

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 lability (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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S105

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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S106

Emotional instability and autism and intellectual disability

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Aim To explore if emotional instability is a useful construct in adults with autism spectrum disorder (ASD) and intellectual disability (ID).

Method/approach The current diagnostic criteria for ASD and ID will be outlined and related to any relevant literature on emotional instability in those with ASD or ID. Recent cross-sectional studies in a clinic and a prison of adults with ASD and/or ID using standardised screening and diagnostic tools will be described.

Findings Current literature indicates there is little research on emotional instability in adults with ASD and ID. Studies across clinic and forensic settings indicate high levels of comorbidity such as attention-deficit hyperactive disorder (ADHD) and mood disorders in adults with ASD and ID.

Conclusion At present emotional instability as a construct may not have validity for adults with ASD and ID but may do in the context of other comorbid conditions such as ADHD.

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Thought and language disorders: Phenomenology and neural pathophysiology

S107

How increasing the effect of rTMS in the treatment of auditory hallucinations in schizophrenia?

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Repetitive transcranial magnetic stimulation (rTMS) shows a high inter-subjects variability in the efficacy of treatment of auditory verbal hallucinations (AVH) in schizophrenia. The aim of this presentation is to demonstrate the involvement of several factors in the efficacy of rTMS such as the frequency of stimulation, the placebo effect and the brain morphology underlying the target of stimulation.

Methods A meta-analysis was conducted to determine the effect sizes of placebo effect in 21 controlled studies on rTMS in the treatment of AVH in schizophrenia. MRI was also acquired in patients treated by rTMS to evaluate the scalp to cortex distances (SDCs) and the gray matter densities (GMDs) at the target of stimulation. Finally, we evaluated the efficacy of high (20 Hz) frequency stimulation in a controlled placebo study.

Results Weak or no placebo effect in the control groups led to reveal a superiority of active rTMS over sham rTMS in the treatment of AVH. Clinical efficacy of rTMS was also correlated with the SCD or the GMD at the region of the target stimulation. Finally, we also demonstrated that more responders were observed after 2 weeks in the active group treated by 20 Hz than in the placebo group.

Conclusion We clearly demonstrated that several factors such as high frequency, the placebo effect, anatomical cortical variations can impact on the efficacy of rTMS. These results fundamentally inform the design and the method of further controlled studies, particularly with respect to studies of rTMS in the treatment of AVH.

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S108

Phenomenology and neural correlates of formal thought disorder

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Speech and language disorders, such as concretism and formal thought disorder (FTD) are core symptoms of Schizophrenia, but do occur to a similar extent in other diagnoses such as bipolar disorder and major depression. We will review clinical rating scales of FTD and introduce a new, validated scale, the TALD. Further, structural and functional brain imaging data will be reviewed and own novel findings presented, relating speech and language dysfunctions to neural networks, within schizophrenia and across the “functional psychoses”. The impact of genetic variance and NNDA receptor blockage on brain function will be addressed with a particular focus on speech and language (dys-) function. We demonstrate, from the genetic to the brain structural and functional level, that particular aspects of the neural language system are disrupted in patients with FTD across traditional diagnoses.

Disclosure of interest The author has not supplied his declaration of competing interest.

Further reading

Kircher T, Krug A, Stratmann M, Ghazi S, Schales C, Frauenheim M, et al. A rating scale for the assessment of objective and subjective formal Thought and Language Disorder (TALD). *Schizophr Res* 2014;160(1–3):216–21.

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S109

Language, psychosis and the brain: Novel insights from a dimensional approach

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The brain mechanisms related to formal thought disorders (FTD) and auditory verbal hallucinations (AVH) appear to be closely linked to structural and functional abnormalities of the language system.

In particular, functional imaging data indicate that several components of the language system are involved in the generation of both, FTD and AVH. Co-activation of the primary auditory cortex (Heschl's gyrus) during verbal thoughts appears to be crucial for the subjective perception of an externally generated voice, and DTI studies indicated that the intrahemispheric fronto-temporal connectivity of the language system is specifically increased in hallucinating patients. On the other hand, FTD are significantly correlated to a gray matter reduction in Wernicke's region along with a hyperactivation of frontal and temporal components of the language system.

AVH are intimately related to a dysfunction of the left hemispheric language system, including the primary auditory cortex and the fronto-temporal fibre tracts connecting Broca's and Wernicke's regions. Co-activation of the primary auditory cortex during verbal thoughts appears to be the basis of the pathological network dynamics during AVH, while FTD are linked to a pathological hyperactivity of central components of the language system. Both phenomena are related to functional imbalances of the language system, and phenomenological differences may depend on the different contributions of the system components.

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