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An overview of the treatment of eating disorders in adults and adolescents: pharmacology and psychotherapy

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Abstract

Purpose: This article provides an overview of current treatment options for adults and adolescents suffering from eating disorders (ED).

Views: ED are prevalent public health problems that considerably impair physical health and disrupt psychosocial functioning. In primary care settings, anorexia nervosa, bulimia nervosa, and binge eating disorder represent the most frequently seen types of eating disorders, in both adults and adolescents. To address these maladaptive eating-related behaviors and concurrent psychiatric symptoms, various pharmacological interventions and specialized psychological treatments have been evaluated and received support to varying degrees by controlled research.

Conclusions: The current literature regarding children and adolescents with eating disorders mainly supports the use of psychological interventions, such as family-based treatment and cognitive behavioral therapy. Due to the lack of robust evidence, the use of psychotropic medications is neither recommended nor approved in this population. For adults with eating disorders, an array of behaviorally focused psychotherapies, along with integrative and interpersonal approaches, can lead to the improvement of symptoms and the achievement of a healthy weight. Moreover, aside from psychotherapy, several pharmacological agents can contribute to the alleviation of eating disorders' clinical characteristics in the adult population. At the moment, the recommended psychotropic medication for eating disorders is represented by fluoxetine for bulimia nervosa and lisdexamfetamine for binge eating disorder.

Key words: anorexia nervosa, bulimia nervosa, binge eating disorder, medication, psychotherapy.

INTRODUCTION

Eating disorders (ED) are conditions characterized by abnormal eating behaviors and excessive preoccupation with food, body weight, and shape. ED stand out as particularly complex multifaceted psychiatric illnesses that can lead to multiple complications, with disruptions in cognitive, emotional, and social functioning [1]. The age at onset for ED is typically in adolescence and young adulthood [2], but a growing body of literature suggests that ED and body image concerns are also present in middle-aged and older women and men [3]. Whereas in other areas of psychiatry the use of psychotropic medication represents the center of the therapeutic management of the case, ED do not benefit from the same wide array of pharmacological agents that can alleviate the specific symptoms. At the moment, there is no medication approved for anorexia nervosa for adults or children and adolescents. Treatment literature supports the use of the antidepressant fluoxetine and central nervous system stimulant, lisdexamfetamine, for bulimia nervosa and binge ED, but only in the adult population. Studies suggest that specialized psychotherapies can be efficient in reducing symptoms of ED, and for children and adolescents these approaches represent the first line of treatment recommended by the major guidelines [4].

RECOGNIZING AND DIAGNOSING EATING DISORDERS

The conceptualization of ED evolved rapidly over the last 10 years [5]. In the current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the most significant changes are represented by the expansion of the ED chapter to "feeding and eating disorders", in which the feeding and ED of infancy or early childhood were added, along with the listing of binge eating disorder (BED) for the first time as a distinct

diagnostic category [6]. Similar modifications were made in the 11th version of the World Health Organization (WHO) International Statistical Classification of Diseases and Related Health Problems (ICD-11). Feeding and eating disorders are no longer viewed as two separate groups but as a single entity and, to align with the DSM-5 classification of ED, BED is also included as an individual ED in ICD-11. Additionally, the descriptions of anorexia nervosa and bulimia nervosa have been updated to include the atypical and developmental variations of clinical presentations [1, 7]. In the ample coverage devoted to this topic in the literature, the most common ED are anorexia nervosa, bulimia nervosa, and BED.

Anorexia nervosa is commonly seen in adolescent girls and young adult women. The peak age of onset in both sex groups is at 15-19 years [8, 9], although recent data show an increasing incidence in younger individuals (< 15 years) [10]. The core symptom of anorexia nervosa according to the ICD-11 diagnosis criteria is low body weight, defined as a body mass index (BMI) less than 18.5 kg/m^2 in adults and BMI-for-age under the fifth percentile in children and adolescents [1]. The accompanying symptoms are represented by an intense fear of weight gain, and a disturbed body image, which lead to the development of persistent behaviors to prevent the restoration of normal weight. These behaviors vary from severe dietary restrictions to purging behaviors, or excessive physical activity [11]. Amenorrhea is no longer mentioned as a diagnostic criterion either in DSM-5 or ICD-11, as it cannot be evaluated in men or premenarcheal females [12]. Anorexia nervosa is characterized by subtype (either restricting or binge-purge), and by severity, using BMI values. The restricting pattern (AN-R) is primarily defined by weight loss accomplished through the patient's engagement in extreme dietary restriction (e.g. fasting 8 hours or more, skipping meals, limiting calorie intake) or exercise. In the binge-purge subtype (AN-BP) patients have regular episodes of binge eating and/or purging behaviors (i.e. self-induced vomiting, abuse of laxatives or diuretics, enemas) aimed at getting rid of ingested food. Individuals can progress from one pattern to another, with a greater possibility of transitioning for AN-R [13]. Anorexia nervosa has the highest mortality rate of all psychiatric disorders, due to its medical and psychological complications. Coexisting psychiatric disorders include anxiety disorders, seen in 25% to 75% of patients with anorexia nervosa, obsessive-compulsive disorder, and alcohol misuse or dependence in, respectively, 15-29%, and 9-25% of individuals with anorexia nervosa [14]. Gastrointestinal, cardiac, pulmonary, musculoskeletal, endocrinal, neurological, and dermatological complications have been reported in patients with anorexia nervosa, as a consequence of prolonged malnourishment and low body weight [15].

Bulimia nervosa has an estimated lifetime prevalence of 3% in females and 1% in males, with a later onset than anorexia nervosa, ranging from 10 to 29 years [16]. The diagnostic criteria for bulimia nervosa consist of the inability to control eating an unusually large amount of food, in a short period, accompanied by repeated inappropriate compensatory behaviors to prevent weight gains, such as fasting, abuse of laxatives and diuretics, or self-induced vomiting, and intensive and vigorous physical activity [8]. Patients with bulimia nervosa usually have a normal or slightly below normal value for BMI, with frequent weight fluctuations. Other psychiatric comorbidities associated with bulimia nervosa are seen in approximately 75% of patients, with anxiety disorder, major depression or dysthymia, borderline personality disorder, self-harm, and drug and alcohol abuse being the most common [16, 17]. Regarding somatic comorbidities, severe electrolyte and acid-base alterations represent the most dangerous medical complications [15].

BED is the most frequent ED, with a lifetime prevalence ranging from 1% to 4.7% and, similarly to anorexia nervosa and bulimia nervosa, a peak age of onset in late adolescence or young adulthood [18, 19]. Binge eating is characterized by recurrent consumption of a large amount of food over a brief period (≤ 2 hours) [20], associated with a subjective loss of control over eating [21]. Almost half of the patients with BED associate ≥ 3 psychiatric disorders. Adults with BED experience anxiety disorders (65%), mood disorders (46%), impulse control disorders (43%), or substance use disorders (23%) as the most frequent psychiatric comorbidities [22]. Obesity and obesity-related diseases are among the most significant medical complications of BED. Moreover, BED may increase the risk of metabolic syndrome and cardiovascular disease, independently, regardless of the risks attributable to obesity [23].

ETIOLOGY OF EATING DISORDERS

The etiological factors of ED are not fully understood. It is presumed that these complex conditions develop from an intricate combination between psychological risk factors, socio-environmental factors, and genetic predispositions [24].

Personality traits associated with all ED are perfectionism, obsessive-compulsive features, dysphoric mood, neuroticism, harm avoidance, low self-directedness, low cooperativeness, and characteristics of avoidant personality disorder [25]. Moreover, specific personality characteristics related to the later development of AN-R are represented by low novelty seeking, anhedonia, reduced social spontaneity, constriction of affect, and emotional responsiveness. Highly impulsive behavior and the tendency to pursue new experiences or sensations have been associated with bulimia nervosa, while BED seems to be

in close relationship with perfectionism and sensation seeking [26].

Various cognitive, behavioral, and emotional risk factors, such as low self-esteem, internalizing or externalizing behaviors during adolescence, risky dieting, shape-related attitudes, behaviors consistent with restraint or disinhibition, and depressive symptoms have been identified as potential precursors of the later development of an ED [27, 28].

Regarding genetic risk factors, all ED were associated with variable heritability rates. Anorexia nervosa seems to be highly familial, with heritability estimates from twin studies ranging from 28% to 74% and a potential risk of eleven times higher than in the general population of female relatives of individuals with anorexia nervosa of developing the disorder themselves [14]. The heritability of bulimia nervosa is estimated to range between 54% to 83% [24] and studies have shown a positive correlation between the relatives of individuals with bulimia nervosa and the personal risk of developing an ED, with a particularly higher risk for bulimia nervosa [29]. Binge eating is also a moderately heritable trait, ranging from 41% to 57% [30]. Significant genetic correlations were observed between binge eating behavior, bulimia characteristics, alcohol dependence, and obesity [31].

Multiple socio-environmental factors have been studied as possible causative factors of ED. Higher parental education levels and internalization of a thin ideal can cause such disorders, especially in female adolescents, while general teasing or teasing specifically related to body weight and shape along with a personal history of trauma confer a higher risk for developing ED for both genders. Moreover, a family history of mood disorders, higher maternal anxiety during pregnancy, maternal overprotection, critical parental comments about body image or eating habits, and poor conflict resolution in families were associated with the later development of anorexia nervosa. Family overeating, the absence or death of a parent, familial separation, or environmental deprivations also revealed an elevated risk of BED [28].

TREATMENT APPROACHES FOR EATING DISORDERS

Psychotherapy

Psychological treatment of adolescents and adults with ED aims to relieve the main and associated symptoms of ED, improve and maintain a normal weight for age and sex, and enhance the quality of life.

Regarding the adult population with anorexia nervosa, a growing body of evidence supports the use of Cognitive Behaviour Therapy-Enhanced (CBT-E), Specialist Supportive Clinical Management (SSCM), or Maudsley Anorexia Nervosa Treatment for Adults (MANTRA). However, no specific approach has shown clear superiority [32]. CBT-E is a highly individualized therapeutic approach, derived from CBT for bulimia nervosa (CBT-BN), which focuses on the maintaining factors of anorexia nervosa psychopathology [33]. The enhanced version of CBT is administered over 40 sessions and can be successfully delivered in both outpatient and inpatient settings [34]. Multiple studies support the use of CBT-E for adults with anorexia nervosa, as it promotes significant improvement in weight and general psychiatric features along with a marked reduction in the ED psychopathology [33, 35]. Other behaviorally focused psychotherapies that have been assessed in treating anorexia nervosa symptoms are cognitive remediation therapy (CRT), exposure and response prevention (AN-EXRP), acceptance and commitment therapy (ACT), and dialectical behavioral therapy (DBT). Some RCTs suggest that these treatments have the potential to improve anorexia nervosa symptoms; however, additional studies are required [34]. Cognitive remediation therapy has been found to be a valuable brief intervention for adults and young people suffering from anorexia nervosa. CRT consists of exercises focused on thinking strategies and processes, encouraging a flexible, big-picture view of the world, instead of directly addressing eating, weight, and shape issues [36]. CRT can be delivered in individual (8-10 sessions) or group formats, in addition to nutrition therapy [37, 38]. An extension of cognitive remediation therapy is cognitive remediation and emotion skills training (CREST). CREST focuses on the socio-emotional difficulties of people with AN, addressing patients' inaptitude in identifying and expressing their emotions and needs [39]. CREST intervention seems to improve social anhedonia and alexithymia; however, future research is needed as firm conclusions have not yet been drawn [40]. The SSCM approach combines aspects of the clinical management of anorexia nervosa symptoms with supportive psychotherapy. SSCM has three phases in which attention is directed towards psychoeducation regarding physical and psychosocial characteristics of anorexia nervosa, resumption of normal eating, and restoration of weight. At the end of the treatment, the main focus is on preventing relapses [41]. Maudsley Anorexia Nervosa Treatment for Adults targets intra- and interpersonal maintaining factors, such as a thinking style characterized by rigidity, detail focus, fear of making mistakes, socio-emotional impairments, beliefs about positive aspects of having anorexia nervosa, and unhelpful responses of others (such as anxiety, worry, blame, criticism, or hostility) [32].

At the moment, the major guidelines recommend family-based treatment (FBT) as the first-line approach for children and adolescents with anorexia nervosa. Family-based treatment emphasizes the role of the family in helping the young person recover from anorexia nervosa, and consists in three major phases, which are typi-

cally delivered throughout 6 to 12 months [13]. In the first phase, the parent or caregiver is responsible for ensuring adequate caloric intake and weight gain. In the second phase, the adolescent is encouraged to take control over their eating behavior; and lastly, the third phase focuses on concerns related to typical adolescent developmental problems [42, 43]. Other therapeutic approaches for adolescents with anorexia nervosa who do not respond to FBT, or have a contraindication for FBT, are represented by adolescent-focused psychotherapy, cognitive behavioral therapy, and systemic family therapy [9].

The first-line therapeutic approach for adults with bulimia nervosa is represented by cognitive-behavioral therapy. Multiple clinical trials have confirmed CBT's superiority when compared to other therapies such as interpersonal psychotherapy (ITP) and psychodynamic psychotherapy (PDT) [44]. CBT-BN leads to a significant improvement in binge eating and purging behaviors, depressive and anxiety symptoms, general psychopathology, self-esteem, and social functioning. CBT-BN is generally delivered in 20 sessions, consisting of three phases, and the estimated rate of full recovery after treatment is 30% to 50% [45]. Despite being an effective treatment for bulimia nervosa, ITP is significantly slower than CBT in ameliorating the core symptoms of it [46]. Regarding the usefulness of psychodynamic psychotherapy for patients with bulimia nervosa, several studies have been conducted. One randomized controlled trial (RCT) concluded that both CBT and PDT improved global ED psychopathology and general psychopathology. However, individuals who received CBT experienced in a shorter period than the PDT group a substantial reduction of core symptoms of bulimia nervosa [47]. In other RCT studies, CBT and PDT had similar results, with both treatments being equally effective in promoting recovery from BN, but a reduced frequency of binge eating and purging favored CBT [48].

Similarly to the recommended treatment for adolescents with anorexia nervosa, the most recent guidelines support the use of family-based treatment in children and adolescents who suffer from bulimia nervosa. CBT is also a suitable treatment for adolescents with bulimia nervosa and it should be considered when FBT is ineffective, unacceptable, or contraindicated [49].

A strong evidence base supports cognitive behavior psychotherapy and interpersonal therapy as the leading psychotherapies for BED. Both approaches have similar short- and long-term results in reducing binge-eating symptoms and associated psychopathology, but neither has a significant effect on the weight loss process [50]. Dialectical behavior therapy may be also a helpful resource in decreasing binge eating episodes, by improving adaptive emotion-regulation skills, even though, like CBT and IPT, it does not have an impact on weight reduction [51]. An alternative therapeutic approach that has the advantage of reducing weight is behavioral weight loss (BWL) treatment. BWL is commonly used for adult obesity; however, it seems to be less effective than other psychotherapeutic strategies in reducing the frequency of binge eating episodes [52]. For adolescents and children with BED, studies investigated the treatment response after CBT, ITP, or DBT sessions were delivered, with positive results regarding the frequency of binge eating episodes for all therapeutic approaches, and an additional improvement of physical appearance self-concept, global self-concept and physical self-worth for CBT [21].

Pharmacological treatment

The pharmacological treatment options approved for ED in the adult population are limited to fluoxetine for bulimia nervosa and lisdexamfetamine for BED. Currently, there is no pharmacological option approved for anorexia nervosa [4, 53]. Regarding children and adolescents with ED, there is no specific pharmacological agent recommended or approved by the major guidelines.

Even though an ample number of studies have been conducted to assess the effect of various pharmacological agents on anorexia nervosa symptoms, no clear conclusions have been reached to date. Tricyclic antidepressants such as amitryptiline and clomipramine showed some impact on hunger, appetite, and energy intake when the treatment was initiated, but had no effect in terms of weight gain. Monoamine oxidase inhibitors (MAOIs) improved mood and anxiety symptoms but were still inefficient in weight gain. Selective Serotonin Reuptake Inhibitors (SSRIs) such as fluoxetine, citalopram, and sertraline were investigated in multiple RCTs [54]. The results of using fluoxetine to treat anorexia nervosa have varied, making firm conclusions difficult. In some studies, fluoxetine was associated with weight gain [53], prevention of relapses after 1-year follow-up period, and reduction of depressive symptoms [55]. However, in other studies fluoxetine either had a negative influence on appetite to the degree of inducing anorexia nervosa [56], or was not associated with any beneficial effects when used in inpatient settings [57], or as an additional treatment to cognitive behavior therapy [58]. Neither citalopram nor sertraline had any effect on weight gain. Citalopram showed an improvement in depression, and obsessive-compulsive symptoms, and studies on sertraline reported a positive impact on depressive symptoms, perception of ineffectiveness, lack of interoceptive awareness, and perfectionism in patients with anorexia nervosa [59]. Systematic reviews conclude that antidepressants show no improvement in weight gain, and their impact on eating symptoms, psychopathology, and prevention of relapses post-weight restoration is yet to be clarified [14].

Regarding antipsychotic agents, the atypical antipsychotics were associated with an alleviation of depressive

symptoms and anorexic rumination in patients with anorexia nervosa. Olanzapine may promote weight gain, reduce anxiety symptoms and improve sleep, as a recent meta-analysis concluded [53]. These results support olanzapine as one of the most promising pharmacological agents to use when treating adults with anorexia nervosa. It is recommended to use a slow up-titration regime, with 2.5 mg/day increments each week, and a maximum dosage of 10 mg/day [60]. In a limited number of studies, aripiprazole was associated with weight gain, a decrease in obsessive eating attitudes, positive effects on symptoms of anxiety and depression, and an improvement in cognitive flexibility. A 2020 case series including eleven female adolescents treated with aripiprazole showed an increase in all patient's BMIs and a reduction of core anorexia nervosa symptoms [61].

Other pharmacological agents such as mood stabilizers (lithium), antihistamines (cyproheptadine), benzodiazepines (alprazolam), opiates (naltrexone) clonidine, oxytocin, N-methyl-D-aspartate agonists and antagonists and ghrelin agonists have been studied. However, there is no significant evidence of their efficacy or safety in anorexia nervosa and they are not recommended as routine care practice [59].

As in the adult population, there is no approval for using any pharmacological treatment for adolescents suffering from anorexia nervosa. Antidepressants such as SSRIs (fluoxetine, fluvoxamine, sertraline), tricyclic antidepressants, and mirtazapine have been studied, but the results have shown little or no difference in terms of weight gain, BMI, anorexia nervosa psychopathology, or depressive and obsessive-compulsive symptoms [62]. Olanzapine seems to have a good tolerability profile, with reports of weight gain and psychopathology improvement. Research papers on risperidone, quetiapine and aripiprazole suggest a potential role in improving weight gain in children and adolescents with anorexia nervosa but, given the lack of larger studies on these agents, further investigations are needed to support their use in clinical practice [63].

A wide variety of pharmacological agents have been evaluated for bulimia nervosa symptomatology, including tricyclic antidepressants, SSRIs, MAOIs, antiepileptic drugs, opioid antagonists, and serotonin 5-HT_3 receptor antagonists.

As noted, the serotoninergic agent fluoxetine is the only medication approved for the treatment of bulimia nervosa. Fluoxetine exerts its therapeutic effect on bulimia nervosa symptoms independently of its effects on mood and is related to the augmentation of satiety mechanisms and a subsequent reduction in binge eating [15]. Typically, a dosage of 60 mg/day of fluoxetine reduces significantly the number of binge eating and vomiting episodes [64] and can be useful in improving symptoms of bulimia nervosa among adult patients who have not responded satisfactorily to psychotherapeutic approaches [65].

Other SSRIs such as citalopram, sertraline, and fluvoxamine have been studied in several RCTs, with positive results in reducing binge eating frequency and purging behaviors, and improvements of anxiety and depressive symptoms [66]. These agents typically represent a second line of treatment in clinical practice. Also, topiramate, an anticonvulsive medication, has demonstrated efficacy in decreasing both binge eating episodes and self-induced vomiting while leading to significant weight loss. Its proposed mechanism of action in the treatment of bulimia nervosa is connected with the inhibition of kainite/amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA) glutamate receptors [67]. In female patients of child-bearing age, clinicians should consider the potential interaction between topiramate and the pharmacokinetics of oral contraceptives. As topiramate and the estrogenic and progestogenic components of oral contraceptives are mainly metabolized by the cytochrome P450 3A4 enzyme system, the efficacy of these types of contraceptives can be diminished. Due to the dose-dependent nature of enzyme induction, high doses of topiramate (> 200 mg/day) can result in contraception failure and increase estrogen clearance in oral contraceptives. However, in several studies the co-administration of topiramate at dosages ≤ 200 mg/day has proven a modest interaction with oral contraceptives, with the likelihood of interference with their efficacy insignificant [68, 69]. The initiation dose should be between 12.5-25 mg/day and progressively increased to 75-200 mg/day to avoid side effects [60]. Studies reported that for patients with treatment-resistant bulimia nervosa, comorbid mood disorders, traumatic brain injury, epilepsy, or with binge eating symptoms after bariatric surgery, topiramate may represent a valid therapeutic approach [70]. Regarding the use of opioid antagonists, and 5-hydroxytryptamine 3 receptor antagonists when treating bulimia nervosa, clinical trial results of these agents are conflicting. For naltrexone, research data suggests that individuals with bulimia nervosa may have better results when the medication is administrated intranasally, or when larger oral doses of naltrexone (up to 400 mg/day) are used [64]. Ondansetron was associated with a significantly greater decrease in the frequency of binge-eating/vomiting episodes, and a significant increase in normal meal patterns, in an RCT performed by Faris *et al*., which included 26 women with severe bulimia nervosa [71].

While the use of fluoxetine was approved for adult patients with bulimia nervosa, for the adolescent population it is not included in the current guidelines [44]. However, in an open clinical trial of ten adolescent patients with bulimia nervosa or ED not otherwise specified, who received fluoxetine 60 mg/day, the results showed good tolerability of the treatment and a significant decrease in binge and purge symptoms [72].

Pharmacotherapy options for BED include antidepressants (SSRIs, serotonin–norepinephrine reuptake inhibi-

tors, and bupropion), anticonvulsants (topiramate), anti-obesity medications (d-fenfluramine, orlistat, sibutramine, and rimonabant), anti-addiction medication (acamprosate, baclofen, and opioid antagonist) and attention-deficit hyperactivity disorder medications (lisdexamfetamine). However, each of these treatments has considerable shortcomings. Antidepressants have a modest effect in reducing binge eating episodes, and have no significant impact on weight loss. In two randomized placebo-controlled trials, flexible-dose topiramate was administrated to obese patients with BED, for a period of 14 and 16 weeks respectively. The results found topiramte to be superior to placebo, with a semnificative reduction of binge eating frequency and binge eating day frequency [73, 74]. Another RCT was conducted to assess the efficacy of topiramate (target daily dose 200 mg) compared to placebo, in 73 obese adults with BED, receiving CBT. The study found topiramate to be well tolerated, efficacious in reducing weight, along with an improvement of binge eating behaviours [75]. However, its use is limited due to its adverse effects, especially on cognitive functions.

Regarding anti-obesity drugs, D-fenfluramine and sibutramine are associated with severe cardiovascular events, and rimonabant can cause important neuropsychiatric adverse effects. Orlistat did not show the same efficacy on weight loss among obese patients with BED, as seen in obese patients without BED, and even if anti-addiction medication showed a potential effect on changing the frequency of binge eating it did not influence the actual time spent binge eating [46].

Initially, lisdexamfetamine dimesylate (LDX) was only approved for the treatment of attention-deficit hyperactivity disorder (ADHD) [76], but in 2015 the Food and Drug Administration (FDA) approved LDX for the treatment of moderate-to-severe BED in adults, in some countries (e.g., US, Canada, Brazil, Puerto Rico, Mexico and Israel). Lisdexamfetamine is a prodrug of dextroamphetamine (d-amphetamine), a central nervous system stimulant [77]. Its mechanism of action in treating BED symptoms is presumed to be through a combination of effects on appetite/satiety, reward, cognitive attention, and impulsivity/inhibition processes, which involve catecholamine neurotransmission in the prefrontal cortex and serotonin receptors [78]. Approval for the use of LDX in the treatment of BED was based on three randomized, placebo-controlled studies: a Phase II study and two large Phase III trials. Currently, it is recommended to initiate the treatment with a dose of 30 mg/day LDX and continue titration in increments of 20 mg weekly, until the recommended target dose of 50 to 70 mg/day is achieved (maximum 70 mg/day) [79].

At the moment, lisdexamfetamine dimesylate treatment for children and adolescents with BED is not approved. However, a retrospective chart review in which 25 children and adolescents with BED receiving treatment with LDX were evaluated, reported an improvement in binge eating symptoms and a good tolerability profile, however with no modification of BMI percentile after treatment. Given the potential role of LDX in reducing BED symptoms, more studies are needed to assess the efficacy, tolerability, and safety in this population [80] (Table 1).

CONCLUSIONS

In addition to their increased risk of medical complications and functional impairment, EDs have been associated with the highest cause-specific mortality rates among mental disorders. Therefore, it is of the utmost importance for clinicians to accurately assess patients'

Table 1. Main pharmacological and psychotherapeutic options for the treatment of eating disorders

	Anorexia nervosa		Bulimia nervosa		Binge eating disorder	
	Adults	**Children and adolescents**	**Adults**	**Children and adolescents**	**Adults**	**Children and adolescents**
Pharmacology	Fluoxetine, citalopram, sertraline, olanzapine, aripiprazole	Olanzapine, risperidone, quetiapine, aripiprazole	Fluoxetine*, citalopram, sertraline, fluvoxamine, topiramate, naltrexone, ondastentron	Fluoxetine	LDX**, topiramate	LDX
Psychotherapy	CBT-E SSCM, MANTRA, CRT CREST	FBT, adolescent-focused psychotherapy, CBT, systemic family therapy	CBT-BN, ITP, PDT	FBT, CBT	CBT, ITP, DBT, BWL	CBT, ITP, DBT

CBT-E – cognitive behaviour therapy-enhanced, SSCMI – Specialist Supportive Clinical Management, MANTRA – Maudsley Anorexia Nervosa Treatment for Adults, CRT – cognitive remediation therapy, CREST – cognitive remediation and emotion skills training, AN-EXRP – exposure and response prevention for anorexia nervosa, ACT – acceptance and commitment therapy, DBT – dialectical behavioral therapy, FBT – family-based treatment, CBT-BN – cognitive-behavioral therapy for bulimia nervosa, ITP – interpersonal psychotherapy, PDT – psychodynamic psychotherapy, BWL – behavioral weight loss, LDX – lisdexafetamine

**Fluoxetine is the only approved medication for bulimia nervosa in the adult population.*

***Lisdexafetamine is the only approved medication for binge eating in the adult population.*

risk factors and behaviors, as early interventions are correlated with superior rates of recovery. For children and adolescents with EDs, psychological interventions represent the most common treatment option to reduce symptomatology and maintain a healthy weight. Additionally, some psychotropic agents have been studied, but not enough data has been collected. For adults, fluoxetine is currently the only medication approved for bulimia nervosa treatment, but topiramate can be regarded as an alternative option for certain populations, as it has been proven to be effective in reducing binging and purging behaviors, along with promoting weight loss. Adults with BED can benefit from the use of lisdexamfetamine, a medication approved and recommended by the guidelines due to its effects on reducing the frequency of binge eating, obsessive thoughts, and compulsions regarding binge eating. Extensive empirical research has also been conducted to identify effective agents with which to treat anorexia nervosa, with a focus on antidepressant and antipsychotic medication, but due to multiple challenges there are no pharmacological treatments approved to date. However, a recent systematic review concluded that low doses of olanzapine can have a potential impact on weight gain, making it a promising treatment option for patients with anorexia nervosa. Finally, additional studies, both psychosocial and pharmacological, are needed in adult and adolescent populations to target the mechanisms underlying EDs, and make further significant advances in treatment.

Conflict of interest

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