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Letter to the Editor

Does vital exhaustion enhance our ability to predict type 2 diabetes?

In a recent prospective study involving 9075 Danish participants, Volden et al. [1] examined the relationship between vital exhaustion (VE) and type 2 diabetes. Based on the finding that stress and depression are predictors of type 2 diabetes, the authors hypothesized that VE too could be a risk factor for type 2 diabetes. The authors found that VE prospectively predicted type 2 diabetes and suggested that assessing VE in clinical practice may help prevent type 2 diabetes. Problematically, the added value of the VE construct with respect to the prediction of type 2 diabetes cannot be established based on the conducted study.

As underlined by the authors themselves, stress and depression have been identified as important risk factors for type 2 diabetes [2,3]. Disconcertingly, however, the authors did not control for stress and depression in their analyses of the relationship between VE and type 2 diabetes. As a result, the central question of whether VE explains unique variance in type 2 diabetes cannot be addressed in the conducted study. We understand that assessing and controlling for previously-identified predictors of a given phenomenon can be methodologically costly. This being noted, in the present case, assessing and controlling for stress and depression would not have been burdensome given the availability of relatively brief measures of the two entities (e.g., the Perceived Stress Scale [4] and the PHQ-9 [5]).

Controlling for depression was crucial, not only because depression is a predictor of type 2 diabetes, but also because the extent to which VE can be distinguished from depressive conditions is, to say the least, unclear [6,7]. Interestingly, the authors displayed the items of the questionnaire that they used for assessing VE (see Figure 1 in the authors' article). Unambiguously, the symptoms listed (e.g., dysphoria, fatigue, suicidal ideation) are depressive in nature. It is unfortunate that the authors did not elaborate on this issue. Indeed, while the authors distinguished VE from depression in their study, the *raison d'être* of such a distinction is difficult to understand in view of the content of the VE measure that the authors used. Construct proliferation threatens theory building and transdisciplinary communication among a scientific community [6-8]. Whenever relevant, efforts should therefore be made to identify redundancy and foster parsimony. In VE research, taking depression into account should not be considered optional.

All in all, we disagree with Volden et al. [1] when they suggest that they may have discovered an additional risk factor for type 2 diabetes. To draw such a conclusion, the authors should have (at least) been able to show that VE explained variance in type 2 diabetes that was not already accounted for by stress and depression. Given that VE is unlikely to reflect any distinct morbid or premorbid phenomenon [6,7], our guess is that the inclusion of stress and depression in the tested models would have rendered the explanatory power of VE non-significant. If this line of reasoning is valid, then recommending that VE be assessed in addition to stress and depression in clinical settings is unwarranted. Such a recommendation may in fact lead to pointlessly increase patients' and clinicians' workload.

Conflict of interest

We declare no competing interests.

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