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# The Obesity Pandemic - Implications for Pakistan

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## Abstract

**Background:** Adverse health outcomes are associated with overweight and obesity. In February 2000, the WHO Regional Office for the Western Pacific, the International Association for the Study of Obesity and the International Obesity Task Force published provisional recommendations for adults for the Asia-Pacific region:

overweight at Body Mass Index (BMI)>23 and obesity at BMI >25.

**Methods:** Data from the National Health Survey of Pakistan, 1990-94 were reanalyzed using BMI cut-offs recommended for Asians to reassess prevalence of overweight and obesity in the adult Pakistani population.

**Results:** Prevalence of obesity (BMI>25) 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 olds, prevalence was 11% for men and 19% for women in rural areas, and 23% and 40% in urban areas for men and women, respectively. Obesity prevalence was directly associated with SES, regardless of residence.

**Conclusion:** In South Asia, including Pakistan, social and environmental changes are occurring rapidly, with increasing urbanization, changing lifestyles, higher energy density of diets, and reduced physical activity. The coexistence of underweight in early life with obesity in adults may presage both a higher prevalence and incidence for noncommunicable diseases (NCDs) such as hypertension and diabetes. Use of BMI >23 for overweight, and BMI >25 for obesity, may provide a more accurate determination of the health of Pakistanis, especially in those with more than one risk factor for NCDs (JPMA 52:342; 2002).

## Introduction

Overweight denotes the presence of excess body weight. Obesity denotes the presence of excess body fat. All obese persons are overweight, but all overweight persons are not necessarily obese as excess body weight may arise from muscle, bone or body water content. Adverse health outcomes associated with overweight and obesity range from increased risk of mortality to non-fatal debilitating disease<sup>1</sup>. Obesity is a major risk factor for cardiovascular disease (CVD) and Type 2 diabetes mellitus (DM) and in the presence of other risk factors for noncommunicable diseases (NCDs) such as smoking, hypertension, elevated blood cholesterol, has a multiplicative effect<sup>2</sup>. In westernized societies or segments of a population, cultural values idealize “thinness”, especially in women, resulting in discrimination and stigmatization of the obese with associated psychosocial problems. The mechanisms regulating body weight are complex, influenced by diverse factors: physiological, societal, environmental, genetic and behavioral. None of these is completely understood. At the individual level, nutritional, metabolic, hormonal and neuronal signals are integrated within the brain to produce changes in behavior (eating, physical activity) and body metabolism (through the autonomic nervous system and hormonal responses) so as to maintain energy balance<sup>1</sup>. Observations of obesity clustering in families led to an early interest in genetic causes for the condition. About 200 different genes or loci have been linked to obesity in humans<sup>3</sup>. However, the pattern of inheritance of obesity suggests that the effect is polygenic, with each variant of many different genes making a small

difference in effect<sup>4</sup>.

Negative health effects of overweight and obesity are influenced by body weight itself, magnitude of weight gain, body fat distribution, and age at which weight gain occurs. Ethnic, gender and age differences exist in body fat deposition<sup>5</sup>. In South Asians, there is a propensity for abdominal fat deposition compared to Europeans<sup>1,6,7</sup>. Also, men have a greater tendency towards abdominal obesity when compared to premenopausal women who deposit fat preferentially in lower body fat depots; at later ages, this difference disappears. Abdominal obesity can be typed as subcutaneous or visceral. Prospective studies have demonstrated that a high accumulation of abdominal adipose tissue, especially visceral type, is important in the development of insulin resistance and glucose intolerance, and is a better predictor of CVD and Type 2 DM than generalized obesity<sup>8-10</sup>.

### **Measurement Issues**

The anthropometric indices body mass index (BMI), waist circumference (WC) and waist-to-hip ratio (WHR), are the simplest and most common methods used for determining overweight and obesity. The BMI (weight (kg)/height squared (m<sup>2</sup>), adjusts weight for height but is more a measure of excess weight versus excess adiposity. The WHR (waist (cm)/hip(cm) is an indicator of body fat distribution and body shape<sup>1,6</sup>. The WC (cm) has the best anthropometric correlation with the amount of visceral abdominal fat<sup>9</sup>. More sophisticated methods exist for measuring adiposity and body fat distribution such as underwater weighing, bioelectrical impedance, computer tomography, ultrasound, and magnetic resonance imaging. Their use is restricted to research and clinical situations as they are complex and expensive.

In 1997 the World Health Organization (WHO) recommended the BMI for measurement of overweight and obesity in adults owing to its: validity in relation to morbidity and mortality outcomes, simplicity, widespread acceptability, and robust nature<sup>1</sup>. These attributes of the BMI were determined largely from studies in Europeans and North Americans, and classified overweight as BMI >25 and obesity as BMI >30. In February 2000, the WHO Regional Office for the Western Pacific, the International Association for the Study of Obesity, and the International Obesity Task Force published provisional recommendations for adults for the Asia-Pacific region: overweight at BMI >23 and obesity at BMI >27 (Table).

**Table: Suggested BMI and WC measurements to determine risk of co-morbidities in Asians (7).**

Classification	BMI (kg/m <sup>2</sup> )	Risk of co-morbidities	
		Waist circumference	
		< 90 (men) <.80 (women)	≥ 90 (men) ≥80 (women)
Underweight	< 18.5	Low (but increased risk of other clinical problems)	Average
Normal range	18.5 - 22.9	Average	Increased
Overweight	≥ 23		
At risk	23 - 24.9	Increased	Moderate
Obese I	25 - 29.9	Moderate	Severe
Obese II	≥30	Severe	Very severe

The recommended lower cut-points for BMI for Asians compared to Europeans are based on studies demonstrating increased risk of co-morbidities at lower BMIs in Asians, who tend to accumulate abdominal fat at lower BMIs<sup>7</sup>. The Table also shows WC cut-points that could be used in conjunction with BMI to improve determination of health status. A higher WC (an indicator of amount of abdominal fat) for a given BMI value implies a higher level of risk; this applies to both sexes. Similar classification schemes have been suggested for Europeans<sup>1</sup>.

While the suggested cut-points for both the BMI and WC are based on limited studies in Asians, there is increasing support for the use of BMI together with WC to more accurately determine an individual's health risk regardless of ethnicity, gender or age,<sup>11,12</sup>.

For children and adolescents, an internationally agreed classification system is more challenging as body height and body composition change at different rates across populations during the different stages of maturation and growth<sup>13</sup>. Also, fewer studies have been conducted in children as compared to adults to determine criteria for distinguishing children as overweight or obese based on morbidity and mortality outcomes. In the absence of outcome-based criteria for children and adolescents, a statistical approach is often adopted. This entails classifying the individual's weight relative to a selected percentile of a reference population based on age, sex, ethnicity, or other group characteristics.

#### **Factors influencing Weight Gain**

The rapid increase in the prevalence of overweight and obesity in both developed and developing countries indicates that the trend is largely due to social, environmental and behavioral changes, rather than changes in hereditary factors<sup>1,14,15</sup>. Modernization and globalization have had both positive and negative effects on populations. Increasing urbanization, changes in traditional family structures, a more mechanized and computerized workplace, globalization of world markets, and economic

transition directly or indirectly affect dietary and physical activity patterns, either through government policy, advertising and media influences, and/or changes in lifestyle. For the majority of persons it is the interaction between genetic factors (which predispose to development of obesity) and an adverse environment (which promotes it) which is responsible for weight gain. However, the role of an individual's genotype and how it interacts with biological, environmental and other factors to increase susceptibility to weight gain is uncertain.

Historically, in developed countries, obesity was associated with affluence; over the last 50 years, however, an inverse association between socioeconomic status (SES) and prevalence of obesity has been observed, particularly in women compared to men<sup>16</sup>. Over 50% of adults in the USA are overweight (BMI  $\geq$ 25) and more than one-fifth obese (BMI  $\geq$ 30)<sup>17</sup>, with similar prevalence rates in the UK<sup>18</sup> and somewhat lower figures in Canada<sup>19</sup>. The nature of the relationship between obesity and SES in developed countries is thought to be bidirectional i.e. lower SES may promote development of obesity; and, the presence of obesity may result in a lower SES. Alternatively, obesity and SES could share common determinants<sup>20</sup>. Other forces, e.g. cultural or traditional influences, may vary by gender, age and population subgroup with different effects on resulting body size and shape. The inverse association between obesity and SES in developed countries is consistent with that observed for other diseases such as CVD. More adverse patterns for CVD outcomes and risk factors (physiological, behavioural, lifestyle, psychosocial) exist for lower SES than higher SES strata<sup>16</sup>.

In developing countries, the demographic and epidemiologic transitions are taking place with varying rates of progress<sup>21</sup>. These countries are also undergoing the nutrition transition, characterized by a change in diet traditionally high in pulses, grains and carbohydrates to foods that are more energy-dense and higher in fats and refined sugars, along with larger quantities of animal protein<sup>22</sup>. The result is a "double burden" arising from health problems associated with both under- and over-nutrition. As socioeconomic conditions of developing countries improve, there is proportional decrease in the population who are underweight and an increase in those who are overweight, shifting the population mean BMI to a higher value. In these countries, obesity is still seen as a sign of prosperity and there is a direct association between obesity and SES.

### **The Situation in Pakistan**

In South Asia there is a growing epidemic of NCDs including hypertension, diabetes, CVD and chronic renal failure associated with increasing levels of high blood pressure, elevated blood cholesterol, obesity, smoking and physical inactivity. Pakistan currently ranks 8th worldwide in terms of diabetes caseload; this is projected to climb to 4th place by the year 2025 (after India, China and the USA) with caseload increasing at more than twice the rate of population growth<sup>23</sup>. Hypertension caseload will also increase but not as steeply as for diabetes<sup>23</sup>. However, as other countries in transition, it still copes with a heavy burden from child and maternal morbidity and mortality, and conditions associated with infection and nutritional deficiencies.

The National Health Survey of Pakistan (NHSP) was conducted 1990-94; participants were stratified according to sex, age, residence, and SES<sup>24</sup>. From the published report of the NHSP, obesity was not distinguished from overweight; persons with a BMI  $>$ 25 were categorized as "overweight/obese". Other studies using data from the NHSP have also not distinguished between overweight and obesity<sup>25,26</sup>. However, previous results and conclusions can be re-examined using the current recommended cut-point of BMI  $>$ 25 to define obesity among Pakistanis.

Overall, prevalence of obesity (BMI  $>$ .25) in adults was consistently higher in women versus men regardless of age group and residence. Within the age group 25-44 years, obesity prevalence was 2.4 times higher in rural versus urban areas for men (9% versus 22%) and 2.6 times higher for women (14% versus 37%), as shown in Figure 1.

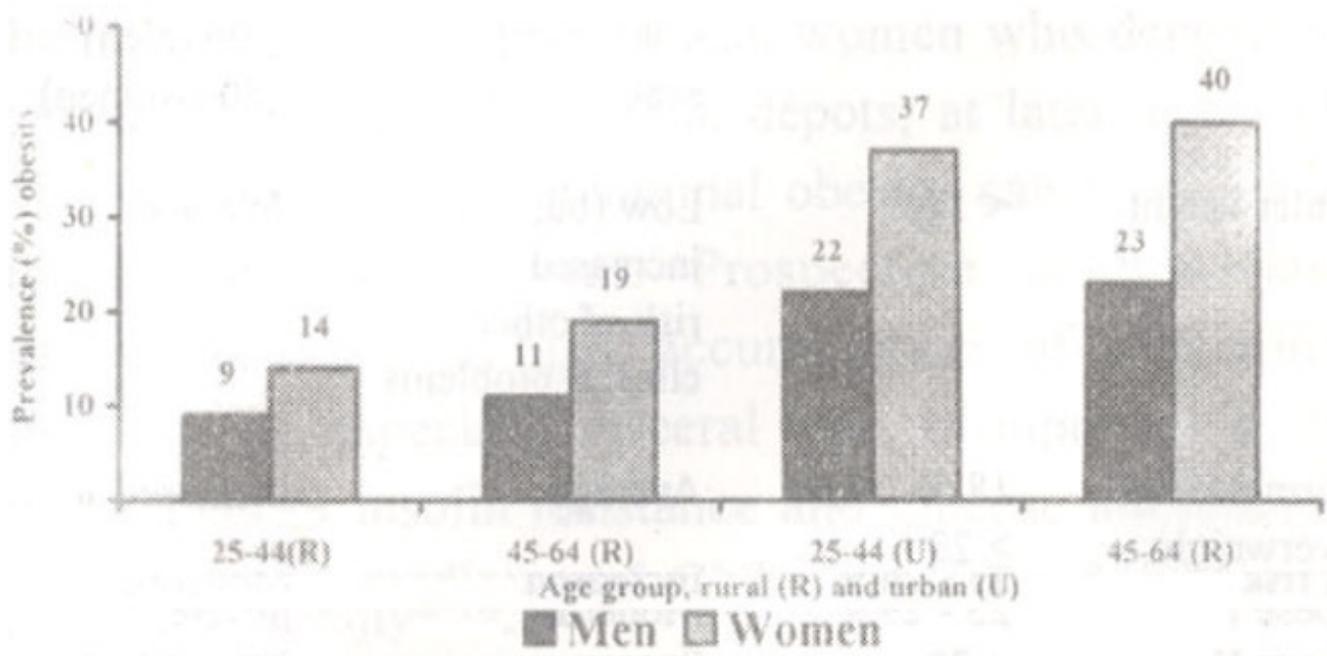


Figure 1. Prevalence of obesity (BMI $\geq$ 25) by age group and residence, for men and women.

For those 45-64 years, obesity prevalence was 2.1 times higher in urban areas compared to rural areas for both sexes. Prevalence was twice as high in urban women compared to urban men: about 40% of women 25-64 years in urban areas are obese, compared to approximately 20% of men. There was a direct association between obesity prevalence and SES in both rural and urban areas, with a greater proportional increase between SES groups in rural versus urban areas (Figure 2).

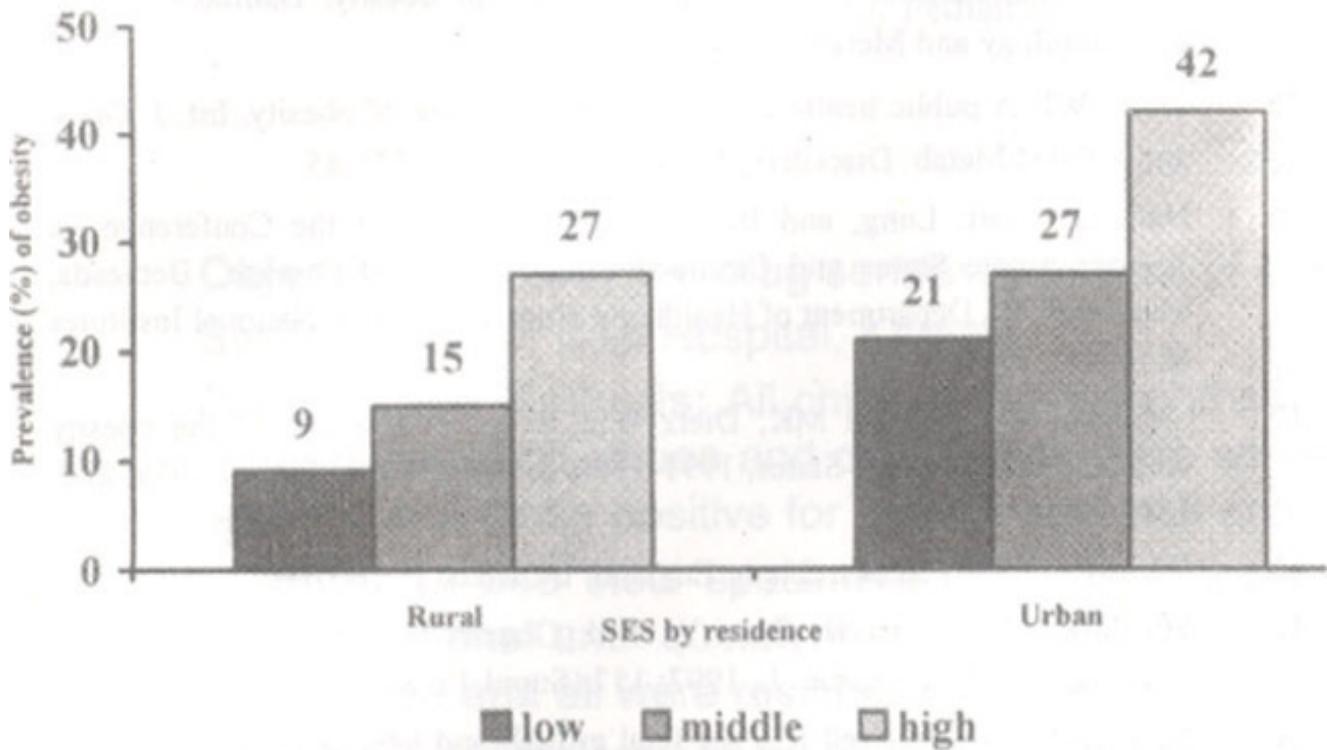


Figure 2. Prevalence of obesity (BMI  $\geq$  25) by SES and residence, for 25-64 years old.

Looking at younger Pakistanis, 40% of 15-24 year olds were categorized as underweight (BMI <18.5). Underweight was more common in rural than in urban areas, and was inversely associated with SES. For children under 5 years (and using WHO criteria), over one third of Pakistani children were stunted, over 14% were wasted and more than one in three children were underweight. These results applied to both urban and rural areas, regardless of sex.

Other related findings from the NHSP include prevalence of high blood pressure in adults around 18%. In terms of dietary patterns, 43% of urban and 17% of rural Pakistanis ate meat more than once per week<sup>27</sup>. Urban residents ate more potatoes, rice and milk compared to rural residents, who ate more pulses, curd and eggs. About 95% of the total sample consumed oil or butter/ghee daily. Thirteen percent of adults had an elevated blood cholesterol (random blood cholesterol >200 mg/dl); women generally had higher levels than men. Overall, 34% of men and 13% of women in Pakistan used tobacco regularly in some form.

Using data from population based surveys conducted by the Diabetes Association of Pakistan and the WHO Collaborating Centre for Diabetes, Pakistan diabetes prevalence is 11%, the highest in South Asia compared to results from similar surveys in other countries<sup>23</sup>.

## Discussion

Obesity is emerging in Pakistan as a public health problem even as it attempts to cope with more traditional problems of undernutrition and infectious diseases. Pakistan is similar to other countries undergoing a number of transitions simultaneously, where obesity affects first urban middle-aged women; with economic development, increasing urbanization and lifestyle changes (including diet and physical activity), obesity then occurs in men and younger women<sup>28</sup>. In Pakistan, higher prevalence of the CVD risk factors high blood pressure, obesity and elevated blood cholesterol are found in the

higher SES groups. This is in direct contrast to developed countries where there has been a reversal of the SES gradient with respect to CVD risk factors in the last century, and prevalence is now higher in the lower SES groups. The notable exception in Pakistan is smoking, which is inversely associated with SES<sup>25</sup>. Again, this reflects the complex and transitional nature of the country.

Using the WHO classification scheme for Asians, the prevalence of obesity in urban women 25-64 years is alarmingly high, around 40%, and is comparable to figures reported from more industrialized countries. Prevalence figures for overweight from the NHSP data were calculated using BMI  $\geq 25$ . Revised calculations using the recommended lower cut-point of BMI  $>23$  for Asians would classify a proportion of the adult Pakistani population as overweight previously considered normal weight. This implies that the health burden from overweight and obesity in Pakistan is currently underestimated. Standardized use of the lower cut-points would promote a change in perception of what constitutes a “healthy” body size, at least initially among Pakistani health professionals. A good example of how cultural values influence perception of body weight is demonstrated by the Arabic word for “health” which is synonymous with “weight”<sup>29</sup>. Also, the use of the lower cut-points should allow a more accurate determination of health risks associated with excess body fat, especially when used in conjunction with the WC measurement, which is a better indicator of abdominal obesity compared to the BMI.

In February 2001 the First World Congress on The Fetal Origins of Adult Disease was held in Mumbai. There is growing evidence in both developed and developing country that small size at birth or in infancy is associated with an increased risk of adverse health outcomes in adulthood, in particular high blood pressure and impaired glucose tolerance, but also Type 2 DM, hypertension and CHD<sup>30-33</sup>. The association is thought to result from impaired fetal growth at critical periods of fetal development, providing a stimulus which leads to changes in birth size and “programs” the body through altered homeostatic mechanisms towards biological risk factors for NCDs and increased susceptibility to disease later in life. One model of CVD etiology arising from this concept is the “thrifty phenotype” hypothesis<sup>33</sup>. This proposes that as an adaptation to undernutrition in fetal life, permanent metabolic and endocrine changes occur which would be beneficial if nutrition remained scarce. If nutrition becomes plentiful, however, these changes predispose to obesity and impaired glucose tolerance and later susceptibility to CVD.

The Metabolic Syndrome refers to the clustering of CVD risk factors such as abdominal obesity, glucose intolerance, hypertension and dyslipidemia, (although there is no consistently used definition in the literature)<sup>1,34,35</sup>. It is suggested that thrifty genes which ensured optimal storage of energy during famine, could contribute to the phenotype of the Metabolic Syndrome<sup>36</sup>. Variants in a number of candidate genes influencing fat and glucose metabolism together with environmental triggers, such as improved nutrition, may allow expression of genetic influences and increase susceptibility to the Syndrome - the “thrifty genotype” hypothesis.

It is still debatable as to what is (are) the predominant model(s) for obesity, Type 2 DM, CVD and other related adult diseases in South Asia, i.e. thrifty genotype, thrifty phenotype, or other possibilities and at what stage and timing in the life cycle stimuli are most critical. What is clear is that social and environmental changes are occurring rapidly in the region, with increasing urbanization, changing lifestyles, higher energy density of diets and reduced physical activity patterns.

Pakistan is an example of a South Asian country challenged by these types of changes. Population-based surveys clearly demonstrate the “double burden” of disease the country is coping with. For hypertension and diabetes, caseloads are expected to double within the next 30 years based on current prevalence figures; many will go undiagnosed and/or uncontrolled. In terms of nutrition, a high prevalence of underweight in younger life coexists with obesity in later life. Such a scenario would predict not only a higher prevalence but also increasing incidence for diseases such as hypertension, Type 2 DM and obesity if evidence continues to grow regarding the association between small size at

birth or in infancy and the development of NCDs in adulthood.

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## References

1. World Health Organization. Obesity: Preventing and managing the global epidemic. Report of a WHO consultation on Obesity. Geneva, WHO, 1998.
2. Blackburn H, Luepker R. Heart disease. In: Last, SM (ed). Maxcy-Rosenau public health and preventive medicine. 12th ed. Norwalk, Conn. Appleton-Century-Crofts, 1986, pp. 1159-93.
3. Lau DCW. Call for action: preventing and managing the expansive and expensive obesity epidemic. *Can. Med. Assoc. J.*, 1999;160:503-5.
4. Sorensen TIA, Soren ME. Obesity genes: identifying single genes involved in polygenic inheritance is not easy. *Br. Med. J.*, 2001; 322: 630-3 1.
5. Kumanyika SK. Special issues regarding obesity in minority populations. *Ann. Int. Med.*, 1993; 119: 650-54:
6. McKeigue PM. Metabolic consequences of obesity and body fat pattern: lessons from migrant studies. In: Chadwick DJ, Cardew OC (eds). *The origins and consequences of obesity*. Chichester, Wiley, 1996, pp. 54-67 (Ciba Foundation Symposium 2001).
7. World Health Organization, Regional Office for the Western Pacific, International Association for the Study of Obesity. International Obesity Task Force. *The Asia-Pacific perspective: redefining obesity and its treatment*. Melbourne, Health Communications Australia, 2000.
8. Lean MEJ, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management. *Br. Med. J.*, 1995; 311: 158-61.
9. Despres JP, Lemieux I, Prud'homme D. Treatment of obesity: need to focus on high risk abdominally obese patients. *Br. Med. J.*, 2001; 322: 716-20.
10. Lamarche B. Abdominal obesity and its metabolic complications: implications for the risk of ischaemic heart disease. *Coron. Artery Dis.*, 1998; 9: 473-81.
11. Singh RB, Mor H, Chen J, et al. Recommendations for the prevention of coronary artery disease in Asians: a scientific statement of the International College of Nutrition. *J. Cardiovasc. Risk*, 1996; 3: 489-94,
12. Seidell JC. Obesity, insulin resistance and diabetes - a worldwide epidemic. *Br. J. Nutr.*, 2000;83:S5-8.
13. Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics*, 1998;101: S497-504.
14. Hodge AM, Zimmet PZ. The epidemiology of obesity. *Baillieres clinical endocrinology and Metab.*, 1994; 8: 577-99.
15. James WP. A public health approach to the problem of obesity. *Int. J. Obes. and Related Metab. Disorders*, 1995; 19(Suppl. 3): S371-45.
16. National Heart, Lung, and Blood Institute. Report of the Conference on Socioeconomic Status and Cardiovascular Health and Disease.. Bethesda, Maryland: US Department of Health and Human Services, National Institutes of Health, 1995.
17. Mokdad AH, Serdula MK, Dietz WH, et al. The spread of the obesity epidemic in the United States, 1991-1998. *J. Amer. Med. Assoc.*, 1999; 282: 1519-22.
18. White C. Obesity rates treble in England. *Br. Med. J.*, 2001; 322: 450.

19. Macdonald S, Reeder B, Chen Y, et al. Obesity in Canada: a descriptive analysis. *Can. Med. Assoc. J.*, 1997; 157 (Suppl 1): S3-9.
20. Schroeder DG, Martorell R. Poor fetal growth and later obesity and chronic disease. In: Pena M, Bacallao J (eds). *Obesity and poverty*. Washington, DC: Pan American Health Organization, 2000.
21. World Health Organization. *The World Health Report 1998*. Geneva: WHO, 1998.
22. Popkin BM. A review of dietary and environmental correlates of obesity with emphasis on developing countries. *Obesity Res.*, 1995; 3: S145-53.
23. White F, Rafique G, Azam I, et al. Diabetes prevalence and projections for Pakistan, and implications of the detection and management of hypertension. 4th International Symposium, Pakistan Hypertension League, Quetta, Pakistan. October 8, 2000.
24. Pakistan Medical Research Council. *National Health Survey of Pakistan*, Islamabad. Network Publication Service, 1998
25. Pappas GA, Gergen PJ. Health status of the Pakistani population: a health profile and comparison with the United States. *Am. J. Public Health*, 2001; 91:93-97,
26. Nanan D, White F. The National Health Survey of Pakistan. review and discussion of report findings pertaining to selected risk factors for cardiovascular disease. *ProCOR Digest*, 1999; 99:6-11.
27. Nanan D, White F. Hypertension in Pakistani women. *Can. S. Cardiol.*, 2000; 16 (Suppl B) 28: B23-B24.
28. James WP, Ralph A. New understanding in obesity research. *Proc. Nutr. Soc.*, 1999; 58: 385-93.
29. Khoury S. A cultural approach to diabetes management in the Middle East. In: *Diabetes Voice*, 2001; 1:23-25.
30. Leon DA. Commentary: getting to grips with fetal programming - aspects of a rapidly evolving agenda. *Int. J. Epidemiol.*, 2001; 30:96-98.
31. Harding SE. The nutritional basis of the fetal origins of adult disease. *Int. J. Epidemiol.*, 2001; 30: 15-23.
32. Terry MB, Susser E. Commentary: the impact of fetal and infant exposures along the life course. *Int. J. of Epidemiol.*, 2001; 30: 95-96.
33. Schroeder DG, Martorell R. Poor fetal growth and later obesity and chronic disease. In: Pena M, Bacallao J. (eds). *Obesity and poverty*. Washington, DC: Pan American Health Organization, 2000.
34. Timar O, Sestier F, Levy E. Metabolic Syndrome X: a review. *Can. J. Cardiol.*, 2000; 16:779-89.
35. Groop L. Genetics of the metabolic syndrome. *Br. J. Nutr.*, 2000; 83: S39-48.
36. Stern MP, Bartley M, Duggirala R, et al. Birthweight and the metabolic syndrome: thrifty phenotype or thrifty genotype? *Diabetes Metab. Res. Rev.*, 2000; 16: 88-93.