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Commentaries

Intermittent explosive disorder and DSM-5: A flawed conceptualization of pathological anger

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DSM Digest

Intermittent explosive disorder (IED) was first introduced into the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III), as one of the impulse control disorders. Since then, there has been a substantial body of literature supporting the paradigm of impulsive aggression (Siever, 2008). If DSM-5's overarching philosophy of clinical utility as 'the highest priority' is to be respected, then, as a bone fide 'disorder', IED has unfortunately fallen far short of this standard. This DSM digest attempts to explain why.

Building upon the stated aims of DSM-5, (American Psychiatric Association, 2013) namely, reducing the 'confusion of disorders' and identifying symptoms that 'cut across' different diagnoses, there is now ample evidence that impulsive aggressive acts are certainly not confined to IED, and that, in practice, they are far more commonly associated with borderline and antisocial personality disorders, as well as with Axis I disorders such as substance abuse disorders and mood disorders (Moeller et al., 2001). As is now readily acknowledged, co-morbidity is more the 'rule' than the exception, with respect to the DSM (First, 2005). This, in turn, continues to leave lingering questions over the construct validity and the 'prototypical' phenomenology of IED, including the very criteria that delineate it from other causes of aggression.

Though allowing for such exclusions, DSM-IV-TR appears to portray IED as an overly simplistic 'failure to resist' aggressive impulses followed by 'relief' of tension. Not only is this anomalous in community practice, it also tends to divorce impulsive aggression from anger or 'the body from the head' when in fact anger and aggression are so inextricably entwined. It also unduly narrows the focus on impulsivity as the *main* factor underlying impulsive aggression when, by its own description, neurological, personality and Axis I disorders should be considered.

This is where the dimensional construct of personality disorders for DSM-5 might have been of greater clinical utility – listing those temperamental and cognitive *predispositions* that are *risk factors* for impulsive aggression (Wilkowski and Robinson, 2008), thereby allowing clinicians the opportunity for more tailored and biopsychosocial interventions. Perhaps then, in categorizing IED as a disorder of impulse control, its proponents were mistakenly generalizing that its phenomenology is indeed similar to other 'so-called' ego-dystonic behaviors, when in reality it is not.

A recent paper cited revised 'integrated criteria' for IED (IED-IR) in advocating for its continued inclusion in DSM-5 (Coccaro, 2011). Its recommendations re: frequency of outbursts appears to have been subsequently incorporated into the final draft of the DSM-5. Nonetheless, the proposed 'regularity' criteria continue to cast doubt on IED's construct validity because they do not meaningfully distinguish 'episodic' from 'generalized' aggression, especially if such episodes occur several times a week or more. Furthermore, the proposed criteria still do not acknowledge *any* specific relational, situational or cognitive antecedents of anger that precipitate such acts of episodic aggression – and which are seen commonly in clinical practice by various different clinical specialties that rely upon the DSM. Lastly, the author acknowledged that the sample group all had personality disorder, and that problems with anger control and its impact on interpersonal relationships made it "very likely" that "most" IED-IR subjects would have a personality disorder.

What binds much of the recent research on IED is that, though its categorization as a 'disorder' may seem questionable, it has emerged more robustly as a behavioral *endophenotype* that cuts across impulsive behaviors and disorders that themselves appear etiologically linked through an 'externalizing spectrum' (Krueger et al., 2007). It has been long established that impulsive aggressive acts are associated with altered central serotonin metabolism (Brown et al., 1982). This has already been recognized for the purposes of targeted pharmacological interventions (Siever, 2008). It would also lend support to IED being better categorized as a

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subtype of borderline and antisocial personality disorder (with quantifiably more severe temperamental instability) or even of post-traumatic stress disorder (PTSD) or major depressive disorder, where there would be particular clinical salience in doing so. Demarcating IED on the basis of biological markers or disproportionality of aggression alone thus remains a flawed logical position from which to argue its separate existence.

Curiously, despite research strides in the area, the phenomenology of anger and its relation to impulsive aggression has continued to be given short shrift by the DSM (Anand and Malhi, 2009). This is intrinsic to the polythetic design of the DSM, whereby more emphasis is given to delineation than to description (Malhi, 2013). In addition, no concerted effort has ever been made to classify pathological anger in its own right. The consequence has been that epiphenomena such as irritability and explosivity tend to be smattered across the Axis I and II spectrum.

In addition, there has been little 'cross-fertilization' of the DSM with psychological research in this clinically important area. Consequently, the salient role of relational aggression (Murray-Close et al., 2010) remains unresolved – and not even linked with the construct of impulsive aggression. Perhaps a dimensional or even iterative approach will reconcile these inconsistencies in future editions. For the moment, however, anger 'as impulse' remains the 'guiding rule' of the newly minted DSM-5.

Declaration of interest

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Evaluation of somatic health care practices in psychiatric inpatient wards

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People with severe mental illness have higher mortality rates due to cardiovascular disease than the general population (De Hert et al., 2011b). Accordingly, many risk factors of cardiovascular disease are associated with mental illness, such as hypertension, abdominal obesity, diabetes and dyslipidemia (Galletly et al., 2012; Ostergaard and Foldager, 2011). Currently, risk factors of cardiovascular disease are frequently underdiagnosed and under-treated in psychiatric inpatients (De Hert et al., 2011a,b). In this respect there are at least three challenges: Firstly, psychiatrists self-reported clinical practice concerning medical illness is not up to date with current guidelines of diagnostics and treatment (Bauer et al., 2008). Secondly, screening rates for cardiometabolic risk factors in psychiatric wards are generally low (Holt et al., 2010). Thirdly, there is a tendency for workers in psychiatry to abrogate responsibility of physical health (Lawn, 2012).

Today, a common solution to under-diagnosing is implementation of screening guidelines. But in doing so, we often forget to ask the essential question: *Why* are our patients under-diagnosed in the first place? To answer this, we must remember three things: (i) Good clinical work is a complex process comprising multiple steps (e.g. clinical examination, treatment) and multiple functions (e.g. nurses, doctors). (ii) In order to find out where to focus efforts of improvement most efficiently, the process must be broken into its components and analysed as such. (iii) Causes for the *same* problem may vary between institutions and departments, and so may the solutions to correct it.

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