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The Role of Genetics, Neurobiology, and Psychosocial Factors in the Development and Treatment of Eating Disorders: A Systematic Review

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Abstract

Eating disorders are severe complications affecting peoples' eating patterns, with erratic practices like purging behaviors, binge eating, and avoidance of food. These disorders result from different causes, including neurotransmitter imbalance, psychological factors like body-shaming, and genetic inheritance. The present review focuses on genetic, neurobiology and psychological factors to provide an understanding of the development of eating disorders and subsequent treatment. The present review was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) checklist that guided the evidence collection in the review to ascertain high quality. The Participant, Interventions, Comparison, Outcomes, and Studies (PICOS) protocol guided the establishment of potential studies' inclusion criteria. The Grading of Recommendations, Assessments, Development, and Evaluation (GRADE) approach was used to assess the certainty of evidence from the individual studies. Two independent reviewers were tasked with study selection, data collection, and study risk of bias assessed. The reviewers used the Cochrane risk of bias tool to assess the risk of bias in the included studies. Fifteen studies met the eligibility tests and were included in the review. A review of the evidence obtained from the included studies revealed that neurobiology, genetics and psychological factors influence the development of eating disorders like binge eating disorders, anorexia nervosa, and bulimia nervosa. In genetics, familial integration and heredity revealed the passing of the genes responsible for the development of anorexia nervosa in subsequent generations. Psychological factors and neurobiology affect self-esteem or body shaming and affect the balance of neurotransmitters in the brain, respectively. The treatment interventions aim to restore neurotransmitter levels and confidence after losing confidence and self-body hate due to bad shape. Genetics, neurobiological factors and psychological status are responsible for the development of eating disorders. Bulimia nervosa, binge eating disorder and purging behaviors result from unregulated 5-HT depletion, can be inherited or can result from body shaming. The evidence shows that eating disorders can be treated based on the aetiologies and pathophysiology.

Keywords: *Eating disorders, treatment, neurobiological factors, genetics, binge eating disorder, bulimia nervosa.*

1.0 Introduction

Eating disorders refer to erratic food intake and behaviors characterized by persistent and severe disturbance of eating patterns, distressed emotions and thoughts. The disorders are common in the general population. However, eating disorders have emerged among young adults and adolescents, especially in developing countries [1], [2]. The increasing incidence of eating disorders, alongside psychological distress, poses a great challenge to the medical fraternity due to the need for effective treatments.

Currently, there are pharmacological and non-pharmacological interventions used to manage eating disorders. The interventions designed to treat eating disorders align with the different causes, including psychological, genetic and neurobiological factors. The genetic aspect of eating disorders regards heredity and familial aggregation [3], [4]. The inheritance of the genes responsible for the development of eating disorders spans the neurobiological factors that involve the dysregulation of neurotransmitters associated with self-esteem, confidence and social security.

Psychological factors play crucial roles in the development and treatment of eating disorders. Negative body talks or body shaming is a crucial trigger of psychological distress, resulting in eating disorders, including emotional eating, and restrained eating [5]. The erratic eating patterns target weight loss or attain a lean body. The psychological aspects pertinent to the development and treatment is a social issue that spans the biological aspects of mental disorders. Body shaming and negative body talks lead to insecurity and body dissatisfaction

Body dissatisfaction remains one of the main causes of eating disorders across the world. Mainly, unhealthy weight loss behaviors, including purging behaviors, binge eating, and bulimia nervosa, are the main causes of eating disorders [6]. Often, obese persons and those who are dissatisfied with their bodies indulge in unhealthy weight loss behaviors, resulting in eating disorders. The current knowledge and evidence from studies suggest that medical professionals should sensitize the public on ideal weight loss practices and behaviors to cushion them from eating disorders.

The current interventions against eating disorders bank on evidence-based approaches and study results proving the effectiveness of the proposed interventions. Studies on the causes of eating disorders inform the potential intervention since treatment measures rely on the cause-and-effect relationships between eating disorders and their causes [7]. The current review investigates the role of genetics, neurobiology and psychological factors in the development of eating disorders. Enhanced understanding of the development of eating disorders based on genetics, psychological factors, and neurobiological aspects provides insights into clinical interventions and approaches that can improve the overall clinical outcomes. The review independently investigates neurobiology, genetics, and psychological factors to provide an understanding of how each of these elements influences eating patterns and can be used to treat the disorders.

2.0 Methods

The PRISMA checklist was used to improve the transparency in the present review [8]. The 27-item checklist informed the breakdown of the review and the evidence therein.

Eligibility criteria

The PICOS protocol guided the inclusion and exclusion criteria. The inclusion criteria determined literature searches to ensure comprehensive, bias-free searches [9]. Studies included in the present review met the following criteria:

P: Persons with eating disorders, BED, and BN who have undergone treatments

I: Cognitive behavioural therapy, MDMA treatment

C: Placebo.

O: Psychological factors associated with BN and BED, including low self-esteem, body dissatisfaction, anxiety, stress.

S: Randomized controlled trials

The following exclusion criteria were used in the present review:

- Studies reporting outcomes irrelevant to the present topic were excluded.
- Low-quality studies that lost data or outcomes.
- Authors' views or opinions were not included in the study.

Information sources

The literature search was performed on electronic databases. The electronic article search was performed on Google Scholar, PubMed, ProQuest, and the Cochrane Library of Randomized controlled trials. Additionally, articles were filtered based on language and data of publication. Only records published in English from January 2013 to November 2023 were considered.

Search strategy

A comprehensive literature search was performed on electronic databases (PubMed, Scopus, Google Scholar, and PsychINFO) using a combination of MeSH terms and keywords associated with neurobiology, genetics, eating disorders, psychological factors, and treatment. Boolean operators combined the keywords and MeSH terms during the literature search. The Boolean operator "AND" combined words with dissimilar meanings, whereas the Boolean operator "OR" combined keywords and MeSH terms with dissimilar meanings.

Selection process

Two independent reviewers (K.O. and K.L.) screened the titles and abstracts of the eligible studies against the inclusion and exclusion criteria. The authors assessed the titles of studies deemed eligible for inclusion. Further, the author critiqued the abstracts of the individual studies to ascertain relevance. The assessment ensured the inclusion of studies reporting outcomes of interest in the present review.

Data collection process

After obtaining the studies that met the inclusion criteria, the independent reviewers banked on a standardized form while extracting the relevant information. The independent reviewers focused on outcomes relevant to the present topic, including the role of genetics, neurobiology, and psychological issues that develop eating disorders. The author separately assessed studies reporting these variables in relation to the topic. Issues arising in the individual studies are discussed and resolved via dialogue. The relevant data were extracted into an Excel sheet.

Data items

The independent reviewers extracted the data from the individual studies, including authors, year of publication, study design, participant features, interventions and the primary outcomes. The primary outcomes of focus regard the role of genetics, psychological factors, and neurobiological aspects pertinent to developing and treating eating disorders. Evidence pertinent to eating disorders, including anorexia nervosa, binge eating disorder, and bulimia nervosa, were collected and recorded in the Excel sheet.

Study risk of bias assessment

The Cochrane Risk of Bias tool was used to assess the risk of bias in the included studies. The tool examines internal validity through domains, including bias due to the randomization process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in the measurement of the outcomes, bias in the selection of the reported result, and the overall risk of bias [10]. The independent reviewers judged the risk of bias in each domain as "Low," "High," or "Unclear."

Synthesis methods

The evidence obtained from the included studies was qualitatively critiqued and discussed for comprehension. The overall outcomes and evidence reported regarding the role of genetics, neurobiology, and psychological factors were analyzed and compared to the existing literature or previous studies to determine whether the evidence aligns with the existing evidence.

Reporting bias assessment

The independent reviewers summarized the risk of bias assessment of the included studies in tables and graphs. The figures and tables highlighted the risk of bias assessment outcomes in every domain, including selection bias, performance bias, detection bias, attrition bias, reporting bias, and other forms of biases. The graphs represented the independent reviewers' judgment of the risk of bias in the individual studies.

Certainty assessment

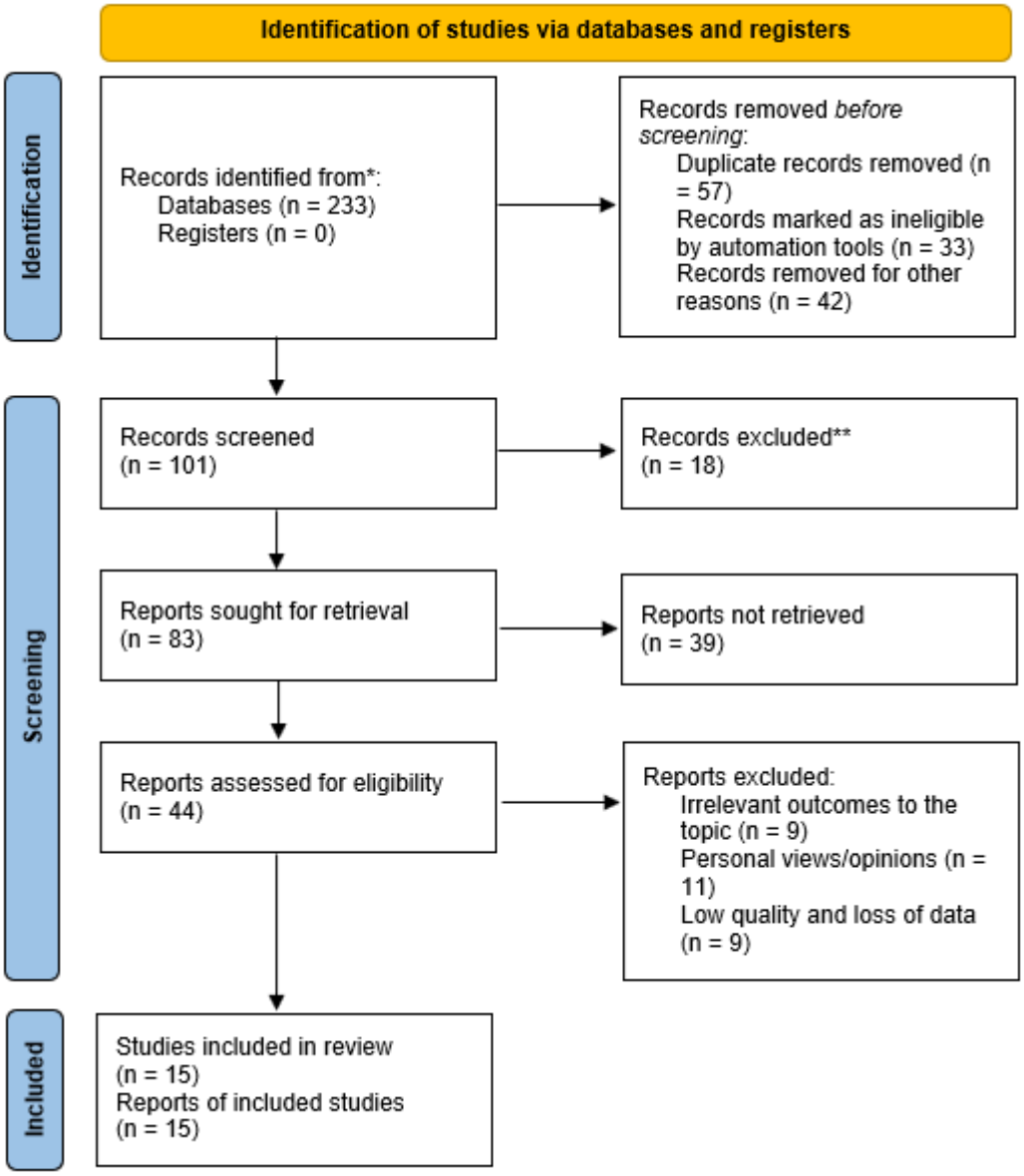
The GRADE approach was used to assess the level of certainty of evidence in the systematic review. The assessment considered factors affecting the certainty of the evidence, including the risk of bias, inconsistency of reported outcomes, indirectness of the available evidence, imprecision of effect estimates, and publication bias [11], [12]. The overall certainty of the evidence of the individual studies depended on the assessment outcomes of the individual domains mentioned above.

3.0 Results

Study selection

The independent reviewers identified 233 articles in the electronic databases in the initial literature search. Out of this, 132 articles were removed before the screening process with reasons, including study duplicates, studies marked as ineligible by automation tools, and other reasons. One hundred and one articles were left for screening, where eighteen articles were excluded. The remaining eighty-three articles were sought for retrieval, where thirty-nine articles were not retrieved. Forty-four articles were assessed for eligibility against the previously stated inclusion criteria. Twenty-nine articles were excluded based on the inclusion criteria, leaving fifteen articles for inclusion in the review. The articles were excluded with reasons, including low-quality studies and loss of qualitative information relevant to the topic, authors' views and opinions about the role of neurobiology, psychological factors and genetics in the development and treatment of treating disorders, alongside irrelevant outcomes (**Figure 1).**

Figure 1: PRISMA flow diagram for the systematic review.



Study characteristics

A total of 15 studies met the eligibility criteria and were included in the present review. The studies reported the role of genetics, psychological factors, and neurobiology in the development of eating disorders. Of course, the 15 studies possessed unique features and characteristics. Four of the fifteen studies were level III evidence, comparative studies reported the role of genetics in the development and treatment of anorexia nervosa [13]–[16]. Even though the level of evidence may be deemed low, the studies reported concise evidence of the role of genetics in the development of eating disorders. Only one study was a cross-sectional study [17]. The remaining ten studies were randomized controlled trials. Out of this, three studies were randomized controlled trials [5], [18], [19], another three studies were double-blind, placebo-controlled pivotal trials [20]–[22], whereas another three studies were placebo-controlled, double-blind cross-over study [23]–[25]. Only one study was a phase one open-label study [26].

The included studies originated from different parts of the world and included multiracial participants. A total of six studies originated from the United States of America, representing the country contributing to the largest number of studies in the review [15], [16], [20]–[22], [26]. Three studies originated from Germany [23]–[25], whereas two studies originated from Norway [18], [19], and China [5], [17] each (**Table 1**). Likewise, one study originated from Denmark [14] and Finland [13], respectively.

Table 1: Study characteristic table

Study ID, year of publication	Study Design	Country of Origin	Participants	Interventions & Main outcomes
Yang et al., 2023	Cross-sectional survey	China	n=815 middle school students, average age: 13.01 years, 368 females & 401 males	WeChat-administrated questionnaires on social networking. The utilization of social platforms impacted on emotional eating and inflicted negative social comparisons
Yang et al., 2022	Cross-sectional study	China	n=5986 participants (45.9% males, 54.1% females, age range: 17-32, average age: 19.8 years)	Investigations on the mediating effects of smartphone addictions on depression revealed the consequential effects like disordered eating and body dissatisfaction.
Mathisen et al., 2020	RCT	Norway	76 females (18-40 years) undergoing physical exercise & dietary therapy and 73 females undergoing cognitive behavioral therapy	The cognitive behavioral therapy and the physical exercise and dietary intervention re-established self-worth and self-regulatory processes by normalizing eating patterns.

Mathisen et al., 2017	RCT	Norway	76 females (18-40 years) of BMI range of 17.5-35.0	the cognitive behavioral therapy & the physical exercise therapy was administered and followed up after 6, 12 & 24 months. These interventions proved to be alternative treatments for BED and BN
Brewerton et al., 2021	Clinical Trial	United States of America	36309 men (37-45% with BN & 21-26% ED).	The effectiveness and application of psychotherapy emerged among patients with ED-PSTD symptoms. This treatment restored body-image perceptions, alleviated socio-emotional processing and rigidity
Brewerton et al., 2023	Clinical Trial	United States of America	884 adults (≥18 years)	Mental health status and symptoms are concurrent
Brewerton et al., 2022	Clinical Trial	United States of America	n=157, 58 females & 89 males	A 6-month follow up study revealed the important intricacies of neurobiology in the development and modulation of mental health among patients.
Raevouri et al., 2008	Clinical trial	Finland	Finnish twins (N=2,426 women,	BN was not significantly prevalent among

			N=1,962 men with known zygosity, age: 22-28 years, sex: 53 males, 292 females	females with same-sex dizygotic or monozygotic pairs. In males, BN was too rare to be linked with zygosity. Thus, the unlikelihood of associating BN with sex composition in twin pairs or zygosity.
Kortegaard et al., 2001	Multicenter clinical study	Denmark	34142 young twins screened for BN	A significant difference across dizygotic and monozygotic pairs of twins denying BN and AN
Klump et al., 2001	Clinical Trial	United States of America	56 biological sibling pairs and 132 participants	59-82% of all participants reported disordered eating (overall eating pathology, body dissatisfaction, weight preoccupation, binge eating), attributing these effects to genetic factors.
Bulik et al., 2006	Clinical Trial	Sweden	31406 male and female twins	The Swedish twin registery reported AN's prevalence of was 1.20% in females and 0.29% in male.
Boehm et al., 2022	Double-blind crossover study,	Canada	22 weight-recovered female patients with a history of anorexia nervosa, and 22	5-HT dysregulation in AN was conformed by tryptophan depletion &

			age-matched female healthy controls	hyperserotonergic functioning
Stending et al., 2023	Double-blind crossover study	Germany	22 ED patients and 25 healthy controls	Decreasing 5-HT complemented tryptophan depletion, thus, disruption of the associated normal brain processes.
Weinert et al., 2020	Placebo-controlled, double-blind cross-over study	Denmark	22 ED patients and 25 healthy controls	Short-term TRP depletion impacts 5-HT alleviates mood and reduces anxiety in AN
Peck et al., 2023	Clinical trial	United States of America	10 females, mean age: 28.3 years, ethnicity: Hispanics & Caucasians	Participants met DSM-V criteria for BN, 25mg of synthetic psilocybin alleviated vital signs of suicidality
Kanen et al., 2021	Double-blind, placebo-controlled, randomized between-groups design	United Kingdom	47 healthy participants	Tryptophan depletion and serotonin significantly implicated psychopathology and affected emotional responsivity.

BN: Bulimia nervosa

BED: Binge eating disorder

DSM-5: Diagnostic and statistical manual of mental disorders-5

MDD: Major depressive disorder

PSTD: Post-traumatic stress disorder

LGBTQ+: Lesbian, gay, bisexual, and transgender community.

MDMA: 3,4-methylenedioxymethamphetamine

CBT: Cognitive behavioral therapy

5-HT: 5-hydroxytryptamine/serotonin

AN: Anorexia nervosa

ATD: Acute tryptophan depletion

Risk of bias in studies

The reviewers assessed the risk of bias and quality of evidence and reported low risk of bias across the studies. The Cochrane risk of bias assessment tool investigated the overall summary of the risk of bias on the domains, including bias due to the randomization process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in the measurement of the outcomes, bias in the selection of the reported result, and the overall risk of bias. The reviewers found a low risk of bias in all the included studies. Additionally, the reviewers summarized the overall risk of bias across the domains and found a low risk of bias.

Results of individual studies

Seven studies reported the role of psychological factors in the development and treatment of BN and BED across different mentally ill patients, including depressed persons, persons with PSTD, LGBTQ+, and MDD [5], [17]–[22], [27]. A preliminary review of evidence obtained from the seven studies strongly suggests that psychological factors predispose individuals to eating disorders. Body shaming, negative body talks and excess weight gain are key contributors to poor psychological status among most patients. The evidence strongly suggested that patients resort to emotional, restrained, and external eating as weight loss measures. Mainly, persons with BED and BN aimed at weight loss to conform to societal and social pressure on them due to excessive weight gain. However, treatment measures, including CBT and MDMA, provided relief from anxiety, low self-esteem, and loneliness resulting from psychological complications associated with eating disorders.

A review of five studies established the role of neurobiology in the development and treatment of eating disorders. The evidence pointed to ATD in the development of eating disorders among patients [23]–[26], [28]. The review established strong evidence suggesting that ATD triggers 5-HT depletion, hence anorexia nervosa. However, the indication of psychotropic medications restored 5-HT, alleviating symptoms of anorexia nervosa [23]. Symptoms of anorexia nervosa improved with the elevation of 5-HT among persons with eating disorders. Additionally, low 5-HT triggered anxiety and stress among patients with anorexia nervosa [24]. However, some of the patients with eating disorders reported short-term improvements, whereas some reported insignificant improvements in symptoms of anorexia nervosa.

Further, four comparison studies examined the role of genetics in the development and treatment of eating disorders in the United States of America, Denmark, and Finland. A preliminary review of the evidence shows that genetics plays a crucial role in the development of anorexia nervosa among twins [13]–[16]. Twins across the countries reported a relatively equal incidence of anorexia nervosa. The evidence asserted the development of anorexia nervosa among twins, creating a general trend of the aetiology of the eating disorder among individuals sharing genes. The general deduction from the evidence presented suggests that genetics and unshared environmental elements lead to the development of anorexia nervosa. However, the genetic aspect of eating disorders does not highlight an effective intervention.

Results of syntheses

The present review investigated the role of psychological factors in the development and treatment of eating disorders among PSTD, LGBTQ+, and MDD patients. The review found

strong evidence that psychological factors predispose mentally ill patients to erratic eating patterns like binge eating behaviors or bulimia nervosa [18]–[22], [27]. The incidence of psychological conditions like low self-esteem, PSTD, MDD, anxiety, loneliness, and the quest for perfectionism aligned with the incidence of BN and BED. The patients tended to control the amount of food due to low psychological status. The results of the present review align with the literature and theoretical perspective, suggesting that poor psychological status results in eating disorders [29], [30]. In response, interventions improving the overall psychological condition alleviated symptoms of eating disorders. The implementation of psychological interventions like CBT improves mood, self-esteem and confidence that most mentally ill patients lack approach to eating. Previous evidence suggests that CBT is an effective intervention against BN, supporting the present evidence [31]. These outcomes emphasize that improving psychological status is a crucial step to eradicating BN and BED.

An investigation on the role of neurobiology in the development and treatment of eating disorders focused on neurobiological aspects. The review established a direct correlation between neurobiology and the development of eating disorders. A preliminary review of evidence obtained from the five studies indicates that low serotonin causes vulnerability to anorexia nervosa [23]–[26], [28]. The evidence demonstrates that dysregulation of serotonin predisposes individuals to anorexia nervosa. The physiological balance of 5-HT is the neurobiological aspect of developing eating disorders. ATD triggers 5-HT depletion or downregulation, resulting in stress, destabilized mood, and overall vulnerability to anorexia nervosa. Even though a section of the patients did not report convincing evidence on the role of neurobiology in the development of anorexia nervosa, the evidence associating low 5-HT with the development of the disorder sufficiently justifies a causal-effect relationship. Additionally, restoration of 5-HT to the ideal physiological levels significantly downregulated anorexia disorder.

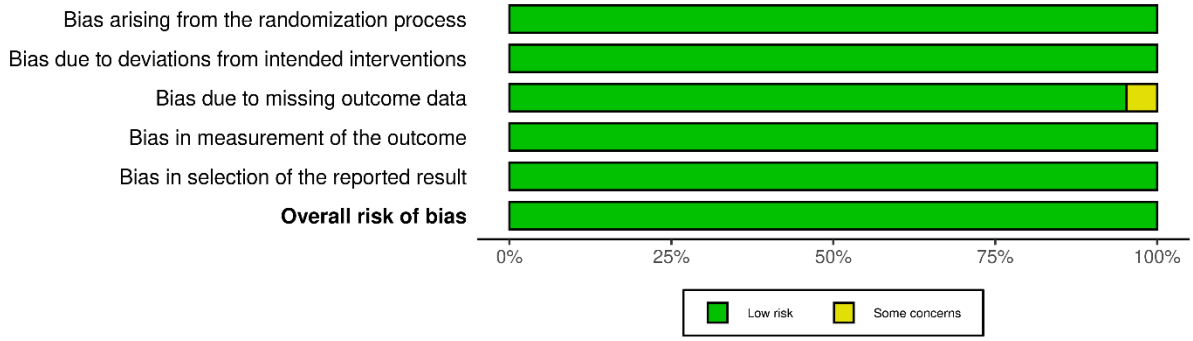
To decipher the role of genetics in the development and treatment of eating disorders, a review of comparative studies reporting the effect of genetics on the incidence and progression of the disorder was assessed. Evidence collected from twins from the United States of America, Finland and Denmark demonstrated the development of anorexia nervosa among twins, with a slight difference in the incidence among males and females [13]–[16]. A notable trend regards the consistent development of anorexia nervosa among twins from different countries. The consistency affirms the role of genetics in the development of anorexia nervosa among twins and heredity. Previous genome-wide association studies and research found that heredity and familial aggregation significantly contribute to the development of eating disorders among children and subsequent generations [32], [33]. Various aspects of genetic involvement in anorexia nervosa development. Genes like *0PRD1*, and *EPHX2* have been reported to cause anorexia nervosa and are known to substantiate the role of genetics in the development of eating disorders.

Reporting biases

The Cochrane risk of bias assessment tool assessed the risk of bias in the individual studies and summarized the overall outcomes of the domains. The authors judged the overall risk of bias in the domains, including bias due to the randomization process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in the measurement of the outcomes, bias in the selection of the reported result, and the overall risk of bias. Based on the risk of bias in the individual domains, the reviewers judged that the overall risk of bias in the included studies was low. All the domains, except bias arising from missing outcome data that reported a slightly unclear risk of bias, were found to have a low risk of bias (**Figure 2**). The

unclear risk of bias in the domain of missing outcome data was negligible and cannot impugn the interpretation of results.

Figure 2: A summary of the risk of bias of the included studies



The reviewers assessed the risk of bias in the individual studies across the domains to determine the risk of bias. According to the reviewers' judgement, all the included studies were found with a low risk of bias across bias due to the randomization process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in the measurement of the outcomes, and bias in the selection of the reported result. Four out of the fifteen studies were found with unclear risk of bias in the domain of bias due to missing outcome data [5], [15]–[17].

Figure 3: Risk of bias outcomes of individual studies

Certainty of evidence

The GRADE tool was used to assess the overall certainty of evidence of studies reporting evidence regarding the psychological factors influencing the development and eating disorders, the neurobiological and genetic factors affecting the development and treatment of eating disorders. The categorized approach collectively assessed the risk of bias, inconsistency, indirectness, imprecision of evidence, alongside other risk of bias of the studies reporting a given outcome. The oval certainty of evidence of the studies in the reported outcomes was low, with a high level of evidence (**Table 2**).

Table 2: GRADE assessment table

No of studies	Design	Risk of bias	Inconsistency	Indirectness[1]	Imprecision	Other[2]	Certainty (Overall score)[3]
Outcome: Psychological factors influencing the development and treatment of eating disorders							
7	Randomized controlled trials and cross-sectional study	No risk of bias	⊕	⊕	⊕	⊕	⊕⊕⊕⊕
Outcome: Neurological factors involved in the development and treatment of eating disorders							
5	Randomized controlled trials	No risk of bias, phase 1 label study, cross-sectional study	⊕	⊕	⊕	⊕	⊕⊕⊕⊕
Outcome:							

4 ⊕⊕⊕⊕ **High** = This research provides a very good indication of the likely effect. The likelihood that the effect will be substantially different** is low.

3 ⊕⊕⊕◯ **Moderate** = This research provides a good indication of the likely effect. The likelihood that the effect will be substantially different** is moderate.

2 ⊕⊕◯◯ **Low** = This research provides some indication of the likely effect. However, the likelihood that it will be substantially different** is high.

1 ⊕◯◯◯ **Very low** = This research does not provide a reliable indication of the likely effect. The likelihood that the effect will be substantially different** is very high.

** Substantially different = a large enough difference that it might affect a decision

			⊕	⊕	⊕	⊕	⊕⊕⊕⊕
Outcome: The role of genetics in the development and treatment of anorexia nervosa							
4	Comparative studies	Not serious risk of bias	⊕	⊕	⊕	⊕	⊕⊕⊕⊕

4.0 Discussion

Eating disorders are severe disorders affecting overall mental and physical well-being. Often, the physical status. Individuals suffering from eating disorders like BED and BN face stigma and intimidation due to poor physical and mental status [34]. The present review found evidence concurring with literature and theoretical orientations on the incidence and treatments of eating disorders. Poor psychological states resulting from mental illnesses, including PSTD, MDD, and anxiety, are associated with eating disorders. Often, the incidence of eating disorders is evidenced through loneliness, low self-esteem, and body image dissatisfaction. The theoretical orientation posits that low self-esteem and body-image dissatisfaction result in emotional eating, restrained eating, and external eating [17]. When left untreated, the eating disorders take a toll on the patients, resulting in depression and uncontrollable feelings of self-hate. The review found that psychological interventions, especially CBT and MDMA administration, improved patients' overall psychological status, providing relief against restrained eating. Reducing societal, family pressure and negative body talks or body shaming significantly alleviates low self-esteem, body dissatisfaction, and overall confidence. Reducing external pressure associated with the different psychological complications improves patients' attitudes towards eating, eradicating emotional eating, binge eating, and emotional eating.

The nexus between neurobiology and eating disorders regards brain structure and functional aspects contributing to binge eating, food restriction, and purging behaviors [35], [36]. The neurobiological aspects unmask psychiatric aspects of eating disorders, including brain structure, functions, and the involvement of neurotransmitters. The present review established substantial evidence pointing at 5-HT's involvement in the development of anorexia nervosa. Five studies reported that low 5-HT levels were associated with AN's development, whereas restoring ideal or physiological 5-HT levels was reported as an effective intervention [23]–[26], [28]. This evidence suggests that low 5-HT causes anorexia nervosa, whereas restoring 5-HT to the desired levels is an effective intervention. The review presented evidence putting neurobiology at the centre of the development and treatment of anorexia nervosa, suggesting the importance of neurobiology in eating disorders. Evidence obtained from the five studies suggested that ATD triggers a series of evidence leading to the development of anorexia nervosa. ATD downregulated 5-HT secretion, resulting in emotional eating, binge eating patterns and behaviors among participants. Additionally, ATD upregulated stress and anxiety towards food, suggesting key roles in the development of anorexia nervosa. This outcome aligns with the theoretical perspective and previous studies where low 5-HT secretion and alterations in 5-HT transportation results in AN [37], [38]. Likewise, the review convincingly proved that elevating 5-HT secretion alleviates symptoms of anorexia nervosa. The review found that modulating 5-HT levels is key to alleviating symptoms of anorexia nervosa.

However, a section of the participants reported short-term alleviation of symptoms of anorexia nervosa [25], and insignificant alleviation of anorexia nervosa [26]. This evidence raises the effectiveness of using a neurobiological approach in the treatment of anorexia disorder. Clinically, the short-term effects indicate possible relapse of the eating disorders due to temporary modulation of 5-HT [39], [40]. The current evidence demonstrates that neurobiology plays a crucial role in the development of eating disorders. This marks a significant point in understanding the incidence and development of eating disorders and the subsequent treatment.

Future studies should consider investigating the role of neurotransmitters in developing and treating neurological disorders. Dysregulation of neurotransmitters, including 5-HT, suggests that medications affecting neurotransmitter secretion influence the development of eating disorders. However, some of the medications are effective interventions for psychiatric illnesses. The development of eating disorders as an adverse effect of psychiatric intervention marks a grey area in psychiatric clinical practices and suggests the need for further studies to harness the incidence of adverse effects and improve clinical outcomes.

The present review examined evidence about the role of genetics in the development and treatment of eating disorders. The study established that heredity and familial integration triggers eating disorders, especially anorexia nervosa. Evidence obtained from four studies indicated the incidence of anorexia nervosa among twins from the United States of America, Finland and Denmark [13]–[16], affirming the role of genetics in the development of eating disorders. However, there was a notable difference in the development of anorexia nervosa among males and females. The disparity in the development of anorexia nervosa among males and females sparks the need for further studies to decipher the rationale for the difference [13].

The treatment of genetic-related eating disorders takes a different path of clinical interventions. Currently, advanced studies, including genome-wide association studies, establish particular genes and the underlying biomolecular processes involved in the disease processes. The genome-wide association studies focus on phenotypic refinement, genetic analysis, nosology and the implementation of endophenotypes in managing gene-associated eating disorders [12]. Currently, emerging evidence posits that the genome-wide associations could be a turning point for the treatment of gene-associated eating disorders.

5.0 Conclusion

In conclusion, the present review unmasked fundamental evidence substantiating a comprehension of the neurobiological, psychological, and genetic factors contributing to the development and treatment of eating disorders. The evidence focused on the multifaceted relationships between eating disorders like BED, BN and anorexia nervosa with neurobiology, genetics and psychological factors among patients. The review yielded fundamental evidence that enhances comprehension of the previously less understood field of psychiatry.

The review convincingly established that BN, BED, and anorexia nervosa result from genetic factors, neurobiological aspects, including brain structure and functions of the neurotransmitters, and the implication of psychological factors among patients. The incidence of eating disorders follows the unique aetiologies that are particular to the different causes. In response, the interventions aim at modulating the psychological effects, genetics, and neurobiological aspects pertinent to the development of eating disorders. Thus, the evidence emphasizes that treatment modalities for the eating disorders.

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