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Review article

A systematic review of temporal discounting in eating disorders and obesity: Behavioural and neuroimaging findings

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ABSTRACT

Objective: Eating Disorders (ED) and obesity are suggested to involve a spectrum of self-regulatory control difficulties. Temporal discounting (TD) tasks have been used to explore this idea. This systematic review examines behavioural and neuroimaging TD data in ED and obesity.
Method: Using PRISMA guidelines, we reviewed relevant articles in MEDLINE, PsycINFO and Embase from inception until 17th August 2016. Studies that reported behavioural differences in TD and/or TD neuroimaging data in ED/obesity were included.
Results: Thirty-one studies were included. Limited data suggest that BN, BED and obesity are associated with increased TD, whilst data in AN are mixed. Aberrant neural activity in frontostriatal circuitry is implicated. TD tasks vary widely and TD in ED/obesity may vary according to factors such as illness stage.
Conclusion: Our findings suggest altered self-regulatory control in ED and obesity. TD tasks are heterogeneous, limiting generalisability of findings. Research into whether TD is multidimensional, along with transdiagnostic neuroimaging research is needed. Assessment of TD may be useful in psychoeducation, outcome prediction and treatment of ED/obesity.

Contents

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1. Introduction

1.1. Eating disorders and obesity

Eating disorders (ED) are serious mental illnesses which affect up to 3.5% of women and 2.0% of men (Schmidt et al., 2016). Anorexia nervosa (AN) has a restrictive (AN-R) and a binge/purge (AN-B/P) subtype: both involve severe food restriction, and AN-B/P also involves binge eating and/or purging behaviours (e.g. vomiting, laxative misuse). Bulimia nervosa (BN) and binge eating disorder (BED) are characterised by frequent binge eating and, whilst BN involves regular compensatory behaviours, BED does not (A.P.A., 2013). Mortality rates in ED are among the highest of all psychiatric conditions (Arcelus et al., 2011; Fichter et al., 2008).

Unlike ED, which are diagnosable psychiatric disorders, obesity is a wide-spread physical health condition. It is characterised by overeating leading to weight gain, and is often associated with reduced psychological well-being (Magallares et al., 2014). Globally, at least 13% of adults are clinically obese and worldwide prevalence rates have more than doubled over the past 40 years (BMI > 30 kg/m^2; WHO, 2015). Raised BMI is a major risk factor for many non-fatal but disabling disorders (e.g. osteoarthritis), is associated with some of the leading causes of death (e.g. diabetes, cardiovascular disease and cancer; Kaiser, 2013; Lu et al., 2014; Yoshimoto et al., 2013) and a BMI indicative of obesity is thought to confer a 2- to 10-year decrease in life expectancy (Whitlock et al., 2009). Obesity is a growing and serious public health issue, it is a major economic and even environmental burden (Dannenberg et al., 2004; Wang et al., 2011).

It is important to emphasise that obesity and ED are distinct physical and psychiatric conditions, respectively. However, comorbidity rates between ED and obesity (lifetime and familial) are high. For example, the prevalence of lifetime obesity in ED is reportedly 28%, ranging from 5% in AN to 87% in BED. Over the last 10 years there has been a threefold increase in obesity in the ED, with prevalence rates predicted to continue to rise. ED patients with lifetime obesity display higher ED severity, worse general psychopathology and have a poorer prognosis than ED patients without lifetime obesity (Villarejo et al., 2012). There also seems to be a clear distinction between obesity and obesity with BED. The latter is associated with more severe obesity, greater medical and psychiatric comorbidity, greater functional impairment and poorer treatment outcomes (Bulik et al., 2002; Fandino et al., 2010; Hsu et al., 1998).

Obesity and ED have a number of overlapping biopsychosocial risk factors, e.g. (epi)genetics, personality traits, ethnicity, adverse events and neurobehavioural processes (Haines et al., 2010; Jacobi et al., 2004; Stunkard, 1988). Studies using a number of neurobehavioural tasks (e.g. Iowa Gambling Task, Wisconsin Card Sorting Test, Stroop, Go-No-Go, Stop Signal Task) demonstrate shared impairments in reward processing and executive functions across ED and obesity. Specifically, reviews and meta-analyses suggest that ED and obesity are associated with comparable neurocognitive difficulties across decision-making, planning, problem solving, cognitive flexibility, reward processing and response inhibition (Bartholdy et al., 2016; Fagundo et al., 2012; Lavagnino et al., 2016; Wu et al., 2014, 2016).

In ED, these difficulties are thought to be underpinned by aberrant frontostriatal neural circuitry, manifesting in impaired regulation of appetite, emotion and self-control (Friederich et al., 2013; Kaye et al., 2011; Kessler et al., 2016; Marsh et al., 2009a,b). More specifically, altered functioning of 'bottom-up' mesolimbic regions (e.g. amygdala, striatum) in conjunction with either reduced or exaggerated 'top-down' cognitive control (via the prefrontal cortex, PFC) are seen as contributing to impulsive (e.g. BN, BED) or exaggerated self-control (e.g. AN) related symptoms/behaviours in ED (Ehrlich et al., 2015; Friederich et al., 2013; Hege et al., 2015; Kaye et al., 2009; Kessler et al., 2016; Marsh et al., 2009b; Sanders et al., 2015).

In people who are obese, impaired executive functions are thought to be underpinned by altered neural activity in areas involved in reward processing (e.g. striatum, insula), emotion and memory (e.g. amygdala), homeostatic regulation of food intake (e.g. hypothalamus) and cognitive control (e.g. PFC) (Carnell et al., 2012; Lavagnino et al., 2016; Pursey et al., 2014; Stice and Yokum, 2016; Tuulari et al., 2015). More specifically, heightened reward sensitivity to food cues (Pursey et al., 2014) in conjunction with reduced cognitive control mechanisms (Tuulari et al., 2015) are proposed to contribute to maladaptive impulse control behaviours in obesity, such as the overconsumption of food. Such processes, similar to those described in addictive behaviours, are likely to contribute to the inability to regulate appetite, food intake and the development of impulsive and often compulsive eating habits (Ziauddeen et al., 2015).

As such, ED, and to a lesser extent obesity, have been considered within spectrum models of self-regulatory control (Brooks et al., 2012; Kaye et al., 2010; Marsh et al., 2007; Piccinni et al., 2015). However, some data suggest that ED may not fit on to a simple continuum. For example, patients with BN are reported to be more impulsive than patients with BED across a number of domains, e.g., self-harm, substance misuse (Hudson et al., 2007). Secondly, whilst