

“Critical slowing down in depression” is a great idea that still needs empirical proof

With great interest we read the paper by van de Leemput et al. (1). The authors conceptualize depression as a complex dynamic system with two distinct states (normal and depressed) and support this idea with dynamic system theory and model simulations. Unfortunately, when presenting empirical evidence for their supposition, the authors fall into the trap of generalizing group-level results to the individual level and mixing up between-subject and within-subject variability, a trap often encountered in the medical field (2).

The authors study depressed and non-depressed persons during a baseline mood-monitoring period and subsequent follow-up. Three indicators of critical slowing down (elevated autocorrelation, variance, and correlation between emotions) were associated with upcoming transitions. This evidence was based on between-subject variability: persons with higher autocorrelation, variance, and correlation compared with others also showed higher (change in) follow-up scores than others. Nevertheless, the authors interpret the evidence as a within-subject effect, using phrases like “increasing autocorrelation,” “rising variability,” and “critical slowing down.” This is questionable for two reasons. First, the design does not allow a within-subject interpretation, because the indicators of critical slowing down were not measured repeatedly within individuals. Second, higher autocorrelation, variance, and correlation may also be explained by higher baseline levels on the emotion variables. Autocorrelation was estimated

in a multilevel model using the interaction between an autoregressive parameter and follow-up depression score. Estimates in multilevel models, however, are a mixture of between- and within-effects, if these effects are not properly disaggregated (2, 3). The autoregressive parameter will then “pick up” variance due to between-subject differences in mean levels. Thus, the higher autocorrelation found in the high-risk group may actually reflect higher baseline levels of the emotions.

Variances were assessed by estimating random effect variances in a three-level model. However, the variance is known to be related to the mean and may be especially low in the case of floor and ceiling effects (4), as in the present study. Thus, also between-subject differences in variance may reflect differences in baseline levels of the emotions. The authors therefore rightly state that the variance may not be the best indicator of critical slowing down (which may also explain why they discount the contradictory evidence of a lower variance in the high-risk group in two of the emotions; their appendix). The correlations between the emotions were estimated by random effect correlations of the three-level model. However, correlation coefficients are affected by the variances of the emotions (5). Because these variances may be confounded by baseline levels of the emotions, the correlations among the emotions may be as well.

To summarize, we think that the supposed within-subject effect can just as well be ex-

plained by between-subject differences in baseline levels of the emotions. Further, whether the assumed “phase transition” really takes place is also uncertain; the change may just as well be gradual or minimal. Therefore, the authors’ conceptualization of depression as a dynamic system is appealing and may be right, but the empirical evidence is weak. Future studies should disaggregate inter- and intraindividual variability more carefully.

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