Depression and cognitive decline

Depressive symptoms and cognitive decline—disentangling the effect of affect

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Time to take depression seriously?

Is depression a risk factor for cognitive decline? Dementia, a potential consequence of cognitive decline, is a devastating disorder. If depression is a risk factor, it is important because it is common. Therefore, even a weak risk-outcome association might represent a substantial population level impact and “account for” a large number of dementia cases. It is not surprising that a growing body of observational research has sought to address this question, including the analysis by Wilson et al in this issue (pp 126–9). However, unfortunately we are still a long way from establishing causation and measurements of exposure leave a lot to be desired.

Several prospective studies have shown that depressive symptoms are associated with an increased risk of developing dementia. It is possible that depression may actually initiate or accelerate neurodegenerative processes. This is obviously of particular interest given the implied possibility of preventing cognitive decline and dementia. However, there are other potential explanations. Depression may simply be a reaction to a perceived deterioration in cognitive function. Alternatively, cognitive function may be impaired during a depressive episode because of reduced attention and motivation, so that someone may present with apparent clinical dementia at a relatively early stage of neurodegeneration. These two explanations are addressed in the analysis carried out by Wilson et al and are not supported by their findings because increased baseline depressive symptoms predicted cognitive decline independently of baseline cognitive function. A third explanation is that depression is a prodromal symptom of dementia rather than a risk factor. This hypothesis is less easy to test. It would predict a greater level of neurodegenerative pathology associated with depressive symptoms in people without clinical dementia in life. However, population-based pathological data are hard to come by. Wilson et al cite a report in press at the time of submission which may shed light on this issue but further research is likely to be required.

So where to go from here? A disappointing feature of most research in this area has been the simplistic approach to depression as an exposure. Some studies focus on categories such as major depression, which poorly characterise late life affective disorder. Most, including that by Wilson et al, do not investigate depression but depressive symptoms. But can a person’s mood state be adequately reflected by summing up a short list of symptoms? What about the perversiveness of individual symptoms themselves, their nature (such as the type of sleep disturbance) or underlying clustering (for example, motivation and affect)? Levels of affective disturbance fluctuate throughout the life course and are likely to have complex, interdependent relations with other aspects of health, such as cognitive function and somatic states (for example, vascular disease). Second analyses of high quality longitudinal datasets have undoubtedly made substantial contributions to this research field, but are invariably limited by measurements that have to cover a large number of objectives. Dysphoric symptoms predict a wide variety of adverse outcomes but are rarely measured in any detail. Perhaps it is time to take depression seriously.

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