Association of obesity with hypertension and type 2 diabetes and the role of the

menopausal status

Kawaljit Kaur Khokhar¹, Gurcharan Kaur², Sharda Sidhu¹

¹ Dept. of Human Genetics, Guru Nanak Dev University, Amritsar, 143005, Punjab, India ²Dept. of Biotechnology, Guru Nanak Dev University Amritsar 143005, Punjab, India **Corresponding Author**: Kawaljit Kaur Khokhar, Dept. of Human Genetics, Guru Nanak Dev University, Amritsar, 143005, Punjab, India.

E-mail: virk.khokhar@yahoo.com

Abstract: Background: In India, Punjab ranks one with 33.9% of population being overweight or obese and the prevalence is higher among women.

Aim: The present cross-sectional study was carried out to find out the association of obesity with hypertension and type 2 diabetes among women and to further assess the impact of the menopausal status on it.

Subjects and Methods: 595 working women (330-pre- and 265-postmenopausal) were personally interviewed for the study. Their body measurements were taken. Blood pressure and blood glucose were checked. Subjects were divided into non-obese and obese groups as per WHO guidelines. Prevalence of hypertension and type 2diabetes was calculated among non-obese and obese subjects as per standardized criteria.

Results: The prevalence of pre-hypertension was 3.44 % and 13.24%, hypertension as 5.17% and 17.65%, and diabetes as 5.17% and 9.19%, respectively among non-obese and obese premenopausal subjects. Similarly, among non-obese and obese postmenopausal women, the prevalence of pre-hypertension was 7.40% and 13.03%, hypertension as 14.82% and 30.25%, and diabetes as 7.41% and 15.55%, respectively. Pearson's correlation co-efficient reflects the association of BMI with Blood pressure.

Conclusions: Obese women are at higher risk of hypertension and type 2 diabetes and the effect of obesity on these diseases seems to be independent of the menopausal status. But as the chi square test didn't reveal the significant differences between non-obese and obese groups, it is suggested that overweight or obesity might be contributing but is not the sole factor in the etiology of hypertension and type 2 diabetes.

Key Words- Premenopausal women, Postmenopausal women, Globesity, Body Mass Index, Hypertension, Type 2 Diabetes

INTRODUCTION

Obesity has become the worst pandemic of the 21st century (Wharton, 2009; Chowbey, 2009).

The pandemic is so great that, it has even spawned a new word "globesity" or worldwide obesity.

Globally, obesity has reached epidemic proportions with more than 1 billion adults being overweight and at least 300 million are clinically obese (WHO, 2003). With the new Asian Body Mass Index (BMI) criteria of overweight at a lower cut-off of $23 \cdot 0 \text{ kg/m}^2$, the number is even higher reaching $1 \cdot 7$ billion people (James *et al.*, 2004; Haslam and James, 2005). So, the obesity epidemic is not restricted to industrialized societies rather this increase is often faster in developing countries than in the developed world. Prentice (2006) reported that the total number of obese or overweight people is projected to grow by 50 percent in the next 10 years, primarily in developing countries.

Obesity is a major risk factor for chronic diseases and plays a central role in the "insulin resistance" or "metabolic" syndrome, which includes hyperinsulinemia, hypertension, hyperlipidemia, type 2 diabetes mellitus (T2DM), and an increased risk of atherosclerotic cardiovascular disease (CVD) (Sinaiko *et al.*, 2001; Ogden *et al.*, 2007; Kelishadi, 2007). About 18 million people die every year from CVDs, for which diabetes and hypertension are the major predisposing factors (Hossain *et al.*, 2007). According to Hall (1990), hypertension is one of the most prevalent and powerful contributors to atherosclerotic CVD and is a common disorder which affects over 40 million individuals in the United States alone.

In recent years, there has been a dramatic increase in the prevalence of obesity in India as well as other regions of the world. India is following a trend of other developing countries that are steadily becoming more obese. According to Singhal (2009), as obesity is becoming more and more common in middle class and neo-rich class in India also, it is no more the disease of high class society or the disease of the rich. It is assumed that there are about 1.5 million people in India who are coming into the group requiring surgical intervention. Obesity is a major chronic disorder, affecting 20%-40% adults in India. The prevalence of obesity is higher among women and also in economically better off individuals and who live in urban areas. Women in higher socio-economic status experience the greater risk for being pre-overweight, overweight and obese (Shukla *et al.*, 2002; Subramanian and Smith, 2006; Nirmala, 2009). Literature supports strongly that the prevalence of obesity is higher among women as compared to men worldwide (Saaristo *et al.*, 2008; Sidik and Rampal, 2009). One of the important reasons for the increased prevalence of obesity among women is the onset of menopause. Obesity among post-

menopausal women has been observed as higher as compared to premenoausal women (Toth, 2000; de Paz *et al.*, 2006; Sharma *et al.*, 2008; Begum *et al.*, 2009; Khokhar *et al.*, 2010).

Punjab is a prosperous state of India and Jalandhar is a prominent city of Punjab. Obesity is emerging as a major problem in Punjab. According to reports of National Family Health Survey (NFHS)- III (2005-6), in India, Punjab ranks one with 30.3% of males and 37.5% of females being overweight or obese. Jalandhar city on one hand is famous for its medical facilities having maximum number of hospitals in Asia, on the other hand obesity and its related diseases like hypertension and T2DM seems to be higher in Jalandhar, especially in women (Kaur *et al.*, 2009: Khokhar *et al.*, 2010). In our previous studies conducted on pre- and postmenopausal women of Jalandhar, we observed 70.30% of pre- and 75.09% of postmenopausal women as obese (BMI>25kg/m²) (Khokhar *et al.*, 2010). The prevalence of hypertension was also studied and it came out that 15.45% of pre- and 27.55% of postmenopausal women were hypertensive (Kaur *et al.*, 2009). Similarly, the prevalence of T2DM was observed as 8.48% and 14.72% in pre- and postmenopausal women, respectively (data not published).

The present cross-sectional study was carried out to see the effect of obesity on the prevalence of hypertension and T2DM among women of Jalandhar and to further assess the effect of the menopausal status on it.

SUBJECTS AND METHODS

595 (330-pre- and 265-postmenopausal) women, working in various educational institutes and hospitals were personally interviewed for the study. A questionnaire was framed to get the desired information. The study was approved by the Ethical Review Committee of Guru Nanak Dev University, Amritsar. The anthropometric measurements like; height and weight were taken on each subject using standard methodology (Weiner and Lourie, 1981). Blood pressure (BP) was measured with mercury sphygmomanometer (Diamond Deluxe BP Apparatus, Pune, India) and stethoscope as per recommendations of Rose and Blackburn (1968). Random Blood Glucose level (RBG) of all the subjects was checked at the time of the interview using a standardized digital glucometer (*Accua Chek Active Glucometer*, Roch Diagnostics, Switzerland). BMI was calculated and subjects were divided in two groups; non-obese and overweight/ obese as per World Health Organization (WHO, 2000) guidelines. Status of hypertension was assessed as per

JNC (VII) guidelines, as reported by Chobanian *et al.*, (2003) and the T2DM was evaluated as per guidelines given by WHO (2006) among non-obese and overweight/ obese subjects separately. For statistical analysis Chi square test and Student's t-test was applied.

RESULTS

1. Prevalence of Pre-hypertension and Hypertension among Non-obese and Overweight/ Obese Subjects

Table 1 depicts the percent prevalence of pre-hypertension and hypertension in non-obese (BMI<23.0 kg/m²) and overweight/obese (BMI \geq 23.0 kg/m²) pre- and postmenopausal women. In case of premenopausal women, the prevalence of pre-hypertension was, 3.44 % (2) and 13.24% (36) in non-obese and overweight/ obese women, respectively. Similarly, in case of postmenopausal women, 7.40% (2) of non-obese and 13.03% (31) of overweight/ obese females were pre-hypertensive. On the other hand, the prevalence of hypertension in non-obese and overweight/ obese premenopausal women was 5.17% (3) and 17.65% (48), respectively.

Table 1: Per cent Prevalence of Pre-Hypertension and Hypertension in Non-Obese (BMI<23.00 kg/m²) and Overweight/Obese (BMI \geq 23.00 kg/m²) Premenopausal (Pre-M) and Postmenopausal Women (Post-M)

	Pre-N	A (330)	Post-M (265)		
Category	BMI <23.0 (58)	BMI≥23.0 (272)	BMI <23.0 (27)	BMI≥23.0 (238)	
Pre-hypertensive	3.44	13.24*	7.40	13.03*	
	(2)	(36)	(2)	(31)	
Hypertensive	5.17	17.65*	14.82	30.25*	
	(3)	(48)	(4)	(72)	
Normotensive	91.38	69.12	77.78	56.72	
	(53)	(188)	(21)	(135)	

*= p<0.05 Digits in parentheses are the number of subjects

Similarly, 14.82% (4) of non-obese and 30.25% (72) of overweight/ obese postmenopausal women were hypertensive. 91.38% (53) of non-obese and 69.12% (188) of obese premenopausal subjects were normotensive, whereas, 77.78% (21) of non-obese and 56.72% (135) of

overweight/obese postmenopausal subjects were normotensive. Although the overweight/obese subjects had 2-3 times rate of pre-hypertension and hypertension as compared to their non-obese counterparts but the chi square test revealed the non-significant difference between the groups.

2. Prevalence of T2DM among Non-obese and Overweight/Obese Subjects

Table 2 shows the prevalence of T2DM in non-obese (BMI<23.00 kg/m²) and overweight/obese (BMI \geq 23.00 kg/m²) pre- and postmenopausal women. The prevalence of T2DM in non-obese and overweight/ obese premenopausal females was 5.17% (3) and 9.19% (25), respectively. The rest 94.83% (55) non-obese and 90.81% (247) overweight/ obese premenopausal subjects were non-diabetic. Similarly, in postmenopausal women, 7.41% (2) non-obese and 15.55% (37) overweight/ obese females were diabetic. The rest of 92.59% (25) non-obese and 84.45% (201) overweight/ obese subjects were non-diabetic. Overweight/ Obese pre- and postmenopausal subjects but the difference between the groups was statistically non-significant.

Table 2: Per cent Prevalence of Type 2 Diabetes Mellitus in Non-Obese (BMI<23.00 kg/m²) and Overweight/Obese (BMI≥23.00 kg/m²) Premenopausal (Pre-M) and Postmenopausal Women (Post-M)

	Pre-N	I (330)	Post-M (265)		
Category	BMI <23.0	BMI≥23.0	BMI <23.0	BMI≥23.0	
	(58)	(272)	(27)	(238)	
Diabetic	5.17	9.19*	7.41	15.55*	
	(3)	(25)	(2)	(37)	
Non-Diabetic	94.83	90.81	92.59	84.45	
	(55)	(247)	(25)	(201)	

*=p<0.05 Digits in parentheses are the number of subjects

DISCUSSION

Obesity is the important modifiable risk factor for hypertension and T2DM. In the current study, it has been seen that the prevalence of pre-hypertension, hypertension and T2DM increased multifold in overweight/obese subjects as compared to their non-obese counterparts. The

prevalence of pre-hypertension and hypertension increased more than three times in overweight/obese premenopausal women and more than two times in overweight/obese postmenopausal women as compared to their non-obese counterparts. Similar was the status of T2DM. In overweight/obese premenopausal subjects, it increased almost two times whereas, in overweight/obese postmenopausal subjects, almost three times higher rate of diabetes was observed as compared to their non-obese counterparts.

Higher prevalence of hypertension and T2DM among overweight/obese women as compared to non-obese women can be related to their higher BMIs. Higher is the BMI among overweight and obese subjects as compared to their non-obese counterparts, higher are the values of Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Pulse Pressure (PP) and Mean Arterial Pressure (MAP) in overweight/ obese subjects, although there is not statistically significant difference between the groups for values of PP in both pre- and postmenopausal and SBP in postmenopausal women only (**Table 3**). Higher values of SBP, DBP, PP and MAP are the indicators of hypertension and T2DM. So, it is suggested that overweight and obese subjects.

The significant association of BMI with SBP, DBP and MAP is also evident from the values of Pearson's correlation coefficient among pre- and postmenopausal women of the present study (**Table 4**). This table shows significant association of BMI with SBP, DBP and MAP among both pre- and postmenopausal women. It is apparent that BMI is positively and significantly correlated with SBP (r=0.261, p<0.001), DBP (r=0.268, p<0.001) and MAP (r=0.231, p<0.001) among premenopausal women. Similarly, significant and positive correlation was observed between BMI and SBP (r=0.261, p<0.001), DBP (r=0.328, p<0.001) and MAP (r=0.307, p<0.001) among postmenopausal women. These findings are in agreement with other studies (Tesfaye *et al.*, 2007; Wang *et al.*, 2010; Raj *et al.*, 2010) which support a strong relationship between BMI and BP across developed and developing countries. Surprisingly, however Danon-Hersch *et al.* (2007) reported that the relationship between BMI and BP decreases over time.

The link between obesity and hypertension is well known, but the exact nature of the association between the two disorders remains unclear. The relationship between obesity and BP appears to be linear and exists throughout the non-obese range. But the strength of the association of obesity with hypertension varies among different racial and ethnic groups. The relationship between

Table 3: Comparison between the mean values of Body Mass Index (BMI), Systolic Blood					
Pressure (SBP), Diastolic Blood Pressure (DBP), Pulse Pressure (PP) and Mean Arterial					
Pressure (MAP) of non-obese and overweight/obese subjects.					

Variables	Non-Obese		Overweight & Obese			P-value	
	Mean	SD	SEM	Mean	SD	SEM	
Preme	Premenopausal Women (58 Non-Obese/272 Overweight & Obese)						
BMI	20.54	1.78	0.23	28.00	3.66	0.23	< 0.001
SBP (mm/Hg)	117.14	16.47	2.16	121.96	17.57	1.09	< 0.05
DBP (mm/Hg)	75.78	10.66	1.40	81.17	11.86	0.74	< 0.01
PP (mm/Hg)	40.36	10.36	1.36	41.36	9.93	0.62	NS
MAP (mm/Hg)	89.33	12.93	1.70	94.35	12.46	0.77	< 0.01
Postmenopausal Women (27 Non-Obese/238 Overweight & Obese)							
BMI	20.55	2.28	0.39	28.93	3.58	0.27	< 0.001
SBP (mm/Hg)	123.77	12.34	2.09	129.95	20.10	1.53	NS
DBP (mm/Hg)	77.20	8.40	1.42	83.29	14.37	1.09	< 0.02
PP (mm/Hg)	46.57	9.57	1.62	46.79	13.04	0.99	NS
MAP (mm/Hg)	93.18	9.19	1.55	98.26	14.32	1.09	< 0.05

obesity and hypertension is growing as an important public health challenge worldwide. Compared with the year 2000, the number of adults with hypertension is predicted to increase by ~60% to a total of 1.56 billion by the year 2025 (Kearney *et al.*, 2005). Based on population studies, risk estimates indicate that at least two-thirds of the prevalence of hypertension can be directly attributed to obesity (Krause *et al.*, 1998). Obesity might lead to hypertension and cardiovascular disease by activating the renin–angiotensin–aldosterone system by increasing sympathetic activity, by promoting insulin resistance and leptin resistance, by increased procoagulatory activity and by endothelial dysfunction. Further mechanisms include increased

renal sodium reabsorption, causing a shift to the right of the pressure–natriuresis relationship and resulting in volume expansion (Wofford and Hall, 2004).

Table 4: Pearson's correlation co-efficient (r) of Body Mass Index (BMI) with Blood Pressure variables (SBP, DBP, PP, MAP) among Premenopausal and Postmenopausal Women

Variable	Premenopausal Women (330)		Postmenopausal Women (265)		
	r	p-value	r	p-value	
SBP	0.261	< 0.001	0.261	<0.001	
DBP	0.268	< 0.001	0.328	<0.001	
РР	0.030	0.665	0.063	0.261	
MAP	0.231	< 0.001	0.307	<0.001	

SBP-systolic blood pressure; DBP-diastolic blood pressure; PP-pulse pressure; MAP- mean arterial pressure

In overweight/obese subjects, adipose tissue might be playing role in the genesis of hypertension. Other epidemiological studies have revealed a strong relation between obesity and hypertension. Onat *et al.* (1997) have suggested that BMI is a strong independent factor for SBP in women. It was further reported that for every 10 mmHg increase in SBP, the risk of Coronary Artery Diseases increases by 22%. Data from the National Health and Nutrition Examination Survey (NHANES) show a remarkable and linear relationship between rise in BMI and systolic, diastolic, and pulse pressures in the American population. In regression models corrected for age-related rise in BP, a BMI gain of 1.7 kg/m² in men and 1.25 kg/m² in women corresponds to an increase in SBP of 1 mmHg (Kissebah and Krakower, 1994). Higher BMI is an indicator of weight gain and it may be suggested that the increase in BP with excess weight gain arises partly because of the release from adipocytes of angiotensinogen (a precursor of angiotensin that has well-known effects on BP), an increase in blood volume associated with the greater body mass, and in response to a rise in blood viscosity. The change in blood viscosity is induced by the 130

release of profibrinogen and plasminogen activator inhibitor from adipocytes with a fall in plasminogen activator (Skurk *et al.*, 2004; Haslam and James, 2005).

This epidemic of obesity and obesity-related hypertension is paralleled by an alarming increase in the incidence of diabetes mellitus and chronic kidney disease. Association between obesity and T2DM has also been reported in literature. Hu et al. (2001) have also reported that diabetes is primarily determined by obesity and lifestyle factors such as diet and exercise and is most frequent in obese individuals over the age of 40 years. In India, there is increasing urbanization and industrialization which has led to physical inactivity, sedentary lifestyle, psychosocial stress and obesity leading to progressive increase in prevalence of T2 DM (Gupta and Pathak, 2003). About 90% of T2DM is attributable to excess weight. Furthermore, approximately 197 million people worldwide have impaired glucose tolerance, most commonly because of obesity and the associated metabolic syndrome. This number is expected to increase to 420 million by 2025. The high risk of both diabetes and CVD associated with obesity in Asians may be due to a predisposition to abdominal obesity, which can lead to the metabolic syndrome and impaired glucose tolerance (Hossain et al., 2007). A 2009 National Health Interview Survey of 88,129 persons found that 28% of U.S. adults over age 20 were obese, and 9% were diagnosed with diabetes. This represents a 44% increase in obesity and a 76% increase in diabetes since 1997 (Barnes, 2009).

Self-management training yields positive effects on essential aspects of diabetes self-care and weight control for people with a high BMI. Modest weight loss, defined as a loss of 5% to 10% of baseline weight, has received increasing attention as a new treatment strategy for overweight and obese patients (Ford *et al.*, 1997; NIH, 1998). As obesity is the root cause of hypertension and T2DM, the stress should be laid on its control from the childhood because childhood obesity leads to adulthood obesity in most of the cases.

CONCLUSIONS

The prevalence of hypertension and T2DM was observed as much higher among overweight/obese pre- and postmenopausal women as compared to non-obese subjects. Although the difference between non-obese and overweight/ obese subjects was statistically non-significant, one thing is sure that that obese women are at higher risk of hypertension and T2DM. As the proportion of differences was almost the same in pre- and postmenopausal women, the

role of obesity in the etiology of hypertension and T2DM seems to be independent of the menopausal status. As the women under study were belonging to working class, may be work stress plays some role in the etiology of hypertension and T2DM. But the association of obesity with hypertension and T2DM can't be ruled out. In women, especially the postmenopausal women who are prone to become obese, the matter should be taken seriously. They must learn to adapt a healthy life style, eat healthy, incorporate physical work out in their daily routines and must try to keep themselves fit by maintaining the required body weight.

LIMITATION OF THE STUDY

As per WHO (2000) guidelines, obesity has been divided into different classes like; Obesity Class-I, Obesity Class-II and Obesity Class-III where hierarchy of the classes goes in proportion with increasing BMI. The health risks associated with increasing BMI are continuous but we didn't consider the association of hypertension and T2DM in proportion to different classes of obesity.

SUGGESTIONS

The prevalence of hypertension and T2DM was observed higher in overweight and obese subjects as compared to their non-obese counterparts but the Chi square test didn't reveal the significant differences between the groups. It is suggested that degree of obesity (Class-I,II and III) should also be considered while assessing the association between obesity and its related disorders in further studies.

DECLARATION OF INTEREST

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

ACKNOWLEDGEMENTS

All the subjects who co-operated and participated in the fulfillment of this study are duly acknowledged. The authors are thankful to U.G.C. for providing financial support for this work.

REFERENCES

Barnes P. 2009. National Health Interview Survey, Centers for Disease Control & Prevention, National Center for Health Statistics.

Begum P, Richardson CE and Carmichael AR. 2009. Obesity in post menopausal women with a family history of breast cancer: prevalence and risk awareness. *Int Semin Surg Oncol* **6**: 1.

Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr., Jones DW, Materson BJ, Oparil S, Wright JT Jr. and Roccella EJ. 2003. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* **289**: 2560-2572.

Chowbey PK. 2009. Bariatric surgery- Surgical management of morbid obesity. In Sinha, R. and Kapoor S. (eds.).*Obesity-A multiple dimensional approach to contemporary global issue*, pp 317-331. Dhanraj book house, New Delhi.

Danon-Hersch N, Chiolero A, Shamlaye C, Paccaud F and Bovet P 2007. Decreasing association between body mass index and blood pressure over time. *Epidemiology* **18**: 493-500.

de Paz IP, Hernando CA and Roldán JO. 2006. Obesity and menopause. *Nutr Hosp* 21(6): 633-637.

Ford ES, Williamson DF and Liu W. 1997. Weight change and diabetes incidence: findings from a cohort of US adults. *Am J Epidem* **146(3)**:214-222.

Gupta OP and Phatak S. 2003. Pandemic trends in prevalence of diabetes mellitus and associated coronary heart disease in India-their causes and prevention. *Int J Diab Dev Ctries* **23**: 37-50.

Hall PM. 1990. Hypertension in women. Cardiology 77 Suppl 2: 25-30.

Haslam DW and James WP. 2005. Obesity. Lancet 366:1197-1209.

Hossain P, Kawar B and Nahas MEI. 2007. Obesity and diabetes in the developing world--a growing challenge. *N Engl J Med* **356**: 213-215.

Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG and Willett WC. 2001. Diet,

lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med 345: 790-797.

James PT, Rigby N and Leach R. 2004. The obesity epidemic, metabolic syndrome and future prevention strategies. *Eur J Cardiovasc Prev Rehabil* **11**: 3-8.

Kaur K, Sidhu S and Kaur G. 2009. Prevalence of Hypertension in working Pre- and Postmenopausal women of Jalandhar City (Punjab). *Indian Journal of Physical Anthropology and Human Genetics* **28** (1-2): 161-168

Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK and He J. 2005. Global burden of hypertension: analysis of worldwide data. *Lancet* **365**: 217-223.

Kelishadi R. (2007). Childhood overweight, obesity, and the metabolic syndrome in developing countries. *Epidemiol Rev* **29**: 62-76.

Khokhar KK, Sidhu S and Kaur G. 2010. Correlation between leptin level and hypertension in normal and obese pre- and postmenopausal women. *Eur J Endocrinol* **163**: 873-878.

Kissebah AH and Krakower GR. 1994. Regional adiposity and morbidity. *Physiol Rev* 74:761–811.

Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK and He J. 2005. Global burden of hypertension: analysis of worldwide data. *Lancet* **365**: 217–223.

Krause RM, Winston M, Fletcher BJ and Grundy SM. 1998. Obesity. Impact on cardiovascular disease. *Circulation* **98**:1472–1476.

Nirmala A. 2009. Obesity and its association with socio-economic status: local and global perspective. In Sinha, R and Kapoor, S (eds). *Obesity-A Multidimentional Approach to Contemporary Global Issue*. pp 195-207. Dhanraj book house, New Delhi, India.

NFHS-III. 2005-06. National Family Health Survey. International Institute for Population Sciences (IIPS). Deonar, Mumbai. http://www.nfhsindia.org/data/pj. Accessed 2010 May, 6.

NIH 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Evidence Report. National Institutes of Health.

Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ and Flegal KM. 2006. Prevalence

of overweight and obesity in the United States, 1999-2004. JAMA 295: 1549-1555.

Onat A, Dursunoglu D and Sansoy V. 1997. Relatively high coronary death and event rates in Turkish women. Relation to three major risk factors in five-year follow-up of cohort. *Int J Cardiol* **61**: 69-77.

Prentice AM. 2006. The emerging epidemic of obesity in developing countries. *Int J Epidemiol* **35**: 93-99.

Raj M, Sundaram KR, Paul M, Sudhakar A and Kumar RK. 2010. Body mass index trend and its association with blood pressure distribution in children. *J Hum Hypertens* **24**: 652-658.

Rose G.A. and H. Blackburn 1968. Cardiovascular Survey Method. WHO Monograph Series: 56:90-95.

Saaristo TE, Barengo NC, Korpi-Hyovalti E, Oksa H, Puolijoki H, Saltevo JT, Vanhala M, Sundvall J, Saarikoski L, Peltonen M and Tuomilehto J. 2008. High prevalence of obesity, central obesity and abnormal glucose tolerance in the middle-aged Finnish population. *BMC Public Health* **8**: 423.

Sharma S, Bakshi R, Tandon VR and Mahajan A. 2008. Postmenopausal obesity. *Journal of Medical education and Research* **10(3):**105-106.

Shukla HC, Gupta PC, Mehta HC and Hebert JR. 2002. Descriptive epidemiology of body mass index of an urban adult population in western India. *J Epidemiol Community Health* **56**: 876-880.

Sidik SM and Rampal L. 2009. The prevalence and factors associated with obesity among adult women in Selangor, Malaysia. *Asia Pac Fam Med* **8**: 2.

Sinaiko AR, Lauer RM and Sanders SP. 2001. End points for cardiovascular drug trials in pediatric patients. *Am Heart J* **142**: 229-232.

Singhal V. 2009. Bariatric surgery-an ultimate solution for morbid obesity, worldwide. In: Sinha,
R. and Kapoor, S. (eds). *Obesity-A Multidimentional Approach to Contemporary Global Issue*.
pp 332-334. Dhanraj book house, New Delhi, India.

Skurk T, Lee YM, Nicuta-Rolfs TO, Haastert B, Wirth A and Hauner H. 2004. Effect of the angiotensin II receptor blocker candesartan on fibrinolysis in patients with mild hypertension. *Diabetes Obes Metab* **6**: 56-62.

Subramanian SV and Smith GD. 2006. Patterns, distribution, and determinants of under- and overnutrition: a population-based study of women in India. *Am J Clin Nutr* **84**: 633-640.

Tesfaye F, Nawi NG, Van Minh H, Byass P, Berhane Y, Bonita R and Wall S. 2007. Association between body mass index and blood pressure across three populations in Africa and Asia. *J Hum Hypertens* **21**: 28-37.

Toth MJ, Tchernof A, Rosen CJ, Matthews DE and Poehlman ET. 2000. Regulation of protein metabolism in middle-aged, premenopausal women: roles of adiposity and estradiol. *J Clin Endocrinol Metab* **85**: 1382-1387.

Wang H, Cao J, Li J, Chen J, Wu X, Duan X, Huang J and Gu D. 2010. Blood pressure, body mass index and risk of cardiovascular disease in Chinese men and women. *BMC Public Health* **10**: 189.

Weiner JS and Lourie JA. 1981: Practical Human Biology. New York: Academic Press, Inc.

Wharton CM. 2009. Obesity, Society, and Health In: Sinha, R. and Kapoor, S. (eds). *Obesity-A Multidimentional Approach to Contemporary Global Issue*. pp 143-151. Dhanraj Book House. New Delhi, India.

WHO. 2000. *The Asia- Pacific Perspective. Redefining Obesity and its Treatment*. International Diabetes Institute. Health communication Australia Pvt. Ltd., Geneva.

WHO. 2006. <u>"Definition and Diagnosis of Diabetes Mellitus and Intermediate</u> <u>Hyperglycemia</u>" (pdf).

http://www.who.int/diabetes/publications/Definition%20and%20diagnosis%20of%20diabetes_n ew.pdf, Retrieved 2007-02-20.

WHO. 2003. Diet, nutrition and prevention of chronic diseases: report of a joint WHO/FAO Expert Consultation. WHO Technical Report Series number 916. Geneva.

Wofford MR and Hall JE. 2004. Pathophysiology and treatment of obesity hypertension. *Curr Pharm Des* **10**: 3621–3637.