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The Characteristics, Risks, Effects, and Pathophysiology of Eating Disorders -A Review of Anorexia Nervosa and Bulimia Nervosa

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Abstract: ***Over the past few centuries, the importance of fitness has gained prominence. Coupled with this is the idea of thinness or the need to be slender and lean as an ideal body type. Thinness and fitness are not the same but are conflated in most messages about being fit. Many individuals, especially adolescents, have been influenced by the ongoing diet culture and social media. They have an ideal body or physique and are increasingly concerned about their weight, shape, size, and body image. As an attempt to lose weight and reach their goals, individuals go to extremes and try to starve themselves and/or excessively exercise, self-induce vomiting, or misuse products like laxatives, diuretics and enemas. Starvation or other psychological discomforts can cause one to binge-eat then purge and feel satisfied for a short period of time, but it eventually leads to self-loathing. This can severely affect the individual both physically and psychologically. As this cycle continues, an eating disorder is developed. Anorexia nervosa and Bulimia nervosa are the most widely recognized, and common eating disorders. Although there is little known about the pathophysiology of eating disorders, the evidence of eating disorders in the context of fitness and thinness, the potential genetic heritability, and developmentally specific age-of-onset shed some light on the aetiology of eating disorders. Research on various reactions of a brain neuropeptide called α-melanocyte-stimulating-hormone (α-MSH), the gut microbiota and brain axis and, the monoamines- dopamine and serotonin aid in the understanding of the pathomechanisms behind eating disorders. This review aims to discuss the characteristics, risk factors, effects and the other features of anorexia nervosa and bulimia nervosa. It also elaborates on the pathophysiology of eating disorders.***
Keywords: Eating Disorders, Psychology, Neuroscience, Anorexia Nervosa, Bulimia Nervosa

I. INTRODUCTION

Eating or feeding disorders are mental disorders that are defined as a persistent disturbance in eating behaviours that cause significant changes in the consumption and absorption of food that lead to impaired physical and psychological functioning.
The different eating disorders (ED) mentioned in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) are Pica, Rumination Disorder, Avoidant/Restrictive Food Intake Disorder (ARFID), Anorexia Nervosa (AN), Bulimia Nervosa (BN), Binge-Eating Disorder (BED), Other Specified Feeding or Eating Disorder (OSFED) and Unspecified Feeding or Eating Disorder (UFED).

A. Anorexia Nervosa

1) Definition: Anorexia is literally defined as ‘a loss of appetite’. It is derived from the Greek word ‘orexis’ which means appetite.

2) Characteristics

a) Significantly low body weight due to restrictive energy consumption. The significantly low body weight is a weight lower than the minimum in the context of age and sex. This adversely affects physical health, and in the case of children and adolescents, disrupts their developmental trajectory.

b) Unwarranted fear of gaining weight or fat even though the individual is at a significantly low weight.

c) Distorted body image and inability to acknowledge their current low body weight.

3) Subtypes

a) Restricting type - Weight loss is achieved through dieting, fasting and excessive exercise.

b) Binge-eating/Purging type - Repeated incidents of binge eating (uncontrollable consumption of unusually large amounts of food in one sitting) or purging (i.e., self-induced vomiting or the misuse of laxatives, diuretics or enemas) behaviour. Over the course of the disorder, crossovers between the subtypes are common, hence diagnosis of subtypes relates to symptoms shown over a period of 3 months rather than a long period.

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4) *Severity:* According to the World Health Organization (WHO) the severity of anorexia nervosa is determined by the current body mass index (BMI) for adults and BMI percentile for adolescents.

Table I - Severity of ANOREXIA NERVOSA BASED on BMI (SOURCE: DSM - 5)

Mild	BMI $\geq$ 17 kg/m^2
Moderate	BMI 16-16.99 kg/m^2
Severe	BMI 15-15.99 kg/m^2
Extreme	BMI < 15 kg/m^2

5) *Prevalence and Development:* Among young females, the prevalence of anorexia nervosa for a period of 12 months is approximately 0.4%. The lifetime prevalence of anorexia nervosa among females is 0.5-2%, but this number might be growing. Cases of anorexia nervosa are rarer in males than in females with an approximate female-to-male ratio of 10:1. Anorexia nervosa usually develops during adolescence or young adulthood and it rarely develops before puberty or past the age 40. It usually starts developing due to a stressful life event but the course and outcomes of the disorder are very different in individuals. Research shows that around 40-80% of individuals with anorexia nervosa show excessive levels of physical activity. They develop a compulsive need to exercise and partake in rigorous physical activity. This hyperactivity plays a major role in the development of anorexia nervosa and it may precede food restriction, or help maintain the disorder and accelerate weight loss alongside restriction of food. The main causes of hyperactivity have been assumed to be dopamine-reinforcing pathways rewarding activity due to the reduced calorie intake and hypothermia which cause hyperleptinemia and thermoregulatory compensation. Enhanced mesolimbic dopamine release is mediated by an activation of the hypothalamo-pituitary-adrenal axis with high blood cortisol levels as a result of hunger and/or hyperactivity causing the feeling of euphoria and reliance. As activity levels persistently increase, a negative energy balance (a condition in which less energy, i.e., food is consumed than expended by metabolism) is created. Most individuals take time to develop anorexia nervosa, starting with changes in eating behaviour that eventually match all the criteria. Some individuals recover after one incident of anorexia nervosa but many have a non-linear recovery. This may include fluctuations in weight and relapses that occur over the course of many years. Outcome studies suggest that a little less than half of anorexia nervosa patients fully recover and a third experience partial remission, but some eating-disorder symptoms persist.

6) *Risk Factors:* Individuals who develop anxiety or obsessive-compulsive disorders during childhood are at a high risk of developing anorexia *nervosa*. It is prevalent in cultures and environments where thinness is valued and encouraged. Fashion models and elite athletes are at high risk as low-fat mass is considered advantageous in those professions. Using functional imaging technology (functional magnetic resonance imaging, positron emission tomography) various brain abnormalities have been identified in patients. There is a good amount of evidence that suggests that genetic factors play a role in vulnerability to eating disorders. Individuals whose first-degree biological relatives have had anorexia and/or bulimia nervosa are at a higher risk for developing both, as well as bipolar and depressive disorders. Those whose relatives have had the binge-eating/purging type are also at a higher risk. The risk of anorexia nervosa or bulimia nervosa among females whose relatives have an eating disorder is 11 times that of the general population. Rates of concordance in anorexia nervosa are higher for monozygotic twins than those for dizygotic twins. Around 58-76% of the variance in the occurrence of anorexia nervosa may be due to genetic factors. The development of secondary sex characteristics during adolescence or young adulthood suggest that puberty might be a risk factor. Oestrogen and other reproductive hormones tend to affect mood and feeding patterns in humans. Information about anorexia nervosa appearing while the hypothalamic-pituitary-gonadal axis matures and the fact that it more commonly occurs in females suggest that some individuals are sensitive to these changes in development. The role of gonadal hormones, development of motivational neurocircuitry, stress on reduced food intake, and excessive physical activity in animals give insight into the pathogenesis of anorexia nervosa. A Genome Wide Association Study (GWAS) done by the Eating Disorders Working Group of the Psychiatric Genomics Consortium (PCG-ED) including 3495 individuals with anorexia nervosa and almost 11,000 controls without eating disorder histories reported significant genetic parallels between anorexia nervosa and other mental disorders such as schizophrenia, personality traits such as neuroticism, and educational attainment. There were also parallels with metabolic and anthropometric traits, as well as, positive genetic correlations with high-density lipoprotein cholesterol and negative genetic correlations with BMI, obesity, fasting insulin and fasting glucose. The discovery of these correlations prompted reconceptualizing anorexia as a psychiatric disorder and as well as a metabolic condition.

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7) *Side Effects and Other Features:* The most commonly observed effect of anorexia nervosa is a significant decrease in metabolic expenditure. Other side effects are the following: Leukopenia (a condition where a person has a reduced number of white blood cells) with a loss of all cell types and apparent lymphocytosis (high lymphocyte (a subtype of white blood cells) count) due to the immune system working to fight off an infection or disease). Mild anaemia (a lack of healthy red blood cells to carry adequate oxygen to the body's tissues), thrombocytopenia (a condition in which one has a low blood platelet count), and bleeding problems can occur. Due to elevated blood urea nitrogen levels, dehydration may occur. Hypercholesterolemia and elevated hepatic enzyme levels are common. Occasionally, hypomagnesemia, hypozincemia, hypophosphatemia and hyperamylasemia are observed. Metabolic alkalosis (elevated serum bicarbonate), hypochloremia, and hypokalaemia may occur due to self-induced vomiting and laxative abuse may lead to a mild metabolic acidosis (an electrolyte disorder characterized by an imbalance in the body's acid-base balance). Thyroid abnormalities include serum thyroxine (T_4) levels generally staying in the range of low-normal, a decrease in triiodothyronine (T_3) levels increase in reverse T_3 levels. Hypothalamic dysfunction is reflected in individuals with amenorrhea and is characterized by low levels of Luteinizing hormone (LH), follicle-stimulating hormone (FSH), estrogen and progesterone. In males, a similar hypogonadism occurs with reduced testosterone levels. Sinus bradycardia is common and arrhythmias occur rarely. Low bone mineral density with specific areas of osteopenia or osteoporosis are seen quite often, increasing the risk of fractures. Due to significant fluid and electrolyte disturbances, diffuse abnormalities reflect a metabolic encephalopathy (a condition in which brain function is disturbed due to different diseases or toxins in the body). Emaciation, hypotension, hypothermia and bradycardia are commonly observed. Lanugo, a fine downy body hair, is developed in certain individuals when a they don't have enough body fat to keep themselves warm. During weight restoration or during the end of laxative and diuretic abuse, peripheral oedemas may develop. A rare symptom is petechiae or ecchymoses, this may be indicative of a bleeding diathesis. Due to hypercarotenemia, some individuals' skin may start to turn yellow. Those who self-induce vomiting may have hypertrophy of the salivary glands. Scars or bruises may develop on the dorsal surface of the hand due to repeated contact with the teeth while trying to induce vomiting. Most physical signs and symptoms of anorexia nervosa are due to starvation. As a consequence of the weight loss, or, in a minority, preceding the weight loss, amenorrhea is common and is an indicator of psychological dysfunction. In pre-pubertal females the menarche might be delayed. Other symptoms are constipation, abdominal pain, cold intolerance, lethargy, and excess energy. Functional limitations related to the disorder may be exhibited by individuals with anorexia. Some are unable to perform well socially and professionally whereas others show significant social distancing or failure to meet academic or career potential.
8) *Mortality Rates and Suicide Risk:* Anorexia nervosa has the highest mortality rate of all mental disorders. It is a chronic illness in around 20% of the individuals who develop it. The crude mortality rate (CMR) is approximately 5% per decade. Deaths are usually associated with medical complications related to the disorder itself or due to suicides. The suicide risk is elevated with reported rates as 12 per 100,000 per year.
9) *Comorbidities:* Various disorders including bipolar, depressive and anxiety disorders commonly occur alongside anorexia nervosa. Symptoms of anxiety disorder may be exhibited prior to or during the course of the eating disorder. Obsessive Compulsive Disorder (OCD) is present in some, especially those with the restricting type. In those with the binge-eating/purging type, alcohol and other substance usage disorders may be present.
10) *Treatment:* The main goal in the treatment of anorexia nervosa is weight restoration. Psychotherapy and medication will not be very useful until the physical and psychobiological consequences are reversed. Inpatient hospitalization to initiate weight gain might be required based on the severity. Meals and exercise will have to be closely monitored. A diet of 2000-4000 kcal per day can be implemented. Psychotherapy is crucial in both group and individual forms, during the weight restoration process. Measures will have to be taken to prevent relapse and the obsession with weight loss will have to be corrected.

B. Bulimia Nervosa

1) *Definition:* Bulimia is thought to be a derivation of the Greek words ‘buos’ and ‘limos’. It translates to ‘ox hunger’.
2) *Characteristics:*

a) Repetitive episodes of binge eating, i.e., eating a significantly large amount of food in a distinct period of time while sensing a lack of control over how much food one is eating. The amount of food consumed is more than most would consume in a similar period of time under similar conditions.
b) Constant measures to compensate for binge-eating and prevent weight gain being taken, such as fasting, excessive exercise, self-induced vomiting and abuse of laxatives, diuretics and other medications.

c) The episodes of binge-eating and inappropriate compensatory measures occur at least once a week for 3 months, on average.
d) Distorted body image and excessive influence of body shape and weight on self-evaluation.
e) Not occurring particularly during episodes of anorexia nervosa.

3) *Severity:* The level of severity is based on the frequency of inappropriate compensatory measures. It may be increased to show other symptoms and the level of functional disability.

Table 2 – Severity of Bulimia NERVOSA BASED on Frequency of Inappropriate Compensatory Behaviours (Source: DSM 5)

Mild	Around 1-3 episodes of such behaviours per week
Moderate	Around 4-7 episodes of such behaviours per week
Severe	Around 8-13 episodes of such behaviours per week
Extreme	14 r more episodes of such behaviours per week

4) *Prevalence and Development:* Among females, the 12 months prevalence of bulimia nervosa is 1%-1.5%. The lifetime prevalence among women is 1-3%. The disorder peaks in older adolescence and early adulthood so the point prevalence is high among young adults. There is very little information about the occurrence in males but bulimia nervosa is fairly uncommon in males in comparison to females, with a female-to-male ratio of 10:1. Males are seriously under-represented in treatment-seeking samples, for reasons currently unknown. Bulimia nervosa is not common among pre-pubertal individuals or those above the age of 40. The binge eating starts to occur frequently after an attempt to lose weight by dieting. The disturbed eating habits commonly last for several years according to clinic samples. Although, the course may be long-term or intermittent and periods of remission and binge eating may occur. Treatment clearly impacts the rate of recovery but many individuals recover without any treatment. Bulimia nervosa can begin during or following a diet. At some point, a compensatory method such as self-induced vomiting or usage of laxatives, diuretics and enemas is discovered to get rid of unwanted calories. This causes fluid loss that leads to weight loss. Medicative stimulants and thyroid replacements are obtained by some and diabetic patients omit insulin administration as an attempt to lose weight. This may feel rewarding and satisfying in the beginning as various foods can be consumed without gaining weight but eventually one loses control over eating habits and appetite. A vicious cycle develops as extreme caloric restriction and inappropriate compensatory behaviours cause hunger that leads to episodes of binge-eating. The frequency of these episodes increases and the quantity of food consumed becomes larger. Low mood, rejection, anxiety, and frustration provoke and precede binges. Other triggers are boredom and consumption of disinhibiting substances such as alcohol. Some individuals may plan binge-eating episodes and store and hide food to do so. There is a high level of secrecy in the planning and execution of the binge. Purging right after the binge offers a sense of relief to many, since it is assumed that they are rid of unwanted calories but self-loathing and anger or frustration usually follow. Individuals may experience a switch from bulimia nervosa to anorexia nervosa. This occurs in a minority of cases (10-15%). Many who have crossovers tend to revert back to bulimia nervosa or have multiple crossovers between the two. Individuals who partake in binge eating but do not take extreme compensatory measures meet the criteria for binge-eating disorder or other specified eating disorder.

5) *Other Features and Side Effects:* Individuals with bulimia nervosa are generally within the normal weight or overweight range, i.e., with a BMI ≥ 18.5 and < 30 in adults. Bulimia nervosa occurs among obese individuals but is fairly uncommon. Typically, individuals with bulimia nervosa, in between binge-eating episodes, restrict their total caloric intake and select low-calorie or 'diet' foods and avoid foods they recognize as fattening or that will likely trigger a binge. Several hundred to 10,000 calories can be consumed in a binge-eating episode but the most characteristic feature of a binge is the sense of losing control over what one consumes. Many individuals that suffer from bulimia have 'safe foods' and 'forbidden foods. The forbidden foods generally trigger binges and make one feel out of control while consuming it. Menstrual amenorrhea commonly occurs in females with bulimia nervosa. It is unknown whether this happens due to fluctuations in weight, nutritional deficiencies, or emotional distress. Due to the purging behaviour, fluid and electrolyte disturbances can cause medically severe issues. Oesophageal tears, gastric rupture and cardiac arrhythmias rarely occur but can be fatal. Among individuals who repeatedly use syrup of ipecac to induce vomiting, serious cardiac and skeletal myopathies have occurred. Those who continuously misuse laxatives may become dependent on them to stimulate bowel movements. Gastrointestinal problems and rectal prolapse are common. Various functional limitations are exhibited in individuals with bulimia nervosa. Only a minority of individuals report severe role impairment. Many persons' social lives are adversely affected.

6) *Risk Factors:* Individuals with weight concerns, low self-esteem, depressive symptoms, social-anxiety disorder, and overanxious disorder during childhood are at a higher risk for the development of bulimia nervosa. Individuals that internalize a thin body ideal tend to develop concerns with their weight, increasing their risk for the development of bulimia nervosa. Those who have experienced sexual or physical abuse during childhood are also at a higher risk. Obesity during childhood and early pubertal maturation increase the risk. There is very little information about the genetic epidemiology of bulimia nervosa in comparison to that of anorexia nervosa. Familial transmission may occur and those with family members diagnosed with bulimia nervosa are at a higher risk. Anorexia nervosa has been suggested as a risk factor for bulimia nervosa as studies suggest that 20-30% of individuals with bulimia nervosa have met the criteria for anorexia nervosa at some point in their lives.
7) *Mortality Rates and Suicide Risk:* Reportedly, individuals with bulimia nervosa have a significantly elevated risk for mortality. The CMR is nearly 2% per decade. These deaths happen either due to suicide or other causes.
8) *Comorbidities:* Most individuals with bulimia nervosa generally experience another mental disorder and the lifetime prevalence of comorbidities have been found in up to 75% of participants with bulimia nervosa. The comorbidities occur across a wide range of mental disorders. Depressive symptoms and bipolar and depressive disorders have an increased frequency in those with bulimia nervosa. Anxiety symptoms or anxiety disorders may also have an increased frequency. Mood disturbances may begin prior to the development of bulimia nervosa, during its course, or following its development. In most cases, the mood and anxiety disturbances cease after effective treatment of bulimia nervosa. Borderline personality disorder and other personality disorders may occur. Among individuals with bulimia nervosa, substance use, especially that of alcohol and stimulants, is at least 30%. Stimulants are usually used as an attempt to control appetite and weight.
9) *Treatment:* Bulimia nervosa patients are usually more open to treatment in comparison to anorexia nervosa patients. There are two methods of treatment. One is a 4-6 month long psychological treatment and cognitive behavioural therapy that focuses on differently organizing the maladaptive thoughts and behaviours that sustain the binge-eating/purging cycle. The second form of therapy is through the usage of antidepressants. Studies show that the use of antidepressants has shown effectiveness in reducing the frequency of binges. Although treatment can be successful, many individuals do relapse. 88 outcome studies were examined and they showed that 5-10 years post presentation, around 50% had completely recovered, 30% had relapsed, and 20% still met all the criteria for bulimia nervosa. Treatment has shown effectiveness in the short term but not much impact over the long term.

C. The Pathophysiology of Eating Disorders

The underlying pathomechanisms of eating disorders are poorly understood and they have an unknown origin. Eating disorders lack Pathophysiological definitions and we rely on the diagnostic criteria mentioned in the DSM-5.

Alterations in brain serotonin (5-hydroxytryptamine - 5-HT) contribute to various aspects of eating disorders such as appetite (satiety), perfectionism, impulsiveness and mood-regulation problems. Increased 5-HT activity causes increased, or prolonged satiety results in restrictive eating behaviours and reduced 5-HT activity leads to a periodic lack of satiation that causes compulsive or binge-eating behaviours. Therefore, it can be understood that the restrictive type of anorexia nervosa corresponds with increased neurotransmission of 5-HT and the binge-purge type of anorexia nervosa or bulimia nervosa coincide with decreased neurotransmission of 5-HT.

Even a minor level of dieting or restriction can cause a reduction in 5-HT activity. Studies have shown that dieting can cause significant changes in 5-HT activity more in women than in men. This indicates that alongside dieting, gender related serotonergic sensitivities can lead to changes in 5-HT transmission and are causative for eating disorders.

Studies show that excessive carbohydrate consumption during binges can cause the levels of large neutral amino acids in the plasma to reduce due to insulin. This increases the availability of

tryptophan (an essential amino acid) in the brain which accelerates 5-HT release, which eventually reduces the number of postsynaptic 5-HT receptors in the brain.

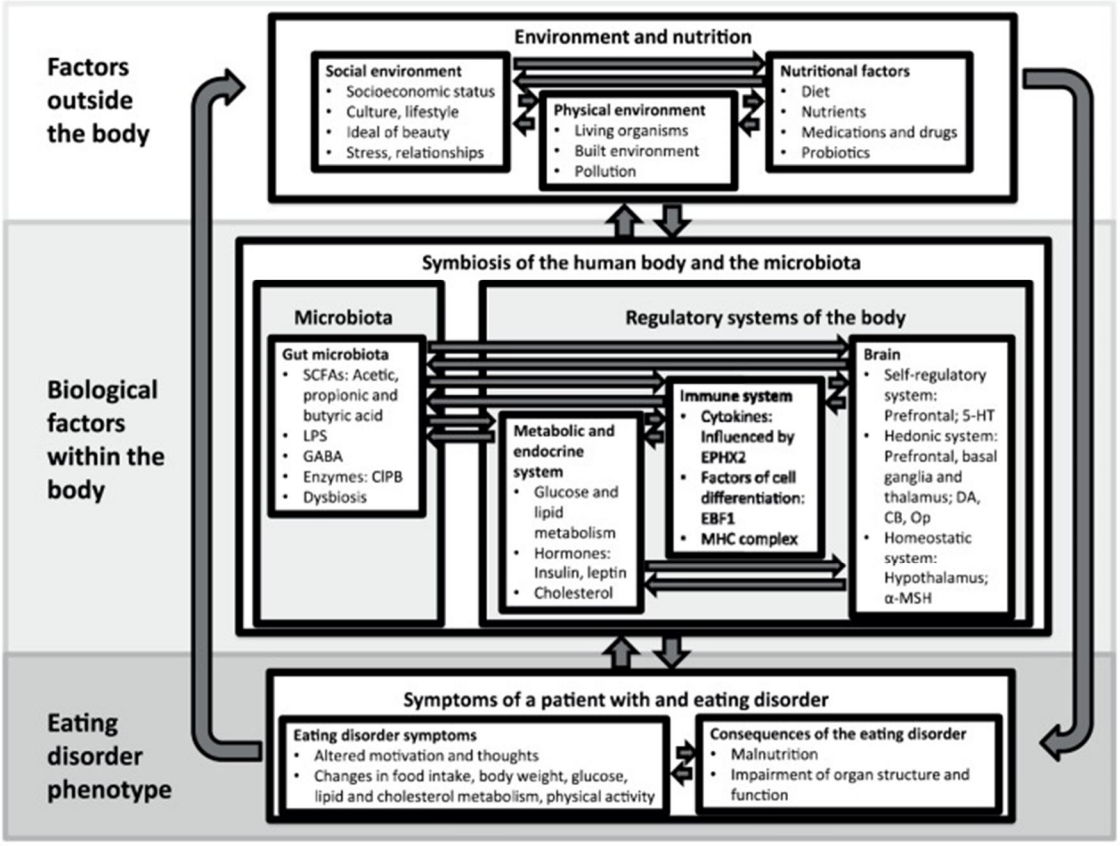
GWASs, epigenetic, gene-expression and gene–gene interaction projects, nutritional genomics suggest that genetics play a large role in the development of eating disorders.

Research about specific gut bacteria also contributes to the information about the pathophysiology of eating disorders. Gut microbiota has been shown to demonstrate involvement in various metabolic functions such as the regulation of weight gain, harvestation of energy from the diet and the secretion of insulin. It is majorly influenced by diet and lifestyle. Gut microbiomes reportedly produce various bioactive metabolic products that enter the systemic circulation. They are capable of producing extreme effects on metabolism, immune function, gene expression, as well as the central nervous system (CNS).

The microbiota is also capable of modulating the human brain. The microbes in the gastrointestinal tract change their host's eating behaviours to increase microbe fitness.

Cravings for foods are generated by specialized gut microbes. The intake of these foods causes dysbiosis, a disruption or imbalance caused in gut microbiome colonies, which alters the production of short-chain fatty acids such as acetic, propionic and butyric acid, and of particular enzymes such as α-MSH. This dysbiosis might interfere with a nutrient-deficient diet and affect energy metabolism and cause malnourishment to persist.

Figure I - A Pathophysiological Model of Eating Disorders Based on Research About Genetics and GUT Microbes (Source: Himmerich, H., Bentley, J., Genetic risk factors for eating disorders: an update and insights into pathophysiology)



Notes: SCFAs- Short Chain Fatty Acids; LPS- LipoPolySaccharides; GABA- Gamma-Aminobutyric Acid; B ClpB- Caseinolytic Protease; EBF1- Early B-Cell Factor 1; EPHX2- Epoxide Hydrolase 2; MHC- Major Histocompatibility Complex; 5-HT- Serotonin; DA- Dopamine; CB- CannaBinoid; Op- Opiods;
α-MSH- Melanocyte-Stimulating Hormone

The pathophysiological model of eating disorders should be based on molecular mechanisms that explain the alterations in appetite and feeding patterns, the mechanisms underlying alterations and diagnostic crossovers between different eating disorders and the risk factors that trigger the final common molecular pathway that leads to anorexia nervosa and/or bulimia nervosa.

Figure 1. is a suggested pathophysiological model of eating disorders based on external and internal factors that contribute to the development of an eating disorder and also the phenotypes of eating disorders.

II. CONCLUSIONS

Eating disorders have been an area of study since the 19th century and their incidence has been rapidly increasing over the past few decades. However, there are several areas which are not completely understood, both in terms of what causes them, and what their consequences are. Most current and available studies are focused on developed western countries such as North America and

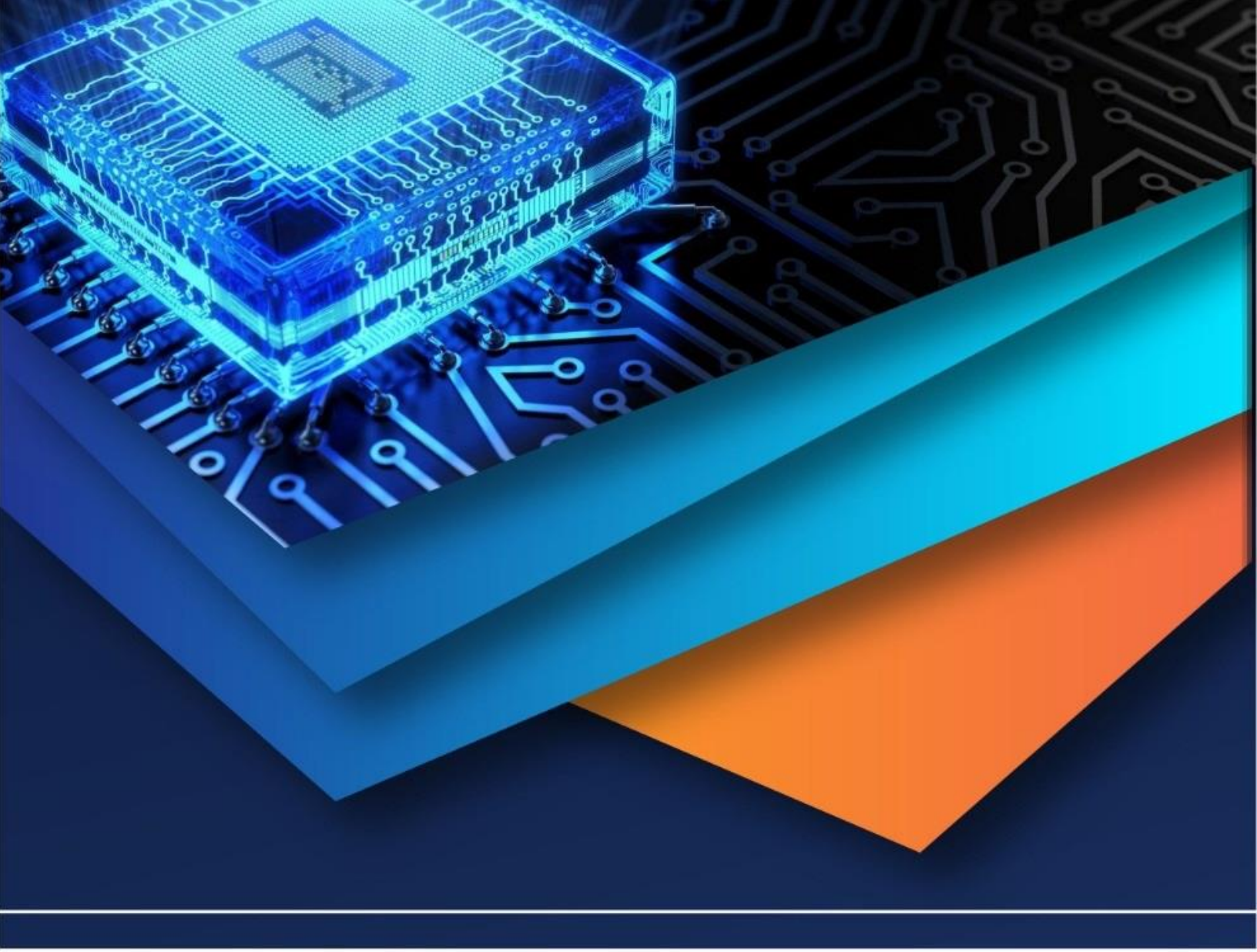
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Europe. Greater awareness about this topic needs to be generated and the stigma around eating disorders should be removed so that more individuals report the disorder and seek treatment and are able to take corrective measures at the right time.

Recent research has shown that eating disorders are not only mental disorders, but also biological disorders. Presently, there is very little information about the pathogenesis and pathophysiology of these disorders. Further research in these areas across diverse populations will aid in both prevention and more effective treatment of eating disorders.

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