



Obesity as a Predisposing Genetic Factor for Development of Pediatric Hypertension in Calabar Municipality, Cross River State, Nigeria

**Nelson Anita Yemi-odae ^{a*}, Unoh Florence Ben ^b,
Nsude Linus Odinakachukwu ^a, Edet Brian Enembe ^c
and Inyang Daniel Ime ^a**

^a Department of Genetics and Biotechnology, University of Calabar, Calabar, Cross River State, Nigeria.

^b Department of Science Laboratory Technology, University of Calabar, Calabar, Cross River State, Nigeria.

^c Department of Public Health, University of Calabar, Calabar, Cross River State, Nigeria.

Authors' contributions

This work was carried out in collaboration among all authors. Author NAY-O designed the study, performed the statistical analysis, and wrote the protocol. Author UFB wrote the first draft of the manuscript. Author EBE managed the analyses of the study. While authors NLO and IDI managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJBGMB/2024/v16i1352

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://www.sdiarticle5.com/review-history/111803>

Original Research Article

Received: 02/12/2023
Accepted: 11/01/2024
Published: 17/01/2024

ABSTRACT

Aims: The study aimed to assess the prevalence of obesity in children, and to evaluate obesity as a risk factor of hypertension among children obtaining treatment at the University of Calabar teaching hospital.

*Corresponding author: E-mail: anitawara@yahoo.com;

Study Design: the study adopted a descriptive cross-sectional survey design involving children between 3 and 18 years attending pediatrics clinic at the University of Calabar teaching hospital was used to achieve the objectives of the study.

Place and Duration of Study: Study was conducted at the University of Calabar teaching hospital Calabar, Cross River state, southern Nigeria between May 2023 and November 2023.

Methodology: data collection was carried out using the following instruments; (a) Semi-structured questionnaire; for anthropometric measurements (the anthropometric measurements were used to determine BMI of respondents) (b) Weighing balance and measuring tape; for measuring the weight and height of respondents in kilogram (kg) and metre (m) respectively (c) Digital blood pressure monitor; for measurement of respondents' blood pressure. Data was collected with the help of the research assistants on a daily basis for 6 weeks from the pediatric unit of the hospital with two research assistants administering the questionnaire while the other took and recorded anthropometric measurements.

Results: BMI percentile of 53 (39.13%) respondents was 95th percentile and above, followed by 35(25.36%) at 5th percentile, 28(20.29%) below 5th percentile, and 22(15.94%) at 85th percentile. 43(31.16%) respondents had elevated blood pressure of 120-129 mm Hg(systolic) and less than 80 mm Hg (diastolic) were recorded for 22(15.94%) and 25(18.12%) respondents respectively. 48(34.78%) respondents had blood pressure higher than 130/80 mm Hg indicative of hypertension.

Conclusion: BP above 130/80 mm Hg indicated a positive correlation between childhood obesity and pediatric high blood pressure.

Keywords: Obesity; predisposing factor; pediatric; hypertension; high blood pressure.

1. INTRODUCTION

“Obesity is the predominant nutritional disorder among children and adolescents in the United States and other regions. A considerable percentage, around 21-24%, of American children and adolescents are categorized as overweight, and an additional 16-18% are classified as obese. The occurrence of obesity is noted to be highest within specific ethnic groups” [1]. “The definition of child obesity commonly relies on the body mass index (BMI) percentile, which evaluates body fat based on a child's weight and height. If a child's BMI is equal to or exceeds the 95th percentile for their age and sex, as determined by growth charts established by health organizations, they are typically considered to be obese” [2]. “The BMI, although not flawless, is a continuous measure of body fatness that takes into account body size and can be easily and reliably quantified in clinical settings” [1]. “It closely correlates with total body fat (TBF), which is estimated using dual-energy x-ray absorptiometry (DEXA) scanning in overweight and obese children. Normal BMI values vary depending on age, sex, and pubertal status” [3]. “Consensus committees have suggested that children and adolescents should be considered overweight or obese if their BMI exceeds the 85th or 95th percentiles, as determined by curves derived from the 1963-1965 and 1966-

1970 NHANES, or exceeds 30 kg/m² at any age” [4].

“Throughout childhood and adolescence, excess fat accumulates when the total intake of energy surpasses the total energy expenditure. This energy imbalance can arise from excessive energy intake and/or reduced energy expenditure, the latter of which is often a consequence of a sedentary lifestyle. This sedentary behavior is particularly associated with excessive television viewing, excessive computer use, and inadequate physical activity. During infancy, excess fat deposition occurs when there is an overabundance of energy provided, especially when there is an alteration in the protein-to-energy ratio. This is frequently observed when feedings are supplemented with additives such as carbohydrates, while the fat and protein content remains the same. Additionally, one study found a higher incidence of obesity in infants who were introduced to solid foods by 4 months of age” [5].

“Childhood obesity predisposes children to insulin resistance, type 2 diabetes, hypertension, hyperlipidemia, liver and renal disease, and reproductive dysfunction. Moreover, it increases the risk of developing obesity and cardiovascular disease in adulthood” [6]. A study by [7] demonstrated “a significant association between low cardiorespiratory fitness, reductions in fitness over time, and weight gain in children aged 6-15

years, as well as an increased risk of being overweight". "Analyses conducted on a cohort of 902 schoolchildren revealed higher waist circumference and disproportionate weight gain over a 12-month follow-up period in children with low cardiorespiratory fitness. The 12-month risk of being classified as overweight was 3.5 times higher in youth with low cardiorespiratory fitness compared to their fit peers" [7].

"Reductions in cardiorespiratory fitness exhibited a significant and independent correlation with increasing BMI. In obese adolescents, the presence of low levels of cardiorespiratory fitness has also been linked to elevated depressive symptoms" [8]. "It is hypothesized that insufficient sleep in young children is associated with an increase in BMI, and this association is believed to be unrelated to other confounding variables such as diet and physical activity" [9]. "Additionally, available data suggests that over a span of five years, an increase in BMI among overweight children aged 6 to 11 is associated with a rise in both systolic and diastolic blood pressure, as well as a decrease in sleep duration" [10].

"Raised blood pressure is a widely acknowledged risk factor for cardiovascular diseases and chronic kidney disease on a global scale" [11-13]. "Furthermore, hypertension significantly contributes to both mortality and disability" [12]. "Previous evidence from both pathophysiology and epidemiology has indicated that childhood hypertension is linked to essential hypertension in adulthood, as well as negative cardiovascular events throughout an individual's lifetime" [14-16]. "The measurement of childhood hypertension is relatively complex and unstable when compared to that of adulthood hypertension" [17,18]. "The prevalence of elevated blood pressure in children, defined as a systolic blood pressure (SBP) or diastolic blood pressure (DBP) equal to or greater than the 95th percentile based on sex, age, and height, has been suggested to exhibit a sustainable decrease of 53.7% in the second visit and 77.7% in the third visit when compared to the initial visit" [19]. "In light of this, the fourth report from the National High Blood Pressure Education Program (NHBPEP) Working Group in the United States has recommended that childhood hypertension be confirmed as high blood pressure on at least three separate occasions, and that the thresholds for high blood pressure

should simultaneously account for variations in age, sex, and body size" [17].

"Speaking from the standpoint of public health, reliable estimations of the prevalence of childhood hypertension are essential in informing appropriate prevention and treatment strategies, as well as evidence-based allocation of healthcare resources and policy development" [20]. Despite the existence of a substantial body of research that has examined the prevalence of hypertension in children and adolescents [21,22], there has been a limited synthesis of global prevalence estimates for childhood hypertension. The objective of the present study was to evaluate the prevalence of obesity in children and to assess obesity as a risk factor for hypertension among children receiving treatment at the University of Calabar Teaching Hospital.

1.1 Childhood Obesity and Health Consequences

The issue of obesity among children and adolescents has gained significant prominence in the medical field. Data from national surveys conducted between the 1960s and 1990s reveal a steady increase in the prevalence of overweight children, rising from 5% to 11% [23]. Researcher also established a link between the surge in obesity rates and an imbalance between energy intake and expenditure, with a positive energy balance closely associated with lifestyle choices and dietary preferences [23]. Furthermore, there is mounting evidence suggesting that an individual's genetic background plays a crucial role in determining the risk of obesity.

Studies have indicated that body mass index (BMI) is heritable to a significant extent, accounting for 25-40% of the variability [24]. However, genetic susceptibility alone is insufficient to influence weight, as it must be combined with environmental and behavioral factors [25]. Genetic factors only account for less than 5% of childhood obesity cases [24]. Therefore, while genetics may contribute to obesity development, it is not the primary cause for the alarming rise in childhood obesity rates. Researchers have also explored the possible influence of basal metabolic rate, which refers to the body's energy expenditure during rest. Basal metabolic rate accounts for 60% of total energy expenditure in sedentary adults. It has been hypothesized that individuals with obesity may have lower basal metabolic rates. However, variations in basal metabolic rates are unlikely to

be responsible for the increasing prevalence of obesity [26].

The escalating epidemic of pediatric obesity has raised significant concerns about its management and associated complications. While prevention should ideally be the main strategy, it can be challenging to identify children at risk of obesity before they become overweight. Even with effective preventive measures, it is expected that many children will still become overweight and require treatment to prevent long-term consequences such as cardiovascular morbidity and mortality [27]. The prevalence of obesity, diabetes, and other nutrition-related chronic diseases is progressively rising. Developed countries have reported the highest prevalence rates of childhood obesity, but developing countries are also experiencing an increase [28].

Comparatively, females are more prone to obesity than males. This is apparently due to inherent hormonal differences [29]. It is now evident that the development of Type 2 Diabetes and Coronary Heart Disease begins in childhood, with childhood obesity serving as a significant contributing factor [30]. Over the past four decades, there has been a remarkable surge in the number of children affected by obesity, particularly in developed nations. Recent studies conducted in various parts of India within the last decade have also shown a similar trend [31-36]. However, this perspective has been challenged in recent years, and it is now regarded as a distinct manifestation of the global malnutrition problem [23].

Given the increasing prevalence of obesity in children, it is accurate to assert that childhood hypertension has experienced an epidemiological shift. The conventional belief has been that hypertension in children is a relatively uncommon condition primarily linked to renal disease. However, in reality, secondary hypertension in children caused by renal disease has become significantly less frequent compared to primary hypertension [37]. Additionally, it has been observed in a substantial pediatric hypertension practice that the typical patient demographic has transformed into an otherwise healthy adolescent with obesity and an amalgamation of the cardiovascular risk factors associated with obesity. These risk factors include a familial history of hypertension and an ethnic predisposition to hypertensive disease.

1.2 Health Implications Childhood Hypertension

Hypertension has been defined as the state in which blood pressure (BP) surpasses a threshold that, when lowered, reduces the cardiovascular risk associated with elevated BP. This threshold, as traditionally reported, has been documented as 140/90 mmHg in adults [38]. The interpretation of BP values in children is influenced by factors such as age, height, and gender. For children, the widely accepted definition of hypertension is characterized by BP values exceeding 95% of the expected BP for their age, gender, and height [39]. In contrast to adults, where essential hypertension is prevalent, children can exhibit both secondary and essential (primary) hypertension. Primary hypertension is notably influenced by factors such as birth weight, maturity at birth, heredity, and diet, while secondary hypertension is influenced by renal abnormalities, coarctation of the aorta, medications, and neoplasm [40].

Presently, hypertension is recognized as the foremost risk factor for morbidity and mortality on a global scale, contributing to 182 million disability-adjusted life years and 10.4 million deaths annually [41]. Hypertension associated with obesity arises from a multifaceted interplay between various mechanisms, including inappropriate activation of the sympathetic nervous system (SNS) and the renin-angiotensin-aldosterone system (RAAS), adipocyte dysfunction, and impaired pressure natriuresis. This condition is further exacerbated by physical compression of the kidneys [42]. It is crucial to acknowledge the increase in RAAS and SNS activation in obesity-related hypertension, as it has implications for the treatment of patients with this condition. Importantly, in obesity, the serum levels of nearly all components of RAAS are elevated, and the amount of adipose tissue present determines the relative quantity of circulating angiotensinogen and angiotensin II [43]. Weight loss, the primary treatment approach for individuals with obesity-related hypertension, leads to a reduction in SNS activity, which directly affects arterial pressure by decreasing peripheral vasoconstriction and indirectly affects arterial pressure by improving pressure natriuresis, resulting in lower intravascular volume. Additionally, weight loss leads to a decrease in renin release from the kidney [44].

Obese children face a significantly elevated risk of cardiovascular diseases (CVD). A study conducted by [45] discovered that these children exhibit higher systolic and diastolic blood pressure (BP) and greater evidence of dyslipidemia and insulin resistance. In fact, 70% of obese children have at least one CVD risk factor, and 39% have two or more. These CVD risk factors, in conjunction with obesity, not only contribute to heart diseases during childhood but also increase the prevalence of CVD risk factors in adulthood, leading to elevated CVD morbidity and mortality rates [46-50].

In addition, the significance of primordial and primary prevention in achieving cardiovascular disease (CVD) risk reduction among young individuals has been emphasized by the American Heart Association and the American Academy of Pediatrics (AAP) [51,52]. A crucial element of this strategy involves regular screening for elevated blood pressure (BP) and hypertension in children. The prevailing guidelines advocate for annual BP measurements in all children aged 3 years and above. Any elevation in BP should be confirmed through repeated measurements, and a diagnosis of hypertension should be assigned to any child with a sustained elevation at or above the 95th percentile when measured by manual auscultation. Furthermore, all children diagnosed with hypertension should undergo evaluation for a potential secondary cause [53].

1.3 Treatment Approach to Obesity-related Hypertension

Regarding the treatment approach to obesity-related hypertension, the primary focus for all children should be on achieving a healthy weight and adopting a lifestyle that promotes heart health. The AAP recommends a staged approach to obesity treatment, with weight loss being recommended for children aged 6 years and above when their BMI falls within the obese category, and weight maintenance being advised for growing children with a BMI within the overweight category [54]. Weight loss is especially critical for children with obesity-related hypertension as it addresses the underlying causes, improves comorbidities, and reduces sympathetic nervous system activation, thereby leading to a reduction in BP [44]. Numerous studies conducted on overweight and obese children aged 6 to 16 years have demonstrated the effectiveness of weight loss in lowering BP. These studies incorporated various interventions

such as diet, physical activity, education, and counseling, and resulted in a decrease in systolic blood pressure (SBP) by 6 to 16 mmHg over periods of 5 to 12 months [55,56, 57].

Changes in diet are also crucial in the treatment of obesity-related hypertension. In 2011, the American Academy of Pediatrics published updated dietary recommendations specifically for hypertension treatment [52]. Regardless of the stage or etiology of hypertension, hypertensive children should follow the cardiovascular health integrated lifestyle diet and the dietary approaches to stop hypertension (DASH) eating plan. This eating plan includes increasing the consumption of fresh vegetables, fruits, and low-fat dairy products, reducing carbohydrate, fat, and processed sugar intake, and limiting or avoiding sugar-sweetened beverages. Additionally, it is important to encourage the intake of foods high in dietary fiber content. Avoiding sugar-sweetened beverages can also contribute to weight loss in children and has been independently associated with a reduction in BP in adults [44]. Studies conducted in 2012 on two randomized controlled trials involving overweight/obese children and normal-weight children respectively, demonstrated that the elimination of sugar-containing beverages led to a decrease in weight and measures of adiposity [58,59].

1.4 Pharmacologic Therapy Approach

The therapeutic approach to treating hypertension in children with obesity should encompass lifestyle modifications. In certain cases, the addition of an antihypertensive medication may be necessary for effective treatment of hypertension. According to [53], children who exhibit symptoms of hypertension and have an underlying cause, such as secondary etiology, type 1 or type 2 diabetes, or end-organ damage like left ventricular hypertrophy, should all be prescribed an antihypertensive medication. Moreover, children who continue to have hypertension after implementing a heart-healthy lifestyle for 6-12 months should also be prescribed medication to lower their blood pressure while they simultaneously focus on weight loss and lifestyle changes.

When selecting a medication, it is crucial to consider the underlying cause of hypertension and address any coexisting conditions. [60] suggests that since the activation of the renin-

angiotensin-aldosterone system (RAAS) is a primary mechanism by which obesity contributes to elevated blood pressure and hypertension, angiotensin converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) would be appropriate initial choices for treating obesity-related hypertension in children.

2. MATERIALS AND METHODS

This study was conducted in Calabar Municipality, in Cross River State, southern Nigeria, which lies between latitude 5° 45 and 5° 75 North of the Equator and longitude 8° 30 and 8° 42 East of the Greenwich Meridian. Temperatures are relatively constant throughout the year with average high temperatures ranging from 25.8 °C/78.5 °F and a low temperature of 23.8 °C. It has an area of 142km² and a population of 179,392 as at the 2006 census. It is a traditional fishing community typical of the Efik people who are the original and major occupants of the area. Major economic activity of the indigenous people include fishing and the making of fishing nets and construction of canoes. Although industrialization has led to the offshoot of other occupations with civil service as a leading occupation of the people. A descriptive cross-sectional survey design involving children between 3 and 18years attending pediatrics clinic at the University of Calabar teaching hospital was used to achieve the objectives of the study. The study population was the 403 pediatric obese and/or hypertensive pediatric patients presenting at the booking pediatrics clinic at University of Calabar teaching hospital during the period of this study.

2.1 Sample Size and Sampling Technique

The sample size was determined statistically by applying the population proportion sample size determination formula:

$$n = Z^2pq/d^2$$

This was concluded by using previous study that assumed children age 3 - 18years with obesity, at the risk of hypertension are 90.0%, and an estimated difference between the actual proportion and the research value to be 0.05 at 95% confidence level $n=z^2p(1-p)/d^2$ sampling distribution of proportion theory.

where;

n = minimum sample size

Z = standard normal deviation at 1.96

p = prevalence rate 90%

$$q = 1-p (1- 0.90) = 0.1$$

d = precision at 0.05

Then,

$$n = (1.96 \times 1.96 \times 0.9 \times 0.1)/0.0025$$

$$n = 138$$

Inclusion Criteria: Children of all gender between ages of 3 and 18 years, with BMI exceeding the 85th or 95th percentiles participated in the study.

Exclusion Criteria: The under five children, and children within the specified age limits who have BMI not exceeding the 85th or 95th percentiles were all excluded.

2.2 Instruments for Data Collection

Questionnaire: semi-structured questionnaire for anthropometric measurements was used to collect data for the study. It consists of Section A (socio- demographic characteristics of parents and children), Section B (on evaluation of obesity and hypertension status of respondents).

Tanita WB-800S Digital Physician Scale and measuring tape: these were used for measuring the weight and height of respondents in kilogram (kg) and metre (m) respectively. The anthropometric measurements were used to determine MBI of respondents.

The A&D Medical UA-767F blood pressure monitor: for measuring blood pressure of respondents.

Validity: the instrument for the study was validated by two experts in pediatrics medicine from University of Calabar Teaching Hospital and in Measurement and Evaluation from University of Calabar.

Reliability: this was obtained by Test-retest method. The questionnaire was pre-tested on 10 respondents similar to target respondents. The structured questionnaire was then be rephrased in relation to the responses from the respondents.

2.3 Data Collection Procedure

Week 1: Three research assistants were trained by the researcher for on the study objectives, purpose and interviewing techniques based on the research instrument. The training included demonstrations and practice in obtaining anthropometric measurements.

Week 2-8: Data was collected with the help of the research assistants on a daily basis from the paediatric unit of the hospital with two research assistants administering the questionnaire while the other one took anthropometric measurements.

2.4 Method of Data Analysis

Data were analysed using SPSS version 20. Descriptive statistics of frequency and percentage were used in the study.

3. RESULTS AND DISCUSSION

3.1 Socio-demographic Characteristics of Respondents

Results on Table 1. shows most respondents fall within 15-18 years age group (40, 28.98%). This was closely followed by 7-10 years (39, 28.26%), 3-6 years (31, 22.46%), and respondents within 11-14year age group (28, 20.29%). More than half (88, 63.77%) of respondents' parents have tertiary education, 28 (20.28%) have primary education, and 22 (15.94%) have studied to secondary education level. Table 1. equally revealed that most parents (97, 70.29%) were married, 30 (21.74%) were either divorced or widowed, and 11 (7.97%) were single mothers. More than half of respondents' parents were employees (87, 63.04%), 41(29.71%), 10 (7.25%) were small business owners.

3.2 Prevalence of Obesity among Children Receiving Treatment at University of Calabar Teaching hospital

Table 2. displays results on prevalence of obesity among children receiving treatment at the University of Calabar teaching hospital. 56 (40.58%) out of 138 respondents affirmed to history of obesity in their respective families, while family history of obesity was not applicable to 82 (59.42%) of respondents. 61 (44.20%) of children participants were already diagnosed with obesity by a health care professional. BMI percentile of 53 (39.13%) respondents was 95th percentile and above, followed by 35(25.36%) at 5th percentile, 28(20.29%) below 5th percentile, and 22(15.94%) at 85th percentile.

3.3 Blood Pressure of Obese Children Receiving Treatment at University of Calabar Teaching Hospital

Results on Table 3 show three categories (normal, elevated, and hypertension) of respondents' blood pressure measurement. Normal blood pressure (less than 120/80 mm Hg) was recorded for 43(31.16%) respondents, elevated blood pressure of 120-129 mm Hg(systolic) and less than 80 mm Hg (diastolic) were recorded for 22(15.94%) and 25(18.12%) respondents respectively. 48(34.78%)

Table 1. Sociodemographic characteristics of preeclampsia respondents

S/N	Variables	Frequency	Percentage (%)
1.	Age range(years)		
	3-6 years	31	22.46
	7-10 years	39	28.26
	11-14years	28	20.29
	15-18years	40	28.98
	138	100	
2.	Parental Education Level		
	Primary	28	20.28
	Secondary	22	15.94
	Tertiary	88	63.77
	138	100	
3.	Parent's Marital Status		
	Single	11	7.97
	Married	97	70.29
	Divorced or Widowed	30	21.74
	138	100	
4.	Occupation		
	Unemployed	41	29.71
	Employee	87	63.04
	Small Business	10	7.25
	138	100	

Source: Field work (2023)

Table 2. Assessment of obesity prevalence among children

S/N	Items	Description	Frequency	Percentage (%)
1.	There is family history of obesity	Yes	56	40.58
		No	82	59.42
			138	100
2.	My child has been diagnosed with obesity by a health care professional	Yes	61	44.20
		No	77	55.80
			138	100
3.	There are current efforts to manage the child's weight	Yes	59	42.75
		No	79	57.25
			138	100
4.	BMI percentile	Less than 5 th percentile	28	20.29
		5 th percentile	35	25.36
		85 th percentile	22	15.94
		95 th percentile and above	53	39.13
			138	100

Source: Field work (2023)

Table 3. Blood pressure assessment of obese children

S/N	Items	Description	Frequency	Percentage (%)
1.	My child's blood pressure has been measured in the last 3 months	Yes	89	64.49
		No	49	35.51
			138	100
2.	Recent blood pressure measurement	Less than 120/80 mm Hg – normal BP	43	31.16
		120-129 mm Hg (Systolic) – elevated BP	22	15.94
		less than 80 mm Hg (Diastolic) - elevated	25	18.12
		higher than 130/80 mm Hg - Hypertension	48	34.78
			138	100
3.	There is history of hypertension in my family	Yes	68	49.28
		No	70	50.72
			138	100
4.	My child been diagnosed of hypertension by a health care professional	Yes	59	42.75
		No	79	57.25
			138	100

Source: Field work (2023)

respondents had blood pressure higher than 130/80 mm Hg which signifies hypertensive condition. Results also revealed the existence of family history of hypertension among 68(49.28%) respondents. 59(42.75%) out of 138 respondents had positive diagnosis for hypertension.

4. DISCUSSION

The prevalence of obesity and hypertension has been steadily increasing among children. Both of these conditions are associated with a higher risk of cardiovascular disease and have a long-term impact, leading to a greater prevalence of heart

disease and mortality in adulthood. The body mass index (BMI), a crucial anthropometric measure of weight and height, provides an unbiased indication of obesity. Since children and teenagers are still growing, the ranges for height, weight, and BMI differ based on age and gender. Consequently, BMI values need to be compared to those of other children of the same age and gender. The majority of participants in this study were adolescents aged 15-18 years, closely followed by children aged 7-10 years. This finding aligns with the European Youth Heart Study, which suggests that obesity is more likely to occur during specific stages of life, such

as early childhood (5-7 years) and adolescence [1]. The accumulation of excess fat during childhood and adolescence is a result of an energy imbalance, where energy intake surpasses energy expenditure. This imbalance can arise from excessive energy consumption and/or reduced physical activity, which is often associated with a sedentary lifestyle characterized by excessive television viewing, computer use, and limited physical activity. In infants, excess fat deposition occurs when there is an excessive supply of energy, particularly when the protein-to-energy ratio is altered. This is frequently observed when feedings are supplemented with carbohydrates or fat while maintaining the same protein content. However, these findings contradict the results reported by [5], who noted an increased incidence of obesity in infants weaned to solid foods by 4 months of age.

The results of this study indicate that 53 respondents had a BMI percentile above the 95th percentile, indicating a high prevalence of obesity. Additionally, there was a positive correlation between family history and BMI percentile, with 40.58% of respondents reporting a family history of obesity. A similar study [61] on families and obesity found that children of overweight parents have an 80 percent likelihood of also being overweight. The influence of weight on genetics and family environment was highlighted in previous research [62], although it was noted that genetics only account for a small portion of this 80 percent chance. [63] supports the notion that a family history of obesity and cardiometabolic diseases are crucial risk factors for the early onset of obesity in childhood and are associated with the severity of obesity [64].

Blood pressure measurements of obese participants revealed that 48 out of 138 respondents (34.78%) had blood pressure levels higher than 130/80 mm Hg, indicating a link between obesity and hypertension. This finding is consistent with the research conducted by [6], which explains the relationship between excess adiposity and elevated blood pressure. It is estimated that obesity contributes to 65-78% of cases of primary hypertension. The mechanisms underlying the relationship between obesity and hypertension are complex and involve factors such as sympathetic nervous system overactivation, stimulation of the renin-angiotensin-aldosterone system, changes in adipose-derived cytokines, insulin resistance, and structural and functional renal changes.

5. CONCLUSIONS

Hypertension has been shown by researchers to increase with obesity in children worldwide. Studies have revealed that weight and hypertension are closely related, and obesity-related blood pressure is a major risk factor for CVDs in societies around the globe. In the present study, elevated systolic BP of 120-129 mm Hg, and diastolic BP of < 80 mm Hg among obese respondents were indicative of possible onset of hypertension. While BP higher than 130/80 mm Hg indicated a positive correlation between childhood obesity and pediatric high blood pressure.

CONSENT

Guardians of participants were informed about the purpose of the study. Informed consent was obtained from the participants in the study and confidentiality was maintained by assuring security and privacy to all participants after being educated on the study and its objectives. Participation in the project was however voluntary.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Schwarz SM. Obesity in Children. Pediatrics: General Medicine; 2023. Available:<https://emedicine.medscape.com/article/985333-overview?form=fpf>
2. WHO. Obesity and overweight. 2018. Available:<https://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight> (Accessed 1 September 2023).
3. Fiore H, Travis S, Whalen A, Auinger P, Ryan S. Potentially protective factors associated with healthful body mass index in adolescents with obese and non-obese parents: a secondary data analysis of the third national health and nutrition examination survey, 1988-1994. Journal of American Diet Association. 1988;106(1):55-64.
4. Flegal KM, Ogden CL, Wei R. Prevalence of overweight in US children: comparison of US growth charts from the centers for disease control and prevention with other reference values for body mass index. American Journal of Clinical Nutrition. 2001;73(6):1086-93.

5. Huh S, Rifas-Shiman S, Taveras E, Oken E, Gillman M. Timing of solid food introduction and risk of obesity in preschool-aged children. *Pediatrics*. 2011;127(3):e544-51.
6. Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. *Gastroenterology*. 2007;132(6):2087-2102.
7. McGavock JM, Torrance BD, McGuire KA, Wozny PD, Lewanczuk RZ. Cardiorespiratory fitness and the risk of overweight in youth: the healthy hearts longitudinal study of cardiometabolic health. *Obesity (Silver Spring)*. 2009;17(9):1802-7.
8. Shomaker LB, Tanofsky-Kraff M, Zocca JM, Field SE, Drinkard B, Yanovski JA. Depressive symptoms and cardiorespiratory fitness in obese adolescents. *Journal of Adolescent Health*. 2012;50(1):87-92.
9. Carter PJ, Taylor BJ, Williams SM, Taylor RW. Longitudinal analysis of sleep in relation to BMI and body fat in children: the FLAME study. *Body Mass Journal*. 2011;342:d2712.
10. Archbold KH, Vasquez MM, Goodwin JL, Quan SF. Effects of sleep patterns and obesity on increases in blood pressure in a 5-Year Period: report from the tucson children's assessment of sleep apnea study. *Journal of Pediatrics*. 2012;25(12):23-45.
11. Danaei G, Lu Y, Singh G. Global burden of metabolic risk factors for chronic diseases collaboration. Cardiovascular disease, chronic kidney disease, and diabetes mortality burden of cardiometabolic risk factors from 1980 to 2010: A comparative risk assessment. *Lancet Diabetes Endocrinology*. 2014;2(8):634-647.
12. WHO. A Global brief on Hypertension: Silent Killer, Global Public Health Crisis: World Health Day. Geneva, Switzerland: World Health Organization; 2013.
13. Zhou B, Bentham J, Di Cesare M. NCD risk factor collaboration (NCD-RisC) worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19.1 million participants. *Lancet*. 2017;389(10064):37-55.
14. Bao W, Threefoot SA, Srinivasan SR, Berenson GS. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: The bogalusa heart study. *American Journal of Hypertension*. 1995;8(7):657-665.
15. Beckett LA, Rosner B, Roche AF, Guo S. Serial changes in blood pressure from adolescence into adulthood. *American Journal of Epidemiology*. 1992; 135(10): 1166-1177.
16. Raitakari OT, Juonala M, Kähönen M. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the cardiovascular risk in young finns study. *JAMA*. 2003;290(17) : 2277-2283.
17. Falkner B, Daniels SR, Flynn JT, et al. National high blood pressure education program working group on high blood pressure in children and adolescents . The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics*. 2004; 114(2): 555-576.
18. Hansen ML, Gunn PW, Kaelber DC. Under diagnosis of hypertension in children and adolescents. 2007; 298: 874–879.
19. Sun J, Steffen LM, Ma C, Liang Y, Xi B. Definition of pediatric hypertension: are blood pressure measurements on three separate occasions necessary? *Hypertension Research*. 2017;40(5):496-503.
20. Akbari M, Moosazadeh M, Ghahramani S. High prevalence of hypertension among Iranian children and adolescents: a systematic review and meta-analysis. *Journal of Hypertension*. 2017;35(6):1155-1163.
21. Noubiap JJ, Essouma M, Bigna JJ, Jingi AM, Aminde LN, Nansseu JR. Prevalence of elevated blood pressure in children and adolescents in Africa: a systematic review and meta-analysis. *Lancet Public Health*. 2017; 2(8):e375-e386.
22. Mccrindle BW. Assessment and management of hypertension in children and adolescents. *National Review Cardiology* . 2010;7(3):155-163.
23. Sahoo K, Sahoo B, Choudhury AK, Sofi NY, Kumar Y, Bhadoria AS. Childhood obesity: causes and consequences. *Journal of Family Medicine and Primary Care*. 2015;4(2):187-191.
24. Anderson PM, Butcher KE. Childhood obesity: Trends and potential causes. *Future Child*. 2006;16:19-45.
25. CDC. Contributing factors; 2023. Available:http://www.cdc.gov//obesity/childhood/contributing_factors.html

26. Neumark-Sztainer D, Wall M, Story M, Standish AR. Dieting and unhealthy weight control behaviors during adolescence: associations with 10-year changes in body mass index. *Journal of Adolescence Health*. 2012;50(1):80-6.
27. Eckel RH, Krauss RM. American Heart Association call to action: obesity as a major risk factor for coronary heart disease. *AHA Nutrition Committee. Circulation*. 1998;97:2099–2100.
28. Popkin BM, Doak, CM. The obesity epidemic is a worldwide phenomenon. *Nutrition Review*. 1998;56:106-14.
29. Gupta RK. Nutrition and the Diseases of Lifestyle. In: Bhalwar RJ, editor. *Text book of public health and community medicine*. 1st ed. Pune: Department of community medicine. AFMC, New Delhi: Pune in Collaboration with WHO India Office. 2009;1199.
30. Bhavé S, Bavdekar A, Otiv M. IAP national task force for childhood, prevention of adult diseases: Childhood obesity. IAP national task force for childhood prevention of adult diseases: childhood obesity. *Indian Pediatrics*. 2004;41:559-75.
31. Chhatwal J, Verma M, Riar SK. Obesity among pre-adolescent and adolescents of a developing country (India). *Asia Pacific Journal of Clinical Nutrition*. 2004;13:231-5.
32. Khadiolkar VV, Khadiolkar AV. Prevalence of obesity in affluent school boys in Pune. *Indian journal of Pediatrics*. 2004;41:857-8.
33. Laxmaiah A, Nagalla B, Vijayaraghavan K, Nair M. Factors affecting prevalence of overweight among 12 to 17 year old urban adolescents in Hyderabad, India. *Obesity*. 2007;15:1384-90.
34. Panjikkaran ST, Kumari K. Augmenting BMI and Waist-height ratio for establishing more efficient obesity percentiles among school children. *Indian Journal of Community Medicine*. 2009;9(34):135-9.
35. Raj M, Sundaram KR, Paul M, Deepa AS, Kumar RK. Obesity in Indian children: Time trends and relationship with hypertension. *National Medicine Journal of India*. 2007;20:288-93.
36. Subramanyam VRJ, Rafi M. Prevalence of overweight and obesity in affluent adolescent girls in Chennai in 1981 and 1998. *Indian Pediatrics*. 1998;40:332-6.
37. Barati L, Radgoodarzi M, Vakili M, Tabatabaizadeh M. Overweight and obesity: worldwide risk factors for pediatric hypertension. *ARYA Atheroscler*. 2022; 18(1):1–6.
38. Schiffrin EL, Calhoun DA, Flack JM. Do we need a new definition of hypertension after SPRINT? *American Journal of Hypertension*. 2016;29:1127-9.
39. Patel N, Walker N. Clinical assessment of hypertension in children. *Clinical Hypertension*. 2016;22:15.
40. Hill KD, Li JS. Childhood hypertension: An underappreciated epidemic? *Pediatrics*. 2016;138. pii:e20162857.
41. GBD. Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the global burden of disease study. *Lancet*. 2017;392:1923-94.
42. Omair AS, Travis JM. Obesity-related hypertension: a review of pathophysiology, management, and the role of metabolic surgery. *Gland Surgery*. 2020;9(1):80–93.
43. Millar PJ, Floras JS. Statins and the autonomic nervous system. *Clinical Science*. 2014;126: 401–15.
44. Brady TM. Obesity-related hypertension in children. *Front Pediatrics*. 2017;5:197.
45. Friedemann C, Heneghan C, Mahtani K, Thompson M, Perera R, Ward AM. Cardiovascular disease risk in healthy children and its association with body mass index: Systematic review and meta-analysis. *Body Mass Journal*. 2012;345:e4759.
46. Berenson GS, Srinivasan SR, Bao W, Newman WP, Tracy RE, Wattigney WA. 1998 Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. *The Bogalusa Heart Study. National England Journal of Medicine*. 2012; 338:1650–6.
47. Brady TM, Fivush B, Flynn JT, Parekh R. Ability of blood pressure to predict left ventricular hypertrophy in children with primary hypertension. *Journal of Pediatric*. 2008;152:73–8.
48. Brady TM, Appel LJ, Holmes KW, Fivush B, Miller ER. Association between adiposity and left ventricular mass in children with hypertension. *Journal Clinical Hypertension*. 2016;18:625–33.
49. Morrison JA, James FW, Sprecher DL, Khoury PR, Daniels SR. Sex and race

- differences in cardiovascular disease risk factor changes in schoolchildren, 1975–1990: the Princeton School Study. *American Journal of Public Health*. 1999;89:1708–1714.
50. Strong JP, Malcom GT, McMahan CA, Tracy RE, Newman WP, Herderick EE. Prevalence and extent of atherosclerosis in adolescents and young adults: implications for prevention from the pathobiological determinants of atherosclerosis in youth study. *JAMA*. 1999; 281:727–35.
 51. Weintraub WS, Daniels SR, Burke LE, Franklin BA, Goff DC, Jr, Hayman LL. Value of primordial and primary prevention for cardiovascular disease: A policy statement from the American Heart Association. *Circulation*. 2011;124:967–90.
 52. EPI. Integrated guidelines for cardiovascular health and risk reduction in children and adolescents; National heart, Lung, and blood institute. expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011;128(5):S213–56.
 53. NHBP. Education program working group on high blood pressure in children and adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics*. 2004;114:555–76.
 54. Spear BA, Barlow SE, Ervin C, Ludwig DS, Saelens BE, Schetzina KE. Recommendations for treatment of child and adolescent overweight and obesity. *Pediatrics*. 2007;120(4):S254–88.
 55. Reinehr T, de Sousa G, Toschke AM, Andler W. Long-term follow-up of cardiovascular disease risk factors in children after an obesity intervention. *American Journal of Clinical Nutrition*. 2006;84:490–6.
 56. Reinehr T, Schaefer A, Winkel K, Finne E, Toschke AM, Kolip P. An effective lifestyle intervention in overweight children: findings from a randomized controlled trial on “Obeldicks light”. *Clinical Nutrition*, 2009;29:331–6.
 57. Rocchini AP, Katch V, Anderson J, Hinderliter J, Becque D, Martin M. Blood pressure in obese adolescents: effect of weight loss. *Pediatrics*. 1988;82:16–23.
 58. Ebbeling CB, Feldman HA, Chomitz VR, Antonelli TA, Gortmaker SL, Osganian SK. A randomized trial of sugar-sweetened beverages and adolescent body weight. *National England Journal of Medicine*. 2012;367:1407–16.
 59. de Ruyter JC, Olthof MR, Seidell JC, Katan MB. A trial of sugar-free or sugar-sweetened beverages and body weight in children. *National England Journal of Medicine*. 2012; 367:1397–406.
 60. Sharma AM, Pischon T, Engeli S, Scholze J. Choice of drug treatment for obesity-related hypertension: where is the evidence? *Journal of Hypertension*. 2001; 19:667–74.
 61. Earhart S. Obesity – The link between your weight and your family; 2010. Available:<https://www.obesityaction.org/resources/obesity-the-link-between-your-weight-and-your-family/>. (Accessed: 18October 2023).
 62. Alfredo MJ, Enriquez L, Moreno-Aliaga MJ, Marti A. Genetics of obesity. *Public Health Nutrition*. 2009;12(1):136.
 63. Corica D, Aversa T, Valenzise M, Messina MF, Alibrandi A, De Luca F, Wasniewska M. Does family history of obesity, cardiovascular, and metabolic diseases influence onset and severity of childhood obesity? *Frontal Endocrinology*. 2018; 9:187.
 64. Shariq OA, McKenzie TJ. Obesity-related hypertension: a review of pathophysiology, management, and the role of metabolic surgery. *Glandular Surgery*.2020;9(1):80–93.

© 2024 Nelson et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
<https://www.sdiarticle5.com/review-history/111803>