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# ADIPOSITY AMONGST BOYS AND GIRLS OF MINGORA CITY, SWAT

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## ABSTRACT

*Background: The dramatic increase in the prevalence of overweight and obesity and its resultant comorbidities are associated with significant health and financial burdens, warranting strong and comprehensive prevention efforts. The objective of the present study was to evaluate and compare the prevalence of obesity in the adolescent boys and girls in Mingora city, district Swat.*

*Methodology: Both boys and girls of age between 10 – 20 years. Two hundred subjects, 100 boys and 100 girls of age included in the study. Using BMI as obesity index, prevalence of obesity in boys was compared with that in girls. The antoropometric and cardiovascular parameters of obese group were also compared with control group.*

*Results: The prevalence of obesity in adolescent boys was 6.7% and 8.35% in adolescent girls. Obese subjects had significantly higher weight, body mass index, and blood pressure values, compared to control group.*

*Conclusion: prevalence of obesity was higher in female subjects.*

*Key words: Weight (Wt), Height (Ht), Body mass index (BMI), Obesity, Blood pressure (B.P).*

## INTRODUCTION

Obesity is defined as a BMI greater than or equals to the 95th percentile for age and gender, overweight as a BMI greater than or equal to the 85th percentile but less than the 95th percentile, and normal weight as BMI less than the 85th percentile.[1] BMI is recognized as one of the most useful indices for adiposity both in children and adults and is highly correlated with weight (0.8 – 0.9) and body fat (0.7 – 0.8). Based on data for children 6 – 15 years, World Health Organization (WHO) has suggested the following cut – off points for BMI as broad guidelines for defining obesity in both sexes.[2] Age ≤ 14 years BMI 19 – 20 (kg/m$^2$), age 15 years BMI 25 (kg/m$^2$), age ≥ 16 years BMI 28 (kg/m$^2$). While Sardinha et al recommends optimum Body Mass Index threshold values to define obesity as follows.[3] Age 10 – 11: Boys 19.0, and Girls 19.6. Age 12 – 13: Boys 19.4, and Girls 21.2, Age 14 – 15: Boys 24.0, and Girls 21.9.

In normal individuals, the percentage of body tissue that is adipose tissue varies by gender (greater in post pubertal females than males) and age (about 12% at birth, increasing to 25% at 5 months, then decreasing to 15% to 18% during puberty).[4]

The changes in body composition that occur during adolescence have been well described. In boys, fat – free mass tends to increase, body fat as a percentage of body weight decreases, and fat tends to be deposited abdominally. In girls, both fat and fat–free mass increase, fat – free mass as a percentage of body weight decreases, and fat tends to be deposited in the buttocks. In both sexes, factors that contribute to the quantity of body fat and abdominal fat distribution appear to increase the risk of subsequent complications.[5]

Only a small percentage of childhood obesity is associated with a hormonal or genetic defect, with the remainder being idiopathic in nature. An endogenous cause for obesity can be either suspected or eliminated from the differential diagnosis in virtually all children based on a careful history and physical examination. In most cases, this should negate the need for expensive and unnecessary laboratory evaluations.[7]

Growth failure characterizes endogenous obesity. Children with an associated genetic or hormonal syndrome are short, usually at or under the 5th percenttile of height for age. Conversely, children with idiopathic obesity are taller, usually above the 50th percentile. Even the onset of a hormonal abnormality in a previously tall child will be marked by a significantly slower rate of growth compared with the child's previous growth curve.[7]

Parental obesity is the most important risk factor for childhood obesity. Twin, adoption, and family studies indicated that inheritance is able to account for 25% to 40% of inter-individual difference in adiposity.[8]

It is possible that the functional organization of the hypothalamic or other neurogenic feeding centers in an obese person is different from that of a non-obese person. Also, there may be abnormalities of neurotransmitters or receptor mechanism in the neural pathways of the hypothalamus that control feeding.[9]

The hormone, Leptin is made almost exclusively in adipose tissue. Low plasma levels of leptin and insulin (e.g., during fasting and weight loss) increase food intake and decrease energy expenditure by stimulating neuropeptide Y (NPY) synthesis, and perhaps by inhibiting sympathetic activity and other catabolic pathways. High leptin and insulin concentrations (e.g., during feeding and weight gain) decrease food intake and increase energy expenditure through release of melanocortin and corticotrophin-releasing hormone. The absence of a functional hormone (or its receptor) leads to uncontrolled food intake and resulting obesity.[10]

Widespread reports indicate that the prevalence of obesity among children and adolescents has been increasing in recent years, just as it has in adults. A recent report of the initial results of 1999 National Health and Nutrition Examination Survey indicates that prevalence rates have increased even further, to 13% of children aged 6 to 11 years and 14% of adolescents aged 12 to 19 years.[11]

Obese children under three years of age without obese patients are at low risk for obesity in adulthood, but among older children, obesity is an increasingly important predictor of adult obesity, regardless of whether the parents are obese. Parental obesity increases the risk of adult obesity among both obese and non-obese children less than 10 years of age.[12]

## SUBJECTS AND METHODS

Two hundred subjects (100 boys and 100 girls) were randomly selected to detect the prevalence of obesity. Hundred subjects, 50 control (non-obese) and 50 obese (age and sex matched) were selected for the comparison of anthropometric and cardiovascular parameters of the two groups. The body mass index and prevalence of obesity in boys were also compared with that in girls.

Boys and girls between ages 10 – 20 years were included in the study.

Subjects were excluded from study participation that had a medical history of disease other than overweight / hypertension or were taking any medication known to affect metabolism.

Health Scale was used to measure weight and height. Weight nearest to 0.5 kg was assessed at 2 different points during interview, and the 2 were averaged for these analyses. Participants were advised to wear normal clothing without shoes, socks, and belts. Height nearest to 0.1 cm was also assessed at 2 different points during interview, and the two readings were averaged for these analyses.

Body mass index (BMI) was determined by dividing weight (wt) in kilogram by height (ht) in meters squared (BMI = kg/m$^2$). Body mass index percentile charts were used to determine obesity in boys and girls.[14]

Blood pressure data was obtained, after at least 5 minutes of rest, with subjects in seated position. A mercury sphygmomanometer, with an appropriate sized cuff covering two third of the upper arm, was used. The onset of the first tapping sound was taken to indicate the systolic blood pressure, while the point of complete disappearance of the sound (Korotkoff V) was taken to indicate diastolic blood pressure. The mean of three reading was recorded.[15] Age and sex specific percentiles of blood pressure measurements charts were used.[16]

## RESULTS

*Table 1, Graph 1:* Show anthroprometric parameters and the prevalence of obesity in boys. Mean age (10 – 15 years) 12.5 ± 1.74: weight, 39.77 ± 12.48, height, 1.45 ± 0.11, BMI, 18.64 ± 3.68, and the prevalence was 6.67%. Mean age (16 – 20 years) 18 ± 1.44, weight, 60.54 ± 9.90, height, 1.71 ± 0.033, BMI, 20.71 ± 3.56 and the prevalence was 6.67%. As shown, the prevalence remained the same from age 10 through 20. The values were expressed as mean ± SD (standard deviation).

*Table 2, Graph 2:* Show anthropometric parameters and the prevalence of obesity in adolescent girls. Mean age (10 – 15 years) 12.5 ± 1.74: weight, 40.57 ± 11.21, height, 1.48 ± 0.78, BMI, 18.29 ± 3.85, and the prevalence was 6.67%. Mean age (16 – 20 years) 18 ± 1.44, weight, 53.17 ± 11.81, height, 1.57 ± 0.027, BMI, 21.57 ± 4.40 and the prevalence was 10%. The values were expressed as mean ± SD (standard deviation).

As shown, the prevalence of obesity in children and adolescents was the same for both sexes till

**Table 1:** *Anthropometric parameters and prevalence of obesity in boys.*

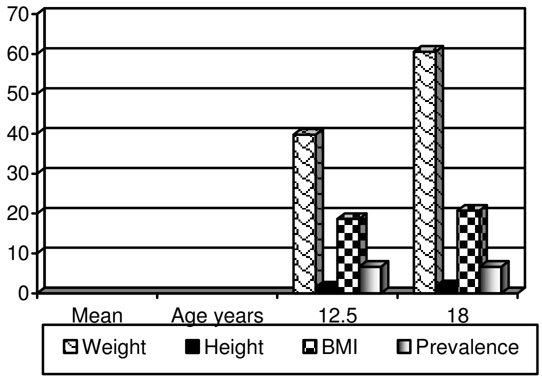
| *Age group years* | *Age (mean)* | *Weight (kg)* | *Height (m)* | *BMI (kg/m$^2$)* | *Prevalence (6.67%)* |
|---|---|---|---|---|---|
| 10 – 15 (n = 50) | 12.5 ± 1.74 | 39.77 ± 12.48 | 1.45 ± 0.11 | 18.64 ± 3.68 | 6.67% |
| 16 – 20 (n = 50) | 18 ± 1.44 | 60.54 ± 9.90 | 1.71 ± 0.033 | 20.71 ± 3.56 | 6.67% |

the age of 15 years. In the late adolescents, the prevalence increased with increase in age and was higher in female than male.

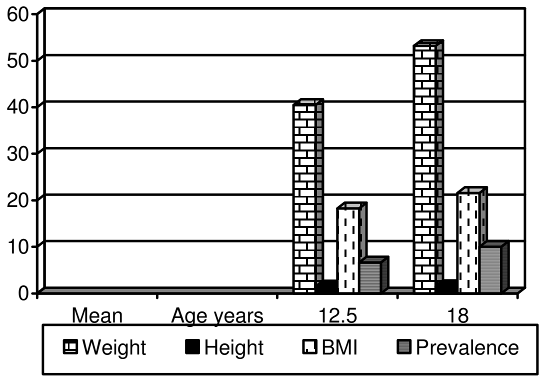
Table 3/ Graph 3 show body mass index and prevalence of obesity in

**Table 2:** *Anthropometric parameters and prevalence of obesity in girls.*

| *Age group years* | *Age (mean)* | *Weight (kg)* | *Height (m)* | *BMI (kg/m²)* | *Prevalence (8.35%)* |
|---|---|---|---|---|---|
| 10 – 15 (n = 50) | 12.5 ± 1.74 | 40.57 ± 11.21 | 1.48 ± 0.78 | 18.29 ± 3.85 | 6.67% |
| 16 – 20 (n = 50) | 18 ± 1.44 | 53.17 ± 11.81 | 1.57 ± 0.027 | 21.57 ± 4.40 | 10% |




**Graph 1:** *Anthropometric parameters and prevalence of obesity in boys.*

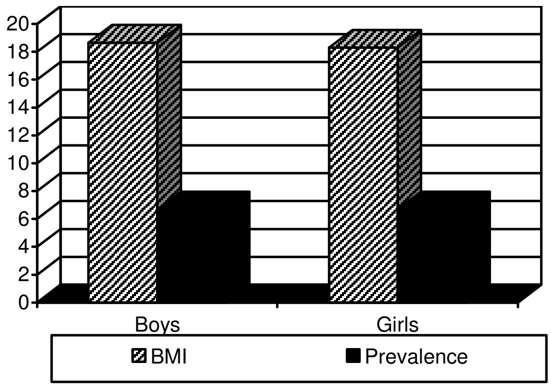



**Graph 2:** *Anthropometric parameters and prevalence of obesity in girls.*

**Table 3:** *Comparison of Body mass index and Prevalence of Obesity in Boys and Girls (Mean age 12.5 ± 1.74).*

| *Parameters* | *Boys* | *Girls* |
|---|---|---|
| BMI | 18.64 ± 3.68 | 18.29 ± 3.85 |
| Prevalence | 6.67% | 6.67% |

boys and girls. Mean age 12.5 ± 1.74, BMI of boys was 18.64 ± 3.68 and that of girls was 18.29 ± 3.85. The difference in BMI of two groups were statistically non significant. The prevalence of obesity of both group also remained the same.




**Graph 3:** *Comparison of Body mass index and Prevalence of Obesity in Boys and Girls (Mean age 12.5 ± 1.74).*

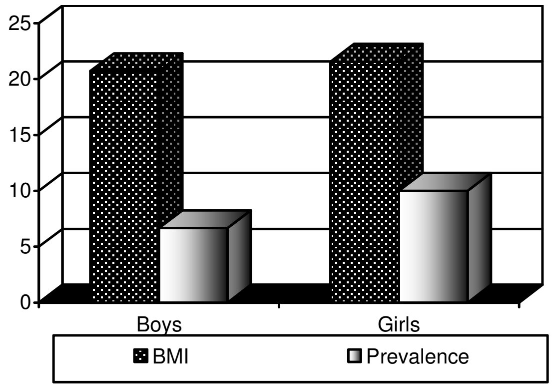
**Table 4:** *Comparison of Body mass index and Prevalence of Obesity in Boys and Girls (Mean age 18 ± 1.44).*

| *Parameters* | *Boys* | *Girls* |
|---|---|---|
| BMI | 20.71 ± 3.56 | 21.57 ± 4.40* |
| Prevalence | 6.67% | 10** |

Values were given as mean ± SD, *p ≤ 0.05 (significant), **p ≤ 0.001 (highly significant)

Table 4 / Graph 4 show body mass index and prevalence of obesity in boys and girls. Mean age 18 ± 1.44, BMI of boys was 20.71 ± 3.56 and that of girls was 21.57 ± 4.40. The difference was statistically significant. The prevalence of obesity in boys was 6.67% and that in girls was 10%. The difference was statistically highly significant.

*Table 5, Graph 5:* The anthropometric parameters such as weight, height, BMI, and both systolic and diastolic blood pressure of group "A" (control) were compared with group B (obese). The mean weight of group "A" was 40 ± 7.27 and that of group "B" was




**Graph 4:** *Comparison of Body mass index and Prevalence of Obesity in Boys and Girls (Mean age 18 ± 1.44).*

**Table 5:** *Comparison of anthropometric and cardiovascular parameters of obese group with control group.*

| *Parameters* | *Control* | *Obese* |
|---|---|---|
| Weight | 40 ± 7.27 | 62 ± 8.43** |
| Height | 1.45 ± 0.08 | 1.44 ± 0.07 |
| BMI | 19 ± 1.29 | 29.9 ± 1.41** |
| Systolic B.P mmHg | 90 ± 11.8 | 135 ± 8.3** |
| Diastolic B.P mmHg | 60 ± 10.9 | 85 ± 6.8** |

Values were given as mean ± SD, *p ≤ 0.05 (significant), **p ≤ 0.001 (highly significant)
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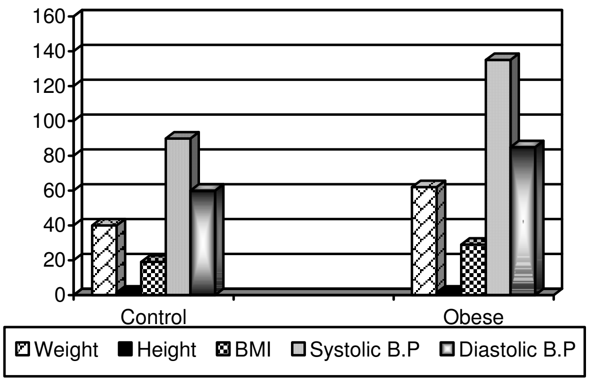


**Graph 5:** *Comparison of anthropometric and cardiovascular parameters of obese group with control group.*

62 ± 8.43. The p-value < 0.001 (highly significant). The mean height of group "A" was 1.45 ± 0.08 and that of group "B" was 1.44 ± 0.068. The difference in height of two groups were statistically non significant. The mean BMI of group "A" was 19 ± 1.29 and that of group "B" was 29.9 ± 1.41. The p-value was < 0.001 (highly significant).

Systolic and diastolic blood pressure of group "A" (control) were also compared with group B (obese). The mean systolic blood pressure of group "A" was 90 ± 11.8 and that of group "B" was 135 ± 8.3. The p-value was < 0.001. The mean diastolic blood pressure of group "A" was 60 ± 10.9 and that of group "B" was 85 ± 6.8. The p-value was < 0.001.

The values are expressed as mean ± SD (standard deviation).

## DISCUSSION

The variation in prevalence of obesity epidemic in various races and communities of the world may be attributed to heredity, age, sex, and diet, eating patterns, life style and / or behavior. Extensive literature exists that documents the relationship between blood pressure (BP) and measures of body fat and fat distribution.[17] Most studies report significant associations for men and women both within population and between populations. Recent studies suggest this relationship has early beginnings, perhaps from birth or may be prenatally.[18]

Obesity tracks from childhood to adulthood,[19] and is strongly related to hypertension in adults.[30] It is suggested that avoidance of obesity should be emphasized in any discussion of blood pressure control in children. At least 30% of obesity begins in childhood. Conversely 50 to 80% of obese children become obese adults.[21]

The present study showed that obesity was prevalent in both adolescent boys and girls in Mingora city of Swat district. The prevalence of obesity in children and adolescents was the same for both sexes till the age of 15 years. In the late adolescents, the prevalence increased with increase in age and still higher in female than male. Also a significant relationship of obesity with both systolic and diastolic blood pressure was found.

Two studies conducted in Chennai, Tamil Nadu, India in 1981 and 1998, showed almost similar results.[22] Ogden et al, also observed almost similar findings and reported that persistently elevated blood pressure levels occur about 9 times more frequently among obese children and adolescents (ages 5 to 18) than in non-obese. Obese children and adolescents are reported to be 2.4 times more likely to have high diastolic blood pressure and 4.5 times more likely to have high systolic blood pressure than their non-obese peers.[14]

Sheila et al, also observed higher rates than the present study and reported that prevalence of overweight amongst Australian children has increased from 11% in 1985 to 20% in 1995. According to WHO at least 50% of adults and 20% of children in U.K. and U.S.A. are currently overweight.[23]

Roberts et al,[24] reported that the United States has one of the highest obesity rates in the world and is the first nation to have people who are both impoverished and obese. The obesity epidemic in also increasing in Europe, Asia and throughout the Americas: as the Indian newspaper The Tribune states.

Obesity also plagues Middle Eastern countries, with 35 percent of Egyptians considered obese, a greater proportion than the population in the USA at 20 percent.[24] Evidence from the National Health and Nutrition Examination survey (NHANES) in the United States and the national study of health and growth in the United Kingdom shows an increasing prevalence of overweight and obesity in young children and adolescents.[25,26]

From the dindings of the study conducted by Danladi et al, it in evident that essential hypertension occurs even in adolescent children, though small, among the anthropometric variables, body weight and BMI tend to the more closely related to both systolic blood pressure and diastolic blood pressure.[27]

Seidell et al, concluded from a multicenter study of women that among anthropometric (AP) variables, BMI was the best overall predictor of both systolic and diastolic blood pressure.[28] The significant positive association between BMI and both systolic and diastolic blood pressure has been reported in studies of African – Americans,[29] Chinese,[30,31] Africans, and Caribbeans.[32] The studies conducted in China, Africa, and the Caribbean are especially noteworthy because significant relationships held even in these lean populations.

Studies of obesity in Asian subjects show that generalized obesity is the major determinant of cardiovascular risk in the Chinese and East Asian subjects while central obesity is associated with greater cardio-vascular risk in South Asians.[33]

It is *concluded* that the significant prevalence of childhood obesity and an associated complication, increased blood pressure, emerges in school – aged children. It is, therefore, suggested that early recognition of excessive weight gain relative to linear growth should become in pediatric ambulatory care settings. BMI should be calculated and plotted periodically and intervention should begin in the first years of school because obesity appears to be well established by then.

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