The Potential of Neurofeedback in the Treatment of Eating Disorders: A Review of the Literature

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Abstract

Neurofeedback is defined as the training of voluntary regulation of localised neural activity using real-time feedback through a brain-computer interface. It has shown initial success as a potential clinical treatment tool in proof of concept studies, but has yet to be evaluated with respect to eating disorders. This paper (i) provides a brief overview of the current status of eating disorder treatments; (ii) describes the studies to date that use neurofeedback involving electroencephalography, real-time functional magnetic resonance imaging or near-infrared spectroscopy; and (iii) considers the potential of these technologies as treatments for eating disorders. Copyright © 2013 John Wiley & Sons, Ltd and Eating Disorders Association.

Keywords

neurofeedback; eating disorders; real-time; neuroimaging; treatment

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Introduction

Eating disorders are typically treated with psychological therapies. However, success rates are modest, particularly for adults with anorexia nervosa (AN). Medication (chiefly antidepressants) has shown some benefit in the treatment of bulimia nervosa (BN) (Shapiro et al., 2007), but little to no benefit has been seen in patients with AN (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). As a consequence, other therapeutic approaches such as those based on neuromodulation have emerged [e.g. attention bias modification treatment (ABMT), transcranial magnetic stimulation (TMS), deep brain stimulation (DBS), transcranial direct current stimulation (TDCS)], which have shown promising results thus far (Renwick, Campbell, & Schmidt, 2013; Van den Eynde & Guillaume, 2013). Although the latter three methods involve electrical stimulation, DBS also requires surgical insertion of an electrode. Although these methods are generally well tolerated, treatments that incorporate elements of both cognitive and neuromodulatory therapies may encapsulate the benefits of both approaches.

Feedback is an essential component of mental health interventions: it is used to increase motivation, facilitate learning and modify thoughts and behaviour (Musiat, Hoffmann, & Schmidt, 2012). Biofeedback describes an intervention that helps the individual to gain voluntary control over physical processes by providing immediate feedback on these or related processes (Nestoriuc & Martin, 2007). Typically, feedback is provided on heart rate, skin conductance, respiratory rate or muscle tension. Biofeedback is used in the treatment of pain (Bergeron et al., 2001), migraine (Nestoriuc & Martin, 2007) or incontinence (Norton, Chelvanayagam, Wilson-Barnett, Redfern, & Kamm, 2003). To date, there are a limited number of studies on the use/value of biofeedback in the treatment of eating disorders. Meule, Freund, Skirde, Vögele and Kübler (2012) used heart rate variability biofeedback in individuals with high levels of food craving and reported reduced craving despite a lack of changes in cardiac control. A gaming intervention for eating disorders, which includes a biofeedback component, is currently being evaluated (Fernandez-Aranda et al., 2012).

Neurofeedback

Neurofeedback is a form of biofeedback that trains individuals to voluntarily regulate their brain activity in response to real-time feedback (Weiskopf, 2012). The level of neural activity in a target area is fed back to the individual using a brain-computer interface, and this provides continuously updated information about their success in regulating their neural activity. Although conventional functional neuroimaging studies examine the relationship between behaviour and neural activity, neurofeedback provides a means of assessing neural response as an independent variable and evaluating its effect on behaviour (Sitaram et al., 2008). As such, it can be used as a means of bridging the gap between psychotherapeutic approaches, which combine approaches focused on altering cognitions to facilitate behaviour change, and neuromodulation that modulates brain activity by using external invasive or non-invasive procedures or equipment (Sitaram et al., 2007). It also provides an opportunity to improve understanding of the neuroscience underlying psychopathology, including that underlying eating disorders.
Neurofeedback trains individuals to directly modulate their own brain activity in a target region voluntarily, testing and adapting strategies by using the feedback provided as an indicator of success (Etkin, 2012). As such, it provides positive reinforcement and can promote feelings of self-efficacy, which may act as an incentive for poorly motivated patients (Linden et al., 2012). This is highly relevant to eating disorders, where patients are often ambivalent about change and where the issue of being in control is of key psychological importance (Fairburn & Harrison, 2003).

To our knowledge, no neurofeedback paradigm has yet been tested in patients with eating disorders. However, to identify the potential use of neurofeedback in eating disorders, we have considered neuroimaging data from patients with eating disorders, together with the results of studies on the use neurofeedback in patients with psychiatric problems that share features with eating disorders. The following section will consider three neuroimaging modalities that are being investigated both in clinical and healthy populations, relating neurofeedback findings using these modalities to the eating disorder literature to evaluate each modality’s potential as a treatment tool in eating disorders.

**Neuroimaging modalities for neurofeedback**

**Electroencephalography**

In electroencephalography (EEG) research, two measures of neural activity are used: event-related potentials and oscillations. Event-related potentials (ERP) are obtained by averaging the EEG signal after an event, that is, the presentation of stimuli, over a large number of trials to remove artefacts and noise (Key, Dove, & Maguire, 2005). This averaging of multiple trials reduces the suitability of ERP for neurofeedback, because immediate feedback cannot be provided. However, Makeig et al. (2002) argue that when analysed individually, ERPs reveal greater information about cortical dynamics. Oscillations form the second EEG measure and are comprised of the spectral content of the EEG signal. To obtain this information, raw EEG data are analysed using a Fast Fourier Transformation to identify the magnitude of neural activity in different frequency bands, such as delta (1-3 Hz), theta (4-7 Hz), alpha (8-12 Hz) or beta (13-30 Hz) (Klimesch, 1999). This transformation can be performed in real-time, thus allowing for oscillations to be used in neurofeedback. EEG signals have excellent temporal resolution, but poor spatial resolution, even when obtained with a high number of channels, and hence, the interpretation of oscillations is limited to its use as general indicators of cognitive performance. For example, increased alpha frequency indicates higher information processing speed (Klimesch, 1999), theta activity is associated with working memory functions and with cognitive control (Klimesch, Schack, & Sauseng, 2005).

**Possible use of electroencephalography feedback in eating disorders**

Most of the EEG research in eating disorders has focused on ERPs or sleep EEG (for a review, see Jauregui-Lobera, 2012). With regard to oscillations, patients with AN are reported to show asymmetry in cortical activation during resting EEG (Casper & Heller, 1991; Silva, Pizzarelli, Larson, Jackson, & Davidson, 2002); however, this asymmetry only partially disappears after weight gain (Grunwald, Weiss, Assmann, & Ettrich, 2004). In addition, patients with AN show reduced frontal activity in the alpha and increased frontal activity in the beta band when underweight (Hatch et al., 2011). In patients with BN, no consistent EEG patterns have been reported (Mitchell, Hosfield, & Pyle, 1983; Pope et al., 1989).

Outside the field of eating disorders, EEG neurofeedback has been extensively studied in attention deficit hyperactivity disorder (ADHD), which is characterised by inattention and impulsivity (Gevensleben et al., 2013). Most commonly, a theta/beta training paradigm is used in neurofeedback for ADHD (Arns, De Ridder, Strehl, Breiter, & Coenen, 2009). It is assumed that in the resting EEG, increased slow wave activity in the theta band and/or reduced wave activity in the alpha and beta band is associated with ADHD. This activity ratio between theta and beta waves might reflect underarousal of the central nervous system (Barry, Clarke, & Johnstone, 2003). In interventions using this paradigm, the individual has to increase activity in the beta range as well as reduce activity in the theta range; therefore, achieving a focused though relaxed state of mind (Gevensleben et al., 2013). A meta-analysis investigating the efficacy of neurofeedback in ADHD identified ten prospective controlled studies (total N = 476), as well as four prospective pre-post studies and one retrospective pre-post design (total N = 718). In this meta-analysis, it was shown that the use of neurofeedback in ADHD has large effect sizes with regard to inattention and impulsivity (Arns et al., 2009). However, only four studies were randomised controlled studies, and not all studies had an active control condition. Recent research suggests EEG neurofeedback in ADHD produces significant and lasting improvements in cognitive and behavioural function that are equal to or of greater magnitude than medication (Meisel, Servera, Garcia-Banda, Cardo, & Moreno, 2013). Bulimic eating disorders (BN and BED) are commonly accompanied by increased impulsivity and inattention (Waxman, 2009) or fully expressed ADHD (Seitz et al., 2013). Patients with ADHD show increased sensitivity to a range of pleasurable stimuli, including food, and it can be argued that rash, reward-driven behaviours, that is, bingeing on highly palatable foods and inattention to long-term consequences (weight gain, negative impact on health) are a central element of the problem (American Psychiatric Association, 2000; Claes, Nederkoorn, Vandereycken, Guerrieri, & Vertommen, 2006; Garrido & Subira, 2013). This behavioural overlap between ADHD and bulimic disorders suggests there is potential for EEG-based neurofeedback in the treatment of at least bulimic eating disorders.

**Evaluation**

There is limited evidence for the efficacy of EEG neurofeedback in other disorders that often have aetiological overlaps with ED, such as depression, anxiety and substance misuse disorders. However, it has been suggested that an asymmetry in alpha activity between anterior left and anterior right brain regions is associated with a bias towards negative emotional stimuli (Rosenfeld, Cha, Blair, & Gotlib, 1995). In a case series using neurofeedback to decrease alpha asymmetry in combination with psychotherapy, patients reported lower depression and alpha asymmetry remained reduced in a long-term follow-up (Baehr, Rosenfeld, & Baehr, 2001). In a more controlled study, participants (N = 12) were randomly assigned to a neurofeedback condition or a mock control
condition (feedback does not reflect brain activity). Compared to controls, those receiving neurofeedback reported elevated mood and felt more energetic (Raymond, Varney, Parkinson, & Gruzelier, 2005). A review of EEG neurofeedback in anxiety disorders has suggested that it can effectively reduce anxiety over the course of the neurofeedback sessions, but emphasised that these effects might not go beyond a placebo effect (Moore, 2000). EEG neurofeedback has also been used in the treatment of patients with substance misuse disorders. In a narrative review of the evidence, Sokhadze, Cannon, and Trudeau (2008) revealed that although promising results were found in several uncontrolled studies using the alpha-theta paradigm, evidence from randomised controlled studies suggest that the effect of EEG neurofeedback is not larger than those of sham treatments or alternative less technically demanding treatments (e.g., meditation). However, it has to be noted that there is a lack of controlled and large-scale trials.

In summary, there is some evidence suggesting that EEG neurofeedback may be of use for the treatment of some eating disorders. Compared to many functional imaging paradigms, EEG is both easier and cheaper to use. In addition, wireless portable EEG devices are increasingly available, which would allow patients to use neurofeedback outside of a laboratory setting and at their own convenience. However, attention has to be paid to ensure that potential effects can be attributed to neurofeedback, by conducting trials which include appropriate control conditions (Egner, Strawson, & Gruzelier, 2002).

Real-time functional magnetic resonance imaging (rt-fMRI)

High spatial resolution of the whole brain can be achieved using functional magnetic resonance imaging (fMRI), unlike alternative modalities such as EEG and near-infrared spectroscopy (NIRS). This non-invasive technique makes use of the magnetic properties of blood to record changes in the ratio of oxygenated and deoxygenated haemoglobin, that is, the blood oxygen level dependent (BOLD) response, as an indirect measure of neural activity. It is based on the fact that in the brain, there is a direct relationship between neural activity and oxygen utilisation, that is, the BOLD response is haemodynamic rather than neural. Since the original development of rt-fMRI by Cox (Cox, Jesmanowicz, & Hyde, 1995), substantial developments in magnetic resonance imaging (MRI) technology, that is, in terms of data acquisition (notably the introduction of echo planar imaging), image processing algorithms and computer hardware, have permitted faster image acquisition and thus improved both sensitivity and speed of fMRI, generalising its applicability to real-time imaging (Sitaram et al., 2013a; Weiskopf, 2012).

Therapeutic applications of neurofeedback using real-time functional magnetic resonance imaging

Real-time fMRI neurofeedback is receiving growing support as a potential treatment modality. A number of reports have demonstrated the capability of healthy individuals to up-regulate or down-regulate neuronal activity in specified brain regions related to given tasks, including (but not limited to) the anterior insula (Veit et al., 2012), rostrolateral prefrontal cortex (Mccaig, Dixon, Keramatian, Liu, & Christoff, 2011), anterior cingulate cortex (Hamilton, Glover, Hsu, Johnson, & Gotlib, 2011) and the amygdala (Zotev et al., 2011). Recently, several proof of concept studies have been published reporting clinical improvement as a result of rt-fMRI neurofeedback in medical and psychiatric patient populations, including patients with Parkinson’s disease (Subramanian et al., 2011), schizophrenia (Ruiz et al., 2013b) and depression (Linden et al., 2012). For example, Linden et al. (2012) reported that patients with non-comorbid unipolar depression that received neurofeedback were able to upregulate activity in the ventrolateral prefrontal cortex and insula by generating positive thoughts. Significant improvements of clinical symptoms were observed in the patient group receiving neurofeedback, compared to a comparison patient group who engaged in the same cognitive strategies without neurofeedback: this suggests that clinical improvement was largely a consequence of the neurofeedback. Inclusion of an appropriate control or sham condition is essential for overcoming any potential confounds, which must be carefully addressed in randomised clinical trials. Providing pseudo-feedback for example, with the control group receiving feedback of the experimental group’s neural activity rather than their own (e.g. Hamilton et al., 2011), or their own feedback from an unrelated brain region (Zotev et al., 2011), may be more appropriate than simply including a group that do not receive any feedback, as providing pseudo-feedback will generate an environment and elicit cognitions or emotions that more closely resemble those in the experimental condition.

Although these findings are suggestive of rt-fMRI neurofeedback being an effective treatment tool, complex cognitions involve the coordination of a network of neural regions and not the activity of any single region in isolation. Researchers have thus begun to explore rt-fMRI neurofeedback from a network-based perspective. This is not only to assess the feasibility of training voluntary self-regulation of regional connectivity, but also to determine the nature and extent of the brain’s plasticity and the dynamic interactions between regions within a target network. Several studies have reported that neurofeedback training in a single region of interest (ROI) results in stronger BOLD signal correlations between the ROI and voxels in functionally-related regions (e.g. Haller et al., 2013; Horovitz, Berman, & Hallett, 2010); this suggests that neurofeedback involves changes in functional connectivity. For example, Lee et al. (2011) employed multivariate pattern analysis using a support vector machine model and Granger Causality Modelling to investigate dynamic changes in the spatial and temporal aspects, respectively. Specifically, they investigated the anterior insula’s connections with other regions involved in emotional processing over five neurofeedback sessions. Over the training sessions, they observed (i) a gradual reduction in the spatial distribution of activated areas; (ii) an initial increase and subsequent pruning of the number of connections between these regions; and (iii) a continuous increase in the strength of these connections. These studies provide important information on the network-wide impact of neurofeedback targeted at a single ROI. It is unclear, however, whether these findings are because of general improvements in the efficacy of activity in the ROIs or are because of a refinement in the cognitive strategies used during regulation. Moreover, these studies provide no indication of whether regional connectivity can be modulated directly (Ruiz, Buyukturkoglu, Rana, Birbaumer, & Sitaram, 2013a). Preliminary evidence of the feasibility of direct
Based neurofeedback training (for review, see Ruiz et al., 2013a) provided with rt-fMRI neurofeedback was shown to be effective in an automatic semantic priming task after learned self-regulation of the connection between the inferior frontal gyrus and the superior temporal gyrus, that is, regions of the frontotemporal network thought to underlie the effect.

The employment of multivariate techniques that allow incorporation and assessment of spatial and temporal relations between regions will vastly improve the ability to examine the effect of neurofeedback on neural networks and the efficacy of network-based neurofeedback training (for review, see Ruiz et al., 2013a). However, current techniques for pattern classification are highly specific to individual participants and require a large amount of preliminary data to train the classifier. The development of subject-independent pattern classifiers for each network will largely overcome this issue and greatly expand the applicability of network-based neurofeedback in rt-fMRI (Ruiz et al., 2013a).

**Possible neural targets for rt-fMRI neurofeedback in eating disorders**

Early studies of fMRI neurofeedback in eating disorders may wish to target neural regions that have been used as targets of other modulatory treatments in eating disorders. For instance, dorsal regions of the prefrontal cortex are often the main targets in trials of TMS (e.g. Broadbent et al., 2011; Uher et al., 2005; Van Den Eynde et al., 2010; Van Den Eynde, Guillaume, Broadbent, Campbell, & Schmidt, 2013), as dorsal circuits are thought to contribute to eating disorder pathology through altered interception or reward processing (Kaye, Fudge, & Paulus, 2009; Van den Eynde et al., 2013). Recovered AN patients show increased lateral prefrontal activity when processing visual food stimuli (Uher et al., 2003), suggesting a positive association between activity in this region and clinical outcome. Thus, dorsolateral regions of the prefrontal cortex can be considered appropriate initial targets for assessment of clinical efficacy of neurofeedback using rt-fMRI. The targets for TMS are restricted to cortical regions as the induced electromagnetic fields fade with distance from the coil (Fitzpatrick & Rothman, 2000). In contrast, the targets for DBS in eating disorders are often subcortical regions involved in the neurocircuitry underlying the processing of reward and anxiety, both of which are considered to be altered in eating disorders and largely contribute to the symptoms. Such targeted regions include the nucleus accumbens (Wu et al., 2012), the anterior cingulate cortex (ACC; Israel, Steiger, Kolivakis, McGregor, & Sadikot, 2010), the subcallosal cingulate (Lipsman et al., 2013), the internal capsule, and bed nucleus of the solitary tract (Barbier, Gabriëls, Van Laere, & Nuttin, 2011). The nucleus accumbens and other striatal regions are key regions in the dopaminergic reward pathway. Abnormalities in this pathway are thought to underlie dysfunctional reward processing in many psychiatric disorders, and thus may be interesting targets for regulation of reward perception (Sulzer et al., 2013b).

One popular direction of research is the relationship between eating disorders and addiction, with eating disorders and obesity being conceptualised in the framework of addiction models (Wilson, 2010). Addictions and eating disorders are often comorbid and share considerable overlap in behavioural and cognitive symptoms (Volkow & Wise, 2005; Wilson, 2010; Wolfe & Maisto, 2000). For example, dysfunctional reward processing can be considered a key maintaining and/or risk factor in both psychiatric conditions. Moreover, addiction and binge-related eating disorders are characterised by impulsivity, compulsivity and the experience of craving (Claes et al., 2006; Davis & Carter, 2009; Friederich, Wu, Simon, & Herzog, 2013). Both types of disorder have been associated with dysfunction in the ACC (e.g. Garavan & Stout, 2005; Schienle, Schafer, Hermann, & Vaitl, 2009), an area central to the initiation of prefrontally-mediated top-down control of behaviour (Garavan & Stout, 2005). This region plays an important role in reward-based decision making and in conflict and error monitoring (Garavan, Ross, Kaufman, & Stein, 2003). Dysfunction in this region may therefore contribute to the disturbed self-regulatory abilities characteristic of substance misuse and binge-eating related disorders (Marsh et al., 2009). Moreover, addiction research has implicated the ACC in the experience of craving, with reports of enhanced ACC activity during craving experiences (e.g. Bonson et al., 2002) and inhibition of activity in this area using TMS has been found to diminish craving-related neural activity (e.g. De Ridder, Vanneste, Kovacs, Sunaert, & Dom, 2011). A recent neurofeedback trial in treatment-seeking smokers revealed that downregulation of activity in the ventral ACC was correlated with reductions in self-reported craving, whereas attempts to increase activity in the prefrontal cortex, an area important for self-control and resisting urges (Koechlin, Ody, & Kouneiher, 2003), were less successful and were not associated with reduced craving (Hanlon et al., 2013). Heart rate variability biofeedback has shown success in reducing food craving in normal and overweight individuals who frequently experiencing strong cravings (Meule et al., 2012). The ACC may therefore be an important target for neurofeedback treatment trials for eating disorders associated with intense cravings for food. Thus, regions involved in reward and craving may constitute important targets in both addictions and eating disorders, with findings providing informative directions for future research in both disorders. Moreover, any differences or similarities in efficacy of neurofeedback between the two disorders will build on our current understanding of the overlap between them and the validity of a model of eating disorders, which includes an addictive component.

Eating disorders are often associated with high anxiety and with high comorbidity, and similarities to anxiety and mood disorders (e.g. Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Thus, brain regions involved in anxiety, stress or emotional regulation may be important targets for neurofeedback studies in eating disorders (Dallman, 2010; Oldershaw et al., 2011). In addition to craving, the ACC is thought to be involved in conflict monitoring and has been demonstrated to contribute to networks activated during affect regulation, alongside the insula, orbitofrontal cortex and lateral prefrontal regions (Veit et al., 2012). Dysfunction in the anterior insula is proposed to contribute to altered interoceptive awareness (Kaye et al., 2009). Regulation of the amygdala, a primary emotion processing centre (Zotev et al., 2011), the anterior insula (Veit et al., 2012) and specific regions of the lateral prefrontal cortex (McCaig et al., 2011) have been successfully accomplished using rt-fMRI neurofeedback in healthy individuals, with reports of clinical benefit associated with successful regulation in patient groups (e.g. Linden et al., 2012), suggesting these are viable targets for neurofeedback in eating disorders.
Evaluation

There is growing evidence for the efficacy of neurofeedback using rt-fMRI in other psychiatric disorders. Eating disorders, similar to other psychiatric problems, are likely to be associated with dysfunction in one or more neural circuits, rather than specific regions. Thus, the ability to image the whole brain at high spatial resolution in fMRI may be advantageous in assessing the efficacy of voluntary regulation in single or multiple localised regions, or the effect of regulation in one region on other areas within a functional neural circuit. However, compared to EEG and NIRS, the applicability of fMRI-based neurofeedback is limited as the scanners cannot be transported. The majority of the rt-fMRI neurofeedback research cited earlier employed either single session protocols (<1 h) involving multiple neurofeedback training runs of less than 10 min each (e.g. Veit et al., 2012; Zotev et al., 2011; ) or multi-visit protocols with involving a few (e.g. 4) short (~10 min) neurofeedback sessions at weekly or biweekly intervals (e.g. Haller et al., 2013; Linden et al., 2012). The high costs for MRI equipment and scanning time are an important consideration, as neurofeedback is likely to be applied clinically in the form of multiple scanning sessions, rather than one extended session. For these reasons, it is likely that neurofeedback in fMRI may be more suitable for use as an adjunct to psychotherapy rather than a replacement.

Near-infrared spectroscopy (NIRS)

Near-infrared spectroscopy is emerging as a suitable candidate for neurofeedback-based interventions. NIRS is an optical spectroscopy technique that makes use of the optimal absorption properties of haemoglobin, the major chromophore in biological tissue, to measure changes in concentration of oxy-haemoglobin and deoxy-haemoglobin to infer underlying physiologic change (Abdelnour & Huppert, 2009).

Unlike photons in the visible spectrum, photons in the NIRS (700-1300 nm) are not absorbed by skin and tissue, instead scatter in soft tissue and bone and are absorbed by chromophores (Macnab, 2009). Oxy-haemoglobin and deoxy-haemoglobin have different patterns of absorption, with optimal absorption of photons emitted at wavelengths of 830 nm and 780 nm, respectively. NIRS is conducted using a cap fitted with an array of fibre optic probes, half of which emit pulses of light at multiple wavelengths into the tissue, the other half serving as sensors that detect and absorb reflected light (Hespos, 2010). Changes in oxygenation of haemoglobin will affect the concentration of photons absorbed by chromophores, that is, chemical groups that absorb light of a particular frequency, and therefore the amount reflected back to the detector probes (Hespos, 2010). In a similar fashion to MRI, NIRS monitors the change in the oxygenation of haemoglobin to infer neural activity under the assumption of neurovascular coupling. Developments in the equipment, that is, increasing the number of channels in the system, and improvements in data processing and analysis methods permit more accurate topographical assessment of real-time regional haemodynamic change (Ehliis, Schneider, Dresler, & Fallgatter, 2013).

Possible neural targets in eating disorders

Although neurofeedback using NIRS has not yet been evaluated in eating disorders, this technique has shown initial success in improving motor function in stroke patients in a recent proof of concept study (Mihara et al., 2013). As there are no existing trials of NIRS neurofeedback in eating disorders, suggestions for neural targets and efficacy predictions can be made based on experimental NIRS research in this psychiatric group.

NIRS research in eating disorders is sparse, with the majority of studies revealing differences in task-related blood perfusion in frontal regions, including the prefrontal cortex, the orbitofrontal cortex and frontotemporal regions, in patients with eating disorders compared to healthy controls (Nagamitsu et al., 2011; Suda et al., 2010b; Sutoh et al., 2013; Uehara et al., 2007). Compared to healthy individuals, adult and adolescent patients with eating disorders show smaller task-related regional haemodynamic changes (e.g. Nagamitsu et al., 2011; Suda et al., 2010b; Sutoh et al., 2013; Uehara et al., 2007), which has been reported to correlate with behaviours relevant to eating disorders. For example, haemodynamic changes in right frontotemporal regions have been reported to negatively correlate with dieting tendency scores, whereas changes in the left orbitofrontal regions correlated with eating restriction and binge eating scores (Suda et al., 2010b). Moreover, disorder-specific changes in perfusion during tasks have been revealed, which relate with disorder-specific behaviours and personality traits. For example, in an examination of prefrontal activity related to self-regulation abilities, Sutoh et al. (2013) revealed that poor inhibition in BN patients was reflected in different patterns of change in oxy-haemoglobin compared to AN patients and healthy controls, who showed no difference in performance or perfusion during the tasks. The authors suggest that such haemodynamic changes in the BN group may reflect a greater effort being required for self-regulation in a group typically characterised as impulsive and lacking control, whereas the lack of haemodynamic change in the AN group may reflect perfectionist and controlling personality traits and behaviours (Sutoh et al., 2013). These studies suggest that the prefrontal cortex is a viable target for neurofeedback in eating disorders, and that the direction of regulation (i.e. up-regulation or down-regulation), and the targeted behavioural response is likely to differ in prefrontal regions between the different eating disorders.

Evaluation

As a result of improvements to NIRS equipment and data processing methods, NIRS is increasingly employed as an experimental tool and offers several advantages as a modality for implementing neurofeedback as a treatment. NIRS equipment is non-invasive, compact and easily portable, and thus can be applied in more naturalistic settings. Moreover, it is relatively insensitive to movement, thus providing an alternative for individuals characterised by excessive movement. As overactivity is a common feature of AN, this feature is an attractive quality of the technique. Moreover, NIRS has been successfully implemented during face-to-face interaction (Suda et al., 2010a), and thus can take place in a more intimate and naturalistic setting compared to neurofeedback using fMRI.

However, NIRS has restricted depth resolution (to only a few millimetres from the scalp), and limited spatial resolution because of the limited number of detector/emitter probes built into the equipment. As a result of this limited resolution, NIRS in adults can only provide information on regional haemodynamic change in cortical regions near the cerebral surface. As the brains of AN
patients are comprised of reduced total grey matter volume and increased cerebrospinal fluid volume, increases in scalp-to-cortex distance may pose an issue (Ehils et al., 2013; Van Den Eynde et al., 2012). Additionally, peripheral perfusion in the scalp (e.g. because of sympathetic arousal) may affect the reliability of NIRS readings. Investigations of the degree to which cortical atrophy and peripheral perfusion affects the sensitivity of NIRS are essential in the evaluation of this technique as both a treatment and research tool in eating disorders.

Other therapeutic applications of real-time imaging

Neurofeedback can be applied clinically both as a means of evaluating current methods of treatment as well as a treatment in itself. For example real-time imaging can be used to further knowledge of the neural effects of psychotherapy. Greater understanding of these effects can lead to improvements in decision making regarding prognosis, suitable pharmacotherapy and may constitute a more objective measure of treatment efficacy in randomised control trials (Linden, 2008). Early studies have revealed that rt-FMRI may be a beneficial adjunct to psychotherapy: patients may benefit from observing physiological changes during therapy, and clinicians may gain a better insight into the efficacy and suitability of treatments (Adcock, Lutomski, Mcleod, Soneji, & Gabrieli, 2005). However, the MR scanning environment may increase anxiety because of the loud noises and feeling of confinement in the scanners, the presence of other staff members (e.g. radiologists) and the reduced directness of the interaction with the therapist may limit the utility of neurofeedback using this procedure (deCharms, 2008). NIRS offers an attractive alternative in this respect; however, the resulting information will be limited to superficial cortical regions.

Overall evaluation of neuroimaging as a potential treatment tool

Initial proof of concept studies of neurofeedback as a clinical tool have yielded promising results; however, this methodology is in its infancy. Randomised blinded clinical trials will be required to evaluate the added value of neurofeedback in the treatment of eating disorders before it can be implemented in clinics. With the inclusion of an appropriate control condition, neurofeedback can be well controlled and standardised, and may not require the presence of a therapist. However, further work will be required to delineate the optimal training approach, for example, with respect to the presentation of information, regulation strategies employed, session length and control condition (for review of issues in fMRI-based neurofeedback, see Sulzer et al., 2013a). These factors must be thoroughly assessed before the expected costs can be calculated and considered in the overall evaluation of neurofeedback as a treatment option.

Research on neurofeedback suggests the technique has a number of advantages. It is non-invasive and combines the treatment principles of cognitive and biological treatments currently available in a novel approach. Neuroimaging is associated with few side effects, although the modality-specific precautions must be assessed. The feedback is interesting and informative to the patient, and success in regulation indicated by feedback can promote self-efficacy and positive reinforcement. This not only encourages positive self-evaluation but also may be a motivational incentive for continuing to engage in treatment. Moreover, initial proof of concept trials have reported transfer of regulation abilities and behavioural effects to later sessions in which online feedback is not provided. This suggests that the clinical benefits associated with learned voluntary neural regulation may outlast the neurofeedback sessions, offering potential for therapy (Zotev et al., 2011). Additional practice outside of the scanner may facilitate consolidation of this skill (Linden et al., 2012). However, more research is needed to evaluate the extent to which observed clinical benefits persist in the longer term.

Conclusion

Through repeated training, individuals can learn to voluntarily regulate localised brain activity, which can consequently influence cognition and behaviour. Early research suggests that neurofeedback may have a unique place in the treatment of psychiatric illness, and may be a useful adjunct to treatment in the clinical management of eating disorders. Early proof of concept studies in eating disorder populations, followed by blinded randomised control trials, are essential in evaluating neurofeedback using various imaging modalities and their respective clinical utility in the treatment of this patient population.

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