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Vital Exhaustion, Burnout, and Other Avatars of Depression

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> "… a science that ignores the mandate for parsimony cannot advance its knowledge base and achieve cumulative knowledge. The purpose of science is to uncover the relatively simple deep structure principles or causes that underlie the apparent complexity observed at the surface structure level… and this is essentially impossible if the mandate for parsimony is not observed."
>
> Le, Schmidt, Harter, and Lauver (1) (p. 112)

Frestad and Prescott (2) conducted a systematic review and meta-analysis of 13 prospective and 3 case–control studies dedicated to the impact of vital exhaustion (VE) (3) on coronary heart disease (CHD). The authors concluded that VE is an *independent* risk factor for incident CHD and heart failure. In our estimation, these conclusions require a more cautious interpretation.

Because depression has been identified as a risk factor for CHD (4–6) and VE has been found to overlap with depression (7–10), depression constitutes a plausible confounder in VE research. Unfortunately, depression was not assessed in most studies included in the systematic review and meta-analysis of Frestad and Prescott (2). This lacuna was mentioned in several of the included studies as a limitation (11–13). In one of the included studies (14), a measure of "negative self-concept" was employed but other depressive symptoms were left unmeasured. Remarkably, in the only included study in which depression (assessed with the Center for Epidemiological Studies Depression scale) was analyzed as a potential confounding variable, VE no longer predicted CHD when depressive symptoms were adjusted for (15). All in all, this state of affairs undermines Frestad and Prescott's conclusions (2). Most probably, VE is *not* associated with incident CHD or heart failure independently of depression (15) because of a problematic overlap of VE with depression (10).

It should be underscored that VE-depression overlap is no surprise in view of the items of the Maastricht Questionnaire (3)—the most widely used measure of VE. Indeed, the items of the Maastricht Questionnaire conspicuously refer to pervasive fatigue and loss of energy, sleep disturbance, cognitive impairment, death wishes, feelings of helplessness, apathy, and hopelessness, irritability, loss of libido, and lowered mood. Most of these symptoms reflect established diagnostic criteria for major depressive disorder and/or persistent depressive disorder (dysthymia), and *all* of them constitute well-identified manifestations of subclinical and/or clinical forms of depression (16–18). It has been argued that VE differs from depression in that (*a*) VE is primarily characterized by lack of energy or fatigue and (*b*) sad mood in VE reflects a demoralization rather than a lowered self-esteem (19). Such arguments do not resist a close examination. First, lack of energy and fatigue often dominate the clinical picture of depression "with atypical features"—a frequently encountered depressive syndrome (20). Second, demoralization is characterized by feelings of impotence, isolation, and despair (21). These feelings are characteristic of depression (16,17).

In our view, there is a worrying tendency in psychology and medicine to let proliferate "depression-like" constructs—a transgression of the scientific canon of parsimony (1). The problem is not limited to VE. Burnout, a condition akin to VE (2,22,23), has been shown to problematically overlap with depression (24,25). Compassion fatigue, a condition that shows particularly blurred definitional contours, is also uncomfortably close to depressive symptomatology (26). The construct of neurasthenia may be part of this confusing trend as well (27–29), although neurasthenia has been elevated to the status of nosological category in the International Classification of Diseases (22). Construct proliferation jeopardizes knowledge growth by undermining theory building and transdisciplinary communication (1). Instead of multiplying unnecessary variations on the theme of depression, we think that researchers should work at better coordinating dimensional and categorical approaches to depression. By enhancing such a coordination, researchers would allow themselves to better take into account both subclinical forms of depression and clinical subtypes of depression (e.g., depression with melancholic features, depression with atypical features) in their empirical and theoretical analyses. Researchers who assume that VE, burnout, compassion fatigue, or neurasthenia lie outside the spectrum of depression should systematically adjust for depressive symptoms in their statistical analyses to corroborate their beliefs.

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Vital Exhaustion and Depression: A Reply to Bianchi and Colleagues

We thank Drs. Bianchi, Schonfeld, and Laurent for their interest in our recent review and for contributing with an insightful critical analysis on the complex relationship between two psychological concepts: vital exhaustion (VE) and depression. The authors have questioned whether there is a well-documented difference between the concepts, referring to the debate on whether VE plays an independent role as psychosocial risk factors for coronary heart disease (CHD).

We agree that a considerable overlap between depression and VE is likely. Conceptually, however, they differ. VE is a psychological construct, characterized by a set of symptoms constituting the items of the Maastricht Questionnaire and is conceptually perceived as a response to prolonged psychological distress. Importantly, there is no consensus regarding a cut-off value for "true" exhaustion. Depression, on the other hand, is psychopathology with very specific diagnostic criteria. Despite its etiological heterogeneity, several neurobiological theories have been proposed as the pathophysiology behind depression (1). Even though characteristics of somatic depression and VE are overlapping in terms of symptoms, the scales do not describe the same entity (2–4). Patients who are vitally exhausted do not necessary meet the criteria for depression and vice versa. There are few studies of this overlap, one suggesting that VE mainly overlaps with the somatic/affective dimension of depression (5). In addition, differences in the relationship of VE and depression to CHD risk factors are also consistent with making a distinction between VE and depression (6).

Although a vast literature documents a positive association between depression and CHD, the role of depression as an independent etiological and prognostic factor in CHD remains controversial (7). A meta-analysis based on 21 etiological and 34 prognostic studies concluded that incomplete and biased reporting of adjustment for traditional cardiovascular risk factors and the severity of coronary disease in published literature may contribute to inflated risk estimates. Should studies of the association between VE and outcome be adjusted for depression? One might also argue that association between depression and CHD should be adjusted for VE. The selection of covariates in multivariable models is complex, and methodological recommendations are that these choices should be guided by theoretical principles (8).

Whether VE captures "subclinical forms of depression" or "clinical subtypes of depression" is yet to be investigated, nevertheless, it can be applied to patients who do not fulfill the diagnostic criteria for "major depressive disorder and/or persistent depressive disorder (dysthymia)." The aim of this meta-analysis and review was to summarize the existing published literature on VE and its role in incident and recurrent CHD. The study conclusion remains unchanged, and existing published research suggests that VE is associated with a significantly increased risk of fatal and nonfatal CHD. We did not conclude that associations between VE and outcome were independent of measures of depression. The link between VE and CHD is complex and difficult to evaluate objectively because of variability in the use of quantitative assessment methods.

We hope that our meta-analysis and review will result in new research on the assessment of VE, biological, and behavioral mechanisms that play a role in the predictive value of VE for adverse cardiovascular outcomes. These studies may lead to a better understanding of the pathological pathways involved in VE that may ultimately result in novel interventions targeted at reducing cardiovascular risk.

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