

## Obesity and the Metabolic Syndrome in Developing Countries

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**Context:** Prevalence of obesity and the metabolic syndrome is rapidly increasing in developing countries, leading to increased morbidity and mortality due to type 2 diabetes mellitus (T2DM) and cardiovascular disease.

**Evidence Acquisition:** Literature search was carried out using the terms obesity, insulin resistance, the metabolic syndrome, diabetes, dyslipidemia, nutrition, physical activity, and developing countries, from PubMed from 1966 to June 2008 and from web sites and published documents of the World Health Organization and Food and Agricultural Organization.

**Evidence Synthesis:** With improvement in economic situation in developing countries, increasing prevalence of obesity and the metabolic syndrome is seen in adults and particularly in children. The main causes are increasing urbanization, nutrition transition, and reduced physical activity. Furthermore, aggressive community nutrition intervention programs for undernourished children may increase obesity. Some evidence suggests that widely prevalent perinatal undernutrition and childhood catch-up obesity may play a role in adult-onset metabolic syndrome and T2DM. The economic cost of obesity and related diseases in developing countries, having meager health budgets is enormous.

**Conclusions:** To prevent increasing morbidity and mortality due to obesity-related T2DM and cardiovascular disease in developing countries, there is an urgent need to initiate large-scale community intervention programs focusing on increased physical activity and healthier food options, particularly for children. International health agencies and respective government should intensively focus on primordial and primary prevention programs for obesity and the metabolic syndrome in developing countries. (*J Clin Endocrinol Metab* 93: S9–S30, 2008)

**N**utritional, demographic, epidemiological, and socioeconomic transitions are occurring in many developing countries. Continuing undernutrition and escalating over nutrition has created double jeopardy of communicable and noncommunicable diseases (NCDs) (1, 2). This double burden poses apparently insurmountable health and economic challenges in resource-constrained populations.

Obesity is a natural consequence of over nutrition and sedentary lifestyle. Persistent obesity dysregulates metabolic processes including action of insulin on glucose-lipid-free fatty acid metabolism and severely affects processes controlling blood glu-

cose, blood pressure, and lipids. Thus begins a cluster of conditions; dysglycemia, dyslipidemia, hypertension, and procoagulant state, known as the metabolic syndrome (3). Data suggest that the obesity and the metabolic syndrome are immediate precursors of type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) (4–7).

Increasing burden of obesity, the metabolic syndrome, T2DM, and CVD in developing countries has created an urgent need to strategize health policies and mass intervention programs to tackle nutrition and continue efforts to manage undernutrition. Clearly these efforts require a thorough understanding of

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Abbreviations: BMI, Body mass index; CVD, cardiovascular disease; FAO, Food and Agricultural Organization; GDP, gross domestic product; HDL-C, high-density lipoprotein cholesterol; IDF, International Diabetes Federation; IFG, impaired fasting glucose; IMCL, intramyocellular triglycerides; NCD, noncommunicable disease; NCEP, ATP III, National Cholesterol Education Program, Adult Treatment Panel III; SES, socioeconomic strata; T2DM, type 2 diabetes mellitus; TFC, transnational food company; WHO, World Health Organization.

factors influencing and driving obesity and the metabolic syndrome in developing countries. In this review, we shall discuss the epidemiology of obesity and the metabolic syndrome, lifestyle and nutritional transitions, determinants, social and economic impacts, and possible solutions for prevention of obesity and the metabolic syndrome in developing countries.

Developing countries have been categorized as per the International Monetary Fund's World Economic Outlook Report, April 2008 (8). The literature search has been carried out using the terms, obesity, insulin resistance, the metabolic syndrome, diabetes, dyslipidemia, nutrition, physical activity, and developing countries, in the medical search database PubMed (National Library of Medicine, Bethesda, MD) from 1966 to June 2008. A manual search of the relevant quoted references was also carried out from the retrieved articles. Data have also been taken from nutritional surveys in different developing countries and web sites and published documents of the World Health Organization (WHO) and Food and Agricultural Organization (FAO). It is important to note that, despite elaborate literature search, data regarding obesity and the metabolic syndrome are not available from many developing countries.

### **Shift towards NCDs in developing countries: a growing burden**

With a substantially high annual rate of increase of obesity and rapid emergence of the metabolic syndrome in most developing countries, the shift in the pattern of NCDs is occurring at a faster rate than it did in the industrialized regions of the world half a century ago (9). Global prevalence of chronic diseases is projected to increase substantially over the next 2 decades in developing countries. Indeed, 60% of the global burden of chronic diseases is expected to occur in developing countries (10). CVD is already the leading cause of mortality in many developing countries (11).

In the World Health Report (1999), it was stated that in 1998, 78% of the burden of NCDs and 85% of the CVD arose from the developing low- and middle-income countries (12). Furthermore, according to projected data, chronic diseases will account for almost three quarters of all deaths worldwide by 2020 and that 71% of deaths due to CVD and 70% of deaths due to diabetes will occur in developing countries (13). In fact, there are more patients with CVD in India and China than in all the economically developed countries (14). Globally, by 2000, 171 million patients with diabetes are projected, which is expected to increase to 366 million in 2030, of which 298 million will be in developing countries (15). The number of people with diabetes is projected to double (during 2000–2030), in three of the six developing regions, including, the Middle East and North Africa, South Asia, and sub-Saharan Africa (15). India has and will continue to have the highest number of patients with diabetes in the world (16).

The resulting increase in morbidity and mortality due to obesity and consequent chronic NCDs is a matter of great concern. Between 1990 and 2020, mortality from CVD in developing countries is expected to increase by 120% for women and 137%

for men (17), which is expected to be substantially greater than from developed countries (29 and 48%, respectively) (18). A near tripling of CVD mortality in Latin America, the Middle East, and sub-Saharan Africa is expected to occur in next 2 decades (19). In India, increase in CVD mortality is expected to reach 2 million by 2010 (20).

### **Epidemiology of obesity and the metabolic syndrome in developing countries**

#### **Obesity (Table 1)**

According to the WHO estimates, the undernourished population in the world has declined and is roughly around 1.2 billion, whereas the overnourished population has increased to 1.2 billion (21). WHO data also show that, globally, there are more than 1 billion adults overweight and 300 million obese people. The problem of obesity is increasing in the developing world with more than 115 million people suffering from obesity-related problems (22). In 1998, Popkin and Doak (23) reported an increase in prevalence of obesity from 2.3 to 19.6% over the last 10-yr period in several developing countries. Obesity rates have increased 3-fold or more since 1980 in Middle East, the Pacific Islands, Australasia, and China (24).

Whereas the prevalence of obesity may not be high in many areas in some developing countries, like China, Japan, and certain African nations, it is extremely high (>75%) in other countries like urban Samoa. Even in low-prevalence countries, the prevalence is significantly high (>20%) in urban areas (23). Recently application of lower cutoff of body mass index (BMI) (Asian criteria of overweight: 23–25 kg/m<sup>2</sup> and obesity: ≥25 kg/m<sup>2</sup>) (25) has led to increase in prevalence figures in several Asian countries (26, 27).

Overall, it appears that overweight and obesity may already be more than underweight and undernutrition in many developing nations. Rural-urban differences in obesity, the metabolic syndrome, and T2DM is seen in most developing countries (28). Furthermore, whereas overweight and obesity in underprivileged people in developed countries is substantial, in developing countries rural-based people are mostly lean and have low prevalence of T2DM and CVD. However, underprivileged people residing in urban areas (mostly rural to urban migrants) show increasing prevalence of overweight/obesity and other cardiovascular risk factors (29).

#### **Abdominal obesity (Table 1)**

Abdominal obesity is an important risk factor for T2DM, the metabolic syndrome, and CVD (30–32) and is particularly prevalent in certain ethnic groups of developing countries (*e.g.* South Asians, Hispanics, *etc.*). In the International Day for Evaluation of Abdominal obesity study (33), waist circumference data from 63 countries showed highest prevalence of abdominal obesity in South Asians compared with north Europeans and other Asian ethnic groups. Of note, 30.9% of men and 32.8% of women in industrial population in India were reported to have abdominal obesity (34). Using Asian cutoffs of waist circumference, 25.7% women and 33.1% men in urban South Korea had abdominal

**TABLE 1.** Prevalence of obesity in developing countries

Author and year	Country/region and urban/rural area	Age (yr)	Sample (n)		Cutoffs of BMI (kg/m <sup>2</sup> ) or waist circumference (cm)	Prevalence of obesity (%)	
			Male	Female		Male	Female
Shapo <i>et al.</i> , 2003 (205)	Albania (Urban) <sup>a</sup>	>25	535	585	BMI ≥ 30	22.8	35.6
Monteiro <i>et al.</i> , 2001 (206)	Brazil <sup>a</sup>	>20	1971	2588	BMI ≥ 30	4.4	12.6
Zaman <i>et al.</i> , 2001 (207)	Bangladesh (rural)	>18	238	272	WC ≥ 94 (M), ≥80 (F)	2.9	16.8
Fezeu <i>et al.</i> , 2006. (36)	Cameroon (urban)	>25	1301	1530	WC ≥ 94 (M), ≥80 (F)	18.0	67.0
Du <i>et al.</i> , 2002 (208)	China <sup>a</sup>	18–49	2796	2936	BMI ≥ 25	15.3	17.1
Gu <i>et al.</i> , 2005 (40)	China <sup>a</sup>	35–74	15540 <sup>b</sup>		BMI ≥ 25	26.9	31.1
Jadue <i>et al.</i> , 1999 (209)	Chile <sup>a</sup>	24–64	1020	2100	BMI ≥ 30	15.7	23.1
Fan <i>et al.</i> , 2008 (210)	Shanghai, China (urban)	20–88	1524	2379	WC ≥ 90 (M), ≥80 (F) BMI ≥ 25	35.4 <sup>b</sup> 43.3 <sup>b</sup>	
Shi <i>et al.</i> , 2008 (211)	Jiangsu, China <sup>a</sup>	>20	2849 <sup>b</sup>		WC ≥ 90 (M), ≥80 (F)	19.5	38.2
Sabanayagam <i>et al.</i> , 2007 (212)	Chinese adults, Singapore (urban)	40–81	402	540	BMI ≥ 25	33.0	34.0
Pang <i>et al.</i> , 2008 (213)	China (rural)	>35	45925 <sup>b</sup>		BMI ≥ 25	17.6	27.3
Zhang <i>et al.</i> , 2008 (214)	China (rural)	>35	29970 <sup>b</sup>		BMI ≥ 25 BMI ≥ 30	15.1 1.2	22.1 2.2
Galal, 2002 (215)	Egypt <sup>a</sup>		1974	2909	BMI ≥ 30	20.0	45.2
Dhurandhar and Kulkarni, 1992 (216)	Western India (urban)	>15 y	791	791	BMI ≥ 30	4.8	7.8
Gupta <i>et al.</i> , 2003 (217)	North India (urban)	≥20	532	559	WC ≥ 102 (M), ≥88 (F)	21.8	44.0
Misra <i>et al.</i> , 2001 (176)	North India (urban) <sup>c</sup>	>18	170	362	BMI ≥ 25	13.3	15.6
Gupta <i>et al.</i> , 2004 (51)	North India (urban) <sup>d</sup>	Mean: 43.2 (M) 44.7 (F)	226	232	BMI ≥ 30, WC ≥ 102 (M), ≥88 (F)	20.8 34.5	32.3 55.6
Prabhakaran <i>et al.</i> , 2005 (218)	North India <sup>~</sup> (industrial population)	20–59	2935 <sup>b</sup>		BMI ≥ 25 WC ≥ 90 (M), ≥80 (F)	35.0 <sup>b</sup> 43.0 <sup>b</sup>	
Misra <i>et al.</i> , 2005 (113)	North India (urban)	38.9	640 <sup>b</sup>		WC ≥ 90 (M), ≥80 (F)	10.1	25.9
Gupta <i>et al.</i> , 2004 (219)	North India (urban)	>20	960	840	WC ≥ 102 (M), ≥88 (F)	25.6	44.0
Deepa <i>et al.</i> , 2007 (220)	South India (urban)	>20	2350		BMI ≥ 25 WC ≥ 90 (M), ≥80 (F)	43.2 56.2	47.4 35.1
Gupta <i>et al.</i> , 2007 (221)	North India (urban)	>20	532	559	BMI ≥ 25	37.8	50.3
Park <i>et al.</i> , 2006 (222)	Korea <sup>a</sup>	20–80	6824 <sup>b</sup>		WC ≥ 90 (M), ≥85 (F)	19.4	22.5
Oh <i>et al.</i> , 2004 (35)	Korea (urban)	30–80	269	505	WC ≥ 90 (M), ≥80 (F)	33.1	25.7
Grabauskas <i>et al.</i> , 2003 (223)	Lithuania <sup>a</sup>	20–64	4337	5440	BMI ≥ 30	16.2	16.0
Benjelloun, 2002 (224)	Morocco <sup>a</sup>	>20	6875	7153	BMI ≥ 25 BMI ≥ 30	25.4 4.3	45.0 16.0
Hodge <i>et al.</i> , 1996 (225)	Mauritius <sup>a</sup>		5021 <sup>b</sup>		BMI ≥ 25	26.1	37.9
			5111 <sup>b</sup>		BMI ≥ 25	35.7	47.7
Al-Lawati <i>et al.</i> , 2003 (38)	Oman (urban)	20–99	1419 <sup>b</sup>		WC ≥ 102 (M), ≥88 (F)	4.7	44.3
Dodani <i>et al.</i> , 2004 (226)	Pakistan (urban)	Mean 41.07	1147 <sup>b</sup>		BMI ≥ 25 BMI ≥ 30	52.2 <sup>b</sup> 18.5 <sup>b</sup>	
Jacoby <i>et al.</i> , 2003 (227)	Peru (urban)	>18	1163	1159	BMI ≥ 30	16.0	22.7
Jahns <i>et al.</i> , 2003 (228)	Russian Federation <sup>a</sup>	19–55	17150 <sup>b</sup>		BMI ≥ 30	7.1	21.6
			9006 <sup>b</sup>		BMI ≥ 30	10.3	19.1
Bovet <i>et al.</i> , 2006 (229)	Republic of Seychelles <sup>a</sup>	25–64	1255 <sup>b</sup>		BMI ≥ 30 BMI ≥ 25	15.0 68.3	35.2 29.3
South Africa Department of Health, 1999 (230)	South Africa <sup>a</sup>	>15	5671	8156	BMI ≥ 30	9.1	29.3
Hodge <i>et al.</i> , 1994 (231)	Samoa <sup>a</sup>	25–74	797	989	BMI ≥ 30	48.7	68.0
Bourne <i>et al.</i> , 2002 (232)	South Africa <sup>a</sup>	>15	4006	5897	BMI ≥ 25 BMI ≥ 30	25.4 6.0	58.5 31.8
Wijewardene <i>et al.</i> , 2005 (233)	Sri Lanka <sup>a</sup>	30–65	2692	3355	BMI ≥ 25	20.3	36.5
Kosulwat, 2002 (234)	Thailand <sup>a</sup>	Adults	NS: 1991 NS: 1996		BMI ≥ 25 BMI ≥ 25	7.7 13.2	15.7 25.0
Florez <i>et al.</i> , 2005 (37)	Venezuela <sup>a</sup>	>20	3108 <sup>b</sup>		WC ≥ 102 (M), ≥88 (F)	42.9 <sup>b</sup>	

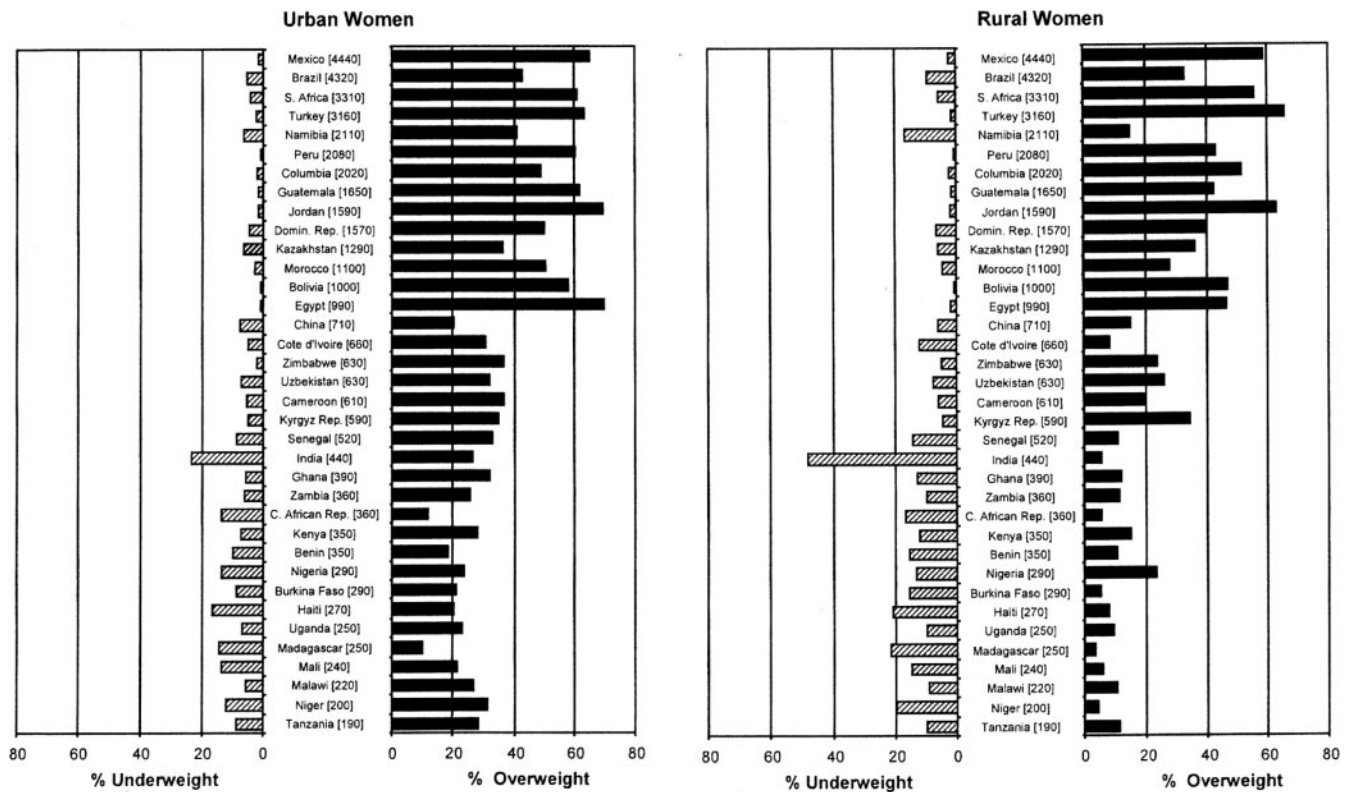
Data are given according to alphabetical order of countries. NS, National survey; M, male; F, female, WC, waist circumference.

<sup>a</sup> Representative of sample total population in the area.

<sup>b</sup> Overall including male and female.

<sup>c</sup> Data from urban slum population of New Delhi, north India.

<sup>d</sup> Data from Punjabi Bhatia community in north India.



**FIG. 1.** Prevalence of overweight [BMI (in kilograms per square meters)  $\geq 25$ ] and underweight (BMI  $< 18.5$ ) in women aged 20–49 yr in 36 developing countries ranked (in brackets) by per capita gross national income (U.S. dollars). Source: Mendez *et al.* (204); originally printed elsewhere (28).

obesity (35). Data from countries in sub-Saharan Africa, South America, and the Middle East also showed similar high prevalence of abdominal obesity: 67% of women and 18% of men in urban Cameroon (36), 43% in Venezuela (37), and 24.6% in Oman (38).

**Obesity in women (Fig. 1)**

Higher prevalence of obesity and abdominal obesity has been seen in women compared with men in many developing countries, consistent with the sedentary lifestyle as discussed later. For example, the prevalence of obesity (standardized for age) in adults in Seychelles was 5-fold more in women compared with men (20.9 *vs.* 4.2%, respectively) (39). Furthermore, in this population, the prevalence of obesity in women increased from 8.9% at age 25–34 yr to 29.4% at age 35–44 yr and reached a plateau thereafter, whereas the prevalence did not change with age in men. Correspondingly, prevalence of T2DM (standardized for age) was also higher in women (women, 4.6% *vs.* men, 3.4%) (39). The age-standardized prevalence of overweight (BMI  $> 25.0$  kg/m<sup>2</sup>) was higher in women (31.1%) than men (26.9%) in China (40). Other developing countries show similar trends regarding higher prevalence of obesity in women: Iran (men, 24% and women, 42%), (41) and Sri Lanka (men, 20.3% and women, 36.5%) (42).

**Obesity in children (Table 2)**

Similar to adults, the prevalence of overweight and obesity and overweight in children in developing countries shows an increasing trend. This is a serious challenge because malnutrition

and stunted growth are often seen to coexist in children, and eliminating undernutrition without increase in obesity is required (43). Since 1986, several surveys in preschool children show increasing obesity in most countries in Latin America and the Caribbean, along with the Middle East and North Africa, which is comparable with prevalence rates of childhood obesity seen in the United States (43). Similar trends have also been observed in India, Mexico, Nigeria, and Tunisia over the past 2 decades (44). The prevalence of obesity in 5- to 12-yr-old children in Thailand increased from 12.2 to 15.6% over a period of 2 yr (24). Increase in the prevalence of overweight among older children and adolescents has been seen as well; from 4.1 to 13.9% between 1975 and 1997 in Brazil, from 6.4 to 7.7% between 1991 and 1997 in China, and from 16 to 24% between 2002 to 2007 in New Delhi, India (45, 46). Furthermore, overweight was more common in urban areas *vs.* rural areas, privately funded schools *vs.* government funded schools (46), girls *vs.* boys (46), and children born to more educated mothers in India (43).

**Epidemiology of the metabolic syndrome (Table 3)**

**Adults**

In line with the rising prevalence of obesity, the metabolic syndrome is also increasing in developing countries. High prevalence of the metabolic syndrome has been reported from sub-Saharan Africa and Middle East countries; South Africa, Mo-

**TABLE 2.** Childhood obesity in developing countries

Author and year	Country	Sample (n)	Age range (yr)	Criteria for measuring overweight/obesity	Prevalence (%)
Hirschler <i>et al.</i> , 2006 (235)	Argentina	321	4.39 <sup>a</sup>	Overweight: BMI $\geq$ 85th percentile, obesity: BMI $\geq$ 95th percentile	At risk of overweight: 19.0 Overweight: 18.4
Silveira <i>et al.</i> , 2006 (236)	Brazil	1420	14–19	Obesity/overweight: BMI $\geq$ 85th percentile	Overweight: 15.2
Liu <i>et al.</i> , 2007 (237)	China	262,738	3.5–6.4	Overweight/obesity: age- and gender-specific BMI (IOTF)	Overweight & obesity: 7.4
Nunez-Rivas <i>et al.</i> , 2003 (238)	Costa Rica	1718	7–12	Overweight: BMI $\geq$ 85th percentile Obesity: 7–9 yr: triceps skinfold $\geq$ 85th percentile for age and sex <sup>b</sup> 10–12 yr: BMI $\geq$ 85th percentile and both triceps and subscapular skinfold thickness $\geq$ 90th percentile	Overweight: 34.5 Obesity: 26.2
Bhardwaj <i>et al.</i> , 2008 (46)	New Delhi, India	3493	14–17	Overweight/obesity: age- and gender-specific BMI cutoffs $\geq$ 85th percentile.	Overall: 24.3 Overweight/obesity: private schools, 29.0; government-funded schools, 11.3
Moayeri <i>et al.</i> , 2006 (239)	Tehran, Iran	2900	11–17	Overweight/obesity: age- and sex-specific cutoff points for BMI	Overweight: 17.9 Obesity: 7.1
Lafta and Kadhim, 2005 (240)	Iraq	8300	7–13	Overweight/obesity: age- and gender-specific cutoff points for BMI (IOTF)	Overweight: 6.0 Obesity: 1.3
Jabre <i>et al.</i> , 2005 (241)	Beirut, Lebanon	234	6–8	Obesity/overweight: age- and gender-specific cut-off points for BMI (IOTF)	Overweight: boys, 26.0; girls, 25.0 Obesity: boys, 7.0; girls, 6.0
Tee <i>et al.</i> , 2002 (258)	Kuala Lumpur, Malaysia	5995	7–10	Overweight/obesity: $\geq$ 95th percentile for age	Overall: 8.4 Overweight: boys, 9.7; girls, 7.1
Sumarni <i>et al.</i> , 2006 (242)	Kuala Lumpur, Malaysia	699, boys	11	Obesity/overweight: age- and gender-specific cutoff points for BMI (IOTF)	Overall: 7.2 Urban, 7.2; rural, 7.0
El-Hazmi and Warsy, 2002 (243)	Saudi Arabia	12071	1–18	Overweight/obesity: age- and sex-specific cutoff points for BMI	Overweight: boys, 10.7; girls, 12.7 Obesity: boys, 6.0; girls, 6.74
Kruger <i>et al.</i> , 2006 (244)	South Africa	1257	10–15	Obesity/overweight: BMI for age (IOTF)	Overweight/obesity: 7.8
Wickramasinghe <i>et al.</i> , 2004 (245)	Sri Lanka	1224	8–12	Obesity/overweight: age- and sex-specific cutoff points for BMI (IOTF)	Obesity: boys, 4.3; girls, 3.1
Langendijk <i>et al.</i> , 2003 (246)	Thailand (northeast)	864	7–9	Obesity: weight-for-height Z-score above 2.0 sd of the NCHS/WHO reference population median	Obesity: 10.8

Data are given according to alphabetical order of countries. IOTF, International Obesity Task Force; NCHS, National Center for Health Statistics.

<sup>a</sup> Mean age.

<sup>b</sup> Using the percentiles by age for children in the United States.

rocco, Oman, Turkey, and Iran showed prevalence of 33.5, 16.3, 21, 33.4, and 33.7%, respectively. The prevalence rates are also high in Venezuela (31.2%) and urban Brazil (25.4%) (37, 47). The situation appears to be similar in South Asian countries. The prevalence of insulin resistance as measured by surrogate markers in Asian Indians residing in India ranged from 20 to 55%; the variation in the prevalence was due to heterogeneity of the population (higher in urban *vs.* rural) and socioeconomic strata (SES) (higher in the people belonging to high SES *vs.* low SES) (48, 49). The recent data show that one fourth to one third of urban population of India has the metabolic syndrome (50). Furthermore, the prevalence is 1.5–2 times higher in women com-

pared with men (34, 50). Interestingly, certain communities in India (*e.g.* Punjabi Bhatia community in north India) have inordinately high tendency to have obesity, T2DM, and the metabolic syndrome (51). Prevalence of the metabolic syndrome in population of Sri Lanka is strikingly high as well; 35% in males and 51% in females ( $n = 16,729$ ) (Wijesuriya M., personal communication). In the Sindh province of Pakistan, overall prevalence of the metabolic syndrome was 34.8 and 49% according to International Diabetes Federation (IDF) and modified National Cholesterol Education Program, Adult Treatment Panel III (NCEP, ATP III) definitions, respectively ( $n = 363$ ) (Basit A., personal communication).

**TABLE 3.** Prevalence of the metabolic syndrome in developing countries

Author and year	Country/region and urban/rural area	Age (yr)	Sample (n)		Criterion for diagnosis	Prevalence (%)	
			Men	Women		Male	Female
Gu <i>et al.</i> , 2005 (40)	China <sup>a</sup>	35–74	15540 <sup>b</sup>		NCEP, ATP III	9.8	17.8
Fan <i>et al.</i> , 2005 (53)	Shanghai, China <sup>a</sup>	Mean 52.4	1218	1957	Modified NCEP, ATP III	22.9	20.8
Yang <i>et al.</i> , 2007 (247)	China <sup>a</sup>	35–74	15838 <sup>b</sup>		IDF	16.5 <sup>b</sup>	
Fan <i>et al.</i> , 2008 (210)	Shanghai, China <sup>a</sup>	20–88	1524	2379	Modified NCEP, ATP III	23.3 <sup>b</sup>	
Misra <i>et al.</i> , 2001 (176)	North India <sup>c</sup>	>18	170	362	Modified NCEP ATP III	15.3 <sup>b</sup>	
Deepa <i>et al.</i> , 2002 (248)	South India (urban)	≥20	1070 <sup>b</sup>		NCEP, ATP III	13.3	15.6
					EGIR	11.2 <sup>b</sup>	
						MIG, 18.7 <sup>b</sup>	
						LIG, 6.5 <sup>b</sup>	
Gupta <i>et al.</i> , 2003 (217)	North India (urban)	≥20	532	559	NCEP, ATP III	9.8	20.4
Ramachandran <i>et al.</i> , 2003 (249)	South India (urban)	20–75	475 <sup>b</sup>		Modified NCEP, ATP III	36.4	46.5
Deepa <i>et al.</i> , 2007 (250)	South India (urban)	≥20	23505		WHO	23.2 <sup>b</sup>	
					NCEP, ATP III IDF	18.3 <sup>b</sup>	
						25.8 <sup>b</sup>	
Gupta <i>et al.</i> , 2004 (219)	North India (urban)	>20	532	559	NCEP, ATP III	22.9	39.9
Misra <i>et al.</i> , 2005 (113)	North India (urban)	Mean; 38.9	640 <sup>b</sup>		Modifications of NCEP, ATP III [BMI ≥ 23 kg/m <sup>2</sup> , WC ≥ 90 (M) ≥ 80 (F) in addition to other criteria]	11.0	10.5
Gupta <i>et al.</i> , 2007 (221)	North India (urban)	>20	532	559	NCEP, ATP III	22.9	31.6
Chow <i>et al.</i> , 2008 (183)	India (rural)	≥30	4535 <sup>b</sup>		Modified NCEP, ATP III	32.5	23.9
Azizi <i>et al.</i> , 2003 (41)	Tehran, Iran (urban)	>20	4397	5971	NCEP, ATP III	24.0	42.0
Oh <i>et al.</i> , 2004 (35)	Korea (urban)	30–80	269 <sup>b</sup>		Modified NCEP, ATP III	29.0	16.8
Park <i>et al.</i> , 2004 (251)	Korea <sup>a</sup>	20–80	3937	4713	Modified NCEP, ATP III	14.2	17.7
Park <i>et al.</i> , 2006 (222)	Korea <sup>a</sup>	20–80	6824 <sup>b</sup>		IDF	13.5	15.5
Gustiene <i>et al.</i> , 2005 (252)	Kaunas, Lithuania	Mean 38.8	192	241	IDF	28.1	16.6
Cameron <i>et al.</i> , 2003 (253)	Mauritius <sup>a</sup>	>24	1473	1698	Modified NCEP, ATP III	10.6	14.7
Aguilar-Salinas <i>et al.</i> , 2003 (254)	Mexico <sup>a</sup>	20–69	2158 <sup>b</sup>		WHO	13.6 <sup>b</sup>	
					NCEP, ATP III	26.6 <sup>b</sup>	
Al-Lawati <i>et al.</i> , 2003 (38)	Oman <sup>a</sup>	>20	695	724	NCEP, ATP III	19.5	23.0
Tan <i>et al.</i> , 2004 (52)	Singapore (Malays, Asian Indians, and Chinese ethnicity) <sup>a</sup>	18–69	4723 <sup>b</sup>		Modified NCEP, ATP III	20.9	15.5
Ozsahin <i>et al.</i> , 2004 (255)	Turkey <sup>a</sup>	20–79	1637 <sup>b</sup>		NCEP, ATP III	23.7	39.1
Onat <i>et al.</i> , 2002 (256)	Turkey <sup>a</sup>	>31	1130	1166	NCEP, ATP III	27.0	38.6
Florez <i>et al.</i> , 2005 (37)	Venezuela <sup>a</sup>	>20	3108 <sup>b</sup>		NCEP, ATP III	31.2 <sup>b</sup>	

Data are given according to alphabetical order of countries. Definitions/criteria for the metabolic syndrome used in the table: (1) NCEP, ATP III: at least three of the following criteria; waist circumference greater than 102 cm (M) and greater than 88 cm (F); triglycerides 150 mg/dl or greater; HDL-C less than 40 mg/dl (M) and less than 50 mg/dl (F); blood pressure 130/85 mm Hg or greater; and fasting blood glucose 110 mg/dl or greater (2) Modified NCEP, ATP III: in addition to NCEP, ATP III criteria, fasting blood glucose greater than 100 mg/dl and ethnic-specific cutoffs of waist circumference greater than 90 cm (M) and greater than 80 cm (F) for Asian populations; (3) IDF, waist circumference is mandatory criterion and any two of the following; triglycerides greater than 150 mg/dl or treated for it; HDL-C less than 40 mg/dl (M) and less than 50 mg/dl (F) or treated for it; blood pressure 130/85 mm Hg or greater or treated for previously diagnosed hypertension, fasting blood glucose 100 mg/dl or greater, or previously diagnosed diabetes; ethnic-specific cutoffs of waist circumference in IDF definition, Europid 94 cm or greater (M) and 80 cm or greater (F); U.S. NCEP, ATP III values applicable; South Asians, 90 cm or greater (M) and 80 cm or greater (F); Chinese, 90 cm or greater (M) and 80 cm or greater (F); Japanese, 90 cm or greater (M) and 80 cm or greater (F); for ethnic South and Central Americans, use South Asian recommendations; sub-Saharan Africans, use European recommendations; Eastern Mediterranean and Middle East (Arab) populations, use European recommendations; for latter three ethnic groups, the recommendations are applicable until more specific data are available; and (4) EGIR, fasting hyperinsulinemia (highest 25%) and at least two of the following: fasting blood glucose 110 mg/dl or greater (excluding diabetes); blood pressure 140/90 mm Hg or greater or treated for hypertension; triglycerides greater than 176 mg/dl (2.0 mmol/liter); or HDL-C less than 40 mg/dl or treated for dyslipidemia, waist circumference 94 cm or greater (M) and 80 cm or greater (F). EGIR, European Group for the Study of Insulin Resistance; M, male; F, female; WC, waist circumference; MIG, middle income group; LIG, lower income group.

<sup>a</sup> Representative sample of total population.

<sup>b</sup> Overall (including both males and females).

<sup>c</sup> Data from urban slum population of New Delhi, north India.

There is a paucity of data on prevalence of insulin resistance and the metabolic syndrome from other south Asian countries: Bangladesh and Nepal.

Prevalence of the metabolic syndrome is rapidly increasing in East Asia and China. Using modified NCEP, ATP III criteria, prevalence was 29.0 and 16.8% in South Korean men and

women, respectively (35). A study done in Singapore not only highlighted change in prevalence when criteria for abdominal obesity were modified but also showed ethnic differences. Tan *et al.* (52) showed that the prevalence was highest in Asian Indians (28.8%), followed by the Malays (24.2%) and then the Chinese (14.8%), and ethnic differences persisted in both genders (52).

Age-standardized prevalence of the metabolic syndrome in China was reported to be 9.8% in men and 17.8% in women and was higher in urban than in rural populations (40). High prevalence of the metabolic syndrome (22.9%) among adults in city of Shanghai, China has been reported, even after adjustment by age and gender (15.3%) (53). Hong Kong Chinese showed high prevalence of several cardiovascular risk factors comprising the metabolic syndrome: 30%, central obesity; 34%, low levels of high-density lipoprotein cholesterol (HDL-C); 20%, hypertriglyceridemia, and 47%, hypertension (54). In many of these studies, higher prevalence rates of the metabolic syndrome in women were reported, similar to the gender differences seen in the prevalence of obesity.

### Children

Data pertaining to the metabolic syndrome in children and adolescents are scarce. This is partly because of lack of consensus on the definition of the metabolic syndrome in children, which is presently defined using different criteria and cutoff points. The recent IDF definition of the metabolic syndrome in children includes waist circumference as mandatory criterion and 2 or more other risk variables (55). The new IDF definition is age specific, taking into account developmental challenges in growing children and adolescents. (Table 4) (56). The prevalence rates vary depending on diverse criteria used. The prevalence of the metabolic syndrome was 5.6% in boys and 6.4% in girls aged 6–12 yr in Taiwan (57, 58). Overall prevalence of the metabolic syndrome in Chinese adolescents was shown to be 3.7%; however, the prevalence was 35.2, 23.4, and 2.3% among adolescents who were overweight (BMI  $\geq$  95th percentile), at risk of overweight (BMI between 85th and 95th percentile), and normal weight (BMI below the 85th percentile), respectively (59). Furthermore, strikingly high prevalence rates of dyslipidemia (61.9%) and low levels of HDL-C (56.1%) and hypertension (16.0%) were seen in

Chinese adolescents 15–18 yr of age (60). In a study in Asian Indian adolescents, we showed that by application of NCEP, ATP III criteria with appropriate percentile cutoff points, the metabolic syndrome was identified in only 0.8% of subjects (61). However, inclusion of BMI and fasting insulin as additional criteria increased prevalence of the metabolic syndrome to 10%. These data indicate that early markers of metabolic derangements, such as fasting insulin, should be included for the definition of the metabolic syndrome in children.

### Phenotype of obesity in developing countries: ethnic specific cutoffs of obesity and abdominal obesity

Phenotype of obesity in several ethnic groups in developing countries appears to be different from that seen in white Caucasians in developed countries. Several investigators have shown that body fat is higher in Asians, particularly south Asians, compared with white Caucasians for the similar level of BMI (27, 62–72). This body composition feature has been documented in Indonesians, Singaporean Chinese, and Malays. For example, at any given percentage of body fat, BMI value of Chinese, Malays, and Asian Indians in Singapore was 3 kg/m<sup>2</sup> lower than that in white Caucasians (73). Furthermore, Indonesians had about a 2 kg/m<sup>2</sup> lower BMI but 3% higher body fat than Dutch Caucasians, suggesting cutoff points for obesity in Indonesians should be 27 kg/m<sup>2</sup> instead of 30 kg/m<sup>2</sup> (74). High percentage of body fat with low BMI value could be partly explained by body build (trunk to leg length ratio and slender body frame), muscularity, adaptation to chronic calorie deprivation, and ethnicity (75). Importantly, obesity-related morbidities (diabetes, hypertension, dyslipidemia) occur more frequently at lower BMI levels in Asians than white Caucasians. In Hong Kong Chinese men, the optimal BMI

**TABLE 4.** The IDF consensus definition of the metabolic syndrome in children and adolescents (56)

Age groups (yr)	Obesity (waist circumference) <sup>a</sup>	Triglycerides	HDL-C	Blood pressure	Glucose (mmol/liter) or known T2DM
6 to <10	$\geq$ 90th percentile	Metabolic syndrome cannot be diagnosed, but further measurements should be made if there is a family history of metabolic syndrome, T2DM, dyslipidemia, CVD, hypertension, and/or obesity			
10 to <16	$\geq$ 90th percentile or adult cutoff if lower	$\geq$ 1.7 mmol/liter ( $\geq$ 150 mg/dl)	<1.03 mmol/liter (<40 mg/dl)	Systolic $\geq$ 130/diastolic $\geq$ 85 mm Hg	$\geq$ 5.6 mmol/liter (100 mg/dl) [if $\geq$ 5.6 mmol/liter (or known T2DM) recommend an OGTT]
16+	Use existing IDF criteria for adults, <i>i.e.</i> Central obesity (defined as waist circumference $\geq$ 94 cm for European men and $\geq$ 80 cm for European women, with ethnicity specific values for other groups <sup>a</sup> ) plus any two of the following four factors: <ul style="list-style-type: none"> <li>● Raised triglycerides: <math>\geq</math>1.7 mmol/liter</li> <li>● Reduced HDL-C &lt;1.03 mmol/liter (&lt;40 mg/dl) in males and &lt;1.29 mmol/liter (&lt;50 mg/dl) in females or specific treatment for these lipid abnormalities</li> <li>● Raised blood pressure: systolic blood pressure <math>\geq</math>130 or diastolic blood pressure <math>\geq</math>85 mm Hg or treatment of previously diagnosed hypertension</li> <li>● IFG: fasting plasma glucose <math>\geq</math>5.6 mmol/liter (<math>\geq</math>100 mg/dl) or previously diagnosed T2DM</li> </ul>				

OGTT, Oral glucose tolerance test.

<sup>a</sup> The IDF consensus group recognizes that there are ethnic, gender, and age differences, but research is still needed on outcomes to establish risk.

cutoff to predict diabetes, hypertension, and dyslipidemia was lower than currently recommended (76). Our data on Asian Indians residing in New Delhi, India, showed that about 66% of men and 88% of women, classified as nonobese based on international cutoff of BMI, had one or more cardiovascular risk factor(s). Furthermore, significantly higher odds ratios for hypertension, T2DM, and hypertriglyceridemia were observed in nonobese subjects having upper quartile of percentage body fat compared with the lowermost quartile (77). Based on these data and those by other investigators, it has been suggested that BMI limits for overweight should be lower for Asian Indians (78–80). Finally, a metaanalysis of data of 13 population-based studies including 239,972 adults in China and Taiwan showed that BMI of 24 kg/m<sup>2</sup> had the best sensitivity and specificity for identification of cardiovascular risk factors, and this new limit would prevent about 50% clustering of risk factors (81). Overall, the results of the studies suggest lowering the BMI limits by about 1–3 kg/m<sup>2</sup> for the diagnosis of overweight for the Asian populations (82).

Although the average value of waist circumference in South Asians is lower than other ethnic groups, abdominal adiposity is significantly more than white Caucasians (65, 83–88). Raji *et al.* (89) showed that at similar value of BMI, migrant Asian Indians had significantly greater total abdominal fat and intraabdominal fat than white Caucasians in the United States. In addition, despite lower BMI and waist circumference, Asian Indians had significantly lower glucose disposal rates during the hyperinsulinemic euglycemic clamp, higher procoagulant tendency, and dyslipidemia compared with white Caucasians (85, 89). Furthermore, we reported significantly high odds ratios for hypertriglyceridemia and hypertension in the normal range of waist circumference (70–80 cm) in Asian Indians residing in India (90). The cutoff points of waist circumference in urban Asian Indians living in India based on receiver operating characteristics curve analysis (using T2DM as a reference) for various morbidities were 90 and 80 cm for men and women, respectively (91, 92). Similarly, lower waist circumference cutoff points than presently accepted have been reported based on the morbidity data for several non-Asian populations of developing countries, including Nigeria, Cameroon, Jamaica, St. Lucia, Barbados (93), Brazil (94), Mexico (95), and Iran (96). It is important to note that South Asians have thick sc adipose tissue as highlighted by the investigators who used skinfold measurements in their investigations (65, 84–88). Higher magnitude of insulin resistance in a BMI- and body fat-matched Asian Indian men than white Caucasians in the United States could be explained by thicker truncal skinfolds in the former (85, 97). A significant association of truncal skinfold thickness (signifying high truncal sc adipose tissue) with fasting hyperinsulinemia in Asian Indian children and adolescents has been reported by us (98). Interestingly, thicker subscapular sc fat in Asian Indians has been recorded at birth and is associated with higher insulin levels when compared with British neonates (68). Finally, histological studies of sc adipose tissue showed that Asian Indians have larger adipocytes (reported to be more resistant to the action of insulin) compared with white Caucasians in the United States (3491 *vs.* 1648  $\mu\text{m}^2$ ;  $P = 0.0001$ ) (97). The clinical implication of the latter finding remains to be investigated.

Fat accumulation in other tissues in which it is not usually deposited (ectopic fat) has the potential to affect insulin sensitivity. Deposition of fat in liver (nonalcoholic fatty liver disease) and muscle [intramyocellular triglycerides (IMCL)] has been shown to correlate with insulin resistance in white Caucasians (99). It is important to note that Asian Indians have higher hepatic triglycerides levels, which are associated with higher insulin and lower adiponectin levels than white Caucasians (100). We recently reported early abnormalities in hepatic gluconeogenesis pathway in nondiabetic obese and nonobese Asian Indians with nonalcoholic fatty liver disease using *in vivo* (31) phosphorus magnetic resonance spectroscopy of liver, signifying that glucose metabolism is dysregulated early in Asian Indians with fatty liver, even when they are nonobese (101). We have also shown excess IMCL deposition in soleus muscle in Asian Indians with abdominal obesity; however, unlike white Caucasians, IMCL did not correlate with fasting insulin levels (102, 103). Finally, presence of excess dorsocervical fat (mild buffalo hump) and excess fat deposition under the chin (double chin) may also signify heightened risk for the metabolic syndrome in urban Asian Indians and may be used as phenotypic markers (104).

Overall, Asians and South Asians could be classified as ‘metabolically obese’ (105), *i.e.* they have several metabolic derangements but are nonobese by conventional BMI standards. These nonobese people usually have high body fat; abdominal adiposity; ectopic fat deposition; and, specifically in South Asians, thick truncal sc fat. These body composition characteristics, individually or in combination, contribute to insulin resistance, dyslipidemia, hyperglycemia, and excess procoagulant factors seen commonly in South Asians (1, 30, 65, 71, 85, 86, 89, 106).

These data have prompted a debate that BMI cutoffs for diagnosis of overweight and obesity should be lower for Asian populations as opposed to currently prevalent international guidelines (70, 107, 108). In a consultation on obesity in Asia and Pacific regions, lower cutoffs for diagnosis of overweight and obesity were suggested; 23 kg/m<sup>2</sup> or greater and 25 kg/m<sup>2</sup> or greater, respectively (25). More recently a WHO expert consultation agreed that Asian populations have different associations between BMI, percentage of body fat, and health risks than do European populations. The experts also opined that the proportion of Asian people with a high risk of T2DM and CVD is considerable at BMI values lower than the existing WHO cutoff point for overweight ( $\geq 25$  kg/m<sup>2</sup>). However, consultation did not suggest a clear BMI cutoff point for all Asians for overweight or obesity. Potential public health action points of BMI (23.0, 27.5, 32.5, and 37.5 kg/m<sup>2</sup>), above which an individual should be targeted for various and health interventions stepwise, were proposed for Asian populations (26). Overall, despite considerable evidence to lower BMI cutoff points for the Asian populations, failure of the WHO and other international agencies to suggest a new BMI cutoff for Asian populations is intriguing. Compared with the data on BMI, the picture is clearer for lower cutoffs for waist circumference for Asian populations for which considerable evidence is also available. These ethnic-specific cutoff points for waist circumference have been taken into consideration by IDF and NCEP, ATP III in recent definitions of the metabolic syndrome (4, 81, 90, 91, 107, 109–112).



## Ethnic groups in developing countries and criteria of the metabolic syndrome

Some ethnic-specific guidelines are available for the definitions of the metabolic syndrome. With new data on cutoffs of waist circumference from Asian populations, the IDF and NCEP, ATP III have introduced modified definitions, which specify ethnic-specific criteria for waist circumference. However, whether predictive value of these definitions for T2DM and CVD is better than previous definitions of the metabolic syndrome for ethnic groups in developing countries has been debated (82). Furthermore, taking in cognizance of distinctive body composition of Asians, and in particular South Asians, new definitions of the metabolic syndrome, including specific body composition characteristics (e.g. truncal sc fat measured by subscapular skinfold thickness), have also been proposed (113). Given some recent evidence that the metabolic syndrome and its components are associated with T2DM but have weak or no association with vascular risk in elderly populations (21), predictive value of any definition of the metabolic syndrome for CVD needs to be investigated further in different ethnic groups in the developing countries. The varying relationships of the components of the metabolic syndrome with CVD risk in different ethnic groups mostly in developing countries necessitates further studies, as discussed below. Detailed discussion on ethnicity and the metabolic syndrome is beyond the scope of this review and can be referred to in our major published review (114).

The first concern with the applicability of the metabolic syndrome criteria to different ethnic groups in developing countries is that the risk imparted by the cutoffs for the criteria (other than waist circumference, discussed above) that comprise it varies according to ethnicity. Data suggest that the hypertension criterion does not accurately indicate CVD risk across populations due to differences in the way hypertension imparts CVD risk across ethnic groups. It is generally accepted that hypertension has the weakest association with insulin resistance among all factors that comprise the metabolic syndrome (115). Interestingly, the Chinese population has a lower prevalence of hypertension than North American Caucasian cohort, but the CVD risk associated with increases in blood pressure is proportionally greater in the Chinese (116). Similarly, Filipino females with a higher prevalence of both hypertension and the metabolic syndrome than white Caucasians did not show excess subclinical atherosclerosis (117). Several studies in the United Kingdom have shown higher prevalence rates of hypertension in populations of African ancestry but lower rates of mortality due to CVD when compared with white Caucasians and South Asians with hypertension (118, 119). Clearly, mismatches between hypertension and CVD risk is evident in several ethnic groups, indicating that the hypertension criteria of the metabolic syndrome should be altered by either changing the cutoffs for different populations or weighting the individual criteria based on their risk contribution to CVD in each population. One could also debate on deletion of hypertension from the criteria of the metabolic syndrome in ethnic groups in which the relationship between hypertension and CVD risk is weak.

Other criteria of metabolic syndrome [serum triglycerides

and HDL-C] should be considered on the similar mode. It has been shown that the NCEP, ATP III criterion, which is most likely to identify insulin resistance in individuals, is the triglycerides to HDL-C ratio (120). It is also known that these factors capture much of the cardiovascular risk associated with the metabolic syndrome. Yet Sone *et al.* (121) noted that in a Japanese population, HDL-C levels were not a significant risk factor for CVD, in contrast to the United Kingdom Prospective Diabetes Study in which HDL-C levels were significantly associated with CVD risk in a British white Caucasian population. Hispanics are known to exhibit higher serum triglyceride and lower HDL-C levels than non-Hispanic Caucasians, (122) yet have similar rates of CVD to non-Hispanic Caucasians (123, 124).

An oft-repeated question is whether blood glucose characterizes cardiovascular risk adequately in different populations. In multiracial population in Singapore, Ma *et al.* (125) noted that mortality risk for T2DM in Asian Indians was higher when compared with Chinese and Malays, yet Chinese and Malays had a higher prevalence of impaired glucose tolerance and impaired fasting glucose (IFG) compared with Asian Indians, suggesting that the progression to T2DM is faster in Asian Indians compared with other Asian ethnic groups. These observations have implications for the relative importance of IFG for CVD risk in different ethnic groups, implying that IFG carries a greater risk for CVD mortality in Asian Indians than other ethnic groups. More accurate standards are needed to weight the risk for elevated blood glucose levels for T2DM and CVD in different ethnic groups in developing countries and alter the cutoffs accordingly, as has been done based on the data from white Caucasian populations (114).

Similarly, differences in the clustering of metabolic parameters have been shown in different ethnic groups in developing countries. The variable clustering of the different components in the metabolic syndrome in different populations suggest varying risk clusters associated with the metabolic syndrome. For example, obesity in African-Americans is more commonly associated with hypertension, whereas obesity-related T2DM is more common in Hispanics (126). Malay females in Mauritius exhibited a relatively high prevalence of hypertension associated with insulin resistance than other ethnic cohorts (127). Thus, there may be combinations of the criteria of the metabolic syndrome that are more appropriate for one ethnic group than another in predicting risk of T2DM and CVD. The evidence we currently have suggests that not only should the cutoffs and weights of the criteria for the metabolic syndrome be altered for different ethnic group in developing countries, but the combinations of the metabolic syndrome criteria for each ethnic group should also be varied. Cumulatively, these data suggest that we need receiver operating characteristics curve analysis-based cutoffs and/or a weighting system for combination of the various criteria of the metabolic syndrome in certain ethnic populations to more accurately capture the risk of T2DM and CVD. Some suggestions for ethnic-specific alterations to define the criteria of the metabolic syndrome are given in Table 5.

Finally, in view of the high and increasing prevalence of obesity and T2DM in children and adolescents, there is an urgent need to establish internationally acceptable criteria for the met-

**TABLE 5.** Summary of suggested ethnic-specific alterations to criteria of the metabolic syndrome

Alterations to criteria
1. Alteration of the cutoffs for metabolic syndrome criteria based on risk thresholds specific for each ethnic population
2. Where no threshold exists, consideration should be given to weighting each criterion differently in terms of its incremental contribution to cardiovascular risk in different ethnic populations
3. Further research to ascertain which combinations of the metabolic syndrome criteria best predict cardiovascular risk in each ethnic group (additional criteria such as age, etc. could be included)
4. A practical risk score similar to the Framingham score should be constructed based on the revised criteria for the metabolic syndrome with the goal of predicting CVD
5. The inclusion and evaluation of easily measurable and cost-effective criteria <sup>a</sup> ( <i>i.e.</i> acanthosis nigricans, subscapular skinfold thickness, buffalo hump, double chin, <sup>b</sup> etc.) in the rubric of the metabolic syndrome to more accurately capture cardiovascular risk should be considered

Adapted from Banerjee and Misra (114).

<sup>a</sup> Suitable for wide application in the developing countries.

<sup>b</sup> Data from Misra *et al.* (104).

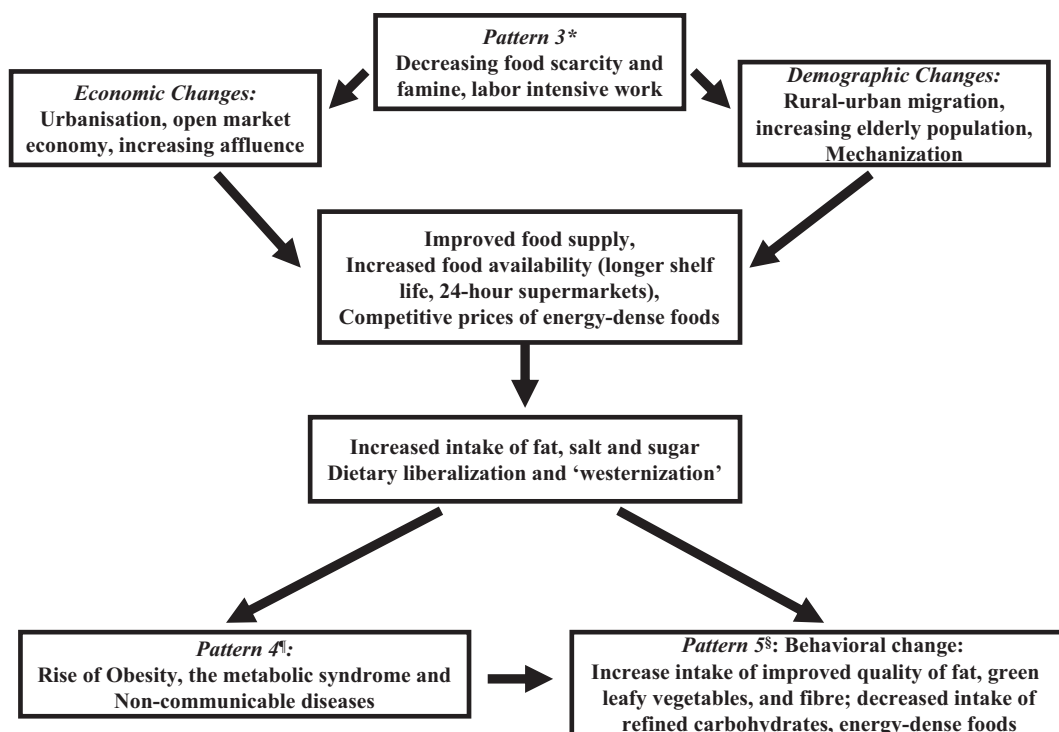
abolic syndrome. It needs to be researched further whether any ethnic-specific cutoffs of anthropometry or other parameters need to be included while proposing a universal definition of the metabolic syndrome in children, similar to the recent IDF definition of the metabolic syndrome in adults (81). In the recently proposed IDF definition of the metabolic syndrome in children (10–16 y), no ethnic differences in various criteria has been properly accounted for and adequately researched. Furthermore, because many criteria of the metabolic syndrome in adults may not

be dysregulated (*e.g.* blood glucose), additional criteria that occur early in phenotype (*e.g.* acanthosis nigricans, truncal skinfolds, buffalo hump, and fasting insulin levels) (46, 61, 98, 104) should be considered in the definition of the metabolic syndrome in children. Clinical utility and advantages over any proposed definition of the metabolic syndrome, whether in adults or children, remains to be researched (55). The resolution of this dilemma would greatly impact the preventive and management strategies in tackling the metabolic syndrome and will have important therapeutic implications.

### Determinants of obesity and the metabolic syndrome in developing countries

#### Transitions

Emergence of obesity and the metabolic syndrome in developing countries is due to a number of factors; the most important are discussed below. Demographic transition (shift to low fertility, low mortality, and higher life expectancy) and epidemiologic transition (from widely prevalent infectious diseases to a pattern of high prevalence of chronic lifestyle-related NCDs) have occurred in developing countries as they become economically more resourceful (socioeconomic transition, shift of people from low SES to high SES), causing significant shifts in dietary and physical activity patterns (nutrition and lifestyle transitions, and stress) (Fig. 2). These changes cause significant effects on body composition and metabolism, often resulting in increase in BMI, excess generalized and abdominal adiposity, deposition of ectopic fat, and increase in dyslipidemia and diabetes.



**FIG. 2.** Relationship between nutrition transition, urbanization, and the rise in obesity and the metabolic syndrome in developing countries. See text for definitions of five patterns of nutrition transition. \*, Pattern 3 may be seen at different rates of progression in different developing countries; †, likely to affect all SES; §, most likely to occur in upper SES.

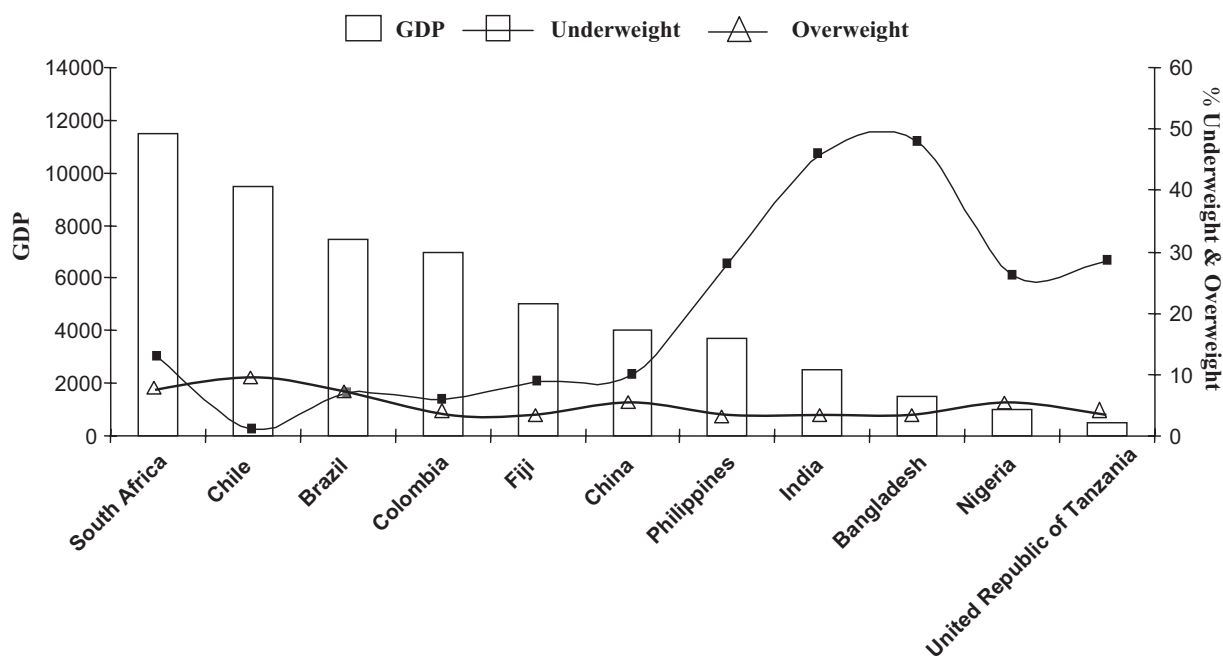


FIG. 3. Relationship between GDP and underweight and overweight children younger than 5 yr of age. Source: Statistics from FAO, 2004 (129).

Each type of transition has an important bearing on occurrence of obesity and metabolic risk factors; however, urbanization, nutrition transition, and increasing affluence remain the most important determinants of such dysmetabolic phenotype (Fig. 3).

**Rapid urbanization (Table 6)**

Urbanization is the prime driver for nutrition transition and emergence of obesity, the metabolic syndrome and other NCDs in developing countries. Remarkably, in 1999, when the world population was nearly 6 billion, a minimal increase in the populations of developed countries (1.9 to 1.21 billion) was seen compared with those of developing countries (4.87 to 6.9 billion), (128), which were increasingly becoming urbanized. Urbanization is variable within developing regions: almost three fourths of the population in Latin America and the Caribbean, whereas 38% in Africa and Asia are urbanized (129). The latter two regions are projected to have almost 50% urbanization by 2020. Specifically, marked increase in urbanization is projected to occur in India, China, Bangladesh, Nigeria, and Tanzania over the next 2 decades (7, 17, 13, 14, and 17%, respectively) (129). The rural-to-urban migration in many of the developing countries exposes migrants to urbanized diets and lifestyle. Studies

done in Brazil and India have clearly shown that many of these migrants, now living in urban slums and city shantytowns, have become obese and also manifest a number of other cardiovascular risk factors (29, 130).

**Nutrition transition**

Five patterns of nutrition transition, derived from historical references of human development, are present and evolve from one category to another in many ethnic groups and disparate geographical locations in developing countries. Pattern 1 nutrition transition is characteristic of hunter-gatherer populations and comprises diets rich in carbohydrates and fiber and low in fat, especially saturated fat, with a high-activity profile and lean body phenotype. In pattern 2, individuals exist in famine-like situation (low calorie, low protein and fat diets) and have growth retardation and low body fat and fat-free mass. In pattern 3, famine vanes, and nutrition improves, with increase in the consumption of fruits, vegetables, and animal proteins, and this pattern is associated with increasing inactivity. Pattern 4 is now most prevalent in developing countries, conducive to development of obesity, the metabolic syndrome, T2DM, and CVD. With improvement in economy, people become affluent and con-

TABLE 6. Urbanization in developing countries

Region/country	Level of urbanization (% of total population in urban settlements)			Urban population (millions)			Rural population (millions)		
	1975	2000	2025	1975	2000	2025	1975	2000	2025
World	37.8	47.4	58.9	1,543	2,890	4,736	2,538	3,201	3,303
Developing regions	26.7	40.5	54.7	809	1,986	3,729	2,223	2,918	3,090
Africa	25.2	37.8	51.7	104	310	752	310	510	702
Asia	24.6	37.6	52.4	593	1,387	2,508	1,813	2,302	2,277
Latin America and Caribbean	61.2	75.4	82.1	196	388	566	124	127	123

Source: Ref. (257). Changes in urban and rural populations by region, 1975–2025.

sume diets high in saturated fats, cholesterol, and refined carbohydrates and low in polyunsaturated fatty acids and fiber, associated with markedly sedentary lifestyle and increased stress. In the last (pattern 5), as people suffering from T2DM and CVD increase in the population, awareness of benefits of balanced diets and regular physical activity increases. Consequently, people attempt to change dietary and physical activity profiles to prevent or delay diseases. This pattern, unlike previous patterns, is driven by an individual's desire to seek healthy behavior, hence may not be evident in large segments of populations, and is likely to be adapted initially by affluent people. Furthermore, impetus to such lifestyle changes may be provided by government's policies, legal actions, and peer practices and hence may vary widely within each country. A rather rapid shift from pattern 3 to 4 in the developing countries is clearly responsible for steep increase in obesity and the metabolic syndrome. As gross domestic product (GDP) of the country increases with improved economy, underweight children decrease, whereas overweight increases predominantly due to nutrition transition (Fig. 3).

Most developing countries in Asia, Latin America, Northern Africa, the Middle East, and the urban areas of sub-Saharan Africa have been experiencing a shift in the dietary patterns over the last few decades. Major dietary changes include a large increase in the consumption of fats, particularly animal fat and added sugar and decrease in cereal and fiber intake. Marked increase in the consumption of egg, poultry, pork, mutton, beef, and milk amounting to 1300 kcal/d has been reported in Chinese adults, proportional to increase in the income in China (131). In India and South Asia, high intake of dairy products, sugar, and hydrogenated vegetable oils (*e.g.* Vanaspati) is widely prevalent (132). Availability of edible vegetable oils for consumption has nearly tripled throughout the developing world (Fig. 4) (129). For example, in China, availability of edible oils has risen 6-fold, whereas intake in the rest of Asia and Oceania tripled over the period from 1961 to 2000, much more than developed nations (129). Increased consumption of edible oils can be explained in

part by rising demand but also import policies, as is evident in the three largest emerging economies, Brazil, China, and India (133).

Importantly, consumption of nontraditional fast foods is occurring at a rapid pace in urban areas of several large cities in South Asia (49). In 1998 U.S.-based transnational food companies (TFCs) invested \$5.7 billion (US) in establishing outlets overseas, including many in developing countries (134). Rapid rise in fast food chains in developing countries is exemplified by the spread of McDonalds outlets globally and in Asia (from 951 outlets in 1987 to 7135 in 2002). Foreign direct investment from U.S.-based supermarket chains is increasing in developing and transition markets, notably in Latin America, Asia, and central and eastern Europe (135). In China, fast food chains increased from three in 1987 to 184 in 1997 and 546 in 2002 (136). In this context, Mexico makes an interesting case study. Between 1995 and 2003, sales of processed foods expanded by 5–10% per year. Recent sales has been particularly rapid for snacks (12% rise from January to June 2004), baked goods (55.4% rise from 2000 to 2003), and dairy products (48.1% rise from 2000 to 2003) (137, 138). Furthermore, calories from carbonated beverages increased from 44 to 61 kcal per capita per day between 1992 and 2000 (139). Lastly, locally made fast foods sold by street vendors in several developing countries, particularly in India and China, are equally unhealthy (140). These food items contain high amounts of trans-fatty acids due to deep frying in cheap and widely available hydrogenated vegetable oils.

**Nutritional intervention programs for undernourished children in developing countries and obesity**

A particularly interesting issue related to increase in childhood obesity is free meal or feeding programs to control undernutrition in developing countries, which may actually lead to increase in adiposity among those with marginal undernutrition (141). In school feeding programs in Chile, nutritional excess was seen with rapid growth and high prevalence of obesity, with 56% of children under the age of 6 yr and 11.1–14.3% of children in first

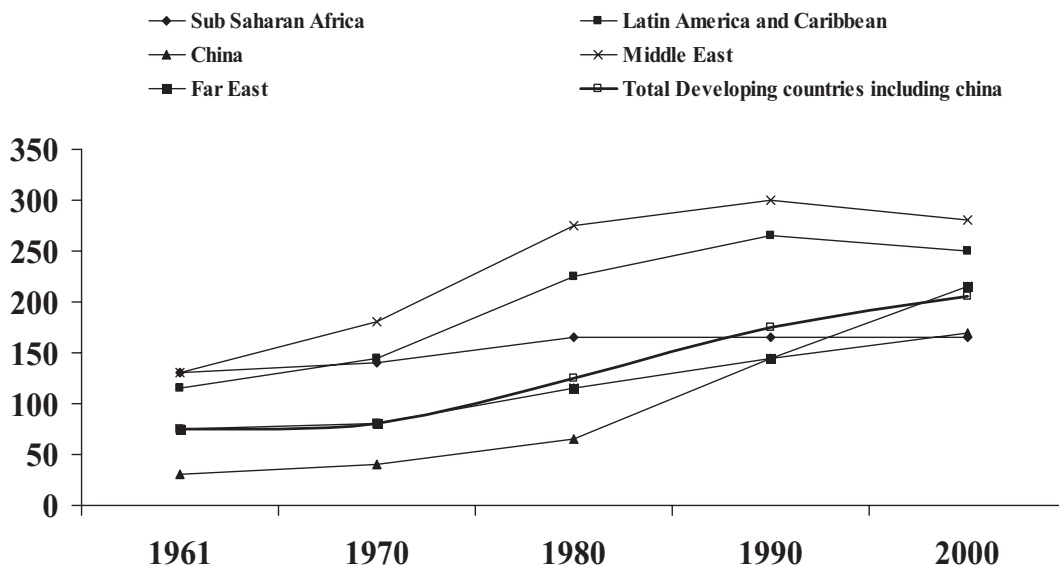


FIG. 4. Regional trends in availability for consumption of edible oils, 1961–2000. Source: Statistics from FAO, 2004 (129).

grade reported to be obese (142). FAO data from 19 Latin American countries show that more than 20% of the population in developing countries received some level of food assistance from nutrition-related programs, whereas prevalence of malnourishment was only 12% (141). It is likely that the scenario is similar in India where free midday meals are provided to schoolchildren who belong to low SES and are studying in government-funded schools.

Thus, nutrition programs have evolved beyond the immediate needs of the malnourished and have the potential to increase obesity epidemic in developing countries. Hence, careful selection of beneficiaries of food assistance programs and determination of the right combination of nutrients/foods, education, and lifestyle intervention is required to optimize nutrition and prevent obesity. Such ongoing programs in other countries to overcome malnutrition should be constantly monitored, reviewed, and revised if needed (141).

### Thrifty genotype/phenotype and maternal-fetal factors

It is postulated that thrifty genotype is propagated by survival during famines and lends heightened tendency to develop obesity and T2DM seen in certain ethnic groups in developing countries with abundant food availability. This concept, although interesting, remains hypothetical and lacks firm evidence in the absence of definite identification of such thrifty genes (143). Furthermore, natural selection by famine and tendency of individuals to gain weight with availability of abundant food should apply to the entire human population and should not be restricted to some subpopulations of developing countries (143, 144). Moreover, different prevalence in obesity rates between genders and even between rural and urban-based populations belonging to low SES indicate significant role of environmental and behavioral factors. Interestingly, in rural Gambia, children born low birth weight remained lean and largely free of metabolic diseases when they grew up as adults if they stayed in a rural setting and followed a frugal and traditional lifestyle (145, 146). These data show relative roles of genetic and environmental/behavioral factors, suggesting that behavioral choices appear to be key mediators of development of obesity (147).

A thrifty phenotype hypothesis that emphasizes fetal undernutrition leading to altered metabolic programming is perhaps a better explanation in resource-poor developing countries because abundant food supply later in life leads to maladaptive increase in weight and increases the risk of NCDs (146, 148, 149). The evidence is available from many developing countries. Asian Indian babies born small and with low birth weight had higher systolic blood pressure and adiposity at 8 yr of age (150). Chinese infants with low birth weight showed several cardiovascular risk factors at age 41–47 yr; hypertension, hyperglycemia, hyperinsulinemia, hypertriglyceridemia, and low levels of HDL-C (151). Furthermore, velocity of weight gain during childhood and catch-up obesity in low-birth-weight Asian Indian babies has been reported to be important for adult-onset hyperglycemia and cardiovascular risk factors (152). Whether low birth weight, childhood catch-up obesity, or increased velocity of weight gain within the normal weight range in childhood are

independent factors or additive in causation of insulin resistance and the metabolic syndrome has not been investigated.

These data have prompted the concept that the metabolic syndrome originates *in utero* and that at this time key metabolic activities may get modulated, even though the exact role of nutritional and genetic factors has not been clearly elucidated. There is preliminary evidence that programming of hormonal systems in response to an adverse fetal environment may play an important role. It is speculated that alterations in neuroendocrine responses to stress, in particular adrenocortical and sympathoadrenal responses, are associated with small size at birth, which may have influence on plasma glucose, lipid concentrations, and blood pressure. These concepts and preliminary data appear interesting but lack confirmatory evidence. Furthermore, the role of micronutrient deficiency(ies) during the perinatal period in development of chronic diseases later in life, although suggested, remains to be conclusively defined (153). Finally, preventive feeding of prepregnant and pregnant women may actually exacerbate the risk of obesity in Asian Indian neonates who have low muscle mass and excess sc adipose (68, 154).

### Physical inactivity

Changes of occupations, advent of newer technologies, and rapid pace of urban life have increasingly resulted in more sedentary work and less energy expenditure. Leisure time activities have also shifted from outdoor play to indoor entertainment; television viewing and computer games (132). In the Philippines and China, shift to more passive commuting modes (*i.e.* increased motorized transportation) has increased physical inactivity (155). The household chores have become more mechanized with the use of multiple domestic automatic devices; microwave, improved food storage, washing machines, vacuum cleaners, *etc.*, especially in India and China. Moreover, increasing mechanization even in rural areas (*e.g.* use of tractors than manual plowing of fields, use of motorized two-wheelers instead of bicycles) has contributed to physical inactivity in developing countries.

Data from 212,021 adults from 51 countries, most of which were developing countries, in the World Health Survey (2002–2003) showed that about 15% of men and 20% of women were at risk for chronic diseases due to physical inactivity (156). The prevalence of physical inactivity at less than the levels recommended for enhancing health was high in developing countries, ranging from 17 to 91% (157). Remarkably, data from Brazil show that 70–80% of the population are inactive (158). In Colombia, an estimated 7.6% of all-cause mortality and 20.1% of mortality due to chronic diseases were attributed to physical inactivity, and an estimated 5% of the mortality due to chronic diseases were preventable if physical inactivity was reduced by 30% (159). Physical inactivity contributed to 9.6% population-attributable risk and was an important risk factor for CVD in Costa Rica (160). Finally, increase in obesity has been directly linked to physical inactivity in Chinese adults (161, 162).

In most of the surveys, women are generally reported to be physically inactive than men, and this may also pertain to ethnic and cultural influences on outdoor activities. Physical inactivity

was more prevalent in women than men in urban east India (85.4 vs. 75.4%) (163) and in Saudi Arabia (98.1 vs. 93.9%) (164). Immigrant women from the Middle East, including Iran and Turkey, had higher prevalence of abdominal obesity than Swedish-born women, with a high degree of physical inactivity during leisure time (165).

Of major concern is that children and adolescents are rapidly showing a decrease in physical activity levels, which is fueling obesity and T2DM at a young age. Urban Asian Indian adolescents who participated regularly in outdoor games had lower prevalence of overweight, with the risk being 3 times higher in those not participating in outdoor games (166). Only 22.4% of Saudi preschool children walked 10,000 steps or more per day (167). Nearly two thirds of Iranian adolescents aged 11–18 yr were physically inactive (168). On account of cultural and weather barriers, adolescent girls had very low levels of physical activity in United Arab Emirates (169). Furthermore, nonobese Iranian children were reported to be more active in sports and went to school by foot compared with obese children who did not participate so much in sports and used mechanized transport (170).

### Socioeconomic factors

The previously held view that people belonging to high SES are more predisposed to develop obesity in developing world is no longer tenable (171). The burden of obesity in developing countries shifts toward the people belonging to lower SES as the country's GDP increases, probably because scarcity of food and/or physically demanding lifestyle become less common, even in people belonging to low SES after the economy improves. Pattern 5 of nutrition transition, in which nutrition and physical activity become more balanced than pattern 4, is mostly applicable to people belonging to high SES. This segment of population can afford relatively more expensive healthy foods and costly but healthy oils and avail of facilities and equipment for physical exercise and thus have far more flexibility in their choices of foods and activity patterns than those belonging to low SES. The latter class of people, although improving in economic status, continue to have low awareness of benefits of diet and physical activity and cannot afford healthier food choices (172). In Brazil, a steep increase in the prevalence of obesity has been seen in people belonging to low SES along with a decline in obesity among the higher-income groups (173–175).

It is important to note that most people belonging to low SES and living in rural areas of the developing countries are lean. However, when these people migrate from rural areas to large metropolitan cities, they quickly acquire risk factors associated with urbanization despite remaining in the same SES as the previous habitat. Specifically they start smoking and consume alcohol, and their diets become imbalanced. Whereas some may still engage in physically intensive jobs, most of them are involved in sedentary jobs (e.g. stationary street hawkers). High prevalence rates of T2DM, obesity, insulin resistance, hypertension, atherogenic dyslipidemia, hyperhomocysteinemia and endothelial dysfunction have been reported by us in this intracountry rural-to-urban migrant population settled in urban slums (29, 90, 176–180). In particular, significant clustering of cardiovas-

cular risk factors was seen in postmenopausal women living in urban slums in India (48, 49, 176, 181). Similar findings have been noted in the urban slum population in Thailand (130, 182).

Recently, increase in obesity and cardiovascular risk factors have been seen even in rural areas of some developing countries (183). It is important to note that many of so-called rural areas are no longer truly rural, and people are increasingly becoming urbanized and mechanized even in locations far from cities. Interestingly, this phenomenon to some extent, also appears to be driven by the so-called 'remittance economy' (money sent home from family members who have become affluent after migration, which leads to relative affluence, even in rural farm-based communities) (147).

### Sociocultural factors

Social stigma against obesity and obsession in some developed countries to remain lean has probably helped limit obesity to some extent. However, this psychosocial attitude toward obesity is not seen in many developing countries. Studies in African-Americans show a lack of social pressure to be thin and reduced stigma toward obesity, leading to higher levels of body satisfaction and acceptance of obesity by the individual and community (184–186). For example, Gambian populations were reported to be more obesity tolerant (acceptance of obese body size as normal) than African-Americans and much more tolerant than white Americans (187). Polynesians, who have one of the highest prevalence rates of obesity in the world, equate large body size with power, beauty, and affluence (188). Similarly, there is a general misconception in parents in India and other developing countries that an obese child is a healthy child, and hence, feel that it is important to feed him/her in excess (46). Furthermore, mothers in India often have traditional belief that feeding oils, *ghee* (clarified butter), and butter to child would be beneficial to growth and impart strength.

Another important factor is increasing pressure on children to perform in academics often forced by parents and teachers, which leads to reluctance of child to take part in sports or any other form of physical activity. Specifically, the majority of children in India are physically inactive when they are studying in classes when major examinations are held (46). Lack of playfields at school and open spaces around home, and decreased stress on games and physical training in schools has further led to decline in physical activity in children. In some developing countries, due to increasing crimes, parents do not allow children to play outside alone. Lastly, but significantly, a steep increase in sedentary activities like television viewing and computer usage has substantially contributed to a rise in obesity in children (46).

### Economic cost of obesity and T2DM in developing countries

Disability, decreased quality of life, greater use of health care facilities, and increased absenteeism have been reported with obesity (189). A strong relationship between BMI and decreased physical functioning with a reduction in overall productivity has been seen (190, 191). A 6-yr study demonstrated that an obese

person experiences a 50% increase in lost productivity and visits a doctor 88% more than a healthy person (192). All these factors increase health care expenditure, leading to slowing of economic growth and development and reduction of GDP; 3.5–7% of direct health care costs in the United States are due to obesity (193). When obesity causes T2DM or CVD, along with attendant complications, the direct and indirect costs rise exponentially and are unlikely to be balanced by meager health budgets and differently aligned health initiatives (more toward communicable diseases) in developing countries.

In developed countries, physical inactivity and consequent diseases, including obesity, are associated with considerable economic burden and account for 1.5–3.0% of total direct health care costs (157). In a review of costs pertaining to diet-related NCDs (CVD, T2DM, and hypertension), Popkin *et al.* (194) placed India in a less advantageous position compared with China. According to these authors, mortality due to diet-related NCDs is expected to increase to 43.3% of all deaths by 2020 in India. The cost will rise substantially as in 1995, total cost of diet-related-NCDs (health and productivity) was \$3.4 billion, costing 1.1% of the GDP in India (194). In Latin America and the Caribbean, the WHO estimated the combined annual direct and indirect costs of obesity-induced diabetes at \$65.2 billion in 2000 (195). In the Pacific islands, the economic consequences of obesity and T2DM have been devastating, costing \$1.95 million in

Tonga and \$13.6 million in Fiji, consuming nearly 60 and 39% of the health budgets, respectively (196). In mainland China, the total medical costs attributable to overweight and obesity in 2003 were estimated at 21.1 billion Yuans (approximately \$2.74 billion), accounting for 25.5% of the total medical costs for chronic diseases and 3.7% of national total medical costs (197). For obesity alone, the health expenses in China were nearly \$50 billion in 2000 and are projected to rise to \$112 billion by 2025 (198).

### Health interventions to reduce obesity in developing countries (Table 7)

Prevention of weight gain by selective interventions directed at population above average risk of developing obesity and targeted prevention directed at high risk individuals is strongly recommended. It is important to emphasize that strategies should be cost effective, culturally sensitive, and adapted to local practices, and messages imparted should be simple and in line with meager health budgets, widespread illiteracy, and unawareness in developing countries. The strategies should incorporate multiple stake holders and include government, physicians, nutritionists, schools, and nongovernmental organizations. Furthermore, intersectoral cooper-

**TABLE 7.** Public health interventions for obesity in developing countries

Item	Public health interventions
Monitoring and surveillance	Periodic monitoring of nutritional and obesity status of children and adults Maintain a nationwide database on secular trends in obesity and diabetes
Education	Nutrition and physical advice through audiovisual media and culturally conducive methods Endorsement of healthy lifestyle by prominent people and local champions
Community	Community fitness programs Organization and participation in health walks and health food festivals Information about nutrition to parents (particularly mothers) Children-specific nutrition information workshops for newly married women
Perinatal and neonatal period	Balanced nutrition to pregnant mothers Encourage breast-feeding Avoidance of catch-up obesity in children Maintenance of correct growth velocity under guidance of physicians Avoid excess nutrition to stunted children
School-based programs	High importance to physical activity Healthy foods in cafeteria, ban on sweetened beverages and energy-dense junk food Training of teachers regarding health education Incorporation of more knowledge about nutrition and physical activity and nutrition-related diseases in school curriculum
Workplace	Establishment of fitness rooms and gyms Healthy food in cafeteria Fitness as incentive to promotions
National Health Authority	Creation of national task force for obesity Decrease in taxes and prices of fruits and vegetables More playgrounds, parks, walking and bicycle tracks Ban on food items containing high amount of trans-fatty acids Restriction of advertisement for commercial foods in television (television prime time and children's programs) Encouragement to transnational food companies to manufacture healthy snacks Banning of unfair nutrition claims for commercial products Disincentive for obese unfit persons in government services
Legislative	Food standards Food labeling Food policy: country-specific guidelines for healthy nutrition for adults and children

ation of multiple governmental departments including health, nutrition, education, agriculture, and legal is required. Clearly the potential of primary care-based programs is underused in developing countries. Contact with health professionals from an early age has been identified as one of the important strategies by the WHO for effective management of overweight and obese children. It is important to impart health education targeting mothers, who traditionally decide about dietary choices and oil purchases for the whole family in developing countries.

### Strategies for children

General strategies for obesity prevention in infants and young children should include promotion of breast-feeding and avoidance of use of added sugars and starches in feeding formula. Overfeeding should be avoided in stunted children. In older children, family-based and school-based health education programs (see below) incorporating healthy food choices and increased physical activity are required. Specific emphasis should be placed on limiting television viewing, promotion of fruits and vegetable intake, and restriction of energy-dense packaged snacks and sweetened carbonated beverages. Firm political will is required from the government and policy makers to carry out the following; ban on sales of junk food and sweetened beverages in and near school premises, mandatory requirement of physical fitness in schools, limiting advertisements of commercial foods on television during prime time, strict regulations regarding trans-fatty acids containing cooking oils for preparation of foods and snacks frequently consumed by children, and encouragement of research into preparation of healthy snacks by the TFCs. Greater emphasis should be placed on teaching about diet and physical exercise in school curriculum. Specifically, teenagers and young adults in urban areas (especially those studying in privately funded schools) and girls should be targeted because high prevalence of obesity has been reported in them (46).

Several interventional programs have been initiated to combat obesity and the metabolic syndrome in children in developed countries. Project SPARK, Planet Health, and PATHWAYS are a few such school-based interventional programs aimed at improving several aspects of obesity-related knowledge, attitudes, and practices and have shown promising results in developed countries (199–201). There is an urgent need of such programs in developing countries as well. Of 20 interventional Chinese studies focusing on the effect of physical activity, dietary intervention, and health education in obesity in children, 17 studies were reported to be effective in reducing obesity (202). On similar grounds, in India, we have initiated comprehensive intervention programs aiming at creating awareness of childhood obesity, the metabolic syndrome, healthy nutrition, and physical activity, namely CHETNA (Children Health Education Through Nutrition and Health Awareness program) and MARG (Medical Education for Children/Adolescents for Realistic Prevention of Obesity and Diabetes and for Healthy AGing) (203). The latter is a large-scale program initiated for the first time in South Asia (aiming to cover more than 500,000 children in 15 cities

in north India). In these programs, education regarding the beneficial effects of a healthy diet and increased physical activity is emphasized to children and also parents and teachers. Messages of healthy diets and lifestyle are given through attractive audiovisual programs and involvement of students in debates, skits, and drama related to health topics.

### Strategies for adults

Both individuals and the state in the developing countries need to make serious efforts to limit the growing morbidity due to obesity and related diseases. Awareness about healthy nutrition and lifestyle should be imparted by physicians and nongovernmental organizations through their networks along with endorsed messages by famous personalities (*e.g.* film celebrities, sportsmen, *etc.*). Governmental strategies should include laws to encourage healthy oil production, discourage trans-fatty acid-based food products, and initiate food labeling with nutritive values of contents. Import duties on unhealthy oils should be increased. The TFCs should be encouraged to produce products free from trans-fatty acids. The government should improve urban planning to create more parks and tracks for jogging and cycling and sport complexes to encourage locally popular sports like football, cricket, *etc.* Audiovisual media can play an important role by televising healthy messages emphasizing the importance of healthy diets and physical activity and awareness about the complications of obesity and diabetes.

### Conclusions

Prevalence of obesity and the metabolic syndrome has shown a rapid rise in developing countries in the past few decades and has led to increased risk of CVD and consequent morbidity and mortality. Because both undernutrition and overnutrition are seen simultaneously in developing countries, the double burden of diseases makes the situation more difficult. The various factors responsible for increasing NCDs are rapid nutrition transition, rural-to-urban migration, increasingly sedentary occupations and lifestyle, and maternal-fetal factors. Both genetic and environmental factors seem to contribute to it; however, the role of environment seems to be predominant. Health interventions required to prevent or reduce the morbidity/mortality need to be addressed in both children and adults. Interventions should be aimed at increasing the physical activity along with healthier food patterns and health education. Successful community-based intervention programs have been reported in developed countries, and a similar approach is required in developing countries. Reports of interventional programs in China and India, especially directed toward children, have shown encouraging results, but large-scale programs involving adults/children are required. Various other health strategies consisting of individual and community initiatives, backed up by governmental and legislative efforts, would also help in minimizing the increasing prevalence of obesity and the metabolic syndrome in developing countries.



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## References

- Misra A 2002 Overnutrition and nutritional deficiency contribute to metabolic syndrome and atherosclerosis in Asian Indians. *Nutrition* 18:702–703
- Reddy KS 2002 Cardiovascular diseases in the developing countries: dimensions, determinants, dynamics and directions for public health action. *Public Health Nutr* 5:231–237
- Grundy SM 2003 Inflammation, hypertension, and the metabolic syndrome. *JAMA* 290:3000–3002
- 2002 Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): final report. *Circulation* 106:3143–3421
- Mensah GA, Mokdad AH, Ford E, Narayan KM, Giles WH, Vinicor F, Deedwania PC 2004 Obesity, metabolic syndrome, and type 2 diabetes: emerging epidemics and their cardiovascular implications. *Cardiol Clin* 22:485–504
- Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC 1994 Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care* 17:961–969
- Rexrode KM, Carey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfer MJ, Willett WC, Manson JE 1998 Abdominal adiposity and coronary heart disease in women. *JAMA* 280:1843–1848
- 2008 International Monetary Fund, World Economic Outlook Database. Nominal GDP list of countries. Data for the year 2007
- Popkin BM 2002 The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr* 5:205–214
- Murray CJL, Lopez AD 1996 The global burden of disease. Boston: Harvard School of Public Health
- World Health Organization: Background. Available at: [http://www.who.int/nutrition/topics/2\\_background/en/index.html](http://www.who.int/nutrition/topics/2_background/en/index.html). Last accessed July 2008
- World Health Organization 1999 The World Health Report: making a Difference. Geneva: World Health Organization
- World Health Organization 1998 The World Health Report: life in the 21st century: a vision for all. Geneva: World Health Organization
- World Health Organization 2002 Diet, physical activity and health (documents A55/16 and A55/16 Corr. 1). Geneva: World Health Organization
- Wild S, Roglic G, Green A, Sicree R, King H 2004 Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care* 27:1047–1053
- Sicree R, Shaw J, Zimmet P 2006 Diabetes and impaired glucose tolerance in India. In: Gan D, ed. *Diabetes atlas*. 3rd ed. Kortrijk (Hevle), Belgium: International Diabetes Federation; 15–103
- Leeder S, Raymond S, Greenberg H, Liu H, Esson K 2004 A race against time: the challenge of cardiovascular disease in developing economies. New York: Columbia University
- Murray CJL, Lopez AD 1996 The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Boston: Harvard School of Public Health
- Yach D, Hawkes C, Gould CL, Hofman KJ 2004 The global burden of chronic diseases: overcoming impediments to prevention and control. *JAMA* 291:2616–2622
- Basnyat B, Rajapaksa LC 2004 Cardiovascular and infectious diseases in South Asia: the double whammy. *BMJ* 328:781
- Sattar N, McConnachie A, Shaper AG, Blauw GJ, Buckley BM, de Craen AJ, Ford I, Forouhi NG, Freeman DJ, Jukema JW, Lennon L, Macfarlane PW, Murphy MB, Packard CJ, Stott DJ, Westendorp RG, Whincup PH, Shepherd J, Wannamethee SG 2008 Can metabolic syndrome usefully predict cardiovascular disease and diabetes? Outcome data from two prospective studies. *Lancet* 371:1927–1935
- World Health Organization Controlling the global obesity epidemic. Available at: <http://www.who.int/nutrition/topics/obesity/en/index.html>. Last accessed July 2008
- Popkin BM, Doak CM 1998 The obesity epidemic is a worldwide phenomenon. *Nutr Rev* 56:106–114
- World Health Organization Global strategy on diet, physical activity and health. Available at: <http://www.who.int/dietphysicalactivity/publications/facts/obesity/en/>. Accessed July 2008
- World Health Organization 2000 The Asia-Pacific perspective. Redefining obesity and its treatment. International Diabetes Institute. Health Communications Australia Pty. Ltd. Available at: <http://www.obesityasiapacific.com/default.htm>. Accessed July 2008
- WHO Expert Consultation 2004 Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 363:157–163
- Dudeja V, Misra A, Pandey RM, Devina G, Kumar G, Vikram NK 2001 BMI does not accurately predict overweight in Asian Indians in northern India. *Br J Nutr* 86:105–112
- Popkin BM 2006 Global nutrition dynamics: the world is shifting rapidly toward a diet linked with noncommunicable diseases. *Am J Clin Nutr* 84:289–298
- Misra A, Sharma R, Pandey RM, Khanna N 2001 Adverse profile of dietary nutrients, anthropometry and lipids in urban slum dwellers of northern India. *Eur J Clin Nutr* 55:727–734
- Misra A, Vikram NK 2003 Clinical and pathophysiological consequences of abdominal adiposity and abdominal adipose tissue depots. *Nutrition* 19:457–466
- Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai Jr P, Razak F, Sharma AM, Anand SS 2005 Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 366:1640–1649
- Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L 2004 Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 364:937–952
- Balkau B, Deanfield JE, Despres JP, Bassand JP, Fox KA, Smith Jr SC, Barter P, Tan CE, Van Gaal L, Wittchen HU, Massien C, Haffner SM 2007 International Day for the Evaluation of Abdominal Obesity (IDEA): a study of waist circumference, cardiovascular disease, and diabetes mellitus in 168,000 primary care patients in 63 countries. *Circulation* 116:1942–1951
- Reddy KS, Prabhakaran D, Chaturvedi V, Jeemon P, Thankappan KR, Ramakrishnan L, Mohan BV, Pandav CS, Ahmed FU, Joshi PP, Meera R, Amin RB, Ahuja RC, Das MS, Jaison TM 2006 Methods for establishing a surveillance system for cardiovascular diseases in Indian industrial populations. *Bull World Health Organ* 84:461–469
- Oh JY, Hong YS, Sung YA, Barrett-Connor E 2004 Prevalence and factor analysis of metabolic syndrome in an urban Korean population. *Diabetes Care* 27:2027–2032
- Fezeu L, Minkoulou E, Balkau B, Kengne AP, Awah P, Unwin N, Alberti GK, Mbanya JC 2006 Association between socioeconomic status and adiposity in urban Cameroon. *Int J Epidemiol* 35:105–111
- Florez H, Silva E, Fernandez V, Ryder E, Sulbaran T, Campos G, Calmon G, Clavel E, Castillo-Florez S, Goldberg R 2005 Prevalence and risk factors associated with the metabolic syndrome and dyslipidemia in White, Black, Amerindian and Mixed Hispanics in Zulia State, Venezuela. *Diabetes Res Clin Pract* 69:63–77
- Al-Lawati JA, Mohammed AJ, Al-Hinai HQ, Jousilahti P 2003 Prevalence of the metabolic syndrome among Omani adults. *Diabetes Care* 26:1781–1785
- Tappy L, Bovet P, Shamlaye C 1991 Prevalence of diabetes and obesity in the adult population of the Seychelles. *Diabet Med* 8:448–452
- Gu D, Reynolds K, Wu X, Chen J, Duan X, Reynolds RF, Whelton PK, He J 2005 Prevalence of the metabolic syndrome and overweight among adults in China. *Lancet* 365:1398–1405
- Azizi F, Salehi P, Etemadi A, Zahedi-Asl S 2003 Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes Res Clin Pract* 61:29–37
- Wijendran V, Bendel RB, Couch SC, Philipson EH, Thomsen K, Zhang X, Lammi-Keefe CJ 1999 Maternal plasma phospholipid polyunsaturated fatty acids in pregnancy with and without gestational diabetes mellitus: relations with maternal factors. *Am J Clin Nutr* 70:53–61
- Martorell R, Kettel Khan L, Hughes ML, Grummer-Strawn LM 2000 Overweight and obesity in preschool children from developing countries. *Int J Obes Relat Metab Disord* 24:959–967

44. de Onis M, Blossner M 2000 Prevalence and trends of overweight among preschool children in developing countries. *Am J Clin Nutr* 72:1032–1039
45. Wang Y, Monteiro C, Popkin BM 2002 Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. *Am J Clin Nutr* 75:971–977
46. Bhardwaj S, Misra A, Khurana, Gulati S, Shah P, Vikram NK 2008 Childhood obesity in Asian Indians: a burgeoning cause of insulin resistance, diabetes and sub-clinical inflammation. *Asia Pac J Clin Nutr* 17(Suppl 1): 172–175
47. Marquezzine GF, Oliveira CM, Pereira AC, Krieger JE, Mill JG 2008 Metabolic syndrome determinants in an urban population from Brazil: social class and gender-specific interaction. *Int J Cardiol* 129:259–265
48. Misra A, Chaudhary D, Vikram NK, Mittal V, Devi JR, Pandey RM, Khanna N, Sharma R, Peshin S 2002 Insulin resistance and clustering of atherogenic risk factors in women belonging to low socio-economic strata in urban slums of North India. *Diabetes Res Clin Pract* 56:73–75
49. Wasir JS, Misra A 2004 The metabolic syndrome in Asian Indians: the impact of nutritional and socio-economic transition in India. *Metab Syndr Relat Disord* 2:14–23
50. Misra A, Misra R, Wijesuriya M 2006 The metabolic syndrome in South Asians. In: Mohan V, Rao HR, Gundu HR, eds. *Type 2 diabetes in South Asians. Epidemiology, risk factors and prevention*. New Delhi: Jaypee Brothers; 76–96
51. Gupta R, Sarna M, Thanvi J, Rastogi P, Kaul V, Gupta VP 2004 High prevalence of multiple coronary risk factors in Punjabi Bhatia community: Jaipur Heart Watch-3. *Indian Heart J* 56:646–652
52. Tan CE, Ma S, Wai D, Chew SK, Tai ES 2004 Can we apply the national cholesterol education program adult treatment panel definition of the metabolic syndrome to Asians? *Diabetes Care* 27:1182–1186
53. Fan JG, Zhu J, Li XJ, Chen L, Lu YS, Li L, Dai F, Li F, Chen SY 2005 Fatty liver and the metabolic syndrome among Shanghai adults. *J Gastroenterol Hepatol* 20:1825–1832
54. Ko GT, Tang JS 2007 Metabolic syndrome in the Hong Kong community: the United Christian Netherlands Community Health Service primary healthcare programme, 2001–2002. *Singapore Med J* 48:1111–1116
55. Zimmet P, Alberti KG, Kaufman F, Tajima N, Silink M, Arslanian S, Wong G, Bennett P, Shaw J, Caprio S 2007 The metabolic syndrome in children and adolescents—an IDF consensus report. *Pediatr Diabetes* 8:299–306
56. The IDF consensus definition of the metabolic syndrome in children and adolescents. Available at: [http://www.idf.org/webdata/docs/Mets\\_definition\\_children.pdf](http://www.idf.org/webdata/docs/Mets_definition_children.pdf). Last accessed July 2008
57. Chu NF, Rimm EB, Wang DJ, Liou HS, Shieh SM 1998 Clustering of cardiovascular disease risk factors among obese schoolchildren: the Taipei Children Heart Study. *Am J Clin Nutr* 67:1141–1146
58. Lee MS, Wahlqvist ML, Yu HL, Pan WH 2007 Hyperuricemia and metabolic syndrome in Taiwanese children. *Asia Pac J Clin Nutr* 16(Suppl 2):594–600
59. Li Y, Yang X, Zhai F, Kok FJ, Zhao W, Piao J, Zhang J, Cui Z, Ma G 2008 Prevalence of the metabolic syndrome in Chinese adolescents. *Br J Nutr* 99: 565–570
60. Li Y, Yang X, Zhai F, Piao J, Zhao W, Zhang J, Ma G 2008 Childhood obesity and its health consequence in China. *Obes Rev* 9(Suppl 1):82–86
61. Vikram NK, Misra A, Pandey RM, Luthra K, Wasir JS, Dhingra V 2005 Heterogeneous phenotypes of insulin resistance and its implications for defining metabolic syndrome in Asian Indian adolescents. *Atherosclerosis* 186: 193–199
62. Deurenberg P, Yap M, van Staveren WA 1998 Body mass index and percent body fat: a meta analysis among different ethnic groups. *Int J Obes Relat Metab Disord* 22:1164–1171
63. Deurenberg-Yap M, Schmidt G, van Staveren WA, Deurenberg P 2000 The paradox of low body mass index and high body fat percentage among Chinese, Malays and Indians in Singapore. *Int J Obes Relat Metab Disord* 24: 1011–1017
64. Deurenberg-Yap M, Schmidt G, van Staveren WA, Hautvast JG, Deurenberg P 2001 Body fat measurement among Singaporean Chinese, Malays and Indians: a comparative study using a four-compartment model and different two-compartment models. *Br J Nutr* 85:491–498
65. Banerji MA, Faridi N, Alturi R, Chaiken RL, Lebovitz HE 1999 Body composition, visceral fat, leptin and insulin resistance in Asian Indian men. *J Clin Endocrinol Metab* 84:137–144
66. Misra A, Athiko D, Sharma R, Pandey RM, Khanna N 2002 Non-obese hyperlipidemic Asian northern Indian males have adverse anthropometric profile. *Nutr Metab Cardiovasc Dis* 12:178–183
67. Yajnik CS 2002 The lifecycle effects of nutrition and body size on adult adiposity, diabetes and cardiovascular disease. *Obes Rev* 3:217–224
68. Yajnik CS, Lubree HG, Rege SS, Naik SS, Deshpande JA, Deshpande SS, Joglekar CV, Yudkin JS 2002 Adiposity and hyperinsulinemia in Indians are present at birth. *J Clin Endocrinol Metab* 87:5575–5580
69. Lubree HG, Rege SS, Bhat DS, Raut KN, Panchnadikar A, Joglekar CV, Yajnik CS, Shetty P, Yudkin J 2002 Body fat and cardiovascular risk factors in Indian men in three geographical locations. *Food Nutr Bull* 23:146–149
70. Misra A 2003 We need ethnic-specific criteria for classification of BMI. In: Medeiros-Neto G, Halpern, A, Bouchrad C, eds. *Progress in Obesity Research: 9. Proceedings of the 9th International Congress on Obesity*, Sao Paulo, Brazil. London: John Libbey Eurotext Ltd.; 547–553
71. Misra A, Vikram NK 2004 Insulin resistance syndrome (metabolic syndrome) and obesity in Asian Indians: evidence and implications. *Nutrition* 20:482–491
72. Misra A 2003 Body composition and the metabolic syndrome in Asian Indians: a saga of multiple adversities. *Natl Med J India* 16:3–7
73. Deurenberg-Yap M, Chew SK, Deurenberg P 2002 Elevated body fat percentage and cardiovascular risks at low body mass index levels among Singaporean Chinese, Malays and Indians. *Obes Rev* 3:209–215
74. Gurrici S, Hartriyanti Y, Hautvast JG, Deurenberg P 1998 Relationship between body fat and body mass index: differences between Indonesians and Dutch Caucasians. *Eur J Clin Nutr* 52:779–783
75. Deurenberg P, Deurenberg-Yap M, Gurrici S 2002 Asians are different from Caucasians and from each other in their body mass index/body fat percent relationship. *Obes Rev* 3:141–146
76. Ko GTC, Chan JCN, Cockram CS, Woo J 1999 Prediction of hypertension, diabetes, dyslipidemia or albuminuria using simple anthropometric indexes in Hong Kong Chinese. *Int J Obes* 23:1136–1142
77. Misra A, Misra R 2003 Asian Indians and insulin resistance syndrome: global perspective. *Metab Syndr Relat Disord* 1:277–285
78. Misra A, Pandey RM, Sinha S, Guleria R, Sridhar V, Dudeja V 2003 Receiver operating characteristics curve analysis of body fat, body mass index in dyslipidaemic Asian Indians. *Indian J Med Res* 117:170–179
79. Vikram NK, Misra A, Pandey RM, Dudeja V, Sinha S, Ramadevi J, Kumar A, Chaudhary D 2003 Anthropometry and body composition in northern Asian Indian patients with type 2 diabetes: receiver operating characteristics (ROC) curve analysis of body mass index with percentage body fat as standard. *Diab Nutr Metab* 16:32–40
80. Mohan V, Deepa M, Farooq S, Narayan KM, Datta M, Deepa R 2007 Anthropometric cut points for identification of cardiometabolic risk factors in an urban Asian Indian population. *Metabolism* 56:961–968
81. International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome. Available at: [http://www.idf.org/webdata/docs/MetS\\_def\\_update2006.pdf](http://www.idf.org/webdata/docs/MetS_def_update2006.pdf). Accessed July 2008
82. Misra A, Vikram NK 2008 Factors, definitions, predictive value, Asian Indian ethnicity: complexities of the metabolic syndrome. *Indian J Med Res* 127: 293–296
83. Miller GJ, Kotecha S, Wilkinson WH, Wilkes H, Stirling Y, Sanders TA, Broadhurst A, Allison J, Meade TW 1988 Dietary and other characteristics relevant for coronary heart disease in men of Indian, West Indian and European descent in London. *Atherosclerosis* 70:63–72
84. Miller GJ, Beckles GL, Maude GH, Carson DC, Alexis SD, Price SG, Byam NT 1989 Ethnicity and other characteristics predictive of coronary heart disease in a developing community: principal results of the St. James Survey, Trinidad. *Int J Epidemiol* 18:808–817
85. Chandalia M, Abate N, Garg A, Stray-Gundersen J, Grundy SM 1999 Relationship between generalized and upper body obesity to insulin resistance in Asian Indian men. *J Clin Endocrinol Metab* 84:2329–2335
86. Kamath SK, Hussain EA, Amin D, Mortillaro E, West B, Peterson CT, Aryee F, Murillo G, Alekel DL 1999 Cardiovascular disease risk factors in 2 distinct ethnic groups: Indian and Pakistani compared with American premenopausal women. *Am J Clin Nutr* 69:621–631
87. Kalhan R, Puthawala K, Agarwal S, Amini SB, Kalhan SC 2001 Altered lipid profile, leptin, insulin, and anthropometry in offspring of South Asian immigrants in the United States. *Metabolism* 50:1197–1202
88. Chowdhury B, Lantz H, Sjostrom L 1996 Computed tomography-determined body composition in relation to cardiovascular risk factors in Indian and matched Swedish males. *Metabolism* 45:634–644
89. Raji A, Seely EW, Arky RA, Simonson DC 2001 Body fat distribution and insulin resistance in healthy Asian Indians and Caucasians. *J Clin Endocrinol Metab* 86:5366–5371
90. Vikram NK, Pandey RM, Misra A, Sharma R, Devi JR, Khanna N 2003 Non-obese (body mass index <25 kg/m<sup>2</sup>) Asian Indians with normal waist circumference have high cardiovascular risk. *Nutrition* 19:503–509
91. Misra A, Vikram NK, Gupta R, Pandey RM, Wasir JS, Gupta VP 2006 Waist circumference cutoff points and action levels for Asian Indians for identification of abdominal obesity. *Int J Obes (Lond)* 30:106–111
92. Misra A, Wasir JS, Vikram NK 2005 Waist circumference criteria for the

- diagnosis of abdominal obesity are not applicable uniformly to all populations and ethnic groups. *Nutrition* 21:969–976
93. Okosun IS, Rotimi CN, Forrester TE, Fraser H, Osotimehin B, Muna WF, Cooper RS 2000 Predictive value of abdominal obesity cut-off points for hypertension in blacks from west African and Caribbean island nations. *Int J Obes Relat Metab Disord* 24:180–186
  94. Velasquez-Melendez G, Kac G, Valente JG, Tavares R, Silva CQ, Garcia ES 2002 Evaluation of waist circumference to predict general obesity and arterial hypertension in women in Greater Metropolitan Belo Horizonte, Brazil. *Cad Saude Publica* 18:765–771
  95. Berber A, Gomez-Santos R, Fanghanel G, Sanchez-Reyes L 2001 Anthropometric indexes in the prediction of type 2 diabetes mellitus, hypertension and dyslipidaemia in a Mexican population. *Int J Obes Relat Metab Disord* 25:1794–1799
  96. Mirmiran P, Esmailzadeh A, Azizi F 2004 Detection of cardiovascular risk factors by anthropometric measures in Tehranian adults: receiver operating characteristic (ROC) curve analysis. *Eur J Clin Nutr* 58:1110–1118
  97. Chandalia M, Lin P, Seenivasan T, Livingston EH, Snell PG, Grundy SM, Abate N 2007 Insulin resistance and body fat distribution in South Asian men compared to Caucasian men. *PLoS ONE* 2:e812
  98. Misra A, Vikram NK, Arya S, Pandey RM, Dhingra V, Chatterjee A, Dwivedi M, Sharma R, Luthra K, Guleria R, Talwar KK 2004 High prevalence of insulin resistance in postpubertal Asian Indian children is associated with adverse truncal body fat patterning, abdominal adiposity and excess body fat. *Int J Obes Relat Metab Disord* 28:1217–1226
  99. Garg A, Misra A 2002 Hepatic steatosis, insulin resistance, and adipose tissue disorders. *J Clin Endocrinol Metab* 87:3019–3022
  100. Petersen KF, Dufour S, Feng J, Befroy D, Dziura J, Dalla Man C, Cobelli C, Shulman GI 2006 Increased prevalence of insulin resistance and nonalcoholic fatty liver disease in Asian-Indian men. *Proc Natl Acad Sci USA* 103:18273–18277
  101. Sharma R, Sinha S, Danishad KA, Vikram NK, Gupta A, Ahuja V, Jagannathan NR, Pandey RM, Misra A, Investigation of hepatic gluconeogenesis pathway in non-diabetic Asian Indians with non-alcoholic fatty liver disease using *in vivo* ( $^{31}\text{P}$ ) phosphorus magnetic resonance spectroscopy. *Atherosclerosis*, in press
  102. Misra A, Sinha S, Kumar M, Jagannathan NR, Pandey RM 2003 Proton magnetic resonance spectroscopy study of soleus muscle in non-obese healthy and type 2 diabetic Asian Northern Indian males: high intramyocellular lipid content correlates with excess body fat and abdominal obesity. *Diabet Med* 20:361–367
  103. Sinha S, Misra A, Kumar V, Jagannathan NR, Bal CS, Pandey RM, Singhania R, Deepak 2004 Proton magnetic resonance spectroscopy and single photon emission computed tomography study of the brain in asymptomatic young hyperlipidaemic Asian Indians in North India show early abnormalities. *Clin Endocrinol (Oxf)* 61:182–189
  104. Misra A, Jaiswal A, Shakti D, Wasir J, Vikram NK, Pandey RM, Kondal D, Bhushan B 2008 Novel phenotypic markers and screening score for the metabolic syndrome in adult Asian Indians. *Diabetes Res Clin Pract* 79:e1–e5
  105. Ruderman N, Chisholm D, Pi-Sunyer X, Schneider S 1998 The metabolically obese, normal-weight individual revisited. *Diabetes* 47:699–713
  106. Dickinson S, Colagiuri S, Faramus E, Petocz P, Brand-Miller JC 2002 Postprandial hyperglycemia and insulin sensitivity differ among lean young adults of different ethnicities. *J Nutr* 132:2574–2579
  107. Misra A 2003 Revision of limits of body mass index to define overweight and obesity are needed for the Asian ethnic groups. *Int J Obes, Relat Metab Disord* 27:1294–1296
  108. Stevens J 2003 Ethnic-specific cutpoints for obesity vs country-specific guidelines for action. *Int J Obes Relat Metab Disord* 27:287–288
  109. Snehalatha C, Viswanathan V, Ramachandran A 2003 Cutoff values for normal anthropometric variables in Asian Indian adults. *Diabetes Care* 26:1380–1384
  110. Molarius A, Seidell JC, Sans S, Tuomilehto J, Kuulasmaa K 1999 Varying sensitivity of waist action levels to identify subjects with overweight or obesity in 19 populations of the WHO MONICA Project. *J Clin Epidemiol* 52:1213–1224
  111. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith Jr SC, Spertus JA, Costa F 2005 Diagnosis and management of the metabolic syndrome. An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Executive Summary. *Cardiol Rev* 13:322–327
  112. Einhorn D, Reaven GM, Cobin RH, Ford E, Ganda OP, Handelsman Y, Hellman R, Jellinger PS, Kendall D, Krauss RM, Neufeld ND, Petak SM, Rodbard HW, Seibel JA, Smith DA, Wilson PW 2003 American College of Endocrinology position statement on the insulin resistance syndrome. *Endocr Pract* 9:237–252
  113. Misra A, Wasir JS, Pandey RM 2005 An evaluation of candidate definitions of the metabolic syndrome in adult Asian Indians. *Diabetes Care* 28:398–403
  114. Banerjee D, Misra A 2007 Does using ethnic specific criteria improve the usefulness of the term metabolic syndrome? Controversies and suggestions. *Int J Obes (Lond)* 31:1340–1349
  115. Diabetes Prevention Program Research Group 2002 Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance. *Hypertension* 40:679–686
  116. Zhang XF, Attia J, D'Este K, Yu XH, Wu XG 2004 Prevalence and magnitude of classical risk factors for coronary heart disease in a cohort of 4400 Chinese steelworkers over 13.5 years follow-up. *Eur J Cardiovasc Prev Rehabil* 11:113–120
  117. Araneta MR, Barrett-Connor E 2004 Subclinical coronary atherosclerosis in asymptomatic Filipino and white women. *Circulation* 110:2817–2823
  118. Cappuccio FP 1997 Ethnicity and cardiovascular risk: variations in people of African ancestry and South Asian origin. *J Hum Hypertens* 11:571–576
  119. Khattar RS, Swales JD, Senior R, Lahiri A 2000 Racial variation in cardiovascular morbidity and mortality in essential hypertension. *Heart* 83:267–271
  120. McLaughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, Simon J, Krauss RM 2005 Is there a simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? *Am J Cardiol* 96:399–404
  121. Sone H, Mizuno S, Fujii H, Yoshimura Y, Yamasaki Y, Ishibashi S, Katayama S, Saito Y, Ito H, Ohashi Y, Akanuma Y, Yamada N 2005 Is the diagnosis of metabolic syndrome useful for predicting cardiovascular disease in Asian diabetic patients? Analysis from the Japan Diabetes Complications Study. *Diabetes Care* 28:1463–1471
  122. Haffner SM, D'Agostino Jr R, Goff D, Howard B, Festa A, Saad MF, Mykkanen L 1999 LDL size in African Americans, Hispanics, and non-Hispanic whites: the insulin resistance atherosclerosis study. *Arterioscler Thromb Vasc Biol* 19:2234–2240
  123. Rewers M, Shetterly SM, Hoag S, Baxter J, Marshall J, Hamman RF 1993 Is the risk of coronary heart disease lower in Hispanics than in non-Hispanic whites? The San Luis Valley Diabetes Study. *Ethn Dis* 3:44–54
  124. Rewers M, Shetterly SM, Baxter J, Marshall JA, Hamman RF 1992 Prevalence of coronary heart disease in subjects with normal and impaired glucose tolerance and non-insulin-dependent diabetes mellitus in a biethnic Colorado population. The San Luis Valley Diabetes Study. *Am J Epidemiol* 135:1321–1330
  125. Ma S, Cutter J, Tan CE, Chew SK, Tai ES 2003 Associations of diabetes mellitus and ethnicity with mortality in a multiethnic Asian population: data from the 1992 Singapore National Health Survey. *Am J Epidemiol* 158:543–552
  126. Cossrow N, Falkner B 2004 Race/ethnic issues in obesity and obesity-related comorbidities. *J Clin Endocrinol Metab* 89:2590–2594
  127. Hodge AM, Boyko EJ, de Courten M, Zimmet PZ, Chitson P, Tuomilehto J, Alberti KG 2001 Leptin and other components of the metabolic syndrome in Mauritius—a factor analysis. *Int J Obes Relat Metab Disord* 25:126–131
  128. Hoffman DJ 2001 Obesity in developing countries: causes and implications. Food, Nutrition and Agriculture, 2001. A publication of the FAO food and nutrition division. Available at: <ftp://ftp.fao.org/docrep/fao/003/y0600m/y0600m04.pdf>. Last accessed July 2008
  129. Food and Agriculture Organization 2004 Globalization of food systems in developing countries: impact on food security and nutrition. Food and Nutrition paper. United Nations: Food and Agriculture Organization of the United Nations
  130. Sawaya AL, Dallal G, Solymos G, de Sousa MH, Ventura ML, Roberts SB, Sigulem DM 1995 Obesity and malnutrition in a shantytown population in the city of Sao Paulo, Brazil. *Obes Res* 3(Suppl 2):107s–115s
  131. Popkin BM, Du S 2003 Dynamics of the nutrition transition toward the animal foods sector in China and its implications: a worried perspective. *J Nutr* 133:3898S–3906S
  132. Popkin BM 2001 The nutrition transition and obesity in the developing world. *J Nutr* 131:871S–873S
  133. Hawkes C 2006 Uneven dietary development: linking the policies and processes of globalization with the nutrition transition, obesity and diet-related chronic diseases. *Global Health* 2:4
  134. Harris JM, Kaufman PR, Martinez SW, Price C 2002 The U.S. Food Marketing System, 2002. Washington, DC: U.S. Department of Agriculture Economic Research Service (agricultural economic report no. 811)
  135. Hawkes C 2005 The role of foreign direct investment in the nutrition transition. *Public Health Nutr* 8:357–365
  136. Pingali P, Khwaja Y 2004 Westernization of Asian diets and the transformation of food systems: implications for research and policy. Annual reports. McDonald Corp.

137. Snipes K 2004 Mexico exporter guide: annual 2004. GAIN report MX 4313. Washington, DC: United States Foreign Agricultural Service
138. 2001 Consulting C Mexico product brief: the Mexican market for processed foods 2001. GAIN report no. MX1099. Washington, DC, U.S. Department of Agriculture Foreign Agricultural Service
139. Arroyo P, Loria A, Mendez O 2004 Changes in the household calorie supply during the 1994 economic crisis in Mexico and its implications on the obesity epidemic. *Nutr Rev* 62:S163–S168
140. Misra A, Khurana L, Isharwal S, Bhardwaj S South Asian diets and insulin resistance. *Br J Nutr*, in press
141. Uauy R, Albala C, Kain J 2001 Obesity trends in Latin America: transiting from under- to overweight. *J Nutr* 131:893S–899S
142. Kain J, Vio F, Albala C 1998 Childhood nutrition in Chile: from deficit to excess. *Nutr Res* 18:1825–1835
143. Prentice AM, Rayco-Solon P, Moore SE 2005 Insights from the developing world: thrifty genotypes and thrifty phenotypes. *Proc Nutr Soc* 64:153–161
144. Prentice AM 2005 Early influences on human energy regulation: thrifty genotypes and thrifty phenotypes. *Physiol Behav* 86:640–645
145. Moore SE, Halsall I, Howarth D, Poskitt EM, Prentice AM 2001 Glucose, insulin and lipid metabolism in rural Gambians exposed to early malnutrition. *Diabet Med* 18:646–653
146. Prentice AM, Moore SE 2005 Early programming of adult diseases in resource poor countries. *Arch Dis Child* 90:429–432
147. Prentice AM 2006 The emerging epidemic of obesity in developing countries. *Int J Epidemiol* 35:93–99
148. Prentice AM 2003 Intrauterine factors, adiposity, and hyperinsulinaemia. *BMJ* 327:880–881
149. Hales CN, Barker DJ 2001 The thrifty phenotype hypothesis. *Br Med Bull* 60:5–20
150. Bavdekar A, Yajnik CS, Fall CH, Bapat S, Pandit AN, Deshpande V, Bhavs S, Kellingray SD, Joglekar C 1999 Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? *Diabetes* 48:2422–2429
151. Mi J, Law C, Zhang KL, Osmond C, Stein C, Barker D 2000 Effects of infant birthweight and maternal body mass index in pregnancy on components of the insulin resistance syndrome in China. *Ann Intern Med* 132:253–260
152. Sachdev HS, Fall CH, Osmond C, Lakshmy R, Dey Biswas SK, Leary SD, Reddy KS, Barker DJ, Bhargava SK 2005 Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurements of body mass index in childhood in the New Delhi birth cohort. *Am J Clin Nutr* 82:456–466
153. Yajnik CS, Deshpande SS, Jackson AA, Refsum H, Rao S, Fisher DJ, Bhat DS, Naik SS, Coyaji KJ, Joglekar CV, Joshi N, Lubree HG, Deshpande VU, Rege SS, Fall CH 2008 Vitamin B(12) and folate concentrations during pregnancy and insulin resistance in the offspring: the Pune Maternal Nutrition Study. *Diabetologia* 51:29–38
154. Yajnik CS, Fall CH, Coyaji KJ, Hirve SS, Rao S, Barker DJ, Joglekar C, Kellingray S 2003 Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. *Int J Obes Relat Metab Disord* 27:173–180
155. Tudor-Locke C, Ainsworth BE, Adair LS, Du S, Lee N, Popkin BM 2007 Cross-sectional comparison of physical activity and inactivity patterns in Chinese and Filipino youth. *Child Care Health Dev* 33:59–66
156. Guthold R, Ono T, Strong KL, Chatterji S, Morabia A 2008 Worldwide variability in physical inactivity a 51-country survey. *Am J Prev Med* 34:486–494
157. Oldridge NB 2008 Economic burden of physical inactivity: healthcare costs associated with cardiovascular disease. *Eur J Cardiovasc Prev Rehabil* 15:130–139
158. Matsudo V, Matsudo S, Andrade D, Araujo T, Andrade E, de Oliveira LC, Braggion G 2002 Promotion of physical activity in a developing country: the Agita Sao Paulo experience. *Public Health Nutr* 5:253–261
159. Lobelo F, Pate R, Parra D, Duperly J, Pratt M 2006 [Burden of mortality associated to physical inactivity in Bogota, Colombia]. *Rev Salud Publica (Bogota)* 8(Suppl 2):28–41
160. Kabagambe EK, Baylin A, Campos H 2007 Nonfatal acute myocardial infarction in Costa Rica: modifiable risk factors, population-attributable risks, and adherence to dietary guidelines. *Circulation* 115:1075–1081
161. Paeratakul S, Popkin BM, Keyou G, Adair LS, Stevens J 1998 Changes in diet and physical activity affect the body mass index of Chinese adults. *Int J Obes Relat Metab Disord* 22:424–431
162. Popkin BM 1999 Urbanization, lifestyle changes and the nutrition transition. *World Dev* 27:1905–1916
163. Ghosh A 2006 Effects of socio-economic and behavioural characteristics in explaining central obesity—a study on adult Asian Indians in Calcutta, India. *Coll Antropol* 30:265–271
164. Al-Nozha MM, Al-Hazzaa HM, Arafah MR, Al-Khadra A, Al-Mazrou YY, Al-Maathouq MA, Khan NB, Al-Marzouki K, Al-Harhi SS, Abdullah M, Al-Shahid MS 2007 Prevalence of physical activity and inactivity among Saudis aged 30–70 years. A population-based cross-sectional study. *Saudi Med J* 28:559–568
165. Daryani A, Berglund L, Andersson A, Kocurk T, Becker W, Vessby B 2005 Risk factors for coronary heart disease among immigrant women from Iran and Turkey, compared to women of Swedish ethnicity. *Ethn Dis* 15:213–220
166. Laxmaiah A, Nagalla B, Vijayaraghavan K, Nair M 2007 Factors affecting prevalence of overweight among 12- to 17-year-old urban adolescents in Hyderabad, India. *Obesity (Silver Spring)* 15:1384–1390
167. Al-Hazzaa HM, Al-Rasheedi AA 2007 Adiposity and physical activity levels among preschool children in Jeddah, Saudi Arabia. *Saudi Med J* 28:766–773
168. Kelishadi R, Sadri G, Tavasoli AA, Kahbazi M, Roohafza HR, Sadeghi M, Khosravi A, Sabet B, Amani A, Ansari R, Alikhassy H 2005 Cumulative prevalence of risk factors for atherosclerotic cardiovascular diseases in Iranian adolescents: IHHP-HHPC. *J Pediatr (Rio J)* 81:447–453
169. Henry CJ, Lightowler HJ, Al-Hourani HM 2004 Physical activity and levels of inactivity in adolescent females ages 11–16 years in the United Arab Emirates. *Am J Hum Biol* 16:346–353
170. Mozaffari H, Nabaei B 2007 Obesity and related risk factors. *Indian J Pediatr* 74:265–267
171. Sobal J, Stunkard AJ 1989 Socioeconomic status and obesity: a review of the literature. *Psychol Bull* 105:260–275
172. 2003 Diet, nutrition and the prevention of chronic diseases. *World Health Organ Tech Rep Ser* 916:i-viii, 1–149, back cover
173. Monteiro CA, Mondini L, de Souza AL, Popkin BM 1995 The nutrition transition in Brazil. *Eur J Clin Nutr* 49:105–113
174. Monteiro CA, Conde WL, Popkin BM 2004 The burden of disease from undernutrition and overnutrition in countries undergoing rapid nutrition transition: a view from Brazil. *Am J Public Health* 94:433–434
175. Monteiro CA, Conde WL, Popkin BM 2002 Is obesity replacing or adding to undernutrition? Evidence from different social classes in Brazil. *Public Health Nutr* 5:105–112
176. Misra A, Pandey RM, Devi JR, Sharma R, Vikram NK, Khanna N 2001 High prevalence of diabetes, obesity and dyslipidaemia in urban slum population in northern India. *Int J Obes Relat Metab Disord* 25:1722–1729
177. Misra A, Pandey RM, Sharma R 2002 Non-communicable diseases (diabetes, obesity and hyperlipidaemia) in urban slums. *Natl Med J India* 15:242–244
178. Misra A, Vikram NK, Pandey RM, Dwivedi M, Ahmad FU, Luthra K, Jain K, Khanna N, Devi JR, Sharma R, Guleria R 2002 Hyperhomocysteinemia, and low intakes of folic acid and vitamin B12 in urban North India. *Eur J Nutr* 41:68–77
179. Vikram NK, Pandey RM, Sharma R, Misra A 2003 Hyperhomocysteinemia in healthy Asian Indians. *Am J Hematol* 72:151–152
180. Sethi A, Misra A, Pandey RM, Luthra K, Devi JR, Sharma R, Khanna N, Dwivedi M, Vikram NK 2002 Soluble inter-cellular adhesion molecule-1 in urban Asian north Indians: relationships with anthropometric and metabolic covariates. *Dis Markers* 18:111–120
181. Wasir JS, Misra A, Vikram NK, Pandey RM, Luthra K 2007 C-reactive protein, obesity, and insulin resistance in postmenopausal women in urban slums of North India. *Diabetes and metabolic syndrome: Clin Res Rev* 1:83–89
182. Bunnag SC, Sitthi-Amorn C, Chandrasert S 1990 The prevalence of obesity, risk factors and associated diseases in Kleng Toey slum and Kleng Toey government apartment houses. *Diabetes Res Clin Pract* 10(Suppl 1):S81–S87
183. Chow CK, Naidu S, Raju K, Raju R, Joshi R, Sullivan D, Celermajer DS, Neal BC 2008 Significant lipid, adiposity and metabolic abnormalities amongst 4535 Indians from a developing region of rural Andhra Pradesh. *Atherosclerosis* 196:943–952
184. Littlewood R 2004 Commentary: globalization, culture, body image, and eating disorders. *Cult Med Psychiatry* 28:597–602
185. Flynn KJ, Fitzgibbon M 1998 Body images and obesity risk among black females: a review of the literature. *Ann Behav Med* 20:13–24
186. Kumanyika S, Wilson JF, Guilford-Davenport M 1993 Weight-related attitudes and behaviors of black women. *J Am Diet Assoc* 93:416–422
187. Siervo M, Grey P, Nyan OA, Prentice AM 2006 A pilot study on body image, attractiveness and body size in Gambians living in an urban community. *Eat Weight Disord* 11:100–109
188. Brewis AA, McGarvey ST, Jones J, Swinburn BA 1998 Perceptions of body size in Pacific Islanders. *Int J Obes Relat Metab Disord* 22:185–189
189. Thompson DL 2007 The costs of obesity: what occupational health nurses need to know. *AAOHN J* 55:265–270
190. Rissanen AM 1996 The economic and psychosocial consequences of obesity. *Ciba Found Symp* 201:194–201; discussion 201–206

191. Seidell JC 1998 Societal and personal costs of obesity. *Exp Clin Endocrinol Diabetes* 106(Suppl 2):7–9
192. Wolf AM, Colditz GA 1994 The cost of obesity: the U.S. perspective. *Pharmacoeconomics* 5:34–37
193. Allison DB, Zannolli R, Narayan KM 1999 The direct health care costs of obesity in the United States. *Am J Public Health* 89:1194–1199
194. Popkin BM, Horton S, Kim S, Mahal A, Shuigao J 2001 Trends in diet, nutritional status, and diet-related noncommunicable diseases in China and India: the economic costs of the nutrition transition. *Nutr Rev* 59:379–390
195. Runge CF 2007 Economic consequences of the obese. *Diabetes* 56:2668–2672
196. Dalton A, Crowley S 2002 Economic Impact of NCD in the Pacific Islands in obesity in the Pacific: too big to ignore. Based on the outcome of the Workshop on Obesity Prevention and Control Strategies in the Pacific, Samoa, September 2000, convened by the World Health Organization Regional Office for the Western Pacific, in collaboration with the Secretariat of the Pacific Community, the International Obesity Task Force, the United Nations Food and Agriculture Organization and the International Life Sciences Institute Available at: <http://www.wpro.who.int/NR/rdonlyres/B924BFA6-A061-43AE-8DCA-0AE82A8f66D2/0/obesityinthepacific.pdf>
197. Zhao W, Zhai Y, Hu J, Wang J, Yang Z, Kong L, Chen C 2008 Economic burden of obesity-related chronic diseases in mainland China. *Obes Rev* 9(Suppl 1):62–67
198. Popkin BM, Kim S, Rusev ER, Du S, Zizza C 2006 Measuring the full economic costs of diet, physical activity and obesity-related chronic diseases. *Obes Rev* 7:271–293
199. Sallis JF, McKenzie TL, Alcaraz JE, Kolody B, Hovell MF, Nader PR 1993 Project SPARK. Effects of physical education on adiposity in children. *Ann NY Acad Sci* 699:127–136
200. Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, Laird N 1999 Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Arch Pediatr Adolesc Med* 153:409–418
201. Caballero B, Clay T, Davis SM, Ethelbah B, Rock BH, Lohman T, Norman J, Story M, Stone EJ, Stephenson L, Stevens J 2003 Pathways: a school-based, randomized controlled trial for the prevention of obesity in American Indian schoolchildren. *Am J Clin Nutr* 78:1030–1038
202. Gao Y, Griffiths S, Chan EY, Community-based interventions to reduce overweight and obesity in China: a systematic review of the Chinese and English literature. *J Public Health (Oxf)*, in press
203. World Diabetes Foundation. Primary prevention in Indian schools. Available at: <http://www.worlddiabetesfoundation.org/composite-1279.htm>. Accessed October 2008
204. Mendez MA, Monteiro CA, Popkin BM 2005 Overweight exceeds underweight among women in most developing countries. *Am J Clin Nutr* 81:714–721
205. Shapo L, Pomerleau J, McKee M, Coker R, Yli A 2003 Body weight patterns in a country in transition: a population-based survey in Tirana City, Albania. *Public Health Nutr* 6:471–477
206. Monteiro CA, Conde WL, Popkin BM 2001 Independent effects of income and education on the risk of obesity in the Brazilian adult population. *J Nutr* 131:881S–886S
207. Zaman MM, Yoshiike N, Rouf MA, Syeed MH, Khan MR, Haque S, Mahtab H, Tanaka H 2001 Cardiovascular risk factors: distribution and prevalence in a rural population of Bangladesh. *J Cardiovasc Risk* 8:103–108
208. Du S, Lu B, Zhai F, Popkin BM 2002 The nutrition transition in China: a new stage of the Chinese diet. In: Caballero B, Popkin BM, eds. *The nutrition transition: diet and disease in the developing world*. London: Academic Press; 205–222
209. Jadue L, Vega J, Escobar MC, Delgado I, Garrido C, Lastra P, Espejo F, Peruga A 1999 [Risk factors for non communicable diseases: methods and global results of the CARMEN program basal survey]. *Rev Med Chil* 127:1004–1013
210. Fan JG, Cai XB, Li L, Li XJ, Dai F, Zhu J 2008 Alcohol consumption and metabolic syndrome among Shanghai adults: a randomized multistage stratified cluster sampling investigation. *World J Gastroenterol* 14:2418–2424
211. Shi Z, Hu X, Yuan B, Hu G, Pan X, Dai Y, Byles JE, Holmboe-Ottesen G 2008 Vegetable-rich food pattern is related to obesity in China. *Int J Obes (Lond)* 32:975–984
212. Sabanayagam C, Shankar A, Wong TY, Saw SM, Foster PJ 2007 Socioeconomic status and overweight/obesity in an adult Chinese population in Singapore. *J Epidemiol* 17:161–168
213. Pang W, Sun Z, Zheng L, Li J, Zhang X, Liu S, Xu C, Hu D, Sun Y 2008 Body mass index and the prevalence of prehypertension and hypertension in a Chinese rural population. *Intern Med* 47:893–897
214. Zhang X, Sun Z, Zheng L, Liu S, Xu C, Li J, Zhao F, Hu D, Sun Y 2008 Prevalence and associated factors of overweight and obesity in a Chinese rural population. *Obesity (Silver Spring)* 16:168–171
215. Galal OM 2002 The nutrition transition in Egypt: obesity, undernutrition and the food consumption context. *Public Health Nutr* 5:141–148
216. Dhurandhar NV, Kulkarni PR 1992 Prevalence of obesity in Bombay. *Int J Obes Relat Metab Disord* 16:367–375
217. Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Kothari K 2003 Prevalence of diabetes, impaired fasting glucose and insulin resistance syndrome in an urban Indian population. *Diabetes Res Clin Pract* 61:69–76
218. Prabhakaran D, Shah P, Chaturvedi V, Ramakrishnan L, Manhapra A, Reddy KS 2005 Cardiovascular risk factor prevalence among men in a large industry of northern India. *Natl Med J India* 18:59–65
219. Gupta R, Deedwania PC, Gupta A, Rastogi S, Panwar RB, Kothari K 2004 Prevalence of metabolic syndrome in an Indian urban population. *Int J Cardiol* 97:257–261
220. Deepa M, Farooq S, Deepa R, Manjula D, Mohan V 2006 Prevalence and significance of generalized and central body obesity in an urban Asian Indian population in Chennai, India (CURES: 47). *Eur J Clin Nutr*, in press
221. Gupta R, Sarna M, Thanvi J, Sharma V, Gupta VP 2007 Fasting glucose and cardiovascular risk factors in an urban population. *J Assoc Physicians India* 55:705–709
222. Park HS, Lee SY, Kim SM, Han JH, Kim DJ 2006 Prevalence of the metabolic syndrome among Korean adults according to the criteria of the International Diabetes Federation. *Diabetes Care* 29:933–934
223. Grabauskas V, Petkeviciene J, Klumbiene J, Vaisvalavicius V 2003 The prevalence of overweight and obesity in relation to social and behavioral factors (Lithuanian health behavior monitoring). *Medicina (Kaunas)* 39:1223–1230
224. Benjelloun S 2002 Nutrition transition in Morocco. *Public Health Nutr* 5:135–140
225. Hodge AM, Dowse GK, Gareeboo H, Tuomilehto J, Alberti KG, Zimmet PZ 1996 Incidence, increasing prevalence, and predictors of change in obesity and fat distribution over 5 years in the rapidly developing population of Mauritius. *Int J Obes Relat Metab Disord* 20:137–146
226. Dodani S, Mistry R, Khwaja A, Farooqi M, Qureshi R, Kazmi K 2004 Prevalence and awareness of risk factors and behaviours of coronary heart disease in an urban population of Karachi, the largest city of Pakistan: a community survey. *J Public Health (Oxf)* 26:245–249
227. Jacoby E, Goldstein J, Lopez A, Nunez E, Lopez T 2003 Social class, family, and life-style factors associated with overweight and obesity among adults in Peruvian cities. *Prev Med* 37:396–405
228. Jahns L, Baturin A, Popkin BM 2003 Obesity, diet, and poverty: trends in the Russian transition to market economy. *Eur J Clin Nutr* 57:1295–1302
229. Bovet P, Shamlaye C, Gabriel A, Riesen W, Paccaud F 2006 Prevalence of cardiovascular risk factors in a middle-income country and estimated cost of a treatment strategy. *BMC Public Health* 6:9
230. 1999 South Africa demographic and health survey. 1998 Preliminary report. Pretoria, South Africa: Department of Health, Macro International Inc., DHS Program
231. Hodge AM, Dowse GK, Toelupe P, Collins VR, Imo T, Zimmet PZ 1994 Dramatic increase in the prevalence of obesity in western Samoa over the 13 year period 1978–1991. *Int J Obes Relat Metab Disord* 18:419–428
232. Bourne LT, Lambert EV, Steyn K 2002 Where does the black population of South Africa stand on the nutrition transition? *Public Health Nutr* 5:157–162
233. Wijewardene K, Mohideen MR, Mendis S, Fernando DS, Kulathilaka T, Weerasekara D, Uluwitta P 2005 Prevalence of hypertension, diabetes and obesity: baseline findings of a population based survey in four provinces in Sri Lanka. *Ceylon Med J* 50:62–70
234. Kosulwat V 2002 The nutrition and health transition in Thailand. *Public Health Nutr* 5:183–189
235. Hirschler V, Gonzalez C, Talgham S, Jadzinsky M 2006 Do mothers of overweight Argentinean preschool children perceive them as such? *Pediatr Diabetes* 7:201–204
236. Silveira D, Taddei JA, Escrivao MA, Oliveira FL, Ancona-Lopez F 2006 Risk factors for overweight among Brazilian adolescents of low-income families: a case-control study. *Public Health Nutr* 9:421–428
237. Liu JM, Ye R, Li S, Ren A, Li Z, Liu Y 2007 Prevalence of overweight/obesity in Chinese children. *Arch Med Res* 38:882–886
238. Nunez-Rivas HP, Monge-Rojas R, Leon H, Rosello M 2003 Prevalence of overweight and obesity among Costa Rican elementary school children. *Rev Panam Salud Publica* 13:24–32
239. Moayeri H, Bidad K, Aghamohammadi A, Rabbani A, Anari S, Nazemi L, Gholami N, Zadhoush S, Hatmi ZN 2006 Overweight and obesity and their associated factors in adolescents in Tehran, Iran, 2004–2005. *Eur J Pediatr* 165:489–493

240. Lafta RK, Kadhim MJ 2005 Childhood obesity in Iraq: prevalence and possible risk factors. *Ann Saudi Med* 25:389–393
241. Jabre P, Sikias P, Khater-Menassa B, Baddoura R, Awada H 2005 Overweight children in Beirut: prevalence estimates and characteristics. *Child Care Health Dev* 31:159–165
242. Sumarni Mohd G, Muhammad Amir K, Ibrahim Md S, Mohd Rodi I, Izzuna Mudla MG, Nurziyana I 2006 Obesity among schoolchildren in Kuala Selangor: a cross-sectional study. *Trop Biomed* 23:148–154
243. El-Hazmi MA, Warsy AS 2002 The prevalence of obesity and overweight in 1–18-year-old Saudi children. *Ann Saudi Med* 22:303–307
244. Kruger R, Kruger HS, Macintyre UE 2006 The determinants of overweight and obesity among 10- to 15-year-old schoolchildren in the North West Province, South Africa—the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. *Public Health Nutr* 9:351–358
245. Wickramasinghe VP, Lamabadusuriya SP, Atapattu N, Sathyadas G, Kuruparanantha S, Karunarathne P 2004 Nutritional status of schoolchildren in an urban area of Sri Lanka. *Ceylon Med J* 49:114–118
246. Langendijk G, Wellings S, van Wyk M, Thompson SJ, McComb J, Chusilp K 2003 The prevalence of childhood obesity in primary school children in urban Khon Kaen, northeast Thailand. *Asia Pac J Clin Nutr* 12:66–72
247. Yang W, Reynolds K, Gu D, Chen J, He J 2007 A comparison of two proposed definitions for metabolic syndrome in the Chinese adult population. *Am J Med Sci* 334:184–189
248. Deepa R, Shanthirani CS, Premalatha G, Sastry NG, Mohan V 2002 Prevalence of insulin resistance syndrome in a selected south Indian population—the Chennai urban population study-7 [CUPS-7]. *Indian J Med Res* 115:118–127
249. Ramachandran A, Snehalatha C, Satyavani K, Sivasankari S, Vijay V 2003 Metabolic syndrome in urban Asian Indian adults—a population study using modified ATP III criteria. *Diabetes Res Clin Pract* 60:199–204
250. Deepa M, Farooq S, Datta M, Deepa R, Mohan V 2007 Prevalence of metabolic syndrome using WHO, ATP III and IDF definitions in Asian Indians: the Chennai Urban Rural Epidemiology Study (CURES-34). *Diabetes Metab Res Rev* 23:127–134
251. Park HS, Oh SW, Cho SI, Choi WH, Kim YS 2004 The metabolic syndrome and associated lifestyle factors among South Korean adults. *Int J Epidemiol* 33:328–336
252. Gustiene O, Slapikas R, Klumbiene J, Sakalauskiene G, Kubilius R, Bagdzeviciute S, Zaliunas R 2005 [The prevalence of metabolic syndrome in middle-aged in Kaunas population]. *Medicina (Kaunas)* 41:867–876
253. Cameron AJ, Shaw JE, Zimmet PZ, Chitson P, Alberti KGMM, Tuomilehto J 2003 Comparison of metabolic syndrome definitions in the prediction of diabetes over 5 years in Mauritius. *Diabetologia* 46:A145–A146
254. Aguilar-Salinas CA, Rojas R, Gomez-Perez FJ, Valles V, Rios-Torres JM, Franco A, Olaiz G, Rull JA, Sepulveda J 2003 Analysis of the agreement between the World Health Organization criteria and the National Cholesterol Education Program-III definition of the metabolic syndrome: results from a population-based survey. *Diabetes Care* 26:1635
255. Ozsahin AK, Gokcel A, Sezgin N, Akbaba M, Guvener N, Ozisik L, Karademir BM 2004 Prevalence of the metabolic syndrome in a Turkish adult population. *Diabetes Nutr Metab* 17:230–234
256. Onat A, Ceyhan K, Basar O, Erer B, Toprak S, Sansoy V 2002 Metabolic syndrome: major impact on coronary risk in a population with low cholesterol levels—a prospective and cross-sectional evaluation. *Atherosclerosis* 165:285–292
257. Haddad L, Ruel MT, Garrett JL 1999 Are urban poverty and undernutrition growing? Some newly assembled evidence: Food Consumption and Nutrition Division, International Food Policy Research Institute. Washington, DC. Available at: <http://www.ifpri.org/divs/fcnd/dp/papers/dp63.pdf>. Last accessed July 2008
258. Tee ES, Khor SC, Ooi HE, Young SI, Zakariah O, Zolkafli H 2002 Regional study of nutritional status of urban primary schoolchildren. Kuala Lumpur, Malaysia. *Food Nutr Bull* 23:41–47