The role of emotional instability in adult ADHD, borderline personality disorder, bipolar disorder, autism and intellectual disability: A transdiagnostic construct or disorder specific syndrome?

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Emotional instability and adult attention-deficit hyperactivity disorder (ADHD)

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Background ADHD is defined in DSM-5 by developmentally inappropriate and impairing levels of inattentive and hyperactive-impulsive symptoms. However, emotional dysregulation is considered to be an associated feature of the disorder that supports the diagnosis of ADHD. The common co-occurrence of emotional liability (EL) in ADHD raises the question of whether EL should be viewed as a component of ADHD or reflecting a comorbid condition.
Aims To address the question of whether EL should be viewed as a third dimension of ADHD.
Method We investigated the association of EL with ADHD and impairment scores, in a sample of adults with ADHD that had been carefully selected for absence of comorbid conditions that could give rise to EL using both rating scale and experience sampling methods to measure emotional instability. We reviewed the effects of stimulants and atomoxetine on EL and the covariation of EL with ADHD inattention and hyperactivity-impulsivity. We further considered the phenotypic and genetic association of EL with ADHD using population twin data.
Results From these studies, we found that EL is strongly associated with ADHD even in non-comorbid cases and gives rise to additional impairments after ADHD symptoms are controlled for in the analysis. Stimulants and atomoxetine both improve EL and these improvements are correlated with changes in ADHD symptoms, indicating a shared treatment response. Genetic model fitting suggests a common pathway model, consistent with a single genetic liability for inattention, hyperactivity-impulsivity and EL.
Conclusions Taken together these findings suggest that EL can be viewed as a third dimension of ADHD. Patients presenting with chronic emotional instability should always be screened for ADHD.
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Emotional instability and borderline personality disorder

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Affective instability is widely regarded as being the core problem in patients with borderline personality disorder (BPD) and the driving force behind the severe clinical manifestations of BPD symptoms. In ICD-10, BPD is even labelled as emotionally unstable personality disorder. In the last years, the advent of electronic diaries, in combination with sophisticated statistical analyses, enabled studying affective instability in everyday life. Surprisingly, most recent studies using state-of-the-art methodology to assess and model affective instability in BPD failed to show any specificity, supporting the idea of a transdiagnostic construct. In addition, dysfunctional emotion regulation strategies revealed results contradictory to current clinical beliefs. Using multiple data sets and multilevel modelling, we will demonstrate that to understand affective instability it is important:
– to statically model basic subcomponents of affective dynamics simultaneously;
– in combination with dysfunctional regulation strategies;
– cognitive processes in everyday life.
Altogether, current research suggests that the dynamics of affective states and their intentional regulation are even more important to psychological health and maladjustment, than the affective states itself. Current initiatives to fundamentally improve psychopathological research are looking at basic physiological processes spanning across disorders. However, these approaches do fall short in understanding human behaviours as dynamical processes that unfold in the broadest setting imaginable – everyday life. Only the combination of basic physiological processes and methods assessing dynamical affective mechanisms in everyday life will enhance our understanding how dysregulations and dysfunctions of fundamental aspects of behaviour cut across traditional disorders.
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Emotional instability and bipolar disorder

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Background Affective dysregulation is a core feature of bipolar disorder (BD) and a significant predictor of clinical and functional outcome. Affective dysregulation can arise from abnormalities in multiple processes. This study addresses the knowledge gap regarding the precise nature of the processes that may be dysregulated in BD and their relationship to the clinical expression of the disorder.
Methods Patients with BD ($n = 45$) who were either in remission or in a depressive or manic state and healthy individuals ($n = 101$) were compared in terms of the intensity, duration and physiological response (measured using inter-beat intervals and skin conductance) to affective and neutral pictures during passive viewing and during experiential suppression.
Results Compared to healthy individuals, patients with BD evidenced increased affective reactivity to neutral pictures and reduced maintenance of subjective affective responses to all pictures. This pattern was present irrespective of clinical state but was more pronounced in symptomatic patients, regardless of polarity. Patients, regardless of symptomatic status, were comparable to healthy individuals in terms of physiological arousal and voluntary control of affective responses.
Conclusion Our study demonstrates that increased affective reactivity to neutral stimuli and decreased maintenance of affective responses are key dimensions of affective dysregulation in BD.
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Emotional instability and autism and intellectual disability

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