

Thematic Article

Cardiovascular risk in the Asia–Pacific region from a nutrition and metabolic point of view: abdominal obesity

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The level of obesity within the Pacific Islands is extremely high and so is the prevalence of weight-related morbidity and mortality. In contrast, the level of obesity, as defined by the standard WHO classification, remains relatively low in most Asian countries, yet rates of obesity-related disease, such as diabetes and cardiovascular disease are increasing rapidly. Many Asian races appear to be susceptible to the development of excessive abdominal fatness, even at low levels of body mass index (BMI). In addition, the health consequences of weight gain appear to occur at much lower levels of BMI and are more intense than in those of European origin. The exact reasons for these ethnic variations in the development of coronary heart disease (CHD) remain unclear. It is likely that genetic differences contribute to this variation in CHD risk, but different dietary and physical activity patterns may also play a role. The advent of modernisation has resulted in marked changes in the level of physical activity and the food supply available throughout the Asia–Pacific region. There has been a shift towards higher intakes of fats (particularly animal fats and vegetable oils) and sugars. Urbanisation and occupational restructuring have reduced daily physical activity levels. As a result, the population mean BMI and consequent illness is increasing in many countries within the region. Recent studies have shown that infants who were undernourished in utero and then born small have a greater risk of developing abdominal obesity and related morbidity as adults. As undernutrition coexists with overnutrition throughout the Asia–Pacific, focusing efforts to improve nutrition during pregnancy will need to be combined with programs to prevent weight gain in adults if CHD and other chronic diseases are to be effectively tackled in the region.

Keywords: abdominal obesity, coronary heart disease risk, modernisation, nutritional programming, visceral fat.

The global problem of obesity

Obesity can now be considered one of the major public health problems facing the world today. Prevalence rates are increasing in all parts of the world, both in affluent western countries and in poorer nations. Men, women and children are affected. Indeed, overweight, obesity and health problems associated with them are now so common that they are replacing the more traditional public health concerns, such as undernutrition and infectious diseases as the most significant contributors to global ill-health.¹ In 1995, the excess adult mortality attributable to overnutrition was estimated to be about 1 million deaths, double the 0.5 million attributable to undernutrition.²

At the physiological level, obesity can be defined as a condition of 'abnormal or excessive fat accumulation in adipose tissue to the extent that health may be impaired'.¹ However, it is difficult to measure bodyfat directly and so surrogate measures such as the body mass index (BMI) are commonly used to indicate overweight and obesity in adults. Additional tools are available for identification of individuals with increased health risks because of 'central' fat distribution, and for the more detailed characterisation of excess fat in special clinical situations and research.

In the new graded classification system developed by the World Health Organization (WHO), a BMI of 30 kg/m² or above denotes obesity (Table 1). There is a high likelihood that individuals with a BMI at or above this level will have excessive bodyfat. However, the health risks associated with

overweight and obesity appear to rise progressively with increasing BMI from a value below 25 kg/m². It has been demonstrated that there are benefits to having a measurement nearer 20–22 kg/m², at least within industrialised countries. To highlight the health risks that can exist at BMI values below the level of obesity, and to raise awareness of the need to prevent further weight gain beyond this level, the first category of overweight included in the new WHO classification system is termed 'preobese' (BMI 25–29.9 kg/m²).

Obesity in the Asia–Pacific region

From a nutrition perspective, research and policy in many Asian countries has focused on undernutrition. However, there are clear indications that a number of these countries are now beginning, or are already experiencing, high levels of overweight and obesity. Urban China, urban Thailand, Malaysia and the Central Asian countries that were members of the Soviet Union before 1992 (such as Krygystan) are all examples. Overweight is also becoming a serious problem in urban India, most notably in the upper-middle class. The situation in China and India is further complicated by the fact

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Table 1. Classification of overweight in adults according to BMI

Classification	BMI (kg/m ²)	Risk of comorbidities
Underweight	< 18.5	Low (but risk of other clinical problems increased)
Normal range	18.5–24.9	Average
Overweight	≥ 25.0	
Pre-obese	25.0–29.9	Increased
Obese class I	30.0–34.9	Moderate
Obese class II	35.0–39.9	Severe
Obese class III	≥ 40.0	Very severe

BMI, body mass index. Taken from Reference 1.

that chronic energy deficiency is still a major problem for large parts of the population

In Japan, although overall rates of obesity remain below 3%, prevalence increased by a factor of 2.4 in the adult male population and by a factor of 1.8 in women aged 20–29 years.¹ The level of obesity among Chinese adults remains low, but the marked shifts in diet, activity and overweight suggest that major increases in overweight and obesity will occur. During the most recent period of the national China Health and Nutrition Survey (CHNS), an ongoing longitudinal survey of eight provinces in China, these data show a consistent increase in adult obesity in both urban and rural areas.³ Changes in diet and activity patterns are rapid in urban residents of all incomes, but are even more rapid in middle and higher income rural residents.

Data from Thailand suggest that obesity rates are increasing, at least in affluent urban populations. In 1991, 3% of men and 3.8% of women were classified as obese in a small study of 66 men and 453 women aged 19–61 years. These figures are higher than those from a similar study in 1985 conducted among a larger group of affluent urban Thais aged 35–54 years.⁴

Trend data from the western Pacific Islands indicate that obesity levels are not only high in these populations, but also that the prevalence of obesity continues to increase considerably in each island.

Abdominal obesity in the Asia–Pacific region

Very few countries in the region have undertaken a systematic collection and analysis of an indicator of abdominal obesity such as waist-to-hip ratio or waist circumference. Ko *et al.* collected waist circumference measures on 1513 Hong Kong Chinese and comparing this data against the WHO criteria for Caucasians found 4.5% of men and 22.5% of women were above the at-risk level.⁵ A recent survey of waist-to-hip ratio in French Polynesia revealed that 47% of men and 70% of women exceed the cut-offs of 1.0 and 0.9, respectively.⁶

Cardiovascular risk and abdominal obesity

Obese individuals have long been known to be at greater risk of cardiovascular disease (CVD). However, early epidemiological studies were only able to demonstrate modest associations between obesity and CVD, especially in younger groups. In short-term studies, the relationship between obesity and CVD mortality was difficult to detect. However, an analysis of the Framingham study which involved a follow-up of over 20 years was able to show a stronger association between

bodyweight and coronary heart disease (CHD).⁷ More recent and better controlled studies have indicated that obesity predisposes to greatly increased CVD morbidity and mortality. In the US Nurses Health Study, a prospective study of 116 000 women, CVD rates among the obese were four times that of the leanest group.⁸ As with subjects from the Framingham study, the risk of CVD rose steady from a BMI of 20 kg/m². Similar results were also found in men in the British regional Heart Study where the level of most CVD risk factors increased steadily with increasing BMI.⁹

A number of other studies have shown a significant relationship between obesity and hypertension, hypercholesterolaemia, low high-density lipoprotein (HDL) cholesterol, hypertriglyceridaemia, hyperinsulinaemia, diabetes mellitus type II, and many clotting and haemostatic conditions.¹⁰ Some of the strongest and most consistent associations have been found for obesity and hypertension and obesity and hypertriglyceridaemia. Between one-quarter and one-half of all obese persons suffer from hypertension and risk estimates suggest that as much as 75% of all hypertension in the US can be attributed to obesity.¹¹ The precise mechanism between rising bodyweight and hypertension is not well understood, but it appears to be associated with renal dysfunction. Thus, the impact of bodyweight may be increased when combined with adverse dietary factors, such as alcohol and sodium/potassium intake.

Over the last two decades, important new information concerning the relationship between obesity and CVD has emerged, which highlights the importance of weight gain and duration of overweight and bodyfat distribution as more important determinants of CVD risk than BMI alone. Both the Health Professionals study and the Nurses Health Study have shown that there is an association between weight gain and CVD, which is independent of total BMI. Those who had gained the most weight in adulthood and who had the longest duration of being overweight, had the highest level of risk for diabetes mellitus type II and CVD.⁸

However, studies of the effects of a central distribution of fat in obesity have demonstrated the strongest associations with CVD mortality and morbidity. Early studies by Larsson *et al.* and Lapidus *et al.* demonstrated a strong association between waist-to-hip ratio and myocardial infarction, stroke and death.^{12,13} Subsequent studies have indicated that it is a high level of intra-abdominal or visceral fat which carries the greatest health risks. Although the waist-to-hip ratios have now been shown to be a poor quantifier of total visceral fat deposits, it allows the differentiation between those obese persons with large central fat

stores and those who store fat peripherally. More recent studies have also found strong associations between central obesity and hypertension, hypertriglyceridaemia, hypercholesterolaemia, hyperglycaemia, hyperinsulinaemia, low HDL levels, diabetes mellitus type II, elevated clotting risk and hyperuricaemia.¹⁰ More importantly, the risk imposed by central obesity was independent of total BMI and was found in those who would not be classified as obese based on their BMI alone. For this reason WHO recommended that a measure of central obesity, such as waist circumference, should be collected together with height and weight as a means of ensuring weight-related health risk for individuals can be properly assessed.

Family studies have suggested that genetics play an important role in determining fat-patterning in individuals and populations. A number of studies have also found behavioural factors such as smoking, physical inactivity, different dietary patterns and excessive alcohol intake as well as psychosocial factors of anger, anxiety and depression are associated with central fat distribution.¹⁴ Bjorntorp has suggested that cortisol release associated with stress may precipitate physiological changes and imbalances which lead to visceral fat accumulation.¹⁵ Only limited work on the relationship between different dietary patterns and the development of visceral obesity has been performed in humans. Although there are clear indications of the benefits of consuming a low-fat diet in the treatment of abdominal obesity,¹⁶ the impact of total fat intake and the composition of dietary fats on visceral fat accumulation is unclear. Animal studies have shown that a high sucrose diet may lead to excessive intra-abdominal fat accumulation.¹⁷

Several mechanisms by which fat distribution may influence CVD risk have been postulated. Visceral adipose tissue is known to be more sensitive to lipolytic hormones, leading to an increased production of free-fatty acids. This can lead to insulin resistance and compensatory hyperinsulinaemia which is believed to be the underlying cause of a cluster of risk factors (glucose intolerance, hypertension, hypertriglyceridaemia, low HDL cholesterol and clotting abnormalities) known as the metabolic syndrome which leads to atherosclerosis and thrombosis risk. Recently, Deprés has also suggested small, dense low-density lipoprotein (LDL) cholesterol particles associated with this syndrome may be responsible for the greatly increased CVD risk in centrally obese individuals.¹⁶

The relationship between abdominal obesity and cardiovascular disease in the Asia-Pacific region

The level of CVD has been rising steadily throughout the Asia-Pacific region for the last two decades and it is now the most common cause of mortality.¹⁸ Recent data presented above have also indicated that the population mean BMI is increasing rapidly throughout the region, yet the level of obesity as defined by WHO (BMI = 30 kg/m²) remains relatively low. This has led many countries within Asia to ignore the problem of obesity as a public health issue and focus their scarce resources on tackling the problems of undernutrition which persist throughout the region. However, evidence is beginning to emerge which demonstrates that obesity, and in particular central obesity is a major problem throughout the region and that it is already associated with rising rates of metabolic disease.

The first indication that peoples of Asian origin were more prone to visceral obesity and CVD at low levels of BMI came from studies involving migrants from south Asia. McKeigue *et al.* found that migrants from India, Pakistan and Bangladesh who settled in England had very much higher rates of CHD than the general population even though they had a lower BMI.¹⁹ Assessment of their CVD risk factors revealed that they had all the markers of the metabolic syndrome and both men and women had a more central distribution of bodyfat for a given BMI when compared to European controls. Subsequent studies of body composition have revealed that most Asian populations have a higher bodyfat content for a given BMI than Europeans,²⁰ while the opposite is true for Pacific Islanders.²¹ Indonesians, for example, have a similar level of fat at a BMI of 27 kg/m² as a Caucasian at a BMI of 30 kg/m² and Polynesians at a BMI of 32–33 kg/m². Limited data has been published on the relationship between BMI and level of CVD risk factors in Asian populations. Work from Hong Kong⁵ and Singapore²² have indicated the presence of a high-level of CVD risk associated with a BMI of 25 kg/m² or less. Similarly, these two studies also demonstrated elevated CVD risk factors at low-levels of waist circumference, even though only a small proportion of the population would be classified within the high-risk waist circumference category using existing WHO standards. Additional work has shown similar results in Korea and Japan, but this has not yet been published outside their countries. Limited prospective studies of BMI and CVD risk in Asian populations has yet been published.

Australian Aborigines have high levels of general and abdominal obesity when assessed against the standard WHO criteria and an alarmingly high-level of CVD mortality and morbidity, although there are many other factors which are likely contributors to this high CVD risk.²³ Pacific Islanders also have high rates of obesity and CVD mortality and morbidity although little work has been done in examining the relationship of bodyweight to CVD risk.

These observations suggested that it may not be appropriate to directly apply the general guidelines for the classification and management of obesity produced at the 1997 WHO consultation on obesity to all Asian and Pacific Island populations. To respond to this issue a committee was established under the joint auspices of WHO (Western Pacific Region) and the Asia and Oceania region of IASO/IOTF. The committee examined the available information about weight status throughout the region and the association between BMI and level of weight-related ill-health. The resulting document – ‘The Asia-Pacific perspective: redefining obesity and its treatment’ presents a regional perspective on these issues and proposes specific diagnostic criteria to define overweight and obesity in Asians using BMI and waist circumference, which reflects the increased risk of disease at lower levels of body fatness (Table 2).²⁴ The document also proposes that a separate classification system be developed for Pacific Islanders but felt that insufficient evidence was available at this time to define the criteria.

The potential impact of fetal undernutrition and later obesity

In countries undergoing transition where overnutrition coexists with undernutrition, the shift in population weight status

Table 2. Proposed classification of weight by BMI and waist circumference in adult Asians

Classification	BMI (kg/m ²)	Risk of comorbidities	Waist circumferences
		< 90 cm (men)	≥ 90 cm (men)
		< 80 cm (women)	≥ 80 cm (women)
Underweight	< 18.5	Low (but risk of other clinical problems increased)	Average
Normal range	18.5–24.9	Average	Increased
Overweight	≥ 23.0		
At Risk	23.0–24.9	Increased	Moderate
Obese class I	25.0–29.9	Moderate	Severe
Obese class II	≥ 30.0	Severe	Very severe

BMI, body mass index. Taken from Reference 24.

Table 3. Subjects with high WHR (abdominal obesity) by grades of BMI in urban Indians (%)

Details	Grades of BMI	Males	Females
Underweight	< 18.5	1.8	1.75
Normal	18.5–24.9	17.8	20.0
Overweight	≥ 25	68.1	58.0

BMI, body mass index; WHR, waist-to-hip ratio. Taken from Reference 31.

has been linked to exaggerated problems of obesity and associated non-communicable diseases in adults. Recent studies have shown that infants who were undernourished in utero and then born small have a greater risk of obesity-related morbidity as adults if they gain weight.^{25,26} In particular, poor intrauterine nutrition appears to predispose some groups to abdominal obesity and results in an earlier and more severe development of comorbid conditions such as hypertension, CHD and diabetes.^{27–29} The apparent impact of intrauterine nutrition on the later structure and functioning of the body has become known as 'programming' and is often referred to as the 'Barker hypothesis,' after one of the key researchers involved in developing this concept. In many parts of Asia, women enter pregnancy malnourished and the problem of undernutrition in utero may be further exacerbated by the low-protein, rice-based diets common in the region.³⁰

The ramifications of programming are immense for countries such as India and China where a large proportion of infants are still born undernourished. If these children are later exposed to high-fat diets and sedentary lifestyles associated with economic transition, and develop into obese adults, then it is likely that they will suffer severe consequences in the form of early heart disease, hypertension and diabetes.

Central obesity is already emerging as a serious problem in India, even at low relative weight.³¹ Among non-overweight urban middle-class residents with BMI less than 25 kg/m², nearly 20% of males and 22% of females had a high waist-to-hip ratio. In overweight subjects with a BMI over 25 kg/m², abdominal obesity was found in a striking 68% of males and 58% of females (Table 3).

Modernisation has brought many social, economic and health benefits to developing countries throughout the world. However, the rapid economic growth, urbanisation and occupational restructuring has also resulted in dramatic changes in diet and physical activity levels that have led to rapid increases in population mean bodyweight. Many countries in the Asia-Pacific region are becoming reliant on non-traditional food sources and there has been a shift towards diets

higher in both animal and vegetable fats (particularly vegetable oils) and sugars.³² This has been proposed as a major factor driving the increase in bodyweight.^{1,30} Escalating levels of obesity together with alcohol abuse and cigarette smoking are associated with high rates of diabetes mellitus type II, hypertension, dislipidaemia and CVD. This has been described as the 'New World Syndrome' and is responsible for the disproportionately high rates of mortality in developing nations and among the disadvantaged ethnic minority groups in developed countries.

Conclusion

Tackling the problem of abdominal obesity in the Asia-Pacific region is one of the greatest public health challenges of the present time. Efforts must be made to restrain the rapid swings in diet and level of physical activity, which are contributing to the problem of overnutrition in adulthood. At the same time, appropriate dietary change needs to be encouraged to help avoid undernutrition in utero which may be programming children to a lifetime of chronic disease brought on by the development of abdominal obesity in later life.

References

1. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO Consultation on Obesity. Geneva 3–5 June, 1997. Geneva: World Health Organization, 1998.
2. World Health Organization. World Health Report 1998. Geneva, World Health Organization, 1998.
3. Popkin BM, Doak CM. The obesity epidemic is a worldwide phenomenon. *Nutr Rev* 1998; 56: 106–114.
4. Leelahagul P, Tanphaicchitr V. Current status on diet-related chronic diseases in Thailand. *Intern Med* 1995; 11: 28–33.
5. Ko GTC, Chan JCN, Woo J, Lau E, Yeung VT, Chow CC, Wai HP, Li JK, So WY, Cockram CS. Simple anthropometric indexes and cardiovascular risk factors in Chinese. *Int J Obesity* 1997; 21: 995–1005.
6. Ministère de la santé et de la recherche, Direction de la santé Institut Malardé. Enquête sur les maladies non transmissibles en Polynésie française. Etude de la prévalence de l'hypertension, du diabète, de la goutte et de l'obésité en relation avec les habitudes alimentaires. September – Nov 1995, Papeete, 1998 (in French).

7. Feinlab M. Epidemiology of obesity in relation to health hazards. *Ann Intern Med* 1985; 103: 1019–1024.
8. Manson J, Willett W, Stamfler M, Colditz GA, Hunter DJ, Hankinson SE, Hennekens CH, Speizer FE. Body weight and mortality among women. *N Engl J Med* 1995; 333: 677–685.
9. Thelle DS, Shaper AG, Whitehead TP, Bullock DG, Ashby D, Patel I. Blood lipids in middle aged British Men. *Br Heart J* 1983; 49: 205–213.
10. Van Gaal LF, Mertens IL. Effects of obesity on cardiovascular system and blood pressure control, digestive disease and cancer. In: Kopelman PJ, Stocks MJ, eds. *Clinical obesity*. Oxford: Blackwell Science, 1998; 205–225.
11. American Heart Association Conference Statement. Obesity: impact on cardiovascular disease. Dallas: American Heart Association, 1998.
12. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Björntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity and risk of cardiovascular disease and death: 13 year follow of participants in a study of men born in 1913. *BMJ* 1984; 288: 1401–1404.
13. Lapidus L, Bengtson C, Laarson C, Pennert K, Ryobo E, Sjöström L. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of participants in a population study of women in Gothenburg, Sweden. *BMJ* 1984; 289: 1257–1261.
14. Wing R, Mathews K, Kuller L, Meilahn E, Plantinga P. Waist to hip ratio in middle aged women. Associations with behavioural and psychosocial factors and with changes in cardiovascular risk factors. *Arterioscler Thromb* 1991; 11: 1250–1257.
15. Björntorp P. Hypothesis: visceral fat accumulation: the missing link between psychosocial factors and cardiovascular disease. *J Intern Med* 1991; 230: 195–201.
16. Deprés JP. The insulin resistance-dyslipidemic syndrome of visceral obesity: effect on patients' risk. *Obes Res* 1998; 1: 8S–17S.
17. Keno Y, Matsuzawa Y, Togunaka K, Fujioka S, Kawamoto T, Kobatake T, Tarui S. High sucrose diet increases visceral fat accumulation in VMH-lesioned rats. *Int J Obesity* 1991; 15: 205–209.
18. Western Pacific Regional Office of the World Health Organization. *Country Health Information Profiles: 1999 Revision*. Manila: World Health Organization, 1999.
19. McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991; 337: 382–386.
20. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta-analysis among different ethnic groups. *Int J Obesity* 1998; 22: 1164–1171.
21. Swinburn BA, Craig PL, Daniel R, Dent DPD, Strauss BJG. Body composition differences between Polynesians and Caucasians assessed by bioelectrical impedance. *Int J Obes* 1996; 20: 889–894.
22. Deurenberg-Yap M, Tan BY, Chew SK, Deurenberg P, van Staveren WA. Manifestation of cardiovascular risk factors at low levels of body mass index and waist-hips ratio in Singaporean Chinese. *Asia Pac J Clin Nutr* 1999; 8: 177–183.
23. O'Dea K, Patel M, Kubisch R, Hopper J, Traianedes K. Obesity, diabetes and hyperlipidaemia in a central Australian Aboriginal community with a long history of acculturation. *Diabetes Care* 1993; 16: 1004–1010.
24. Western Pacific Regional Office of the World Health Organization, The International Association for the Study of Obesity, The International Obesity TaskForce. *The Asia-Pacific perspective: redefining obesity and its treatment*. Sydney: Health Communications Australia, 2000.
25. Ravelli GP, Stein ZA, Susser M. Obesity in young men after famine exposure in utero and early infancy. *N Engl J Med* 1976; 295: 349–353.
26. Jackson AA, Langley-Evans SC, McCarthy HD. Nutritional influences on early life upon obesity and body proportions. In: Chadwick DJ, Cardew GC, eds. *The origins and consequences of obesity*. Chichester: Wiley, 1996; 118–137.
27. Law CM, Barker DJ, Osmond C, Fall CH, Simmonds SJ. Early growth and abdominal fatness in adult life. *J Epidemiol Community Health* 1992; 46: 184–186.
28. Schroeder DG, Martorell R, Flores R. Infant and child growth and fatness and fat distribution in Guatemalan adults. *Am J Epidemiol* 1999; 149: 177–185.
29. Barker DJP. Foetal undernutrition and obesity in later life. In: Guy-Grand B, Ailhaud G, eds. *Progress in obesity research: 9*. London: John Libbey, 1999; 623–625.
30. James WPT. Long term fetal programming of body composition and longevity. *Nutr Rev* 1997; 55: S31–S34.
31. Gopalan C. Obesity in Indian urban 'middle class'. *Bull Nutr Found India* 1998; 19: 1–5.
32. Drewnowski A, Popkin BM. The nutrition transition: new trends in the global diet. *Nutr Rev* 1997; 55: 31–43.