ORIGINAL ARTICLE

OBESITY INDICATORS AND CARDIOVASCULAR PARAMETERS IN OBESE AND NON-OBESE SUBJECTS

Rabia Siddiqui, Noorunnisa*, Muhammad Abdul Azeem, Nudrat Khan*, Muhammad Younus Jamal Siddiqui***

Department of Physiology, Bahria University Medical & Dental College, *Dewan Medical and Dental College, **United Medical & Dental College, Karachi

Background: Sedentary life style and faulty dietary habits result in obesity. Although, extensive studies are available on the effects of obesity on anthropometric (ANPP) and cardiovascular parameters (CVP) their statistical correlations between obese and non-obese persons are very few. This study was aimed to find the correlation between different ANPP and CVP with BMI and their correlation in obese and non-obese individuals. **Methods:** This cross-sectional data obtained from 30 non-obese and 30 obese individuals (age 21–60 year) was used to determine the Pearson's correlation of BMI with ANPP such as height, weight, Triceps Skin Girth (TSG), waist circumference (WC), and CVP such as pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP). **Results:** Significantly greater mean values were found for weight, BMI, TSG and WC in obese group except for height, in order of Height>Weight>WC>TSG for correlation with BMI. Significantly higher mean values found for SBP, DBP, MAP and PP in obese except for PR being non-significantly different between two groups. The CVP fell in order of PR>MAP>DBP>SBP> PP for correlation with BMI. This correlation was positive for all the parameters except for height (both obese and non-obese), TSG and PP (only non-obese) being negatively correlated with BMI. **Conclusion:** On the basis of the order determined for ANPP and CVP, the short height and higher PR have better correlation in obese group representing the best indicators of obesity, involving greater risk for cardiovascular diseases.

Keywords: Obesity, Anthropometric, Pearson's correlation, cardiovascular diseases

INTRODUCTION

Obesity is an abnormal accumulation of body fat, usually 20% or more over an individual's ideal body weight. In developed countries, sedentary life style and faulty dietary habits result in an imbalance between energy intake and energy expenditure leading to obesity. Obesity is a global epidemic becoming a threat to healthy populations in increasing number of countries.[1,2] The use of Body mass index (BMI) started to track the epidemic of obesity in the early years of 1980's[3] is still used. BMI of 25.9–29 is considered overweight, and ≥30 is considered obese. Measurements and comparisons of waist and hip circumference can also provide some information regarding risk factors associated with weight.

According to National Health and Nutritional Examination Survey (NHANES), from 1988–1999, the prevalence of obesity increased from 22.9% to 30.5%.[4] In Pakistan prevalence of obesity in 25–44 year olds in rural areas was 9% for men and 14% for women; in urban areas, prevalence was 22% and 37% for men and women, respectively. For 45–64 year old, prevalence was 11% for men and 19% for women in rural areas, and 23% and 40% in urban areas for men and women, respectively.[5] Studies have estimated that in 2007–2008 prevalence of obesity was 32.2% among adult men and 35.5% among adult women in United States.[6] The mechanism for excessive weight gain is clear that occurs due to a complex combination of factors, i.e., genetic factors, eating habits, patterns of physical activity being the most important one. Obesity can also be a side-effect of certain disorders and conditions, including: psychological factors, such as depression and low self-esteem emotional and social problems.

Over the years concern has been grown on the fact that increasing obesity among adults might affect their cardiovascular health. When a person accumulates adipose tissue a variety of changes occur in the cardio-respiratory structure and function.[7] Hence obesity may affect the heart and lungs through its influence on the known risk factors of hypertension, dyslipidemia and glucose intolerance. The cardiovascular disorders due to obesity result in increased mortality from coronary artery disease, heart failure, arrhythmias and sudden death.[8,9]

Few studies have been done to see the correlation of BMI with cardiovascular parameters[10] BMI with ANPP[11] and some have been done to see the correlation of anthropometric indices and CVP[12,13], but none have been done to see the correlation of ANPP and CVP together with BMI in both obese and non-obese for comparison. The aim of this study was to assess the correlation between different anthropometric and cardiovascular parameters with obesity indicators (BMI, WC, and TSG) and correlation of CVP and WC vs

TSG, to ascertain clinical conditions in obese groups by comparison with non-obese ones.

MATERIAL AND METHODS

This was a cross-sectional, observational study. Selected subjects (from the staff of Dewan Medical & Dental College, and general population) were divided into two groups. In Group-A, 30 non-obese individuals and in Group-B, 30 obese individuals were selected having the age range of 21–60 year.

Healthy Individuals with BMI 30 Kg/m^2 in the age group of 21–60 years were classified as obese. Healthy individuals with BMI of 18.50–24.99 Kg/m^2 in the age group of 21–60 years were included as controls in this study. Subjects with BMI between 25–29.9, subjects with medical problems especially cardio-respiratory illnesses and smokers were excluded from the study.

Ethics were carefully observed by taking the consent from all the individuals. A brief history including smoking history and a clinical examination of the cardiovascular system and respiratory system was done to exclude medical problems. Physical examination of all subjects included measuring height in meters, weight in Kg and body mass index was derived by Quetelet's index. Waist circumference (WC) was measured to the nearest Cm in the standing position at the end of gentle expiration. Triceps skin fold girth was measured using the Vernier Caliper, midway between the acromion and olecranon processes of scapula and ulna. Systolic and diastolic blood pressures were measured with mercury sphygmomanometer, and radial pulse rate/min was recorded. Other cardiac parameters like MAP.

SPSS-20 was used for data analysis which is presented as Mean±SE along with Student's *t*-test. Pearson's correlation coefficient was used to identify relationship between various parameters, and $p\leq 0.05$ was considered statistically significant.

RESULTS

A total of 60 subjects were included in the study. Their mean height was significantly lesser in obese group than non-obese, however, the mean weight and BMI were significantly greater ($p<0.0005$) in obese group. Mean values of WC and TSG were found significantly ($p<0.0005$) greater in obese group. There were significantly higher mean values of SBP, DBP, MAP ($p<0.0005$) and PP ($p<0.05$) in obese group. However, the mean values of pulse rate showed not significant ($p>0.05$) difference between two groups, being higher in obese (Table-1).

Correlations of various ANPP and CVP, with BMI between obese and non-obese groups have been presented in Figure-1A a–j and Figure-1B i–x.

The correlations for all the CVP (obese person only), are found to be positive and significant with BMI at the level of 0.05 except that the correlation of PR was significant at 0.01 (Figure-1B i–x).

Statistical comparison of WC with CVP had positive and significant correlation for all parameters for obese and non-obese except with SBP and MAP in non-obese, where it was non-significant (Table-2).

Our results regarding Pearson correlation obtained between waist circumference and triceps girth for obese & non-obese groups demonstrated positive and significant correlation at the level of 0.01.

Correlation of pulse rate with SBP, DBP and MAP was found to be positive and significant with r=0.5, in obese group. However, in non-obese group it was negative and non-significant (-0.1 for SBP, -0.2 for DBP and -0.2 for MAP).

Table-1: Comparison of anthropometric and cardiovascular parameters between obese and non-obese groups

Parameters	Obese n=30	Non-obese n=30
ANTHROPOMETRIC		
Age (years)	38.15±1.50	
Height (Cm)	154.22±0.06	166.72±0.04*
Weight (Kg)	92.67±1.99	61.23±0.89*
BMI	40.87±1.48	22.81±0.34*
Waist Circumference (Cm)	103.22±1.56	83.58±1.16*
Triceps Skin Girth (Cm)	3.19±0.12	1.92±0.05*
CARDIOVASCULAR		
Pulse Rate/min	78.33±0.87	75.33±1.30**
Systolic BP (mmHg)	128.13±1.92	113.03±2.12*
Diastolic BP (mmHg)	85.03±1.34	75.90±1.46*
Mean Arterial Pressure (mmHg)	99.50±1.46	88.37±1.55*
Pulse Pressure (mmHg)	43.13±1.13	38.57±1.30***

*$p<0.0005$; **$p>0.05$; ***$p<0.05$

Table-2: Correlation between WC and CVP

WC vs CVP	Obese (n=30)	Non-obese (n=30)
Systolic	0.433*	0.176
Diastolic	0.520**	0.386*
Mean Arterial Pressure	0.503**	0.317

*Correlation is significant at the level 0.05 (2-tailed); **Correlation is significant at the level 0.01 (2-tailed)

Table-3: Correlation between cardio-vascular parameters in obese and non-obese

	Obese (n=30)	Non-Obese (n=30)
Pulse Rate		
Systolic	0.573**	-0.123
Diastolic	0.512**	-0.191
Mean Arterial Pressure	0.576**	-0.177
Systolic Blood Pressure		
Diastolic	0.797**	0.695**
Mean Arterial Pressure	0.931**	0.891**
Pulse Pressure	0.710**	0.403*

*Correlation is significant at the 0.05 level (2-tailed);
**Correlation is significant at the 0.01 level (2-tailed)

A
ANTHROPOMETRIC

B
CARDIOVASCULAR

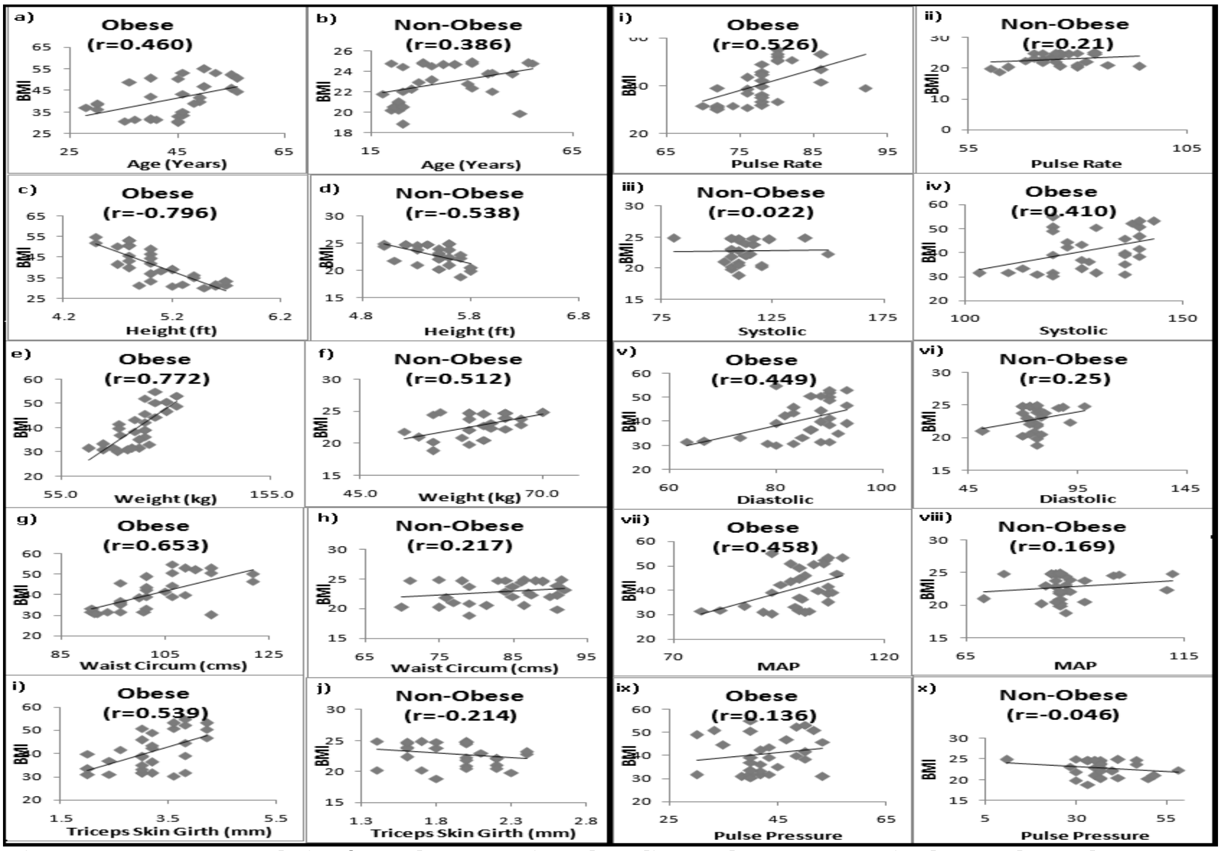

Figure-1: Pearson's correlation for anthropometric and cardiovascular parameters in obese and non-obese groups

DISCUSSION

Studies[11,14] have been published for individual indicators of obesity, without including all CVP and ANPP parameters. Further, specific differentiation of correlation between obesity indicators and CVP and CVP themselves are not reported.

Significantly lesser height in obese than non-obese group in our study indicates that short height is one of obesity indicators. However, it should meet the cut-off values of BMI, above normal as standardised by American National Heart Lung Association (ANHLA)[15]. The importance of shortness of body height in obesity is also proven in another study[16] demonstrating strong association of short stature individuals with high BMI. They also found strong association between obesity and short stature even after the adjustment of diet, physical activity, and many environmental factors.[16] The prevalence of short stature was also reported 19.6% and 15.4% in men and women, respectively by indicating it an independent risk factor for overweight and high Waist Hip Ratio (WHR) in women.[17] A study conducted in Egypt[18] has also supported this hypothesis. Our finding of significantly lesser height in obese persons is in conformity with earlier reports.

Mean weight in our study was expectedly higher with BMI and found significantly (p<0.0005) greater in obese. The mean values of BMI matches with earlier reports[19]. Persons having short height and cut-off values of body weight above over-weight are the main anthropometric characters of obesity, determined in our study.

Commonly in clinical settings, studies on obesity also include measurements relevant to body fatness but, their associations are poorly known and procedures are needed to facilitate the interpretation of these measurements regarding indicators, WC and TSG.[20] In our study, both of these are found significantly greater in obese group (irrespective of gender); our study has indications similar to ANHLA[15] measurements, regarding WC for obesity in particular and TSG in general. However, other studies regarding correlations between selected ANPP measurements and estimates of percent and total body fat are also available.[20,21] In one of these studies[20] comparisons among these correlations, for children and adults of each sex, lead to conclusions that the TSG was the best single indicator of percent body fat in children and women. Our results demonstrate the mean values of TSG, irrespective of gender, within the earlier reported range.

Obesity has its profound effect on cardiovascular parameters.[11,22] Our results showing significantly higher values of all the cardiovascular parameters in obese group with the exception of non-significantly higher PR when compared with non-obese group confirm these findings. These results represent a probable activation of sympathetic nervous system with concomitant deactivation of parasympathetic function in obese group[14] responsible to elevate blood pressure. In another study, percent body fat had been related directly to the pulse rate.[23] In the present study pulse rate was non-significantly higher, indicating an unexpected finding which cannot be explained on the basis of established obesity related cardiovascular changes.

A study[11] showed correlation between ANPP and CVP with obesity indicators like BMI, WC and TSG. However, they did not identify the best indicator that brings change in most of the CVP, by comparing between obese and non-obese. There is no study to-date that identified the best ANPP on the basis of Pearson's test that can reflect obesity, by comparing between obese and non-obese.

A direct relation has been found between BMI and age, which was comparatively poor in non-obese group, indicating greater rise of BMI with age in obese than non-obese group. Similarly, positive correlation has been reported in children >7 years of age and onward.[21] It is to be noted that non-obese with poor '*r*-value' are also having positive and direct relation between BMI and age. If degree of obesity is to be correctly determined with age, a correction factor should be calculated with respect to increase in BMI with the age of normal or non-obese persons and to be subtracted from BMI values of obese persons.

Both weight and height were also correlated with BMI to assess which of these two parameters is more associated with obesity. In earlier studies in young adults, BMI is affected in a subtle, but opposite manner in both sex in terms of their height.[24] Although the body mass index is widely used as a surrogate measure of adiposity, it is moderately associated (r=0.3) with height among children.[25] In one of the research reports, adult height was well predicted from childhood, with strong positive correlations (r=0.7 for both sexes) with BMI between ages 7 and 33 years.[12] In the present study similar strong but negative correlation of height (r=-0.8) was observed with BMI in obese group, and r=-0.5 in non-obese. Difference in correlation values between above mention study[12] and the present study reflects difference in age range. Deurenberg *et al*[21] stated that after the age of 16 years the positive correlation of the BMI with body height disappeared and became even slightly, but significantly negative. In the present study the age group is above 20 years. Similar to our study, Diverse Population Collaborative Group (DPCG)[26] demonstrated negative correlation in the age group of >25 years and older. There was a positive correlation between BMI and weight for both obese (r=0.8) and non-obese (r=0.5) groups. Similar results have been reported by DPCG[26], with higher correlation of body weight with BMI. This is in conformity with our results. Body weight correlates better than height with obesity. The differences in the *r*-value and slope of regression line in obese and non-obese in our study demonstrates greater change in BMI per unit change in both the height and weight in obesity, irrespective of their negative or positive correlation.

BMI has shown positive correlation with WC and TSG, in obese group. In earlier studies[27,28], similar results have been reported. WC is a better indicator of obesity than TSG when they are correlated with BMI in general. According to above discussed results of correlation (ANPP vs BMI), the order is Height>Weight>WC>TSG>Age.

Associations between BMI and CVP like, PR, SBP, DBP, MAP and PP have been consistently observed for obese in several studies[14,29]. In the present study, both obese and non-obese have been correlated for comparison regarding BMI vs CVP. Our results show positive correlation of all of these CVP with BMI for both the obese and non-obese groups, except PP where non-obese showed negative correlation with BMI. This correlation for all of the above mentioned CVP was poor in non-obese groups. In an earlier study the association between BMI and BP was examined in three obese populations across Africa and Asia[30], showing significant and positive correlation of BMI with both SBP and DBP for obese in all the three populations. Obesity determined in terms of BMI is responsible to shift the positive correlation of CVP with BMI towards higher values and thus our correlation study confirms earlier studies that represents higher values of CVP with BMI involving greater risk for cardiovascular diseases including high cholesterol, essential hypertension and coronary heart diseases as also mentioned by ANHLA and Blood institute as well as WHO[31].

Our results are different for the correlation of BMI with Pulse pressure (PP) than remaining CVP in terms of low values of correlation. The negative correlation in non-obese suggests that some compensatory mechanism is probably responsible to decrease PP with BMI, while this compensatory effect vanishes in obesity leading to positive correlation of PP with BMI. In an earlier study[10], PP was reported higher in the lean (BMI<25) than in the overweight (BMI≥25). Another study[32] has reported an independent association of increase in BMI with decrease in the arterial compliance. The inverse relation observed in our study between BMI and PP in non-obese may help to explain previous reports of increased cardiovascular risk among lean versus obese subjects. Correlation of CVP with BMI falls in the order of PR>MAP>DBP>SBP>PP.

The distribution of body fat has been shown to be an important determinant of cardiovascular disease risk. The intra-abdominal fat has been identified with adverse lipid profile and hypertension in various types of patients.[26] WC and TSG are helpful parameter for measuring the abdominal and general obesity. There was a positive correlation of WC with SBP, DBP and MAP for both obese and non-obese groups. This correlation was poor in non-obese group being 0.2, 0.4 and 0.3 for SBP, DBP, and MAP respectively. One study[11] examined similar correlation between some ANPP and cardiovascular parameters which were found to be positive and significant. In another study[33] waist circumference had a high correlation with systolic and diastolic BP. Our results are in conformity with them for correlation between CVP and WC. Our study also demonstrated correlation between WC and TSG for obese and for non-obese groups. In an earlier study[34] the best correlation of fat distribution is regarded to the WC among all body measures for body fat. The above studies indicate that WC is an important indicator of obesity in terms of body fat contents along with TSG. On the other hand, in non-obese group of our study, the poor correlation exhibits only non-associated changes among them, if any.

In obesity a variety of adaptations and alterations in cardiovascular structure and function occur and reflected in its well defined parameters such as PR, SBP, DBP, MAP and PP. In previous studies, the correlation of these parameters with BMI or WC, TSG and waist hip ratio (WHR) were extensively reported.[11,14,35] in obese persons. However, none of the study has yet reported correlation between CVS parameters themselves in obese and non-obese persons. In the present study these correlations between CVP has provided important inter-relations. These interrelations demonstrated positive and significant correlations of pulse rate with SBP, DBP and MAP, in obese group. However, in non-obese group it is negative and non-significant. It is an important finding indicating that in normal persons a probable rise in PR may not be compulsorily associated with a simultaneous rise in other CVP, mentioned above. In obesity this correlation has been found to shift towards higher *r*-values indicating that a minor increase in pulse rate may be responsible for a concomitant rise in SBP, DBP and MAP. In an earlier study[14] statistically significant increase in these parameters had been shown in obese subjects when compared to non-obese.

SBP and DBP frequently display positive relationship with a certain ratio depending upon the pathophysiology involved and provided that persons are not suffering from isolated hypertension (ISH). In the present study, Pearson's correlation between SBP and DBP for both the obese and non-obese groups was found to be r=0.8 and r=0.7 respectively. It clearly indicates that these persons are not suffering from ISH. In an earlier study, the correlation between SBP and DBP in hypertensive patient (24 hours ambulatory assessment) was r=0.74±0.14 (r>0.5 in 95% of patients).[24] It is therefore, suggested that obesity and hypertension are though separate clinical conditions, but both of them correlate SBP and DBP with more or less similar *r*-values.

MAP is closer to the arithmetic average of SBP and DBP. The correlation between SBP and MAP in our study is r=0.9 for both obese and non-obese group. Previous researches also showed that the average values of SBP and DBP were highly correlated with MAP, with Spearman correlations ranging from 0.88 to 0.94 (p<0.001) in men.[23] Therefore, it is suggested that MAP rises with either increase in SBP and DBP irrespective of obesity.

The pulse pressure has been considered an index of pulsatile component of the cardiac cycle. There is a relationship between pulse pressure and heart disease.[36,37] Vaccarino *et al*[38] investigated that the pulse pressure, which is a measure of arterial stiffness, is also an independent predictor of the incidence of coronary heart disease, congestive heart failure and overall mortality among community-dwelling elderly. In the present study an attempt was also made to correlate SBP and PP which shows the value of r=0.7 for obese and r=0.4 for non-obese groups. This indicates that PP is not only a measure of arterial stiffness but can also indicate the development of other CV diseases related to obesity. Earlier evidence[39] also suggests a similar finding in one study where systolic blood pressure correlates highly with pulse pressure in univariate analysis.

CONCLUSION

The order of Pearson's correlation for anthropometric parameters with BMI is Height>Weight>WC>TSG. Short height followed by over-weight are the main anthropometric characters of obesity. Correlation of CVP with BMI falls in the order of PR>MAP>DBP>SBP>PP. The inverse relation between BMI and PP in non-obese group is important to understand CV risk in the lean versus obese subjects.

ACKNOWLEDGEMENT

Technical assistance from Mr. Nadir Ansari in statistical analysis and script writing is greatly appreciated.

REFERENCES

1. Park K. Textbook of Preventive and Social Medicine. 18th ed. Banarsidas Bhanot, India: 2005;316–9.
2. World Health Organization. Obesity: Preventing and managing the global epidemic. WHO Technical Report Series No. 894. Geneva. 2000.

3. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: The National Health And Nutrition Examination Surveys, 1960–1991. JAMA 1994;272:205–11.
4. Flegal KM, Carrol MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2008. JAMA 2002;288:1723–7.
5. Nanan DJ. The Obesity Pandemic –Implications for Pakistan. J Pak Med Assoc 2002;52:342.
6. Katherine MF, Margaret DC, Cynthia LO, Lester RC. Prevalence and trends in obesity among US adults, 1999–2008. JAMA 2010;303(3):235–41.
7. Kasper DL, Braundwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL. Harrison's Principles of Internal Medicine, Vol. 1. 16th ed. United States of America: The McGraw Hill Companies, 2005;422–9.
8. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The Evidence Report: National Institutes of Health. Obes Res 1998;6(Suppl 2):51S–209S.
9. Despres, Jp. Health consequences of visceral obesity. Ann Med 2001;33:534–41.
10. Martins D, Tareen N, Pan D, Norris K. The relationship between body mass index and pulse pressure in older adults with isolated systolic hypertension. Am J Hypertens 2002;15(6):538–43.
11. Abiodun AG, Egwii MO, Adedoyin RA. Anthropometric indices associated with variation in cardiovascular parameters among primary school pupils in ILE-IFE. Int J Hypertens 2011;2011:186194. doi: 10.4061/2011/186194.
12. Power C, Lake JK, Cole TJ. Body mass index and height from childhood to adulthood in the 1958 British Born Cohort. Am J Clin Nutr 1997;66(5):1094–110.
13. Ofuya ZM, Goergewill AA, Agu GO. A study of cardiovascular parameters in obese and non obese subjects residents in Port Harcourt. African J Appl Zool Environ Biol 2005;7:11–3.
14. Shekharappa RK, Johncy S, Mallikarjuna PT, Vedavathi KJ, Jayarjan MP. Correlation between body mass index and cardiovascular parameters in obese and non-obese in different age groups. Int J Biol Med Res 2011;2(2):551–5.
15. Zhang C, Rexrode KM, van Dam RM, Li TY, Hu FB. Abdominal obesity and the risk of all-cause, cardiovascular, and cancer mortality: Sixteen years of follow-up in US women. Circulation 2008;117:1658–67.
16. Sichieri R, Dos Santos, Barbosa F, Mouva EC. Relationship between short stature and obesity in Brazil: A multilevel analysis. Br J Nutr 2010;103(10):1534–8.
17. Velásquez-Meléndez G, Martins IS, Cervato AM, Fornés NS, Marucci MF, Coelho LT. Relationship between stature, overweight and central obesity in the adult population In São Paulo, Brazil. Int J Obes Relat Metab Disord 1999;23(6):639–44.
18. Sahar A. Ibrahim, Maysa A. Samy, Azza O. L. Saleh, Gulsen S. Ahmed, May K. Matter. Obesity among Egyptian adults with short stature. J T U Med Sci 2010;5(2):98–104.
19. Body mass index and health. A publication of the USDA Center for Nutrition Policy and Promotion March 2000. Page 1–2. http://www.cnpp.usda.gov/publications/nutritioninsights/insight16.pdf
20. Roche AF, Sievogel RM, Chumla WC, Webb P. Grading body fatness from limited anthropometric data. Am J Clin Nutr 1981;34(12):2831–8.
21. Deurenberg P, Westrate JA, Seidell JC. Body mass index as a measure of body fatness: Age and sex specific prediction formulas. Br J Nutr 1991;65(2):105–14.
22. Sesso HD, Stampfer MJ, Rosner B, Hennenkens CH, Gaziano JM, Manson JE, *et al*. Systolic and diastolic blood pressure, pulse pressure and mean arterial pressure as predictors of cardiovascular disease risk in men. Hypertension 2000;36:801–7.
23. Gavish B, Ben-Dar IZ, Bursztyn M. Linear relationship between systolic and diastolic blood pressure monitored over 24 H: Assessment and correlates. J Hypertens 2008;26(2):199–209.
24. Mandel D, Zimlichman E, Mimouni FB, Grotto I, Kreiss Y. Height-related changes in body mass index: A reappraisal. J Am Coll Nutr 2004;23(1):51–4.
25. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivisan SR, Berenson GS. Inter-relationships among childhood BMI, childhood height, and adult obesity: The Bogalusa Heart Study. Int J Obes 2004;28:10–6.
26. Diverse Population Collaborative Group. Weight-height relationships and body mass index: Some observations from the diverse population collaboration. Am J Phys Anthropol 2005;128(1):220–9.
27. Pratesi S, Peternostro F, Tani A, Sassoli C, Cappellini AC. Waist circumference correlates with body mass index (BMI) in school-aged children. Italian J Anat Embryol 2011;116(1 Supplement):150.
28. Nevill AM, Stewart AD, Olds T, Holder R. Relationship between adiposity and body size reveals limitations of BMI. Am J Phys Anthropol 2006;129(1):151–6.
29. Yar T. Resting heart rate and its relationship with general and abdominal obesity in young male Saudi university students. Pak J Physiol 2012;6(1):6–13.
30. Tesfaye F, Nawi NG, Van Minh H, Byass P, Berhane Y, Bonita R, *et al*. Association between body mass index and blood pressure across three populations in Africa and Asia. J Hum Hypert 2007;21:28–37.
31. Must A, Spadano J, Eugenie H, Coakley MA, Alison EF, Colditz G, *et al*. The disease burden associated with overweight and obesity. JAMA 1999;282(16):1523–9.
32. Kwagyan J, Tabe CE, Xu S, Maqbool AR, Gordeuk VR, Randall OS. The impact of body mass index on pulse pressure in obesity. J Hypertens 2005;23(3):619–24.
33. Maffeis C, Pietrobelli A, Grezzani A, Provera S, Tato L. Waist circumference and cardiovascular risk factors in prepubertal children. Obesity Research 2001;9:179–87.
34. Daniels SR, Khoury PR, Morrison JA. Utility of different measures of body fat distribution in children and adolescents. Am J Epidemiol 2000;152(12):1179–84.
35. Farida M, Ammarah, Sara, Momina, Sadaf M, Madiha A. Waist hip ratio and body mass index in women of different age groups. Pak J Physiol 2012;8(1):49–51.
36. Kannel WB, Tavia G, Schwartz MJ. Systolic versus diastolic blood pressure and risk of coronary heart disease. Am J Cardiol 1971;27(4):335–46.
37. Mitchell GF, Moyé LA, Braunwald E, Rouleau JL, Bernstein V, Geltman EM, *et al*. Sphygmomanometrically determined pulse pressure is a powerful independent predictor of recurrent events after myocardial infarction in patients with impaired left ventricular function. Circulation 1997;96:4254–60.
38. Vaccarino V, Holford TR, Krumholz HM. Pulse pressure and risk for myocardial infarction and heart failure in the elderly. J Am Coll Cardiol 2000;36(1):130–8.
39. Klassen PS, Lowrie EG, Reddan DN, Delong ER, Coladonato JA, Szczech LA, *et al*. Association between pulse pressure and mortality in patients undergoing maintenance hemodialysis. JAMA 2002;287(12):1548–55.

Address for Correspondence:

Professor Dr. Muhammad Abdul Azeem, Department of Physiology, United Medical & Dental College, Korangi, Ibrahim Heydri, Karachi, Pakistan. **Cell:** +92-300-2510190
Email: azenmu@gmail.com