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Increase in blood pressure precedes clinical depression in elderly primary care patients

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Previous studies on the relationship between blood pressure (BP) and depression have produced inconsistent results (Paterniti *et al*., 2000; Lenoir *et al*., 2008; Stewart and Hirani, 2010; Siennicki-Lantz *et al*., 2012). There have been no studies examining changes in BP prior to depression over a relatively long period. Participants in this study were primary care patients aged 60 years or older who screened negative for depression using the Center for Epidemiologic Studies Depression Scale (CES-D < 16) and had no prior depression diagnosis. Participants were screened from 1991 to 1993 during regularly scheduled primary care appointments at an urban public health system (Callahan *et al*., 1994). Participants were then followed through December 31, 2010.

The study end point was defined as the time to a physician diagnosis of clinical depression based on International Classification of Disease codes. For patients not diagnosed with depression, date of the last encounter with the health system was their study endpoints. Clinical information was extracted from the Regenstrief Medical Record System including all height, weight, systolic BP, diastolic BP from outpatient visits, and 15 diagnoses (hypertension, diabetes, atherosclerotic vascular disease, coronary artery disease, peripheral artery disease, congestive heart failure, cerebrovascular disease, cancer, anemia, chronic obstructive pulmonary disease, arthritis, liver disease, renal disease, thyroid disease, hyperlipidemia, and dementia). Age, gender, race, years of education, and history of smoking were collected at enrollment.

Repeated systolic and diastolic BP from enrollment to study endpoints were used as dependent variables in mixed effect models with random intercepts and random slopes while

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adjusting for time dependent covariates of patients' medical conditions. The BP measures were aligned to each patient's study endpoint, indexing a BP measure by the number of years this measure was taken before the patient's endpoint.

A total of 2581 participants were included with a median of 21 BP measures per patient. The mean times from enrollment to endpoints for the depression and no-depression groups were 7.2 years (*SD* = 4.6) and 7.9 years (*SD* = 5.8), respectively.

Participants in the incident depression group had lower systolic BP at enrollment than those without depression ($p = 0.0057$) and were younger, more likely to be a woman, had a higher mean body mass index, and a higher rate of arthritis at enrollment ($p < 0.0001$). No other differences in rates of medical conditions were evident and the total numbers of comorbid conditions did not differ between the two groups at enrollment ($p = 0.1699$).

A significant interaction between depression group and time was found for systolic BP indicating that systolic BP of those with incident depression increased significantly more than in those without depression ($p < 0.0001$) while adjusting for age, gender, race, body mass index, smoking, and medical conditions. Figure 1 illustrates the model by plotting predicted mean systolic BP over time for participants with incident depression and those without depression. An increasing trend is seen in systolic BP over time for the incident depression group, whereas the no-depression group showed a slight decrease in systolic BP toward the study endpoint. No significant main effects or interactions were found when diastolic BP was used as the dependent variable.

In this depression-free cohort of older primary care patients followed for 18 years, we found that increases in systolic BP preceded the diagnosis of depression. The increase in systolic BP in participants with incident depression cannot be attributed to differences in medical conditions including cardiovascular diseases as these conditions were adjusted for in the models. The mechanism underlying the increase in systolic BP in participants with incident depression is not clear. Although increasing BP may act as an independent risk factor for late-life depression, it is also likely that both BP and depression were affected by other underlying factors such as stress, inflammation, or incipient vascular diseases.

To our knowledge, this is the first study examining longitudinal trends in BP in elderly participants with incident late-life depression. The inclusion of longitudinal BP measures over a long period made it possible to examine BP trends in participants prior to depression diagnosis. Our study also has the advantage of excluding participants with baseline depression so that BP changes prior to new depression diagnosis can be examined. Our results suggest that increases in BP may be an important indicator for incipient late-life depression as well as for cardiovascular diseases in the elderly population.

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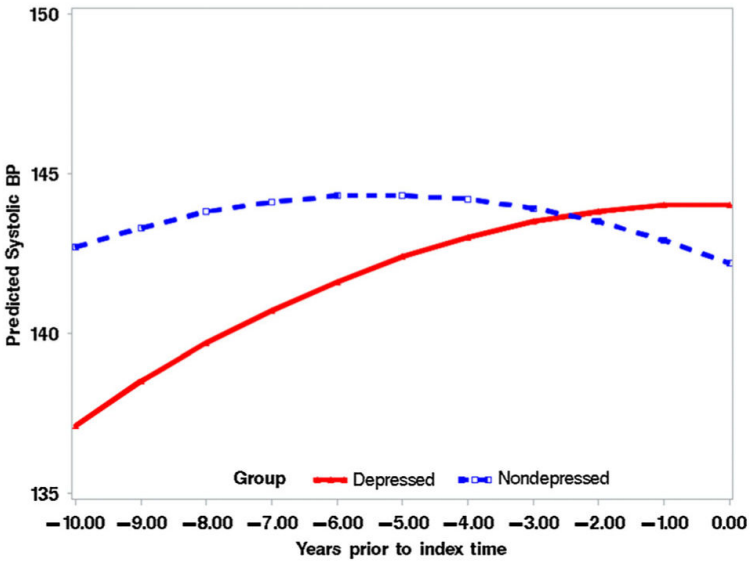

Figure 1.

Predicted mean systolic blood pressure over time for participants with incident depression and those without depression based on the final mixed effect model.