Depressive Symptoms, Risk of Stroke and the Stress Response

To the Editor:

In their analysis of the association between depressive symptoms and an increased risk of stroke, Salaycik et al.1 conclude that depressive symptoms are independent risk factors for incident stroke/TIA in individuals below the age of 65 years. They also mention a bibliographic search about possible causes for this relationship: genetic and biological markers that have been implicated include increased levels of fibrinogen, platelet activation, and catecholamines, or inflammatory markers such as C-reactive protein. The association between depression and the risk of stroke, they continue, may also be attributed to poor adherence to prescribed medical regimens, including medication, diet, and exercise.

Depression is a silent killer, but how and why? Can a deregulated stress response be one of the culprits? Available evidence suggests that we can include depression among other chronic diseases with inflammatory components, as we do now with obesity or diabetes.2 Moreover, depression consistently shows a deregulated stress response.3 Intensity of immune-inflammatory responses to stress can be of different magnitude among different patients. (For example, early attacks to the inflammatory responses to stress can be of different magnitude among different patients. (For example, early attacks to the inflammatory side. These patients will have few complications derived directly from their mental stress. Could it be the case for people above 65 years old? Further research is required to give an affirmative answer in this point. For the others, a proatherosclerotic deregulated response will result irrespectively of their predominant immuno-suppression or their main response toward the inflammatory side. Finally, some patients may conserve the equilibrium between both arms of the stress response, but with an abnormally high set-point for stopping the response, a proatherosclerotic process also. Here, complications such as cardio- or cerebrovascular disease can be anticipated.2,5

Pharmacological intervention to improve this pathological stress response could reduce patients’ comorbidities with profound positive effects on the quality of their lives. However, how do we know which patients with depression will benefit with those therapies? Standardized psychosocial stress significantly increases salivary α-amylase via adrenergic mechanisms6; consequently, if a deregulated stress response is the integrator of the many markers of vascular vulnerability in patients with depression, those depressed patients in high risk of experiencing a stroke incident may present elevations in the salivary amylase. Hence, I propose that further studies relating depression and stroke will be improved if they include measurements of salivary α-amylase.

Disclosures

None.

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*Stroke*. 2007;38:e34; originally published online April 19, 2007;
doi: 10.1161/STRKEAHA.106.481655
*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/38/6/e34

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