Commentary

Depression deconstruction lessons from psychosomatic research

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In the review by Poole and colleagues in this issue of Journal of Psychosomatic Research, it is suggested that depression that develops in the aftermath of an acute coronary syndrome (ACS), such as a myocardial infarction (MI), may have a different etiology than depression as is observed in the general population. Interestingly, the authors make a case that post-MI depression results from a somatic reaction to a physiological process or an adaptive response to a somatic stressor, which is not necessarily a physical condition. This is in line with the authors' concept of an inflammation-based somatic subtype of depression as is observed in the general population. Interestingly, in this general population sample, they found that especially cognitive symptoms within the depression spectrum were associated with several clinical characteristics, including higher neuroticism and lower introversion scores, longer depression duration and more chronicity. In contrast, previous work on post-MI depression has suggested that specifically somatic or neurovegetative symptoms are associated with poor cardiovascular outcomes. For instance, in cardiac patients, the treatment effects on depressive symptoms after 8 weeks for four antidepressants (fluoxetine, sertraline, mirtazapine and citalopram) were highly similar to those reported by Turner and colleagues in unselected patients using FDA data. Similarly, although randomised comparisons of psychotherapeutic interventions have not always been very successful, there is much evidence to believe that these interventions are less effective in patients with ACS than in those without.

One interesting aspect that Poole and colleagues touched upon is the reference to the fascinating work by Appels and colleagues on vital exhaustion. The concept of vital exhaustion was developed first as a form of mental and physical distress preceding ACS, but later on also as a sequel of ACS with potentially cardiotoxic properties. Of interest, vital exhaustion was thought to consist of a combination of negative cognitions, vital exhaustion was thought to consist of a combination of negative cognitions, feelings of exhaustion and irritation, which is consistent with the reference to the fascinating work by Appels and colleagues on vital exhaustion. The concept of vital exhaustion was developed first as a form of mental and physical distress preceding ACS, but later on also as a sequel of ACS with potentially cardiotoxic properties. Of interest, vital exhaustion was thought to consist of a combination of negative cognitions, feelings of exhaustion and irritation, which is consistent with the reference to the fascinating work by Appels and colleagues on vital exhaustion. The concept of vital exhaustion was developed first as a form of mental and physical distress preceding ACS, but later on also as a sequel of ACS with potentially cardiotoxic properties. Of interest, vital exhaustion was thought to consist of a combination of negative cognitions, feelings of exhaustion and irritation, which is consistent with the reference to the fascinating work by Appels and colleagues on vital exhaustion.

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depression is slightly different than vital exhaustion as they consider it to be a subtype of depression, i.e., fulfilling the criteria for MDD. Still, perhaps insights from the literature on the etiology (e.g., the role of inflammation in vital exhaustion [15]) and treatment of vital exhaustion (e.g., relaxation therapy, [16]) might help in improving the efficacy of depression treatment.

I believe that Poole and colleagues are teaching us a valuable lesson in depression deconstruction, and this may well be a lesson that goes beyond the field of cardiopsychiatry and psychosomatic research. Only the future can tell whether the identification of inflammation-based depression will be truly influential. Its relevance, I believe, will depend on the question of whether we will be able to develop and test interventions that are specifically effective in reducing inflammation-based symptoms of depression.

References