

The time is right to launch large-scale controlled treatment effectiveness studies of early-onset binge eating disorders and bulimia nervosa in student populations

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Commentary on: Kessler R.C. *et al.* (2014). A comparative analysis of role attainment and impairment in binge-eating disorder and bulimia nervosa: results from the WHO World Mental Health Surveys. *Epidemiology and Psychiatric Sciences* (doi:10.1017/S2045796013000516).

Eating disorders have often been neglected in epidemiological surveys. The emergence of binge eating disorders (BED) as a relatively common form of problem has increased the power of the epidemiological approach which can therefore be a means of setting eating disorders within a broader context. The majority of cases of bulimia nervosa (BN) and BED that are ascertained using epidemiological case finding methods are not in treatment. Without this type of investigation their needs would be neglected. This paper is a synthesis of several surveys using the standardized methodology of the WHO World Mental Health (WMH) initiative. This includes a coordinated series of community epidemiological surveys examining social and vocational functioning.

In this commentary, I have chosen to frame the findings within the literature from clinical research. In contrast to epidemiological studies that focus on the more common form of eating disorder (BED and bulimia nervosa) for the most part clinical research focuses on anorexia nervosa. Anorexia nervosa is the most visible eating disorder; it has an earlier age of onset and so draws in close others who are prompted to act. Therefore the proportion of cases that are not known to services is lower than for those eating disorders characterized by loss of control over eating (Micali

et al. 2013). It follows that combining the literature from epidemiological and clinical studies may help build a coherent picture of eating disorders as a whole. There are however similarities as well as differences in terms of risk, prognosis and clinical features across the spectrum of eating disorders.

The report notes the importance of co morbidity in explaining long-term disability. An important and unique facet of eating disorders is that this co morbidity may be specifically and causally linked to the eating disorder itself rather than being a consequence of an underlying shared risk factor (such as childhood trauma, perinatal adversity etc.) or a more random association. This is because the symptoms have an impact on nutrition and appetite. Some of the physical and psychological morbidity found in longitudinal studies may be a consequence of specific symptoms (Johnson *et al.* 2002). Starvation impacts on brain plasticity and reduces cognitive, emotional and social flexibility and competence. Habitual abnormal patterns of eating and digestive processes have consequences on the mechanisms underpinning brain/gut synchrony and control of metabolic balance and reward.

A study by Field and colleagues on a sample of 8594 female adolescents (mean age 12, s.d. 1.6) found that BN, BED purging disorder and eating disorders not otherwise specified were longitudinally associated with obesity, depression, substance and alcohol use (Field *et al.* 2012). This trajectory of behaviours and symptoms fits with what is known from preclinical studies. For example, animals exposed to an intermittent pattern of fasting and feasting on high palatability foods show changes in behaviour such as binge eating and a predisposition to select substances of abuse (summarized in a recent review (Avena & Bocarsly, 2012). Moreover, the associated changes in neural circuits and neurotransmitters resemble those that are seen in preclinical studies of addiction in which

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impulsive behaviours become compulsive habits. Negative mood develops in the withdrawal state. Thus abnormal eating behaviours in adolescence can be a gateway to the addictions.

The report also highlights the higher education attainments in this group. The authors speculate that this might be a consequence of high levels of perfectionism. An alternative hypothesis is that high IQ may also contribute. A systemic review concluded that people with anorexia nervosa have a higher than average IQ (Lopez *et al.* 2010). Also the offspring of women with an eating disorder had a higher IQ and working memory (Kothari *et al.* 2012). In relation to this aspect of functioning one assumption that has been made in this project is 'we assumed conservatively that all respondents had normative transitions for their countries (e.g., graduating from primary school in the US at age 14 and from secondary school at age 18)' might not be true especially in the context of anorexia nervosa where schooling is often interrupted by the illness.

Social functioning is considered using marital status as a marker. The authors speculate that overweight status i.e. obesity which is a common consequence of BED might decrease marital eligibility. Indeed this explanation would explain the differential pattern between BN and BED. An alternative explanation is that the lower rates of marriage may be a marker of a more widespread disturbance in social function. For example, there is poor social function before the onset of the illness. This predicts onset and long-term outcome and this is at the heart of recent explanatory models of risk and maintaining factors (Treasure, 2013).

The authors conclude by raising an important question 'whether successful early intervention to treat BED and BN might either prevent the onset or reduce the severity of these secondary disorders'. There are data from the seminal Maudsley, small randomized controlled trial that suggests that early (within the first 3 years of onset), effective (family therapy) for anorexia nervosa has a positive effect on the outcome for a minimum of 5 years (the length of time of follow-up). The group in which the illness had already persisted for over 3 years failed to respond to both forms of treatment (individual and family) and had a less favourable course over 5 years. It is uncertain whether a similar consequence of prolonged untreated BN and BED has a similar effect and reduces the response to treatment and overall outcome. Cognitive behavioural therapy (CBT), the gold standard treatment for bulimia nervosa, produces only moderate-sized symptom change. The question as to whether some of the variation in outcome might relate to the duration of untreated illness has not yet been studied.

Finally, I wholeheartedly endorse the conclusion 'the time is right to launch large-scale controlled treatment effectiveness studies of early-onset BED and BN in student populations to trace out long-term preventive effects on secondary disorders'. We certainly have the tools to make this project feasible. Guided self-care treatments (books, web-based) are effective (Perkins *et al.* 2006) and benefit for both adults and adolescents and are of, easily disseminated and with less cost than the standard CBT (Mitchell *et al.* 2011; Crow *et al.* 2013; Wagner *et al.* 2013b, a). This study has set the context for the problem, suggests key outcomes that can be measured and has made a strong call for action. Let's go.

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Conflict of Interest

None

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