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Depressive symptoms are associated with allostatic load among community-dwelling older adults

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HIGHLIGHTS

- We analyzed associations of allostatic load with late-life depressive symptoms.
- Greater allostatic load was associated with more severe depressive symptoms.
- We observed associations with overall, affective, and somatic depressive symptoms.
- The strength of these associations appears to be of clinical significance.
- Future research should explore factors driving these associations.

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ABSTRACT

The allostatic load model has been used to quantify the physiological costs of the body's response to repeated stressful demands and may provide a useful, integrative perspective on the various correlates of late-life depressive symptoms. We interviewed 125 Rochester, NY adults, ranging in age from 67 to 94 years. We employed an allostatic load score as a measure of multisystem dysfunction in hypothalamic–pituitary–adrenal axis function, immune function, anabolic activity, and cardiovascular activity. Overall, affective, and somatic depressive symptom scores were computed using the 20-item Center for Epidemiologic Studies Depression Scale. Multiple linear regression models were used to estimate associations between allostatic load scores and affective, somatic, and overall depressive symptoms. Among our sample of mean age 76.1 years, the one-week prevalence of clinically significant depressive symptoms was 12.8%. In models adjusting for demographic, socioeconomic, and health-related factors, higher allostatic load scores were associated with elevated scores for overall, affective, and somatic depressive symptoms: beta = 1.21 (95% CI = 0.38, 2.05); beta = 0.14 (95% CI = 0.040, 0.24); beta = 0.60 (95% CI = 0.23, 0.97), respectively. Our results suggest that allostatic load measure is associated with late-life depressive symptoms. This association appears to be of clinical significance, as the magnitude of the effect size was comparable (but opposite in direction) to that of antidepressant use. Future research should examine the inter-relationships of allostatic load, psychological stress, and late-life depressive symptoms.

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1. Introduction

Depressive symptoms are common among older adults in the U.S., with estimates of the one-week prevalence of clinically significant depressive symptoms ranging from 8% to 16% [1–3]. As the U.S. population continues to age, the public health burden of late-life depressive symptoms can be expected to increase. The U.S. Census Bureau projects that by the year 2050 adults over the age of 65 will comprise over 20% of the U.S. population as compared to about 13% currently [4,5]. Thus, the absolute number of older adults affected by depressive symptoms will increase substantially, and understanding the psychosocial and physiological correlates of depressive symptoms among community-dwelling older adults will become increasingly crucial.

The established psychosocial and physiological correlates of late-life depressive symptoms are diverse [6]. Individuals affected by physical conditions spanning multiple systems of the body, such as cardiovascular disease, diabetes, sleep disturbance, cognitive dysfunction

Abbreviations: HPA, hypothalamic–pituitary–adrenal; SNS, sympathetic nervous system; BMI, body mass index; MIEIHS, Mindfulness to Improve Elders' Immune and Health Status; MMSE, Mini-Mental State Examination; DCS, diurnal cortisol slope; IL-6, interleukin-6; IGF-1, insulin-like growth factor 1; CES-D, Center for Epidemiologic Studies Depression Scale; CHAMPS, Community Healthy Activities Model Program for Seniors.

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(e.g. Parkinson's disease, dementias), and hyperactivity of inflammatory pathways are more likely to experience depressive symptoms [1,2,7,8]. In addition to physical conditions, psychosocial factors linked with late-life depressive symptoms include trait neuroticism, multiple dimensions of poor socioeconomic position, poor social support, stressful life events, and elevated levels of perceived stress [2,6,7,9].

The 'allostatic load model' may provide a useful integrative perspective on these various psychological and physiological correlates of late-life depressive symptoms. Sterling and Eyer developed the concept of 'allostasis' to describe an organism's ability to adjust its physiological functioning in response to the environment [10]. McEwen and Stellar extended this conceptual model to explain how psychosocial phenomena may result in lasting physiological changes in the body [11,12]. This model has been used to quantify the physiological costs of the body's response to either repeated stressful demands or inadequate responses to these demands [12,13]. It posits that repeated or inadequate physiological adaption to social and environmental stress over time, as mediated through the dysregulation of glucocorticoids such as cortisol via the hypothalamic–pituitary–adrenal (HPA) axis and catecholamines via the sympathetic nervous system (SNS), can result in dysfunction of the body's cardiovascular, immune, and metabolic systems [13,14]. Independent of McEwen, Björntorp and Rosmond suggested how a cluster of metabolic and cardiovascular symptoms might be associated with HPA axis (named Metabolic Syndrome X) and stress [15,16]. This dysregulation of cortisol and of downstream physiologic systems may promote depressive symptoms [17]. In summary, repeated adaptation to stress is thought to result in dysfunction of the HPA axis, and the resulting dysregulation of cortisol and of downstream physiologic systems may be associated with depressive symptoms.

Although this model suggests that the link between allostatic load and depressive symptoms is plausible, and despite the fact that many of the standard physiological components of an allostatic load summary measures have been studied in relation to depressive symptoms [6], only a few analyses have examined the association. Analyses of 972 Taiwanese older adults (mean age 67.7 years at baseline) demonstrated significant associations between elevated allostatic load scores (greater multisystem dysfunction) and greater overall depressive symptoms at baseline and three years later [18,19]. Another study also provided support for a relationship between allostatic load and depressive symptoms (at baseline and three years later) among 58 adults of mean age 67.6 years [20]. In a more recent analysis of a large, nationally-representative sample, a physiological dysfunction summary measure, guided by the allostatic load model, was associated with multiple dimensions of depressive symptoms [21].

In this analysis, we examine the association between a measure of allostatic load and overall depressive symptom severity using a sample of community-dwelling older adults. In addition to examining overall (i.e. global) symptoms, we also considered affective and somatic depressive symptoms. These two dimensions were chosen because studies increasingly suggest that physiological factors may be associated with specific dimensions of depressive symptoms [22–25]. Two such symptom clusters are "affective" (e.g. guilt, dysphoria) and "somatic" (e.g. sleep irregularities, lack of energy, changes in appetite) symptoms. The literature on depressive symptoms rarely delineates the affective and somatic dimensions of depressive symptoms, yet distinguishing these dimensions may be important because they may be associated with unique risk factors and independent neurobiological pathologies in the context of late-life depression [26,27].

2. Material and methods

2.1. Study population

We followed up and collected new data on participants who previously participated in the Mindfulness to Improve Elders' Immune and Health Status (MIEIHS) study, a randomized controlled trial, completed in 2009, conducted to study Mindfulness Based Stress Reduction and its effects on immune function [28]. Study participants were community-dwelling older adults of at least 65 years of age that were recruited by newspaper advertisements and flyers. Eligibility requirements stipulated that subjects be 65 years of age or older and English speaking. Participants with prescription antidepressant or anxiolytic medication must have had a stable regimen for eight weeks prior to enrollment (except for persons taking sedative-hypnotic sleep medication, low level psychotropic medication for pain management, or beta-blockers for heart conditions). Major, uncorrected sensory impairments or cognitive impairment (defined as a score of 24 or lower on the Mini-Mental State Examination (MMSE)) were considered exclusionary, based on previous recommendations [29]. Exclusions were also made for individuals with major depression with psychotic features, psychosis, lifetime history of schizophrenia, bipolar disorder, organic brain syndrome, mental retardation, or a history of substance abuse within the previous year. Psychiatric-based exclusion criteria were assessed through the Structured Clinical Interview for DSM-IV (SCID) and clinical interview. The MIEIHS study consisted of a baseline assessment with additional assessments at 8 weeks, 11 weeks, and 32 weeks. Various physiological and psychological measures were taken across these four periods.

For this 2010 follow-up study, the 204 subjects that completed all phases of the MIEIHS were re-contacted. The MMSE was re-administered and subjects with a total score of 24 or lower were excluded from follow-up participation. Aside from this, all eligible subjects that completed the MIEIHS study (including both arms of the clinical trial) were offered participation in the follow-up study. Successful telephone contact was made for 165 (81%) subjects and ultimately, interviews were conducted on and full data were available for 125 (61%) subjects. Informed consent was obtained from each participant and the University of Rochester's Research Subjects Review Board approved the study protocol.

2.2. Biomarker measurement

Our allostatic load summary measure was constructed using seven component measures, encompassing neuroendocrine function, cytokine status, metabolism, and cardiovascular function. These include diurnal cortisol slope (DCS), interleukin-6 (IL-6), insulin-like growth factor 1 (IGF-1), waist-to-hip ratio, resting heart rate, systolic blood pressure, and diastolic blood pressure. The DCS, waist-to-hip ratio, resting heart rate, and blood pressure measures were collected during the 2010 follow-up study, while IL-6 and IGF-1 were collected during the original MIEIHS study.

Various measures of HPA axis function status have been proposed, including measures of the cortisol awakening response and the DCS. We selected the DCS because it is thought to reflect chronic stress conditions and has been found to be a particularly sensitive marker of psychiatric disturbances, particularly those involving stress (e.g. post-traumatic stress disorder, depression, generalized anxiety disorder), as well as physical health measures (e.g. obesity, cardiovascular disease) [30]. With the DCS, it is thought that conditions of chronic stress are associated with flatter diurnal pattern, and thus averaged coefficients of smaller magnitude are thought to be indicative of greater stress and poorer HPA axis functionality [30]. We characterized the diurnal cortisol secretion pattern with six salivary cortisol samples obtained over two consecutive days. On each these days, samples were collected: (1) immediately after the subject awakened, (2) 2.5 h after awakening, and (3) and at bedtime. Subjects were asked to passively drool into labeled, polypropylene tubes at each collection period and document the time of collection. After all six samples were collected, sealed tubes were mailed in padded envelopes to study staff. Upon arrival the sealed tubes were stored in a −80 °C freezer. Samples were later thawed and assayed for cortisol using a high sensitivity enzyme-linked immunoassay kit (Salimetrics LLC, PA). The calculation of a DCS involved using linear regression to estimate the best-fitting line through the cortisol values

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