Response to Letter Regarding Article, “Psychosocial Distress and Stroke Risk in Older Adults”

We appreciate the letter by Drs Egido and Garcia commenting on our recent report in *Stroke,* which identified increasing levels of psychosocial distress as a significant predictor of incident stroke, particularly hemorrhagic stroke and stroke mortality in a US population-based cohort of black and white community residents aged 65 years. Egido et al recently reported similar results in a case-control study of 150 younger patients with stroke (mean age, 53.8; range, 18–65 years) from a hospital-based stroke unit in Madrid, Spain, and 300 age-matched neighborhood controls. In their study, patients with stroke were significantly more likely than controls to report moderate and high scores on the Holmes and Rahe Social Readjustment Rating Scale, a measure of perceived stressfulness of recently experienced life events. This association was maintained after adjusting for demographics, health behaviors, medical history and chronic conditions, and sleep quality. The fact that our 2 studies, using different study methodologies, with one focused on a younger patient population and the other focused on a community-dwelling cohort of older adults, find similar associations lends credence to the conclusion that psychosocial distress is an important risk factor for stroke outcomes.

As Egido and Garcia point out, in our analyses of stroke mortality, we adjusted for history of stroke, a highly significant covariate in the model (hazard ratio [HR], 3.24; 95% confidence interval [CI], 2.23–4.72) in which each 1-SD higher distress score predicted 29% excess risk of stroke deaths (HR, 1.29; 95% CI, 1.10–1.52). We did not report in the article an additional analysis of stroke mortality that excluded all persons with a history of self-reported stroke before the second cycle of data collection in our cohort. These analyses, which included 3,424 participants and 98 stroke deaths, were very similar to the reported findings; indeed, the HR was higher in this risk factor–adjusted model (HR, 1.37; 95% CI, 1.13–1.66) than in the model that adjusted for stroke history. Even among older adults without a history of stroke, distress strongly predicted risk of dying from stroke after taking into account known stroke risk factors.

Egido and Garcia question our point about the critical biological pathways not being ischemic in origin; in fact, we observed a significant association between distress and incident nonhemorrhagic ischemic stroke in the minimally adjusted models (HR, 1.12; 95% CI, 1.01–1.24). When known stroke risk factors were added to the model, the HR for distress was reduced and became nonsignificant (HR, 1.02; 95% CI, 0.91–1.15), implying that the link between greater distress and ischemic stroke is largely explained through these important risk factors. In contrast, the relationship between distress and incident hemorrhagic stroke effectively was not influenced by these same stroke risk factors. The point we wish to emphasize is that this pattern of findings suggests that pathways linking distress with nonischemic strokes are important to identify and are seemingly unrelated to known stroke risk factors. But this does not negate the fact that distress also was related to ischemic strokes in our analyses. It may be that the association of stress with ischemic stroke outcomes is stronger in younger populations. Stroke by and large is a disease of the elderly, yet it obviously occurs in many individuals aged <65 years. Thus, it is a task of future research to further investigate nontraditional factors, such as psychosocial stress or distress that may contribute to risk for stroke in middle-aged and younger populations and to clarify the pathways through which stress may lead to hemorrhagic stroke.

Disclosures

None.

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