

# NUTRITIONAL CONSEQUENCES OF THE AFRICAN DIASPORA

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■ **Abstract** Along with their foods and dietary customs, Africans were carried into diaspora throughout the Americas as a result of the European slave trade. Their descendants represent populations at varying stages of the nutrition transition. West Africans are in the early stage, where undernutrition and nutrient deficiencies are prevalent. Many Caribbean populations represent the middle stages, with undernutrition and obesity coexisting. African-Americans and black populations in the United Kingdom suffer from the consequences of caloric excess and diets high in fat and animal products. Obesity, non-insulin-dependent diabetes mellitus, hypertension, coronary heart disease, and certain cancers all follow an east-to-west gradient of increasing prevalence. Public health efforts must focus not only on eradicating undernutrition in West Africa and the Caribbean but also on preventing obesity, hypercholesterolemia, and their consequences. Fortunately, a coherent and well-supported set of recommendations exists to promote better nutrition. Implementation of it founders primarily as a result of the influence of commercial and political interests.

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INTRODUCTION

In the 400 years following Columbus’ landing on the island of Hispanola, 11–13 million people were transported from sub-Saharan Africa to provide the primary productive force in the New World colonies. The Africans carried into diaspora by the European slave trade have grown into large populations in North America, the Caribbean, Brazil and northern South America, and, via more recent secondary migration, the United Kingdom. Smaller populations dot the coast of Central America or have merged with the European majority in Argentina and other Andean countries. The contemporary social and economic environments for Africans vary more than for any other recognized macro-population group, ranging from traditional subsistence agriculture in much of West Africa and parts of the Caribbean, to urban and peri-urban small-market economies in other parts of the Caribbean, to post-industrialized societies in the United States and the United Kingdom. The contrasts in economic conditions between these populations mirror health and nutrition contrasts. In keeping with the historical experience of other populations, moving across this spectrum of social settings from traditional agriculture to industrialization, and as national and individual wealth increases, nutrition-related concerns shift from privation (e.g. childhood stunting) to surfeit (e.g. obesity). The African diaspora therefore offers a unique opportunity to observe the nutrition transitions among contemporary populations sharing a common historical and genetic origin.

HISTORICAL BACKGROUND TO THE  
AFRICAN DIASPORA

The ownership and exploitation of one human being by another has existed as a societal institution throughout history. Slaves have occupied a surprisingly wide range of social roles, although in recent history they have most often been consigned to manual work under brutal conditions, usually with a minimum of food, clothing, and shelter. Enslaved persons were considered chattel and as such were denied freedom of activity and movement, had no rights in courts of law, and could be sold or given away. Until the time of the Atlantic Slave Trade, which had its beginning in the late fifteenth century, most slaves were war captives or debtors (men who sold themselves and/or their wives and children to liquidate debt) (9). Gradually through the Middle Ages, agricultural slavery in Europe evolved into tenant farm and manorial systems. Tenant farmers were bound to the landowners by debt, and serfs pledged their labor and loyalty in return for protection and the

use of a piece of land for subsistence. Though infrequently realized, it was legally sanctioned for a serf, through grueling labor and frugality, to buy his plot of land from the lord of the manor and become a freeman.

Slavery was not eradicated from European society, however. The practice of using slaves for domestic work continued, and sometimes people were sold into slavery as punishment. Slavery in Spain and Portugal continued through the Middle Ages, and from the time of first contact, the Hispanic colonists, to satisfy their need for human labor, instituted forced labor systems on the indigenous peoples of South America, the Caribbean, and Mexico. These systems were not sustainable, however, because of the brutality and overwork imposed on the Indian laborers and the susceptibility of the Indians to such diseases as smallpox, diphtheria, and tuberculosis brought by the colonists. Also, being on their home territory, the Indians who survived often escaped or banded together in revolt (9, 107, 118).

Soon, the Hispanic colonists turned to the nascent Portuguese slave trade on the West African coast to satisfy their need for labor. The first Africans enslaved by the Portuguese were settled in Europe in the late 1400s. In 1510, 250 Africans living in Spain were shipped to the island of Hispanola by the king of Spain to work in the gold mines (107). The first direct voyage from the islands of Cape Verde and São Tome off the western coast of sub-Saharan Africa to the Caribbean was made in 1533 and carried more than 500 Africans to labor on the sugar plantations. Hispanic colonists found the use of the Africans advantageous because they were skilled agriculturists, miners, and metalworkers, had a facility for languages, and were amenable to conversion to Christianity, and those who survived the march to the African coast and the passage to the Americas appeared to have immunity to many dangerous diseases. At least these are the historically recorded reasons; undoubtedly the already established European view that Africans were subhuman also played a critical role. By 1619, one million Africans were enslaved in the Americas, mostly on sugar plantations.

In this same year, a Dutch ship needing supplies docked at Jamestown, Virginia, and traded Africans for food. Although the change was gradual, indentured servitude in the United States was ultimately replaced by racial slavery. The demand for labor in the Americas was met by a systematized triangular trade in which English ships transported goods to the west coast of Africa. After exchanging goods for people, they continued on to the Americas, where they exchanged the people for agricultural produce, then sailed back to England. This arrangement grew into one of the most efficient international enterprises of its time, creating huge fortunes in England, France, and Portugal. By the same token, the extensive nature of the slave trade led to endemic warfare, radically changing the indigenous societies of the coast of Africa. Between the first transport of African slaves in 1533 and the last in 1870, approximately 11–13 million Africans were captured and sold at the coast, up to 1.5 million died on the Middle Passage to the Americas, and 10%–20% died within the first year in the Americas (9, 29, 107). Between 75% and 90% of Africans taken to the New World were transported from ports on the west coast

of Africa between Senegambia in the north and Angola in the south (21, 29, 107) (Figure 1).

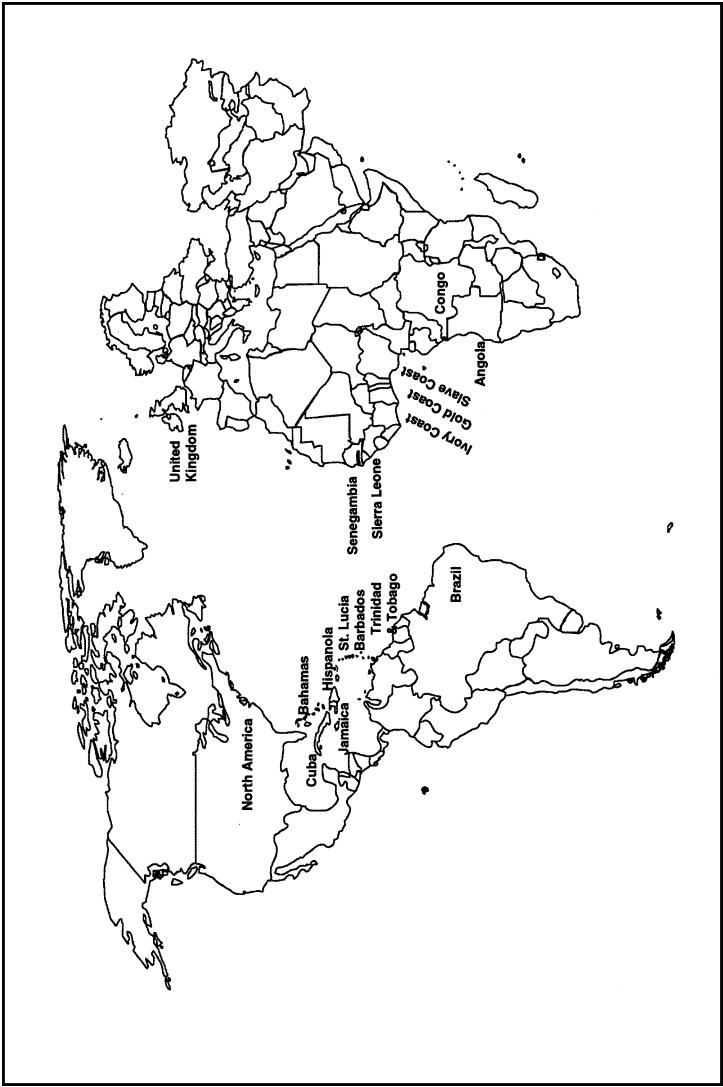
Based on the capture, enslavement, and transport of humans to wholly new cultures, the shipping business, the plantation owners of the South, and the merchants and shipbuilders of the North collectively accumulated much of the capital necessary to fuel the Industrial Revolution. In the broadest sense, therefore, African peoples laid one of the critical foundation pieces of the contemporary capitalist world. Unlike other immigrants, however, this sacrifice has never been rewarded, either in cultural or economic terms.

The former slave-holding colonies and nations in the Americas achieved their large current populations of Africans in separate ways. Brazil, for example, imported the largest number of slaves; Nigeria is the only nation with a larger black population. The United States, destination for only 5% of slaves, experienced rapid natural expansion. The Caribbean islands—Cuba; the former West Indies, including the Dominican Republic, Puerto Rico, Trinidad, and Tobago; the former British West Indies, including Jamaica, Barbados, and the Bahamas; and the former French West Indies, including Haiti—continually imported large numbers of slaves because of high mortality (9, 107). In addition, there are sizeable populations of African origin living in postcolonial Europe, most notably Britain, where people from the West Indies and British West Africa immigrated to meet labor demands after World War II (16, 102).

In addition to the large-scale forced migration of Africans, the triangular trade transferred cultural, agricultural, and dietary practices from Africa to the New World and back again. Crops such as sorghum, pearl millet, African rice, cowpea (black-eyed peas), African yams, okra, watermelon, bottle gourd, and fluted pumpkin were indigenous to Africa (47, 104). Yams were cultivated as early as 17,000–18,000 years ago on the African continent (47).

Although it is uncertain whether deep frying originated in Africa or the middle East, this cooking method traveled to the Americas with Africans. *Akara* from Nigeria, a fritter made by frying cowpea flour in palm oil, is a clear example of the movement of both crops and cooking method. The cowpea, palm oil, and frying method came to the New World with the Africans, probably Yoruba or Hausa slaves. The same fritter is made in Brazil (*acaraje*), Trinidad (*akkra*), and the French islands (*acrats*). Also, the foods in Bahia—the colonial settlement in northeastern Brazil where the Portuguese first brought Africans to work on plantations—are based on tropical oils, palm oil, and coconut. A rice porridge made with coconut milk called *acaca* is made in Colombia and northeastern Brazil. An identical dish with the same name exists in the Republic of Benin and in Nigeria (104).

Crops carried to Africa from the Americas by the Portuguese that became crucial to African agriculture include peanuts, sweet potatoes, and manioc (cassava). Additional foods carried to Africa by Europeans include maize, guavas, lima beans, pumpkins and squash, avocados, tomatoes, pineapples, papayas, and cashew nuts (104).



**Figure 1** Map of the Atlantic Ocean showing the primary regions involved in the slave trade between 1533 and 1870. The regions on the West African coast represent primary deportation sites whereas those in the Americas represent primary countries of importation. The United Kingdom is labeled as the site of secondary migration in the twentieth century, primarily from the English-speaking Caribbean.

## DIET IN AFRICA AND THE DIASPORA

Contemporary dietary data are sparse for sub-Saharan Africa in general and for West Africa specifically. Numerous studies list cassavas, yams, maize, or millet and sorghum as the top contributors of energy (8, 11, 80, 94, 108). The most prominent feature of this diet is the high degree of monotony, in terms of both the raw foodstuffs and the methods of preparation. The majority of available data indicate that 20%–25% of kilocalories in both rural and urban diets is supplied by fat (20, 55, 69, 88; E Choboso, unpublished information), most of which is palm, peanut, and corn oil. Recent studies from Cameroon suggest a higher proportion of kilocalories from fat (i.e. more than 40%); however, this estimate seems implausibly high (72, 101). In this study, cassavas, palm wine, and cocoyams were listed as the primary sources of energy in rural areas whereas in urban areas meat and fish were the top energy contributors.

Food-frequency questionnaire data collected in Spanish Town, Jamaica, indicate a higher percentage of kilocalories derived from fat (101; TE Forrester, unpublished information) than was observed in West Africa, 27%–30% versus 20%–25%. The primary energy sources listed in Jamaica were rice and peas (101). Consistent with trends in industrialized nations, it was estimated that the diet of African-Caribbeans living in Manchester, England, had a fat content between 32% and 35% (100). The chief energy contributors for this population were meat, rice, and peas.

Based on the results of the second National Health and Nutrition Examination Survey (NHANES II), the primary sources of kilocalories for all people in the United States were white breads, cookies and donuts, and meat (10, 70). Self-reported dietary data from NHANES III, however, suggest that diets of all ethnic groups have become more healthful, i.e. over the course of 30 years, the percentage of kilocalories from fat dropped from 38% (NHANES I) to 34% (NHANES III) (93). Still, the diet of African-Americans is characterized as high in fat and salt and low in fruits and vegetables (60). A comparison of diets indicated that those of southern-born African-Americans living in Harlem were less healthful than those of African-Americans born in other regions of the United States or in the Caribbean. African-Americans born in the Caribbean reported the lowest intake of fat (43).

In general, dietary data record an increase in the percentage of energy derived from fat as one moves from West Africa, to the Caribbean, to the United States and United Kingdom, as well as a marked increase in the consumption of refined foods and meat products. At the far end of the spectrum, the African influence has been completely submerged by commercial food patterns; a mixed or syncretic pattern remains in some regions of the Americas (e.g. Jamaica and Bahia, Brazil). For the great majority of West Africans, certainly for the 75% of the population living on subsistence farms, every day brings a meal much like the one the day before, and the ones of centuries before.

## NUTRITION TRANSITION AMONG POPULATIONS OF THE DIASPORA

The usual diet of rural, agriculturist West Africa and that of the industrialized United States represent the early and late stages, respectively, of what has been termed the nutrition transition (90). The changes in nutrient intake and dietary patterns effected by populations as social, cultural, and economic changes decisively influence the public health and thereby the course of the epidemiologic transition (90, 111). The epidemiologic transition can be defined as the changes in patterns of health, morbidity, and mortality that result from demographic shifts and associated economic and societal changes (111). Although these processes are most often used to describe the transition one population undergoes in a linear fashion over time, the African diaspora provides an opportunity to examine and compare separate stages of related populations at a single point in time. When viewed as a whole, the populations of the African diaspora represent differing stages of the transition while the individual countries are undergoing the transition at varying rates themselves.

Although the concept of integrated societal transitions is a useful organizing principle, much heterogeneity exists across societies and subpopulations within those societies. The demographic, epidemiologic, and nutrition transitions interact such that in the earliest stage, the high fertility and high mortality rates of traditional societies influence and are influenced by high prevalences of infectious disease and undernutrition. As societies shift to relatively low fertility rates and an increase occurs in the mean age of the population, noncommunicable diseases come to predominate, due in large part to shifts in dietary patterns (30, 90). Historically in countries now fully industrialized, as personal and community resources improved, the nutrition transition involved changes from traditional, agriculturally based low-fat, high-fiber diets to diets rich in animal fats, rich in refined and processed foods, and relatively low in fiber (111). In recent decades, the rapid urbanization of developing countries (44, 98) and the dramatic increase in the availability of inexpensive vegetable fats (30) have altered the historical pattern of the nutrition transition. In much of the developing world, the absolute number of poor and undernourished people living in urban areas has increased, as has the urban share of overall poverty and undernutrition (44). This pattern of urbanization, in combination with increased consumption of dietary fat and refined foods and changes in physical activity levels (30, 34, 91, 98), has created a situation in which the nutrition and epidemiologic transitions are occurring at ever-lower levels of economic development (30), and noncommunicable diseases are coexisting with infectious and nutrient-deficiency diseases (92, 99, 111).

Much of West Africa can be considered as being in the early stage of the transition processes. Sub-Saharan Africans contend with very high rates of morbidity and mortality from infectious disease, a situation that is worsening as the prevalence of HIV/AIDS increases (81), and they continue to struggle with undernutrition,

manifested most prominently as stunting in children (5). In many Caribbean and South American countries of the diaspora, the nutrition and epidemiologic transitions are well under way, accompanied by increases in dietary fat intake and the prevalence of obesity and associated chronic diseases (38, 45, 116). At the same time, undernutrition among children continues to exist in many communities (75, 87, 99). The United States and United Kingdom represent the late stage of the epidemiologic and nutrition transitions. Chronic, degenerative diseases of nutrition excess are the leading causes of ill health among both African-Americans and the black populations of the United Kingdom (15, 78).

## UNDERNUTRITION AMONG CHILDREN OF THE DIASPORA

Although in the late twentieth century large-scale famines and frank starvation became relatively rare, except in regions embroiled in war or civil unrest (40, 45), undernutrition is still prevalent in many parts of West Africa and in some regions of the Caribbean and Latin America (33, 87).

Of the regions relevant to this report, West Africa experiences by far the greatest degree of undernutrition. In reporting the proportion of chronically undernourished people, the Food and Agriculture Organization of the United Nations estimates the minimum energy requirements of populations, i.e. 1.54 times the estimated average basal metabolic rate, and compares that with the available food supply (14, 35). Based on these criteria, between 1990 and 1996, 40% of sub-Saharan Africans did not have access to adequate supplies of energy (35). This burden is disproportionately experienced by children and childbearing women and is most extreme in populations undergoing governmental instability and/or armed conflicts (40). In children, depending on the time of deprivation, inadequate energy and/or protein intake manifest as stunting, i.e. height-for-age more than two standard deviations (SD) below the US National Center for Health Statistics reference values, or wasting, i.e. more than two SD below means of weight-for-height (113). Stunting, of course, reflects more than physical insults; more than acute wasting, it has been linked to delayed mental development and measurable deficits in behavior and cognitive performance in school-aged children (3, 42, 112).

A 1993 nutritional survey among Nigerian children between the ages of 6 and 71 months reported a national prevalence of 38% for stunting and 19% for wasting (33). This high degree of stunting has been observed in other West African surveys among preschool children, e.g. 61% stunted and 7% wasted in a smaller group of Nigerian children (2), and 27% stunted and 4% wasted in northern Ghana (106). Stunting is diagnostic of low energy intakes during critical stages of development; however, data collected by our group in rural southwestern Nigeria (Yoruba land) may offer some insight into the potential for catch-up growth in this population. Over 47% of the boys and 17% of the girls between the ages of 12 and 19 years had heights more than two SD below the mean of comparably aged African-American



children. By adulthood, however, less than 10% of either gender was of short stature (25). The implications of this apparent adolescent catch-up growth for long-term health and cognitive performance are not clear and need further investigation.

Vitamin A deficiency is highly prevalent in most of the developing world, including West Africa. The overall prevalence of subclinical deficiency was reported as 9%–25% in Ghanaian and Nigerian preschoolers (33, 106). The highest rate of clinical vitamin A deficiency, presenting as xerophthalmia, was reported as 1% in the Sahel and the northern arid regions of those countries (1, 5, 33). Although most West African dietary patterns include the intake of red palm oil, which has very high concentrations of carotenoids, and seasonal consumption of vitamin A-containing fruits, there is some evidence that vitamin A-rich foods are introduced into children's diets at a late stage (33). Iron-deficiency anemia among children and women of childbearing age is a significant problem in much of West Africa. Numerous surveys have estimated the prevalence rates of anemia to be from 55% to 92% in children (1, 33, 48, 106), 27% in adults, and 53% in pregnant women (48). There are many contributing factors to the high rates of iron deficiency, including the low intake of meat, fish, and vitamin C-containing fruits, the high intake of plant foods with low concentrations of bioavailable iron, and the endemic malaria parasitemia (33). As noted, most rural diets consist of relatively few foods (101; A Luke, unpublished information), which undoubtedly contributes to documented vitamin A and iron deficiencies and has a negative impact on overall health.

In the Caribbean and Brazil, the prevalence of childhood stunting and wasting have decreased significantly over the past 3–4 decades as economic and social conditions have improved (45, 75, 103). Based on national nutrition survey data, the prevalence of undernutrition in Brazil, defined as weight-for-age more than two SD below the National Center for Health Statistics reference means, fell by more than 60% between 1975 and 1989 (75), with an additional 20% drop between 1989 and 1994 (87). The cities were found to have about half the rate of undernutrition (10%) of the rural areas (19%) (74), and the highest prevalence was in the traditionally poor north and northeast regions of the country (74, 75). Similar decreases over the past three decades have been reported for Jamaica, e.g. between 1970 and 1985, the rate of undernutrition decreased from 10.8% to 8.0% (87, 103). Many of the Caribbean islands currently report similar levels of low weight-for-age (87, 103). The primary exception to this late-twentieth-century improvement in the nutritional status of children has been in economically depressed, politically unstable Haiti, where the prevalence of severe stunting in five-year-old children was greater than 40% in 1996 (87). Much of the regional decrease in rates of undernutrition in the Caribbean and Brazil has been attributed to modest improvement in family incomes associated with a substantial increase in health services and programs (75, 103, 112).

Although undernutrition is present among African-Americans and among the black populations in the United Kingdom and in the wealthier of the Caribbean nations, such as the Bahamas, the far greater concern is that of the dramatically increasing prevalence of overweight, obesity, hypercholesterolemia, and associated

health risks among children (41, 103). These findings conform to the expectations for populations in the final stage of the epidemiologic and nutrition transitions. There continues to be a real need among governmental and health organizations to improve the nutritional status among children while avoiding the problems associated with increased buying power.

ADULT ANTHROPOMETRICS AND OBESITY

The long-term impact of diet and nutrition, as well as overall health status, can be observed from the comparison of adult anthropometric values within and across the populations of the African diaspora. Comparison of heights and weights of adults within a population over time provides an indication of changes in the availability of energy and nutrient sources, balanced against the requirement for physical activity. Anthropometric and dietary data were collected in the late 1950s in seven villages in Nigeria (80) and in urban and rural sites in Jamaica (4). One of the first databases in the United States in which race was identified was the first National Health and Nutrition Examination Survey (NHANES I) (77). Mean height, weight, and body mass index (BMI) from these early studies were compared with data from more recent population-based surveys conducted by the International Collaborative Study on Hypertension in Blacks (ICSHIB) (see Table 1), described in more detail

TABLE 1 Change in height, weight, and body mass index (BMI) of Nigerian, Jamaican, and African-American adults<sup>a</sup>

Determinant	N	Height (cm)		Weight (kg)		BMI		Reference
		M	W	M	W	M	W	
Nigeria								
1959, rural <sup>b</sup>	448	158.5	167.7	50.5	58.4	20.0	20.4	80
1995, rural	1666	157.9	168.3	58.5	62.5	22.9	22.7	25
Difference		−0.6	+0.6	+8.0	+4.1	+2.9	+2.3	
Jamaica								
1963, urban	736	159.0	170.2	60.3	66.1	23.9	22.8	4
1995, urban	1031	161.9	172.9	74.0	71.7	28.2	23.9	25
Difference		+2.9	+2.7	+13.7	+5.6	+4.3	+1.1	
US								
1971–1974, national	1333	162.7	174.7	71.5	78.1	26.9	25.6	78
1995, urban	1151	163.9	177.0	82.2	84.6	30.5	26.9	25
Difference		+1.2	+2.3	+10.7	+6.5	+3.6	+1.3	

<sup>a</sup>Mean values; ages 25–55 years. M, Men; W, women.

<sup>b</sup>Includes ages >13 years.

below (25). Although the sampling methods were not the same in the early and recent surveys, there was overlap in the sampling areas. For Nigeria, the 1959 data used in Table 1 were those collected from adults aged 25–55 years in two of the seven total villages in which cassavas and yams were the primary sources of energy; this diet was comparable to that of the ICSHIB sample (80). The 1959 Jamaican data were those collected in urban areas, again comparable to the ICSHIB sample (4). The ICSHIB sample from the United States was not significantly different in terms of height and weight from the African-American NHANES III sample (59).

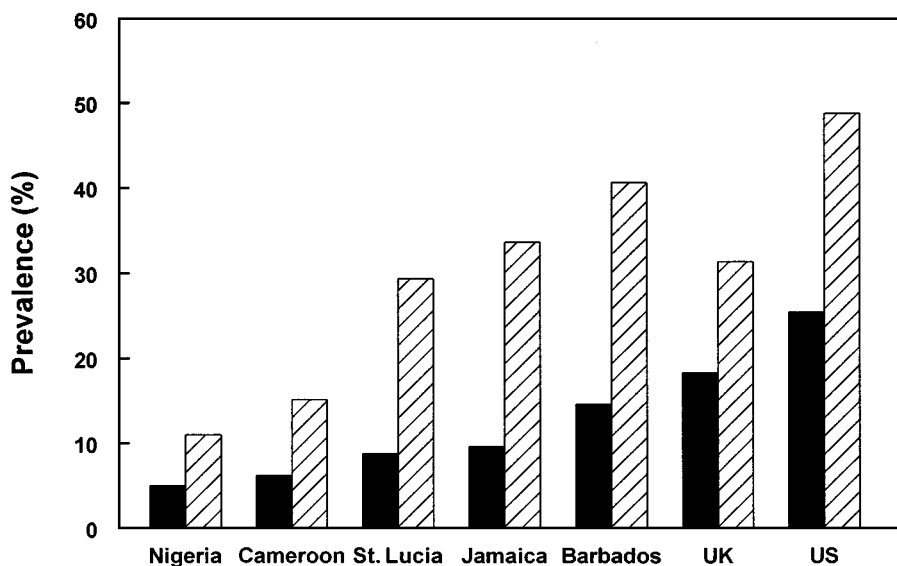
As can be observed from Table 1, average height increased slightly between the surveys among the Jamaican and US samples but did not change over the 35-year interval in Nigeria. In contrast, weight and BMI increased between the surveys across all sites and for both genders, with the largest increases in BMI occurring among women. These data suggest that the availability of energy for adults has increased in each of these three populations over the 25–35 years between surveys. The lack of change in mean height among Nigerian adults further suggests that there was no increase in the supply of energy and/or protein during the vulnerable growth periods in childhood (113). These comparisons of adult anthropometric values over time provide some evidence of the extent of the nutrition transition within populations; from these data it is clear that women of African origin, particularly in Jamaica and the United States, have been affected by the changes in dietary patterns more than men.

The ICSHIB study was a large-scale project designed to describe the biological evolution of hypertension over the course of the African diaspora (6, 25). The aims of the first phase of the study were to determine the magnitude of associations between blood pressure and known major risk factors in each population, to compare levels of blood pressure and hypertension prevalence, and to determine the extent to which contrasts between populations were due to differences in the level of risk factors (6). The initial phase of the ICSHIB study was a population-based survey of over 10,000 individuals, aged 25–74 years, from West Africa (Nigeria, Cameroon), the Caribbean (Barbados, Jamaica, St. Lucia), the United States (Chicago, Illinois), and the United Kingdom (Manchester). In each site, height, weight, waist and hip circumferences, blood pressure, and urinary sodium and potassium were measured using standardized protocols (6, 25). In Nigeria, Jamaica, and the United States, body composition was estimated using bioelectrical impedance analysis (65, 66).

As illustrated by the reduced data sets presented in Table 1, for each of the measured anthropometric variables there was a distinct gradient in the mean values, with lowest levels observed in Nigeria and rural Cameroon and highest levels found in the United States, for both women and men (95). For example, height increased from a mean of 158.2 cm for Nigerian women to 163.4 cm for African-American women, with values in the middle for Barbados and Jamaica (160.5 cm). The same pattern was observed for height of men, and for weight and waist and hip circumferences of both genders. Mean body weight ranged from 57 kg for Nigerian women to 83 kg for US women, and from 62 kg among Nigerian men to 85 kg for US men. Waist circumferences among women ranged from a low of 74 cm to a

high of 91 in Nigeria and the United States, respectively, with the same degree of variation in hip circumference (83, 95). Among men, waist circumferences ranged from 77 cm in Nigeria to 92 cm in the United States. In contrast to the wide variation in waist and hip circumferences, the mean waist-hip ratios did not vary across sites. Men from Nigeria, rural Cameroon, Barbados, and the United States had the same mean waist-hip ratio (0.88–0.89), whereas women from all sites except rural Cameroon did not differ in this measurement (0.79–0.82) (25, 95).

BMI displayed an increasing gradient in mean values from West Africa to the Caribbean to the United States for both men and women (53, 95). Among men, the lowest BMIs were from Nigeria (mean = 21.7) and rural Cameroon (mean = 23.5). BMI values were slightly higher among men in the Caribbean, Jamaica (mean = 23.8), St. Lucia (mean = 24.3), and Barbados (mean = 25.9), and highest in the United States (mean = 27.1). For women, there was a dramatic increase in mean values between West Africa and the Caribbean and the United States. In Nigeria and rural Cameroon, mean BMI of women was 22.6 and 23.5, respectively, whereas in Jamaica it was 27.9, in St. Lucia 27.3, in Barbados 29.4, and in the United States 30.8. There was a marked east-to-west increasing gradient in the prevalence of obesity, defined as a BMI  $\geq 30.0$  (79, 121) (Figure 2). The prevalence of obesity was lowest for Nigerian men (5%) and highest for African-American women (49%). In the three sites where body composition was estimated, Nigeria, Jamaica, and the United States, the levels of fat-free mass did not differ for



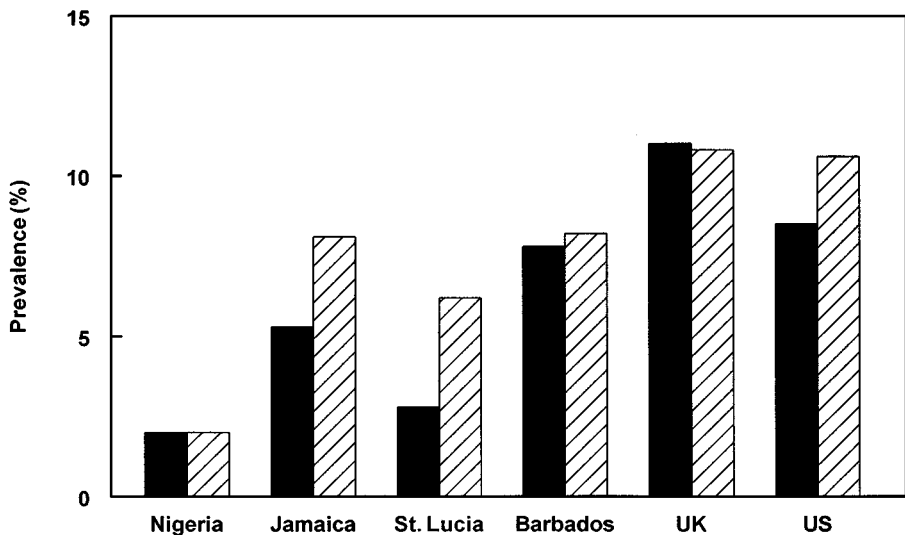
**Figure 2** Age-adjusted prevalence of obesity, defined as body mass index  $\geq 30$ , in seven populations of the African diaspora by gender; men are represented by solid bars, women by hatched bars. (Data from References 25 and 95.)

men (mean = 57 kg) or women (mean = 45 kg) (65). The difference in weight between these populations, as described above, was due to significant differences in fat mass within each gender across the three populations. Mean percentages of body fat levels were 11%, 19%, and 25% for men and 25%, 36%, and 40% for women in Nigeria, Jamaica, and the United States, respectively. Not only did adults in Jamaica and the United States have greater mean levels of body fat, the relationships between adiposity and BMI were shifted to the left, such that a BMI of 25 represented a body fat level of 16.4%, 22.2%, and 25.8% for Nigerian, Jamaican, and US men, respectively (65). The same shift was observed in women.

Obesity, of course, is a syndrome with a variety of potential underlying causes. Whatever the specific etiologies for the increases in obesity prevalence across the African diaspora, the consequences are readily observable in the morbidity and mortality patterns resulting from obesity-related chronic diseases, most notably non-insulin-dependent diabetes mellitus (NIDDM), hypertension and cerebrovascular disease, and coronary heart disease (CHD).

## DIABETES IN THE DIASPORA

More than any other chronic disease, NIDDM is most strongly associated with obesity, total body fat, and abdominal fat (82, 89). As a shadow cast by obesity, there is a distinct east-to-west gradient in the prevalence of NIDDM among adults of the diaspora (Figure 3) (26). In 1901, Albert Cook, a medical missionary to Uganda,



**Figure 3** Age-adjusted prevalence of non-insulin-dependent diabetes mellitus, by self-report or fasting blood glucose  $\geq 6.7$  mmol/liter, in six populations of the African diaspora by gender; men are represented by solid bars, women by hatched bars. (Data from Reference 26.)

recognized that diabetes was relatively rare in sub-Saharan Africa, reporting that "... diabetes is rather uncommon and very fatal..." (see 71). Since the 1960s, several studies have attempted to determine the prevalence of NIDDM in sub-Saharan Africa (57, 71, 86). Based on population samples ranging in age from 30 to 64, prevalences between 0.5% and 4% have been reported (26, 71, 76, 109). The exception appears to be the more urbanized, industrialized South Africa, where rates between 4.8% and 8.0% were recorded, and where obesity is also relatively prevalent (62, 73). The low prevalence rates observed in much of Africa may also be affected by the lack of treatment, and by the consequent high mortality. A recent study in rural and poor urban Nigeria suggests that as many as 20% and 35% of the elderly population were hyperinsulinemic and insulin resistant, respectively (31). It is hard to reconcile these data with estimates from the same communities of the prevalence of frank NIDDM being 2%, however, and this needs to be investigated further (26, 97). Even with the very low prevalence of NIDDM among indigenous Africans [rates are higher among Africans of Asian origin (56, 71)], a consistent positive association has been reported with BMI (26, 62, 109).

The positive association between BMI and NIDDM persists across the African diaspora, at both the individual and the population level. In the Caribbean the prevalence among black adults, especially women, increases sharply compared with those in West Africa (Figure 3). Based on self-report and/or fasting blood glucose concentration  $\geq 6.7$  mmol/liter (120), the age-adjusted prevalences were 2.8%, 7.8%, and 5.3% for men and 9.0%, 8.4% and 10.4% for women in St. Lucia, Barbados, and Jamaica, respectively (26). Using a more stringent diagnosis based on an oral glucose-tolerance test in the same Jamaican population, Wilks et al reported a prevalence of 9.8% for men and 15.7% for women for NIDDM (117). In the Jamaican sample, the population's attributable risk for diabetes, i.e. the proportion of NIDDM in the population that is due to the exposure of being overweight (i.e. BMI  $\geq 25$ ) was 66% and of having a waist-to-hip ratio greater than the median (0.80) was 80% (117). A multicenter study on NIDDM conducted in nine Brazilian cities between 1986 and 1988 reported a mean prevalence of 7.6% for the urban population aged 30–69 years, with higher rates in Sao Paulo (9.7%) and Porto Alegre (8.9%) (87). With the rate of obesity increasing in Brazil (74, 99), it is likely that current rates of NIDDM currently exceed these estimates.

In population-based samples from Chicago, Illinois, and Manchester, England, the overall age-adjusted prevalence of NIDDM among blacks was reported to be 10.6% and 10.8%, respectively (26, 97). In the United States, as in the Caribbean populations, the rate was higher for women than men (12.3% vs 8.5%, respectively). Among the African-Caribbean adults living in Manchester, however, no difference between genders was reported in this study (26). Separate studies conducted in London reported the prevalence of NIDDM to be somewhat higher than the Manchester study, about 15% and 18%, respectively, for men and women originating from the West Indies (15, 19).

Not only is NIDDM more prevalent among blacks in the western hemisphere relative to West Africa, it is also more prevalent among blacks than whites in both

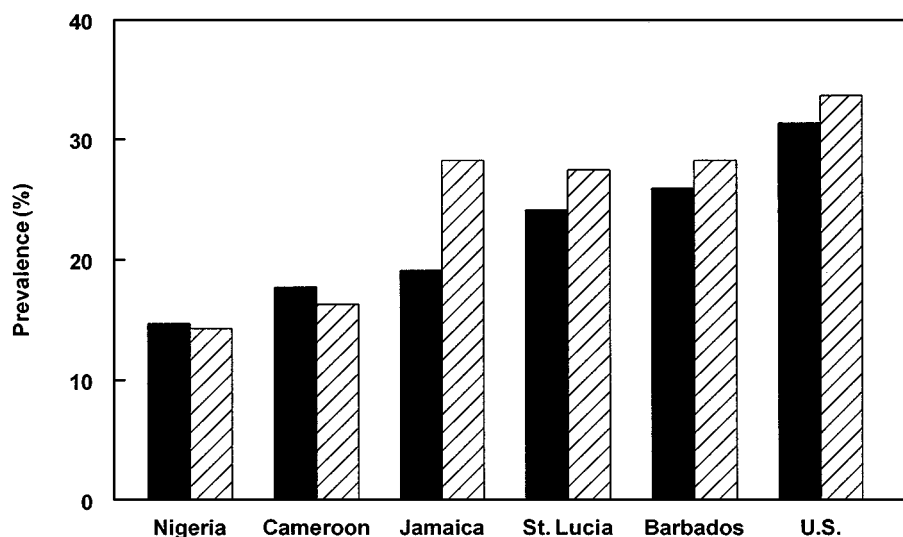
the United States (17, 56) and the United Kingdom (15). Although obesity is clearly a primary risk factor for the development of NIDDM, it does not completely explain the excess incidence of NIDDM in African-Americans. In a 16-year follow-up to NHANES I, African-American adults had one and a half to two times the incidence of NIDDM at comparable levels of obesity (64). Not only are the prevalence and incidence of NIDDM greater, health outcomes such as end-stage renal disease (28), retinopathy (46), and lower-extremity amputations (61) tend to be more frequent and severe among African-Americans. In contrast, an 18- to 20-year follow-up of diabetics in the United Kingdom reported comparable BMI and blood pressure but lower serum cholesterol levels and much lower risk ratios for all-cause (0.59) and coronary heart disease mortality (0.37) for African-Caribbean relative to European diabetics (19).

The prevalence of NIDDM increases colinearly with obesity among populations of the African diaspora, and blacks in the West currently have higher rates and suffer more comorbidities than do whites. With particular regard to NIDDM, a clear goal of public health nutritionists will be to minimize the increase in obesity that occurs as populations undergo the nutrition transition.

## HYPERTENSION IN THE DIASPORA

Hypertension is the most common cardiovascular condition in the world; the life-time risk approaches 50% in most populations. Among the known risk factors for hypertension, nutritional aspects of lifestyle figure prominently, including obesity, a low intake of fruits and vegetables, and a high intake of sodium. Persons of African descent in the United States have been recognized since the 1930s to have a higher incidence of this condition than do whites (105). Early on, a tendency developed to think of the black experience with hypertension as an exceptional case and, despite limited evidence, to speculate that the condition was common in Africa (114). Recent large-scale surveys have in fact demonstrated low prevalences in rural Africa, with the expected gradient to urban areas, the Caribbean, and the United States paralleling trends in obesity and sodium intake (25, 53, 54). Among black populations in the West Indies, the hypertension burden is similar to that of US and European whites (i.e. 25%) (25), whereas in the United States, prevalences are significantly higher (i.e. 35%) (see Figure 4). The pattern of vascular disease, especially stroke, follows the gradient one would anticipate given the population levels of hypertension (84). The extent to which these trends are simply the result of multiple environmental exposures or unique predisposition on the part of this population group is much debated (27). In general, however, it is now clear that the pattern observed across populations of the African diaspora is consistent with what might be predicted from the epidemiologic experience.

In effect, the case for "black exceptionalism" rests on the slope of the cross-cultural gradient. That is, it remains possible that the rate of increase, given the exposures, is steeper than would be observed for other groups because the prevalences



**Figure 4** Age-adjusted prevalence of hypertension, defined as systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg, or having treatment for hypertension, in seven populations of the African diaspora by gender; men are represented by solid bars, women by hatched bars. (Data from Reference 25.)

in the United States and the United Kingdom are higher than for whites. Of course, this exercise ignores the potential contribution of other less well-characterized factors, such as socioeconomic status and psychosocial stressors. Furthermore, it is not often recognized that among Finns, Russians, and Poles, hypertension rates are similar to those of African-Americans (22); whether all these populations achieve this exceptional status by the same route, namely nutritional excesses, or whether different combinations of risk factors are present in each group has not been well studied.

A more quantitative formulation of this question, that is, what proportion of the change in prevalence can be attributed to measured risk factors, quickly reveals the limitations of the epidemiologic method. A multistage analysis is required to answer this question. First, similar relationships need to be observed at the individual level between the exposures and the outcomes, e.g. salt and blood pressure. Second, the trends in exposures need to be collinear across populations, making it possible to ignore interactions, and the absence of unmeasured confounders must be assumed. If these conditions are met, an ecologic analysis could be undertaken, with the population as the unit of analysis and mean risk factor as the exposure. Clearly for diseases like hypertension, where one is trying to model lifetime effect of multiple, poorly characterized exposures, this analysis is approximate. Unfortunately, this imprecision has been interpreted as license to invoke genetic predisposition as the cause of more hypertension among blacks than whites in the United States and the United Kingdom (119).



An examination of the relative impact of specific nutritional factors across the African diaspora is complicated by the imprecision of the survey methods and the interactions that occur between them. Recent data suggest that in such low-risk groups as rural Nigerians, the associations between measurable exposures and blood pressure at the individual level are stronger than among high-risk groups, where multiple exposures coexist (12, 52). Thus, the correlation between 24-h sodium excretion and blood pressure was as high as 0.4 among rural Nigerian men compared with 0.10–0.15 for “westernized” populations (54). Preliminary data also suggest that the blood pressure response to changes in dietary sodium in these communities may be larger than seen elsewhere (A Adeyemo, unpublished information). By the same token, the correlation between BMI was also slightly greater in African groups (53). Parallel research on pathophysiologic measures, such as circulating hormone levels, reinforces the impression that the within-person relationships are better defined among the low-risk groups, implying that the underlying control mechanisms have become less disordered (37). This pattern, which has been confirmed in studies of experimental animals (H Jacobs, unpublished information), suggests that further insights into the evolution of disease patterns could be obtained by comparative studies across these populations (27). The potential also exists to use this “population laboratory” in combination with modern molecular genetic techniques to examine gene-environment interactions, and as a unique epidemiologic resource, the African diaspora could make it possible to address entirely new questions. Excessive concern with prevalence estimates and comparisons between whites and blacks has prevented many investigators from seeing this opportunity (119).

Although the quantitative results are only approximate, it is abundantly clear that nutritional status accounts for the majority of the increase in hypertension risk comparing Africans to African-Americans (53, 83). Not only are black populations in the west more overweight, they also consume much more sodium and have differing dietary patterns than do West Africans, e.g. Nigerians do not add salt to their food once it is prepared (54; A Adeyemo, unpublished information). The opportunities for prevention are therefore substantial. In fact, it is difficult to see how any progress toward prevention of this disease will take place without successful programs to reduce sodium in the food supply, maintain normal body weight, and increase intake of fruits and vegetables.

## OTHER NUTRITION-RELATED OUTCOMES

Data on prevalence and incidence rates for many other nutrition- and obesity-related chronic diseases, e.g. certain cancers, osteoarthritis, and sleep disorders, are sparse or nonexistent for much of sub-Saharan Africa and the Caribbean. The interaction of overall nutritional status and/or specific nutrients and cancer, in particular colon, breast, and prostate cancers, are extremely active areas of research. We hope future cross-cultural studies will shed light on the roles nutrition plays in the development and progression of cancer in black populations. CHD is another

such disease; in this case, however, some data are available. Although limited, available data suggest a prevalence pattern among populations of African origin similar to that observed for NIDDM and hypertension, i.e. very low rates in West Africa, increasing rates in the Caribbean, and relatively high rates in the United States and United Kingdom. As with the other two diseases, the pattern for CHD follows the epidemiologic and nutrition transitions of the diaspora.

In Africa and much of the Caribbean, the proportion of mortality attributable to CHD is difficult to estimate with precision because of the overall dearth of vital status information (24). Much of the data for Africa come from the 1960s and 1970s, during which time CHD was documented at autopsy as the cause of death in 0.5% of examined cases in Uganda, 1.5% in Ibadan, Nigeria, and 1.2% in Accra, Ghana (49). In the Caribbean, the available mortality statistics suggest an increased rate of death due to CHD. Approximately 8% of all deaths in Brazil in 1995 and 23% in Cuba in 1996 were attributed to CHD, although it must be remembered that these are not race-specific data and the majority of Brazilians and Cubans are not of African descent (122). In Jamaica, 13.1% of all deaths between 1996 and 1998 were due to CHD (87). In the United States, CHD is the leading cause of death among both blacks and whites, with death rates of 99.4 and 86.4, respectively, per 100,000 resident population in 1996, or about 20% of all deaths for both races (78). Although the overall incidence rates are comparable between the races (23), blacks in the United States suffer a higher rate of fatal events, and for the first time since the CHD data were recorded in vital statistics, the age-adjusted death rates from CHD are higher among black than white men (63). The well-documented decline in CHD mortality since the mid-1960s has been steady among whites whereas the rate of decline has decelerated among blacks such that since 1980, the absolute mortality gap between blacks and whites has steadily increased (63). Among adults in the United Kingdom, black immigrants from the Caribbean and West Africa tend to have a significantly lower incidence of and mortality from CHD than do either their South Asian counterparts or European whites (7, 15, 115).

There are several causal risk factors for CHD, most significantly serum lipid levels, hypertension, smoking, and physical inactivity, which likely impact the differential CHD mortality rates observed in the populations of interest (58, 76). Differences in dietary patterns and obesity prevalence across the diaspora have influenced population-level serum lipid concentrations. Both population-based surveys (51, 85, 96) and those among targeted subgroups, i.e. civil servants (13) and rural and urban elderly (31), reported low total serum cholesterol concentrations among Nigerians relative to contemporary mean values for African-Americans. For example, mean serum cholesterol concentrations for Nigerian and African-American adults from the ICSHIB survey were 3.75 and 5.00 mmol/liter, respectively (96). Jamaican serum cholesterol values from the same survey fell between these two, 4.73 mmol/liter (R Wilks, unpublished information). In each of the NHANES and the Coronary Artery Risk Development in Young Adults studies, total cholesterol concentrations for African-American adults have been comparable to those for whites, whereas high-density lipoprotein cholesterol (HDL) levels

tended to be higher among African-Americans, particularly men (23, 39, 110). In contrast, HDL levels for Nigerians and Jamaicans were lower than for African-Americans but comparable to whites (96; R Wilks, unpublished information). The differences in serum lipid levels observed among the populations of the diaspora are likely the result of high intakes of total and saturated fats in the West, which will increase both total and HDL cholesterol levels, and lower intakes of fats in Africa and the Caribbean, resulting in lower serum levels and lower risk for CHD mortality.

## SUMMARY

Millions of Africans were sacrificed through the trans-Atlantic slave trade to lay a critical piece of the foundation for the modern capitalist world. They brought with them many of their foods and dietary customs, which have had an impact on nutritional status throughout the Americas. The descendants of the African slaves in diaspora now represent populations at varying stages of the nutrition transition. West Africans, in the early stages of the transition, consume diets relatively low in fat and containing few highly processed foods. Undernutrition among children exists, and obesity and its adverse health consequences, e.g. NIDDM, hypertension, and CHD, are uncommon in most West African countries. Diets of Caribbean populations tend to have somewhat higher levels of fat, between 25% and 30% of total kilocalories. Undernutrition among children has declined sharply in the past few decades, but the prevalence of obesity and its sequelae are much higher in most Caribbean countries than in West Africa. The United States and the United Kingdom represent the last stages of the transition, in which public health concerns have shifted almost completely from undernutrition and deficiency diseases to those of excess, as illustrated by the very high prevalence of obesity and chronic diseases among black adults in these countries. It is hoped that with the awareness arising from public health research, the last stage of the nutrition transition will eventually be one in which obesity, NIDDM, CHD, and hypertension will be prevented or controlled and the majority of diets will be nutritionally replete. The African diaspora provides the opportunity for the investigation of the nutrition transition and dramatically illustrates the need for public health measures to be implemented in the Caribbean and West Africa to eradicate undernutrition while preventing obesity and its consequences.

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## LITERATURE CITED

1. Adelekan DA, Adeodu OO. 1998. Anaemia in Nigerian mothers and their children: relative importance of infections and iron deficiency. *Afr. J. Med. Sci.* 28:185–87
2. Adelekan DA, Fatusi AO, Fakunle JB, Olotu CT, Olukoga IA, et al. 1997. Prevalence of malnutrition and vitamin A deficiency in Nigerian preschool children subsisting

- on high intakes of carotenes. *Nutr. Health* 12:17–24
3. Allen LH. 1993. The nutrition CRSP: What is marginal malnutrition, and does it affect human function? *Nutr. Rev.* 51:255–67
  4. Ashcroft MT, Ling J, Lovell HG, Miall WE. 1966. Heights and weights of adults in rural and urban areas of Jamaica. *Br. J. Prev. Soc. Med.* 20:22–26
  5. Ashworth A, Dowler E. 1991. Child malnutrition. See Ref. 32, pp. 122–33
  6. Ataman SL, Cooper R, Rotimi C, McGee D, Osotimehin B, et al. 1996. Standardization of blood pressure measurement in an international comparative study. *Clin. Epidemiol.* 49:869–77
  7. Balarajan R. 1991. Ethnic differences in mortality from ischaemic heart disease and cerebrovascular disease in England and Wales. *Br. Med. J.* 302:560–64
  8. Banea-Mayambu J-P, Tylleskar T, Tylleskar K, Gebre-Medhin M, Rosling H. 2000. Dietary cyanide from insufficiently processed cassava and growth retardation in children in the Democratic Republic of Congo (formerly Zaire). *Ann. Trop. Paediatr.* 20:34–40
  9. Blackburn R. 1997. *The Making of New World Slavery*. New York: Verso. 602 pp.
  10. Block G, Dresser CM, Hartman AM, Carroll MD. 1985. Nutrient sources in the American diet: quantitative data from the NHANES II survey. *Am. J. Epidemiol.* 122:27–40
  11. Brieger WR. 1985. Food groups in cultural perspective. *Trop. Dr.* 15:42–43
  12. Bunker CH, Okoro FI, Markovic N, Thai N, Pippin B, et al. 1996. Relationship of hypertension to socioeconomic status in a West African population. *Ethn. Health* 1:33–45
  13. Bunker CH, Ukoli FA, Okoro FI, Olomu AB, Kriska AM, et al. 1996. Correlates of serum lipids in a lean black population. *Atherosclerosis* 123:215–25
  14. Buttler F. 2000. Ending hunger in developing countries. *Contemp. Sociol.* 29:13–27
  15. Cappuccio FP, Cook DG, Atkinson RW, Strazzullo P. 1997. Prevalence, detection, and management of cardiovascular risk factors in different ethnic groups in South London. *Heart* 78:555–63
  16. Carlson E, Kipps M, Thomson J. 1984. Influences on the food habits of some ethnic minorities in the United Kingdom. *Hum. Nutr. Appl. Nutr.* 38A:85–98
  17. Carter JS, Pugh JA, Monterrosa A. 1996. Non-insulin-dependent diabetes mellitus in minorities in the United States. *Ann. Intern. Med.* 125:221–32
  18. Chadwick DJ, Cardew G, eds. 1996. *The Origins and Consequences of Obesity*. New York: Wiley. 278 pp.
  19. Chaturvedi N, Jarrett J, Morrish N, Keen H, Fuller JH. 1996. Differences in mortality and morbidity in African Caribbeans and European people with non-insulin dependent diabetes mellitus: results of 20-year follow up of a London cohort of a multinational study. *Br. Med. J.* 313:848–52
  20. Cole AH, Taiwo OO, Nwagbara NI, Cole CE. 1997. Energy intakes, anthropometry and body composition of Nigerian adolescent girls: a case study of an institutionalized secondary school in Ibadan. *Br. J. Nutr.* 77:497–509
  21. Conniff ML, Davis TJ. 1994. *Africans in the Americas*. New York: St. Martin's. 356 pp.
  22. Cooper RS. 1998. Geographic patterns of hypertension: a global perspective. In *Hypertension Primer: The Essentials of High Blood Pressure*, ed. LJ Izzo, H Black, pp. 150–53. New York: Lippincott, Williams & Wilkins. 374 pp.
  23. Cooper RS, Ford E. 1992. Comparability of risk factors for coronary heart disease among blacks and whites in the NHANES-I Epidemiologic Follow-up Study. *Ann. Epidemiol.* 2:637–45
  24. Cooper RS, Osotimehin B, Kaufman JS, Forrester T. 1998. Disease burden in sub-Saharan Africa: What should we conclude

- in the absence of data? *Lancet* 351:208–10
25. Cooper RS, Rotimi C, Ataman S, McGee D, Osotimehin B, et al. 1997. The prevalence of hypertension in seven populations of West African origin. *Am. J. Public Health* 87:160–68
  26. Cooper RS, Rotimi CN, Kaufman JS, Owoaje EE, Fraser H, et al. 1997. Prevalence of NIDDM among populations of the African diaspora. *Diabetes Care* 20:343–48
  27. Cooper RS, Rotimi CN, Ward R. 1999. The puzzle of hypertension in African Americans. *Sci. Am.* 280:56–63
  28. Cowie CC, Port PK, Wolfe RA, Savage PJ, Moll PP, Hawthorne VM. 1989. Disparities in incidence of end-stage renal disease according to race and type of diabetes. *N. Engl. J. Med.* 321:1074–79
  29. Curtin PD. 1969. *The Atlantic Slave Trade: A Census*. Milwaukee: Univ. Wisconsin Press. 338 pp.
  30. Drewnowski A, Popkin BM. 1997. The nutrition transition: new trends in the global diet. *Nutr. Rev.* 55:1905–16
  31. Ezenwaka CE, Akanji AO, Akanji BO, Unwin NC, Adejuwon CA. 1997. The prevalence of insulin resistance and other cardiovascular disease risk factors in healthy elderly southwestern Nigerians. *Atherosclerosis* 128:201–11
  32. Feachem RG, Jamison DT, eds. 1991. *Disease and Mortality in Sub-Saharan Africa*. London: Oxford Univ. Press. 356 pp.
  33. Fed. Ministry Health Soc. Serv./USAID/VITAL/OMNI. 1996. *The Nigeria Micronutrient Survey 1993. Final Report*. Lagos, Nigeria: Fed. Ministry Health Soc. Serv. 105 pp.
  34. Ferro-Luzzi A, Martino L. 1996. Obesity and physical activity. See Ref. 32, pp. 207–27
  35. Food Agric. Org. 1998. *The State of Food and Agriculture 1998*. Rome: FAO. 396 pp.
  36. Ford ES, Giles WH, Croft JB. 2000. Prevalence of nonfatal coronary heart disease among American adults. *Am. Heart J.* 139:371–77
  37. Forrester T, Cooper R, Bennet F, McFarlane-Anderson N, Puras A, et al. 1996. Angiotensinogen and blood pressure among blacks: findings from a community survey in Jamaica. *J. Hypertens.* 14:315–21
  38. Forrester T, Wilks R, Bennett F, McFarlane-Anderson N, McGee D, et al. 1996. Obesity in the Caribbean. See Ref. 18, pp. 17–36
  39. Freedman DS, Strogatz DS, Williamson DF, Aubert RE. 1992. Education, race, and high-density lipoprotein cholesterol among US adults. *Am. J. Public Health* 82:999–1006
  40. Goldman R. 1999. Food and food poverty: perspectives on distribution. *Soc. Res.* 66:283–304
  41. Gortmaker SL, Dietz WH, Sobol AM, Wehler CA. 1987. Increasing pediatric obesity in the United States. *Am. J. Dis. Child.* 141:535–40
  42. Grantham-McGregor SM, Cumper G. 1992. Jamaican studies in nutrition and child development, and their implications for national development. *Proc. Nutr. Soc.* 51:71–79
  43. Greenberg MR, Schneider D, Northridge ME, Ganz ML. 1998. Region of birth and black diets: the Harlem Household Study. *Am. J. Public Health* 88:1199–202
  44. Haddad L, Ruel MT, Garrett JL. 1999. Are urban poverty and undernutrition growing? Some newly assembled evidence. *World Dev.* 27:1891–904
  45. Hagley KE. 1993. Nutrition and health in the developing world: the Caribbean experience. *Proc. Nutr. Soc.* 52:183–87
  46. Harris EL, Feldman S, Robinson CR, Sherman S, Georgopoulos A. 1993. Racial differences in the relationship between blood pressure and risk of retinopathy among individuals with NIDDM. *Diabetes Care* 16:748–54

47. Harris JB. 1998. *The Africa Cookbook*. New York: Simon & Schuster. 383 pp.
48. Hercberg S, Galan P, Dupin H. 1987. Iron deficiency in Africa. *World Rev. Nutr. Diet.* 54:201–36
49. Hutt MSR. 1991. Cancer and cardiovascular disease. See Ref. 32, pp. 221–40
50. James WPT, Ferro-Luzzi A, Waterlow JC. 1988. Definition of chronic energy deficiency in adults. Report of a working party of the International Dietary Energy Consultative Group. *Eur. J. Clin. Nutr.* 42:969–81
51. Kadiri S, Salako BL. 1997. Cardiovascular risk factors in middle aged Nigerians. *East Afr. Med. J.* 74:303–6
52. Kaufman J, Rotimi C, Cooper R. 1999. Blood pressure change in Africa. A case study from Nigeria. *Hum. Biol.* 71:641–57
53. Kaufman JS, Durazo-Avizu RA, Rotimi CN, McGee DL, Cooper RS, ICHSHIB Invest. 1996. Obesity and hypertension prevalence in populations of African origin: results from the International Collaborative Study on Hypertension in Blacks. *Epidemiology* 7:398–405
54. Kaufman JS, Owoaje EE, James SA, Rotimi CN, Cooper RS. 1996. Determinants of hypertension in West Africa: contribution of anthropometric and dietary factors to urban-rural and socioeconomic gradients. *Am. J. Epidemiol.* 143:1203–18
55. Kigutha HN. 1997. Assessment of dietary intake in rural communities in Africa: experiences in Kenya. *Am. J. Clin. Nutr.* 65:1168–72S
56. King H, Rewers M, WHO Ad Hoc Diabetes Rep. Group. 1993. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. *Diabetes Care* 16:157–77
57. Kinnear TWG. 1963. The pattern of diabetes mellitus in a Nigerian teaching hospital. *East Afr. Med. J.* 40:288–94
58. Knuiman JT, West CE, Katan MB, Hautvast JGAJ. 1987. Total cholesterol and high density lipoprotein cholesterol levels in populations differing in fat and carbohydrate intake. *Arteriosclerosis* 7:612–19
59. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. 1994. Increasing prevalence of overweight among US adults. *JAMA* 272:205–11
60. Kumanyika S. 1993. Diet and nutrition as influences on the morbidity/mortality gap. *Ann. Epidemiol.* 3:154–58
61. Lavery LA, Ashry HR, van Houtum W, Pugh JA, Harkless LB, Basu S. 1996. Variation in the incidence and proportion of diabetes-related amputations in minorities. *Diabetes Care* 19:48–52
62. Levitt NS, Katzenellenbogen JM, Bradshaw D, Hoffman MN, Bonnici F. 1993. The prevalence and identification of risk factors for NIDDM in urban Africans in Cape Town, South Africa. *Diabetes Care* 16:601–7
63. Liao Y, Cooper RS. 1995. Continued adverse trends in coronary heart disease mortality among blacks, 1980–91. *Public Health Rep.* 110:572–79
64. Lipton RB, Liao Y, Cao G, Cooper RS, McGee D. 1993. Determinants of incident non-insulin-dependent diabetes mellitus among blacks and whites in a national sample. *Am. J. Epidemiol.* 138:826–39
65. Luke A, Durazo-Arvizu R, Rotimi C, Prewitt TE, Forrester TE, et al. 1997. Relation between body mass index and body fat in black population samples from Nigeria, Jamaica, and the United States. *Am. J. Epidemiol.* 145:620–28
66. Luke AH, Rotimi CN, Cooper RS, Long AE, Forrester TE, et al. 1998. Leptin and body composition of Nigerians, Jamaicans, and US blacks. *Am. J. Clin. Nutr.* 67:391–96
67. Macfarlane S, Racelis M, Muli-Muslime F. 2000. Public health in developing countries. *Lancet* 356:841–46
68. Martorell R, Kettel Khan L, Hughes ML, Grummer-Strawn LM. 2000. Obesity in women from developing countries. *Eur. J. Clin. Nutr.* 54:247–52

69. Mazengo MC, Simell O, Lukmanji Z, Shirima R, Karvetti R-L. 1997. Food consumption in rural and urban Tanzania. *Acta Trop.* 68:313–26
70. McDowell MA, Briefel RR, Alaimo K, Bischof AM, Caughman CR, et al. 1994. Energy and micronutrient intakes of persons age 2 months and over in the United States: Third National Health and Nutrition Examination Survey, Phase 1, 1988–91. *Adv. Data Natl. Cent. Health Stat.* 255:1–23
71. McLarty DG, Pollitt C, Swai ABM. 1990. Diabetes in Africa. *Diabetes Med.* 7:670–84
72. Mennen LI, Mbanya JC, Cade J, Balkau B, Sharma S, et al. 2000. The habitual diet in rural and urban Cameroon. *Eur. J. Clin. Nutr.* 54:150–54
73. Mollentze WF, Moore AJ, Steyn AF, Joubert G, Steyn K, et al. 1995. Coronary heart disease risk factors in a rural and urban Orange Free State black population. *S. Afr. Med. J.* 85:90–96
74. Mondini L, Monteiro CA. 1997. The stage of nutrition transition in different Brazilian regions. *Arch. Latinoam. Nutr.* 47:17–24
75. Monteiro CA, Benicio MH, Iunes R, Gouveia NC, Taddei JAAC, Cardoso MAA. 1992. Nutritional status of Brazilian children: trends from 1975 to 1989. *Bull. WHO* 70:657–66
76. Muna WFT. 1993. Cardiovascular disorders in Africa. *World Health Stat. Q.* 46:125–32
77. Natl. Cent. Health Stat. 1979. *Weight by Height and Age for Adults 18–74 Years: United States, 1971–1974. Vital and Health Stat., Ser. 11, No. 208.* Washington, DC: US Gov. Print. Off.
78. Natl. Cent. Health Stat. 1998. *Health, United States, 1998 with Socioeconomic Status and Health Chartbook.* Hyattsville, MD: Natl. Cent. Health Stat. 460 pp.
79. Natl. Inst. Health. 1998. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. *Obes. Res.* 6:51–209S
80. Nicol BM. 1959. The protein requirement of Nigerian peasant farmers. *Br. J. Nutr.* 13:307–20
81. Ofofu-Amaah S. 1991. Disease in sub-Saharan Africa: an overview. See Ref. 32, pp. 119–21
82. Okosun IS, Cooper RS, Rotimi CN, Oso-timehin B, Forrester T. 1998. Association of waist circumference with risk of hypertension and type 2 diabetes in Nigerians, Jamaicans, and African-Americans. *Diabetes Care* 21:1836–42
83. Okosun IS, Forrester TE, Rotimi CN, Oso-timehin BO, Muna W, Cooper RS. 1999. Abdominal obesity in six populations of West African descent: prevalence and population attributable fraction of hypertension. *Obes. Res.* 7:453–62
84. Okosun IS, Muna WFT, Cooper R. 1998. International epidemiology of stroke in African populations outside the United States. In *Stroke in Blacks: A Guide to Management and Prevention*, ed. PB Gorelick, ES Cooper, RF Gillum, pp. 70–83. New York: Karger. 230 pp.
85. Ononogbu IC. 1979. Comparison of high density lipoprotein and serum cholesterol levels in a European and an African community. *Atherosclerosis* 34:49–52
86. Osuntokun BO, Akinkugbe FM, Francis TI, Reddy S, Osuntokun O, Taylor GOL. 1971. Diabetes mellitus in Nigerians: a study of 832 patients. *West Afr. Med. J.* 20:295–312
87. Pan Am. Health Org. 1999. *Basic Country Health Profiles, Summaries.* <http://www.paho.org>
88. Phillips PG. 1954. The metabolic cost of common West African agricultural activities. *J. Trop. Med.* 57:12–20
89. Pontiroli AE, Galli L. 1998. Duration of obesity is a risk factor for non-insulin-dependent diabetes mellitus, not for arterial hypertension or hyperlipidaemia. *Acta Diabetol.* 35:130–36
90. Popkin BM. 1994. The nutrition transition

- in low-income countries: an emerging crisis. *Nutr. Rev.* 52:285–98
91. Popkin BM, Paeratakul S, Zhai F, Ge K. 1995. A review of dietary and environmental correlates of obesity with emphasis on developing countries. *Obes. Res.* 3:145–53S
  92. Popkin BM, Richards MK, Monteiro CA. 1996. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *J. Nutr.* 126:3009–16
  93. Popkin BM, Siega-Riz AM, Haines PS. 1996. A comparison of dietary trends among racial and socioeconomic groups in the United States. *N. Engl. J. Med.* 335:716–20
  94. Ross PJ, Etkin NL, Muazzamu I. 1996. A changing Hausa diet. *Med. Anthropol.* 17:143–63
  95. Rotimi CN, Cooper RS, Ataman SL, Osotimehin B, Kadiri S, et al. 1995. Distribution of anthropometric variables and the prevalence of obesity in populations of West African origin: the International Collaborative Study on Hypertension in Blacks. *Obes. Res.* 3:95–105S
  96. Rotimi CN, Cooper RS, Marcovina SM, McGee D, Owoaje E, Ladipo M. 1997. Serum distribution of lipoprotein(a) in African Americans and Nigerians: potential evidence for a genotype-environmental effect. *Genet. Epidemiol.* 14:157–68
  97. Rotimi CN, Cooper RS, Okosun IS, Olatunbosun ST, Bella AF, et al. 1999. Prevalence of diabetes and impaired glucose tolerance in Nigerians, Jamaicans and US blacks. *Ethn. Dis.* 9:190–200
  98. Ruel MT, Haddad L, Garrett JL. 1999. Some urban facts of life: implications for research and policy. *World Dev.* 27:1917–38
  99. Sawaya AL, Dallal G, Solymos G, de Sousa MH, Ventura ML, et al. 1995. Obesity and malnutrition in a shantytown population in the city of São Paulo, Brazil. *Obes. Res.* 3:107–15S
  100. Sharma S, Cade J, Griffiths S, Cruickshank K. 1998. Nutrient intakes among UK African-Caribbeans: changing risk of coronary heart disease. *Lancet* 352:114–15
  101. Sharma S, Cade J, Jackson M, Mbanya JC, Chungong S, et al. 1996. Development of food frequency questionnaires in three population samples of African origin from Cameroon, Jamaica and Caribbean migrants to the UK. *Eur. J. Clin. Nutr.* 50:479–86
  102. Shyllon F. 1993. Blacks in Britain: a historical and analytical overview. In *Global Dimensions of the African Diaspora*, ed. JE Harris, pp. 223–48. Washington, DC: Howard Univ. Press. 532 pp.
  103. Sinha DP. 1988. Nutrition in the English-speaking Caribbean: a brief review of the changes over the last three decades. *Ca-janus* 21:113–32
  104. Sokolov R. 1991. *Why We Eat What We Eat*. New York: Summit. 255 pp.
  105. Stamler J, Stamler R, Pullman TN, eds. 1967. *The Epidemiology of Hypertension*. New York: Grune & Stratton. 472 pp.
  106. Takyi EEK. 1999. Nutritional status and nutrient intake of preschool children in northern Ghana. *East Afr. Med. J.* 76:510–15
  107. Thomas H. 1997. *The Slave Trade*. New York: Simon & Schuster. 908 pp.
  108. Thomas HM. 1972. Some aspects of food and nutrition in Sierra Leone. *World Rev. Nutr. Diet.* 14:48–58
  109. van der Sande MAB, Bailey R, Faal H, Banya WAS, Dolin P, et al. 1997. Nationwide prevalence study of hypertension and related non-communicable diseases in the Gambia. *Trop. Med. Int. Health* 2:1039–48
  110. Van Horn LV, Ballew C, Liu K, Ruth K, McDonald A, et al. 1991. Diet, body size, and plasma lipids-lipoproteins in young adults: differences by race and sex. *Am. J. Epidemiol.* 133:9–23



111. Vorster HH, Bourne LT, Venter CS, Oosthuizen W. 1999. Contribution of nutrition to the health transition in developing countries: a framework for research and intervention. *Nutr. Rev.* 57:341–49
112. Waterlow JC. 1979. Childhood malnutrition—the global problem. *Proc. Nutr. Soc.* 38:1–9
113. Waterlow JC. 1994. Childhood malnutrition in developing nations: looking backward and looking forward. *Annu. Rev. Nutr.* 14:1–19
114. White PD. 1967. Hypertension and atherosclerosis in the Congo and in the Gabon. See Ref. 105, pp. 150–54
115. Wild S, McKeigue P. 1997. Cross sectional analysis of mortality by country of birth in England and Wales, 1970–92. *Br. Med. J.* 314:705–10
116. Wilks R, McFarlane-Anderson N, Bennett F, Fraser H, McGee D, et al. 1996. Obesity in peoples of the African diaspora. See Ref. 18, pp. 37–53
117. Wilks R, Rotimi C, Bennett F, McFarlane-Anderson N, Kaufman JS, et al. 1999. Diabetes in the Caribbean: results of a population survey from Spanish Town, Jamaica. *Diabetic Med.* 16:875–83
118. Williams E. 1970. *From Columbus to Castro. The History of the Caribbean.* New York: Random House. 576 pp.
119. Wilson TW, Grim CE. 1991. Biohistory of slavery and blood pressure differences in blacks today. *Hypertension* 17:1122–28
120. World Health Org. 1980. *WHO Expert Committee on Diabetes Mellitus. Second Rep.* Geneva, Switzerland: WHO. 262 pp.
121. World Health Org. 1998. *Obesity. Preventing and Managing the Global Epidemic. Rep. WHO Consult. Obes.* Geneva, Switzerland: WHO. 276 pp.
122. World Health Org. 2000. *World Health Statistics Annual Mortality Data. WHO Statistical Information System.* <http://www.who.org>