

and 54% are not fully euthymic between episodes (Kupka *et al*, 2001).

Children of mothers with bipolar I disorder have increased rates of both unipolar disorder and ADHD, further suggesting a neurobiological overlap of these three diagnoses. Hirshfeld-Becker *et al* (2006) report significantly higher rates (23.5%) of ADHD in offspring of parents with bipolar disorder compared with psychiatric comparison parents (8.4%) and non-psychiatric comparison parents (4.2%).

Drug treatments also overlap. Stimulant-type medication has been used in bipolar depression, and newer medications such as atomoxetine have similar pharmacological characteristics to some antidepressants (Lydon & El-Mallakh, 2006). Catecholaminergic antidepressants are not only potentially of benefit in ADHD but may be less likely to destabilise bipolar disorder.

There is thus a clinical and neurobiological overlap between ADHD, bipolar and unipolar disorder. Asherson *et al*'s timely editorial has reminded us that ADHD in adults should not be overlooked and that further research is needed to clarify its impact on other adult psychopathology and comorbidity, particularly in mood disorders.

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Authors' reply: Kuan & Young point out that further research into the role of mood symptoms in attention-deficit hyperactivity disorder (ADHD) is essential. In a recent study of 141 adults with ADHD, 95% were found to have mood symptoms, chiefly mood instability (Kooij, 2007). We observe that in adult ADHD mood instability frequently responds to stimulants over the same time course as core ADHD symptoms, an observation reported by others. This has led to the suggestion that mood dysregulation might represent a core impairment in adult ADHD, perhaps related to the same processes that cause dysregulation of other executive processes.

Despite these observations the relationship of ADHD to mood disorders is controversial. The controversy has arisen in the context of paediatric bipolar disorder, where the distinction from ADHD is made difficult if one chooses to view irritability as a sufficient manifestation of bipolar disorder and if the requirement for episodicity is not strictly applied. However, available validation studies for the construct of paediatric bipolar disorder use elation and/or grandiosity as cardinal symptoms, rather than irritability. Narrowly defined paediatric bipolar disorder can be differentiated from ADHD, shows longitudinal stability and has plausible familial aggregation patterns (Geller & Tillman, 2005; Geller *et al*, 2006). Recent evidence suggests that the narrowly defined disorder can be distinguished at the behavioural and electrophysiological level from broadly construed disorder (Rich *et al*, 2007). Conversely, it has been argued that the intensity of irritability (Mick *et al*, 2005) and its temporal pattern (chronic or episodic) can distinguish paediatric bipolar disorder from ADHD (Leibenluft *et al*, 2006). The family study of Hirshfeld-Becker *et al* (2006) is intriguing, yet the sample size is small (12 families with bipolar I disorder, 11 with bipolar II disorder), and further work is needed to clarify the rates of ADHD among relatives with narrowly defined *v.* broadly defined bipolar disorder.

One of the main questions to be addressed relates to how valid a diagnostic concept broadly defined bipolar disorder is, or whether mood instability/irritability in the presence of ADHD may be more

adequately described by a new dimension, such as mood dysregulation (Brotman *et al*, 2006). Until the relevant empirical data become available, we see merit in maintaining the classic definition of mania, so that a diagnosis of bipolar disorder requires euphoria, grandiosity and episodicity, and the differential between ADHD and bipolar disorder remains explicit.

Brotman, M. A., Schmajuk, M., Rich, B. A., et al (2006) Prevalence, clinical correlates, and longitudinal course of severe mood dysregulation in children. *Biological Psychiatry*, **60**, 991–997.

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