

Review

Migration and its impact on adiposity and type 2 diabetes

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Abstract

In this review, we discuss the impact of migration on the incidence and prevalence of obesity and type 2 diabetes mellitus (T2DM) in different ethnic groups and populations. We also analyze the determinants of such phenomena in view of the global increase in the migration and escalating prevalence of obesity and T2DM. The risk escalation of the obesity and T2DM followed a gradient, as migrants (Blacks, Hispanics, Chinese, South Asians, etc.) became more affluent and urbanized, indicating an important role of environmental factors. A stepwise increase in the prevalence of obesity in Blacks along the path of migration (5% in Nigeria, 23% in Jamaica, and 39% in the United States) is a classic example. Furthermore, South Asian migrants, who are particularly predisposed to develop insulin resistance and T2DM, showed nearly four times prevalence rates of T2DM than rural sedentary populations. Similar observations were also reported in intracountry migrants and resettled indigenous populations. The determinants were found to include nutrition transition, physical inactivity, gene-environment interaction, stress, and other factors such as ethnic susceptibility. However, certain contradictory trends were also seen in some migrant communities and have been explained by various phenomena such as healthy migrant effect, “salmon bias”, and adherence to traditional diets.

A review of the evidence suggests a critical role of environmental factors in conferring an increased risk of obesity and T2DM. The important contributory factors to this phenomenon were urbanization, mechanization, and changes in nutrition and lifestyle behaviors, but the role of stress and as yet unknown factors remain to be determined. © 2007 Elsevier Inc. All rights reserved.

Keywords:

Migration; Type 2 diabetes; Obesity; Physical activity; Nutrition; Ethnicity

Introduction

Migration of populations is an ancient phenomenon, happening from the time of our ancestors in Africa. A variety of reasons may contribute to this phenomenon. Migration can occur due to “push factors,” i.e., poverty, war, etc., in the native country, or “pull factors,” i.e., toward better educational, financial, or career opportunities in the country of migration. One of the largest migrations occurred to the United States from Europe in the 18th and 19th centuries.

More recently, migration has become much more frequent with increasing global opportunities in business and technology and frequent international travel. It provides a unique opportunity to study the influence of various environmental factors on secular trends and risk factors of various predominantly environmentally influenced diseases such as obesity and type 2 diabetes mellitus (T2DM). The purpose of this paper is to review the effect of migration on the incidence and prevalence of T2DM and obesity in different ethnic groups and populations. We also discuss the various determinants of such phenomena in different migrant ethnic groups followed by a discussion on migration-linked determinants of adiposity, T2DM, and cardiovascular disease (CVD).

The search strategy for literature review was as follows. An electronic literature search was carried out by using the terms “immigrants or migrants and obesity and diabetes or type 2 diabetes mellitus” in the databases of 1) PubMed

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(National Library of Medicine, Bethesda, MD, USA) from 1966 to March 2007 and 2) Current Contents (Institute for Scientific Information, Thomson Scientific, Philadelphia, PA, USA). The studies were selected based on their relevance and importance in the context of the topic. Those appearing in high-impact and internationally acknowledged journals were given preference. A manual search of the relevant articles from the published references was also conducted. Databases, non-indexed publications, and web sites of reputed medical research and public health institutions of the United States, United Kingdom, Canada, India, and other countries were also researched using general web-based search engines.

Definition and patterns

The term “migrant populations” usually refers to migration from a native country to another country; however, in a broader perspective, it may be applied to the migration from one habitat to another within a country (e.g., rural-to-urban migration). In the early 19th century, migrants represented mainly people who were laborers, farm workers, refugees, etc., but lately, academically accomplished, and economically well-off migrants have begun to settle in developed countries due to industrial globalization and availability of professional opportunities in open-market economies. With an increasing period of stay and successive generations being born in the adopted countries, the original language, ethnic, and lifestyle practices are gradually replaced by locally prevalent practices, a process termed “acculturation.” Hence, for scientific research, comparisons are made between morbidity and mortality data of the migrant populations with similar data of people living in the original country of residence (“sedentees”). In such a situation, the essential and basic assumption is that the migrants are true representatives of the population of the country of origin and have a genetic make-up similar to those of sedentees.

Migration and diseases

It has been long observed that the processes of urbanization or westernization associated with migration lead to the availability and abundance of calorie-dense/low-fiber foods and the adoption of sedentary lifestyles. This has consequently led to increased risks of morbidity and mortality from chronic diet and lifestyle-related diseases. The pattern has been seen in cross-country and intracountry migration in developed and developing countries.

Several studies during the past four decades have showed an escalation of risk in CVD and other diseases as migrants have become more affluent and urbanized. Haenszel and Kurihara [1] studied first-generation (“Issei”) and second-generation (“Nisei”) Japanese migrants to Hawaii and the western United States and demonstrated that the mortality

rates of certain cancers (e.g., colonic carcinoma) in the migrants had become nearly equal to those seen in U.S. Caucasians. Standardized mortality ratios of intestinal cancer for Japanese Issei migrants and U.S. White men were 374 and 489, respectively. Conversely, breast cancer risks were low and similar to those of the sedentary Japanese population.

In other studies, Marmot et al. [2] and Robertson et al. [3] used comparable examining methods in 11 900 men of Japanese ancestry 45–69 y of age and reportedly found the age-adjusted prevalence rates for coronary heart disease (per 1000 individuals) as determined by electrocardiogram to be 5.3 in Japan, 5.2 in Hawaii, and 10.8 in California. This pattern was similar to the trends seen for the total cholesterol concentrations in these populations. These observations, despite the widely prevalent smoking practices observed in Japan, highlighted the effect of migration on cardiovascular risk escalation because the results were unexplainable by taking only the conventional risk factors into account. The risk of myocardial infarction (MI) has been found to be lower in southern than in northern European countries, and the lower rate of MI in the Mediterranean regions of Europe has been suggested to be due to a potential role of the traditional Mediterranean diets in the prevention of MI. But, in the previous two decades, a tendency to adopt “westernized” food habits, due to migratory factors, even in southern regions of Europe is reflected by an increase in the prevalence of obesity [4].

However, the studies on migrant populations have not yielded consistently converging scientific viewpoints. Interestingly, contradictory reports of healthier or less healthy migrant populations than the sedentees have been demonstrated [5,6]. The “epidemiologic paradox” of low all-cause mortality in migrant Hispanics living in the southwest United States despite their poor socioeconomic stratum has intrigued epidemiologists [5]. Other investigators have also reported increased CVD mortality risk in U.S.-born Mexican Americans than in Mexican immigrants despite low socioeconomic status [7], which also showed a similar paradox. The argument in this case has been that such lower mortality rates, particularly in recently migrated individuals, are probably due to more favorable health behavior (e.g., continued laborious lifestyle and frugal diets of recently migrated people) or selection of “healthy migrants.” The “healthy-migrant effect” could also be due to strict health stipulations and eligibility criteria required for migration to that country (e.g., Australia) [8]. Findings of foreign-born Latina women being less likely to have low-birth-weight babies than U.S.-born Latina women were also attributed to the fittest people being selected during the immigration process [9]. Conversely, such data may be due to “salmon bias.” This phenomenon is believed to be due to the desire of elderly migrants to die in their birthplace. Such individuals become “statistically immortal” because these deaths are not included in the mortality data of the adopted country. However, reliable data confirming the salmon-bias effect

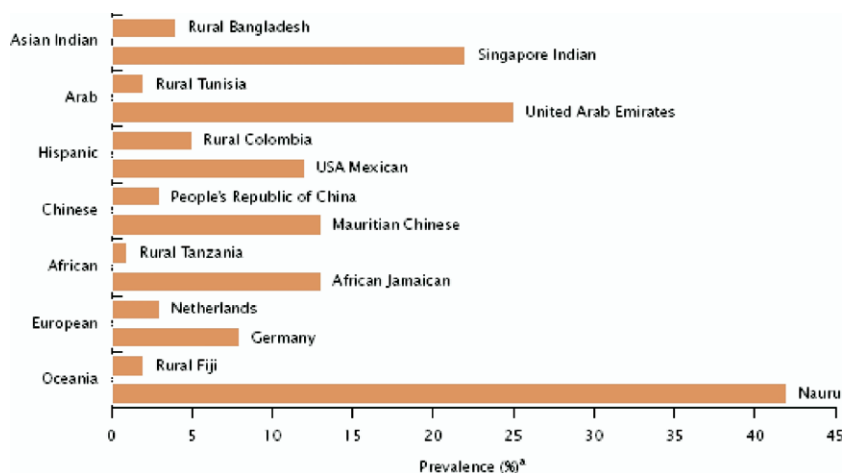


Fig. 1. Prevalence estimates of diabetes in select populations and regions. Reproduced with permission from International Diabetes Federation [11].

and its statistical significant contribution are still lacking. It is also possible that prevalence rates of some diseases do not increase after migration, probably suggestive of a genetic interplay or a lack of environmental effects on its pathophysiology, whereas escalation of others, particularly the lifestyle-related diseases, largely bearing environmental influences do show an increase. Overall, the healthy-migrant effect may be due to multiple factors, but why it is applicable specifically to some migrant populations is not clear.

Obesity and T2DM in different ethnic migrant populations

Since Taylor and Zimmet [10] reviewed the impact of migrant studies in the epidemiology of T2DM two decades ago, no review of this subject has been published. In view of the global increase in migration and the escalating prevalence of T2DM and the variable increase in various regions of the world (well documented by the World Health Organization [WHO] and the International Diabetes Federation (Fig. 1 [11]), the issue assumes even greater importance in terms of studying the recent trends. In the following sections, we present evidence regarding various migrant populations, including Blacks, Hispanics, South Asians, Japanese, Chinese, Turks, and others.

Blacks

During the four centuries after Columbus arrived in the Caribbean, nearly 12 million Black people left sub-Saharan Africa for the Western Hemisphere as involuntary migrants, and the destinations included the Caribbean islands, the Americas, and Europe. African migrants provided most of the labor in the mines, plantations, and construction works of the New World, and these African migrants and their descendants constituted the African Diaspora. The people of African origin in these countries, however, usually belong

to a low socioeconomic stratum and generally have unsatisfactory health indicators. Many investigations have highlighted the health and nutritional transitions of the African Diaspora. Although populations in many countries in western Africa are undernourished or have started experiencing a nutritional transition, African Americans and Blacks in the United Kingdom are at the other end of the spectrum by showing caloric excess and its consequences [12].

In a series of studies, Luke et al. [12–14], Rotini et al. [15], and Okosun et al. [12–16] compared body composition and metabolic covariates in the people of African origin residing in Nigeria, the Caribbean, and the United States (countries nearly approximating the path of migration of Blacks from Africa several centuries ago). The investigators used a standardized protocol of anthropometric measurements on 1054 Black men and women in a cross-sectional study. They reported that, for both genders, indices of obesity were lowest in people from Nigeria and highest in people living in the United States, and values in people from Jamaica were intermediate [13]. For example, the respective average values of body mass index (BMI) were 23, 26, and 28 kg/m² in people from Nigeria, Jamaica, and the United States, respectively [15]. A similar study of 4623 men and women of the African Diaspora showed stepwise increases in prevalences of obesity: 5% in Nigeria, 23% in Jamaica, and 39% in the United States [14]. Others have reported similar observations for insulin resistance and T2DM in people of African origin [15,17].

The prevalence of hypertension (commonly seen as an associated risk factor with obesity and T2DM) followed a similar gradient as seen with metabolic variables, being lowest in those residing in rural Africa in an age-stratified sampling of 9102 men and women 25–74 y of age [18]. These observations point to the fact that the prevalence of chronic diseases in the migrant population differed in three different migrant regions, probably due to different environmental influences, despite the similar genetic origin of the sample.

Hispanics

Hispanics migrated to the United States four centuries ago, with further migration occurring between 1910 and 1930 and during the mid-19th century. By 2000, Hispanics had become one of the largest minority groups in the United States and their numbers are still growing rapidly. However, Hispanics have remained an economically poor ethnic group in the United States. It has been a matter of debate that, despite language and cultural barriers and poor health care access, Hispanics have low all-cause mortality. This fact stands vindicated based on the national mortality statistics and a cohort study involving 700 000 respondents in a 9-y follow up of the mortality data matched to the National Death Index [19,20]. However, higher sex- and age-adjusted mortality rates (per 1000 person-years) have been reported in U.S.-born Mexican Americans (5.7) compared with non-Hispanic Whites (3.8) and Mexican Americans born elsewhere (3.6) [21]. This could be due to the fact that the process of acculturation, with adoption of a “Westernized” dietary and lifestyle habits over a long span of time, led to increased morbidity and, hence, mortality in U.S.-born Mexican Americans compared with foreign-born Mexican American migrants [21].

Recently, several reports have emphasized the adverse profile of obesity-related diseases in Hispanic populations in the United States. A low level of acculturation in Hispanics has been associated with high rates of obesity, abdominal obesity, and other cardiovascular risk factors. The data analysis of the Third National Health and Nutrition Examination Survey (1988–94), involving 2781 men and women (25–64 y of age) and a Hispanic survey of 5180 adult men and women (18–74 y of age) clearly demonstrated that the average value of BMI in successive generations of Hispanic migrants showed an increase when using linear regression models of BMI covariates [22,23]. Mean values of BMI were 25.9, 26.0, and 25.5 kg/m² in men and 26.6, 25.9, and 26.2 kg/m² in women of Mexican American, Cuban American, and Puerto Rican origins, respectively. However, relative to the first generation, the increase in BMI units was 1.15 in men and 1.76 in women in the second generation and 0.83 in men and 1.83 in women in the third generation [23]. These investigators used multivariate logit techniques to understand the ethnic, age, gender, and intergenerational patterns of adolescent obesity [23,24]. The high rates of obesity and T2DM in the migrant Hispanic populations were also demonstrated in New Mexico in the United States more than a decade ago in a study of 1175 Hispanic adults, which suggested the importance of genetic factors [25]. An increased occurrence of T2DM and obesity in Hispanics despite a low level of acculturation is also explained by the hypothesis that, because Hispanics are genetically admixed with American Indians, this phenomenon may be genetic in origin and result from genes derived from American Indians [25]. The cross-sectional survey data from the Third National Health and Nutrition Examination Survey (1988–

1994) further showed a remarkably high prevalence of T2DM, dyslipidemia, and the metabolic syndrome in Mexican Americans [26]. The unadjusted and age-adjusted prevalences of the metabolic syndrome were 21.8% and 23.7%, respectively, for men and women in the general U.S. population, with Mexican Americans being reported to have the highest age-adjusted prevalence of the metabolic syndrome (31.9%) [26]. These observations suggest that, for development of obesity and T2DM, an interplay among environmental, social, and genetic factors is important in migrant populations.

South Asians

Under the British occupation, South Asians migrated to various commonwealth countries as laborers to work at the plantations or as industrial workers. Subsequently, Asian Indians migrated to other countries also as traders, businessmen, and technologically accomplished engineers and doctors.

Lifestyle changes and increasing affluence have caused a high prevalence of obesity, insulin resistance, and T2DM in migrant South Asians [27–29]. The prevalence of the metabolic syndrome and CVD was found to be highest in South Asians in the United Kingdom in a cross-sectional study [30] (Fig. 2). The average values of BMI, blood pressure, lipids, blood glucose, and insulin resistance of the migrant Asian Indians tended to be higher than those of urban- or rural-based sedentees in India [28,31–33]. For example, migrant Asian Indians living in the United Kingdom were more obese, had higher levels of blood pressure, total cholesterol, and blood glucose, and were more insulin resistant

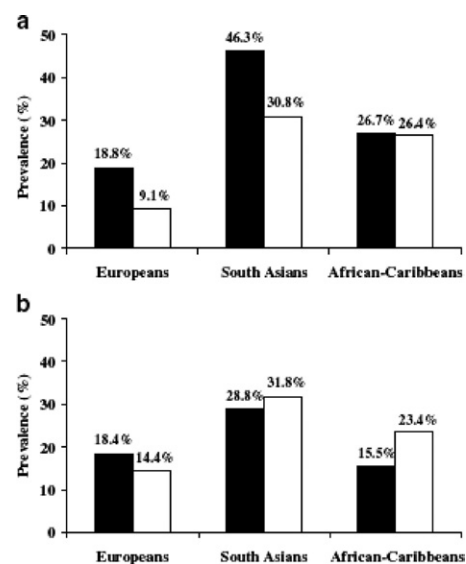


Fig. 2. Age-standardized prevalence of the metabolic syndrome as defined by the World Health Organization criteria (a) or the National Cholesterol Education Program, Adult Treatment Panel III criteria (b) in men (black bars) and women (white bars) divided according to ethnicity. Reproduced from Tillin et al. [30] (with permission).

than their siblings living in Punjab, India [32]. Similarly, in a comparison of Gujaratis (originating from the state of Gujarat, India) in Britain with non-migrant Gujaratis in India, the former had higher mean values of BMI, blood pressure, lipids, non-esterified fatty acids, and C-reactive protein and a higher dietary intake of calorie and fat; however, the prevalence of T2DM was similarly high in both samples [33].

Importantly, the prevalence rate of T2DM in migrant Asian Indians has been found to be consistently higher than in other ethnic groups [34,35]. For example, Asian Indian communities in South Africa showed 11–13% prevalence of T2DM, higher than the native Cape colored and Bantu ethnic groups [10]. Interestingly, these investigators also showed that highly inbred and culture-preserving Tamil Hindus had the highest prevalence of T2DM, 37% in those older than 25 y of age [10]. Important observations from the Diabetes Epidemiology Collaborative analysis of Diagnostic Criteria in Europe (DECODA) study (including 11 cohort studies and comprising 24 335 subjects) on the age- and sex-specific prevalence of T2DM and impaired glucose regulation in four Asian countries, i.e., India, China, Singapore, and Japan, showed that Asian Indians have the highest prevalence of T2DM [36]. Interestingly, a retrospective analysis showed that, during 1993–2001, the greatest increase in the prevalence of T2DM (68%) in the elderly in the United States was seen in Asian populations [37]. Furthermore, a recent multisite study showed stepwise increases in prevalence of T2DM from rural India (8.4%), urban India (13.6%), and in Asian Indians settled in the United States (17.4%) (A. Misra, unpublished data).

Various investigators have stressed adverse attributes of body composition in the pathophysiology of insulin resistance, the metabolic syndrome, and T2DM in Asian Indians and South Asians. Specific features include excesses in body fat, truncal subcutaneous fat, and abdominal adiposity and low muscle mass [29,38–41]. Migration leads to a further increase in adiposity in these regional adipose tissue depots. Importantly, ethnic differences in insulin sensitivity between South Asian and White Caucasian adolescents (14–17 y of age) could be explained by ethnic differences in body fat. Ehtisham et al. [42], in their community-based cross-sectional cohort study, conclusively showed that South Asian adolescents were more insulin resistant, with more body fat than White European Caucasian adolescents, and this may contribute to their increased risk of developing T2DM [42] (Fig. 3).

Over the previous two decades, India and other South Asian countries have been undergoing rapid demographic nutritional transition and urbanization. The changing dietary profile currently includes non-traditional “Westernized” foods, particularly consumed by younger and economically well-off people [43]. Obesity and the metabolic syndrome are also becoming increasingly noticeable in urban areas, particularly in children [28,39,44–48]. The prevalence of T2DM has approximately doubled over the previous two

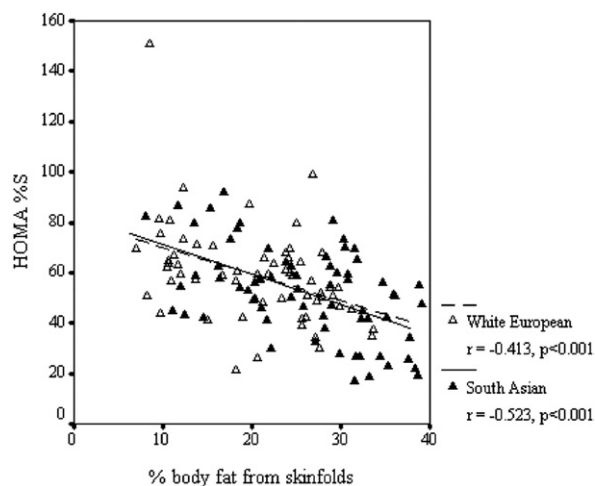


Fig. 3. Scatter plot of HOMA analysis of insulin sensitivity against percent body fat by ethnicity, with linear regression lines for each ethnic group. Reproduced from Ehtisham et al. [42] (with permission). HOMA, Homeostatic Model of Assessment.

decades in the rural and urban populations in India [40,49,50]. Apart from adults, T2DM has been known to increase in children and adolescents [51,52], which reflects substantial and continuous changes in nutrition-related behaviors and lifestyle in the younger population [43]. The environmental influences appear to play an important role in the prevalence of chronic disease in migrant Asian Indian populations besides ethnic susceptibility.

Other migrant populations

Chinese

The Chinese migration to the United States and Canada started in the 1850s, first to California (*gum shan*, gold mountain) in search of gold and later as railway workers. The migrant Chinese living in North America reportedly had higher rates of obesity than the sedentees in a study involving 2488 subjects [53]. Similarly, in a random clustered sample study of 5080 adults 25–74 y old, Chinese migrants to Mauritius were shown to have a higher age-standardized prevalence of impaired glucose tolerance and T2DM based on WHO criteria. The age- and sex-standardized prevalences (percentages) of impaired glucose tolerance and T2DM in Chinese immigrants were 16.6 and 11.9, respectively [54]. The prevalence of CVD was six times greater (24%) among Chinese migrants to Mauritius than among Chinese in Beijing, China (4%), with higher average cholesterol levels among Mauritius-based Chinese (5.4 mmol/L) than in those living in Beijing (4.3 mmol/L) [55]. Similar to other Asian populations, obesity and the metabolic syndrome are currently increasing in urban China (Table 1) [56]. A cross-sectional survey of a nationally representative sample of 15 540 Chinese adults 35–74 y of age substantiated these data [56]. The increased prevalence of these morbid factors also is currently being seen in the urban Chinese population,

Table 1
Crude and age-standardized prevalences of at least one component of the metabolic syndrome* in China. Adapted from Gu et al. [56] (with permission).

	Number of metabolic abnormalities				
	1	2	3	4	5
Crude	35.2 (0.5)	19.6 (0.4)	9.6 (0.3)	3.5 (0.2)	0.7 (0.1)
Men	37.8 (0.8) [†]	18.0 (0.6) [†]	7.7 (0.4) [†]	1.9 (0.2) [†]	0.2 (0.1) [†]
Women	32.6 (0.7)	21.1 (0.6)	11.6 (0.5)	5.1 (0.3)	1.2 (0.2)
Urban	32.4 (0.9) [‡]	23.1 (0.8)	12.3 (0.6) [‡]	3.3 (0.3) [‡]	0.5 (0.1) [‡]
Rural	39.1 (0.9)	17.0 (0.7)	6.6 (0.5)	1.6 (0.3)	0.1 (0.1)
Women					
Urban	28.1 (0.8) [‡]	19.0 (0.7) [‡]	13.1 (0.6) [‡]	6.4 (0.4) [‡]	1.8 (0.3) [‡]
Rural	33.8 (0.9)	21.7 (0.8)	11.4 (0.6)	4.9 (0.4)	1.0 (0.2)

Data are percentage (SE).

* Based on ATPIII criteria.

[†] $p < 0.05$ for men versus women.

[‡] $p < 0.05$ urban versus rural.

apart from migrant Chinese in various parts of the world, and is probably due to rapid nutritional and lifestyle transitions [56]. He et al. [57] studied a nationally representative cohort of 169 871 men and women ≥ 40 y of age and showed that CVD is currently the leading cause of death in China and that T2DM was among the top five causes of death in urban Chinese residents. Overall, a clear shift in the prevalence of chronic diseases has seemed to occur in the Chinese population within the intercountry and intracountry migrants.

Japanese

A high prevalence of T2DM in Japanese Nisei men and women was reported in the United States [58] and Brazil [59,60] in cross-sectional, population-based studies. The results seem to be significantly influenced by an accumulation of excess intra-abdominal fat [61]. An important lifestyle factor considered in the development of T2DM in Japanese Americans appears to be dietary saturated (animal origin) fat and physical inactivity, and the development of insulin resistance also seems to be probably related to excess abdominal adiposity [58].

Similar to the high prevalence of T2DM seen in Japanese Americans, a cross-sectional study comparing first (Issei) and second (Nisei) generations of Japanese Brazilians 40–79 y of age showed that the prevalence of T2DM in migrant Japanese Brazilians was higher than the rates reported for Japan at comparable age groups [59]. A higher incidence of T2DM, an increased insulin resistance, and a higher total cholesterol and serum triacylglycerol levels were seen in Japanese Americans than in native Japanese [62]. The prevalence of diabetes was seen to be two to three times higher among Japanese migrants living in Hawaii, who also showed higher fasting and post-postprandial insulin concentrations than the Japanese living in Hiroshima [63]. The proportion of deaths attributed to ischemic heart disease was found to be higher in diabetic and non-diabetic

Japanese Hawaiians than in diabetic sedentees in Japan. Even the offspring of Japanese migrants living in Hawaii showed a higher proportion of deaths than did sedentees in Japan [64]. Although the proportional death rate from diabetes in the 1950s was about half in Japanese Hawaiians compared with Caucasian Hawaiians, it increased sharply to 1.6 times compared with Caucasian in the 1970s [64].

Greeks, Turks, and other European populations

An interesting “morbidity-mortality paradox” has been reported in Greek migrants to Australia [65]. The data in the 1980s suggested that first-generation Greeks in Australia were the second longest-lived population among all populations including the sedentees in Greece [66]. Intriguingly, such a low mortality rate was seen despite high prevalences of obesity, hyperlipidemia, hypertension, and physical inactivity [67–69], and this paradox has been explained on the basis of adherence to their traditional “healthy” Mediterranean diets [65]. These Mediterranean diets generally contain larger amounts of antioxidant carotenoids (especially lutein), which may decrease cardiovascular risk, although this issue needs further investigation. However, interestingly, at the same time, Australia-born Greeks demonstrated considerably higher standardized mortality ratios suggestive of strong dietary and other lifestyle influences [65,70]. Despite belonging to low socioeconomic strata and indulging in heavy smoking, migrants from southern Europe and North Africa to France have lower mortality and better health in comparison with local French subjects, probably explained by adherence to a Mediterranean diet [71].

Since the 1960s, numerous healthy Turkish people have migrated to Germany, and those who had lived in Germany for at least 10 y showed higher total cholesterol concentrations than sedentees in Turkey. The Geissen study involving 480 male and female Turkish immigrants in Germany showed that dyslipidemia was the highest among all cardiovascular risk factors in both genders [72]. Total cholesterol levels were comparable to those of other Western countries and were remarkably higher than those reported for the population in Turkey. However, high-density lipoprotein cholesterol concentrations were low and found to be similar to those in the sedentees in Turkey [72]. Based on these factors, it was concluded that Turkish immigrants in Germany have developed a lipid profile similar to that of Western populations. Based on these observations, it has been speculated that the incidence of CVD is likely to increase in Turkish migrants in future.

Samoans and Tokelauans

American Samoans, inhabitants of an unincorporated territory of the United States located in the South Pacific, have undergone demographic and nutritional transitions. High prevalence rates of obesity, hypertension, and dyslipidemia were observed in urbanized and migrant Samoan populations in Hawaii and western United States [73–75]. It is emphasized that parental ties were linked to substantially

lower blood pressures, and a practice of Samoan traditions favored lower blood pressure among migrant men, whereas Westernized dietary practices favored higher blood pressures [73]. A study of 2657 Samoan adults showed an increase in the frequency of obesity in Samoan men with increasing modernity of residence or occupation, with women also demonstrating a pattern of higher adiposity in more modern jobs [74]. Moreover, Samoans living in the San Francisco Bay area, interestingly, had an average height between the 25th and 50th percentiles, whereas approximately 50% of them exceeded the 95th percentile for body weight in the U.S. population [75]. Such obesity was accompanied by elevated blood pressure and, in females, by elevated fasting plasma glucose [75]. Prevalences of T2DM in American Samoans were 25% and 15% versus 3.3% and 5.4% in sedentary Samoan men and women, respectively. Moreover, 4-y incidences of T2DM (measured from 1990–1991 to 1994–1995) were 12.4% and 8.5% in American Samoans compared with 2% and 2.6% in men and women, respectively, from Samoa [76]. The pattern emphasized that the modernization of health trends and lifestyle habits in the migrant community led to an increase in morbidity status. Similar observations have been reported for Tokelauans [77].

Other ethnic groups and populations

Italian and Greek migrants to Australia have a three times higher cumulative incidence of T2DM (2.4% and 2.6%, respectively) than do Australia-born (0.7%) individuals [69]. Italian migrants to Belgium also showed higher insulin requirements (in diabetics) and higher glycosylated hemoglobin concentration than Belgian subjects [78]. The prevalence of T2DM was found to be high in Arab Americans in a randomized study of 20- to 75-y-old adults (15.5% in women and 20.1% in men) but was similar to the prevalence rates reported in urban Arab populations living in native countries [79].

Intracountry migration and resettlements

The migration within a country may occur from a rural to an urban habitat or from the traditional habitat to government-selected reservations or missions. The following descriptions of such migrant populations provide a reflection of how nutritional and lifestyle transitions have affected the health status of people living within the same country.

The Aboriginal and Torres Strait Islanders, traditionally being hunters and gatherers, were resettled in government-made reservations. However, they remain Australia's most disadvantaged population, with poor health and living conditions. Remarkably, nearly 75% of women and 50% of men and an increasing number of children of such "urbanized" aboriginals are obese and show high prevalences of the metabolic syndrome and T2DM, as demonstrated by age-stratified data analysis and cross-sectional studies [80,81].

In India and other South Asian countries, a rural-to-urban area migration is occurring rapidly. In their previous rural habitat, these people worked in the fields and plantations, consumed frugal diets, and showed a low prevalence of obesity, T2DM, or CVD [82]. Such populations, upon migration to urban resettlement colonies, became inactive, obese, insulin resistant, and dyslipidemic and acquired several other cardiovascular risk factors [40,45,83–85]. Considerable adverse changes in their dietary pattern were also observed despite continued poverty and low resources [40].

An adverse coronary risk profile was reported among rural-to-urban migrant populations living in urban slums undergoing a stressful socioeconomic transition [85]. A Brazilian survey of 535 poor families comprising 2411 individuals living in city shantytowns showed a high prevalence of malnutrition commonly associated with obesity [86]. The coexistence of disease associated with urbanization along with malnutrition in the economically disadvantaged population represents the "twin burden" of diseases and has been also reported from many other developing countries [86,87].

The migration of Native American Indians through the "ice-free" corridor or through glaciers in North America exposed them to cold harsh climates, intense physical exertion, and diets low in fat and carbohydrates; after resettlement, they were exposed to high energy, sugar, and fatty diets and an urbanized lifestyle. These influences led to high insulin resistance, obesity, and T2DM [88,89].

International adoptees

The international adoptees comprise an interesting sample of migrants who are born in developing countries but are settled in developed countries from an early age, mostly during childhood. In a population-based study, Johansson-Kark et al. [90] investigated the prevalence of overweight in 275 026 young international adoptees, born in 1973–1977 and living in Sweden at 17 y of age, and compared the data with those of non-adopted subjects. Interestingly, heterogeneity in overweight according to country of origin was seen, with the highest being in those from South America [90]. The data did not sufficiently point toward the environmental influences on the risk factor, and differences in prevalence of overweight between various groups of adoptees and between adopted and non-adopted subjects were probably thought to be due to diversity in genetic susceptibility to overweight. More epidemiologic and metabolic studies are required to demonstrate the influence of a "foreign" lifestyle in young adoptees, particularly in those originally belonging to developing countries.

Pitfalls of the migrant studies

Studies relating to migrant populations are difficult to perform because of numerous and continuously changing

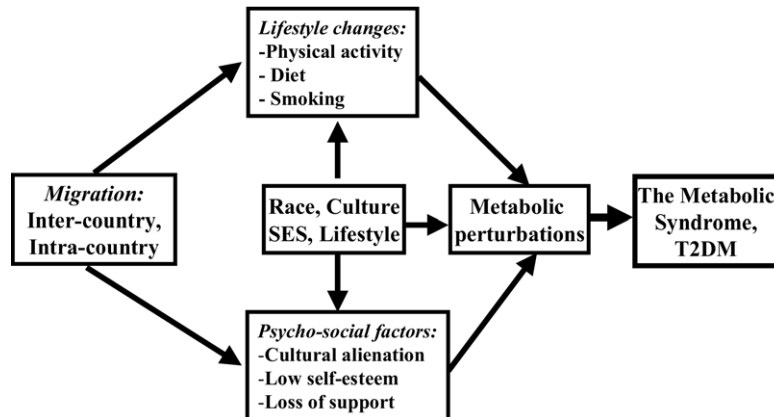


Fig. 4. Interplay of multiple factors after migration. The end result may be early-onset metabolic syndrome and T2DM. SES, socioeconomic stratum; T2DM, type 2 diabetes mellitus.

variables. First, the migrant population may not typically represent the general characteristics of the sedentary population, and they may be healthier or less healthy than sedentaries due to various factors outlined previously. Second, the increasing global reach of the Western food industry, spurred on by the barrage of advertisements in audiovisual media, is radically influencing the dietary patterns of children and young individuals in many developing countries. Hence, the nutritional habits, body composition, and metabolic attributes commonly seen in first-generation migrants may have been acquired by similarly aged sedentaries in developing countries. Third, some studies of migrant populations have required long periods to complete, and during this time the prevalence of obesity and T2DM in sedentaries may have increased, making the initial comparisons erroneous [91]. Moreover, the apparent increase in metabolic risk may take several years to occur after being exposed to changed lifestyle factors after migration. In addition, the morbidity and mortality data depend on the awareness of the population to the disease, primary prevention strategies, and the level of medical care available in the respective countries.

Determinants of obesity and T2DM in migrant populations

Although the following section lists individual factors, usually there is a complex interplay of several factors, which may determine the adverse metabolic changes and the prevalence of T2DM on migration (Fig. 4).

Nutritional transition

A major factor responsible for many of the above-mentioned disorders is the nutritional transition of migrants. Recent studies have shown that most developed countries have a converging dietary pattern: high calories, saturated

fat, simple sugars, and low intakes of dietary fiber, fruits, and vegetables. This “pattern four” of nutritional transition is commonly associated with obesity and T2DM [92]. And in a population probably genetically programmed (but modifiable) in utero to abdominal obesity, diabetes (T2DM and gestational), and CVD, these conditions may be rapidly acquired on a migration-induced nutritional transition. Several studies on migrants have identified nutritional transition confirming to the “pattern four”; however, the extent varies with each ethnic group [53,93,94].

The migrant Japanese Americans show a conversion to diets higher in animal fat and simple carbohydrates and take less complex carbohydrates [95]. Consumption of animal fat and simple carbohydrates (sucrose and fructose) were at least twice as high in Japanese living in Hawaii as in sedentary Japanese in Hiroshima. Conversely, Japanese in Hiroshima consumed about twice the amount of complex carbohydrate as the Hawaiian Japanese [64]. Similarly, two cross-sectional surveys in a randomly chosen population found that the energy intakes from dietary fats (percentage) among Japan-born subjects were 27.2 for men and 26.2 for women, whereas the respective figures for Brazil-born Japanese were 30.1 and 29.5 [96]. The Chinese living in China consumed more carbohydrate but less fat and were leaner than Chinese settled in North America [53]. The transition in the habitual diet with increasing (saturated) fat and decreasing fruit and vegetable intakes was thought to be the reason for increasing chronic diseases in Caribbean migrants to Britain [97].

Interestingly, a common West African ancestry but different body composition has been largely accounted for by the vastly different food intakes and physical activity patterns in the country of migration. For example, 20–25% energy of rural and urban diets in West Africa is supplied by fat from palm, peanut, and corn oils [98], whereas it was 27–39% in those living in Jamaica [12] and 32–35% in African Caribbeans living in Manchester, United Kingdom [99]. Changes have been identified in the specific dietary

nutrients in the selected populations. For example, fish intake was reduced as Japanese moved from Japan to Brazil, which correlated with cardiovascular risk [60]. The increase in salt intake correlating to high blood pressure was reported in Tokelauan migrants to New Zealand [91].

Interestingly, Anderson et al. [100] reported that the nutritional transition in Italian migrants to Scotland was seen only in the subsequent generation, whereas South Asian migrants to Scotland developed an adverse dietary profile in the first generation. Mostly, however, the dietary change in migrant South Asians has been gradual, depending on the length of residence in the adapted country, age group, and whether the subject was a first- or a subsequent-generation migrant. The “dietary acculturation” included decreased vegetarian status and use of South Asian ethnic foods, inclusion of Westernized food items, and alteration in the meal pattern. The changes in the dietary profile conforming to urban diets and increased salt intake have also been seen in intracountry rural-to-urban migrants in India and other countries [45,101]. The dietary “acculturation,” however, may be highly variable within the same ethnic group [102]. The age and economic status of the migrants also influence their dietary choices. Cross-sectional studies of randomly sampled younger migrants found that they changed their food habits more readily to more energy-dense foods [103,104]. Other perceived barriers to maintaining customary eating habits in migrants were the high cost of fruits and vegetables, greater availability of convenience foods, and the limited variety of foods of choice available in the migrant country [103]. Interestingly, increasing migration may result in discernible changes in the food habits of the non-migrant people in the adapted country, making migration-associated food changes “bidirectional” [105]. This bidirectional food acculturation, as seen in the Australian eating patterns, became “Asianized” [105]. However, the impact of such Asianized diets as prevalent in some of the developed countries (e.g., United Kingdom) on metabolic and cardiovascular risk factors in White Caucasians has not been investigated.

There is a paucity of data on the dietary intervention studies in migrant populations. In Australian Aboriginals, Rowley et al. [106] conducted sequential, periodic, cross-sectional risk factor surveys to evaluate the effectiveness of a community-directed intervention program to reduce cardiovascular risk through dietary modifications. The study showed that a dietary intervention program consisting of decreased saturated fat and sugar intake and increased fruit and vegetable intake significantly decreased hypercholesterolemia and other cardiovascular risk factors [106]. Clearly, such studies are needed for other migrant populations.

Physical inactivity

Usually there is a marked decrease in the physical activity of migrants. For example, a more sedentary lifestyle was

typically seen in Australian Aboriginals and Tokelauans after migration [107]. Similar observations were also seen in Myanmar youth migrating to Thailand [108] and Chinese migrating to North America [53]. However, such a change may be less striking in those who were already urbanized and sedentary before migration, as may be recently seen in technologically skilled migrants. Intracountry migrants often take sedentary jobs that are markedly different from their previous labor-intensive work [45]. These adverse lifestyle habits could be improved if interventions for healthy lifestyle could be effectively delivered and sustained. The sustainable improvements observed for dietary intake and level of physical activity were justified convincingly in a study of a remote Australian Aboriginal community [109]. The study evaluated the health outcomes of a cohort of high-risk individuals followed over 4 y [109]. These investigators reported that involvement of high-risk subjects in preventive diet and/or exercise strategies was associated with protection from increases in blood glucose and serum triacylglycerols, with significant reductions in fasting insulin concentrations [109].

Gene-environment interactions

Most chronic diseases (including obesity, T2DM, and CVD) result from the interaction between genetic susceptibility and environmental factors, including diet, smoking, and exercise [110]. Evidence shows that genes are involved in determining enzymes, receptors, cofactors, and structural components involved in the metabolism of lipids, lipoproteins, and proteins involved in inflammation and coagulation, and genetic risk modulates relations between dietary factors and adiposity [111]. The migrant data are suggestive of the possible interaction between genes and environment in the development of T2DM [112]. Rapid transitions in diet and lifestyle environmental factors seen in migrant populations and during transition from poverty to affluence may influence heritability of the variant phenotypes that are dependent on the nutrient environment for their expression. However, with the majority of these genes being polymorphic, some genes may respond to nutritional modulation, whereas others may not indicate any response [113]. The discernibility of gene-environmental interaction and the role of either component in a particular setting are often difficult to judge and interpret. In contrast to environmental “modernization” or “Westernization” with acculturation, a “post-modernization” process has been also seen. The prevalence of T2DM was seen to decline in Mexican Americans in San Antonio, with acculturation to the healthier attitudes and behaviors of “postmodernized” American society [114]. A similar trend also seen in the upper socioeconomic stratum in developing countries probably indicates the predominant influence of acquired lifestyle factors often overriding genetic influences. The subtle effects of gene-environment interactions are undoubtedly important and merit a detailed discussion, which is beyond the scope of the present review.

Stress

Many scientists consider migration a “risk factor,” because people undergo significant stress due to a new environment, social, economic, and language disparities, and the job challenges faced during this period. The migrant populations often lack social support and often are socially isolated. These factors may play a significant part in stress-induced hormonal and metabolic derangements. Interestingly, in this context, migrant Tokelauans in New Zealand had higher 24-h urinary catecholamine levels compared with natives living in Tokelau, consistent with the increased number of stressors [115]. Males may undergo more stress in the migrant populations such as Polynesians who follow a male-dominated family structure, which may contribute to hypertension [77]. Importantly, psychological stress and low self-esteem in migrants are also responsible for an increased frequency of smoking [101], alcohol consumption [116], unhealthy dietary practices, and physical inactivity, which may lead to metabolic disturbances.

Other factors

Epidemiologists have described ethnic susceptibility of some populations to develop a specific disease, given the right environmental stimulus. For example, Samoans are highly susceptible to develop obesity [74,75], whereas South Asians are susceptible to develop insulin resistance and T2DM [31]. An excess nutrient supply in those with “early-life adverse events” such as low birth weight and fetal growth retardation has often been cited as a factor responsible for increased rates of obesity, hypertension, and T2DM in migrants from developing countries having a frequent prevalence of malnutrition [117–119].

Summary

Environmental factors seem to play a critical role in conferring an increased risk of obesity, insulin resistance, T2DM, and CVD. Although the remnant genetic effect has been dominant in certain situations, influences of environmental and acquired factors on cardiovascular and metabolic risks often override genetic influences. Most environmental factors are due to technologic and social progress: urbanization, mechanization, changes in nutrition, physical activity, smoking, and alcohol intake. The role of stress and ethnicity in the pathogenesis of adiposity and related metabolic diseases needs to be systematically studied.

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References

- [1] Haenszel W, Kurihara M. Studies of Japanese migrants. I. Mortality from cancer and other diseases among Japanese in the United States. *J Natl Cancer Inst* 1968;40:43–68.
- [2] Marmot MG, Syme SL, Kagan A, Kato H, Cohen JB, Belsky J. Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: prevalence of coronary and hypertensive heart disease and associated risk factors. *Am J Epidemiol* 1975;102:514–25.
- [3] Robertson TL, Kato H, Rhoads GG, Kagan A, Marmot M, Syme SL, et al. Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California. Incidence of myocardial infarction and death from coronary heart disease. *Am J Cardiol* 1977;39:239–43.
- [4] Iacoviello L, Arnout J, Buntinx F, Cappuccio FP, Dagnelie PC, de Lorgeril M, et al. Dietary habit profile in European communities with different risk of myocardial infarction: the impact of migration as a model of gene-environment interaction. The IMMIDIET Study. *Nutr Metab Cardiovasc Dis* 2001;11(suppl):122–6.
- [5] Markides KS, Coreil J. The health of Hispanics in the southwestern United States: an epidemiologic paradox. *Public Health Rep* 1986; 101:253–65.
- [6] Rubia M, Marcos I, Muennig PA. Increased risk of heart disease and stroke among foreign-born females residing in the United States. *Am J Prev Med* 2002;22:30–5.
- [7] Stern MP, Wei M. Do Mexican Americans really have low rates of cardiovascular disease? *Prev Med* 1999;29(6 pt 2):S90–5.
- [8] Holdenson Z, Catanzariti L, Phillips G, Waters AM. A picture of diabetes in overseas-born Australians. Bulletin number 9. AIHW catalog number Aus 38. Canberra: AIHW; 2003.
- [9] Kelaher M, Jessop DJ. Differences in low-birthweight among documented and undocumented foreign-born and US-born Latinas. *Soc Sci Med* 2002;55:2171–5.
- [10] Taylor R, Zimmet P. Migrant studies in diabetes epidemiology. In: Mann JI, Pyorala K, Teuscher A, editors. *Diabetes in epidemiological perspective*. Edinburgh: Churchill Livingstone; 1983, p. 58–77.
- [11] International Diabetes Federation. *Diabetes atlas*. Executive summary. 2nd ed. Available at: <http://www.eatlas.idf.org/webdata/docs/Atlas%202003-Summary.pdf>. Accessed May 12, 2007.
- [12] Luke A, Cooper RS, Prewitt TE, Adeyemo AA, Forrester TE. Nutritional consequences of the African diaspora. *Annu Rev Nutr* 2001;21:47–71.
- [13] Luke A, Durazo-Arvizu R, Rotimi C, Prewitt TE, Forrester T, Wilks R, et al. Relation between body mass index and body fat in black population samples from Nigeria, Jamaica, and the United States. *Am J Epidemiol* 1997;145:620–8.
- [14] Luke A, Guo X, Adeyemo AA, Wilks R, Forrester T, Lowe W Jr., et al. Heritability of obesity-related traits among Nigerians, Jamaicans and US black people. *Int J Obes Relat Metab Disord* 2001;25:1034–41.
- [15] Rotimi CN, Cooper RS, Okosun IS, Olatunbosun ST, Bella AF, Wilks R, et al. Prevalence of diabetes and impaired glucose tolerance in Nigerians, Jamaicans and US blacks. *Ethn Dis* 1999;9:190–200.
- [16] Okosun IS, Rotimi CN, Forrester TE, Fraser H, Osotimehin B, Muna WF, et al. 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* 2000;24:180–6.
- [17] Osei K. Metabolic consequences of the West African diaspora: lessons from the thrifty gene. *J Lab Clin Med* 1999;133:98–111.
- [18] Kaufman JS, Durazo-Arvizu RA, Rotimi CN, McGee DL, Cooper RS, et al. Obesity and hypertension prevalence in populations of African origin. The Investigators of the International Collaborative Study on Hypertension in Blacks. *Epidemiology* 1996;7:398–405.

- [19] Rosenwaike I. Mortality differentials among persons born in Cuba, Mexico, and Puerto Rico residing in the United States, 1979–81. *Am J Public Health* 1987;77:603–6.
- [20] Sorlie PD, Backlund E, Johnson NJ, Rogot E. Mortality by Hispanic status in the United States. *JAMA* 1993;270:2464–8.
- [21] Wei M, Valdez RA, Mitchell BD, Haffner SM, Stern MP, Hazuda HP. Migration status, socioeconomic status, and mortality rates in Mexican Americans and non-Hispanic whites: the San Antonio Heart Study. *Ann Epidemiol* 1996;6:307–13.
- [22] Sundquist J, Winkleby M. Country of birth, acculturation status and abdominal obesity in a national sample of Mexican-American women and men. *Int J Epidemiol* 2000;29:470–7.
- [23] Khan LK, Sobal J, Martorell R. Acculturation, socioeconomic status, and obesity in Mexican Americans, Cuban Americans, and Puerto Ricans. *Int J Obes Relat Metab Disord* 1997;21:91–6.
- [24] Popkin BM, Udry JR. Adolescent obesity increases significantly in second and third generation U.S. immigrants: the National Longitudinal Study of Adolescent Health. *J Nutr* 1998;128:701–6.
- [25] Samet JM, Coultas DB, Howard CA, Skipper BJ, Hanis CL. Diabetes, gallbladder disease, obesity, and hypertension among Hispanics in New Mexico. *Am J Epidemiol* 1988;128:1302–11.
- [26] Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 2002;287:356–9.
- [27] McKeigue PM, Marmot MG, Syndercombe Court YD, Cottier DE, Rahman S, Riemersma RA. Diabetes, hyperinsulinaemia, and coronary risk factors in Bangladeshis in east London. *Br Heart J* 1988;60:390–6.
- [28] Misra A, Vikram NK. Insulin resistance syndrome (metabolic syndrome) and Asian Indians. *Curr Sci* 2002;83:1483–6. Available at: <http://tejas.serc.iisc.ernet.in/~cursci>.
- [29] Banerji MA, Faridi N, Alturi R, Chaiken RL, Lebovitz HE. Body composition, visceral fat, leptin and insulin resistance in Asian Indian men. *J Clin Endocrinol Metab* 1999;84:137–44.
- [30] Tillin T, Forouhi N, Johnston DG, McKeigue PM, Chaturvedi N, Godsland IF. Metabolic syndrome and coronary heart disease in South Asians, African-Caribbeans and white Europeans: a UK population-based cross-sectional study. *Diabetologia* 2005;48:649–56.
- [31] Misra A, Vikram NK. Insulin resistance syndrome (metabolic syndrome) and obesity in Asian Indians: evidence and implications. *Nutrition* 2004;20:482–91.
- [32] Bhatnagar D, Anand IS, Durrington PN, Patel DJ, Wander GS, Mackness MI, et al. Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India. *Lancet* 1995;345(8947):405–9.
- [33] Patel JV, Vyas A, Cruickshank JK, Prabhakaran D, Hughes E, Reddy KS, et al. Impact of migration on coronary heart disease risk factors: comparison of Gujaratis in Britain and their contemporaries in villages of origin in India. *Atherosclerosis* 2006;185:297–306.
- [34] McKeigue PM, Pierpoint T, Ferrie JE, Marmot MG. Relationship of glucose intolerance and hyperinsulinaemia to body fat pattern in south Asians and Europeans. *Diabetologia* 1992;35:785–91.
- [35] Anand SS, Yusuf S, Vuksan V, Devanese S, Teo KK, Montague PA, et al. Differences in risk factors, atherosclerosis and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic Groups (SHARE). *Indian Heart J* 2000;52(suppl):S35–43.
- [36] Qiao Q, Hu G, Tuomilehto J, Borch-Johnsen K, Ramachandran A, Mohan V, et al. Age- and sex-specific prevalence of diabetes and impaired glucose regulation in 11 Asian cohorts. *Diabetes Care* 2003;26:1770–80.
- [37] McBean AM, Li S, Gilbertson DT, Collins AJ. Differences in diabetes prevalence, incidence, and mortality among the elderly of four racial/ethnic groups: whites, blacks, Hispanics, and Asians. *Diabetes Care* 2004;27:2317–24.
- [38] Raji A, Seely EW, Arky RA, Simonson DC. Body fat distribution and insulin resistance in healthy Asian Indians and Caucasians. *J Clin Endocrinol Metab* 2001;86:5366–71.
- [39] Misra A. Body composition and the metabolic syndrome in Asian Indians: a saga of multiple adversities. *Natl Med J India* 2003;16:3–7.
- [40] Misra A, Pandey RM, Devi JR, Sharma R, Vikram NK, Khanna N. High prevalence of diabetes, obesity and dyslipidaemia in urban slum population in northern India. *Int J Obes Relat Metab Disord* 2001;25:1722–9.
- [41] Dudeja V, Misra A, Pandey RM, Devina G, Kumar G, Vikram NK. BMI does not accurately predict overweight in Asian Indians in northern India. *Br J Nutr* 2001;86:105–12.
- [42] Ehtisham S, Crabtree N, Clark P, Shaw N, Barrett T. Ethnic differences in insulin resistance and body composition in United Kingdom adolescents. *J Clin Endocrinol Metab* 2005;90:3963–9.
- [43] Wasir JS, Misra A. The metabolic syndrome in Asian Indians: the impact of nutritional and socio-economic transition in India. *Metab Syndr Relat Disord* 2004;2:14–23.
- [44] Misra A, Vikram NK, Sharma R, Basit A. High prevalence of obesity and associated risk factors in urban children in India and Pakistan highlights immediate need to initiate primary prevention program for diabetes and coronary heart disease in schools. *Diabetes Res Clin Pract* 2006;71:101–2.
- [45] Misra A, Sharma R, Pandey RM, Khanna N. Adverse profile of dietary nutrients, anthropometry and lipids in urban slum dwellers of northern India. *Eur J Clin Nutr* 2001;55:727–34.
- [46] Mohan V, Shanthirani S, Deepa R, Premalatha G, Sastry NG, Saroja R. Intra-urban differences in the prevalence of the metabolic syndrome in southern India—the Chennai Urban Population Study (CUPS No. 4). *Diabet Med* 2001;18:280–7.
- [47] Vikram NK, Misra A, Pandey RM, Luthra K, Wasir JS, Dhingra V. Heterogeneous phenotypes of insulin resistance and its implications for defining metabolic syndrome in Asian Indian adolescents. *Atherosclerosis* 2006;186:193–9.
- [48] Vikram NK, Misra A. Adiponectin levels in postpubertal Asian Indian adolescents: relationships with insulin resistance and C-reactive protein. *Metabolism* 2004;53:1336–41.
- [49] Ramachandran A, Snehalatha C, Vijay V. Temporal changes in prevalence of type 2 diabetes and impaired glucose tolerance in urban southern India. *Diabetes Res Clin Pract* 2002;58:55–60.
- [50] Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Kothari K. Prevalence of diabetes, impaired fasting glucose and insulin resistance syndrome in an urban Indian population. *Diabetes Res Clin Pract* 2003;61:69–76.
- [51] Vikram NK, Tandon N, Misra A, Srivastava MC, Pandey RM, Mithal A. Correlates of type 2 diabetes mellitus in children, adolescents and young adults in north India: a multisite collaborative case-control study. *Diabet Med* 2006;23:293–8.
- [52] Ramachandran A, Snehalatha C, Satyavani K, Sivasankari S, Vijay V. Type 2 diabetes in Asian-Indian urban children. *Diabetes Care* 2003;26:1022–5.
- [53] Lee MM, Wu-Williams A, Whittemore AS, Zheng S, Gallagher R, Teh CZ, et al. Comparison of dietary habits, physical activity and body size among Chinese in North America and China. *Int J Epidemiol* 1994;23:984–90.
- [54] Dowse GK, Gareeboo H, Zimmet PZ, Alberti KG, Tuomilehto J, Fareed D, et al. High prevalence of NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. Mauritius Non-communicable Disease Study Group. *Diabetes* 1990;39:390–6.
- [55] Li N, Tuomilehto J, Dowse G, Alberti KG, Zimmet P, Min Z. Electrocardiographic abnormalities and associated factors in Chinese living in Beijing and in Mauritius. The Mauritius Non-Communicable Disease Study Group. *BMJ* 1992;304(6842):1596–601.
- [56] Gu D, Reynolds K, Wu X, Chen J, Duan X, Reynolds RF, et al. Prevalence of the metabolic syndrome and overweight among adults in China. *Lancet* 2005;365(9468):1398–405.

- [57] He J, Gu D, Wu X, Reynolds K, Duan X, Yao C, et al. Major causes of death among men and women in China. *N Engl J Med* 2005;353:1124–34.
- [58] Fujimoto WY, Bergstrom RW, Boyko EJ, Kinyoun JL, Leonetti DL, Newell-Morris LL, et al. Diabetes and diabetes risk factors in second- and third-generation Japanese Americans in Seattle, Washington. *Diabetes Res Clin Pract* 1994;24(suppl):S43–52.
- [59] Franco LJ. Diabetes in Japanese-Brazilians—influence of the acculturation process. *Diabetes Res Clin Pract* 1996;34(suppl):S51–7.
- [60] Mizushima S, Moriguchi EH, Ishikawa P, Hekman P, Nara Y, Mimura G, et al. Fish intake and cardiovascular risk among middle-aged Japanese in Japan and Brazil. *J Cardiovasc Risk* 1997;4:191–9.
- [61] Boyko EJ, Fujimoto WY, Leonetti DL, Newell-Morris L. Visceral adiposity and risk of type 2 diabetes: a prospective study among Japanese Americans. *Diabetes Care* 2000;23:465–71.
- [62] Nakanishi S, Okubo M, Yoneda M, Jitsuiki K, Yamane K, Kohno N. A comparison between Japanese-Americans living in Hawaii and Los Angeles and native Japanese: the impact of lifestyle westernization on diabetes mellitus. *Biomed Pharmacother* 2004;58:571–7.
- [63] Hara H, Egusa G, Yamakido M, Kawate R. The high prevalence of diabetes mellitus and hyperinsulinemia among the Japanese-Americans living in Hawaii and Los Angeles. *Diabetes Res Clin Pract* 1994;24(suppl):S37–42.
- [64] Kawate R, Yamakido M, Nishimoto Y, Bennett PH, Hamman RF, Knowler WC. Diabetes mellitus and its vascular complications in Japanese migrants on the Island of Hawaii. *Diabetes Care* 1979;2:161–70.
- [65] Kouris-Blazos A. Morbidity mortality paradox of 1st generation Greek Australians. *Asia Pac J Clin Nutr* 2002;11(suppl 3):S569–75.
- [66] Young C. Selection and survival: immigrant mortality in Australia. Studies in adult migrant education. Canberra: Department of Immigration and Ethnic Affairs, Australia Government Printing Services; 1986.
- [67] Bennett SA. Inequalities in risk factors and cardiovascular mortality among Australia's immigrants. *Aust J Public Health* 1993;17:251–61.
- [68] Kouris-Blazos A, et al. Health and nutritional status of elderly Greek migrants to Melbourne, Australia. *Age Ageing* 1996;25:177–89.
- [69] Hodge AM, English DR, O'Dea K, Giles G. Increased diabetes incidence in Greek and Italian migrants to Australia: how much can be explained by known risk factors? *Diabetes Care* 2004;27:2330–4.
- [70] Young C. Mortality, the ultimate indicator of survival: the differential experience between birthplace groups. In: Donovan J, d'Espaignet EM, Merton C, van Ommergen A, editors. Immigrants in Australia: a health profile. Canberra: Australian Government Publishing Service; 1992, p. 34–70.
- [71] Darmon N, Khlal M. An overview of the health status of migrants in France, in relation to their dietary practices. *Public Health Nutr* 2001;4:163–72.
- [72] Porsch-Oezcuernomez M, Bilgin Y, Wollny M, Gediz A, Arat A, Karatay E, et al. Prevalence of risk factors of coronary heart disease in Turks living in Germany: the Giessen Study. *Atherosclerosis* 1999;144:185–98.
- [73] Hanna JM. Psychosocial factors in blood pressure variation: a comparative study of young Samoans. *Soc Biol* 1996;43:169–90.
- [74] Bindon JR, Baker PT. Modernization, migration and obesity among Samoan adults. *Ann Hum Biol* 1985;12:67–76.
- [75] Pawson IG, Janes C. Massive obesity in a migrant Samoan population. *Am J Public Health* 1981;71:508–13.
- [76] Tsai HJ, Sun G, Weeks DE, Kaushal R, Wolujewicz M, McGarvey ST, et al. Type 2 diabetes and three calpain-10 gene polymorphisms in Samoans: no evidence of association. *Am J Hum Genet* 2001;69:1236–44.
- [77] Salmond CE, Prior IA, Wessen AF. Blood pressure patterns and migration: a 14-year cohort study of adult Tokelauans. *Am J Epidemiol* 1989;130:37–52.
- [78] Selvais PL, Hermans MP. Characterization of type 2 diabetes mellitus in first generation Italian migrants to Belgium. *Acta Clin Belg* 2005;60:362–8.
- [79] Jaber LA, Brown MB, Hammad A, Nowak SN, Zhu Q, Ghafoor A, Herman WH. Epidemiology of diabetes among Arab Americans. *Diabetes Care* 2003;26:308–13.
- [80] Piers LS, et al. Relation of adiposity and body fat distribution to body mass index in Australians of Aboriginal and European ancestry. *Eur J Clin Nutr* 2003;57:956–63.
- [81] Daniel M, Rowley KG, McDermott R, O'Dea K. Diabetes and impaired glucose tolerance in Aboriginal Australians: prevalence and risk. *Diabetes Res Clin Pract* 2002;57:23–33.
- [82] Kutty VR, Soman CR, Joseph A, Pisharody R, Vijayakumar K. Type 2 diabetes in southern Kerala: variation in prevalence among geographic divisions within a region. *Natl Med J India* 2000;13:287–92.
- [83] Misra A, Chaudhary D, Vikram NK, Mittal V, Devi JR, Pandey RM. 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* 2002;56:73–5.
- [84] Sethi A, Misra A, Pandey RM, Luthra K, Devi JR, Sharma R, et al. Soluble inter-cellular adhesion molecule-1 in urban Asian north Indians: relationships with anthropometric and metabolic covariates. *Dis Markers* 2002;18:111–20.
- [85] Misra A, Vikram NK, Pandey RM, Dwivedi M, Ahmad FU, Luthra K, et al. Hyperhomocysteinemia, and low intakes of folic acid and vitamin B12 in urban North India. *Eur J Nutr* 2002;41:68–77.
- [86] Sawaya AL, Dallal G, Solymos G, de Sousa MH, Ventura ML, Roberts SB, Sigulem DM. Obesity and malnutrition in a Shantytown population in the city of Sao Paulo, Brazil. *Obes Res* 1995;3(suppl 2):107s–15.
- [87] Misra A. Overnutrition and nutritional deficiency contribute to metabolic syndrome and atherosclerosis in Asian Indians. *Nutrition* 2002;18:702–3.
- [88] Wendorf M. Diabetes, the ice free corridor, and the Paleoindian settlement of North America. *Am J Phys Anthropol* 1989;79:503–20.
- [89] Bindon JR, Baker PT. Bergmann's rule and the thrifty genotype. *Am J Phys Anthropol* 1997;104:201–10.
- [90] Johansson-Kark M, Rasmussen F, Hjert A. Overweight among international adoptees in Sweden: a population-based study. *Acta Paediatr* 2002;91:827–32.
- [91] Salmond CE, Joseph JG, Prior IA, Stanley DG, Wessen AF. Longitudinal analysis of the relationship between blood pressure and migration: the Tokelau Island Migrant Study. *Am J Epidemiol* 1985;122:291–301.
- [92] Lieberman LS. Dietary, evolutionary, and modernizing influences on the prevalence of type 2 diabetes. *Annu Rev Nutr* 2003;23:345–77.
- [93] Trostler N. Health risks of immigration: the Yemenite and Ethiopian cases in Israel. *Biomed Pharmacother* 1997;51:352–9.
- [94] Shannon C. Acculturation: Aboriginal and Torres Strait Islander nutrition. *Asia Pac J Clin Nutr* 2002;11(suppl 3):S576–8.
- [95] Hara H, Egusa G, Yamakido M. Incidence of non-insulin-dependent diabetes mellitus and its risk factors in Japanese-Americans living in Hawaii and Los Angeles. *Diabet Med* 1996;13(suppl 6):S133–42.
- [96] Cardoso MA, Hamada GS, de Souza JM, Tsugane S, Tokudome S. Dietary patterns in Japanese migrants to southeastern Brazil and their descendants. *J Epidemiol* 1997;7:198–204.
- [97] Mennen LI, Jackson M, Sharma S, Mbanya JC, Cade J, Walker S, et al. Habitual diet in four populations of African origin: a descriptive paper on nutrient intakes in rural and urban Cameroon, Jamaica and Caribbean migrants in Britain. *Public Health Nutr* 2001;4:765–72.
- [98] Kigutha HN. Assessment of dietary intake in rural communities in Africa: experiences in Kenya. *Am J Clin Nutr* 1997;65(suppl):1168S–72.
- [99] Sharma S, Cade J, Griffiths S, Cruickshank K. Nutrient intakes among UK African-Caribbeans: changing risk of coronary heart disease. *Lancet* 1998;352(9122):114–5.

- [100] Anderson AS, Bush H, Lean M, Bradby H, Williams R, Lea E. Evolution of atherogenic diets in South Asian and Italian women after migration to a higher risk region. *J Hum Nutr Diet* 2005;18:33–43.
- [101] Steyn K, Kazenellenbogen JM, Lombard CJ, Bourne LT. Urbanization and the risk for chronic diseases of lifestyle in the black population of the Cape Peninsula, South Africa. *J Cardiovasc Risk* 1997;4:135–42.
- [102] Wenkam NS, Wolff RJ. A half century of changing food habits among Japanese in Hawaii. *J Am Diet Assoc* 1970;57:29–32.
- [103] Papadaki A, Scott JA. The impact on eating habits of temporary translocation from a Mediterranean to a Northern European environment. *Eur J Clin Nutr* 2002;56:455–61.
- [104] Sharma S, Cade J, Riste L, Cruickshank K. Nutrient intake trends among African-Caribbeans in Britain: a migrant population and its second generation. *Public Health Nutr* 1999;2:469–76.
- [105] Wahlqvist ML. Asian migration to Australia: food and health consequences. *Asia Pac J Clin Nutr* 2002;11(suppl 3):S562–8.
- [106] Rowley KG, Su Q, Cincotta M, Skinner M, Skinner K, Pindan B, et al. Improvements in circulating cholesterol, antioxidants, and homocysteine after dietary intervention in an Australian Aboriginal community. *Am J Clin Nutr* 2001;74:442–8.
- [107] O’Dea K. Preventable chronic diseases among indigenous Australians: the need for a comprehensive national approach. *Heart Lung Circ* 2005;14:167–71.
- [108] Howteerakul N, Suwannapong N, Than M. Cigarette, alcohol use and physical activity among Myanmar youth workers, Samut Sakhon Province, Thailand. *Southeast Asian J Trop Med Public Health* 2005;36:790–6.
- [109] Rowley KG, Daniel M, Skinner K, Skinner M, White GA, O’Dea K. Effectiveness of a community-directed ‘healthy lifestyle’ program in a remote Australian aboriginal community. *Aust N Z J Public Health* 2000;24:136–44.
- [110] Vincent S, Planells R, Defoort C, Bernard MC, Gerber M, Prudhomme J, et al. Genetic polymorphisms and lipoprotein responses to diets. *Proc Nutr Soc* 2002;61:427–34.
- [111] Greenfield JR, Samaras K, Jenkins AB, Kelly PJ, Spector TD, Campbell LV. Moderate alcohol consumption, dietary fat composition, and abdominal obesity in women: evidence for gene-environment interaction. *J Clin Endocrinol Metab* 2003;88:5381–6.
- [112] Nemoto M, Sasaki T, Deeb SS, Fujimoto WY, Tajima N. Differential effect of PPARgamma2 variants in the development of type 2 diabetes between native Japanese and Japanese Americans. *Diabetes Res Clin Pract* 2002;57:131–7.
- [113] Singh RB, Niaz MA. Genetic variation and nutrition in relation to coronary artery disease. *J Assoc Phys India* 1999;47:1185–90.
- [114] Stern MP, Knapp JA, Hazuda HP, Haffner SM, Patterson JK, Mitchell BD. Genetic and environmental determinants of type II diabetes in Mexican Americans. Is there a “descending limb” to the modernization/diabetes relationship? *Diabetes Care* 1991;14:649–54.
- [115] Jenner DA, Harrison GA, Prior IA. Catecholamine excretion in Tokelauans living in three different environments. *Hum Biol* 1987;59:165–72.
- [116] Ostbye T, Welby TJ, Prior IA, Salmond CE, Stokes YM. Type 2 (non-insulin-dependent) diabetes mellitus, migration and westernisation: the Tokelau Island Migrant Study. *Diabetologia* 1989;32:585–90.
- [117] Fall CH, Barker DJ. The fetal origins of coronary heart disease and non-insulin dependent diabetes in India. *Indian Pediatr* 1997;34:5–8.
- [118] Stein CE, Fall CH, Kumaran K, Osmond C, Cox V, Barker DJ. Fetal growth and coronary heart disease in south India. *Lancet* 1996;348(9037):1269–73.
- [119] Olatunbosun ST, Bella AF. Relationship between height, glucose intolerance, and hypertension in an urban African black adult population: a case for the “thrifty phenotype” hypothesis? *J Natl Med Assoc* 2000;92:265–8.