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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.

Keywords

Neurocognitive training; Inhibitory control training; Eating disorders

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

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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 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.)

Preliminary data for CRT

At this time, 14 studies have investigated the use of CRT for the treatment of AN (see reviews by Tchanturia and colleagues; Tchanturia, Lloyd, & Lang, 2013; Tchanturia, Lounes, & Holttum, 2014). Overall, CRT has demonstrated acceptability and feasibility for use in both inpatient and outpatient settings (Easter & Tchanturia, 2011; Tchanturia et al., 2013) and dropout rates range from 10 to 15%, as compared to approximately 60% for most treatment trials for AN (Tchanturia et al., 2013). Although results are mixed, several studies have revealed medium-to-large effect sizes in improvements in cognitive flexibility from

pre- to post-treatment (Brockmeyer et al., 2014; Lock et al., 2013; Tchanturia et al., 2013). However, there is minimal support for the long-term transfer of such improvements in cognitive flexibility to symptom improvement (Lock et al., 2013). In addition, formal assessment of the putative mediators (set-shifting and central coherence) of CRT has yet to be formally examined.

Limitations of existing research on CRT

Although this research provides initial promise for CRT as an acceptable treatment for AN, several limitations exist in the current literature. First, there is a notable lack of a control group in most studies, which raises concerns regarding confounds (e.g., expectancy effects, regression to the mean) in addition to the critical question of whether improvements in cognitive flexibility may be attributable to practice effects (both from baseline assessment and tasks throughout the course of CRT) rather than true improvements in cognitive flexibility. Also, because most studies of CRT have been conducted in an inpatient setting, it is possible that improvements in cognitive flexibility could be attributable to weight gain achieved through treatment, as starvation/malnourishment can cause impairments in brain function and neuropsychological performance (McCormick et al., 2008). One study of the existing 14 on CRT included computerized set-shifting trainings as part of the CRT training program (Brockmeyer et al., 2014), but otherwise the adaptations on the traditional in-person format and frequency of CRT have been minimal.

Also unknown is whether improvements in cognitive flexibility achieved through CRT persists over time. Some of the initial work has failed to demonstrate long-term improvements in neurocognitive performance (Tchanturia et al., 2013). Additionally, further investigation is needed to determine how best to integrate CRT with other interventions and to understand whether integration produces a synergistic effect. Finally, research on CRT for ED has generally been limited to AN samples, with little work having been conducted with BN and BED.

Extending neurocognitive training paradigms for treatment of conditions related to ED

Although CRT shows promise as an adjunctive treatment for ED, the current paradigm targets only cognitive flexibility. As we have discussed, EF deficits in EDs also include poor inhibitory control and working memory. Training in these domains has shown promise in the treatment of other psychiatric disorders (Kurtz et al., 2007) and in populations who experience subclinical eating problems (Houben & Jansen, 2011; Veling, Aarts, & Papies, 2011). Of note, most of the programs described below are different from most investigations of CRT in that that they involve automated, computer-based, high-frequency practice sessions. These trainings require repeated and rapid practice of tasks that target underlying implicit processes involved in executive control. Extensive evidence exists to indicate that intensive and frequent drill-and-practice sessions (delivered by a computer) are the most effective at improving deficits, particularly when administered for several weeks (Vinogradov et al., 2011). To date, computer-assisted cognitive remediation treatments have yielded little clinical or functional benefit to patients without concurrent functional or

behavioral skills training (Bowie et al., 2012; Spierer, Chavan, & Manuel, 2013). However, based on two meta-analyses of general cognitive remediation training for schizophrenia (which included studies designed to address multiple deficits and computer-assisted training studies), computer-based training can augment the effects of behavioral therapy when the two modalities are administered concurrently (McGurk et al., 2007; Wykes et al., 2011). Based on these results, and the overlap in types of EF deficits targeted in the training programs described above, we propose computer-based neurocognitive training as an adjunctive therapy to enhance existing evidence-based treatments.

CRT for EDs primarily targets explicit decision-making skills through verbal discussion of these processes with a trained therapist, and fails to reach the frequency or intensity of practice that may be required for long-lasting changes in EF. Computer-based training can be administered at high frequencies at very little cost, and moreover can be delivered in the home. Both strength and duration of improvements in neurocognitive deficits have been shown to be associated with training session dosage (McGurk et al., 2007), suggesting the potential for computer-based neurocognitive training for EDs. Moreover, the low cost of delivery and automated format of delivery make this type of treatment highly disseminable. The potential applications of computerized cognitive training for other psychiatric disorders to EDs are described below.

Cognitive flexibility training

Implementation of computerized training of cognitive flexibility may help to address the limitations of current CRT paradigms for EDs. These trainings would provide direct, implicit training of cognitive flexibility, and may yield greater improvements than explicit, discussion-based training. In addition, improvements from these computerized sessions may have a “bottom-up” effect and improve patients' ability to engage with treatment and reduce symptomatic behavior during treatment. For example, in AN, increased flexibility allows patients to generate alternatives to rigid food and exercise rules, thereby improving one's ability to comply with treatment recommendations for more flexible and varied eating patterns.

Computerized cognitive training has shown promise in the treatment of other psychiatric disorders that are characterized by impaired cognitive flexibility, including major depressive disorder, schizophrenia, and substance use disorders. Among patients with depression, computerized cognitive flexibility training (with no therapist intervention) yielded improvements in cognitive flexibility which also generalized to other EF domains (Elgamal et al., 2007). However, the training failed to yield improvements in depression symptoms, and other results suggest that clinical benefits of such a training can only be observed when administered in conjunction with psychotherapy (Bowie et al., 2013). Several studies have demonstrated that high-frequency, computerized cognitive training ameliorates deficits in cognitive flexibility among those with schizophrenia (Bellucci, Glaberman, & Haslam, 2003; Dickinson et al., 2010; Urben et al., 2012). Results on generalization of cognitive gains to clinical and psychosocial improvements are mixed (Cavallaro et al., 2009; Urben et al., 2012). Indeed, most studies that yielded improved behavioral outcomes were confounded by the inclusion of psychotherapeutic intervention, which included practice of specific

behavioral strategies to improve psychosocial functioning (Bowie & Gupta, 2013; Wykes et al., 2011). Among substance abuse patients, two studies have shown that computer-assisted cognitive training (which included a cognitive flexibility component) enhanced engagement in treatment and subsequent abstinence rates (Fals-Stewart & Lam, 2010; Grohman & Fals-Stewart, 2003). Overall, in conjunction with one study that has used a computerized CRT training for AN (Brockmeyer et al., 2014), these results suggest that computerized training has the potential to address deficits in cognitive flexibility among EDs. The extent to which neurocognitive gains may augment the efficacy of standard behavioral treatment is still debated, but it is likely that a combination of computer training and clinician-guided therapy may have the most benefit.

Inhibitory control training

Inhibitory control training (ICT), also known as response inhibition training, has been used to reduce a range of problematic and impulsive behaviors (Houben & Jansen, 2011; Houben et al., 2011; Jones & Field, 2013) among non-clinical samples, but the most relevant successes have been those that reduce disinhibited eating. Food-cue-specific ICT paradigms have shown preliminary efficacy at reducing impulsive responses to visual cues for highly palatable foods (Houben, 2011; Houben, Wiers, & Jansen, 2011). Training also generalizes to reduced consumption of the trained stimulus, such that certain “trigger” items (e.g., candy, potato chips) can be targeted for inhibition and reduced consumption (Veling et al., 2011; Veling, Aarts, & Stroebe, 2013). However, caution must be used when interpreting these preliminary results; the majority of ICT studies for eating behavior have included only non-clinical samples, and are limited methodologically. For example, Houben and colleagues used a paradigm in which the control group received an intervention which appeared to actually train subjects to be more impulsive (Houben, 2011; Houben & Jansen, 2011), and the efficacy of the ICT intervention may have been therefore exaggerated in their study results. Indeed, two other studies have found no transfer of cognitive gains to improvements in eating behavior (Guerrieri, Nederkoorn, & Jansen, 2007, 2012). Recent studies have improved upon these initial paradigms with better-controlled studies which compared effects of cue-specific and/or general inhibition training to more valid control conditions (Jones & Field, 2013; Veling et al., 2013). Cue-specific ICT has been found to facilitate weight loss (Allom & Mullan, 2015; Veling et al., 2014), and has also been shown to reduce laboratory selection (Koningsbruggen et al., 2014; Veling et al., 2013) and consumption (Houben & Jansen, 2015) of palatable foods. However, only one of these studies included a follow-up period of at least one week, and results showed abatement of cognitive gains over time (Allom & Mullan, 2015). Therefore, the durability of these training effects has yet to be demonstrated. While IC training has shown preliminary promise in reducing impulsive eating behavior, evidence for treatment effects must be extended to clinical populations and improved to yield long-term influence on behavior.

The impulsive eating behavior addressed by these studies is similar in nature to the impulsive and disinhibited behavior that characterizes binge-eating-spectrum pathology (BN, BED, AN B/P), and an exploration of the utility of ICT as a treatment adjunct appears warranted. The stimulus-specific nature of ICT paradigms may allow for individualized training programs that include response inhibition training for an individual's most frequent

"trigger" foods that are typically eaten under such emotional circumstances. However, binge eating is more entrenched and emotionally complex than disinhibited eating experienced by dieters (Haedt-Matt & Keel, 2011; Munsch et al., 2012; Schag et al., 2013). Although food-cue-specific inhibitory control deficits among ED patients who engage in binge eat are well-documented (and are qualitatively similar to non-ED dieters' deficits), bulimic symptoms are also frequently tied to a negative affective state, rather than a simple desire for a "tempting" food (Haedt-Matt & Keel, 2011). Existing ICT paradigms therefore may require modification in order to address impulsive binge eating and purging behaviors, which often occur specifically under a negative affective state (Haedt-Matt & Keel, 2011). Of note, results from one study of ICT for alcohol consumption suggest that training may modify affective associations with the target stimulus (which would be binge foods for BN or BED) (Houben et al., 2011). Taken together, existing evidence suggests that exploration of the efficacy of ICT for binge-eating pathology (perhaps as an adjunctive therapy to CBT) is justified.

Working memory training

Improvements during treatment for EDs are associated with concurrent improvements in working memory (Lena & Fiocco, 2004; Moser et al., 2003), and it is likely that observed working memory deficits in ED patients interfere with progress in treatment. For example, individuals with poor working memory may experience greater preoccupation with thoughts about food, weight, and shape, which may result in greater difficulty in processing new and particularly complex information, such as that delivered during a psychotherapy session (Kemps et al., 2006).

Working memory capacity may be of particular importance for successful ED treatment, and has the potential to provide both direct and indirect benefits (Morrison & Chein, 2011). For example, an individual must engage in self-regulation in order to reduce the occurrence of symptomatic behavior. Basic working memory training could be provided in conjunction with a specific plan for reducing symptoms and normalizing eating behavior, and could directly enhance patients' ability to follow through with treatment guidelines (such as resisting the urge to binge, or following a clinician-prescribed dietary plan). Training may also support the processing of complex information (Carretti, Borella, & De Beni, 2007). Indirectly, improved ability to engage with treatment guidelines (in and out of therapy sessions) may result in improved self-efficacy and improve motivation for recovery.

Working memory trainings have been developed and tested for a range of psychopathology, with much of the extant work focused on targeting working memory deficits in children with ADHD (Klingberg, 2010). This work, much of which is conducted by Klingberg and colleagues, has demonstrated the short-term efficacy of computerized trainings in improving working memory capacity in those with ADHD (Klingberg, Forssberg, & Westerberg, 2002; Klingberg et al., 2005), and often, the transfer of effects to other domains, such as response inhibition (Klingberg et al., 2005). In addition, working training appears to yield increases in neural activity in prefrontal and parietal areas, providing evidence for training-induced plasticity in the neural regions subserving working memory (Olesen, Westerberg, & Klingberg, 2004). Growing empirical support exists for the utility of working memory

training in the treatment of other adult psychiatric disorders with executive deficits similar to those identified in EDs (Fisher et al., 2010; Morrison & Chein, 2011; Wolinsky et al., 2009), including schizophrenia. Similar to EDs, schizophrenia is a psychiatric disorder in which working memory deficits are well-established and known to interfere with treatment efficacy and interpersonal functioning in affected patients (Green et al., 2000). Some results suggest that working memory training may yield greater transfer effects and may promote generalization of skills and improved behavioral outcomes compared to other neurocognitive training paradigms, though the extent of far transfer from working memory training is still debated (Jaeggi et al., 2014). Computer-based working memory training for patients with schizophrenia has been shown to yield specific cognitive improvement (Fisher et al., 2010). As with CRT, cognitive gains appear to transfer to psychosocial functioning only with concurrent behavioral skills training (Medalia & Saperstein, 2013). Thus, computer training is likely best suited as an adjunct to CBT-E.

Working memory training has also been shown to reduce behavioral impulsivity during decision-making tasks in individuals with stimulant dependence, and to decrease alcohol consumption in problem drinkers at one-month follow-up (Bickel et al., 2011; Houben et al., 2011), but replication is required in order to determine whether these results generalize to substance abuse patient populations. Impulsivity is known to underlie several eating disorder behaviors, including binge eating and purging (Wonderlich, Connolly, & Stice, 2004), and there is also substantial co-morbidity among EDs and substance use (Dawe & Loxton, 2004). Therefore, effective training paradigms for substance use are also likely to be effective for EDs if adapted appropriately for AN, BN, and BED.

Future directions

The existing body of research suggests that impairments in EF, particularly in the domains of set-shifting, central coherence, inhibitory control, and working memory, are related to the development and maintenance of disordered eating behavior (Dickinson et al., 2010; Lopez et al., 2009). CRT shows initial promise for improving EF, though more research is needed to establish whether these programs result in symptom reduction or greater responsiveness to conventional behavioral treatment (McGurk et al., 2007; Vinogradov et al., 2011). Moreover, at this time, the putative mediators (set-shifting and central coherence) of CRT have yet to be formally examined. For example, Dingemans and colleagues (2014) reported significant improvements in quality of life and ED symptomology in the treatment as usual + CRT versus the treatment as usual group, but changes in neuropsychological functioning were equivalent between groups, indicating CRT may be operating through a mechanism other than improving cognitive flexibility. It may be that CRT affects other EF domains beyond set-shifting and central coherence, indicating that paradigms specifically targeting each deficit may not be necessary to improve treatment response. However, it is also possible that CRT may be operating through other non-specific therapy effects. Identifying the mechanisms of action in CRT will help to better understand how symptom improvements are effected as well as to refine the treatment to include those components that effect change.

Dingemans and colleagues also observed differences in response to CRT such that those with poorer set-shifting at baseline had greater quality of life outcomes in the CRT

condition. These results suggest that CRT, and perhaps neurocognitive training programs more generally, yields the most benefit for those with the most prominent deficits. These results also raise the intriguing possibility that strengthening executive capacities could benefit individuals more generally in their everyday lives. Future research should continue to identify who may potentially benefit most from adjunctive neurocognitive training programs, with particular foci on identifying baseline severity as a potential predictor of response and examination of overall quality of life as an outcome variable. Finally, though some work has begun to establish how changes in cognitive flexibility are related to underlying neurobiological mechanisms (Garrett et al., 2014), there is little known about how and whether CRT specifically alters these mechanisms. Evaluating neurobiological changes as a result of CRT treatment will help to evaluate the specific changes that then impact executive function. Additional future research directions specific to CRT have been published elsewhere, cf., Tchanturia (2014).

Although relatively minimal work has been conducted in the area of neurocognitive training for eating pathology beyond CRT, computer-based trainings have been used to train the same types of neurocognitive deficits in related conditions with similar symptom patterns (Dickinson et al., 2010; Wykes et al., 2011) and may similarly prove beneficial for eating pathology. More work is needed to understand whether these computer-based training programs similarly impact EF deficits in EDs, and whether changes are associated with symptom change. The only existing study utilizing computer-based model of neurocognitive training for EDs demonstrated promising findings for the utility of computer-based training (Brockmeyer et al., 2014). Given that computerized neurocognitive training paradigms are potentially more disseminable than in-person CRT, future research should continue to examine preliminary and/or analogue effects of computerized neurocognitive trainings on EF and symptom change in individuals with eating pathology.

Across neurocognitive training programs, there is minimal knowledge about how long and how often neurocognitive training should be administered to be most effective or how to best integrate programs into existing treatment. According to Vinogradov and colleagues (2011), trainings must occur on a daily basis for many weeks or months before neurocognitive changes occur to the extent that the changes will persist and transfer to behavior outside of the task being trained; however, what dose specifically is required remains unknown. For EDs in particular, the available literature to inform these types of decisions when designing a training program is sparse. Moreover, little is known about the long-term maintenance of skills acquired from neurocognitive training (McGurk et al., 2007; Vinogradov et al., 2011). Computerized neurocognitive training paradigms will not take the place of traditional behavioral treatments, but may serve as a valuable adjunctive intervention. Further research should examine the additive benefit of such paradigms to traditional psychosocial treatments, such as CBT or even psychopharmacological interventions.

In sum, in order to develop more effective training programs, additional research is needed to better understand (1) which neurocognitive deficits contribute to symptoms and are amenable to training programs, (2) which training programs promote maximal gains and generalization (and how behavioral skills training might maximize potential for clinical benefit), (3) the intensity and duration of training that are necessary to observe improvement,

and (4) the potential utility of training paradigms that allow patients to engage in more frequent and consistent training (e.g. at home computerized training programs). Lastly, we need additional research on how best to incorporate cognitive training programs into existing psychosocial treatments to promote long-term behavior change. Without additional research in these domains, the creation of empirically-based neurocognitive training programs that can promote long-term reductions in disordered eating symptoms will be limited. It is our hope that the successes of neurocognitive training programs within other psychiatric disorders and the strong rationale for incorporating these techniques into treatments for eating disorders will spur others to continue work in this promising area.

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