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Addressing Childhood Obesity: An Interdisciplinary Approach in Biochemical Aspects, Public Health, Community Health, Epidemiology, and Paediatrics

Naseabah Ali Mohammed Abu Haddash, Waheid Hadi Mohammed Abu Hadash, Waseem Hadi Mohammed Abu Hadash, Emad Hussain Mohammad Alagsam, Ghala Mohammed Bin Ibrahim Shajiri, Ali Mohammed Ali Kulaybi, Yahya Mohamed Gtabri Daghreri, Mohammed Rashed Mohammed, Mohammed Abduh Alshafyie, Ahmed Ibrahim Ibrahim Alkhawaji, Hanan Abdulrahman Abdulah Ali, Nawal Hassan Ali Moharid, Aisha Hassan Ali Moharid, Areej Ali Ali Awaji, Hanan Mohammed Abdullah Sarafi, Thuraya Saeed Mohammed Al Zahrani, Ahmed Ibrahim Mohammed Khuli, Nojood Mohammed Ahmed Hakami

Ministry of Health, Saudi Arabia

Abstract

Background: Childhood obesity is a significant global health concern associated with a spectrum of comorbidities, including type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD). The complex interplay of genetic, physiological, environmental, and social factors has heightened its prevalence, warranting multidisciplinary intervention strategies.

Aim: This study explores the latest developments in the pathophysiology, comorbidities, biochemical aspects, and management strategies for childhood obesity, emphasizing type 2 diabetes and CVD risk.

Methods: A thorough literature review was conducted using PubMed to analyze original and review articles in English addressing pediatric obesity, T2DM, and CVD risk factors. Studies on epidemiology, pathophysiology, and interventions were included.

Results: Childhood obesity prevalence is rising, exacerbated by socioeconomic, genetic, and environmental factors. Key findings highlight the significant role of adverse childhood experiences and metabolic dysregulation in obesity's pathogenesis. Obesity-induced insulin resistance and β-cell dysfunction underpin the early onset of T2DM, with youth exhibiting more severe disease progression than adults. Treatment strategies include lifestyle modifications, pharmacotherapy, and bariatric surgery, yet their efficacy in pediatrics remains suboptimal. Comorbidities, including NAFLD and psychosocial impacts, further complicate management.

Conclusion: Childhood obesity requires an interdisciplinary approach that integrates public health, pediatrics, epidemiology, and community health. Strategies should prioritize early intervention, preventive measures, and tailored therapeutic approaches to mitigate long-term health impacts.

Keywords: childhood obesity, type 2 diabetes mellitus, cardiovascular disease, pediatric health, metabolic dysregulation, public health interventions.

1. Introduction

As an epidemic that cuts across national, cultural, and economic borders, childhood obesity has emerged as a major worldwide health concern [1, 2]. A complex interplay of genetic, physiological, environmental, and social factors leads to this chronic illness [3]. The early emergence of certain comorbidities, such as type 2 diabetes mellitus (T2DM), cardiovascular disease (CVD) risk, and nonalcoholic fatty liver disease (NAFLD), has been closely associated with the rising frequency of juvenile obesity [4, 5]. The goal of this mini-review is to give a brief overview of the most recent advancements in the pathophysiology of juvenile obesity, with a particular emphasis on type 2 diabetes in children, as well as the mediators that influence the link between childhood obesity and an increased risk of cardiovascular disease. It also discusses the most recent research on the efficacy and applicability of metabolic surgery, medication, and lifestyle modifications in the treatment of obesity, type 2 diabetes, and cardiovascular disease risk factors. With an emphasis on recent research, a thorough search of PubMed was carried out, examining both original and reviewed English-language publications on childhood obesity, pediatric type 2 diabetes, and risk factors for CVD in children.

The epidemiology of childhood obesity

Both domestically and internationally, the prevalence of childhood obesity is on the rise, which is alarming [1, 2]. The prevalence of obesity in children and adolescents aged 2 to 19 years, as measured by a body mass index (BMI) at or above the 95th percentile for age and sex, is 19.7% overall, according to recent statistics from the United States (2017–2020) [6]. Compared to their non-Hispanic White and Asian peers, children who are Hispanic or non-Hispanic Black have a much greater prevalence of obesity [6]. Furthermore, a number of factors, such as the child's age, the degree of obesity, parental obesity, and the trajectory of BMI during childhood, affect the persistence of obesity into adulthood [7]. Obesity in adolescents increases the likelihood that

*Corresponding author e-mail: nabuhaddash@moh.gov.sa (Sarah Abdullah Al Muhandir)
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they will remain obese throughout adulthood. The severity of the illness is directly correlated with the likelihood of problems connected to obesity [4, 5]. From 1% in the early 1970s to 6.1% in 2017–2018, the percentage of children and adolescents with severe obesity—defined as a BMI of 120% or more of the 95th percentile or ≥35, whichever is lower—has sharply increased, nearly quadrupling over the past 50 years, with additional increases noted during the COVID-19 pandemic [5, 8]. To better track children with severe obesity, the CDC has modified its BMI charts to include extra percentiles and extend to a BMI of 60 [9, 10].

Pathophysiology

In the past, obesity was seen through the prism of personal preference, especially with regard to diet and exercise. A variety of biochemical, physiological, social, and environmental variables are involved in the far more complex pathophysiology of pediatric obesity. The five areas of social determinants of health—economic stability, neighborhood and built environment, community, social context, and education access and quality—all have a major impact on the prevalence of childhood obesity [11]. Obesity development has been closely associated with adverse childhood experiences (ACEs), especially those that impact females [12]. By modifying metabolic, immunologic, and epigenetic mechanisms essential to energy balance, ACEs and more general social determinants of health both raise the risk of obesity [13]. Together, these elements add to the rising prevalence of childhood obesity and make it more difficult to lower its prevalence and lessen its long-term health effects.

Over the last ten years, there has been a growing recognition of the important role that biological and genetic factors play in the development and progression of obesity [14]. Between 40% and 75% of the variation in obesity is thought to be due to hereditary factors [15]. Obesity is caused by specific genetic abnormalities, including those in the leptin melanocortin pathway, among which mutations in the melanocortin 4 receptor (MC4R) route are the most common. Approximately 2–6% of children who are obese before the age of five carry these mutations [16]. Additionally, obesity is frequently seen in a number of syndromes, including Bardet-Biedl and Prader-Willi syndromes. Nonetheless, most cases of obesity are polygenic, meaning that several genes contribute to its development, or multifactorial in nature [17]. The development of obesity is also significantly influenced by environmental variables. Obesity has been associated with the use of fast food, high glycemic index foods, sugar-sweetened beverages, and large portion sizes [18]. Obesity is also influenced by sedentary behavior, which includes more time spent on screens for leisure. Sedentary behavior has a negative impact on sleep, replaces physical exercise, and degrades diet quality, among other processes, which all contribute to weight increase [20].

Obesity has been causally linked to inadequate sleep duration and abnormal sleep patterns [21, 22]. Weight gain is encouraged by circadian misalignment, in which eating and waking take place during the body's normal sleep cycle. The underlying mechanisms include changes in food choices and intake, decreased physical activity, and disturbances in hormones that regulate hunger (e.g., peptide-YY, ghrelin, and leptin) [22]. Obesity and the metabolic disorders that accompany it are also influenced by environmental and nutritional factors throughout the intrauterine and early infancy stages. The birth weight and future childhood obesity risk of a kid are predicted by the mother's pre-pregnancy weight and gestational weight increase [23]. It has been demonstrated that breastfeeding has a preventive effect against childhood obesity [24]. Rapid weight gain in infancy is associated with an increased risk of obesity in later childhood and may occasionally be a sign of an underlying genetic etiology [25].

Significant weight gain has been linked to a number of medications, including glucocorticoids, antipsychotics (such olanzapine and risperidone), and antiseizure medications. Less than 1% of obese children have endocrine abnormalities, which include growth hormone insufficiency, Cushing syndrome, hypothyroidism, and pseudohypoparathyroidism type 1a. Children who acquire weight for endocrine reasons usually have short stature or reduced growth velocity. According to this research, energy homeostasis may be impacted by variations in the gut microbiota composition between obese and normal-weight people [26]. Additionally, a higher prevalence of obesity in later life has been associated with the use of antibiotics during pregnancy and infancy [27]. Hyperphagia and reduced energy expenditure can be caused by hypothalamic dysfunction, including damage to the ventromedial or paraventricular parts of the hypothalamus from diseases like brain tumors, trauma, encephalitis, radiation, or surgery [28].

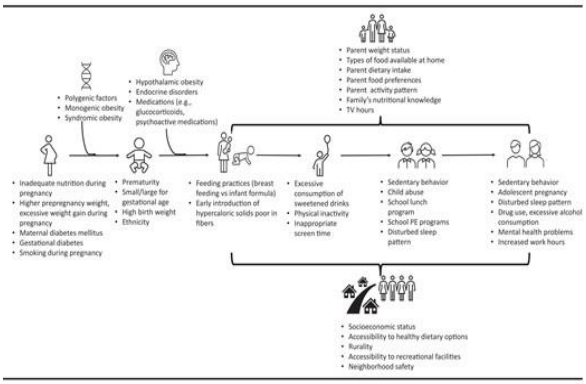

Figure 1: Contribution to Development of Childhood obesity.

Problems Associated with Obesity

Children who are obese are far more likely to acquire type 2 diabetes mellitus (T2DM) and other cardiovascular disease (CVD) risk factors. Nonalcoholic fatty liver disease (NAFLD), obstructive sleep apnea, asthma, pseudotumor cerebri, and musculoskeletal and orthopedic conditions are further consequences linked to obesity. Depression, anxiety, low self-esteem, social isolation, and disrupted peer interactions are among the psychological effects of obesity that are frequently underestimated, especially in children [29]. The link between childhood obesity, type 2 diabetes, and the risk of cardiovascular diseases is the main emphasis of this mini-review, even though these problems must be addressed.

Diabetes as Complications:

The incidence of Type 2 Diabetes Mellitus (T2DM) in teenagers has been closely linked to the rising prevalence of obesity. The mechanisms behind the development of type 2 diabetes in obese children are examined in this part, along with the distinctive characteristics of pediatric type 2 diabetes in comparison to adult-onset type 2 diabetes and the most recent developments in pharmacological and surgical treatments for treating pediatric type 2 diabetes.

According to a cross-sectional, multicenter study conducted in the United States, the prevalence of type 2 diabetes in young people nearly doubled between 2001 and 2017, rising from 0.34 per 1000 adolescents to 0.67 per 1000 youth [30]. T2DM prevalence varies significantly by race and ethnicity, with non-Hispanic Black and Native American kids having the greatest rates [31]. Moreover, T2DM is more common in kids and teenagers from low socioeconomic backgrounds [32]. Notably, T2DM has become more common and severe during the COVID-19 pandemic, which may indicate that the worldwide crisis has exacerbated pediatric obesity and diabetes rates [33]. Obesity-induced insulin resistance (IR), which is associated with adipocyte dysfunction—a critical component in the development of both type 2 diabetes and cardiovascular disease—is a significant risk factor for pediatric T2DM. Adipokine dysregulation, oxidative stress, endoplasmic reticulum stress, and persistent low-grade inflammation are some of the multifactorial processes that underlie IR in obesity [34]. Increased levels of free fatty acids and adipokines, including resistin, leptin, interleukin-6, and tumor necrosis factor α, are caused by obesity and hinder the action of insulin by increasing the synthesis of glucose in the liver and decreasing the uptake of glucose by skeletal muscles [35, 36]. Insulin resistance is further made worse by decreased expression of adiponectin, a protein generated from adipocytes that is insulin-sensitizing [37]. This series of metabolic disturbances leads to insulin resistance and compromised β-cell activity, which is a major factor in the development of type 2 diabetes in obese children [38, 39].

In addition to obesity, genetic and environmental variables also play a role in the pathophysiology of type 2 diabetes in children. Children from high-risk ethnic groups or those with a family history of type 2 diabetes had a more marked progression from prediabetes to the disease. Seven loci linked to youth-onset type 2 diabetes were discovered by the ProDiGY Consortium through a genome-wide association study, providing insight into the genetic foundations of the condition [40]. By raising total body fat, especially visceral fat, environmental variables such excessive calorie intake, poor food, sedentary lifestyles, and decreased physical exercise also contribute to the pathophysiology of type 2 diabetes. T2DM is also characterized by dysregulated α-cell function, which exacerbates hepatic glucose production and postprandial hyperglycemia by impairing postprandial glucagon suppression [41, 42]. Insulin secretion and glucose metabolism are mostly controlled by incretins, which are gut hormones secreted by enteroendocrine cells in response to dietary intake. The degree of obesity in individuals with type 2 diabetes is correlated with a considerable reduction in the postprandial release of glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 (GLP-1) [43, 44]. Oral glucose tolerance tests, however, did not reveal any appreciable variations in incretin hormone levels between children with obesity and those with prediabetes and normal glucose tolerance [46].

The clinical course of juvenile type 2 diabetes is significantly different from that of adult-onset type 2 diabetes. The increased prevalence of poor glycemic control in children with type 2 diabetes may be explained by the fact that youth had nearly twice the insulin levels of adults and much lower insulin sensitivity, according to a study comparing obese adolescents to matched adults. This phenomenon is explained by the increased load on β-cells, which deteriorates glycemic control and causes premature β-cell failure [47]. Furthermore, about 50% of young people fail metformin treatment, compared to 21% of adults, making the cumulative failure rate of metformin therapy in young people more than twice as high as that of adults [48, 49]. Furthermore, microvascular problems such diabetic retinopathy, neuropathy, and nephropathy appear earlier and worsen more quickly in teenagers. According to a study of 500 young individuals with type 2 diabetes, 60.1% experienced microvascular problems, and after 15 years, the cumulative incidence of diabetic nephropathy was 54.8% [50]. Additionally, despite the youth's relatively young age, significant cardiovascular events such as myocardial infarction, stroke, and congestive heart failure were noted [50]. The identification of pediatric-specific glycemic thresholds as indicators of long-term problems and the pressing need for age-appropriate management techniques for pediatric type 2 diabetes are both highlighted by these findings.

Risk Factors for Cardiovascular Disease and Type 2 Diabetes

Due to a complex interaction between hyperglycemia, insulin resistance (IR), genetic predisposition, elevated body mass index (BMI), and environmental factors, children with Type 2 diabetes mellitus (T2DM) frequently exhibit multiple cardiometabolic risk factors, such as dyslipidemia and hypertension [51]. Among 283 pediatric T2DM patients in a study by the SEARCH for Diabetes in Youth , 39% had triglyceride levels above 150 mg/dL, 44% had high-density lipoprotein cholesterol (HDL-C) levels below 40 mg/dL, 33% had total cholesterol levels over 200 mg/dL, and 9% had low-density lipoprotein cholesterol (LDL-C) levels above 160 mg/dL [51]. Subsequent analysis of the TODAY trial showed that 4.5% of participants undergoing LDL-lowering therapy or had LDL-C values higher than 130 mg/dL; after 36 months of follow-up, this percentage rose to 10.7% [52]. Additionally, 11% of individuals had hypertension at the beginning of the research, and 21% of respondents had triglyceride levels exceeding 150 mg/dL at baseline. Microalbuminuria rose from 6.3% at baseline to 16.6% at the end of the research, and 33% of adolescents had hypertension by the end of the study [50]. Compared to adolescents with type 1 diabetes, adolescents with type 2 diabetes are far more likely to experience problems from their diabetes, and these consequences typically appear sooner in the course of the disease [53].

Risk of Cardiovascular Disease Associated with Obesity

Although childhood obesity and eventual adult cardiovascular disease (CVD) have a complex association, obesity is a major factor in the worsening of CVD risk. This section gives a quick summary of the main mediators that link juvenile obesity to an elevated risk of CVD, with a focus on the significance of novel biomarkers that are essential for detecting and tracking pediatric obesity-related cardiovascular risk.

Because childhood obesity is directly linked to deteriorating cardiometabolic outcomes, its persistence into adulthood is especially worrying. Childhood obesity has been identified as a critical factor in the early development of atherosclerosis disease thanks to landmark investigations like the Bogalusa Heart Study, the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study, and the Muscatine Study [54, 55, 56]. The clinical importance of childhood cardiovascular risk factors and their role in vascular organ damage were further highlighted by these research. The relationship between childhood obesity and modifiable cardiovascular risk factors, such as dyslipidemia, hypertension, and vascular changes, has been further investigated [57–59]. The prevalence of CVD risk factors increased in a dose-dependent manner, with the highest rates seen in obese adolescents and the lowest in those of normal weight, according to data from the National Health and Nutrition Examination Survey (NHANES), which covered adolescents aged 12 to 19 from 1999 to 2008 [59]. The majority of infants are born with Ideal Cardiovascular Health (ICVH) [62,63], and only certain genetic or congenital problems impact this baseline state, despite the fact that obesity plays a major role in the course of CVD. A protracted, asymptomatic phase characterized by subclinical alterations usually precedes the clinical manifestation of CVD, and the loss of ICVH is a gradual and ongoing process [62, 64, 65]. Accordingly, CVD is sometimes thought of as a continuum that advances from low-risk to intermediate-risk, which is marked by subclinical alterations, and finally to high-risk states with clinical CVD. The absence of effective, long-term strategies for the prevention and management of these risks, as well as the difficulty of evaluating risk during the subclinical stage of atherosclerosis in youth, are among the major obstacles to the early prevention and management of obesity and its progression to CVD.

Since excess adiposity plays a major role in the development of cardiovascular disease (CVD) risk factors, it poses a serious obstacle to preserving Ideal Cardiovascular Health (ICVH) during youth [64]. It is crucial to remember that not all obese children develop metabolic comorbidities, such as cardiovascular disease [54, 55, 66–68]. Finding risk factors and biomarkers is crucial for both following the trend toward CVD and detecting ICVH worsening early on, as most pediatric cases do not exhibit overt CVD [69, 70]. Although conventional risk factors like blood pressure (BP), lipid levels, glucose concentrations, and an unfavorable body mass index (BMI) offer useful information, they are insufficiently sensitive to take into consideration the variability in children's susceptibility to CVD and risk prediction [62,71]. Reliable methods of tracking vascular and structural alterations are provided by imaging markers of CVD, such as carotid intima-media thickness, pulse wave velocity, and flow-mediated dilatation [72,73]. However, the availability of these methods in standard clinical settings is restricted due to their high cost and specialized skill requirements. Alternative clinical indicators, such as wrist circumference, which has been linked to both left ventricular hypertrophy and insulin resistance (IR), have been proposed by several research [74, 75]. Notwithstanding these encouraging results, additional validation is required due to the inconsistent outcomes [76]. Furthermore, new biomarkers might identify changes in the evolution of CVD risk earlier than conventional markers [4, 62, 71, 72, 77, 78]. The prediction accuracy of conventional CVD risk variables may be improved by these novel biomarkers [62, 71, 79]. Despite progress, we still don't fully grasp the mechanisms and intermediate biomarkers that connect childhood obesity to CVD [62, 68, 71]. The importance of early prevention was emphasized in recent National Heart, Lung, and Blood Institute (NHLBI) proceedings, which highlighted the pressing need for improved biomarkers and innovative monitoring techniques to track the progressive loss of ICVH and the onset of CVD risk [62]. The effectiveness of early interventions may be increased by implementing preventative measures before clinical symptoms of CVD appear by finding biomarkers that represent the downstream effects of excess adiposity. Dysfunction in different signals across numerous organs, such as the gut, liver, muscle, and adipose tissue, as well as interactions within and between these systems, orchestrate the complex processes that contribute to the continuum of CVD risk [68]. Changes in these signaling pathways brought on by obesity promote and quicken the development of CVD risk by causing endothelial damage, myocardial and vascular remodeling, and the start of atherosclerotic processes [4, 68, 71, 77, 80].

Biochemical Aspects of Obesity:

Obesity is a complex metabolic disorder characterized by excessive fat accumulation resulting from an imbalance between energy intake and expenditure. It is associated with numerous biochemical alterations that influence lipid metabolism, glucose homeostasis, and inflammatory pathways. Understanding the biochemical underpinnings of obesity is essential for developing effective prevention and treatment strategies.

Adipose Tissue Dysfunction

In obesity, adipose tissue, a primary energy storage site, undergoes hypertrophy (increase in cell size) and hyperplasia (increase in cell number). This expansion leads to adipocyte dysfunction, triggering the release of excess free fatty acids (FFAs) into the bloodstream. Elevated FFAs impair insulin signaling in peripheral tissues such as skeletal muscle and liver, contributing to insulin resistance. Furthermore, adipose tissue acts as an endocrine organ, secreting various adipokines, including leptin, adiponectin, and resistin. Obese individuals often exhibit leptin resistance, reducing the hormone's ability to suppress appetite and regulate energy expenditure. Concurrently, levels of anti-inflammatory adiponectin decrease, exacerbating metabolic disturbances.

Inflammation and Oxidative Stress

Chronic low-grade inflammation is a hallmark of obesity. Enlarged adipocytes recruit immune cells, particularly macrophages, to the adipose tissue. These

immune cells release pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP). These cytokines interfere with insulin receptor signaling, amplifying insulin resistance. Additionally, obesity-induced oxidative stress arises from excessive production of reactive oxygen species (ROS) due to mitochondrial dysfunction in adipose tissue and other organs. ROS not only damage cellular components but also activate inflammatory pathways, creating a vicious cycle of metabolic dysfunction.

Lipid and Lipoprotein Metabolism

Obesity alters lipid metabolism, leading to dyslipidemia, a condition characterized by elevated triglycerides, low high-density lipoprotein (HDL) cholesterol, and high low-density lipoprotein (LDL) cholesterol levels. Excess FFAs from adipose tissue are transported to the liver, promoting triglyceride synthesis and accumulation. This contributes to the development of non-alcoholic fatty liver disease (NAFLD), a common comorbidity in obesity. The imbalance in lipid profiles increases the risk of cardiovascular diseases, a leading cause of morbidity and mortality in obese individuals.

Glucose Metabolism and Insulin Resistance

Obesity significantly impacts glucose metabolism. Insulin resistance, a key feature of obesity, arises from the combined effects of increased FFAs, inflammatory cytokines, and impaired adipokine secretion. As insulin sensitivity declines, glucose uptake by muscle and adipose tissue decreases, while hepatic glucose production increases. Over time, the pancreas compensates by producing more insulin, resulting in hyperinsulinemia. If this compensatory mechanism fails, type 2 diabetes mellitus (T2DM) develops.

Hormonal Regulation

The hypothalamus plays a central role in energy balance by integrating signals from hormones such as leptin, ghrelin, and insulin. In obesity, dysregulation of these hormonal pathways disrupts appetite control and energy expenditure. Elevated levels of ghrelin, a hunger-stimulating hormone, further perpetuate overeating, while leptin resistance diminishes satiety signaling. The biochemical aspects of obesity encompass a wide range of alterations in adipose tissue function, inflammatory responses, oxidative stress, lipid metabolism, and hormonal regulation. These changes collectively contribute to the development of metabolic syndrome, diabetes, cardiovascular diseases, and other obesity-related complications. Addressing these biochemical mechanisms through targeted interventions is vital for combating obesity and its associated health risks (**Figure 2**).

Interventions of Public Health:

The Prevention of Obesity

It is crucial to define obesity prevention before examining how Precision Population Health (PPH) may improve childhood obesity prevention. Frameworks for preventing other chronic diseases, including diabetes or cardiovascular disease, provide important insights because obesity is a chronic, relapsing disorder. Primary prevention in this context refers to measures taken to stop obesity before it starts. In order to stop the growth of obesity and lessen related medical consequences, secondary prevention involves early detection, diagnosis, and treatment. Lastly, controlling existing obesity to lessen its effects and stop new complications is the main goal of tertiary prevention. Primary prevention measures have received a lot of attention lately when it comes to preventing childhood obesity.

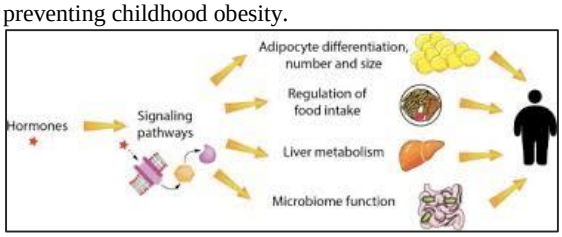

Figure 2: Biochemical Aspects of Obesity.

Establishing surroundings that support health and lower risk factors for childhood obesity is the goal of primary preventive initiatives. Examples include universal laws like the breastfeeding-friendly hospital effort, front-of-package nutrition labeling, charges on sugar-sweetened beverages, limitations on food marketing, and school-based initiatives that have been put into place in many different countries [81, 82]. Many of these policies, nevertheless, are still either sometimes implemented or do not have full legal enforcement. Additionally, there is little data showing long-term decreases in the prevalence of childhood obesity, despite the fact that these actions are essential for promoting good starts in childhood [82]. The limitations of single-policy interventions, which can influence health behaviors but fall short in addressing the complex factors that contribute to obesity, may be the cause of this lack of long-lasting effect. Numerous programs to reduce obesity have been put into place in communities, schools, and families around the world. While physical activity interventions alone had no effect on older children up to the age of 18, a Cochrane analysis found that combining physical activity with dietary interventions reduced the risk of obesity in children under five by a modest amount [83]. Similar to initiatives at the policy level, school-based nutritional interventions seem to affect behavior but only slightly lower children's body mass index (BMI) [84]. Strengthening current policies and investigating the combined benefits of various policy initiatives are essential going forward. Furthermore, it is important to investigate how PPH might provide novel insights into the social and environmental elements affecting childhood obesity. Such knowledge could guide the creation of evidence-based, more successful policies and actions.

Precision Public Health (PPH) and Childhood Obesity Prevention

Precision Public Health (PPH), a new idea with many definitions, is still not widely used in scientific literature, especially when it comes to studies on obesity. PPH's poor field visibility was brought to light in 2018 and continues to this day [85]. Only 85 publications with the term "precision public health" in their titles were found by a PubMed search on January 11, 2023; by March 1, 2023, that number had slightly increased to 86. Only two hits, both written by the same study group and unrelated to childhood obesity, appeared when the search term "obesity" was added [86, 87]. Based on a selection of publications, this review takes a wide view of PPH and looks at developments in the study of pediatric obesity in relation to surveillance, risk factor identification, intervention, and implementation.

Surveillance as a Foundation for Prevention

Strong surveillance systems are essential to PPH and public health in general. Numerous countries have put in place mechanisms to keep an eye on the health of their children; the Childhood Obesity Surveillance Initiative (COSI) in Europe is a prime example. For more than ten years, COSI, which is overseen by the World Health Organization, has gathered standardized data on more than 300,000 elementary school students, including determining variables and BMI readings [88]. Although this data is used to inform national and European policy, it does not have a widely usable digital interface. There are also cutting-edge surveillance techniques, such a pan-European initiative that used smart gadgets to collect a variety of data from 5,500 kids, such as accelerometry, food images, and surveys [89]. Although concerns about informed consent, privacy, and inclusivity were brought to light, the project transformed these data into dashboards for stakeholders. Additionally, utilizing tiny spatial units pertinent to policy-making, a worldwide geospatial mapping endeavor examined the incidence of overweight children under five in 105 nations. An example of how research might address scientific concerns and education policy is the easily navigable visualization tool used in this study [90].

Risk Factor Identification and Prediction

Understanding the risk factors for childhood obesity has improved because to data-driven research, which frequently strikes a balance between dataset size and detail. While bigger cohorts give broad demographic patterns, smaller research with established methodologies offer more detailed information. Historical data sets are still useful for studying long-term effects. There is, however, a dearth of research specifically utilizing PPH for risk factors for pediatric obesity. The socioeconomic determinants of childhood obesity are the subject of two noteworthy studies. A study conducted in the United States linked the prevalence of obesity to neighborhood opportunity indices by combining data from 54 cohorts [91]. The results showed that lower social vulnerability and greater opportunity were linked to decreased rates of obesity. A Finnish study that looked at socioeconomic factors influencing obesity found that household income and parental education were important drivers [92]. The need to merge various datasets to guide PPH therapies is emphasized in both studies. Childhood BMI trajectories indicate future risks of obesity-related disorders, such as type 2 diabetes and cancer, in addition to socioeconomic factors [93-95]. For example, a Danish study used large national registers to link childhood BMI patterns to cancer incidence [96]. These findings are further supported by genetic epidemiology, as research using Mendelian Randomization indicate that childhood obesity affects disease risk, however the results differ depending on the condition [96–100]. A growing tool for predicting obesity is electronic health records, or EHRs. Using machine learning to evaluate EHR data, two extensive studies conducted in the United States and Spain were able to predict childhood obesity with intermediate accuracy [101, 102]. Converting these discoveries into PPH applications is still difficult, though. In order to create comprehensive preventative measures, future research should combine individual-level data with environmental and social issues, such as marketing, pollution, and access to green places.

Intervention, Evaluation, and Implementation:

Studies on preventing childhood obesity that meet the exact criteria of precision public health (PPH) are currently lacking in the literature. As a result, there is not enough information available about the assessment and application of PPH in this situation. The COVID-19 pandemic has produced notable instances of PPH applications, wherein extensive monitoring systems and sophisticated data processing enabled quick and focused testing and vaccination campaigns [103]. However, such quick and targeted therapies are not appropriate for obesity because it is a non-communicable illness with a variety of phenotypes and phases of progression [104]. Additionally, PPH has demonstrated potential in genetic testing for disorders with distinct genetic markers. A hospital-based screening program for a genetic condition that primarily affects people of African descent, for example, showed how genomic medicine could lessen health disparities [105]. This pilot project demonstrated that screening and patient information sharing are acceptable, providing a basis for larger-scale research and potential PPH applications. Because obesity is polygenic, genetic testing has limited application in preventing obesity, despite its potential. Genetic testing is still an underutilized strategy for PPH in this area unless certain genotypes or other omics markers linked to obesity are found [106]. In conclusion, although PPH approaches have potential, the complicated and multifaceted character of the disease limits their current relevance to the prevention of pediatric obesity. In order to ensure scalability and equity in public health policies, future initiatives should focus on integrating genetic and omics insights into tailored interventions.

Future Directions:

The emergence of big data presents a great opportunity to advance PPH techniques due to its unparalleled extent, diversity, and velocity. Big data alone, however, won't be sufficient to address the intricate problems facing public health. 33 planned and five organic data sources were assessed for their usefulness in assisting PPH in the prevention of obesity in a scoping review carried out in Queensland, Australia. The review evaluated aspects such data age, granularity, usability, and accessibility. Both forms of data were available, however compared to organic data, planned data was found to be older, more comprehensive, and simpler to use. The results brought to light a number of factors that need to be considered in order to effectively use these resources, such as data access costs, legal constraints, ethics, and technological skills. Even in obesity research, there is limited use of the various data sources that are available, making their incorporation into current public health programs inadequate [107].

There are many obstacles to overcome when incorporating various data sources into obesity prevention plans. Designed datasets are not always easy to utilize and can demand significant time commitments. In a similar vein, organic data is usually unstructured, which makes scientific research challenging. Furthermore, the representativeness of organic data is frequently questioned, and methodological challenges arise due to their vast number and interconnectivity. Data

generalizability is further complicated by participation bias, namely the underrepresentation of people from lower socioeconomic positions (SEPs) [108-110]. These prejudices highlight how crucial it is to make sure PPH programs take socioeconomically disadvantaged groups' vulnerabilities into account. In order to prevent escalating already-existing health disparities, PPH must aggressively address larger environmental and socioeconomic causes, as childhood obesity disproportionately impacts these communities. Complex dataset analysis is made possible by cutting-edge data processing technologies like artificial intelligence and machine learning. Nevertheless, these instruments are constrained by the presumptions and judgments incorporated into their algorithms [111-113]. Integrating disparate datasets is still a major difficulty, especially when trying to comprehend the complex biology of obesity. Enhancing data accessibility and sharing for public health objectives is the goal of initiatives such as the European Health Data Space. However, databases must include comprehensive data, such as socioeconomic and environmental determinants of health, to guarantee that they support effective obesity prevention initiatives [114]. It will take creative approaches and teamwork from a variety of partners to create effective, fair primary prevention plans for childhood obesity if PPH is to make significant strides [115].

Interventions of Family Medicine for Childhood Obesity:

Family medicine plays a pivotal role in addressing childhood obesity, given its holistic approach to patient care and emphasis on prevention, early detection, and long-term management. The family physician often serves as the first point of contact for healthcare, making them uniquely positioned to implement interventions that address the multifaceted causes of obesity, including genetic, behavioral, environmental, and social determinants.

Preventive Interventions and Early Detection

Prevention forms the cornerstone of family medicine's approach to childhood obesity. Family physicians focus on early identification of risk factors, such as excessive weight gain in infancy, familial history of obesity, and unhealthy dietary habits. Regular growth monitoring using tools like body mass index (BMI) percentiles is a standard practice in primary care settings. By identifying trends toward overweight or obesity early, family physicians can initiate timely discussions with families about lifestyle modifications. Screening for comorbidities associated with obesity, including type 2 diabetes, hypertension, and dyslipidemia, is another essential aspect of intervention. Early detection allows for the implementation of tailored interventions that can mitigate the long-term health risks associated with these conditions. Family physicians often incorporate evidence-based guidelines, such as those from the American Academy of Pediatrics (AAP), to ensure comprehensive screening and risk assessment.

Nutritional Counseling and Lifestyle Modifications

One of the core interventions in family medicine is providing nutritional counseling. Physicians guide families in adopting healthier dietary patterns, focusing on balanced meals rich in fruits, vegetables, whole grains, and lean proteins while minimizing the consumption of sugar-sweetened beverages and processed foods. Family physicians also educate parents on portion control and the importance of structured meal times to avoid overeating. Lifestyle interventions often extend beyond dietary advice to include promoting physical activity. Family physicians encourage children and their families to engage in age-appropriate physical activities for at least 60 minutes daily, as recommended by global health authorities. Additionally, physicians address sedentary behaviors, such as excessive screen time, by advocating for structured routines that prioritize active play and limit passive activities.

Behavioral Interventions and Family-Centered Care

Behavioral interventions are integral to addressing childhood obesity. Family physicians employ motivational interviewing techniques to assess readiness for change and foster a supportive environment for behavioral modification. By involving the entire family in the intervention process, physicians emphasize the shared responsibility of creating a health-promoting environment at home. Family-centered care is particularly effective in modifying behaviors contributing to obesity. By addressing parental attitudes, knowledge, and practices regarding nutrition and physical activity, family physicians can influence the broader familial context in which children develop their habits. This approach is especially critical in low-resource settings, where socioeconomic factors often limit access to healthy food and recreational opportunities.

Management of Comorbidities and Coordination of Care

Family physicians play a central role in managing obesity-related comorbidities. Pharmacological interventions, though less commonly used in children, may be considered for specific cases under expert guidance. Referral to subspecialists, such as pediatric endocrinologists or dietitians, is facilitated by family physicians when additional expertise is required. In managing conditions such as type 2 diabetes or hypertension, family physicians ensure continuity of care by coordinating between primary care and specialized services. The integration of psychological support into obesity management is another critical intervention. Recognizing the psychological impact of obesity, including low self-esteem and body image concerns, family physicians may refer children and families to mental health professionals. Cognitive-behavioral therapy (CBT) has shown efficacy in helping children develop healthier habits and coping mechanisms.

Community Engagement and Advocacy

Family physicians often extend their interventions beyond the clinic by engaging in community-based efforts to address childhood obesity. Participation in school health programs, local fitness initiatives, and public health campaigns allows family physicians to advocate for policies promoting healthy environments. Examples include advocating for healthier school lunches, safe recreational spaces, and education programs targeting childhood nutrition and physical activity. Advocacy efforts also focus on addressing systemic barriers, such as food insecurity and socioeconomic disparities, that contribute to childhood obesity. By collaborating with policymakers and public health organizations, family physicians work to create supportive environments that facilitate healthier lifestyle choices for families.

Cultural and Individualized Approaches

Family medicine recognizes the importance of cultural competence in effectively addressing childhood obesity. Physicians tailor interventions to align with the cultural and religious practices of families, ensuring recommendations are both feasible and respectful of individual beliefs. For example, dietary advice may consider traditional food preferences, and exercise recommendations may be adapted to suit cultural norms regarding gender and physical activity. Individualized care plans, which consider the child's age, developmental stage, and family dynamics, enhance the effectiveness of interventions. By involving children in goal-setting and decision-making, family physicians foster autonomy and empower patients to take an active role in their health. The interventions of family medicine in addressing childhood obesity emphasize prevention, early detection, and comprehensive management of the condition and its associated comorbidities. Through nutritional counseling, behavioral support, and community engagement, family physicians address the root causes of obesity while promoting sustainable lifestyle changes. By adopting a family-centered and culturally sensitive approach, family physicians ensure that interventions are inclusive, equitable, and tailored to the unique needs of each family. These efforts not only improve individual health outcomes but also contribute to broader public health objectives aimed at curbing the obesity epidemic.

Conclusion:

Childhood obesity poses a critical challenge to global public health, demanding urgent and coordinated action across disciplines. Its multifaceted etiology, involving genetic predisposition, environmental influences, and socioeconomic determinants, necessitates a comprehensive understanding to inform effective strategies. The strong association between childhood obesity and type 2 diabetes mellitus (T2DM) underscores the importance of early detection and intervention. Unlike adults, children with T2DM exhibit accelerated disease progression, characterized by severe insulin resistance, β-cell dysfunction, and a high prevalence of comorbidities. These complications, including cardiovascular disease (CVD), nonalcoholic fatty liver disease (NAFLD), and psychosocial impairments, significantly impact quality of life and long-term health outcomes. The findings emphasize the necessity for multifaceted interventions. Lifestyle modifications, though foundational, often yield limited success in pediatric populations. Pharmacological treatments and bariatric surgery offer potential, but their implementation must be judicious, given the unique physiological and psychological needs of children. Importantly, public health initiatives should focus on preventive measures, including promoting healthy eating habits, reducing sedentary behaviors, and addressing socioeconomic disparities that exacerbate obesity risk. Integrating educational campaigns into the community and school programs can play a pivotal role in fostering healthier lifestyles. Interdisciplinary collaboration among healthcare providers, public health professionals, and policymakers is critical to addressing this epidemic. By combining medical expertise with community engagement and policy reform, we can create sustainable systems to combat childhood obesity. Future research should prioritize longitudinal studies to evaluate the effectiveness of emerging therapies and interventions tailored to diverse pediatric populations. Addressing childhood obesity is not merely a medical challenge but a societal responsibility, pivotal to safeguarding the health of future generations.

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