

Letter to the Editor

Testing Criteria for Hypomania: Author's Reply to Peele

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TO THE EDITOR:

Professor Peele (George Washington University, Rockville, MD) questioned if the diagnostic criteria for hypomania tested in this Journal (1) could also be applied to mania, supporting the diagnostic utility of overactivity (increased goal-directed activity) for mania, too (Letter to the Editor 07–19–07). According to Kraepelin (2), mania and hypomania were on a continuum of severity, sharing the same basic domains of excitement of mood, thinking, and activity. Differently from DSM-IV, Kraepelin did not set any priority among the basic domains of hypomania/mania (mania could even be diagnosed if mood was depressed, such as in the mixed state “depressive mania”). This dimensional, no-priority of symptoms approach to hypomania and mania has recently been supported by epidemiological and clinical studies, finding a continuum of symptoms in mania and hypomania, and between mania and hypomania (3). Two to three factors have been found in hypomania, one including elated overactivity, and one including irritable risky behaviours (reviewed by Benazzi (3)). Many and different multivariate analyses of mania, differently from hypomania, have found a more complex symptom structure, including factors/clusters/subtypes (reviewed by Benazzi (4)). Some psychometric studies of mania have supported “activation” as its basic feature (3,4). The current diagnostic criteria for mania and hypomania, instead, set priority of mood change (elevated and/or irritable mood, criterion A) over a group of symptoms including overactivity (criterion B). While psychometric studies of hypomania are consistent, not only on its factorial structure, but also on the priority of overactivity (at least at the same level of mood change), this is not the case for mania, even if overactivity is its most common sign (5).

Findings in hypomania cannot be applied to mania. While there seems to be a grading of severity of manic/hypomanic

symptoms, the priority of overactivity in mania, as it is for hypomania, has to be supported by more studies. One basic difference in the study of hypomania versus mania is that the clinical picture of hypomania is based mainly on the recall of episodes (as it is uncommon for a bipolar II patient to present for treatment of hypomania, which has often improved functioning), while studies of mania are usually based on directly observed inpatients.

That the symptom structure of mania may be different from that of hypomania can be inferred from some basic data, such as the outpatient nature and the often improved functioning of hypomania, and the often inpatient, psychotic, and severely impairing functioning of mania. In hypomania overactivity is often goal-directed and often improves functioning, while in mania overactivity may or may not be goal-directed and markedly impairs functioning. These clinical features suggest a more complex nature of mania, making unlikely a simple overlap of the most discriminative symptoms (i.e., the core features, such as overactivity) of mania and hypomania (at least for the most severe mania, given the continuum of symptoms between mania and hypomania) (3,4).

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