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Research report

Could training executive function improve treatment outcomes for eating disorders? ☆

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ABSTRACT

Current gold standard treatments for eating disorders (EDs) lack satisfactory efficacy, and traditional psychological treatments do not directly address executive functioning deficits underpinning ED pathology. The goal of this paper is to explore the potential for enhancing ED treatment outcomes by improving executive functioning deficits that have been demonstrated to underlie eating pathology. To achieve our objective, we (1) review existing evidence for executive functioning deficits that underpin EDs and consider the extent to which these deficits could be targeted in neurocognitive training programs, (2) present the evidence for the one ED neurocognitive training program well-studied to date (Cognitive Remediation Therapy), (3) discuss the utility of neurocognitive training programs that have been developed for other psychiatric disorders with similar deficits, and (4) provide suggestions for the future development and research of neurocognitive training programs for EDs. Despite the fact that the body of empirical work on neurocognitive training programs for eating disorders is very small, we conclude that their potential is high given the combined evidence for the role of deficits in executive functioning in EDs, the initial promise of Cognitive Remediation Training, and the success in treating related conditions with neurocognitive training. Based on the evidence to date, it appears that the development and empirical evaluation of neurocognitive training programs for EDs is warranted.

Current treatments for eating disorders

Eating disorders (EDs) are serious psychiatric illnesses and the efficacy of existing cognitive behavioral treatments is unsatisfactory (Brownley et al., 2007; Bulik et al., 2007; Shapiro et al., 2007). Innovative treatment methods may be needed to improve outcomes. Anorexia nervosa (AN) has the highest mortality rate of all psychiatric conditions (Birmingham et al., 2005), but the efficacy of existing treatments for adults with AN is limited (Carter et al., 2011; Zipfel et al., 2014). Family-based treatment for adolescents with AN is considered an effective treatment, though its efficacy is moderate at best, with a large percentage of adolescents showing continued symptoms after a full dose of treatment (APA Presidential Task Force on Evidence-Based Practice, 2006). Generally, AN patients are often resistant to beginning treatment (Serpell et al., 1999; Vitousek, Watson, & Wilson, 1998), have poor treatment adherence and acceptance (Halmi et al., 2005), and commonly drop out of treatment (Halmi et al., 2005; Kahn & Pike, 2001; Mahon, 2000; Surgenor, Maguire, & Beumont, 2004). Treatments for bulimia nervosa (BN) and binge eating disorder (BED) are more effective than treatments for AN, but are well short of satisfactory (Kass, Kolko, & Wilfley, 2013). Cognitive Behavioral Therapy (CBT) for BN and BED, including an enhanced, transdiagnostic version, Cognitive Behavioral Therapy-Enhanced (CBT-E), demonstrates the best outcomes to date for these disorders (Byrne et al., 2011; Excellence, N.I.f.C., 2011; Fairburn et al., 2009; Hay et al., 2009; Shapiro et al., 2007; Wonderlich et al., 2013). Interpersonal Psychotherapy (IPT) for BN also has strong empirical support, but has consistently yielded outcomes that are comparable or slightly worse than those of CBT-E (Spielmans et al., 2013). However, one of the most comprehensive and recent studies of CBT-E found that by the end of treatment, only 38.6% of patients with BN met remission criteria and by 60-weeks follow-up, 45.6% met remission criteria (Byrne et al., 2011). Although CBT and IPT are relatively effective treatments for BN, the fact that over 50% of patients are partially or fully symptomatic after CBT-E suggests additional room for improvement (Fairburn et al., 2009).

Aims of the current paper

Deficits in neurocognitive functioning (described in greater detail below) have been hypothesized to contribute to the development

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and maintenance of eating pathology. Addressing these deficits may be one method for improving existing treatment of EDs. This paper will explore the potential for enhancing treatment outcomes through the training of neurocognitive deficits that appear to underlie eating pathology. First, we will briefly review existing evidence for executive functioning deficits that underpin EDs, and briefly present the evidence for the one ED neurocognitive training program well-studied to date (Cognitive Remediation Therapy). We will then (1) discuss the utility of neurocognitive training programs that have been developed for other psychiatric disorders with similar deficits, and (2) provide suggestions for the future development and research of neurocognitive training programs for EDs.

Neurocognitive deficits in eating disorders

Many of the observed neurocognitive deficits implicated in EDs occur in the area of executive function (EF; Kanakam & Treasure, 2013; van Elburg & Treasure, 2013; Van den Eynde et al., 2011). EF is an umbrella term that refers to a set of neuropsychological processes (primarily centered in prefrontal regions) that govern higher-level, goal-directed behavior (Miyake et al., 2000). A set of meta-analyses (Lang et al., 2014; Lopez et al., 2008; Roberts et al., 2007; Wu et al., 2013, 2014) provide strong evidence that deficits in set-shifting (i.e., difficulty flexibly adjusting behavior and cognitions in order to achieve goals in accordance with changing rules or situational demands; Roberts, Tchanturia, & Treasure, 2010; Roberts et al., 2007; Tchanturia et al., 2011; Wu et al., 2014), central coherence (i.e., biased emphasis on small details and a weak ability to integrate information into a gestalt; Lopez et al., 2009), and working memory (i.e., the inability to temporarily store and attend to goal-relevant information, while ignoring distracting or irrelevant informational inputs; Duchesne et al., 2010; Svaldi, Brand, & Tuschen-Caffier, 2010; Zakzanis, Campbell, & Polsinelli, 2010) are present across ED diagnostic groups, and that the magnitude of impairment is similar among AN and BN subtypes (Lang et al., 2014; Roberts et al., 2007). Additionally, poor inhibitory control (i.e., the ability to inhibit a prepotent response) has been observed in EDs characterized by binge eating (Fischer, Smith, & Anderson, 2003; Rosval et al., 2006; Wu et al., 2013). Converging evidence that deficits in EF are present in adolescents who have a short duration of ED illness (Darcy et al., 2012, 2014; Fitzpatrick et al., 2012), and that deficits remain following symptom remission (Holliday et al., 2005; Lopez et al., 2009; Roberts et al., 2007, 2010), suggest that these traits could be causal rather than consequential (though there is likely to be some degree of bi-directionality) (Kanakam & Treasure, 2013).

Most existing ED therapies do not directly address potential EF maintenance factors, and thus perhaps miss a vital treatment target. Moreover, deficits in EF may interfere with the ability to successfully engage in and benefit from behavioral treatment (Fowler et al., 2006; Lena & Fiocco, 2004; Svaldi et al., 2010). Experts in the field have recently called for the development of treatments that directly target relevant maintenance factors (such as EF) that are not addressed in CBT, with the hope of improving treatment outcomes (Wonderlich, 2013). Neurocognitive training, described below, is one possible adjunctive treatment method to target EF, which has the potential to enhance outcomes for EDs. The degree to which traditional therapies such as CBT affect EF is unknown, although this is an area ripe for future investigation. It should be noted that other treatment approaches, such as repetitive Transcranial Magnetic Stimulation (rTMS) and direct current stimulation (Grall-Bronnec & Sauvaget, 2014), can target brain regions subserving EF (e.g., dorsolateral prefrontal cortex) and may be viable methods for targeting EF deficits. These interventions represent potential treatments for EDs, but are somewhat different from the training approaches described in the remainder of the article in that they noninvasively deliver stimulation to small regions of the brain and cannot be defined as training approaches. For the remainder of the paper we focus specifically on neurocognitive training paradigms, although additional research on alternative approaches for improving EF in EDs may suggest other viable options.

Neurocognitive training for related conditions

A growing body of work has demonstrated that psychiatric illness is associated with dysfunction across prefrontal, fronto-limbic, and fronto-striatal neural systems (Vinogradov, Fisher, & de Villers-Sidani, 2011). These systems are associated with a diverse range of cognitive functioning including perception, cognition, social interactions, emotion regulation, and motivation (Eisenberg & Berman, 2010; Hartley & Phelps, 2010; Koob & Volkow, 2010; Price & Drevets, 2010; Vinogradov, Fisher, & Nagarajan, 2013). The plasticity observed in neural circuitry across the lifespan, combined with alterations in neural functioning associated with an individual's specific learning history, supports the assumption that brain functions can be trained (Vinogradov et al., 2013). The associations among dysfunction in neural systems, deficits in neurocognition, maladaptive behaviors, and cognitions have prompted researchers to examine whether interventions that improve neural functioning can result in improvements in psychiatric symptoms.

Although preliminary, initial reviews suggest that neurocognitive training can produce alterations in brain regions, neural circuitry, and behaviors, at least for behaviors similar to the training paradigm and potentially to more extended real-world behaviors (Vinogradov et al., 2013). Cognitive remediation emerged as a possible adjunct to standard treatments for schizophrenia over 50 years ago, and reviews assessing a wide variety of rehabilitation approaches have documented a moderate effect size of $d = 0.41$ for cognitive improvement and $d = 0.36$ for functional outcome, providing support for the efficacy of this approach in psychiatric populations (McGurk et al., 2007). Despite a strong rationale for investigating neurocognitive training in other disorders with observed neuropsychological deficits, neurocognitive training has only recently been investigated in disorders beyond schizophrenia. At this time, the only existing *direct* EF training approach that has been tested with EDs is Cognitive Remediation Therapy (CRT).

Cognitive remediation therapy for AN

At this time, most research on CRT has focused solely on AN, which is briefly discussed below.

Description of CRT

CRT is not designed as a stand-alone treatment for AN (i.e., its aim is not on weight gain or improving disordered eating cognitions); rather, it is designed as an adjunctive treatment, with the aim of (1) encouraging retention in targeted AN treatment by adding an engaging and interactive therapy component, and (2) decreasing cognitive rigidity in the hope of facilitating better utilization of skills provided in traditional therapies. CRT is an in-person intervention that aims to increase cognitive flexibility by identifying problems with inflexible thinking and practicing tasks meant to increase cognitive and behavioral flexibility (Easter & Tchanturia, 2011). With the exception of one study that examined CRT in the form of 21 sessions of computerized set-shifting trainings in conjunction with nine in-person sessions (Brockmeyer et al., 2014), CRT has typically been delivered for 8–10 weekly in-person sessions, and is conducted either individually or in group format. ED-related behaviors are not directly discussed, as the content of the group focuses on cognitive process and not on the specific content of cognitions. (The format and content of CRT has been comprehensively reviewed elsewhere; cf., Tchanturia, 2014.)