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Eating disorders need more experimental psychopathology

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ABSTRACT

Eating disorders are severe and disabling mental disorders. The scientific study of eating disorders has expanded dramatically over the past few decades, and provided significant understanding of eating disorders and their treatments. Those significant advances notwithstanding, there is scant knowledge about key processes that are crucial to clinical improvement. The lack of understanding mechanisms that cause, maintain and change eating disorders, currently is the biggest problem facing the science of eating disorders. It hampers the development of really effective interventions that could be fine-tuned to target the mechanisms of change and, therefore, the development of more effective treatments. It is argued here that the science of eating disorders and eating disorder treatment could benefit tremendously from pure experimental studies into its mechanisms of change, that is, experimental psychopathology (EPP). To illustrate why eating disorders need more EPP research, some key symptoms - restriction of intake, binge eating and body overvaluation - will be discussed. EPP studies challenge some generally accepted views and offer a fresh new look at key symptoms. This will, consequently, better inform eating disorder treatments.

1. Introduction

Eating disorders are severe, often disabling and frequently chronic mental disorders (Klump, Bulik, Kaye, Treasure, & Tyson, 2009) with high mortality and suicide rates (Fichter & Quadflieg, 2016; Hoang, Goldacre, & James, 2014). They are characterised by overvaluation of weight/shape and abnormal eating, including severe restriction of food intake and/or frequent binge eating and purging behaviours, such as self-induced vomiting and misuse of laxatives. The striving for an ever thinner body leads to severe emaciation in anorexia nervosa (AN) while other eating disorder patients may range between slightly underweight and severely overweight. The lifetime prevalence of eating disorders is about 5%: 0,6% for AN, 1% for bulimia nervosa (BN) and 3% for binge eating disorder (BED) (Treasure, Claudino, & Zucker, 2010), while the prevalence of 'other specified feeding or eating disorders' and 'unspecified feeding or eating disorders' is not known. The scientific study of eating disorders has expanded dramatically over the past few decades (Theander, 2002). It has delivered significant understanding of the clinical dynamics of eating disorders, as well as knowledge of risk factors and the - generally limited - effects of treatments. Those significant advances notwithstanding, there is scant knowledge about the key processes that are crucial to clinical improvement. This lack of understanding about the mechanisms of change currently is the biggest problem facing the science of eating disorders. It hampers the development of really effective interventions that could be fine-tuned to target these mechanisms of change and, therefore, the development of more effective treatments.

In order to answer the seemingly simple question of which mechanisms have to be targeted in order to really reduce eating disorder psychopathology, an elaborate group of behavioural, cognitive, neurocognitive and interpersonal processes have to be taken into account. While an extensive range of sophisticated models on the development and maintenance of disordered eating were described, only a small percentage had progressed beyond mere description towards the development of interventions (Pennesi & Wade, 2016) which is remarkable because the success rates of eating disorder treatments are, in general, modest. Many eating disorder patients drop out of treatment, do not or scarcely benefit from treatment, become chronic or soon relapse after an initial success (Bergh et al., 2013; Bulik, 2014; Galsworthy-Francis & Allan, 2014; Watson & Bulik, 2013). Effective treatments require understanding of the involved change mechanisms: why do treatments work? The experimental study of maintenance mechanisms and mechanisms of change, that is, the key processes that are crucial to clinical improvement, might help to advance the

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treatment of eating disorders. It is argued here that the science of eating disorders and eating disorder treatment could benefit tremendously from in particular *experimental* studies into these mechanisms.

2. Methodological issues

The methodological concerns in eating disorders research are not inherently different from other sciences (Van den Hout, Engelhard & McNally, in press). Our knowledge of risk factors for the development of eating disorders expanded but most of the well-known risk factors come from cross-sectional studies and sometimes from longitudinal studies (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). These cross-sectional and longitudinal studies are well suited for the spotting of associations but they do not per se demonstrate causality, even not when the risk factor precedes the eating disorder symptoms in time. Though it is acknowledged that the study of risk factors is an important first step into factors involved in the development of eating disorders, as all causal factors are risk factors, temporal associations cannot simply be interpreted as causal associations, for other – 'third' – variables could cause both the risk factor and the development of the eating disorder. Therefore, intervening on risk factors to prevent the development or worsening of eating disorders, is premature as long as its causal status is unknown: if the risk factor is not causal, the intervention might not be targeting the key mechanisms.

Likewise, randomised controlled trials (RCTs) intervene on alleged causal or maintaining factors during interventions, but they do not necessarily elucidate the mechanisms at work. Treatment usually means that many factors are manipulated at the same time while it is not precisely clear which components of the intervention are responsible for the treatment effect. The identification of mediators might represent potential mechanisms of change, but while mechanisms of change always are mediators, mediators are not always mechanisms of change (Kraemer, Wilson, Fairburn, & Agras, 2002; Laurenceau, Hayes, & Feldman, 2007). Besides, this could be considered a cumbersome way of searching for these mechanisms: RCTs require the opportunity to study large samples of eating disorder patients and carrying out a methodologically sound intervention study is an extremely laborious, costly and time-consuming process. And if correlates are manipulated that are no risk or causal factors, the RCT is a waste of time (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

It is not argued here that longitudinal studies and RCTs examining processes are not useful, on the contrary, they are very valuable. It is argued here that such a study requires, amongst other things, a strong theoretical model on the dynamics of eating disorders, in particular about the key processes that are crucial to clinical improvement. The testing of treatment effects and processes, using sophisticated models and analyses, is therefore preferably preceded by a research phase in which less costly and less time-consuming laboratory experiments are carried out to test the causality of alleged change mechanisms. Experimental psychopathology exactly does that.

3. Experimental psychopathology (EPP)

EPP refers to the experimental study of mental disorders; experimental psychopathologists bring psychopathology to the laboratory. They carry out well-controlled laboratory experiments with humans in which they manipulate variables that are assumed to be related to the appearance or disappearance of symptoms (Abramson & Seligman, 1977; Van den hout et al., in press; Vervliet & Raes, 2013). Experiments can provide a rigorous examination of clinical processes (Zvolensky, Lejuez, Stuart, & Curtin, 2001) that do not merely describe clinical issues but try to explain and predict them (Van den hout et al., in press). EPP research is considered the interface between fundamental experimental psychology and clinical psychology (Van den hout et al., in press; Vervliet & Raes, 2013; Zvolensky et al., 2001). To understand which factors cause or maintain eating disorders, the manipulation of these factors in well-controlled experimental studies is preferably tested in healthy, non-afflicted individuals. The researcher aims to mimic abnormal processes in healthy individuals by manipulating an alleged causal variable, to test whether the activation of this factor is sufficient for the hypothesised effect to occur. For example, if it is argued that emotional overeating follows from appetitive conditioning while in a sad mood, a laboratory study could study appetitive conditioning (e.g., a neutral cue is repeatedly followed by eating a piece of chocolate while another neutral cue is repeatedly followed by no intake) in healthy volunteers who are in a manipulated sad vs. neutral mood (e.g., by listening to sad or neutral music). If participants in the emotional condition (sad mood) eat significantly more than participants in the neutral control condition during a cued bogus taste test after appetitive conditioning, one can conclude that being emotional facilitates cued overeating. This hypothesis needed to be tested in healthy participants: if it was tested in a clinical or subclinical sample of emotional eaters, their test behaviour could follow from the emotional eating instead of leading to it. Emotional eaters might overeat after emotion induction because they always do this, while the researcher wants to know whether being emotional during appetitive conditioning induces cued overeating in healthy people.

There are ethical issues involved in the mimicking of symptoms in healthy non-afflicted participants by the manipulation of a variable (Zvolensky et al., 2001). This is especially true when experiments are done that potentially induce full psychopathology in healthy volunteers, which ethical committees will, rightly, not approve: ethical concerns constrain the severity of symptoms that can be induced (Sher & Trull, 1996). The psychological manipulations that are used in typical EPP research are quite weak imitations of real causes, and also the effects are usually very weak imitations: mild symptoms of transient duration. In the example above, the induced sad mood is just short and mild lowering of one's mood and the test eating does not involve real binge eating but a relatively higher intake compared to a neutral condition who did not undergo the manipulation of the alleged cause. The aim is not to model an entire disorder or to induce full-blown symptoms, but to test causation: does the activation of factor A lead to the occurrence of the (miniature) symptom B?

Another way to determine the causal status of a factor, in addition to the induced mimicking of symptoms, is to reduce or remove the factor in analogue samples; nonclinical individuals who show subclinical symptoms. If the factor is causal, the symptoms will reduce when the factor is removed. For example, inhibition training reduces 'go' associations and intake in chocolate cravers (Houben & Jansen, 2015) and mirror exposure increases body satisfaction in participants high in body dissatisfaction (Jansen, Voorwinde, Hoebink, Rekkers, Martijn & Mulkens, 2016).

Both ways of studying causality, the induction and reduction of symptoms by manipulating (inducing vs. removing) the alleged causal or maintaining variable, are typical for EPP. It is a most valuable and effective way to learn more about mechanisms that maintain the disorder and, in this way, EPP findings might inform clinical treatment. The translation to actual clinical treatment is a next step in EPP research: removal of the hypothesised causal factor should reduce symptoms in patients. If it does, the EPP model has strong predictive validity, that is, "performance in the model predicts performance in the disorder" (Vervliet & Raes, 2013, p. 2241).