Letter by Egido and Garcia Regarding Article, “Psychosocial Distress and Stroke Risk in Older Adults”

To the Editor:

We read with great interest the article by Henderson et al., in which authors report the effects of psychosocial distress measured by 4 psychosocial indicators on stroke mortality and incidence in a population-based cohort of older black and white adults. Sample size considered was 4120 and 2649 for the mortality and incident stroke analysis, with a follow-up of 6.7 years (SD, 3.5) and 6.0 years (SD, 3.0), respectively.

In this study, psychosocial assessment was done at just one point in time; as the authors discuss, it was not possible to determine whether changes in distress levels occurred or if such changes could influence stroke risk. Data acquired in a case-control study, previously reported by our group, may be of help and complementary in this question, as the scales selected for measurement of psychophysical stress provided information about the psychosocial problems in the previous 4 weeks, the profile of general health status with reference to the previous month, and the stressful experiences lived over the previous year of the first incident stroke. The presence of type A behavior was also assessed, and the results obtained are coherent in terms of relationship of stroke with stress between both studies. As compared with a case-control design, cohort studies do not have a possible recall bias. Noteworthy, history of stroke was also significantly higher at baseline in the Chicago Health and Aging Project population more-stressed quartiles. To investigate this issue further, it would be of interest to report whether deaths and strokes occurred close to the second cycle of interviews of Chicago Health and Aging Project data collection or later in time.

However, we disagree with the authors about the pathways by which distress may increase stroke risk when they say interestingly, “our analyses of incident stroke events show that the critical biological pathways may not be ischemic in origin.” The vascular risk factors load in that old population could make the sample size insufficient to maintain the statistical significance when analyzing ischemic stroke. That is the main reason we selected a population from 18 to 65 years. Our sample cases (90% were of ischemic origin) compared with controls and following adjustment for confounding variables had a 4-fold stroke risk when living under stressful conditions in the previous year and doubled the risk if presented high levels of competitiveness and aggression. This relationship was not also modified by sex.

Finally, although the design of both studies precludes establishing a causal relationship, scientific evidence is added as results are plausible, consistent with previous work, and there is a biological gradient and temporality. Reversibility remains to be clarified, so further interventional studies are granted to understand stress risk pathways for cerebrovascular disease and lead to future preventive strategies to reduce stroke burden.

Disclosures

None.

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