A summary of risk factors and protective factors in eating disorders

William Waugh[1,2], Ahad Jan[1], Carol Kan[1,2,3,4]

1. Vincent Square Eating Disorder Service, London, UK
2. Central and North West London NHS Foundation Trust, London, UK
3. King's College London, London, UK
4. Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

✉ Corresponding author: William Waugh. Email: william.waugh@nhs.net

Abstract

Given the increasing prevalence of eating disorders, a greater awareness of risk factors among clinicians is essential. This should enable appropriate screening for at-risk groups, reducing the risk of developing eating disorders and prompting individualised treatment programmes.

The risk factors for eating disorders are multifactorial, and we will discuss factors under the following domains: demographic, biological and developmental, personal, psychological and sociocultural. Protective factors will also be discussed.

Introduction

A recent meta-analysis showed that the prevalence of eating disorders in the UK is higher than previously estimated, with a lifetime mean of 8.4% for women and 2.2% for men (1). Furthermore, there is evidence to suggest that prevalence rates have increased significantly between the periods of 2000 to 2006 and 2013 to 2018 (1). An increasing prevalence has led to an increasing demand on specialist eating disorder services, resulting in longer waiting times and a comparatively higher "illness threshold" for patients to gain access to specialist treatment (2).

Patients with eating disorders are therefore increasingly being managed by non-specialist services, including child and adolescent/general adult mental health teams, general medical teams and general practitioners. If prevalence and illness thresholds continue to rise, the severity of eating disorder illness that is expected to be managed by non-specialist services is also likely to rise.

The 2017 parliamentary ombudsman report on the avoidable deaths of three patients with eating disorders (3) highlighted how harm can be prevented with prompt and effective intervention. The subsequent call for a review of junior doctor training in eating disorders revealed that the average medical school graduate receives less than two hours of teaching on eating disorders throughout their entire undergraduate medical degree (4). Medical education has not kept up with increasing clinical demands. This is a particularly pressing issue given that eating disorders are associated with high levels of physical and psychiatric comorbidity and a high mortality rate (5). Non-specialists may be asked to manage complex and risky patients in a community setting with minimal training, in a very specialised area of psychiatry. This includes general adult psychiatrists and GPs. GPs are frequently the first point of contact with health services for those with eating disorders, and early detection from GPs may contribute to better outcomes (6). The inverse is also true – lack of detection or poor care from GPs may cause overall worse outcomes (7). Early detection of an eating disorder (8) and subsequent early intervention (9) have been demonstrated to have a significant impact on recovery, symptom burden and mortality. In primary care, the focus must be on early detection and care planning, which should include discussion with more specialist services but may place a large burden of the responsibility for delivery of care on the general practitioner.

A key element of early detection and care planning is health professionals' knowledge of the risk and protective factors associated with eating disorders, especially during early contacts with health services in, for example, primary care (6). In this review, we will discuss risk factors and protective factors around eating disorders, specifically focussing on anorexia nervosa, bulimia nervosa and binge eating disorder, although many of these risk factors will also be applicable to eating disorder not otherwise specified and avoidant/restrictive food intake disorder diagnoses.

Risk factors

Identification of groups at risk of developing eating disorders allows for focused monitoring, targeted prevention strategies and earlier intervention, all of which contribute to more favourable outcomes. However, in order for risk factors to be clinically useful, they need to be both sensitive and specific. In this section, we will focus on risk factors that are both sensitive and specific to eating disorders in demographic, biological and developmental, personal, psychological and sociocultural domains.

Demographic risk factors

Age and gender are well-known risk factors for eating disorders. Women develop eating disorders at rates significantly higher than men (10). The reported differences between men and women are variable depending on the source; some studies show that women have a lifetime risk almost four times higher than men (1). In addition, rates of eating disorders in women and men have changed substantially in the past decades; males comprised only 10% of patients diagnosed with eating disorders in the 1960s (11), compared to up to 25% in 2020 (1). Evidence suggests that men are historically less likely to receive an eating disorder diagnosis and cases of men with an eating disorder were considered vanishingly rare (12). It is only in the past two to three decades that diagnoses of eating disorders, and particularly anorexia nervosa, in men have become more widely accepted. This is attributed to greater understanding of eating disorders amongst professionals and an overall increasing prevalence of eating disorders. The impact of changing gender roles in society is difficult to measure but may also play a role in the changes in male-to-female risk ratios in eating disorders (13).

Academic and societal concepts of gender and sexual identity are changing and rates of eating disorders among LGBTQI groups are significantly higher than those in the general population; some studies suggest that over 50% of LGBT adolescents have received an eating disorder diagnosis, whilst over 60% have engaged in at least one disordered eating behaviour (14), compared to 13% and 22% in general population, respectively (14, 15). Our current understanding of the impact of gender on eating disorders is limited, with the complex intersectionality between gender, sexual identity and eating disorders remaining unexplored (16).

This is further complicated by adolescence being the peak period of eating disorders risk (10), with the risk highest between the ages of 15 and 19 (17). Those with earlier age of puberty onset are also at increased risk (18). Factors such as bodily changes, identity formation and transition to adulthood have all been implicated in this risk period (18). A growing body of research has highlighted the role of oestrogen, and oestrogen receptor genes, in the development of eating disorders – this may contribute to the increased risk both around puberty in females (18), and also around menopause (19).

Studies on the impact of ethnicity and ethnic minority status on the risk of developing an eating disorder are far from conclusive; some studies suggest no differences in risk based on ethnicity (19) whilst others report that being mixed race may increase risk and being from black/Hispanic backgrounds may reduce risk (20). Higher parental socioeconomic status has been associated with increased risk of developing an eating disorder, especially anorexia nervosa (21), in contrast to other major mental illnesses; however, the validity of these findings is now being questioned, with the suggestion that increased risk may in fact be better explained by other factors, such as higher educational attainment, which is linked to anorexia nervosa (22).

Biological and developmental risk factors

Genetic factors are thought to play a significant role in the development of eating disorders, with heritability estimates from twin studies in the range of 48% to 74% for anorexia nervosa, 55% to 62% for bulimia nervosa and 39 to 45% for binge eating disorder (23).

In addition, genome-wide association studies have revealed extensive genetic overlap between anorexia nervosa, psychiatric disorders and related traits, such as bipolar disorder and neuroticism (24).

The interplay between genetic factors and eating disorders is further complicated by intergenerational association. Studies have reported that an individual with a parent who has a history of an eating disorder is twice as likely to develop an eating disorder than those with parents who have no history of eating disorders (25). In addition, family history of any affective disorder has been shown to increase the risk of developing an eating disorder (26), and the closer the relative, the higher the risk conferred. It is likely that there is a degree of overlap between genetic risk and environmental/social factors when a relative is in very close proximity to the patient, such as a parent or sibling. Our comprehension of the impact of these genetic and environmental factors on the risks of developing an eating disorder remains limited at present. It is likely that there is an epigenetic contribution to the development of eating disorders, given the apparent interaction between genetic risk factors and environmental stressors; however, the evidence remains limited at present (27).

Regarding biological risk factors, satiety and ovarian hormones have been linked to the core symptoms of eating disorders (28). Imbalances in glutamatergic and dopaminergic mesolimbic reward pathways have also been implicated, showing abnormalities similar to those seen in drug addiction in patients with binge eating disorder (29). Glucocorticoids, stress response and dysregulation in these axes have been linked to modulation of appetite; however, further study is required to establish direct links (30).

Childhood obesity has been linked to subsequent development of bulimia nervosa or binge eating disorder (31), whereas low childhood body mass index has been associated with anorexia nervosa (32). Together with evidence indicating an association between childhood energy balance disorder and later development of eating disorders (33), these vulnerabilities suggest a metabolic causative factor for eating disorders (32).

Certain long-term physical illnesses, such as coeliac disease (34), type 1 diabetes mellitus (35) or serious childhood illnesses requiring hospital admission (36) have also been associated with the development of disordered eating behaviours. Inflammatory bowel diseases, namely Crohn disease and ulcerative colitis, show a complex bidirectional relationship with eating disorders (37). There is evidence of bidirectional diagnostic overshadowing; an increased fear of food-related symptoms in those with IBD may induce disordered eating behaviours.

Research has suggested that the prevalence of eating disorders among people with type 1 diabetes is 7% (38), the association being strongest with bulimia nervosa (39). The latter is sometimes referred to as "diabulimia" and involves the misuse of insulin to prevent weight gain. It is possible that the biological and psychological implications of having type 1 diabetes increase the risks of subsequent eating disorders (40).

Neurotransmitter imbalances are linked to the perpetuation of anorexia nervosa and bulimia nervosa, particularly serotonin (41). This imbalance can heighten anxiety and perpetuate the obsessive thoughts around food and body image in eating disorders. Compared with findings in anorexia nervosa, serotonergic changes in binge eating disorder appear to be inconsistently skewed toward reduction of serotonin activity (41). In addition, dysregulation of appetite-related hormones has been shown to contribute to the perpetuation of binge eating disorder, though the mechanism remains unclear (42).

Neurodevelopmental disorders such as attention deficit hyperactivity disorder (ADHD) (43) and autism (44) both show high levels of co-occurrence with eating disorders. It is not clear whether these should be considered risk factors; however, it is important to be aware that someone with, for example, ADHD is more likely to meet the diagnostic criteria for an eating disorder (45). It is also important to be aware that presentations may vary with the presence of a concomitant neurodevelopmental disorder (46).

Finally, gut microbiota is an emerging area of interest, with some evidence suggesting patients with anorexia nervosa have a very different gut microbiome profile from those of the general population (47). It is unclear if the difference observed reflects a predisposing factor for anorexia nervosa or the effects of starvation. In addition, differences/changes in microbiome profile have been observed in factors reported earlier, such as anxiety and depression symptomology (48), immune system defence mechanisms and the metabolism of insulin (47).

Psychological risk factors

Certain personality traits, such as perfectionism (49, 50), body dissatisfaction and negative emotionality (51), have all been linked with the development of eating disorders in the context of a social setting that idealises thinness. In the context of an eating disorder, perfectionist traits are mostly associated with restriction as they can drive individuals to maintain rigid eating patterns, reinforcing their perceived control (52). Other traits implicated in the development of eating disorders include low self-esteem, negative body image and feelings of inadequacy (53). Impulsivity and difficulties with emotional regulation are more closely associated with bingeing/purging behaviours (54).

Diagnoses of affective disorders such as anxiety or depression are common in those diagnosed with eating disorders (55). Low self-esteem has been shown to be a universal risk factor for eating disorders (37); it has even been hypothesised that low self-esteem directly causes eating disorders (56), and it has been robustly demonstrated that self-esteem and risk of developing an eating disorder follow a reciprocal relationship, that is, the lower the self-esteem, the higher the risk.

Some individuals with eating disorders struggle with emotional regulation and may use food intake or food restriction as a coping mechanism to alleviate distress (57). For example, in binge eating disorder, emotional regulation through binge eating plays a significant role in the perpetuation of the illness; individuals use food as a coping mechanism to alleviate stress, anxiety or depression (58). The negative reinforcement of emotional relief maintains the pattern of compulsive eating, as the emotional relief is frequently only short-lived and is replaced with more negative feelings resulting directly from the binge (59).

Difficulties in emotional regulation may also play a key role in maintaining the symptoms of eating disorders (60). Chronic starvation in anorexia nervosa can lead to anhedonia and worsen existing comorbidities which can, in turn, lead to emotional distress which acts to perpetuate the illness; thus, difficulties in the regulation of emotions act as both a predisposing and a perpetuating factor (61, 62).

Individuals with eating disorders may feel compelled to maintain the secrecy of their behaviours. Strong feelings of shame or guilt are associated with symptoms of disordered eating (62), and this can lead to social withdrawal and isolation which can perpetuate their illness (63). For example, social isolation has been shown to contribute to the perpetuation of anorexia nervosa, as individuals with anorexia withdraw from social activities and relationships, limiting their opportunities for external influence and support (64). Social isolation has also been shown to play a role in the perpetuation of binge eating disorder, as the individual's withdrawal from others combined with the cycle of overeating followed by negative emotions appears to increase the intensity of symptoms, which further reinforces the illness and the social isolation (65).

Motivation to recover or to change disordered eating behaviours is a difficult-to-quantify risk factor for both the per-

petuation and precipitation of an eating disorder. It is variable across a patient's lifetime, appearing to increase with age (66) and being influenced by a range of lifestyle and social factors (67).

Personal risk factors

A personal history of mental illness has been shown to increase risk of developing an eating disorder (68); in fact, many patients with eating disorders have been shown to meet diagnostic criteria for anxiety disorders prior to developing symptoms of disordered eating (69).

A personal history of trauma seems to compound all other risk factors as well as conferring increased risk itself (69). Traumatic experiences or adverse life events may act as triggers for the development of an eating disorder, pushing individuals toward restrictive eating as a method of regaining a sense of control. These can include experiences such as childhood maltreatment (70), insecure attachment (71), teasing, bullying or social exclusion (72), or childhood sexual abuse (73). Studies have shown that when patients diagnosed with eating disorders are compared with a control group, significantly more patients with eating disorders than community controls have experienced either a severe life event or a period of marked difficulties during the year before onset of their illness (64). The most common serious life stressors before onset involved close relationships with family and friends (interpersonal events) (64). Of all those diagnosed with eating disorders who had experienced either a severe life event or period of marked difficulties during the year before the onset of their illness, those with bulimia nervosa had most commonly experienced a serious life stressor, whereas difficulties with sexuality seem to be specifically related to the onset of anorexia nervosa (64). Social stressors and problems affecting interpersonal relationships can result in the formation of a vicious cycle, as problems with interpersonal relationships are secondary consequences of an eating disorder and have been shown to contribute to the perpetuation and perseverance of the illness (52).

We have previously mentioned that the highest-risk age group is those between 15 and 19 years of age; two factors that appear to be closely linked to this age group and act as major contributing factors are the impact of dieting behaviours and the impact of academic stress (i.e., the pressure to succeed academically). It was noted that female adolescents who severely dieted were 18 times more likely to develop an eating disorder than those who did not (74). Literature on the impact of academic stress on eating disorders is lacking but it has been implicated in contributing to stress in the years prior to diagnosis (75), and also implicated as an exacerbating factor in those already diagnosed, causing emotional distress which is manifested as symptoms of disordered eating (76). Transitioning to higher education has been reliably shown to increase risk – this appears to be as a result of a combination of factors including age group, academic pressure, peer influence and the impact of social media (77).

The relationship between eating disorders and recreational activities, such as sport, is complex. However, it seems that, in general, sports that involve a subjective judgement of performance (e.g., ballet, gymnastics), as well as sports that are weight category–dependent or in which weight has a significant impact upon performance (78), confer higher risks of eating disorders, whilst sports that involve more objective judgement of performance by final score (e.g., football, netball) and are not weight category–dependent may in fact confer protection against the development of eating disorders (79, 80). Among professional sports, the risk is higher (81), and specific links to situations like being weighed in public or attempts to reduce weight in order to "make weight" for a competition or event have been identified as risk factors (78).

The home environment is closely linked to development of eating disorders, with family dysfunction and particularly negative food-related experiences involving the family being closely associated with increased disordered eating (82). In particular, childhood undereating and fussy eating have been linked to the development of anorexia nervosa, whilst childhood overeating has been linked to the development of binge eating disorder (83).

Sociocultural risk factors

Idealisation of thinness is a strongly contributing predisposing factor, both in general terms as societal beauty standards have shifted over the 20th and 21st centuries (84) and specifically within families and social groups where the phenomenon of "fat talk" (topics of conversation around eating and body image, specifically disparaging or negative comments about size and body shape in relation to eating) has been shown to be strongly correlated with the presence of symptoms of disordered eating (85). Sociocultural influences, such as the idealisation of thinness (including media exposure, pressures for thinness, thinness expectancies) contribute to body dissatisfaction and may prompt maladaptive eating patterns (86).

In addition to the increasing prevalence of unrealistic/unachievable beauty standards, there has also been an increasing normalisation of diet culture and this, combined with the idealisation and pursuit of thinness, exerts a great deal of social pressure on vulnerable individuals and contributes to a distorted body image (87). Distorted body image is implicated in the genesis of all eating disorders, but in binge eating disorder the dieting and restriction, often in response to societal pressures, can escalate into episodes of compulsive overeating (88). Dieting alone puts an adolescent at significantly increased risk of developing an eating disorder (74).

Peer pressure and societal ideals have also been shown to contribute to the perpetuation of bulimia nervosa (89).

Reflections from patients who have recovered from bulimia nervosa have cited the desire to conform to societal expectations regarding body image as a strong factor in the propagation of the cycle of binge eating and purging, and this is likely to be applicable to symptoms of disordered eating in general.

Social media have been well documented to contribute both to the development of disordered eating but also to relapse risk in those in recovery, through the mechanisms of social comparison, thin ideal internalisation and self-objectification, as well as driving body dissatisfaction (90). This is especially true in highly visual social media (91) and appears to be related to exposure to idealised standards of thinness/leanness/muscularity; however, social media literacy and positive self-image appear to protect against these risks (90).

Protective factors

Definitions of "protective factors" vary across the literature. However, for the purposes of most research on eating disorders (and for the purposes of this paper), the following well-established summary is sufficient. A psychosocial protective factor is that which modulates or in some other way reduces the impact of a risk factor or risk factors on a person's development, contributing to an overall positive outcome and (of particular relevance in eating disorders) promoting the "establishment and maintenance of self-esteem and self-efficacy" (92). Many of the protective factors we think about in the prevention and treatment of eating disorders are not solely specific to disordered eating but will apply to a wide range of mental health difficulties or diagnoses (93). In a similar vein, protective factors do not necessarily apply universally, and it has been demonstrated robustly in the literature that demographic factors such as socioeconomic status, gender and ethnicity (among others) will influence the impact of protective factors (94). The significance of specific protective factors varies across the research, but it is relevant to note that many of these protective factors (e.g., social and emotional support, family environments) are also modifiable.

Beginning with the home environment during development, and beginning with perhaps the broadest and least specific protective factor, it has been shown that feeling satisfied with one's family life between the ages of 10 and 12 years is linked to a reduction in levels of disordered eating in early adolescence and, thus, a (non-specific) feeling of satisfaction in one's family life might be protective against the development of eating disorders (95). In a similar vein, higher levels of familial "connectedness" (96) also appears to convey protection against developing disordered eating behaviours. This may link to the fact that the structure and cohesiveness of the family unit has also been shown to play an important role, as having a higher number of full siblings has been associated with a lower risk of developing an eating disorder (whilst having a higher number of half siblings appears to be associated with a higher risk of developing an eating disorder) (97). There is strong evidence to show that living in a two-parent family reduces the risk of disordered eating, even in the presence of other risk factors (98).

A more specific measure of the effect of the home environment is attitudes towards weight and eating within the family home. Evidence suggests that conversations around healthy eating in the home convey protection against disordered eating behaviours (99), whilst conversations focused solely on weight (100) or critical comments about weight are associated with an increase in disordered eating behaviours (101) – linking to earlier discussions around "fat talk". A high frequency of meals eaten as a family has been shown to be protective against disordered eating behaviours (102) and this appears to show a stronger association when also combined with a positive atmosphere at the dinner table (100), although other data indicates that frequency alone is insufficient to have a protective effect, and that only when frequency is combined with a positive atmosphere is the protective impact present (96).

The importance of family support should not be underestimated, both as a protective factor and as a positive influence on treatment outcomes. Adolescents who reported higher levels of unconditional parental support also had lower rates of developing eating disorders, and a higher level of unconditional parental support has been shown to be protective against the impact of negative life events (103). Symptom burden has also been shown to be lower in those with high levels of family support, for example, purging behaviours (104).

Personality traits are often cited in the development of eating disorders, particularly in anorexia. When examining protective factors, a higher level of personal resilience is associated with better outcomes (improved psychological health and a reduction in symptom burden) (105). Positive self-esteem/self-image is also demonstrated to be both protective against development of eating disorders and associated with better outcomes (106). In the same way that lack of emotional regulation is associated with the development of, for example, binge eating disorder (107), strong emotional regulation (differentiated from emotional suppression) is shown to be protective against developing an eating disorder (108).

Personal resilience is also protective, and levels of family support and care experienced during adolescence are directly correlated with levels of personal resilience (109). Research therefore supports both early family involvement in care, as well as care planning that looks at the patient not just as an individual but as a member of the family unit (94) and examines the relationships and dynamics present within the home.

Relevance to treatment

Eating disorders are serious psychiatric illnesses with high burdens of morbidity and mortality. Early intervention has

been shown to be beneficial in improving treatment outcomes and reducing the impacts of symptom and illness burden, such as developmental disruption. One model of early intervention that has demonstrated promising results is the First Episode Rapid Early intervention for Eating Disorders (FREED) program which aims to address "barriers to early, effective eating disorder treatment in emerging adults aged 16 to 25 years" (110). The program targets those at the onset of their illness and works to address the risk factors around the patient, in addition to treating the patient themselves. The results show a significant increase in body mass index compared to those receiving treatment as usual, as well as a reduction in intensity of service usage (111). Furthermore, those treated under the FREED program also experience a reduction in symptom burden and an increase in self-acceptance. They also reported being able to build a support network and focus on life outside their eating disorder (112). Other studies have reported the effect of eating disorder prevention programmes on university campuses on the risk of students developing an eating disorder (113). More research is needed to design and evaluate preventive eating disorder interventions, with the potential of scalability to population level.

Summary and clinical implications

As inferred from the range of risk factors discussed here, the development of an eating disorder is a multifactorial process. The range of risk factors discussed here are summarised in Figure 1 and it is helpful in clinical practice to conceive of these as "vulnerability factors" that can be accrued and put someone at lesser or greater risk. The quote "genetics loads the gun but the environment pulls the trigger" (114) is a useful way to conceptualise this, and also to understand why two seemingly very similar patients may present very differently.

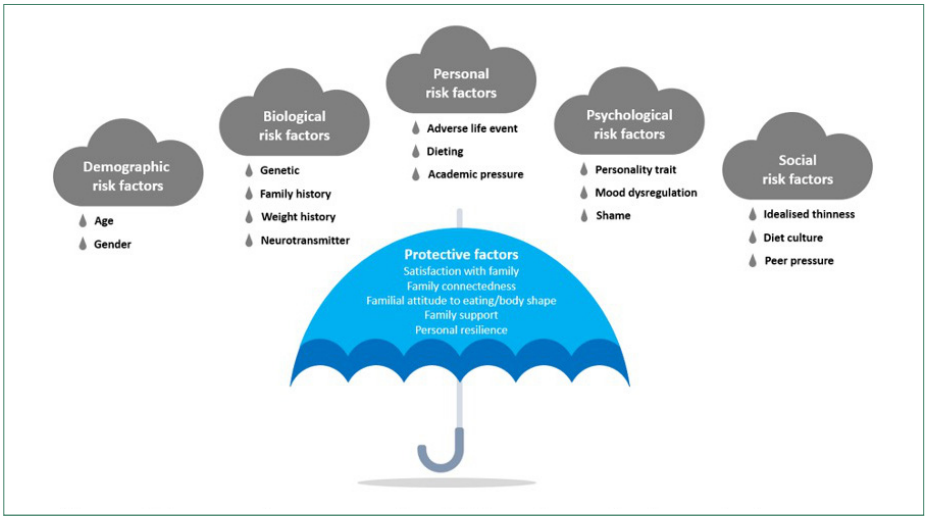

Figure 1: Diagrammatic representation of risks and protective factors in the development of eating disorders

The key clinical implications and messages from this discussion should be that eating disorders, whilst serious, are also treatable and, to some extent, preventable. The prognosis is drastically improved by early intervention, and early identification is vital for interventions to be provided in a timely manner. Eating difficulties cannot be viewed in isolation but must be viewed in the context of their own individual situation; risk and protective factors should be reviewed to form a working care plan alongside the patient and their support network. With knowledge of these factors, clinicians will be more able to provide effective early care for patients, reducing the disease burden for individuals, their loved ones and for society. Furthermore, with these aims in mind whilst care planning, clinical teams should be more able to take a truly patient-centred approach to holistic care planning.

References

1. Galmiche M, Déchelotte P, Lambert G, Tavolacci MP. Prevalence of eating disorders over the 2000–2018 period: a systematic literature review. The American Journal of Clinical Nutrition. 2019;109(5):1402-13.
2. Beat Eating Disorders. Lives at risk: The state of NHS adult services 2020. Accessed 02 January 2024. Available from: https://www.beateatingdisorders.org.uk/about-beat/policy-work/policy-and-best-practice-reports/lives-at-risk-the-state-of-nhs-adult-services/.
3. Parliamentary and Health Service Ombudsman. Ignoring the alarms: How NHS eating disorder services are failing patients 2017. Accessed 08 Sept 2024. Available from: https://www.ombudsman.org.uk/sites/default/files/page/FINAL%20FOR%20WEB%20Anorexia%20Report.pdf.
4. Ayton A, Ibrahim A. Does UK medical education provide doctors with sufficient skills and knowledge to manage patients with eating disorders safely? Postgraduate Medical Journal. 2018;94(1113):374-80.
5. Peebles R, Sieke EH. Medical complications of eating disorders in youth. Child and Adolescent Psychiatric Clinics. 2019;28(4):593-615.
6. Yeo M, Hughes E. Eating disorders: early identification in general practice. Australian Family Physician. 2011;40(3):108-11.
7. Rowe E. Early detection of eating disorders in general practice. Australian Family Physician. 2017;46(11):833-8.
8. Cadwallader JS, Godart N, Chastang J, Falissard B, Huas C. Detecting eating disorder patients in a general practice setting: a systematic review of heterogeneous data on clinical outcomes and care trajectories. Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity. 2016;21:365-81.
9. Kalindjian N, Hirot F, Stona A-C, Huas C, Godart N. Early detection of eating disorders: a scoping review. Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity. 2021:1-48.

10. Hudson JI, Hiripi E, Pope Jr HG, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biological Psychiatry. 2007;61(3):348-58.
11. Andersen AE, Holman JE. Males with eating disorders: challenges for treatment and research. Psychopharmacology Bulletin. 1966;33(3):391-7.
12. Murray SB, Nagata JM, Griffiths S, Calzo JP, Brown TA, Mitchison D, et al. The enigma of male eating disorders: a critical review and synthesis. Clinical Psychology Review. 2017;57:1-11.
13. Sweeting H, Bhaskar A, Benzeval M, Popham F, Hunt K. Changing gender roles and attitudes and their implications for well-being around the new millennium. Social Psychiatry and Psychiatric Epidemiology. 2014;49(5):791-809.
14. Lopez-Gil JF, Garcia-Hermoso A, Smith L, Firth J, Trott M, Mesas AE, et al. Global proportion of disordered eating in children and adolescents: a systematic review and meta-analysis. JAMA Pediatrics. 2023;177(4):363-72.
15. Rienecke RD. Family-based treatment of eating disorders in adolescents: current insights. Adolescent Health, Medicine and Therapeutics. 2017;8(null):69-79.
16. Gordon AR, Beccia AL, Egan N, Lipson SK. Intersecting gender identity and racial/ethnic inequities in eating disorder risk factors, symptoms, and diagnosis among US college students: an intersectional multilevel analysis of individual heterogeneity and discriminatory accuracy. International Journal of Eating Disorders. 2023;57(1):146-61.
17. Van Son GE, van Hoeken D, Bartelds AI, Van Furth EF, Hoek HW. Time trends in the incidence of eating disorders: a primary care study in the Netherlands. International Journal of Eating Disorders. 2006;39(7):565-9.
18. Klump KL. Puberty as a critical risk period for eating disorders: a review of human and animal studies. Hormones and Behavior. 2013;64(2):399-410.
19. Baker JH, Runfola CD. Eating disorders in midlife women: a perimenopausal eating disorder? Maturitas. 2016;85:112-6.
20. Van Dyne A, Washington N, Villodas M, Cronan T. Racial/ethnic differences in anorexia and bulimia diagnoses among US college students. Eating Behaviors. 2023;50:101779.
21. Koch SV, Larsen JT, Plessen KJ, Thornton LM, Bulik CM, Petersen LV. Associations between parental socioeconomic-, family-, and sibling status and risk of eating disorders in offspring in a Danish national female cohort. International Journal of Eating Disorders. 2022;55(8):1130-42.
22. Schilder CM, Sternheim LC, Aarts E, van Elburg AA, Danner UN. Relationships between educational achievement, intelligence, and perfectionism in adolescents with eating disorders. International Journal of Eating Disorders. 2021;54(5):794-801.
23. Yilmaz Z, Hardaway JA, Bulik CM. Genetics and epigenetics of eating disorders. Advances in Genomics and Genetics. 2015:131-50.
24. Bang L, Bahrami S, Hindley G, Smeland OB, Rødevand L, Jaholkowski PP, et al. Genome-wide analysis of anorexia nervosa and major psychiatric disorders and related traits reveals genetic overlap and identifies novel risk loci for anorexia nervosa. Translational Psychiatry. 2023;13(1):291.
25. Bould H, Sovio U, Koupil I, Dalman C, Micali N, Lewis G, et al. Do eating disorders in parents predict eating disorders in children? Evidence from a Swedish cohort. Acta Psychiatrica Scandinavica. 2015;132(1):51-9.
26. Hudson JI, Pope HG, Jonas JM, Yurgelun-Todd D. Family history study of anorexia nervosa and bulimia. The British Journal of Psychiatry. 1983;142(2):133-7.
27. Hübel C, Marzi SJ, Breen G, Bulik CM. Epigenetics in eating disorders: a systematic review. Molecular Psychiatry. 2019;24(6):901-15.
28. Culbert KM, Racine SE, Klump KL. Hormonal factors and disturbances in eating disorders. Current Psychiatry Reports. 2016;18:1-16.
29. Yohn SE, Galbraith J, Calipari ES, Conn PJ. Shared behavioral and neurocircuitry disruptions in drug addiction, obesity, and binge eating disorder: focus on group I mGluRs in the mesolimbic dopamine pathway. ACS Chemical Neuroscience. 2019;10(5):2125-43.
30. Kuckuck S, van Der Valk ES, Scheurink AJ, van Der Voorn B, Iyer AM, Visser JA, et al. Glucocorticoids, stress and eating: the mediating role of appetite-regulating hormones. Obesity Reviews. 2023;24(3):e13539.
31. Stabouli S, Erdine S, Suurorg L, Jankauskienė A, Lurbe E. Obesity and eating disorders in children and adolescents: the bidirectional link. Nutrients. 2021;13(12):4321.
32. Stice E, Gau JM, Rohde P, Shaw H. Risk factors that predict future onset of each DSM–5 eating disorder: Predictive specificity in high-risk adolescent females. Journal of Abnormal Psychology. 2017;126(1):38.
33. Han JC, Weiss R. Obesity, metabolic syndrome and disorders of energy balance. Sperling Pediatric Endocrinology. Elsevier (2021). p. 939-1003.
34. Nikniaz Z, Beheshti S, Abbasalizad Farhangi M, Nikniaz L. A systematic review and meta-analysis of the prevalence and odds of eating disorders in patients with celiac disease and vice-versa. International Journal of Eating Disorders. 2021;54(9):1563-74.
35. Hanlan ME, Griffith J, Patel N, Jaser SS. Eating disorders and disordered eating in type 1 diabetes: prevalence, screening, and treatment options. Current Diabetes Reports. 2013;13:909-16.
36. Breithaupt L, Köhler-Forsberg O, Larsen JT, Benros ME, Thornton LM, Bulik CM, et al. Association of exposure to infections in childhood with risk of eating disorders in adolescent girls. JAMA Psychiatry. 2019;76(8):800-9.
37. Di Giorgio FM, Melatti P, Ciminnisi S, Cappello M. A narrative review on eating disorders and disordered eating in inflammatory bowel diseases: need for increased awareness. Dietetics. 2023;2(2):150-60.
38. Winston AP. Eating disorders and diabetes. Current Diabetes Reports. 2020;20:1-6.
39. Ismail K. Eating disorders and diabetes. Psychiatry. 2008;7(4):179-82.
40. Harrison A, Zaremba N, Brown J, Allan J, Konstantara E, Hopkins D, et al. A cognitive behavioural model of the bidirectional relationship between disordered eating and diabetes self care in people with type 1 diabetes mellitus. Diabetic Medicine. 2021;38(7):e14578.
41. Steiger H. Eating disorders and the serotonin connection: state, trait and developmental effects. Journal of Psychiatry and Neuroscience. 2004;29(1):20-9.
42. Baenas I, Miranda-Olivos R, Solé-Morata N, Jiménez-Murcia S, Fernández-Aranda F. Neuroendocrinological factors in binge eating disorder: a narrative review. Psychoneuroendocrinology. 2023;150:106030.
43. Bleck JR, DeBate RD, Olivardia R. The comorbidity of ADHD and eating disorders in a nationally representative sample. The Journal of Behavioral Health Services and Research. 2015;42:437-51.
44. Tchanturia K. What we can do about Autism and Eating Disorder comorbidity. European Eating Disorders Review. 2022 Sept; 30(5):437-41.

45. Biederman J, Ball SW, Monuteaux MC, Surman CB, Johnson JL, Zeitlin S. Are girls with ADHD at risk for eating disorders? Results from a controlled, five-year prospective study. Journal of Developmental and Behavioral Pediatrics. 2007;28(4):302-7.
46. Carpita B, Muti D, Cremone IM, Fagiolini A, Dell'Osso L. Eating disorders and autism spectrum: links and risks. CNS Spectrums. 2022;27(3):272-80.
47. Santonicola A, Gagliardi M, Guarino MPL, Siniscalchi M, Ciacci C, Iovino P. Eating disorders and gastrointestinal diseases. Nutrients. 2019;11(12):3038.
48. Seitz J, Trinh S, Herpertz-Dahlmann B. The microbiome and eating disorders. Psychiatric Clinics. 2019;42(1):93-103.
49. Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME. Risk factors for bulimia nervosa: a community-based case-control study. Archives of General Psychiatry. 1997;54(6):509-17.
50. Fairburn CG, Cooper Z, Doll HA, Welch SL. Risk factors for anorexia nervosa: three integrated case-control comparisons. Archives of General Psychiatry. 1999;56(5):468-76.
51. Leon GR, Fulkerson JA, Perry CL, Keel PK, Klump KL. Three to four year prospective evaluation of personality and behavioral risk factors for later disordered eating in adolescent girls and boys. Journal of Youth and Adolescence. 1999;28:181-96.
52. Treasure J, Willmott D, Ambwani S, Cardi V, Clark Bryan D, Rowlands K, et al. Cognitive interpersonal model for anorexia nervosa revisited: The perpetuating factors that contribute to the development of the severe and enduring illness. Journal of Clinical Medicine. 2020;9(3):630.
53. Stankovska G, Memedi I, Nuhii N. Relationship Between Bulimia Nervosa and Psychological Problems in Period of Adolescence: Perfectionism, Self-Esteem, and Beyond. *In* Eating Disorders. Cham. Springer International Publishing (2023). p. 723-745.
54. Lavender JM, Mitchell JE. Eating disorders and their relationship to impulsivity. Current Treatment Options in Psychiatry. 2015;2:394-401.
55. Bulik CM. Eating Disorders and Obesity: A Comprehensive Handbook. New York, NY. Guilford Press New York (2002).
56. Colmsee I-SO, Hank P, Bošnjak M. Low self-esteem as a risk factor for eating disorders. Zeitschrift für Psychologie. 2021;229(1):48-69
57. Vanderlinden J, Dalle Grave R, Fernandez F, Vandereycken W, Pieters G, Noorduin C. Which factors do provoke binge eating? An exploratory study in eating disorder patients. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity. 2004;9:300-5.
58. Lobo I, da Luz FQ, Hay P, Gaeta TL, Teixeira PC, Cordás TA, et al. Is binge eating associated with poor weight loss outcomes in people with a high body weight? A systematic review with meta-analyses. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity. 2023;28(1):89.
59. Moore CF, Sabino V, Koob GF, Cottone P. Pathological overeating: emerging evidence for a compulsivity construct. Neuropsychopharmacology. 2017;42(7):1375-89.
60. Engel SG, Wonderlich SA, Crosby RD, Mitchell JE, Crow S, Peterson CB, et al. The role of affect in the maintenance of anorexia nervosa: evidence from a naturalistic assessment of momentary behaviors and emotion. Journal of Abnormal Psychology. 2013;122(3):709.
61. Wildes JE, Marcus MD, Bright AC, Dapelo MM. Emotion and eating disorder symptoms in patients with anorexia nervosa: an experimental study. International Journal of Eating Disorders. 2012;45(7):876-82.
62. Rodgers RF, Paxton SJ. The impact of indicated prevention and early intervention on co-morbid eating disorder and depressive symptoms: a systematic review. Journal of Eating Disorders. 2014;2(1):1-10.
63. Johnson C, Larson R. Bulimia: An analysis of moods and behavior. Psychosomatic Medicine. 1982;44(4):341-51.
64. Schmidt U, Tiller J, Blanchard M, Andrews B, Treasure J. Is there a specific trauma precipitating anorexia nervosa? Psychological Medicine. 1997;27(3):523-30.
65. Mason TB, Heron KE, Braitman AL, Lewis RJ. A daily diary study of perceived social isolation, dietary restraint, and negative affect in binge eating. Appetite. 2016;97:94-100.
66. Casasnovas C, Fernández-Aranda F, Granero R, Krug I, Jiménez-Murcia S, Bulik C, et al. Motivation to change in eating disorders: clinical and therapeutic implications. European Eating Disorders Review 2007;15(6):449-56.
67. Venturo-Conerly KE, Wasil AR, Dreier MJ, Lipson SM, Shingleton RM, Weisz JR. Why I recovered: a qualitative investigation of factors promoting motivation for eating disorder recovery. International Journal of Eating Disorders. 2020;53(8):1244-51.
68. Hilbert A, Pike KM, Goldschmidt AB, Wilfley DE, Fairburn CG, Dohm F-A, et al. Risk factors across the eating disorders. Psychiatry Research. 2014;220(1-2):500-6.
69. Brewerton TD. Stress, trauma, and adversity as risk factors in the development of eating disorders. The Wiley Handbook of Eating Disorders. Wiley (2015). p. 445-60.
70. Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. American Journal of Psychiatry. 2002;159(3):394-400.
71. Jewell T, Collyer H, Gardner T, Tchanturia K, Simic M, Fonagy P, et al. Attachment and mentalization and their association with child and adolescent eating pathology: a systematic review. International Journal of Eating Disorders. 2016;49(4):354-73.
72. Cardi V, Tchanturia K, Treasure J. Premorbid and illness-related social difficulties in eating disorders: an overview of the literature and treatment developments. Current Neuropharmacology. 2018;16(8):1122-30.
73. Solmi M, Radua J, Stubbs B, Ricca V, Moretti D, Busatta D, et al. Risk factors for eating disorders: an umbrella review of published meta-analyses. Brazilian Journal of Psychiatry. 2020;43:314-23.
74. Patton GC, Selzer R, Coffey C, Carlin JB, Wolfe R. Onset of adolescent eating disorders: population based cohort study over 3 years. BMJ. 1999;318(7186):765-8.
75. Krafchek J, Kronborg L. Stressful life events experienced by academically high-achieving females before the onset of disordered eating. Roeper Review. 2018;40(4):245-54.
76. Hubert Lacey J, Coker S, Birtchnell S. Bulimia: Factors associated with its etiology and maintenance. International Journal of Eating Disorders. 1986;5(3):475-87.
77. Laboe AA, Shah J, Fitzsimmons-Craft EE. The transition to college as a risk factor for eating disorders. Eating Disorders: An International Comprehensive View. Springer (2023). p. 1-17.
78. Currie A. Sport and eating disorders-understanding and managing the risks. Asian Journal of Sports Medicine. 2010;1(2):63.
79. Iuso S, Bellomo A, Pagano T, Carnevale R, Ventriglio A, Petito A. Sport activity as risk or protective factor in feeding and eating disorder. Behavioral Sciences. 2019;9(12):143.
80. Bratland-Sanda S, Sundgot-Borgen J. Eating disorders in athletes: overview of prevalence, risk factors and recommendations for prevention

and treatment. European Journal of Sport Science. 2013;13(5):499-508.

81. Milano W, Milano L, Capasso A. Eating disorders in athletes: from risk management to therapy. Endocrine, Metabolic and Immune Disorders-Drug Targets. 2020;20(1):2-14.
82. Kluck AS. Family factors in the development of disordered eating: integrating dynamic and behavioral explanations. Eating Behaviors. 2008;9(4):471-83.
83. Herle M, De Stavola B, Hübel C, Abdulkadir M, Ferreira DS, Loos RJ, et al. A longitudinal study of eating behaviours in childhood and later eating disorder behaviours and diagnoses. The British Journal of Psychiatry. 2020;216(2):113-9.
84. Keel PK, Klump KL. Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. Psychological Bulletin. 2003;129(5):747.
85. Ousley L, Cordero ED, White S. Fat talk among college students: How undergraduates communicate regarding food and body weight, shape and appearance. Eating Disorders. 2007;16(1):73-84.
86. Culbert KM, Racine SE, Klump KL. Research Review: What we have learned about the causes of eating disorders–a synthesis of sociocultural, psychological, and biological research. Journal of Child Psychology and Psychiatry. 2015;56(11):1141-64.
87. Allison S, Warin M, Bastiampillai T. Anorexia nervosa and social contagion: clinical implications. Australian and New Zealand Journal of Psychiatry. 2014;48(2):116-20.
88. Hagan MM, Shuman ES, Oswald KD, Corcoran KJ, Profitt JH, Blackburn K, et al. Incidence of chaotic eating behaviors in binge-eating disorder: contributing factors. Behavioral Medicine. 2002;28(3):99-105.
89. Rossotto E, Rorty-Greenfield M, Yager J. What causes and maintains bulimia nervosa? Recovered and nonrecovered women's reflections on the disorder. Eating Disorders. 1996;4(2):115-27.
90. Dane A, Bhatia K. The social media diet: A scoping review to investigate the association between social media, body image and eating disorders amongst young people. PLOS Global Public Health. 2023;3(3):e0001091.
91. Sharma A, Vidal C. A scoping literature review of the associations between highly visual social media use and eating disorders and disordered eating: a changing landscape. Journal of Eating Disorders. 2023;11(1):170.
92. Rutter M. Psychosocial resilience and protective mechanisms. American Journal of Orthopsychiatry. 1987;57(3):316-31.
93. Levine MP, Smolak L. The role of protective factors in the prevention of negative body image and disordered eating. Eating Disorders. 2016;24(1):39-46.
94. Langdon-Daly J, Serpell L. Protective factors against disordered eating in family systems: a systematic review of research. Journal of Eating Disorders. 2017;5(1):1-15.
95. Allen KL, Gibson LY, McLean NJ, Davis EA, Byrne SM. Maternal and family factors and child eating pathology: risk and protective relationships. Journal of Eating Disorders. 2014;2:1-14.
96. Neumark-Sztainer D, Wall M, Story M, Sherwood NE. Five-year longitudinal predictive factors for disordered eating in a population-based sample of overweight adolescents: Implications for prevention and treatment. International Journal of Eating Disorders. 2009;42(7):664-72.
97. Ahrén JC, Chiesa F, Koupil I, Magnusson C, Dalman C, Goodman A. We are family - parents, siblings, and eating disorders in a prospective total-population study of 250,000 Swedish males and females. International Journal of Eating Disorders. 2013;46(7):693-700.
98. Croll J, Neumark-Sztainer D, Story M, Ireland M. Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. Journal of Adolescent Health. 2002;31(2):166-75.
99. Berge JM, MacLehose R, Loth KA, Eisenberg M, Bucchianeri MM, Neumark-Sztainer D. Parent conversations about healthful eating and weight: associations with adolescent disordered eating behaviors. JAMA Pediatrics. 2013;167(8):746-53.
100. Neumark-Sztainer DR, Wall MM, Haines JI, Story MT, Sherwood NE, van den Berg PA. Shared risk and protective factors for overweight and disordered eating in adolescents. American Journal of Preventive Medicine. 2007;33(5):359-69. e3.
101. Cordero ED, Israel T. Parents as protective factors in eating problems of college women. Eating Disorders. 2009;17(2):146-61.
102. Neumark-Sztainer D, Eisenberg ME, Fulkerson JA, Story M, Larson NI. Family meals and disordered eating in adolescents: longitudinal findings from project EAT. Archives of Pediatrics and Adolescent Medicine. 2008;162(1):17-22.
103. McVey GL, Pepler D, Davis R, Flett GL, Abdolell M. Risk and protective factors associated with disordered eating during early adolescence. The Journal of Early Adolescence. 2002;22(1):75-95.
104. Perkins DF, Luster T, Jank W. Protective factors, physical abuse, and purging from community-wide surveys of female adolescents. Journal of Adolescent Research. 2002;17(4):377-400.
105. Calvete E, Las Hayas C, Del Barrio AG. Longitudinal associations between resilience and quality of life in eating disorders. Psychiatry Research. 2018;259:470-5.
106. Vanderlinden J, Kamphuis J, Slagmolen C, Wigboldus D, Pieters G, Probst M. Be kind to your eating disorder patients: the impact of positive and negative feedback on the explicit and implicit self-esteem of female patients with eating disorders. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity. 2009;14:e237-e42.
107. Leehr EJ, Krohmer K, Schag K, Dresler T, Zipfel S, Giel KE. Emotion regulation model in binge eating disorder and obesity-a systematic review. Neuroscience and Biobehavioral Reviews. 2015;49:125-34.
108. Prefit A-B, Candea DM, Szentagotai-Tătar A. Emotion regulation across eating pathology: a meta-analysis. Appetite. 2019;143:104438.
109. Shastri PC. Resilience: Building immunity in psychiatry. Indian Journal of Psychiatry. 2013;55(3):224.
110. Allen KL, Mountford V, Brown A, Richards K, Grant N, Austin A, et al. First episode rapid early intervention for eating disorders (FREED): from research to routine clinical practice. Early Intervention in Psychiatry. 2020;14(5):625-30.
111. Fukutomi A, Austin A, McClelland J, Brown A, Glennon D, Mountford V, et al. First episode rapid early intervention for eating disorders: a two-year follow-up. Early Intervention in Psychiatry. 2020;14(1):137-41.
112. Potterton R, Austin A, Flynn M, Allen K, Lawrence V, Mountford V, et al. "I'm truly free from my eating disorder": emerging adults' experiences of FREED, an early intervention service model and care pathway for eating disorders. Journal of Eating Disorders. 2021;9(1):1-14.
113. Harrer M, Adam SH, Messner EM, Baumeister H, Cuijpers P, Bruffaerts R, et al. Prevention of eating disorders at universities: a systematic review and meta-analysis. International Journal of Eating Disorders. 2020;53(6):813-33.
114. Ramos RG, Olden K. Gene-environment interactions in the development of complex disease phenotypes. International Journal of Environmental Research and Public Health. 2008;5(1):4-11.