Recent advances and future directions in the management of eating disorders

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Abstract

The aim of this paper is to describe recent and future advances in eating disorder treatment. The development of new treatments represents a pressing issue, given that current treatments, which are predominantly forms of psychotherapy or inpatient treatment (for those at high medical risk), have variable and limited efficacy, with short-term recovery levels between 30% to 50% for the first stage of treatment. If initial treatment efforts fail, there is uncertainty about what strategy should be next considered. Developments in aetiological understanding, through defining biomarkers, working with animal models and neurobiological investigations, have encouraged the consideration of a broader range of treatments. Brain-directed strategies such as neurostimulation, exposure-based learning, psychedelics, metabolically targeted treatments including leptin and incretin receptor agonists, and the therapeutic use of the arts have been recently investigated. New technologies such as virtual reality–based and brain training–based interventions have been found to enhance standard forms of treatment. Unfortunately, the limited funding for eating disorder research has meant that advances into clinical practice have been limited. However, given the size of the problem and its protracted nature, it is important that work to progress and evaluate new treatment pathways, such as that in depression or schizophrenia, is prioritised.

1. Introduction

Progress in eating disorder research has been hampered by the poor level of funding for research and service development in this area. Treatment guidelines for eating disorders have remained relatively unchanged over the last 10 to 20 years, recommending various forms of psychotherapy that produce moderate effects. Involving the family in the treatment of anorexia nervosa (AN) produces larger effects, especially in adolescence (1).

Recent developments in our understanding of the aetiology of eating disorders have inspired new approaches. It should be acknowledged that family members of people with eating disorders have played an important role in this change of focus by supporting and/or founding charities that funded the acquisition of the large cohorts of people with AN that are needed for genetic studies. These studies have led to the discovery that both neural and metabolic systems are associated with the risk of developing AN (2, 3), shifting the emphasis from social onto biological causes. Collaborative efforts between research centres have enabled the impact of AN on brain structure and function to be more clearly defined. For example, the ENIGMA consortium has reported on the profound impact of starvation on brain structure and function, whereby AN, compared to other psychiatric disorders, has the greatest loss in cortical thickness (4). This has led to research aimed at improving neuroplastic processes (5). This paper aims to provide an overview of some these new approaches.

1.1 The varieties of eating disorders

The standard varieties of eating disorders defined in the DSM-5 include AN, bulimia nervosa (BN), binge eating disorder (BED), avoidant/restrictive food intake disorder (ARFID), pica and rumination disorder. The literature relating to the latter three conditions is limited and will not be considered in this paper.

1.2 Models of eating disorders

Table 1 outlines some of the factors thought to be associated with the predisposition, precipitation and perpetuation of eating disorders. Theoretically, treatment should target these factors, but there are gaps in the status of this knowledge. Although there was an initial emphasis on transdiagnostic processes, some of the biological aspects diverge. Most of the information on environmental or developmental predisposing factors is sourced from longitudinal studies and relates to binge-spectrum disorders, given their higher prevalence. In contrast, clinical studies and research into biomarkers focuses mainly on AN, often within the inpatient context.

1.3 Predisposing factors

The genetic risk profile for eating disorders is broadly similar to many other psychiatric disorders, although the profile for AN has most overlap with that of obsessive-compulsive disorder (3), and the profile for binge-spectrum disorders overlaps with addictions and attention deficit hyperactivity disorder (2). Interestingly, the metabolic genetic risk profile differs widely across the eating disorder spectrum. The profile for binge-spectrum disorders is associated with

	AN	BN	BED
Predisposing traits	Compulsivity	Impulsivity	Impulsivity
	Sensitivity to punishment	Sensitivity to reward	Sensitivity to reward
	Satiation sensitivity	-	Appetite sensitivity
Environment	Trauma	Trauma++	Trauma++
	Social comparison	Social comparison	Social comparison
Trigger	Negative energy balance	Negative energy balance	Negative energy balance
Perpetuating	↓ Brain volume	-	Addictive process:
	↓ Social cognition		↑ Craving
	↓ Memory		↓ Reward
	Psychosexual regression - isolation	-	Self-disgust, shame
	Stress, anxiety, treatment-resistant depression	Stress, anxiety, treatment-resistant depression	Stress, anxiety, treatment-resistant depression

Table 1. Models of eating disorders: factors that contribute to predisposition, precipitation and perpetuation
Abbreviations: AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder

higher waist and hip circumference, being overweight, obesity and extreme body mass index (BMI) (2), whereas the profile of AN is in the opposite direction (negative relationship with body size, obesity, BMI, insulin resistance, fasting insulin and leptin) (3). Another contrast is that binge-spectrum disorders have increased sensitivity to reward, whereas AN is also associated with sensitivity to punishment (6, 7). These temperamental traits and behavioural styles shape the background predispositions to the form that an eating disorder might take.

1.4 Precipitating and perpetuating factors

Trends of eating disorders over time have shown a steady increase in binge-spectrum disorders from 1979, following Russell's seminal paper (8), whereas the incidence of AN has remained roughly steady, apart from an increase in people aged 10 to 14 years (9). However, since the COVID-19 pandemic, the incidence of severe cases of AN has increased (10). The time course of the increase in binge-spectrum disorders parallels the increase in obesity in many populations. Changes in diet (particularly associated with the hyperabundance of ultra-processed foods), eating behaviour, food poverty and weight stigma have been implicated in all weight and eating conditions. These specific environmental contexts combine with other forms of marginalisation or trauma, as well as genetic predisposition, to increase the risk of developing an eating disorder.

Environmental background adversity is less pronounced with AN; however, the prolonged starvation that follows from the onset of the restricted eating leads to medical instability, with secondary effects that impact on brain structure and function. These consequences can impact on psychosexual development and lead to problems in social cognition and memory, which may impact on the ability to develop a recovery identity (11). These brain changes and the protracted course of AN can lead to social alienation, stress and the development of a form of depression or anhedonia that is resistant to standard pharmacological treatments.

2. First-line treatments

For binge-spectrum disorders (BN and BED) and AN, first-line treatments consist of cognitive behavioural therapy (CBT)-based approaches adapted for eating disorders. Involving the family in treatment at an age-appropriate level is particularly important for AN (1). Additionally, inpatient, day patient or home-based treatment strategies are used if the physical state is compromised (12).

Treatments for AN that focus on the underlying predisposing traits and the secondary impact of starvation include the Maudsley Model of Anorexia Nervosa Treatment for Adults (MANTRA) (13, 14) and temperament-based therapy with support (15).

Approximately one-third of adult cases of AN do not respond to these first-line approaches. New approaches to treatment are being considered for these groups with a longstanding form of illness, who often have additional comorbidities, and develop medical, psychological and social consequences from protracted ill health (16).

3. Second-line treatments

A variety of new approaches have been introduced to target some of the emotional, social and cognitive features of eating disorders that are associated with a poor response to first-line treatments. For example, new strategies to augment treatment for binge-spectrum disorders, focusing on moderating impulsivity, have been introduced, whereas a focus on compulsive patterns of behaviour have directed new approaches for AN. These potential new second-line treatments are discussed in the following sections.

3.1 New psychotherapeutic and cognitive strategies

3.1.1 Externalising strategies

One of the unique and puzzling aspects of AN is that many patients do not consider themselves to have a problem. Indeed, they may value aspects of their eating disorder. This can cause disruptions in their relationships with supporters (professionals and family), who are often extremely concerned, and can lead to delays in seeking treatment and decreased motivation to engage in treatment.

Many psychological therapies for AN, such as family-based therapy and MANTRA, use externalisation strategies to modify this ambivalence about recovery. Externalising refers to the process whereby the individual is guided, through various methods, to re-perceive their illness as separate to, rather than a part of, the self. Art therapies, often with the use of forms of transitional objects (letters, photographs, playlists, toys etc.), are used to externalise the illness and may be useful in individual or group approaches (17).

New techniques to augment externalisation include dialogue therapy, which uses chair work, where the patient converses with different aspects of the self, or the self in different stages of recovery, to explore ambivalence (18). Avatar treatment (developed for the treatment of schizophrenia) is a similar approach in which the therapist speaks as the eating disorder through a digitally constructed bespoke avatar and coaches the patient to talk back to the AN voice (19, 20). A small feasibility study using this avatar-based approach for eight sessions in 10 patients with AN found lower levels of distress associated with the eating disorder voice and higher levels of self-compassion. Feedback from patients was that avatar therapy had helped with their ability to stand up to the illness, make positive changes around eating and increase their motivation to recover (20). Additionally, a study of computer-assisted avatar-based treatment in subclinical eating disorder cases (n = 48) found a reduction in eating disorder–specific symptomatology (21).

3.1.2 Exposure-based treatments

Exposure-based treatments involve engagement with, rather than the avoidance of, emotionally laden triggering situations relating to food, body image and social encounters, which are common sources of fear and distress in AN.

Supported eating is a key component of the higher levels of care for AN (22, 23) but is less frequently used as a part of outpatient care (24). However, creative use of virtual exposure has offered the possibility of using new strategies to combat fear and avoidance in the outpatient setting. Virtual reality (VR) can facilitate novel and personalised exposure experiences and encompass a range of engagement with a wide variety of food-related (25-27), body-image and socially threatening stimuli (28, 29), in a variety of contexts.

Exposure treatment involves a process of reversing fear by developing inhibitory learning (30). The feared outcome from eating in eating disorders is usually an expected future consequence, such as becoming "fat", and facing the associated social stigma. This cannot be disconfirmed in the short term. However, approaches such as imaginal exposure, which target these future expectancies, has shown good effects when used in a transdiagnostic sample (31, 32). Body image problems can also be addressed in VR with the use of avatars that allow users to experience different body perspectives (33, 34). A meta-analysis of VR techniques for people with binge-purging eating disorders found reductions in binge eating and body image problems (35). Moreover, a promising study found that VR-based body exposure therapy was an effective second-line treatment for those with BN and BED who had a poor outcome with CBT (36).

3.2 Brain stimulation techniques

A variety of new techniques have been developed to stimulate specific areas of the brain, both invasively, with intracranial electrodes, or through using non-invasive brain stimulation, such as magnetic or direct-current stimulation (37).

3.2.1 Transcranial magnetic stimulation (TMS)

TMS is a brain stimulation technique that is modulated over a specific area of the brain to enhance or inhibit electrical activity by increasing or decreasing the excitability of targeted neurons (38). Pulsing patterns of TMS are varied depending on the desired outcome, ranging from single pulses that last milliseconds to repeated trains lasting minutes (39).

TMS to the dorsolateral prefrontal cortex (dlPFC) has been applied to participants with a wide variety of behavioural traits that are relevant for people with eating disorders, such as decision-making, risk-taking and impulsivity. The dlPFC has been linked to food craving and consumption and is implicated in self-control in a dietary context (38). In trials with a sham comparison group, significant therapeutic effects were found for cravings (large effects), depressive symptoms (moderate effects), obsessive or compulsive symptoms and anxiety (small effects) (40).

A literature review of 16 studies using TMS applied to people with AN found that the treatments were safe and well accepted (41). A pilot randomised trial included in the systematic review above was conducted on 34 patients (mean age of 34 years, with an average BMI of 16 and an average illness duration of 14 years). Participants were randomised to real or sham TMS applied to the left dlPFC. The treatment was acceptable and at four months produced a large

reduction in depression (42). Although there was little impact on weight or eating at four months, their preference for higher-calorie foods was increased (43). A subgroup of 24 patients were followed for 18 months, during which those allocated to the sham condition were offered active treatment. Increases in BMI were found in both real (mean = 2.13, standard deviation (SD) = 3.2) and sham (mean = 0.79, SD = 1.56) TMS groups, with a moderately larger effect in those initially allocated to real TMS (44). The participants reported acceptability of the treatment, noting that they became more positive, open-minded and flexible following the intervention, but they also noted that it was time-consuming (45).

3.2.2 Transcranial direct current stimulation (tDCS)

tDCS generates a low-intensity electrical current between two electrodes to modulate activity in underlying neuronal circuits (46). As described in the TMS section above, the target has often been the dlPFC. A systematic review of tDCS to the dlPFC in people with binge-type eating disorders and behavioural addictions found seven papers reporting a reduction in food cravings (47). Later studies including sham-controlled designs also reported small-to-moderate reductions in food cravings (48, 49), and a meta-analysis found that inhibitory control was increased (50). Overall, tDCS has shown potential in improving the symptoms of BN in adults.

A recent systematic review examined the effect of tDCS in children and young people, concluding that studies with a more robust design are needed to determine the place of this intervention in clinical treatments (51).

3.2.3 Deep brain stimulation

Invasive brain stimulation strategies have been tested in small groups of patients with severe AN, exploring target areas including the subcallosal cingulate cortex (SCC), nucleus accumbens, bed nucleus of the stria terminalis and ventral anterior limb of the internal capsule. There have been several systematic reviews summarising the evidence. A review of 11 studies published between 2010 and 2020 found that out of 53 patients who began with an abnormal BMI before treatment, 22 patients (41.5%) had achieved normal BMI on follow-up (52) and a later systematic review of 11 studies (n = 36 patients with a low BMI of 12.58 ± 1.4 and a protracted illness) reported some benefits, concluding that stimulation directed at the SCC produced the most change in BMI at six months (53).

3.2.4 Brain training to overcome extremes in emotional, behavioural or social styles

A systematic review found that a variety of impulsivity-targeted approaches aimed to strengthen inhibition have shown promise in the treatment of binge eating (54). An app-based inhibitory control training towards palatable binge foods (the Food Training app) was explored in a feasibility study as an augmentation to treatment as usual, where 80 patients with BN were randomised to treatment as usual with or without augmentation with the Food Training app. Those randomised to the app showed a reduction in preferences towards binge foods and in eating disorder psychopathology (55, 56). Inhibitory control training in combination with neurostimulation techniques was also used successfully for people with binge-type eating disorders (57).

Cognitive bias modification strategies have been developed to target habitual eating disorder attitudes and behaviours including concerns about appearance and self-worth (58). For example, a small positive result was found in a feasibility study which trained people to moderate their negative interpretation of ambiguous social stimuli (59).

3.3 Pharmacological treatments

New international guidelines on the pharmacological treatment of eating disorders have been produced by the World Federation of Societies of Biological Psychiatry (WFSBP) (60). In AN, there is a limited recommendation for olanzapine because the available evidence is restricted to weight gain, with an uncertain effect on psychopathology. In BN, there are recommendations for fluoxetine or topiramate. For BED, lisdexamfetamine and topiramate might be considered. However, these drugs are not yet licenced for this indication in the UK.

This section will explore pharmacological treatments that are currently in development for the treatment of eating disorders, focusing on AN.

3.3.1 Cannabinoids

A feasibility study of dronabinol (delta-9-tetrahydrocannabinol) reported positive outcomes in a double-blind crossover trial (61, 62). However, this level of evidence was only given a grade of 2 by the WFSBP guidelines and this controlled drug is not licensed for use in AN in the UK.

3.3.2 Psilocybin

Psilocybin is a hallucinogenic with mechanistic action via serotonin pathways (5-HT) and has high affinity for the 5-HT2A receptor (63). Serotonergic dysfunction has been considered to contribute to the appetite and mood problems seen in AN (64). One hypothesis is that psilocybin may inhibit maladaptive behaviours and thought patterns that contribute to AN maintenance (65, 66). Furthermore, trials using psilocybin in treatment-resistant depression have demonstrated safety (67) and improvements in depression severity and anxiety (68, 69),which are two typical comorbidities of AN.

An open-label feasibility study, including 10 adult participants with AN (both in the acute or partial remission state), showed encouraging results (70). Participants were given 25 mg of synthetic psilocybin accompanied by psychological support. After three months, five patients had an increase in BMI and four participants had a decrease in weight concern. There were minimal adverse events, although two participants developed asymptomatic hypoglycaemia post-treatment, which resolved within a day. Several small randomised trials are currently underway to examine the feasibility and preliminary efficacy.

3.3.3 Ketamine

Ketamine is a N-methyl-D-aspartate receptor antagonist and dissociative anaesthetic that in low doses (for example, 0.5 mg/kg intravenously) has antidepressant effects. Several narrative reviews have presented the case for using ketamine in patients with AN, due to its rapid (albeit transient) antidepressant effects and targets on neuroplastic processes (71-73), and its impact on animal models of AN (74). A 1998 case series of people with AN given a ketamine infusion found that 9 of 15 had a reduction in depression and improvements in AN behaviour and psychopathology (75). Other case series of ketamine treatment for AN support this finding, demonstrating reductions in depression and improvements in eating behaviour (76-78). There has also been a case report of recovery in an individual with severe BN (79). Larger case series and feasibility studies are in progress.

3.3.4 Human recombinant leptin (metreleptin)

A series of studies have examined the hypothesis that hypoleptinaemia might be involved in the psychopathology of AN. A Mendelian randomisation study concluded that low endogenous leptin synthesis was a risk factor for developing AN (80). Furthermore, in activity-based (running wheel) animal models of AN, leptin delivered subcutaneously through mini pumps suppressed the development of hyperactivity following food restriction (81).

A case series of three female patients with acute AN treated with metreleptin for 6 to 14 days reported a pronounced and beneficial clinical response in terms of depression, activity and some aspects of eating disorder psychopathology which disappeared when the metreleptin was stopped (82). A case report of a male adolescent showed a similar profile of short-term effects (83), and a case report of a female adolescent showed an increase in appetite during the metreleptin phase of the study with continued weight gain of 15 kg over the following six months (84). The main outcomes that improved quickly were mood, hyperactivity and endocrine markers, whereas improvements in appetite were more variable.

3.3.5 Appetite-regulating hormones

Appetite-regulating hormones are of interest for both eating disorders and obesity (85, 86). A range of new treatments have been developed that target hormonal pathways, including incretin signalling, and introduced for the treatment of obesity. Preliminary studies have suggested that liraglutide (87) and semaglutide (88) reduce binge eating and weight in people with obesity and binge eating symptoms. Moreover, dulaglutide had a similar effect on people with type 2 diabetes and binge eating symptoms (89), as did liraglutide in higher-weight patients with binge eating symptoms (90). However, minimally supervised or unsupervised use of these drugs can cause problems, such as a severe lack of appetite and thirst (91).

3.4 The use the arts as adjunctive therapy

The MRC in the UK is funding research that incorporates the voice of lived experience and arts into treatment (92). Research in inpatients with AN has shown that most patients use music to cope with difficult emotions and as a distraction from their thoughts and feelings (93). A systematic review of studies on the effects of music in people with or at risk for EDs suggested that the therapeutic application of music may be beneficial in patients with AN and BN (94, 95). This review included, for example, a study by Cardi et al. (96) which found that inpatients with AN benefited from listening to classical music, which reduced distress and increased the consumption of a smoothie test meal. A systematic review of three studies using arts strategies (97) found improvements in quality of life and psychopathological symptoms, with qualitative findings indicating greater self-expression, self-awareness, new perspectives, distraction and pride. The evidence base for such therapies shows potential but is still in development.

4. The future: understanding the mechanisms underpinning eating disorders

New technologies have been used to define the biomarkers associated with eating disorders and to explore their functional role in animal models of all eating disorders, such as the food addiction, activity-based, restraint or social stress paradigms (98). Biomarkers in the brain, body and microbiome have been found, including the gut microbiome (99), immune and endocrine markers such as neurotrophins and proinflammatory cytokines (42, 100), markers of neuronal damage (for example, neurofilament light (101)) and also structural brain markers (4, 102-104).

New possible biomarkers of interest include liver-expressed antimicrobial peptide (LEAP-2) and acyl-Co-A-binding protein. Anomalies in the reciprocal relationship between ghrelin (GHR; orexigenic) and LEAP-2 (which reduces appetite) have been found in eating disorders (105) and obesity (106). Undernutrition is usually marked by low levels of LEAP-2, however patients with AN had an increase in both LEAP-2 levels and ghrelin in the acute, poorly nourished

state (107). Additionally, those with stable weight restoration six months after discharge from inpatient care had higher levels of LEAP-2 than those with continuing problems, indicating the potential for LEAP-2 changes to predict unstable remission (107). Another potential biomarker, acyl-Co-A-binding protein, is an orexigenic biomarker which is modulated by stress in animal models and was found to be lower in patients with AN (108, 109) and associated with a poor prognosis in those with the binge purge form of AN (110). These models and the mechanisms they have revealed may lead to potential new treatments. For example, donepezil, an acetyl cholinesterase inhibitor, was found to reduce self-starvation behaviours in the activity-based anorexia model (111, 112) and improved weight and eating behaviours in a small case series (n = 6) of people with AN (113). The further identification of predictive and prognostic biomarkers is a priority in the efforts towards stratifying and identifying personalised treatments for individuals with eating disorders.

5. Conclusions

As research into the aetiology of eating disorders increases, a wider range of therapeutic targets is revealed. However, there remain many unanswered questions about how to personalise treatments. One possibility is to introduce a tiered care pathway for people with eating disorders, such as that shown in Table 2. A pathway for binge-spectrum disorders already exists, starting with guided self-help, followed by CBT. The next step might include adjunctive VR exposure-based treatment (36) and/or medication, such as lisdexamfetamine or possibly incretins.

Tier	Treatment for binge-spectrum eating disorders	Treatment for anorexia nervosa
1	Guided self-help	Recruiting and skilling up social support (family treatments) by building attachments
2	CBT	CBT, MANTRA supplemented by motivational and externalisation strategies
3	Virtual reality-augmented psychotherapy targeting food and body image and social relationships Cue exposure-focused treatments	Virtual reality-augmented psychotherapy targeting food and body image and social relationships Cue exposure-focused treatments
4	Augmentation with lisdexamfetamine or topiramate (to augment treatment) TMS	Augmentation with TMS and/or psychodelics (psilocybin, ketamine)
5	Incretin receptor agonists if comorbidity with obesity or type 2 diabetes (work in progress)	Appetite system targets

Table 2: A potential tiered treatment pathway for eating disorders

Abbreviations: CBT = cognitive behavioural therapy; MANTRA = Maudsley Model of Anorexia Nervosa Treatment for Adults; TMS = transcranial magnetic stimulation

The pathway for AN is less clear cut. It may be that non-invasive neurostimulation techniques, typical/atypical forms of psychedelic medication (e.g., psilocybin, ketamine) or treatments derived from novel aspects of the appetite system are introduced in future, but currently there are only small feasibility studies investigating these. Further, a wide number of outcomes spanning 6 to 18 months may need to be considered, as changes in the traditional metrics of treatment success (e.g., weight and eating disorder psychopathology) may be slow, although there may be proximal indicators of change (e.g., depression, anhedonia, quality of life (114)).

A range of services that facilitate a variety of outcomes may be needed. Music or other creative therapies might be considered as add-ons to treatment as usual for all eating disorder presentations, both for emotional coping and to maximise opportunities for the development of a multifaceted identity.

All patients would benefit from no waiting list delays (115), as any approach to get help might represent a shift in their ambivalence, opening a crucial window in their readiness for change. Other key facets of an optimal service are a team with a wide range of clinical expertise, including collaboration with carers and peer support workers who can represent beacons of hope that recovery is possible, no matter how severe or protracted the illness. Nevertheless, it is apparent that there currently exists a gap in treatment provision for individuals who may not be at the stage of readiness (both in motivation and ability) for change. The development of treatment approaches to maximise both motivation and neurobiological ability (i.e., neuroplasticity) for change is needed.

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