



The Prevalence of Obesity in Children

Anayitullah Kawa^{1*}, Dr. Pavan Kumar Talupuru²

1. Research Scholar, Sunrise University, Alwar, Rajasthan, India
atkawa1979@gmail.com,

2. Professor, Physiotherapy Department, Sunrise University, Alwar, Rajasthan, India

Abstract: Over the last several decades, juvenile obesity has become a far more common problem worldwide. Since childhood obesity accounts for the bulk of obesity in adults, paediatricians should be concerned about it and should implement treatments during this time. Obesity has a number of negative effects on children, including insulin resistance type 2 diabetes, cholesterol, polycystic ovary syndrome, pulmonary and orthopaedic illnesses, and psychological issues. It is also linked to higher morbidity and mortality in adulthood. Obesity is a condition that is influenced by both hereditary and environmental factors. Since there are few effective treatments for this condition, obesity prevention is essential. To safeguard youngsters, food control and exercise must be promoted, encouraged, and given top priority.

Keywords: Physical activity, physical fitness, obesity, overweight, children

----- X -----

INTRODUCTION

Every person aspires to have healthy kids who will live long lives and become healthy adults. The cornerstone for a healthy adult life is a good childhood. Childhood habits have a long-term effect on one's health and happiness. To foster healthy behaviours for optimum health, parents, families, communities, and society all have a duty.¹ Due to the fact that many unique adult health issues have their roots in childhood, it is obvious that in many nations this goal may not be realised for a number of years. "Childhood Obesity" is one of these health issues. Every mother desires to have plump children. As a result, obesity is now the most prevalent and significant nutritional disease among children. Many moms think that children will eventually shed the puppy fat they had as young children. The risk of adult obesity, however, seems to be higher among children who are fat before the age of five.²⁻⁵

Regardless of age, sex, or ethnicity, childhood obesity is a global epidemic that transcends geographical and economic boundaries. It also affects all socioeconomic levels. It has become an epidemic not just in affluent nations but also in emerging nations like India that are going through a fast epidemiological transformation.⁶

Obesity has recently emerged as one of the most prevalent nutritional illnesses in the world, approximating a pandemic and also being dubbed the disease of the 21st century that threatens the profitability of providing basic healthcare.⁷ Globally, the incidence of childhood obesity is rising quickly. In the last 30 years, childhood obesity has increased by a factor of three. Currently, there is an epidemic-like rise in the prevalence of obesity and overweight children and adolescents that has affected, globally, 20–25 percent of children and 45–50 percent of teenagers⁹. Previously thought to be the curse of wealthy western nations, it has already reached pandemic proportions in China and India as well. India and other developing nations

find themselves in the odd circumstance of having to cope with both extremes of the nutritional problem continuum. On the one hand, the pandemic of malnutrition has long been popular.⁸

It has been shown that the foetal stage and the first few months of life are crucial for the child's future development. It has been shown that nutrition is significant as a tendency for lower and accelerated deposition of fats leading to obesity throughout these early phases of development, which include the impacts of nursing and weaning. The export of the western way of life, together with inappropriate diets and low levels of physical activity, seem to pose a serious threat to the population growth that many nations would want to see. Fast food and sugar-laden soft drinks were discovered to be important.⁹ Effective intervention on the suggested idea of food and physical activity inside the family, school, and community setting from a young age was shown to be important in Europe, Asia, and the Pacific nations where sedentary lifestyle was on the rise.

The teenage years are a key time for development. Physical changes include development, the beginning of females' menarche, a rise in muscle and fat mass, and higher-than-average nutritional requirements all occur during this time. The key factor believed to be driving the pandemic is urbanisation, which has a tremendous influence on socioeconomic level, lifestyle, and the globalisation of food markets. Additionally, societal elements including shifting dietary customs and attitudes toward food also have a role in the prevalence and occurrence of obesity. Obese or very overweight people run a number of risks.¹⁰

There is now strong evidence that fat is a multi-factorial illness, with genetic, demographic, and lifestyle factors all contributing. Age, race, and gender are inborn characteristics that can't be changed, and neither can family history of obesity. Lifestyle factors that contribute to obesity are typically reversible.¹¹ In fact, prior studies have indicated that bad eating habits and inactivity, as well as sedentary behaviour, are all contributing factors to juvenile obesity. Studies have shown that various lifestyle variables, such as soda intake or computer usage, are linked to childhood obesity. Adolescents' BMI was shown to be linked to bad eating habits and inactivity, according to data on lifestyle variables connected with obesity.¹²⁻¹⁵

Weight gain and obesity are both caused by an imbalance in calorie intake and calorie expenditure. Because of sedentary jobs, changing transportation modes, and rising urbanisation, there has been a worldwide rise of energy-dense meals that are rich in fat and an increase in physical inactivity.

Overweight and obese children are typically the consequence of environmental and sociological shifts, as well as the absence of supporting policies in areas such as health, agriculture, transportation, urban planning, environment, food processing/distribution/marketing/education. Marketing and the price of food and drink may have an especially negative impact on children. Obesity in children is clearly linked to the marketing of harmful foods and beverages to youngsters. Energy-rich, nutrition deficient meals are also readily available at a cheap cost.¹⁶

Infants who are exclusively breastfed during the first six months of life are less likely to become overweight or obese. Continuous sleep deprivation makes the brain more sensitive to food odours, according to a research by the Cognitive Neuroscience Society (2017). As a result, they're more prone to nibble on junk food.¹⁷⁻¹⁸

According to Dr.Choubey, 60 per cent of instances of obesity are caused by bad dietary habits, and cases in the 11-18-year-old age bracket have climbed by 15 to 20 percent in recent years

According to a recent study by Professor Dolton in the United Kingdom, 35-40 percent of the world's youngsters are obese because of their parents.¹⁹

Childhood obesity is linked to a variety of family behaviours that have changed considerably over the last several decades, including the decline in breastfeeding and the rise in the use of newborn formula. As technological items such as televisions and video games keep kids cooped up indoors, less of them go outdoors to play. School-age children are being driven to school by their parents rather of walking or bicycling to a bus stop or straight to school. Children's pester power, their capacity to persuade adults to do what they want, rises as family size decreases. Because of this, they may more easily get their hands on high-calorie treats like candy and soda. Childhood obesity rates are influenced by the social setting of family mealtimes.²⁰⁻²¹

Childhood obesity has been exacerbated in recent decades as a result of quick and dramatic social change. For both adults and children, eating and physical activity habits are significantly impacted by the social and physical settings in which they occur. Obesity has been linked to urbanization-related eating habits such as frequenting fast-food restaurants and consuming disproportionately large servings both at home and at restaurants.²²⁻²⁴

MATERIAL AND METHODS

Adolescents with Obesity and overweight were found to be 8.88% and 15.1% respectively. The difference between the two percentages was around 5 percent, with a 95 percent confidence range for predicting the difference between two proportions.²⁵

$$n = \left[\frac{Z^2_{1-\alpha/2} (P_1*(1-P_1) + P_2* (1-P_2))}{d^2} \right] = \left[\frac{(1.96)^2 (0.888*(1-0.888)+0.151*(1-0.151))}{(0.05)^2} \right] = 350$$

Each group must have a minimum sample size of 350 people.

The participants in the research were adolescents between the ages of 13 and 16 who were in the 7th and 11th grades of school. The primary goal of this research is to examine the impact of fitness on adolescents who are fat or overweight. Adolescence is a time of rapid physical and mental development, making it an ideal time to do research on adolescents. As the pubertal phase approaches, the fat mass begins to accumulate and the elements influencing body composition, such as social, cultural, physical, and environmental, are critical. Any weight gain at this stage in life is associated with a slew of potential health issues in the future. The greatest way to ensure that children grow up fit and healthy is to target their fitness and avoid obesity among this specific demographic.²⁶

RESULTS

Choosing a sample size: There was a 5% risk difference and a 95% confidence range for predicting the difference between the two proportions based on earlier research that found obesity to be 8.88% and

overweight to be 15.1 percent among teenagers.²⁷

$$n = [Z^2_{1-\alpha/2} (P_1*(1-P_1) + P_2*(1-P_2))]/d^2 = [(1.96)^2 (0.888*(1-0.888)+0.151*(1-0.151))/(0.05)^2] = 350$$

The total minimum sample size for each group = 350.

Data was examined using Microsoft Excel and the Graph Pad Prism version 5.0 statistical programme. For continuous normal data, Mean SD was used, while for continuous non-normal data, Median IQR was used. One-way analysis of variance, Bonferroni's multiple comparison test, Kruskal Walli's multiple comparison test, and Dunn's multiple comparison test were used to compare three sets of data. Unpaired t-tests were used to compare continuous normal data between two groups, whereas Mann Whitney U tests were used to compare continuous non-normal data between two groups. This study was deemed statistically significant because of its low threshold for significance (p0.05).²⁸

Table 1: Compares the age (years) of girls and boys in the obesity, overweight, and normal weight categories.

Gender	Groups	N	Range	Mean	SD	P-Value
Boys	Obese	220	13 to 16	14.48	1.01	0.005
	Overweight	184	13 to 16	14.49	1.00	
	Normal	208	13 to 16	14.21	0.83	
Girls	Obese	130	13 to 16	14.48	0.95	0.002
	Overweight	166	13 to 16	14.20	0.91	
	Normal	142	13 to 16	14.10	0.86	

Study of the data:The exam by Kruskal Walli.

Conclusion: Obese, overweight, and normally weighted people all had different ages in the study.²⁹

Table 2: There are several comparisons among obese, overweight and normal groups in relation to age (years) for both girls and boys

Gender	Multiple Comparisons	Mean Difference	Significant? 0.05?	P <	Summary
--------	----------------------	-----------------	--------------------	-----	---------

Boys	Obese vs Overweight	0.01	No	NS
	Obese vs Normal	0.27	Yes	*
	Overweight vs Normal	0.28	Yes	*
Girls	Obese vs Overweight	0.27	Yes	*
	Obese vs Normal	0.38	Yes	**
	Overweight vs Normal	0.11	No	NS

An analysis of data: The multiple comparisons test developed by Dunn and Bonferrini

Conclusion: For the variable age (years), there was a substantial difference between the obese and the overweight, the obese and the normal, the overweight and the normal.³⁰

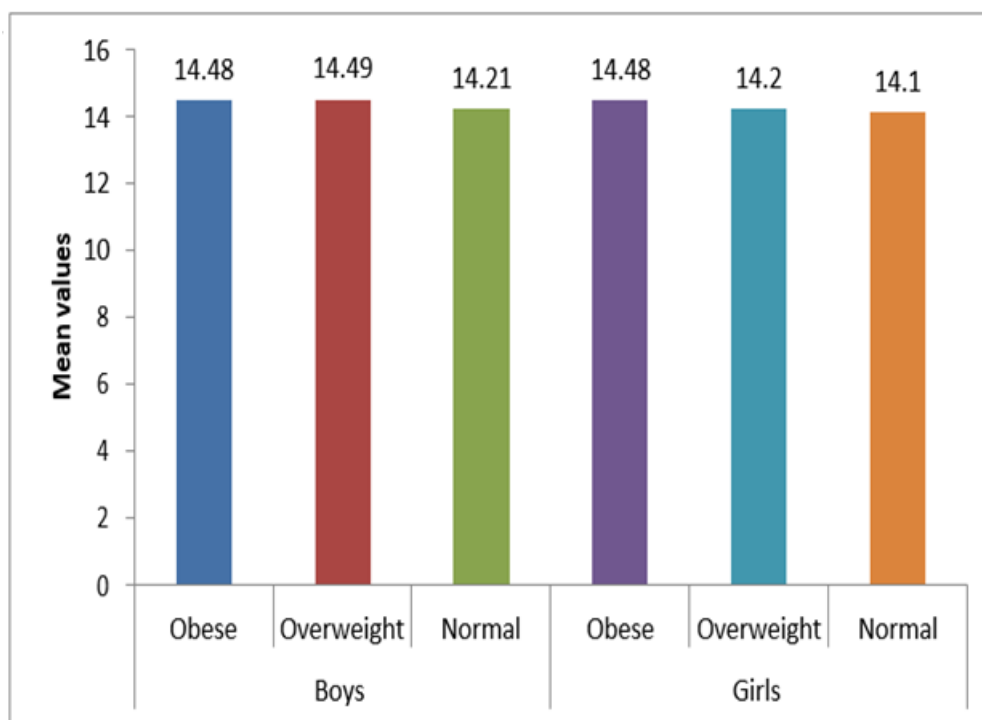


Figure 1: The simple mean bar graph for the comparison of obesity, overweight, and normal subgroups for the variable age (years) in children and adolescents

Age differences between obese children and normal-weight children are seen in this graph. Obese males had a mean value of 14.48 and a standard deviation of 1.01. For males, the average value was 14.49 and the standard deviation was 1.00. The average weight of males was 14.21 pounds, with an SD of 0.83 pounds. Among obese and OW boys, the mean difference was 0.01, whereas the difference between obese & normal boys was 0.27, and the difference between OW boys & normal boys was 0.28. There was a P-value of 0.005, which is less than 0.05. In this way, it is clear that the groups differed significantly.³¹

Obese females had a mean BMI of 14.48 and an SD of 0.95. Overweight (OW) girls had a mean value of 14.28 and a standard deviation of 0.91. Girls with normal weight had a mean of 14.10 pounds and an SD of 0.86 pounds. There was a mean difference of 0.27 between obese and OW, 0.38 between obese and normal and 0.11 between obese and normal females. The p-value was 0.02 that was less than the 0.05 cutoff point. This demonstrates that the groups of females differed significantly.

CONCLUSION

Children's obesity and serious co-morbidities have epidemic proportions. Since the 1980s, a number of hereditary and environmental variables have come together, increasing the incidence of obesity. The involvement of leptin in controlling energy intake and expenditure, as well as hyperinsulinemia and its side effects, has been recognised as a part of the pathophysiological changes associated with obesity. It is now recognised that several additional Exicans and anorexics have an impact on the control of food intake. However, prevention is essential since there are few effective treatments for this illness. Pediatricians and medical professionals should keep a tight eye out for any signs of rapid weight gain in proportion to linear development. To safeguard youngsters, food control & increased physical activity should be encouraged, promoted, & given top priority. Dietary strategies should promote moderation and diversity with the aim of establishing healthy eating patterns for life. To get insurance to cover the sickness, advocacy is required.³²

References

1. Alemzadeh R, Rising R, Lifshitz F. Obesity in Children. In Pediatric Endocrinology 5th edition Vol. 1, Chapter 1 ed. F. Lifshitz. Informa NY. 2007.
2. Allison DB, Casey DE. Antipsychotic-induced weight gain: a review of the literature. J Clin Psychiatry 2001; 62(suppl 7):22–31.
3. American Obesity Association. AOAFact Sheets Obesity in the US. 2004; www.obesity.org/subs/fastfacts/obesity_youth.shtml.
4. Betts P, Mulligan J, Ward P, Smith B, Wilkin T. Increasing Body Weight predicts the earlier onset of insulin dependant diabetes in childhood: testing the 'accelerator hypothesis' Diabet Med 2004;22:144-151.
5. Branson R, Potoczna N, Kral JG, Lentes K-U, Hoehe MR, Horber FF. Binge Eating as a Major Phenotype of Melanocortin 4 Receptor Gene Mutations. New Eng J Med 2003;348:1096-1103.
6. Controlling the Global Obesity Epidemic. World Health Organization Obesity and Overweight Fact

Sheet. 2003.

7. Decaluwe V, Braet C. Prevalence of binge-eating disorder in obese children and adolescents seeking weight-loss treatment. *Int J Obes Relat Metab Disord* 2003;27: 404–409.
8. Farooqi IS, Keogh JM, Yeo GSH, Lank EJ, Cheetham T, O’Rahilly S. Clinical Spectrum of Obesity and Mutations in the Melanocortin 4 Receptor Gene. *New Eng J Med* 2003;348:1085-1095.
9. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA* 2005;293:1861-1867.
10. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. *JAMA* 2003;289:187–193.
11. Ford ES. The epidemiology of obesity and asthma. *J Allergy Clin Immunol* 2005;115: 897–909.
12. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. *Pediatrics* 2005;115: 22-27.
13. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. *JAMA* 2004;291:2847-2850.
14. Janssen I, Katzmarzyk PT, Srinivasan SR, Chen W, Malina RM, Bouchard C, Berenson GS. Combined influence of body mass index and waist circumference on coronary artery disease risk factors among children and adolescents. *Pediatrics* 2005;115:1623-1630.
15. List JF, Habener JF. Defective Melanocortin 4 Receptors in Hyperphagia and Morbid Obesity. *N Eng J Med* 2003;348:1160-1163.
16. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA* 2004;291: 1238–1245.
17. Nead KG, Halterman JS, Kaczorowski JM, Auinger P, Weitzman M. Overweight children and adolescents: a risk group for iron deficiency. *Pediatrics* 2004;114:104–108.
18. Neumark-Sztainer D, Hannan P. Weight-related behaviors among adolescent girls and boys: results from a national survey. *Arch Pediatr Adolesc Med* 2000;154: 569–577.
19. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, Hayflick L, Butler RN, Allison DB, Ludwig DS. A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med* 2005;352:1138-1145.
20. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* 2003; 138:24–32.
21. Rankinen T, Zuberi A, Chagnon YC, Weisnagel SJ, Argyropoulos G, Walts B, Périusse L, Bouchard C. The human obesity gene map: the 2005 update. *Obesity* 2006;14:529-644.

22. Reaven GM. Role of insulin resistance in human disease. *Diabetes* 1988; 37: 1595–1607.
23. Schaffler A, Schölmerich J, Büchler C. Mechanisms of disease: adipocytokines and visceral adipose tissue—emerging role in nonalcoholic fatty liver disease. *Nat Clin Pract Gastroenterol Hepatol* 2005;2:273-280.
24. Shaw GM, Velie EM, Schaffer D. Risk of neural tube defect-affected pregnancies among obese women. *JAMA* 1996;275: 1093–1096.
25. Strollo PJ Jr., Rogers RM. Obstructive sleep apnea. *N Engl J Med* 1996; 334:99–104.
26. Stunkard AJ, Harris JR, Pedersen NL, McClearn GE. The body-mass index of twins who have been reared apart. *N Engl J Med* 1990; 322:1483–1487.
27. Timm NL, Grupp-Phelan J, Ho ML. Chronic ankle morbidity in obese children following an acute ankle injury. *Arch Pediatr Adolesc Med* 2005;159:33-36.
28. Valle M, Martos R, Gascon F, Canete R, Zafra MA, Morales R. Low-grade systemic inflammation, hypoadiponectinemia and a high concentration of leptin are present in very young obese children, and correlate with metabolic syndrome. *Diabetes Metab* 2005;31:55-62.
29. Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *Am J Clin Nutr* 2005;81:555-563.
30. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, Allen K, Lopes M, Savoye M, Morrison J, Sherwin RS, Caprio S. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* 2004;350:2362-2374.
31. Williams J, Wake M, Hesketh K, Maher E, Waters E. Health-related quality of life of overweight and obese children. *JAMA* 2005;293:70-76.
32. Yajnik CS, Yudkin JS. The Y–Y paradox. *Lancet* 2004; 363:163.