

Metabolic Syndrome and Binge Eating in Children: A Comprehensive Review

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ABSTRACT

Children who binge eating are at a higher risk of becoming overweight. The Metabolic Syndrome (MetS) significantly predicts Type 2 diabetes and coronary heart disease in obese children and adolescents. The accumulation of cardiovascular risk factors known as Metabolic syndrome (MetS) elevates low serum High-Density Lipoprotein (HDL) cholesterol levels, hypertension, hypertriglyceridemia, cardiovascular disease risk, central adiposity, and fasting blood glucose levels in Low- and Middle-Income Countries (LMIC's). This study aims to illuminate new and existing ideas about Metabolic Syndrome (MetS) in children, from definition to diagnostic techniques. Relevant published articles up to 2024 were systematically retrieved from Google Scholar, MEDLINE/PubMed, SCOPUS, EMBASE, and WOS databases using keywords such as Metabolic syndrome, obesity, eating disorders, mental health screening, binge eating, and childhood obesity. The study examined the nutritional composition and metabolic characteristics of obese children and adolescents receiving treatment, along with their binge eating symptoms. We conclude from this analysis that Metabolic Syndrome (MetS) in children and adolescents is becoming an increasingly important public health issue in low- and middle-income nations with rising obesity rates. People who are overweight or obese have a substantially higher prevalence. Children and adolescents are at increased risk for developing metabolic syndrome due to the growing prevalence of obesity. An increase in body fat associated with binge eating helped to explain some of the negative consequences on children's metabolic health. Binge eating is an early behavioural indicator that may be targeted by obesity and metabolic syndrome therapy.

Keywords: Metabolic Syndrome, Obesity, Eating Disorders, Mental Health Screening, Binge Eating, Childhood Obesity.

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Received: 16-07-2025;

Revised: 03-09-2025;

Accepted: 28-10-2025.

INTRODUCTION

Binge Eating Disorder (BED) is characterized by repeated episodes of binge eating in which the person eats massive amounts of food and loses control over their eating habits. The eating disorder known as binge eating disorder is characterized by frequent, unpleasant bouts of binge eating that lack the improper compensatory behaviours seen with bulimia nervosa (Bechara *et al.*, 2002). The World Health Organization (WHO) has issued guidelines for treating children's obesity, overweight, and Binge Eating (BE) habits. In 2014, approximately 41 million children under the age of five were overweight or obese globally. In addition to the potential for physical and psychological effects throughout childhood, these children run the risk of developing

cardiovascular disease and diabetes and dying young. Primary healthcare providers should measure the weight and height of all newborns and children under five years old. Provide nutrition as well as physical activity counselling to parents and caregivers, encourage and promote exclusive breastfeeding for the first six months of a child's life and continue it for a full 24 months or longer, and create a suitable management strategy if a child is found to be overweight or obese (Güngör, 2014). A balanced diet with a range of food categories is crucial, according to the WHO. Fruits, vegetables, whole grains, dairy products, and lean proteins should all be consumed by children.

According to the guidelines, children should consume certain nutrients according to their developmental stage and age, such as enough protein for healthy growth and development, enough iron and calcium, as well as vitamins D, C, and A and added sugars and salt should be in an adequate amount. Throughout the day, children should eat regular meals and snacks. Their general health and energy levels may be impacted by missing nutritional



DOI: 10.5530/jyp.20250040

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meals. Children should be encouraged to drink water and minimize sugary drinks for proper hydration. Eat fewer highly processed meals that are heavy in artificial additives, bad fats, and added sugars. Eating meals as a family encourages healthy eating practices. Therefore, controlling children's binge eating requires early intervention as well as a supportive environment.

The World Health Organization Mental Survey Study (Kessler *et al.*, 2013), which included community surveys of 24,124 participants, found that lifetime prevalence rates in upper-middle-class and high-income countries averaged 1.4%. The information demonstrated that women are especially at risk and that the disease has become more prevalent in more recent cohorts. Using data from the Norwegian Twin Registry, (Reichborn-Kjennerud *et al.*, 2004) study discovered a 6-month prevalence of 3.8% in males and 5.2% in women. Hudson *et al.*, (2007) observed a 3.5% lifetime prevalence in their National Comorbidity Replication Study (Hudson *et al.*, 2007). It has also been demonstrated that individuals who are obese have higher rates of BED, with prevalence rates often falling between 3.5% and 5.5% (Herpertz *et al.*, 2006).

Children's Binge Eating Disorder (BED) is a serious problem that has to be understood to determine its demographic characteristics and the treatment, as well as its preventative implications. Research shows that in population samples, 1-3% of children and adolescents suffer from BED; in clinical settings, the prevalence is greater (Allen *et al.*, 2013). Though it may affect kids as young as five, BED is most often identified in adolescence and teenagers (Crow *et al.*, 2014). According to continued research, the frequency of BED reports is higher in girls than in boys, with a ratio of around 3:1. But BED in men is similarly higher and often unreported (Swanson *et al.*, 2011). All socioeconomic backgrounds exhibit BED, albeit children from lower socioeconomic status homes may not have as much access to therapy (Hay *et al.*, 2015). Many other ethnic groups have greater rates of BED than their Caucasian counterparts; studies have shown that African American and Hispanic children had higher rates of BED (Micali *et al.*, 2015). Therefore, it is essential to comprehend the demographic parameters of BED in children to implement guidelines and efficient interventions.

Metabolic Syndrome (MetS) refers to a set of cardiovascular risk factors that are common in obese Children and adolescents. These risks include dyslipidemia, increased blood pressure, altered glucose metabolism, and abdominal obesity (Wilson *et al.*, 2005). Childhood obesity rates are increasing, which is contributing to an increase in MetS risk in Children and adolescents (Seo *et al.*, 2021). We need a better understanding of MetS's pathogenesis, risk factors, and treatment techniques for children. The most common risk factors include increased blood pressure, obesity, reduced High-Density Lipoprotein Cholesterol (HDL-C), altered glucose metabolism, and elevated triglycerides (Reaven, 2002). One known effect of obesity is metabolic syndrome, which

can start in adolescence. Undoubtedly, not every child who is fat will have every obesity-related issue. It is yet unclear which obese children are more likely to develop MetS (Jankowska *et al.*, 2021). Despite attempts to encourage weight loss, obesity and overweight in children and adolescents are becoming more common. Disorder of binge eating is a phenomenon that is intimately linked to obesity; however, it is not limited to the condition (Ogden *et al.*, 2006). The sensation that one has lost control over one's eating habits and frequent bouts of binge eating, which entail ingesting vast quantities of food in a short period of time, are characteristics of a recently suggested mental condition termed binge-eating disorder. Although the binge-eating disorder is linked to subjective discomfort, bulimia nervosa patients self-induce vomiting or misuse laxatives as part of their purging behaviours (Wonderlich *et al.*, 2009). It is common, becoming more frequent, and often linked to other issues with mental and physical health, such as obesity (Roehrig *et al.*, 2009). In the US and Western Europe, its cumulative prevalence ranges from 1% to 3%. Research evaluating elements of the metabolic syndrome in compulsive eaters, but not all (Wonderlich *et al.*, 2009), research examining the frequency of compulsive eating in patients diagnosed with type 2 diabetes has raised the possibility of a connection between the binge eating disorder and metabolic syndrome. However, conclusions on the nature of the relationship between metabolic syndrome and binge eating disorder are limited since a large portion of the previous study was cross-sectional and may have been biased in terms of selection and reporting (Mitchell *et al.*, 2006).

METHODOLOGY

In 2024, an overview of literature review was carried out and updated in early 2025, mainly using Google Scholar, MEDLINE/ PubMed, SCOPUS, EMBASE, and WOS databases to analyze studies from the last decade (up to 2024) related to Metabolic syndrome and binge eating in children. The review included English-language articles exploring Dietary patterns, Binge-Eating Disorder (BED) and its pharmacotherapy, Metabolic Syndrome, Differences and connections between Dietary patterns, BE and MetS, Physiological Outcomes of Obesity in Children, Epidemiology and Mechanism and Risk Factors in Children. The search employed keywords such as Metabolic syndrome, obesity, eating disorders, mental health screening, binge eating, and childhood obesity. The results were compiled to highlight gaps in future research that need to be addressed.

RESULTS

Epidemiology

According to data gathered by the NHANES (2003-2004), 17.1% of kids and teens (2-19 years old) in the U.S. had a BMI that was at or above the 95th percentile for their respective ages. According to estimates from Brazil's Consumer Expenditure

Survey (2002-2003), 2.3% of kids and adolescents (10-19 years old) and 16.7% of overweight persons were obese (IBGE, 2006). According to the International Obesity Task Force guidelines, the age and sex-specific BMI cutoffs were set, which translated into a body mass index of between 25 and 30 at the age of 25, respectively, for these rates. Brazilians between the ages of 10 and 11 had the highest prevalence of overweight (22.4%).

Researchers observed varying prevalence estimates, ranging from 4.2% to 9.2% when estimating the prevalence of MS using the same data but different definitions. A few small-scale investigations conducted recently across India assessed the frequency of MS in teenagers (Kapil and Kaur, 2010). In Delhi, children from upper socioeconomic strata (6-18 years old) had a prevalence of 6.5%. In a similar vein, 2.6% of teenagers in Jammu and Kashmir were identified to have the condition. Another study that included Indian teenagers living in urban settings indicated that the prevalence was 4.3% and 3.0%, respectively, according to the NCEP ATP III and IDF criteria. In another study, 4.3% of Asian Indian youths had MS (Vikram *et al.*, 2005). There are no national figures available for the prevalence of MS in teenagers in India; estimates come from small-scale or regional research. The incidence and burden of MS in teenagers aged 10 to 19 throughout the country, as well as any related sociodemographic differences, are presented in this research.

Point prevalence rates varied from 0.6% (six to twelve months before becoming pregnant in a sample of expectant women) to 34.1% (in a type two diabetes sample) (Easter *et al.*, 2013), using a DSM-IV standard, and from 0.6% (in a sample of female college and high school students) (Machado *et al.*, 2012), to 1.7% (in a sample of relatives of first-degree of BED patients) utilizing the suggested DSM-V criteria. These approximations are not restricted to estimations based on populations. In line with DSM-IV requirements, the 12-month incidence rate of binge eating disorder, not just based on population estimates, ranged from 0.1 per cent (in an analysis of adults over 18 in a broad population) (Preti *et al.*, 2009), to 1.1 per cent (in a sampling of those individuals who were classified as Latino). Based on DSM-IV requirements, the average lifetime incidence of BED varied between 0.17% among a healthy adult female identical twin sample, as well as 8.8% in an out-patient bipolar disorder specimen (McElroy *et al.*, 2011). Additionally, using the revised DSM-5 requirements, the lifespan incidence of BED varied from 0.22% in a grown-up female identical twin sample to 3.6% in a first-degree family member sample of BED victims. These results go beyond population-based estimates.

Overview of Metabolic Syndrome

Metabolic Syndrome is "a combination of the most hazardous heart attack risk aspects: prediabetes and diabetes, abdominal weight gain, elevated cholesterol levels, and raised blood pressure," by the International Diabetes Federation." It is estimated that

20-25% of adults worldwide suffer from MetS. Although the exact cause of Metabolic Syndrome is still unknown, research has shown that insulin resistance and central obesity play major roles. Insulin resistance is most likely the cause of several aspects of MetS, while this is still up for discussion. Increased fasting glucose and decreased glucose tolerance are signs of insulin resistance, which is frequently brought on by aberrant gluconeogenic enzyme expression. Further insulin release is induced by this metabolic state, which ultimately leads to hyperinsulinemia. Then, hyperinsulinemia mimics the liver's Srebp-1c transcription factors, which cause hypertriglyceridemia and hepatic steatosis (Haas and Biddinger, 2009). Furthermore, excessive synthesis and release of insulin by pancreatic β -cells may lead to their fatigue and demise, which will cause Type 2 Diabetes to start. Obesity plays a major role in Metabolic Syndrome (MetS), as evidenced by the association between the most common type of insulin resistance and malfunction of adipose tissue in the abdomen.

According to Goodman *et al.*, 2007, the frequency of diagnoses of metabolic syndrome reported at baseline that are confirmed during follow-up testing indicates that adolescents with metabolic syndrome have relatively poor long-term stability. Despite whatever metabolic syndrome criteria are used, Goodman discovered that only half of the cases of the illness in teens stay stable during a three-year follow-up period (Goodman *et al.*, 2007).

Obesity is thought to be the primary cause of the metabolic syndrome, which is characterized by several physiological abnormalities, including increased blood pressure, high levels of triglycerides, modest HDLC, raised glucose, and obesity in the abdomen, which raises the risk of type 2 diabetes and cardiovascular illnesses. All weight groups of children and adolescents experience LOC (loss of control) eating, which is a defining characteristic of binge eating (Schlüter *et al.*, 2015). The stated LOC eating rate falls between 23% to 31% (He *et al.*, 2016). Negative health effects, such as excessive weight gain (kg/m², BMI).

Even though obesity is the primary cause of metabolic syndrome, binge eating may contribute to metabolic dysfunction in a way that goes beyond being overweight, according to research on adult samples. Thus, eating behaviors that are out of control might be a modifiable risk factor for those with a metabolic condition. Since some children and adolescents may not continue to participate in LOC eating (Tanofsky-Kraff *et al.*, 2012), moderating factors could provide light on the connection between LOC eating and unfavourable circumstances involving weight.

Metabolic syndrome, or MetS, and obesity are two growing global public health concerns (Arcelus *et al.*, 2011). In industrialised countries, these disorders have significantly increased in frequency during the last thirty years (Van Vliet-Ostapchouk *et al.*, 2014). In 1975, there were 34 million men and 71 million women who

were obese; by 2014, there were 266 million men and 375 million women (“Trends in Adult Body-mass Index in 200 Countries From 1975 to 2014: A Pooled Analysis of 1698 Population-based Measurement Studies With 19.2 Million Participants,” 2016). Obese people are more likely to have Metabolic Syndrome than people of normal weight; about 60% of obese male and 50% of obese females have MetS (Park *et al.*, 2003).

Furthermore, it should be noted that BED is characterised by recurrent instances of binge eating, which are associated with a LOC and significant distress when compensatory weight-reducing behavior is not consistent (DSM-IV) (DeZwaan, 2001). In the average adult population, BED prevalence ranges from 2% to 5%. However, this percentage increases to 30% in a group of adult obese people seeking obesity therapy (Dingemans *et al.*, 2002). Furthermore, it is well recognized that the disease often shows up in people with a history of early-onset obesity, a more frequent weight-shifting pattern, and a more serious type of obesity (Borges *et al.*, 2002). BED may also affect children and teenagers, and its symptoms can vary widely (from preclinical behaviors to the entire illness satisfying all DSM-IV criteria) (Lamerz *et al.*, 2005). For the apparent reason that, among other potential impacts, kids and teens who report BE acquire more weight over time than adolescents and kids without such a history, it is imperative to detect BE episodes to give appropriate treatment (Field *et al.*, 2003).

Dietary patterns

The amounts, ratios, variety, or combinations of various foods and drinks in diets, as well as the regularity of their consumption, are referred to as dietary patterns. Dietary recommendations may be made more quickly and with more knowledge when the intake pattern is assessed, which is why the pattern has attracted a lot of interest. This is because foods and nutrients can interact when consumed together.

Eating Disorder (ED) patients' diets, energy consumption, binge episodes, and macronutrient intake were examined. Patients with Bulimia Nervosa (BN) consumed between 7101 and 9360 kcal per day and between 3030 and 4479 kcal during each binge episode, according to objective lab data (Mitchell *et al.*, 2011). Binge eaters with BN consume more fat than non-binge eaters, but they consume equivalent amounts of carbohydrates and protein. BNers consume fewer calories during binge episodes. BN and controls consume the same amount of daily energy as carbs, but BN consume far more fat and much less protein (Engel *et al.*, 2009). Energy usage showed that BED sufferers utilized higher energy on binge and non-binge days (Raymond *et al.*, 2011). No difference was found in the average intake of controls and non-binge days (McGowan *et al.*, 2012). The “Eating Disorders” Examination Questionnaire (EDE-Q) found that BED patients binge eat 10.7 to 17 times every 28 days (Bartholome *et al.*, 2012). BED patients consume more fat and less protein on binge days

compared to non-binge days, even while obese controls eat less protein and more fat than women with BED (Hudson *et al.*, 2012). These findings raise the question of whether obese and BED women eat different macronutrients.

Binge-Eating Disorder (BED)

People of various genders, ages, colors, ethnicities, weights, body types, sexual orientations, and socioeconomic backgrounds can be impacted by eating disorders. Females are more likely than men to suffer anxiety, tension, and sadness (2-3 times more common) (Bourdier *et al.*, 2017), which may be related to their higher body dissatisfaction and increased propensity to feel these emotions (Peebles and Sieke, 2019). More importantly, eating disorders have been connected to many health issues, including type 2 diabetes, cardiovascular disease, diabetes, gallbladder disease, hypertension, being overweight, and even death (Streatfeild *et al.*, 2021). According to estimates, BED costs USD 11,808 per person (Eddy *et al.*, 2016). Thankfully, 60% of individuals can recover with therapy (Wonderlich *et al.*, 2009).

Although the “Diagnostic and Statistics Manual of Mental Disorders”, 4th Edition (DSM-IV), appendix provided tentative criteria, BED research, which is recognized as a unique mental condition in the DSM-5th Edition, has continued. Since then, a large amount of data has accumulated to support the BED diagnosis's validity and clinical usefulness (Tanofsky-Kraff, M. 2009). Human health is negatively impacted by binge eating disorder, which affects 9% of the general population in Western nations like the US. One of the medical conditions and mental health issues associated with its increasing prevalence is obesity (Field *et al.*, 2003b). Excessive food consumption more than the body needs, caused by disordered eating behaviors, is one of the features of binge eating disorder. Overindulging in food might be a clear indication of discomfort. Stated differently, binge eating disorder is a state in which an individual has a desire to consume due to certain stimuli. Self-induced vomiting is more common in bulimia nervosa patients, while it may also happen in BED cases (Hudson *et al.*, 2007). Compared to other eating disorders, BED is more prevalent worldwide, with an estimated lifetime incidence of 1.9% (Kessler *et al.*, 2013). It is linked to decreased quality of life, psychological distress, mental comorbidity, and functional disability. Growing data suggest that BED and binge eating behavior in general are linked to obesity in addition to significant mental and psychological morbidity (Bulik *et al.*, 2002). Furthermore, an increasing amount of prospective research indicates a link between the onset of obesity and Binge Eating Disorder (BED). Lastly, BED seems to be linked to higher levels of health discontent, higher usage and expenses of health services and higher mortality (Ágh *et al.*, 2015).

MetS is a significant cardiovascular disease risk factor (McNeill *et al.*, 2005); however, it can also affect kids and teenagers. Moreover, it is believed that psychological disturbance raises

the chance of Metabolic syndrome. Compared to patients with modest psychological anguish, those with severe psychological problems at baseline had a more than twofold increased risk of developing MetS. Anxiety and major depressive disorders are linked to an increased risk of MetS, according to pooled data from cross-sectional studies (Odd Ratio = 1.82; 95 per cent CI = 1.06-3.14 and Odd Ratio = 1.07, 95% CI 1.01-1.12) (Tang *et al.*, 2016). Several factors, including poor diet, sedentary lifestyle, and sleep length, have been associated with metabolic syndrome, or MetS, and obesity (Hruby and Hu, 2014). Binge Eating (BE) is clinically linked to obesity and MetS (Bulik *et al.*, 2002; Hudson *et al.*, 2007). BE is characterized by eating large amounts of food quickly and feeling unable to stop. In binge eating disorder, recurrent episodes of eating disorders happen no less often than once every three weeks. A lack of compensatory behaviors, such as increased exercise or vomiting after binge eating, feelings of sadness, embarrassment, and disgust with oneself after BE, and significant pain from binge episodes are further characteristics of BED (Grilo *et al.*, 2012). Obesity severity, as well as general psychopathology including impulsivity and rage (Amianto *et al.*, 2015), have been linked to BED. Typically, pharmaceutical and psychosocial therapies are used to treat BED (Williams *et al.*, 2019). Obese binge eaters participate in unhealthy eating patterns and behaviors that may put them at higher risk for MetS as compared to non-BE obesity sufferers (Ndubuisi *et al.*, 2022). For instance, in both men and women with extreme obesity, hypercaloric food intake has been associated with increased lipid levels and a raised risk of non-alcoholic fatty liver disease (Alciati *et al.*, 2011). According to a long-term investigation, binge eaters had a higher chance than non-binge eaters of developing newly identified MetS components. Despite evidence that obesity and BE co-occur, little is understood about the psychological characteristics of those who have both disorders. In particular, it is yet unknown how psychological characteristics relate to the different developmental paths of diets, BE, and obesity problems.

Special issue in pharmacotherapy for binge eating disorders

The preliminary diagnostic criteria for BED in the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th Edition) facilitated the rapid rise of academic studies on the illness (Grilo *et al.*, 2012). The body of research indicating BED is a pharmacologically responsive illness is increasing, which validates the disorder's diagnostic status (Ndubuisi *et al.*, 2022; Alciati *et al.*, 2011; Hudson, Lalonde, *et al.*, 2010; McCuen-Wurst *et al.*, 2017; Solmi *et al.*, 2021; Tanofsky-Kraff *et al.*, 2012), as represented in Table 1. Despite the argument that BED should only be used as a marker for more general psychological illnesses rather than an independent diagnosis, there is mounting evidence that binge eating disorder is a distinct phenomenon in and of itself (Stunkard and Allison, 2003). For instance, melanocortin 4 receptor mutations and BED were found to be related in a group of people with extreme obesity, according to Branson *et al.* 2004 (Hebebrand *et al.*, 2004). Moreover, pharmacological studies in BED patients have demonstrated a rising degree of methodological sophistication over time. More recent medication studies have included participants with a BED diagnosis based on the criteria for DSM-IV and comparable main outcome measures (number of days or bouts of excess feeding), conventional techniques for assessing anthropometric parameters, and psychopathology.

Clinical research on anxiety disorders has also shown a generally robust and diversified trend in the placebo-response (Versiani, 2000). As a result, it's critical to comprehend the features of the BED medication placebo response. A subgroup of BED patients who showed improvement during the placebo run-in stage of a medication study was examined by Jacobs *et al.*, 2005.

A mean response rate of 27% to placebo was observed. The quality of life, social functioning, and severity of binge eating were all less impaired in placebo responders at baseline than in placebo non-responders, but at the 1-year follow-up, their symptoms had returned. These early results point to the disorder's unexpected

Table 1: Binge Eating Disorder (BED) and Outcomes.

Age	Findings	Population	Outcome	References
Adult population. Mean age was 43.8%	All were overweight.	135 Obese males and females	31% of those who overindulged in food had metabolic disorders.	(McCuen-Wurst <i>et al.</i> , 2017)
Adult population	All had hypertension. 60% had Hyperglycemia.	268 individuals	The Metabolic Syndrome risk factors may be triggered by binge eating disorder.	(Ndubuisi <i>et al.</i> , 2022)
Adult population	Obese.	Not confirmed	The metabolic syndrome and BED are strongly correlated.	(Solmi <i>et al.</i> , 2021)
34 - 75 years old	All were overweight.	15,500 binge-eating population	Obesity is more likely to develop in those with BED.	(Tanofsky-Kraff <i>et al.</i> , 2012)
5 - 12-year-old children	Overweight.	180 children with Binge eating behaviour	Because binge eating increases triglycerides, it may cause metabolic disorders to develop.	(Tanofsky-Kraff <i>et al.</i> , 2009)

Table 2: Pharmacological interventions for Binge eating disorder in randomized controlled trials.

Diagnosis	Treatment duration (weeks)	N ^a	Medication dose/day	% Reduction in binge eating frequency ^d	% Remission at the end of treatment ^e	Depression outcome	Weight outcome Weight change (kg)	References
DSM-IV BED Overweight	6	16/19 15/19	CIT Plc (40-60 mg, average: 58 mg)	67.3 ^{b,c} 40.3 ^c	56 ^c 27 ^c	CIT=Plc	-2.1 ^b +0.2	(McElroy <i>et al.</i> , 2011)
DSM-IV BED Obesity	12	23/30 25/30	SIB (15 mg) Plc	66 ^b 41	52 ^c 32 ^c	SIB>Plc ^b	-7.4 ^b +1.4	(Apolinário-Hagen <i>et al.</i> , 2020)
DSM-IV BED Overweight	6	23/30 23/30	FLX (20-80 mg, mean: 71 mg) Plc	70 ^{b,c} 55 ^c	57 ^c 25 ^c	FLX>Plc ^b	-3.9 ^b +0.7	(Arnold <i>et al.</i> , 2002)
DSM-IV BED Obesity	8	12/14 12/14	d-FEN (30 mg) Plc	72.7 ^b 0	80 ^b 33 ^c	d-FEN=Plc	--- ---	(Stunkard and Allison, 2003)
DSM-IV BED Overweight	9	29/42 38/43	FVX (50-300 mg, mean: 260 mg) Plc	--- ---	45 ^{b,c} 24 ^c	FVX=Plc	-1.3 ^b -0.4	(Hudson <i>et al.</i> , 2007)

course as a feature of BED, which might assist in explaining the phenomenon of placebos seen in BED medication studies.

Currently, pharmacological therapies are regarded as the 1st choice BED therapy (DeZwaan, 2001). While combination therapy seems acceptable for obese or overweight individuals with BED, little research has been done on this approach. A third potential topic for BED treatment study is the combination of pharmacotherapy and psychotherapy, given the effectiveness of several pharmacological drugs in the various symptom categories of BED (Devlin, 2001). Pharmacological interventions (Apolinário-Hagen *et al.*, 2020; Arnold *et al.*, 2002; Stunkard and Allison, 2003) for BED in randomized controlled trials is represented in Table 2.

Finally, it's critical to note that the exact mechanism by which pharmaceutical treatments for BED work is uncertain. While classifying the medications is a useful strategy in this review, it is easy to assume that antidepressants, anticonvulsants and anti-obesity agents work for BED because of their respective actions against obesity, anticonvulsants, and mood stabilizing agents. With just two relatively unique chemicals having been demonstrated to be effective, this may or may not be the case with anticonvulsants. It is likely the case for anti-obesity drugs, and it may be the case for antidepressants. With just two relatively unique chemicals having been demonstrated to be effective, this may or may not be the case with anticonvulsants.

In the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), except for the Stunkard *et al.*, 1996 trial, which found that binges were lessened in patients with acceptable plasma levels of d-fenfluramine, the word "remission"

is used in all studies to refer to the cessation of binges. Additionally, Plc (Placebo), SER (Sertraline), FLX (Fluoxetine), d-FEN (d-Fenfluramine), FVX (Fluvoxamine), TOP (Topiramate), SIB (Sibutramine), CIT (Citalopram), and other medications are categorized in this way in research. A person is considered obese if their Body Mass Index (BMI) is more than 30 kg/m² and overweight if it is more than 85% of the ideal body weight.

Differences and Connection between Dietary Patterns, Binge Eating and Metabolic Syndrome (MetS)

The differences among dietary patterns, binge eating, and metabolic syndrome show that Western, Mediterranean, and DASH diets are representations of dietary patterns. The Western diet is linked to high-calorie consumption, processed carbohydrates, and saturated fats. It could make the metabolic syndrome worse. Fruits, vegetables, fish, and olive oil are abundant in the Mediterranean diet. It's connected to a lower chance of diabetes and heart disease as well as weight reduction. Whole grains, lean meats, and low-fat dairy products are highlighted in the DASH Diet. Additionally, it is advantageous for metabolic health (Angelico *et al.*, 2023). Binge eating, however, also affects behavior and metabolism. This behavior entails devouring a lot of food quickly, often coupled with losing control. Binge eating habits may raise the risk of metabolic disorders, according to Metabolic Impact (Roehrig *et al.*, 2009). It covers dietary impact, which includes healthy diets (that emphasize whole, plant-based foods) and negative effects (such as excess caloric consumption and Western dietary patterns) as well as positive effects (such as Mediterranean and DASH diets) (Esposito *et al.*, 2007). The

connection between metabolic syndrome, binge eating, and dietary patterns shows that, adopting a nutritious diet that consists mostly of fruits, vegetables, nuts, low-fat dairy products, and legumes is linked to a 46% lower chance of developing metabolic syndrome. Blood pressure, waist circumference, and blood glucose levels are only a few of the specific metabolic syndrome components that dietary pattern helps to regulate (Kheirandish *et al.*, 2024). An elevated risk of metabolic syndrome is linked to a Western diet pattern that emphasizes high consumption of red meat, processed meat, fried meals, and refined carbs (Lottenberg *et al.*, 2012). Nevertheless, no meaningful association was discovered in fully adjusted models between the elements of the metabolic syndrome and the Western eating pattern. But, in Binge Eating and Metabolic Syndrome, those who binge eat and are obese may be more susceptible to developing metabolic syndrome. Excessive calorie intake, or hypercaloric food intake, has been connected to raised lipid levels in individuals with severe obesity and non-alcoholic fatty liver disease (Conti *et al.*, 2020). Consequently, implementing a nutritious eating plan may aid in the prevention and management of metabolic syndrome.

DISCUSSION

Research over the past decade has revealed a notable connection between binge eating behaviors and the emergence of metabolic syndrome in children and adolescents. While excessive weight gain has long been recognized as a result of binge eating, accumulating evidence indicates that metabolic health can also be compromised even when weight changes are only being the reason. For instance, children who report frequent episodes of binge eating are at an increased risk of developing adverse metabolic profiles, including higher triglycerides and greater visceral fat, compared to peers without these eating patterns.

Notably, this association is influenced by more than just body mass. Studies have shown that the metabolic complications seen in youth with binge eating can occur even after adjusting for changes in BMI, highlighting binge eating itself as a behavioral risk marker. The role of dietary patterns is equally significant. High intake of simple sugars and processed foods, commonly reported among youth with binge eating tendencies, is linked to worsened lipid profiles and overall metabolic risk.

Demographic variables such as gender, ethnicity, and socioeconomic status further influence both the rates and effects of binge eating and metabolic syndrome. Female children are more frequently diagnosed, but underdiagnosis still exists among male and minority groups. Importantly, psychological factors such as depression and anxiety can interact with binge eating and metabolic dysfunction, suggesting the need for comprehensive assessment and prevention strategies.

CONCLUSION

Metabolic syndrome issues have emerged as major health issues in the past few years. Cardiovascular disease and central obesity, as compared to peripheral obesity, which is characterized by elevated cortisol production, are arguably the biggest risk factors for metabolic syndrome. In particular, it is yet unknown how psychological characteristics relate to the different developmental paths of diets, BE, and obesity problems. It concluded that childhood binge eating may be associated with metabolic dysfunction; However, the connection between childhood body weight and binge eating mostly explains these connections. The detection of metabolic syndrome in children and adolescents who were underweight, normal weight, overweight, or obese demonstrated the triple burden of malnutrition in these countries. However, further research must be done to determine all potential causes. According to a number of studies on binge eating disorders, those who have the illness are more likely to acquire metabolic syndrome.

Given the serious long-term risks associated with both binge eating and metabolic syndrome-including elevated odds for adult cardiovascular disease and diabetes-it is crucial to implement early recognition and intervention. Pediatric evaluations should routinely include screening for disordered eating behaviors, especially in children with obesity or a family history of metabolic problems. Early dietary guidance and psychological support can mitigate the risk for metabolic complications, offering hope for improved outcomes in this vulnerable population.

ACKNOWLEDGEMENT

The authors would like to express sincere gratitude to Department of Pharmacy, Faculty of Pharmaceutical Sciences at Maharishi Markandeshwar (Deemed to be University), Mullana for the support to carry out this review as a part of my Ph.D. work.

ABBREVIATIONS

MetS: Metabolic Syndrome; **BE:** Binge eating; **ED:** Eating Disorder; **BN:** Bulimia Nervosa; **BED:** Binge eating disorder; **WHO:** World Health Organization; **HDL-C:** High-Density Lipoprotein Cholesterol; **PCOD:** Polycystic Ovarian Syndrome; **EDE-Q:** Eating Disorders Examination Questionnaire; **DSM:** Diagnostic and Statistical Manual of Mental Disorders; **LOC:** Loss of Control; **DASH:** Dietary Approaches to Stop Hypertension.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Cite this article: Monga P, Das R, Agrawal BK, Mehta DK. Metabolic Syndrome and Binge Eating in Children: A Comprehensive Review. *J Young Pharm.* 2025;17(4):824-32.