences for the old-old and oldest-old (Baltes 1997; Baltes and Lindenberger 1997; Smith and Baltes, 1997). In this respect, there could be a ceiling effect on successful adaptation to the accumulation of age-related losses, and thus there could be an age-related threshold of mental well-being.

There is an abundance of research on the prevalence, risk factors, prognosis, and consequences of late-life depression (e.g., Alexopoulos 2005; Blazer 2003; Newman, Sheldon, and Bland 1998; Østbye et al. 2005; Smith and Baltes 1997). However, the reliance on cross-sectional data, among other fundamental limitations, has hitherto

constrained research into aging-related changes in late-life depression. The usage of cross-sectional data (or between person comparisons) is inappropriate for estimating age effects on late-life depression. The proper estimation of age effects requires longitudinal observations (or within person comparisons) in order to disentangle changes associated with period- or cohort-specific effects from changes associated with life-stage or chronological effects (Karel 1997; Yang 2007). Hence, most earlier estimates of late-life depression were unable to generate a reliable understanding about age effects, leaving a crucial question unanswered: Does the prevalence of late-life depression increase, decrease, or remain stable as senior-aged people get older?

We use a longitudinal approach to examine if age has an independent effect on the course of depression for Canadian seniors. Our research improves on prior longitudinal studies in several important respects. First, our empirical analysis models age-related changes in the risk of major depressive disorder (MDD) and subsyndromal depression (SSD), in addition to measuring changes in depressive symptoms. To our knowledge, all previous longitudinal studies consider depressive symptoms alone, which is an insufficient for determining age-related changes in the prevalence of late-life MDD or SSD. Furthermore, our measurement of depressive symptoms and depression is based on a clinician-administered scale, which is a preferred assessment of late-life depression and is an improvement over self-reported measures (Nguyen and Zonderman 2006). Second, our regression models adjust for different types of dementia, which often present symptoms that mimic or are otherwise mistaken for depressive symptoms (Ballard, Bannister, and Oyebode 1996). Third, our study sample includes both community-

dwelling and institution-dwelling seniors, whereas most prior longitudinal studies follow community-dwelling seniors.

PRIOR STUDIES

Although most previous studies on the relationship between aging and late-life depression depend on cross-sectional data, there are important exceptions. For example, Yang (2007) examines if age influences growth in late-life depressive symptoms in cohort-specific terms, using longitudinal data from a community-dwelling US sample. She observes a gross age effect on late-life depression, and that, in general, aging appears to produce a monotonic increase of depressive symptoms. However, Yang's findings also demonstrate that, while there is an age-related increase of depressive symptoms in late-life, this pattern of growth unfolds through an indirect process. As certain risk factors of depression become more prevalent at older ages, such as chronic illnesses and physical disabilities, these age-related conditions trigger corresponding increases of depressive symptoms, but aging per se is not a risk factor. Moreover, Yang's findings suggest that the overall effect of age on late-life depressive symptoms is cohort-driven. In accordance, there is substantial inter-cohort variation in the average levels and growth of late-life depressive symptoms; i.e., depressive symptoms increase with age for some cohorts, whereas these decrease with age for others. This inter-cohort variation in the trajectories of late-life depression is attributable to commensurate differences in martial status, sex composition, socio-economic status, and physiological decline.

There are a few other notable studies that use a longitudinal approach to model the age-depression relationship in late life. Anstey et al. (2007) demonstrate that the level of depressive symptoms increases over time for seniors of all ages. However, the rate of

change in depressive symptoms is steeper for seniors aged 75 and older in comparison to seniors of younger ages. This age-time interaction shows that the growth in the level of depressive symptoms accelerates year-after-year after age 75. Anstey et al. indicate that this growth in depressive symptoms corresponds to age-related declines in functional and cognitive well-being. According to Nguyen and Zonderman (2006), the level of depressive symptoms in late-life remains stable until about age 70, but begins to mount thereafter. Upon reaching old-old age, there are linear age-related increases of somatic complaints and a lack of well-being (hopelessness), and these age effects are independent of co-morbid conditions. Rothermund and Brandtstädter (2003) also observe that the level of depressive symptoms is stable until age 70 and then begins to increase. Hence, the authors identify two distinct phases of depressive symptomatology in late life. In the first phase, seniors tend to adapt to typical age-related losses and challenges, and thus depressive symptoms do not increase over time among the young-old. However, in the second phase, the age-related accumulation of these losses and challenges eventually overtaxes personal coping resources, leading to a subsequent incline of depressive symptoms.

Although these studies used prospective data to make inferences on age-depression relationship for seniors, each has one or more of the following data limitations: 1) the measures of depressive symptoms are based on self-reported data, 2) the analyses do not account for dementia, and 3) the study samples are limited to community-dwelling seniors.

For several reasons, assessing depression in older adults appears to require a different approach than for non-senior adults. First, some studies observe that self-

reported scales tend to over-estimate depression among seniors through attributing somatic symptoms (e.g., chronic fatigue, weight loss, insomnia), bereavement, or other distress to depression, even though other medical or psychosocial problems could be responsible for these symptoms (Karel 1997; Kessler et al. 1992). Second, different assessment techniques can result in inconsistent estimates. Nguyen and Zonderman (2006) observe that standardized scales (self-reported measures) tend to indicate a positive curvilinear (or U-shaped) pattern of late-life depression, whereas clinical diagnoses tend to indicate a negative curvilinear (or an opposite) pattern. Third, as Watson et al. (2004) suggest that, conventional instruments, including the Geriatric Depression Scale (GDS) and the Center for Epidemiological Studies – Depression (CES-D), are inappropriate instruments for assessing clinical depression in the old-old and oldest-old. The authors report that two-week follow-ups to GDS and CED-D assessments, which geriatric psychiatrists conducted using the Structural Clinical Interview for DSM (SCID) disorders, indicated that standardized cut-points underestimated both major depressive disorder and subsyndromal depression in old-old seniors. Overall, the literature indicates that clinician-based diagnoses represent a superior tool for assessing late-life depression.

To be accurate, the estimation of late-life depression requires controlling for symptoms of different types of dementia. This is because there is a strong concurrence of depression and dementia symptoms (Ballard, Bannister, and Oyebode 1996). In Canada, the reported prevalence of MDD is 9.5 percent for seniors with dementia, which is 5 times higher than the general prevalence of late-life depression (Government of Canada 2006). The clinical presentation of dementia tends to overlap the clinical features of

depression, including apathetic mood, concentration problems, and anhedonia. This duplication of symptoms implies that it is crucial to disentangle dementias from depression in order to obtain valid measurements (Alexopoulos et al. 2002). Moreover, self-reports of depressive symptoms may be unreliable scales for measuring depression in demented patients because dementia-related impairments in concentration, communication abilities, and personal insight can distort self-assessments (Bedard and O'Donnell 2003; Shankar and Orrell 2000).

For two fundamental reasons, the exclusion of full-time residents of institutions (e.g., nursing homes, hospitals) could introduce a selection bias into estimates of age effects on late-life depression. First, age increases the risk of institutionalization. Using the 2001 Canadian Census data, we found that, of Canadians aged 85-89, about 17% of males and 27% of females are full-time residents of institutions. Among 90-94 year-olds, 30% of males and 44% of females are full-time institution residents (the complete table of age and institution status is available on request). Second, the prevalence of MDD for Canadian seniors in long-term care facilities is 3-4 times higher that in the general population (Conn 2002). When full-time residents of institutions are included, the 12-month prevalence of late-life MDD escalates from about 2 percent to between 10 and 15 percent. Thus, excluding institution residents could underestimate or otherwise miss important age-related changes in late-life depression.

DATA AND METHODS

Data

Our empirical analysis is based on 3 waves of Canadian Study of Health and Aging (CSHA) data. The CSHA is a longitudinal, population-based epidemiological survey and consists of a nationally representative sample of 10,263 Canadians aged 65 and older residing in communities and institutions. The sample excluded seniors from the Yukon and Northwest Territories (remote areas), Indian reserves, military bases, individuals with a life-threatening illness (e.g., terminal cancer, conditions that require life support), and those unable to communicate in English or French. The participants were assessed at 5-year intervals in 1991, 1996, and for a final time in 2001. The main objective of the CSHA was to determine the prevalence of dementia for seniors, its risk factors, and its impact on informal caregivers. The CSHA also collected detailed clinical data on several additional health topics, including chronic illness, functional and cognitive well-being, neurological health, and depressive symptoms.

The community-based sample was selected using an age- and sex-stratified sampling design, over-sampling persons over age 75. All participants were randomly selected from 36 major Canadian cities and their neighboring areas (strata). The institution-based sample also used an age- and sex-stratified sampling design, and included seniors living in nursing homes, chronic care facilities, hospitals, and various other types of institutional settings. As with the community sample, institutionalized persons over age 75 were over-sampled. The total sample of the CSHA consists of 9,008 seniors from communities and 1,255 from institutions. The overall participation rate was 72.1% for the community sample and 81.7% for the institution sample. The overall sample represents approximately 60% of the Canadian population aged 65 and older in 1991 (for details see McDowell et al. 1994).

The current study focused on respondents who completed a full clinical examination, which included a physician-made assessment of DSM-III-R depressive symptoms. The total Wave 1 sample has 2,914 seniors, including 1,255 institutional residents. The original sample reduced to 1,149 seniors (693 community participants and 456 institutional participants) in Wave 2 due to attrition, with a participation rate of 84.5% for the community sample and 90% for the institution sample. In Wave 3, the study sample reduced to 317 (213 community participants and 104 institutional participants), with participation rates of 89.4% for the community sample and 90.6% for the institution sample (CSHA 2008).[1] We merged all 3 waves and created a "person-year" dataset for statistical analyses, amounting to 4,380 "person-year" observations. The *N* reduced to 3,652 after eliminating cases with missing data.[2]

Measures

Our dependent variables are total depressive symptoms (TDS), major depression (MDD), and subsyndromal depression (SSD). CSHA physicians assessed depressive symptoms using Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R) criteria. The DSM-III-R is a standard instrument for depression diagnosis and is reliable for diagnosing depression in older adults (Alexopoulos et al. 1993; Segal et al. 1993). The assessment of depressive symptoms was based on a 12-item scale, including depressed mood (sadness), diminished interest or pleasure (anhedonia), feelings or worthlessness or guilt, recurrent thoughts of death or suicide ideation, appetite or weight changes, sleep disturbances, inability to concentrate, psychomotor agitation/retardation, and fatigue or decreased energy. We measured TDS as a "continuous" variable (number of symptoms)

derived from this scale. Following DSM conventions, we constructed dichotomous variables to indicate MDD and SSD. The DSM cutoff for MDD is the presence of ≥5 depressive symptoms, which must include either sadness or anhedonia, and persistence of symptoms for 2 continuous weeks or longer. The cutoff for SSD is 2-4 depressive symptoms, including either sadness or anhedonia, and persistence of symptoms for at least 2 continuous weeks.

Table 1 presents variable definitions, descriptive statistics, and their standard deviations for the community and institution samples. For each variable, a bivariate significance test between the two groups was conducted, and *p* values for the tests are reported in the last column of the table. All statistics reported in the table are based on Wave 1 data.

<Table 1 about Here>

All measures of depressive symptoms and depression are time-variant. Table 1 shows a prevalence of MMD of 2.6% within the community sample and 7% within the institution sample. The prevalence of SSD is 3.5% among community-dwelling seniors and 4.1% among institutionalized seniors.

The primary independent variable is age and it was measured as a continuous variable and mean-centered. We generated a quadratic term for age because the relationship between aging and our outcome variables could be non-linear. Our baseline regression models also contain control variables for sex, marital status, place of residence, visible minority status, and education. Marital status was measured as a 4-level categorical variable: Never married, separated or divorced, widowed, and married or cohabiting. We measured place of residence as a dummy variable, indicating whether the

participant lived in a rural area. We used another dummy variable, indicating whether the participant belonged to a visible minority group. Formal education was measured in years and mean-centered. With the exception of age, these variables are time-invariant and were measured at Wave 1. Information on marital status and place of residence is unavailable at Waves 2 and 3. Although the rate of rural-urban movement (migration) is likely negligible among Canada's older population, the incidence of widowhood rises with age, which is known to have detrimental impact on mental health (Umberson, Wortman, and Kessler 1992).

To adjust for medical comorbidies, we considered three health indicators. Activities of daily living dependencies (ADLD) was measured on a 9-item scale. CSHA physicians assessed ADLD status during the formal clinical examinations. Respondents were asked about difficulties with doing outside work, paying bills (handling money or day-to-day purchases), going out alone, preparing their own meals, getting dressed or undressed, grooming, bathing or taking a shower, toileting, and finding their way in familiar areas. We measured ADLD as a sum of affirmative ratings (responses) to these items, which are time-variant.

Chronic illness was also diagnosed by CSHA physicians. This variable was measured using a 25-item inventory, representing various illnesses and conditions, such as history of seizures, stroke, neurological symptoms, diabetes, and cardiac symptoms. Chronic illness was measured as a sum of illnesses and conditions at Wave 1, and is a time-invariant variable because no comparable measurements were available at the follow-up waves.

The CSHA went through considerable effort to make an accurate diagnosis of dementia. The final diagnosis for each participant was determined at a "case conference," evaluating the findings from the screening interview, family and cognitive history, a preliminary clinical examination, a series of neuropsychological tests, a physician's mental assessment and neuralgic exams, a CT scan (for those with a preliminary diagnosis of vascular dementia or Alzheimer's disease with vascular components), and hematological and biochemical tests (for those suspected to have dementia or delirium). Each participant was then classified into one of the 7 categories: 1) no cognitive loss (normal), 2) cognitive loss but no dementia, 3) probable Alzheimer's disease, 4) possible Alzheimer's, 5) vascular dementia, 6) other specific dementia (e.g., Parkinson's, Pick's, Huntington's), and 7) unclassified dementia. For our study, we combined the last two categories.

Statistical Method

We estimated the effect of age on depression using generalized linear mixed model (GLMM) techniques, which account for the repeated observations on older persons collected in the CSHA. The GLMM model is an extension of generalized linear models (GLMs) for longitudinal data (Breslow and Clayton 1993; McCulloch and Searle 2001; Wolfinger and O'Connell 1993). Like the GLM, the GLMM model assumes that the response distribution (conditional on the random effects) belongs to the exponential family of distributions. However, unlike the GLM, which is constructed under the assumption of the independence of the data, the GLMM assumes that response data are correlated. In this sense, the GLMM extends the GLM by incorporating random effects in

the model, allowing for individual-specific (conditional) and population-average (marginal) inference (SAS Institute 2006). In other words, by applying the GLMM to correlated data, we are able to draw inference from both changes *within* individuals over time (aging effects) and differences *between* individuals across age groups (cohort effects) (Diggle et al. 2002).

In this study, the GLMMs were estimated using the GLMMIX procedure, an add-on product in SAS 9.1 (SAS Institute 2004). GLMMIX fits GLMMs using Taylor-series techniques (linearization methods) and a doubly iterative fitting algorithm. Parameters were estimated using the restricted pseudo-likelihood method (Wolfinger and O'Connell 1993). Wald-type test statistics and confidence intervals were constructed using linearization methods. In GLMMs, the log-likelihood of the data cannot always be derived, hence it is difficult, and sometimes infeasible, to compute likelihood-based tests and statistics. Thus, likelihood ratio tests are unavailable in the current version of the GLMMIX procedure (SAS Institute 2006). For goodness-of-fit statistics, we instead presented twice the negative of the residual log likelihood (in the final model) as well as the ratio of generalized chi-square statistics and corresponding degrees of freedom. The latter is a measure of the residual variability in the marginal distribution of the response data (an estimate of the scale parameter). The quality of the fit is generally acceptable if the ratio is not appreciably different from 1 (Littell et al. 2006).

FINDINGS

Total Depressive Symptoms

Table 2 presents GLMM models of age effects on changes in total depressive symptoms (TDS), including selected independent variables. The TDS variable is considered because it is more sensitive than standard cutoffs for MDD, which require the presence of either sadness or anhedonia for a positive diagnosis and do not differentiate between a MDD patient with 5 depressive symptoms and another with 12 symptoms. We included regressions for TDS because it can measure increases (or decreases) in the number of symptoms among MDD patients, such as an increase from 5 to 10 depressive symptoms between time points, which could represent important age-related changes that would otherwise be undetected.

<Table 2 About Here>

Model 1 in Table 2 examines the relationship between age and TDS, after adjusting for the effects of socio-demographic characteristics. The results indicate that age has a significant ($p < .01$) positive linear effect. That is, the total number of depressive symptoms tends to increase as seniors age. Model 2 introduces a quadratic term for age because the age-TDS relationship could be non-linear. The quadratic term has a non-significant effect, indicating that the age effect on TDS appears to be linear. Model 3 combines institution residence with Model 1 variables. This reduces the significance level ($p < .05$) for the age effect, but the positive linear effect persists. This model demonstrates that a high average number of depressive symptoms among institutionalized seniors does not inflate the general age effect. However, institution-dwellers do indeed experience a higher average number of depressive symptoms than community-dwellers. Model 4 introduces controls for ADLD, chronic illness, and dementia to the baseline variables. This model accounts for the age effect on TDS. The

presence of ADLD, chronic illness, or dementia are all significant predictors of TDS. Model 5 re-introduces institution residence, but this model does not improve upon Model 4. To examine whether age effects differ according to institutional-status and gender, Model 6 includes an interaction of age-institution and Model 7 includes an interaction of age-female. Both interactions are non-significant. Overall, Table 2 shows that age-related increases in ADLD and dementia account for corresponding increases of TDS. Table 2 also indicates that both random intercepts and residuals are significant in all 7 models, suggesting significant inter-person and intra-person differences in TDS, after controlling for the covariates.

Major Depressive Disorder

Table 3 presents GLMM models for age effects on the risk of MDD. This table presents 7 models using the same model specifications as in Table 2. The effect of age is non-significant in Model 1. The age effect becomes significant ($p < .01$) after a quadratic term of age is introduced. The quadratic term is significant ($p < .01$) in a positive direction. The risk of MDD follows a U-shaped trajectory for seniors. Unlike Table 2, the subsequent model specifications, which consider institution residence, health indicators, and interactions, cannot account for this age effect. That said, most dementias (except Alzheimer's disease), ADLD, and chronic conditions are significant predictors of MDD. Models 6 and 7 show that the selected interactions do not have a significant effect on the risk of MDD. In all models, the effects of random intercepts are not significant. This indicates that inter-person differences in the risk of MDD are non-significant after

controlling for covariates. Hence, the fixed effects of the explanatory variables account for most of the person-to-person variation in the risk of MDD.

<Table 3 About Here>

To illustrate the non-linear effect, we plotted the relationship between age and the risk of MDD in Figure 1. The two MDD trajectories presented in Figure 1 represent the effects of age with and without controls. The estimates for age and age square come from Model 5 in Table 3, but the comparable estimates for the curve without controls are from unreported analysis. Our focus is on the shape of the relationship not the rate (the probability) of MDD, which is re-scaled for the purpose of presentation. As Figure 1 illustrates, the risk of MDD decreases in early old-age and then begins to increase after a certain age threshold is crossed. Specifically, the relationship changes its direction after age 79 (80 in the model without controls, and 83 in the model with controls) for older Canadians.

<Figure 1 About Here>

Subsyndromal Depression

Table 4 presents 7 GLMM models for age effects on the risk of SSD with the same model specifications as used before. We measured the age-related risk of SSD because the literature indicates that SSD (i.e., depression below standard MDD thresholds) is an under-diagnosed and serious illness among seniors (e.g., Hybels, Blazer, and Pieper 2001; VanItallie 2005).

<Table 4 About Here>

Table 4 shows that, in all model specifications, age has a non-significant effect on the risk of SSD. In Model 7, there is some evidence suggesting that the effect of age differs between women and men (p = .08). For women, age appears to have a negative effect (b = -.021), but an opposite effect for men (b = .027). Moreover, as Model 7 indicates, ADLD, chronic illnesses, cognitive impairment, and some dementias are significant risk factors for SDD. Again, Alzheimer's disease has a non-significant effect on SSD. Like the models of MDD, the effects of random intercepts are small and non-significant in all 7 models.

DISCUSSION AND CONCLUSION

This research project examined whether aging influences the risk of major depression (MDD), the risk of subsyndromal depression (SSD), and changes in level of depressive symptoms (TDS) among Canadian seniors. Our empirical models adjusted for co-morbid depression (i.e., depression associated with the presence of chronic illness or ADL dependencies), dementias, and institution residence. These variables are well-established risk factors of late-life depression (Alexopoulos 2005; Blazer 2000). Hence, observed age effects on depression could correspond to these conditions, rather than being a pure aging effect. Although some studies account for dementia and some studies consider both community-level and institution-level populations, no previous studies consider all of these variables, using longitudinal data. In these respects, this study improves on pre-existing estimates of age effects on late-life depression.

We examined TDS (Table 2) because this measurement is sensitive to age-related changes in depressive symptoms. The literature indicates that even 1 or 2 symptoms can

be harmful for seniors (Chopra et al. 2005). This measurement allowed us to detect aging-related increases (or decreases) in depressive symptoms that clinical measures (diagnostic cut-offs) could miss. Our initial results indicated that there is an age-effect on TDS, as the level of depressive symptoms tends to increase with age. However, this increase represents co-morbid depression, for the introduction of controls for functional limitations, chronic illness, and dementia account for this age effect. This result supports the evidence that late-life depressive symptomatology is an indirect function of age, and is manifest through age-related physiological declines (see Yang 2007). This indicates that physiological variables mediate the age-depression relationship, meaning that age alone does not have a significant effect on changes in TDS.

While TDS increase with age, albeit as an indirect effect of physiological declines, there is a non-linear age effect on the risk of MDD. That is, the risk of MDD decreases in young-old age and then increases at around age 80. Unlike in our measurement of TDS, the subsequent model specifications, which consider institution residence, health and physiological indicators, and other variables, could not account for this age effect. This finding is consistent with previous research. For example, Alexopoulos (2005) demonstrates that the prevalence of MDD doubles for seniors after the threshold into oldest-old age is crossed. Hence, there appears to be an age limit on successful aging that is germane to major depression. This implies that MDD could have a vascular or another neurological-based factor that precipitates depression in advanced age.

According to prior studies, SSD is a potentially serious threat to well-being in geriatric populations (e.g., VanItallie 2005). Though we cannot discount that SSD is a

serious and under-diagnosed condition among seniors, there is generally a non-significant relationship between aging and the risk of SSD. That being said, our analysis did capture a relatively weak gender-specific age effect on SSD. The risk of SSD appears to decline with age for women, but rises with age for men. All in all, the risk of SSD appears to be more or less stable across age for Canadian seniors.

The aging effects this study details present two important implications. First, aging increases depressive symptoms through physical declines, which implies that a physical health problem could represent a double threat for well-being (successful aging) among seniors. An accumulation of depressive symptoms is a consequence of age-related biological decline, and increases in chronic diseases, disabilities, and dementias in particular. This confirms that co-morbid depression is a common circumstance of the advanced aging process. Hence, it may be insufficient to focus on physical ailments alone, for these could also involve comorbid depression. In addition, what some health care providers may diagnose to be minor ailments in terms of their physical consequences could be quite serious in terms of mental health outcomes. In other words, health care for seniors should consider these less obvious consequences of physical-based ailments, and be attentive to the mediating effect of these ailments on depressive symptomatology among seniors.

Second, the age pattern of the risk of MDD is somewhat unsettling, because it could involve an immutable change in health status. After considering important health status variables, among other risk factors, our results illustrate that after a decreasing effect, aging corresponds to an increase in the risk of MDD. This result appears to indicate a higher MDD risk among the oldest-old compared to other seniors. The

principal reasons for this elevated risk are unclear from the present research, but the literature suggests that brain deterioration (apart from dementia) is responsible for this outcome. For example, there is evidence of vascular depression (i.e., depression that is precipitated through cerebrovascular disease) or depression that is related to other brain degeneration (Alexopoulos 2005). This physiological-based pathology implies that normal treatments (e.g., anti-depressants or psychotherapeutic interventions) could be ineffective in certain cases of late-life depression, depending on the underlying cause. In these cases, treating the physical symptoms could represent the most effective approach to treating depression. In general, we need to consider the fact that biological wear-and-tear is a threat to mental well-being for seniors, and especially oldest-old seniors.

This study has a number of data limitations that could influence our conclusions. First, the panel data are unbalanced because of sample attritions, and this constricted our choice of model selection because of difficulties in getting alternate statistical models (e.g., growth curve models) to converge. Second, our data source collected information on some covariates (e.g., marital status, chronic conditions) at the baseline wave but not in the follow up waves. This means that the effects of changes in these independent variables cannot be captured with regards to their influence on depression. Third, the CSHA dataset does not include information on a number of well-established mental health indicators, such as income, coping, and social support. Future research should examine whether these resources influence the age-depression relationship among older persons.

ENDNOTES

1. Given advanced ages of the participants in the study sample, high rates of attritions across the 10-year interval were expected. To provide some descriptive information about those who were "lost" in the follow-ups, we estimated two logistic (fixed-effects) models, identifying the "risk" factors associated with attritions at Waves 2 and 3, respectively. The results are generally consistent with expectations. For example, age has nonlinear "U-shaped" effects in both models, suggesting that the rate of "attrition" (failure in the follow-ups) accelerates with age. The rate was lower for female participants (at Wave 3 only), but higher for widowed participants, institutional residents, and those who had cognitive impairments (dementia or Alzheimer's disease). The rate of attrition also increased with the number of chronic illnesses/conditions, but decreased with education (at Wave 3 only). The logistic regression results are available from the authors.

2. Missing data (item non-responses) can bias regression estimates and statistical inference. For our study sample, 728 (16.6%) observations have missing values for the response measures used in the analysis. To assess the impact of the missing data, we conducted a sensitivity analysis, using multiple imputation (MI) techniques (Rubin 1987). We re-estimated the GLMM models in Tables 2 – 4 using imputed (complete) datasets. A comparison of these findings indicates that there is minimal substantive difference in regression estimates and statistical inference. Hence, we decided to report the GLMM findings without imputations (the imputation results are available are from the authors).

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Table 1 Definitions and Descriptive Statistics of the Variables Used in the Analysis: Canadians (Age 65+) in the CSHA, 1990 (Wave 1)

		Community Sample		Institution Sample		Difference
Variable	Definition	M or %	S. D	M or %	S. D	(*p* value)
Depressive Symptoms [ab]	DSM-III-R scale (12 items, Cronbach's a = .74; range: 0 - 12).	1.20	1.77	1.76	2.18	<.001
Major Depressive Disorder [ab]	Dummy indicator (1 = yes, 0 = no)	2.6%	—	7.0%	—	<.001
Subsyndromal Depression [ab]	Dummy indicator (1 = yes, 0 = no)	3.5%	—	4.1%	—	0.629
Age[a]	Age in years (range: 65 - 107)	80.12	6.87	81.37	7.84	<.001
Female[c]	Dummy indicator (1 = female, 0 = male)	59%	—	70%	—	<.001
Marital status[c]						
Single	Dummy indicator (1 = yes, 0 = no)	8.6%	—	19.5%	—	<.001
Separated/divorced	Dummy indicator (1 = yes, 0 = no)	3.7%	—	6.0%	—	0.014
Widowed	Dummy indicator (1 = yes, 0 = no)	47.5%	—	59.2%	—	<.001
Married/Cohabiting	Reference group	40.2%	—	15.4%	—	<.001
Rural residence[c]	Dummy indicator (1 = rural, 0 = urban)	14.6%	—	6.6%	—	<.001
Visible minority[c]	Dummy indicator (1 = yes, 0 = no)	1.9%	—	0.8%	—	0.021
Education[c]	Formal education in years (range: 0 - 21)	8.57	4.14	8.79	3.55	0.170
Activities of daily living dependencies[ab] (ADLD)	Number of limitations of activities of daily living (range: 0 - 9)	0.58	1.39	2.22	2.51	<.001
Chronic illness [bc]	Number of chronic illnesses/conditions (range: 0 - 25)	3.46	2.25	3.98	2.32	<.001
Dementia diagnosis[ab]						
Cognitive loss but no dementia	Dummy indicator (1 = yes, 0 = no)	29.9%	—	35.8%	—	0.004
Probable Alzheimers	Dummy indicator (1 = yes, 0 = no)	9.5%	—	17.4%	—	<.001
Possible Alzheimers	Dummy indicator (1 = yes, 0 = no)	6.1%	—	12.4%	—	<.001
Vascular dementia	Dummy indicator (1 = yes, 0 = no)	4.4%	—	8.6%	—	<.001
Other/unclassified dementia	Dummy indicator (1 = yes, 0 = no)	2.9%	—	7.9%	—	<.001
Normal	Reference group	47.2%	—	17.8%	—	<.001
N (person-period observations)		1,553		869		

Note: Significance tests were obtained from bivariate analyses using data from wave 1 of the CSHA (*N* = 2,422).

[a] Time-dependent measure.

[b] See text for details.

[c] Time-invariant measure.

Table 2 Generalized Linear Mixed Models of Total Depressive Symptoms on Age and Selected Variables: Canadians (Age 65+) in the CSHA, 1990 - 2001 (*N* = 3,652)

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Age[a]	0.013 ***	-0.096	0.010 **	-0.001	0.000	0.003	-0.001
Age square[a]	—	0.001	—	—	—	—	—
Female[b] (1 = yes)	0.117	0.113	0.112	0.159 **	0.160 **	0.163 **	0.101
Marital status[b]							
Single	-0.180	-0.186	-0.357 ***	-0.210 *	-0.265 **	-0.278 **	-0.266 **
Separated/divorced	-0.022	-0.024	-0.161	-0.168	-0.211	-0.222	-0.210
Widowed	0.102	0.104	-0.002	0.038	0.005	0.001	0.005
Married/Cohabiting[c]							
Rural residence[b] (1 = yes)	-0.366 ***	-0.369 ***	-0.306 ***	-0.321 ***	-0.306 ***	-0.306 ***	-0.307 ***
Visible minority[b] (1 = yes)	0.417	0.413	0.498 *	0.435	0.461 *	0.470 *	0.461 *
Education[b]	-0.015 *	-0.015	-0.014	-0.007	-0.007	-0.007	-0.007
Living in institution (1 = yes)	—	—	0.495 ***	—	0.160 **	0.743	0.160 **
ADL dependencies[a]	—	—	—	0.075 ***	0.065 ***	0.065 ***	0.065 ***
Chronic illness[a]	—	—	—	0.183 ***	0.181 ***	0.181 ***	0.181 ***
Dementia diagnosis[a]							
Cognitive loss but no dementia	—	—	—	0.500 ***	0.482 ***	0.477 ***	0.482 ***
Probable Alzheimers	—	—	—	0.373 ***	0.348 ***	0.349 ***	0.348 ***
Possible Alzheimers	—	—	—	0.288 **	0.260 **	0.259 **	0.260 **
Vascular Dementia	—	—	—	0.657 ***	0.636 ***	0.631 ***	0.636 ***
Other/unclassified dementia	—	—	—	1.020 ***	0.983 ***	0.978 ***	0.983 ***
Normal[c]							
Age × institution	—	—	—	—	—	-0.007	—
Age × female	—	—	—	—	—	—	0.001
Intercept	0.469	4.880	0.540	0.398	0.367	0.142	0.404
Random effects							
Intercepts	1.443 ***	1.446 ***	1.381 ***	1.195 ***	1.185 ***	1.187 ***	1.186 ***
Residuals	2.270 ***	2.267 ***	2.273 ***	2.180 ***	2.184 ***	2.183 ***	2.184 ***
- 2 Restricted Log Likelihood	14965	14977	14921	14674	14673	14680	14681
Generalized Chi-square / DF	2.270	2.270	2.270	2.180	2.180	2.18	2.18

[a] Time-dependent measure.

[b] Time-invariant measure.

[c] Reference category.

*p < .10, **p < .05, *** p < .01 (two-tailed test)

Table 3 Generalized Linear Mixed Models of Major Depressive Disorder on Age and Selected Variables: Canadians (Age 65+) in the CSHA, 1990 - 2001 (*N* = 3,652)

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Age[a]	-0.002	-0.462 ***	-0.400 **	-0.458 **	-0.428 **	-0.651 **	-0.087
Age square[a]	—	0.003 ***	0.002 **	0.003 **	0.003 **	0.004 **	0.000
Female[b] (1 = yes)	0.096	0.072	0.067	0.130	0.149	0.151	16.606
Marital status[b]							
Single	-0.413	-0.437	-0.793 **	-0.461	-0.636 *	-0.649 *	-0.629 *
Separated/divorced	0.251	0.247	-0.047	0.153	-0.010	-0.015	-0.023
Widowed	0.088	0.113	-0.120	0.050	-0.085	-0.097	-0.072
Married/Cohabiting[c]							
Rural residence[b] (1 = yes)	-0.435	-0.457	-0.352	-0.421	-0.385	-0.393	-0.379
Visible minority[b] (1 = yes)	0.122	0.090	0.283	0.074	0.168	0.155	0.178
Education[b]	-0.033	-0.032	-0.030	-0.020	-0.021	-0.022	-0.021
Living in institutions (1 = yes)	—	—	0.882 ***	—	0.491 **	-14.870	0.491 **
ADL dependencies[a]	—	—	—	0.097 **	0.068	0.068	0.066
Chronic illness[a]	—	—	—	0.139 ***	0.132 ***	0.133 ***	0.132 ***
Dementia diagnosis[a]							
Cognitive loss but no dementia	—	—	—	0.945 ***	0.873 ***	0.870 ***	0.876 ***
Probable Alzheimers	—	—	—	0.312	0.230	0.230	0.235
Possible Alzheimers	—	—	—	0.143	0.027	0.028	0.038
Vascular Dementia	—	—	—	1.099 ***	1.023 ***	1.013 ***	1.022 ***
Other/unclassified dementia	—	—	—	1.452 ***	1.327 ***	1.329 ***	1.336 ***
Normal[c]							
Age × institution	—	—	—	—	—	0.385	—
Age^2 × institution	—	—	—	—	—	-0.002	—
Age × female	—	—	—	—		—	-0.424
Age^2 × female	—	—	—	—		—	0.003
Intercept	-2.834 ***	15.838 ***	13.214 *	14.561 *	13.276 *	22.162 *	0.092
Random effects							
Intercepts	0.578	0.540	0.472	0.528	0.513	0.528	0.512
– 2 Residual Log Pseudo Likelihood	21275	21415	21836	22148	22274	22308	22297
Generalized Chi-square / DF	0.620	0.640	0.680	0.610	0.620	0.620	0.620

[a] Time-dependent measure.

[b] Time-invariant measure.

[c] Reference category.

*p < .10, **p < .05, *** p < .01 (two-tailed test)

Table 4 Generalized Linear Mixed Models of Subsyndromal Depression on Age and Selected Variables: Canadians (Age 65+) in the CSHA, 1990 - 2001 (N = 3,512)

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Age[a]	0.008	0.217	0.007	-0.002	-0.003	0.004	0.027
Age square[a]	—	-0.001	—	—	—		
Female[b] (1 = yes)	0.035	0.041	0.033	0.032	0.023	0.028	3.933
Marital status[b]							
Single	-0.660 *	-0.652 *	-0.726 *	-0.630 *	-0.539	-0.565	-0.533
Separated/divorced	-0.615	-0.614	-0.661	-0.718	-0.640	-0.661	-0.642
Widowed	-0.064	-0.069	-0.103	-0.123	-0.059	-0.069	-0.057
Married/Cohabiting[c]							
Rural residence[b] (1 = yes)	-0.028	-0.023	-0.010	0.012	-0.010	-0.006	0.006
Visible minority[b] (1 = yes)	1.042 **	1.049 **	1.066 **	1.082 **	1.049 **	1.065 **	1.052 **
Education[b]	-0.020	-0.020	-0.019	-0.013	-0.013	-0.013	-0.011
Living in institution (1 = yes)	—	—	0.176	—	-0.281	1.251	-0.279
ADL dependencies[a]	—	—	—	0.111 **	0.129 ***	0.127 ***	0.131 ***
Chronic illness[a]	—	—	—	0.148 ***	0.152 ***	0.152 ***	0.154 ***
Dementia diagnosis[a]							
Cognitive loss but no dementia	—	—	—	0.811 ***	0.846 ***	0.833 ***	0.852 ***
Probable Alzheimers	—	—	—	0.174	0.212	0.212	0.228
Possible Alzheimers	—	—	—	0.355	0.415	0.413	0.413
Vascular Dementia	—	—	—	0.831 **	0.863 **	0.850 **	0.886 **
Other/unclassified dementia	—	—	—	0.111	0.167	0.152	0.169
Normal[c]							
Age × institution	—	—	—	—	—	-0.018	—
Age × female	—	—	—	—		—	-0.048 *
Intercept	-3.644 ***	-12.128	-3.614 ***	-4.052 ***	-3.968 ***	-4.574 ***	-6.507 ***
Random effects							
Intercepts	0.270	0.269	0.272	0.244	0.255	0.264	0.239
– 2 Residual Log Pseudo Likelihood	21107	21134	21112	21709	21688	21676	21782
Generalized Chi-square / DF	0.790	0.800	0.790	0.780	0.770	0.760	0.780

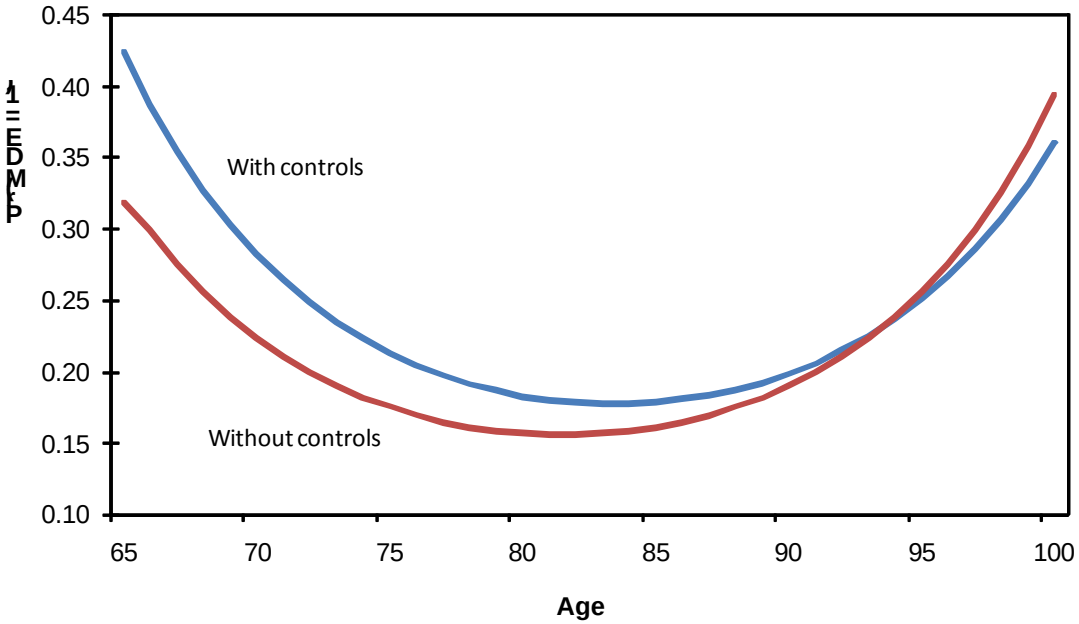
[a] Time-dependent measure.

[b] Time-invariant measure.

[c] Reference category.

*p < .10, **p < .05, *** p < .01 (two-tailed test)

Figure 1. Age Pattern of Major Depressive Disorder (CSHA, 1990 - 2001)



Note: Pr(MDE = 1) is re-scaled.

Source: Canadian Study of Health and Aging, 1990 - 2001.