Prog Neuropsychopharmacol Biol Psychiatry. 2009 Feb 1;33(1):86-93. doi: 10.1016/j.pnpbp.2008.10.007. Epub 2008 Oct 27.

Classifying mood disorders by age-at-onset instead of polarity.

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Abstract

BACKGROUND: Polarity is the pillar of the current categorical unipolar-bipolar division of mood disorders. However, genetic studies on these polarity-based phenotypes have been largely inconclusive. Recent clinical and epidemiological studies seem to support more of a continuum than a splitting of mood disorders. A reshaping of the classification of mood disorders thus seems required. Age-at-onset and recurrence have been suggested to be more clinically and genetically useful in the phenotyping of mood disorders.

STUDY AIM: To test a classification of mood disorders based on age-at-onset, and to delineate its phenotypes.

METHODS: A total of 441 consecutive bipolar II disorder (BP-II) and 289 unipolar major depressive disorder (MDD) outpatients, presenting for treatment of a major depressive episode (MDE) in a clinical and research private practice, were assessed by a mood disorder specialist psychiatrist (FB) using a Structured Clinical Interview for the DSM-IV, modified for better probing past hypomania [Benazzi, F. Bipolar disorder-focus on bipolar II disorder and mixed depression. Lancet 2007a;369: 935-945]. The sample was divided according to age-at-onset. Age-at-onset was defined by the age at onset of the first MDE. Early-age-at-onset (EO) was defined as age at onset before 21 years, late-age-at-onset (LO) as onset at or after age 21 years. The study's current goal had not been planned when data were recorded between 1999 and 2006. Variables were compared in EO versus LO mood disorders, investigating phenotype differences. The main focus was on 'classic' diagnostic validators: MDE clinical picture, gender, course, and family history. Age, gender, BP-II, and mania/hypomania family history (possible confounding) were controlled for in the analyses. Logistic regression was used.

RESULTS: First, EO was regressed on each variable, one at a time, to find significant associations. Second, EO was regressed on all of the variables whose odds ratio (OR) was statistically significant in the previous analyses in order to find independent predictors. Independent predictors of EO mood disorder were history of hypomania, high recurrence, atypical depression, and family history of mania/hypomania. Controlling for BP-II (in addition to age and gender) did not impact the findings. The highest OR was that between EO and high recurrence (OR=4.00). Distinguishing MDE symptoms of EO mood disorder included hypersomnia and psychomotor agitation when controlling for age and gender, and, by controlling also for BP-II, hypersomnia only.

DISCUSSION: A close association among EO mood disorder, high recurrence, and bipolarity (history of hypomania, family history of mania/hypomania) was found. Compared to most previous studies testing EO versus LO in bipolar (mainly BP-I) or in unipolar MDD samples, the present study tested a mixed BP-II and MDD sample and controlled for polarity, reducing, as much as possible, the impact of polarity on the findings. EO (below age 21 years) was distinguished by hypersomnic depression, high recurrence, high history of hypomania, and high history of mania/hypomania. Replications are needed, especially in mixed samples also including BP-I. Results, if replicated, could have implications not only for clinical and genetic studies, but also for treatment (e.g., mood stabilizers could have better long-term effects than antidepressants in EO mood disorders, antidepressants could have negative long-term effects on EO).

PMID: 18992784 [PubMed - indexed for MEDLINE]