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Review Article

Cardiometabolic Risks in Adolescent Obesity

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Abstract

Currently, childhood obesity is a major public health issue in the Western world. There has been evidence citing that although obesity is a major problem during adolescence, it could also be the beginning of more serious conditions during adulthood. Because of this, numerous studies have been conducted to understand childhood obesity, most of which rely on cardiometabolic risk factors to predict and determine the prevalence of childhood obesity. This study aims to correlate childhood obesity with cardiometabolic factors to generate insights on forthcoming studies on this subject matter.

Methods: This study relies on research into the verified risks linked to childhood obesity. Reliance of secondary sources of data, which include journals and textbooks that are relevant to this study were also included. Database (PubMed, Medline, Google Scholar) searches including key terms such as "childhood obesity", "cardiometabolic risks", "childhood obesity risks" and "metabolic syndromes" were used. Strategically, articles were included as early as the 1970s to track the changes in diet and lifestyle, which could be implicated as risk factors. This study consists of different populations of children from various parts of the U.S., which in consequently explores the implications of socioeconomic differences.

Results: It has been shown that childhood obesity is associated with numerous cardiometabolic risks that can affect both children and adults. Additionally, evidence shows that childhood obesity has a high morbidity and mortality. It also has been shown that childhood obesity has been found to lead to exacerbation cardiovascular risk factors during adulthood. The evidence generated on this subject is not on par with meta-analyses and randomized controlled trials as the majority of these studies were non-experimental.

Conclusion: The study into the association between childhood obesity and cardiometabolic risk factors as well as adult cardiometabolic risk factors underlines the requisite for more research on this subject matter. Suggested studies should include the restructuring of the general management of childhood obesity as well as associated risks for current and future generations. Although there is considerable information available on childhood obesity and related cardiometabolic risk factors, the validity as well as reliability that more assertive studies such as Random Controlled Trials (RCTs) and meta-analyses have been yet to be included.

Keywords: Childhood obesity; Public health; Cardiometabolic factors; Cardiometabolic risks

Introduction

Childhood obesity has been shown to include various comorbidities. Common examples include prediabetes and type II diabetes. Additionally, high blood pressure, hypercholesterolemia, hyperlipidemia, obstructive sleep apnea, asthma, non-alcoholic fatty disease, orthopedic disease, abnormalities of the female reproductive system an some forms of cancer [1] have been shown to be linked. It would be remiss to not mention the psychological and social burdens (stigmatization, bullying,

and discrimination) that are also related [2,3]. It has been shown that children who are severely obese risk developing cardiovascular disease relative to the case of those who are less obese and non-obese children. These risks mainly comprise of hypertension, high fasting glucose, high total cholesterol, low LDL cholesterol and high triglyceride levels. There is a concomitant rise in the comorbidities of childhood obesity with the increasing severity of obesity [4]. Unfortunately, childhood complications and these poor health outcomes usually persist into adulthood, as seen in clinical cases, which most commonly include cardiovascular disease, metabolic disease and obesity.

Multiple studies seek to establish cardiovascular and metabolic comorbidity rates in obese children; however few studies seem to encompass these trends for long periods of time. This study seek sot establish the risks associated with childhood obesity dating back as far as 1970 with a focus on lifestyle changes. For instance, it is likely that diet has changed in the last five decades and has affected rates of obesity and associated cardiometabolic risk factors. This study will consider studies conducted on different populations of children from different parts of the United States, as well as explore the socioeconomic background and how that plays a role in childhood obesity and understanding how this can be used as a determinant for making predictions about childhood obesity. Nevertheless, this study mainly focuses on determining how this problem of obesity can be a marker for future cardiometabolic problems and provide insight into the strategies for prevention of both cardiovascular and metabolic conditions that are attributable to childhood obesity.

The term cardiometabolic risk describes the risks of developing cardiovascular disease attributable to a multitude of risk factors. Although there is an established mechanism to prevent cardiovascular disease, there are still patients suffering from CVD. This can be explained because most of the patients are unable to stick to such lifestyle interventions that are prescribed to avoid CVDs [5]. Statistical evidence cites at lease 2/3 of the U.S. population is obese. Of which there is an 18% prevalence of obesity in childhood and adolescence [6]. This shows that although there have been thorough deviations to lessen the burden

of CVD; statistics show that non-adherence is a major contributor.

A well-documented comorbidity of obesity is type II diabetes. At the current time, there is significant concern with childhood diabetes as the age of onset has been decreasing. It has been shown children with age as low as seven years have been developing type II diabetes. A major interest with this is the development of micro vascular and macrovascular complications in these children when they develop into young adults [7]. The explanation for childhood diabetes and its epidemic still remains unknown.

The cardiometabolic risk factor that childhood obesity proposes includes elevated systolic blood pressure, diastolic blood pressure, triglycerides, fasting glucose and low HDL [8]. Correspondingly, a decrease in physical activity is also linked to a high risk of obesity. With less physical activity being correlated to a high risk of obesity, adult health can be shown to be significantly compromised as the level of physical activity during childhood being low to the extent of reaching global pandemic levels. Cardiometabolic risk factors that develop during childhood become essential risk factors for diseases such as coronary heart disease, diabetes and stroke when one gets to adulthood [8].

In the past, BMI has been used as the accepted screening tool for obesity in adulthood, or simply obesity in children mainly because of simplicity and the strong correlation with CMR [9]. However, in order to better stratify risk, there are three obesity subcategories that have been suggested by the American Heart association. Utilizing this new stratification, class III obese children had two times more risk of hyperglycemia and hypertension in adulthood (Skinner, Perrin, and Moss. This preludes the idea that there are other factors at play that lead to development of risk factors in children than simply being obest.

Hypotheses

- There is a strong correlation between cardiometabolic risks and childhood obesity
- Childhood obesity is linked to adult cardiovascular risk factors

Methods

The study involves research into the necessary risks that are associated to childhood obesity. Reliance on secondary sources of data which includes journals and textbooks relevant to the study has been used. Searching for articles through various databases, including search term such as “childhood obesity”, “cardiometabolic risks”, “childhood obesity risks” and “metabolic syndromes” etc. done. The study involved using Google scholar as the primary database, where the search -term “Cardiometabolic risk factors in children” generated 69,400 results. The articles were selected based on the suitability of their titles. Studies included are those that have a direct correlation with the current study. The majority of the studies were mainly descriptive, non-experimental, and explanatory studies. Nevertheless, the study included illustrative mixed method studies, meta-analyses and systematic reviews in the study. (See details in the evidence tables in the Appendix section).

The initial study explores the factors that relate childhood obesity and then moves into cardiometabolic risk factors, genetic factors as well as factors that predispose children to obesity. The study addresses cardiometabolic factors that are linked to the problem and those which predispose children to childhood obesity. It then seeks to establish current preventive and curative measures for childhood obesity as well as the management of various cardiometabolic factors.

Results and Discussion

The findings below were made from the evidence collected on the studied topic. Correlation was formed that is supported by the evidence on the subject. This approach allows for the synthesis of evidence on the subject and to draw a valid conclusion, as well as ultimately making appropriate recommendations.

Childhood obesity is a significant health problem that is on the cusp of becoming an epidemic. It is linked with the development of adult obesity and being overweight as well as cardiometabolic risk factors of obesity into adulthood [10]. When exposed to obesity-associated cardiometabolic risk factors, the development of atherosclerotic conditions such as changed vascular structure and function is likely to occur [10]. Early diagnosis and treatment will allow a better outcome yield. The cardiometabolic risk factors can be compounded by tobacco smoking, male gender, physical inactivity and diabetes [11]. Nonetheless, obesity is a standalone independent risk factor for worsened cardiometabolic factors.

The genetic study on susceptibility to metabolic syndromes oftentimes seems conflicting with a rare chance of reproducibility of results. Notwithstanding, researchers believe it is imperative to establish the genetic foundation of metabolic syndrome so it can help in early detection and management. The reasoning behind variability of results from genetic studies includes a lack of standard definition for “metabolic syndrome”, multiple combinations of the phenotypes even with a single description, ethnicity as well as gender differences [12]. Lifestyle unfortunately has not been taken seriously in these studies either, as evidence points to metabolic syndromes being linked as a fine interplay between genetic predisposition and environmental influences.

Obesity in children is recognized as a major contributor to cardiometabolic risks. Adiposity tests can be used to classify children who are likely to have cardiometabolic opportunities. Evidence suggests that anthropometric measurements when assessing adiposity are just as rational as tests derived from DXA. Nonetheless, due to higher precision levels in predicting insulin resistance, studies suggest the use of both DXA-derived adiposity indicators and indirect rules [13]. The DXA approach works on the principle of different tissues receiving low-energy x-rays differently. It is appropriate for estimating fat free and fat mass and composition of the regional body. Some reservations about this approach include high costs, which are not easily accessible, and that the scanning areas may be restricted only to people who can fit within the scanning area [14]. Consequently, it could be suggested from the evidence presented here that a combination of DXA-derived methods and anthropometric tests could be used as a reliable indicator of insulin resistance, a significant risk factor for cardio metabolism. It is appropriate for estimating fat-free and fat mass and composition of the regional body. Some reservations about this approach include high costs, which are not easily

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Birth weight studies have shed some light on prenatal causes and children's development of obesity. For high birth weight and low birth weight, studies have often used specific cut-off points. Nonetheless, weight and obesity are continuum variables, suggesting a review of approaches used to study obesity and its associated risk factors for cardio metabolism [15]. Throughout childhood and adulthood, high birth weight from maternal gestational diabetes is associated with obesity. For example, research carried out between 1936 and 1983 on Danish youth showed that the proportion of people who became overweight between 6 and 13 years of age increased [15].

The children of parents with Type II diabetes had significantly higher birth weight, higher weight at all ages, high levels of insulin, glucose, triglycerides, VLDL cholesterol, and LDL cholesterol at adulthood in the Bogalusa Heart Study. Such proof was derived from research carried out since the 1970s. Long-term research on the subject could therefore provide data that could assist in treating overweight and obesity to boost cardiometabolic risk factors.

Several candidate genes are thought to play a role in animal models ' energy control or play a role in extreme or monogenic types of obesity [12]. We were tested for their association with obesity-related characteristics. Nonetheless, only a few have been effectively associated with obesity or some of its risk factors. Small sample sizes that result in low statistical power, low number of variants and low biological insights to inform the gene candidacy foundation are a significant problem with most studies.

Obese children are likely to develop adult obesity, which is associated with an increased risk of morbidity, evidence suggests. The development of adult diabetes, coronary heart disease, and some cancers excluding breast cancer was correlated with a high childhood BMI [16]. In predicting adult morbidity of obesity, childhood BMI is not so accurate. Evidence suggests that high childhood (Above 12) BMI can be attributed to only about 3 percent of future diabetes and 22 percent of future hypertension. Research also shows that in children who were obese and overweight, only about 20% of adult cancers were detected [16].

Evidence suggests that while BMI in childhood was used to explain the frequency of certain morbi). Ditties in adulthood, it is not adequate to be used as an adult morbidity predictor [16]. Most of the morbidity of adult obesity arises in healthy people as children. Consequently, addressing childhood obesity as the primary path to adult obesity burden reduction may not provide a sustainable solution.

For some reason, it is difficult to observe the epidemiological patterns of childhood obesity while obesity is widely studied in children; identifying obesity in a population that has to adjust body size has become increasingly difficult [17]. This problem was solved by using Z-statistics, however. In addition, the urban population is highly mobile, making it increasingly difficult to carry out the required follow-up, which could take statistically significant results from childhood obesity studies for up to 40 years. Nevertheless, a small group of dedicated investigators held long-term records of CVD. The Bogalusa Heart Research, for example, was conducted in the 1970s and is one of the researchers ' main initiatives [17]. The study has contributed to the publication of medical reports by more than 1,000 papers Current scientists have a better perspective on the mechanisms resulting in medically evident CVD because of such studies.

Although BMI is the most common anthropometric tool used for weight examination and obesity diagnosis, it is associated with certain limitations. It is associated, for example, with outcomes and mortality associations in the form of U or J [18]. This leads to the "obesity paradox" in which people with patients with elevated BMI and chronic diseases tend to have greater chances of survival than patients with non-obesity [18]. BMI cannot be relied on to differentiate between lean body weight and fat body weight which causes high body weight.

Discussion

For some reason, it is difficult to observe the epidemiological patterns of childhood obesity. While obesity is widely studied in children, identifying obesity in a population that has to adjust body size has become increasingly difficult [17]. This problem was solved by using Z-statistics, however. In addition, the urban population is highly mobile, making it increasingly difficult to carry out the required follow-up, which could take statistically significant results from childhood obesity studies for up to 40 years. Nevertheless, a small group of researchers have held long-term reports of CVD.

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Establishing the evolution of end-organ damage that occurs slowly over decades involves the use of approaches that can help to compensate for unavoidable changes in one or more risk factors occurring and severity. For example, assume that child X is eight years old with moderate obesity and slightly elevated blood pressure, both of which remain relatively mild for the next three decades and child Y is eight years old with average weight and

BP while slowly becomes overweight and extremely hypertensive over the years.

Highly capable and innovative statistical tools are needed to analyze the effect of various risk factors on the different trajectories of comorbid conditions in a comprehensive manner. Analytical methods used in cardiometabolic research are therefore important in assessing

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Intrauterine over nutrition is a major risk factor for cardio metabolism and a primary determinant of the development of obesity and type II diabetes. Studies have established intrauterine and postnatal pathways that may be associated with increased adiposity risk and type II diabetes [19]. The factors associated with these pathways can be Trans generational and are a risk factor with significant implications for obesity and type II diabetes development. Fetal life is a significant determinant of later growth, including adult life, according to Barker's hypothesis [19]. In the 1990s, the theory was based on available epidemiological evidence pointing to fetal life, which determined the risk of childhood obesity, which was therefore thought to imply adolescence and adult life. Subsequently, studies identified developmental mechanisms involved in adiposity and type II diabetes development [19]. Intrauterine conditions such as over-nutrition are known as major risk factors for increased adiposity, impacting the adipoinsular axis of the fetus. Excess intrauterine maternal fuel disrupts the regulation of energy and appetite and the metabolism of adipocytes [20]. The adipoinsular shaft has hormones like insulin and leptin, which are essential determinants of adiposity and diabetes-related outcomes. Thus, the vicious transgenerational cycle begins with fetal intrauterine exposure and has severe effects on the onset of obesity and type II diabetes [21, 22]. Obesity as a predictor of morbidity in adulthood.

Obesity has been attributed to cardiovascular disease-related risk factors, including high blood pressure, insulin resistance, and dyslipidemia, which are the three key elements of metabolic syndrome. As a result, new health programs have arisen to treat problems in young people such as fatty liver disease, obesity, diabetes, and hypercholesterolemia [17]. Some cardiometabolic risk factors, including fatty streaks in blood vessels and arteriosclerosis precursors, are observed in children. These patterns are associated with high and low density lipoprotein levels [17]. Among younger adults, early exposure to medical complications such as hypertension and dyslipidemia is associated with the risk of cardiovascular disease.

Obese children are likely to experience adult obesity, which is associated with an increased risk of morbidity, evidence suggests. The prevalence of adult diabetes, coronary heart disease, and some cancers besides breast cancer was associated with a high childhood BMI [16]. When predicting obesity morbidity when adults, childhood BMI is not so accurate. Evidence suggests that high childhood (Above 12) BMI can be attributed to only about 3 percent of future diabetes and 22 percent of future hypertension. Research also shows that in children who were obese and overweight, only about 20% of adult cancers were detected [16]. Evidence suggests that while BMI in childhood was used to explain the frequency of certain morbidities in adulthood, it is not adequate to be used as an adult morbidity predictor [16]. Most of the morbidity of adult obesity occurs in healthy people as children. Consequently, addressing childhood obesity as the primary path to adult obesity burden reduction may not provide a sustainable solution.

Obesity and the risk factor for cardio metabolism might run through families. There is sufficient evidence to suggest that children may develop cardiovascular disease and obesity predispositions [23]. Different studies have identified candidate genes related to obesity susceptibility. These include studies of animal studies, linkage studies, Mendelian syndromes, and expression studies, among other genetics association studies. These include studies of animal studies, linkage studies, Mendelian syndromes, and expression studies, among other genetics association studies. These experiments were aimed at finding genes that predispose individuals to obesity. Many of the candidate genes are thought to play a role in animal models ' energy control or play a role in extreme or monogenic types of obesity [12]. They were tested for their association with obesity-related characteristics. Nonetheless, only a few are effectively associated with obesity or some of its risk factors. Small sample sizes that result in low statistical power, low number of variants and limited biological insights to inform the gene candidacy base are a significant problem with most studies.

Genetic studies on metabolomic syndrome vulnerability often

disagree with the rare chance of findings being reproducible. Nevertheless, researchers believe that identifying the genetic basis of metabolic syndrome helps to identify and treat metabolic syndromes early on. Some reasons for variability in genetic study results include lack of a standard definition of metabolic syndromes, multiple combinations of phenotypes even with one description, gender differences, and ethnicity [12]. In addition, other factors, such as lifestyle, have been suggested to be taken seriously in these studies. Evidence indicated that the interplay between genetic predispositions and environmental influences is the result of metabolic syndromes.

Adiposity as BMI as a measure

It is known that childhood obesity contributes significantly to cardiometabolic risks. Adiposity tests can be used to classify children who are likely to have cardiometabolic opportunities. Evidence suggests that anthropometric measurements when assessing adiposity are just as rational as measures derived from DXA. Nonetheless, due to higher precision levels in predicting insulin resistance, studies suggest the use of both DXA-derived adiposity tests as well as indirect rules [13]. The DXA approach works on the principle of different tissues receiving low-energy x-rays differently. It is appropriate for estimating the fat-free and fat mass and composition of the regional body. Some reservations about this approach include high costs, which are not easily accessible, and that the scanning areas may be restricted only to people who can fit within the scanning area [14]. Consequently, it could be suggested from the evidence presented here that a combination of DXA-derived methods and anthropometric tests could be used as a reliable indicator of insulin resistance, a significant risk factor for cardio metabolism.

Waist circumference (WC) is an important factor associated with obesity in childhood. It is a crucial factor in the assessment of abdominal adiposity in adolescents, according to Holman, Carson, and Janssen [24]. It is also an important predictor of risk factors for cardio metabolism than BMI [24]. In many studies, Waist circumference was used to predict morbidity and mortality that the BMI could predict. Measuring WC > However, some authors doubt the medical usefulness of WC based on the fact that WC does not provide further data beyond what is obtained through the BMI. While there is insufficient evidence to support WC predicting cardiovascular risk when regulated by other factors such as hypertension, hypercholesterolemia, and apolipoprotein rations, its importance as an important risk factor for cardio metabolism cannot be ignored.

Janiszewski, Janssen, & Ross [25] find that WC helps to predict diabetes outside of cardiometabolic risk factors and BMI. On the other hand, they find that after considering common cardiometabolic risk factors, BMI could not effectively predict diabetes. Researchers report that while higher levels of WC and BMI are highly associated with risk of CVD, the effect of elevated levels is negated after risk factors are controlled. The U.S. and Canadian health guidelines recommend the use of toilet for a more educated evaluation. Researchers find that WC is more effective in predicting hazard (fivefold) when comparisons are made with regulated BMI and lifestyle factors from a variety of WC categories. WC contributes significantly to the prediction of the incidence of diabetes, making it an important risk factor for cardio metabolism [25].

Childhood obesity and adult cardiovascular disease

There are variations between children and adults in the concept of obesity. While the concept of obesity in adults refers to some associated risk points, childhood obesity depends on a reference population [26]. A BMI equal to or above 30 in adults means a person is obese. In children and youth, on the other hand, obesity is diagnosed with a BMI greater than or equal to the age- and sex-specific 95th percentile of the 2000 growth charts of Centers for Disease Control and Prevention [26]. There is a very strong relationship in the future between childhood obesity and CVD growth. Due to childhood obesity, the high risk of CVD can be due to the specific risk of CVD posed by or risk of developing other risk factors for CVD in adult life [9].

There is a strong correlation between childhood obesity and CMRs such as high systolic blood pressure, diastolic blood pressure, triglycerides, fasting lipoprotein (HDL) and fasting glucose [8]. Less physical activity is associated with a high risk of obesity. With insufficient levels of physical activity during adolescence to meet global levels of pandemics, and high prevalence of obesity, adult wellbeing is greatly impaired. The cardiometabolic risks that arise during childhood become major risk factors for diseases like coronary heart disease, diabetes, and stroke as you reach adulthood [8]. Throughout adolescence, low levels of physical activity, combined with obesity, indicate cardiometabolic risk factors. Physical activity is a modifiable wellness practice that could go a long way in reducing cardiometabolic risk factors by following suggested strategies. Physical activity in obese children has been shown to reduce cardiovascular risks [8].

It has been shown that obesity raises the risk of coronary heart disease and stroke 2-4 times during puberty. In studies that tracked cardiometabolic risk factors from childhood to adulthood, the most important cardiovascular risk mediator was childhood obesity. Indeed, even in studies where childhood obesity and cardiometabolic risk factors were found to be small, childhood obesity was still the primary driver of adult CRMs [27].

Adult obesity rates have doubled over the past three decades. Even with lower rates of obesity in children, the rate of growth has exceeded the rate of adults. Such results are troubling as obese children are predisposed as adults to be obese relative to healthy children. There is also evidence that obese children are more likely to develop cardiovascular disease than obese adults, and weight loss is not enough to prevent cardiovascular disease [17]. It can be inferred from the patterns of rising childhood obesity that risks associated with adults are likely to increase over the years.

High blood pressure

Previously, hypertension in childhood was considered to be rare. It was considered due to underlying factors such as parenchymal renal disease when it occurred. Primary hypertension in children, however, is now established. Childhood obesity is deeply involved in the high prevalence of childhood hypertension and other obesity-related morbidities. Therefore, hypertension in childhood is a common condition of health among young people.

The prevalence of obesity and overweight in various European countries is projected to be between 20-30 percent combined [28]. It has been found that the prevalence of obesity varies from developed to developing countries. In developed countries, the

incidence of obesity among people with lower socio-economic status is higher, with people with lower prevalence from the highest economic situation. In developing countries, however, it is the children of higher socio-economic status who are prone to being obese and overweight, while children of lower socio-economic status are protected from being overweight and obese. Therefore, during childhood, there is a correlation between obesity and overweight. The recent past has seen a large increase in obesity and overweight. Overweight and obesity rates are increasing at a higher rate in some countries such as South Africa, China, and Thailand. Obesity and overweight are estimated to have a global prevalence of 2.5 between 1990 and 2010 [28].

The question of hypertension in children is calculated by testing whether the diastolic or systolic blood pressure for sex, age, and height is greater than the 95th percentile based on the normative data obtained from evaluating broad BP readings databases taken from healthy children [28]. Approximately 5 percent of children and adolescents have elevated B.P from this definition. Nevertheless, from 1974 to 2012, the prevalence of hypertension in childhood has been between 2 and 3.5%, although more recent studies report higher rates of between 4 and 5%. One of the successful studies is the Houston BP testing campaign. This obtained children's data on repeated BP tests in public schools. After three screenings, the project found that BP was about 3.2 percent among children. However, it is interesting that during the first screening exercises; about 20 percent of the children in the project had a high level of BP [28]. The study also found that the pre-hypertensive stage was 9.5 percent, while the hypertensive age was 9.4 percent. There is therefore a need for repeated testing to establish hypertension in the childhood [28].

Hypercholesterolemia

Hypercholesterolemia is another important risk factor for cardio metabolism. Hypo cholesterolemia, even for young people, is associated with coronary heart disease. Evidence suggests screening for cholesterol by the child-parent. It is easier to identify parents with family hypercholesterolemia when a child is recognized as having family hypercholesterolemia [29]. It is essential to identify hypo cholesterolemia in children as early as possible to initiate early interventions to prevent open cardiovascular disease. At the adolescent level, where there is evidence of benefits, Statin may be given to children [29]. Hypercholesterolemia is another important risk factor for cardio metabolism. Hypo cholesterolemia, even for young people, is associated with coronary heart disease. Evidence suggests screening for cholesterol by the child-parent. It is easier to identify parents with family hypercholesterolemia when a child is recognized as having family hypercholesterolemia [29]. It is essential to identify hypo cholesterolemia in children as early as possible to initiate early interventions to prevent open cardiovascular disease. At the adolescent level, where there is evidence of benefits, Statin may be given to children [29]. Statin may be given to parents as well, and it is important as it helps the child when a parent's death is avoided.

Breakfast eating is associated with a reduced risk of cardiometabolic diseases and obesity. Evidence suggests that breakfast leads to reducing the risk of obesity by metabolism and energy balance processes [29]. It is argued that breakfast affects hormones and other metabolic factors that regulate energy use, appetite, processing, and blood glucose regulation [30]. While breakfast has been correlated with practical effects on cardiometabolic risk factors, studies that monitor breakfast composition have little evidence to suggest. Investigations on the impact of breakfast consistency on cardiometabolic factors need to be carried out in the future.

Hyperlipidemia

Because cardiovascular disease is a major cause of death in the US, the atherosclerotic process is believed to start during childhood. In children aged 10 years and in coronary arteries in people aged 20 years, fatty streaks in the aorta were detected. For people with elevated serum total lipoprotein and low density cholesterol, arteriosclerotic lesions are normal. Obese kids have heavy lipids in the blood. High LDL-c, TG levels and low HDL-care in children with overweight and obesity and the association between these will causes the cardiovascular diseases. Insulin resistance in obese children is associated with high levels of TG and LDL, but not with levels of HDL-c and TC.

Sleep apnea

OSA is known as a risk factor for cardiovascular disease due to its association with obesity. Another cardiometabolic risk associated with obesity in children is sleep apnea. For healthy individuals there is a reduction in the tone of airway musculature with the pharyngeal dilator operation that holds the airway patent. Therefore, even though children may have occasional breathing delays (10-15 seconds), there is no substantial limit to airflow. As a result, paO_2 could fall between 2 and 4 mmHg, rising the end-tidal CO_2 by 3-4mmHg.

Obstructive Sleep Apnea (OSA) is a sleep disorder characterized by the absence of nasal airflow despite chest wall movement and abdominal movement for at least two children's breaths. OSA is closely linked to snoring, recurrent or complete apnea, and upper airway obstruction. Snoring is not, however, associated with OSA, as neuromuscular compensation occurs in children during routine snoring.

Because of mechanical and neuronal causes, airway collapse is associated with airway obstruction during OSA. Adenoid / tonsil hypertrophy that narrows the lumen of the airway. An estimated 2 percent of children are associated with mechanically obstructing relatively large tonsils and adenoids. Nevertheless, OSA is a combination of both mechanical obstruction and decreased muscle activity of the pharyngeal dilator. For children with OSA, the muscle tone is diminished, leading to shortened and obstructed airways, hence the upper airway obstruction.

Evidence has shown that OSA and obesity are strongly correlated. Two sets of observations support the association: high OSA prevalence among obese children and a higher number of overweight children with OSA. Therefore, the two factors are likely to potentiate and coexist. OSA is present in up to 60 percent of obese children. A research by Marcus et al. [31] found that about 46% of obese children had OSA, while Silvestri et al. [32] found that 59% of children were overweight in a study examining children's sleep disorders. Normally, OSA will decrease significantly when adeno-tonsillectomy is performed in children, only persisting in about 10-20 percent of healthy children. However, even after adeno-tonsillectomy, 50% of obese children continue to experience OSA. In obese children, adeno-tonsillar

hypertrophy is more common than in healthy children, indicating another causal factor in the disorder. The high prevalence of adeno-tonsillar hypertrophy is suspected to be due to endocrine-mediated somatic development leading to larger fat pads, soft palate, and tongue.

Asthma

The prevalence of asthma is increasing and affects about 10 percent of school-age children in the US. Differences in the prevalence of asthma in ethnic and racial terms correlate with those of obesity. The prevalence of asthma among African Americans and Hispanics is higher, especially those from Puerto Rico compared to the case of children from other racial and ethnic groups in the United States [33]. Some reasons for childhood asthma prevalence include early-age allergy susceptibility and inadequate caregiver control of asthma. Truncal adiposity is a major contributor to asthma and other pulmonary deficits during obesity. Truncal adiposity is associated with the diaphragm's mechanical drawbacks as a result of excess fat and is associated with reduced functional residual capacity (FRC), reduced residual volume (RV) and expiratory reserve volume (ERV) [33]. When the RFC is small, it affects the bronchial smooth muscle stretch to the tidal volume exhalation terminal, resulting in increased breathing effort with only daily inspiration. In the second (FEV1) and forced vital capacity (FVC), obese children may have a higher expiratory volume than normal-weight children [33]. Truncal adiposity is associated with the diaphragm's mechanical drawbacks as a result of excess fat and is associated with reduced functional residual capacity (FRC), reduced residual volume (RV) and expiratory reserve volume (ERV) [33]. The combination of lung volume and mechanical restraint shows how obesity in children is predisposed to lower the FEV1/FVC ratio.

Limitations and Future Direction

The research is heavily dependent on previous study data. The study finds that in general, the majority of studies are descriptive and explanatory. More experimental studies, stronger RCTs, meta-analyzes, and systematic reviews are needed. Studies rely heavily on their research sources, which may ultimately affect their integrity and validity. As a result, the study used few meta-analyses, RCT, and systematic reviews. Nevertheless, the research makes an important finding that there is a need for quality studies on the subject. To achieve this goal, experimental studies are needed.

Conclusions and Recommendations

There is a consensus that there is a close relationship between obesity and cardiometabolic risk factors. Specific cardiometabolic factors are associated with obesity, with studies showing the relationship between the two. The evaluation of BMI as a determinant of obesity and its effectiveness in predicting the incidence of obesity and cardiometabolic risk factors is an ongoing research. In the study of obesity and cardiometabolic risk factors, several difficulties are faced, including the concept of obesity, particularly in children-related studies. Furthermore, long-term studies involving children are made difficult by the transient existence of communities, making it difficult to monitor changes in participants in the study.

Research from the study shows that in addition to weight and obesity management, more needs to be done to treat obesity and cardiometabolic risk factors. This is because obesity and cardiometabolic factors use genetic and environmental factors to build their base. Nevertheless, changes in lifestyle have a significant impact on obesity and cardiometabolic risk factors prevention and management. An important observation made in this analysis is that either cohort studies or consensus groups are the majority of evaluations. Although there is sufficient information on childhood obesity and related risk factors for cardio metabolism, stronger, valid and reliable evidence such as RCTs and meta-analysis is needed.

This paper's recommendations focus on high-quality evidence produced through analysis. There is a need for substantially high-quality data, such as through processes such as meta-analysis and RCTs, to help make significant progress in research on the subject. Therefore, data analysis approaches need to be updated to help explain emerging trends such as why some children may develop mild obesity and mild BP after childhood and retain a mild severity of cardiometabolic risk factors over the years, while others may grow the same at a faster rate in life despite having normal childhood without obesity and other cardio metabolic risk factors.

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Appendices

#	Author & Date	Type of Evidence	Sample and Setting	Relevant Findings	Observable Measures	Limitations	Level of Evidence
1	Wang H, Blanco E, Algarin C, Peirano P, Burrows R, Reyes M, Gahagan S(2016) [8]	Non-experimental study	223	Weight status and physical activity influence cardiometabolic risk factors in adolescents	MVPA, triglycerides, systolic blood pressure, weight	Small sample size making it difficult to track small changes	III High
2	Hetheringt on-Rauth M, Bea JW, Lee VR, Blew RM, Funk J, Lohman TG, Going SB (2017) [13]	Non-experimental Study	288	Adiposity is directly related to cardiometabolic risk factors except for fasting glucose	HOMA-IR, TG, HDL-C, LDL-C,	Simple classification pf BMI into normal, overweight, and obese groups instead of using BMI as a continuous predictor in various regression models.	III High
3	Nielsen LA, Nielsen TRH, Holm JC (2015) [23]	Meta-synthesis	N/A	Familial predispositions imply children acquiring cardiovascular disease and obesity	Obesity, CVD, Type II diabetes	The study does not explore familial preferences to second – degree relatives	II Good
4	Ayer J, Charakida M, Deanfield	Systematic Review	8	Obesity alters the function and structure of the cardiovascular	Obesity, CVD	Does not fully explore factors that could change the	II Good

	JE, Celermajer DS (2015) [14]			system in an unfavorable way		risk of cardiometaboli c risk factors	
5	Bastien M, Poirier P, Lemieux I, Despres JP (2014) [8]	Non-experimental study	N/A	Adipose tissue is an essential determinant of cardiovascular risk factors	BMI, Adipose tissue	N/A	III High
6	Jokinen E (2015)	Non-experimental study	N/A	Forms a correlation between obesity/overweight and cardiovascular disease	LDL, cholesterol levels, BP, tobacco smoking, diabetes, gender, physical activity	N/A	III High
7	Herouvi D, Karanasio s E, Karayiann i, C, Karavana ki, K (2013) [10]	Non-experimental	N/A	Explores methods of measurement of endothelial dysfunction in obese children and adolescents	Arterial stiffness, carotid intima, endothelial function	N/A	III Good
8	McMullen S (2014) [20]	Non-experimental study	N/A	Explores how the long-term risk of metabolic and CVD due to childhood obesity can be mitigated	N/A	N/A	III High
9	Nadeau KJ, Maahs DM,	Explanator y mixed method	N/A	Explore the impact of childhood obesity on adult	N/A	N/A	II

	Daniels SR, Eckel RH (2011) [15]	design Level II Quantitative Study		obesity, surrogate markers of CVD, components of metabolic syndrome, and the development of CVD			
10	Perng W, Gillman M W, Fleisch AF, Michalek RD, Watkins SM, Isganaitis E, Oken E (2014)	Non-experimental study	262	Branched amino acid and androgen metabolites are linked to adiposity, and cardiometabolic risk during mid-childhood and that maternal obesity may contribute to altered offspring BCAA metabolism	HOMA-IR, triglycerides, leptin, adiponectin, hsCRP, IL-6		III High
11	Nadeau KJ, Maahs DM, Daniels SR, Eckel RH (2011) [15]	Explanatory (Level II)	N/A	Explore treatment strategies for obesity to improve cardiometabolic risk factors and adverse clinical outcomes	N/A	N/A	II High
12	Aguilera CM, Olza J, Gil A (2013) [12]	Explanatory mixed methods (Level I)	N/A	Metabolic syndrome is a result of the interaction between genetic and environmental factors	TG, HDL-c, BP, Fasting plasma glucose	N/A	I High
13	Llewellyn A, Simmonds M, Owen CG, Woolacott N (2016) [16]	Systematic Review Meta-analysis	37	Childhood obesity increases the chances of adult obesity but not sufficient to be a predictor of adult obesity.	BMI, diabetes, coronary heart disease	N/A	I High

14	Litwin SE (2014) [17]	Explanatory mixed method design Level I Quantitative Study	N/A	Establishes a correlation between childhood obesity and cardiovascular disease	N/A	N/A	I High
15	Wald DS, Bestwick JP, Wald NJ (2007) [30]	Meta-analysis	13	Established a population screening strategy that could help prevent medical consequences for hypercholesterolemia	LDL	N/A	I High”

Appendix 1: Individual Evidence

"Level I • Experimental study • Randomized controlled trial (RCT) • A systematic review of RCTs with or without meta-analysis • Explanatory mixed method design that includes only a Level I quantitative research	Three sources	I	• An illustrative mixed-method article establishing genetics and the environment have a role in the development of the metabolic syndrome • Systematic review/meta-analysis creating that childhood predisposes a child to adult obesity but not a reliable predictor of adult obesity • Meta-analysis for measurement of hypercholesterolemia (recommendations)
Level II • Quasi-experimental studies • A systematic review of a combination of RCTs and quasi-experimental studies, or quasi-experimental studies only, with or without meta-analysis • Explanatory mixed method design that includes only a Level II quantitative study	Four source	I	• Studies address components, development, impact, and management of various cardiometabolic risk factors, obesity, and overweight.
Level III • Non-experimental study • A systematic review of a combination of RCTs, quasi-experimental and nonexperimental studies, or nonexperimental studies only, with or without meta-analysis • Qualitative research or meta-synthesis • Exploratory, concurrent, or multiphasic mixed-methods studies • Explanatory mixed method design that includes only a level III quantitative study	7 article	III	• Tried to establish the correlation between various cardiometabolic factors and obesity/overweight; as well as methods of measurement for the cardiometabolic risk factors
Level IV • Opinions of respected authorities and reports of nationally recognized expert committees or consensus panels based on scientific evidence	Nil		NA

Level V • Evidence obtained from literature or integrative reviews, quality improvement, program evaluation, financial evaluation, or case reports • The opinion of nationally recognized expert(s) based on experiential evidence	Nil		NA”

Appendix 2: Individual Evidence levels