Obesity and Diabetes in Blacks

F. Xavier Pi-Sunyer, MD

Diabetes mellitus is more prevalent in the American Black population than in the White population. The prevalence is increasing in Blacks, and there is evidence that it is accompanied by a greater severity of diabetic complications. In addition, mortality figures are higher in Blacks, and Black women are more seriously affected than Black men. Although the reasons for this are unclear, some factors stand out as important. These include obesity, socioeconomic status, and genetics. Obesity is a severe problem in Blacks, particularly in women. Both the degree and the distribution of fat may contribute greatly to the prevalence of diabetes in Blacks. Although the prevalence of obesity is higher in the poor economic groups, multivariate analysis suggests that poverty cannot explain all of the excess obesity that occurs in the Black population. More research is needed into a possible genetic predisposition of Blacks to obesity and diabetes and into the interrelationship between the two conditions in this racial group. *Diabetes Care* **13 (Suppl. 4):1144–49, 1990**

The prevalence of both obesity and diabetes mellitus is higher in American Blacks than in American Whites (1,2). Whether it is the higher prevalence of obesity that leads to the greater prevalence of diabetes is discussed herein. This article also reviews other potential reasons for the high levels of diabetes in American Blacks.

ASSOCIATION BETWEEN OBESITY AND DIABETES

In all ethnic and racial groups, a strong association exists between obesity and diabetes mellitus. Obesity is the most important general environmental determinant in the manifestation of diabetes in all peoples. West and Kalbfleisch (3), years ago, in a series of population studies involving many geographical areas, races, and cultures, noted a marked correlation between the prevalence of overweight and diabetes. This observation has been confirmed by many studies (4,5).

There is a strong correlation between relative weight and prevalence of type II (non-insulin-dependent) diabetes in population groups (1). Diabetes is 2.9 times higher in overweight than nonoverweight people in the Second National Health and Nutrition Examination Survey (NHANES II) data. Other cross-sectional studies have also shown that diabetic patients have an increased relative weight (6). There have been prospective studies in several countries, including the United States (7,8), Norway (9), Sweden (10), and Israel (11), which have shown that increasing weight increases the risk of diabetes.

Excess body fat, whatever the determinants, frequently results in a significant impairment of health (12,13). Obesity, even independent of diabetes, is a risk factor for hypertension and cardiovascular disease (7). When it is combined with diabetes, the risk of acquiring these illnesses can be greater, leading to significantly greater morbidity and mortality.

OBESITY

Both genetics and environment are likely to be involved in the pathogenesis of obesity. Some studies have made

From the Department of Medicine and Obesity Research Center, St. Luke's/Roosevelt Hospital Center, Columbia University, New York, New York.

Address correspondence and reprint requests to F. Xavier Pi-Sunyer, MD, Department of Medicine and Obesity Research Center, St. Luke's/Roosevelt Hospital Center, Columbia University, New York, NY 10025.

it clear that there is a strong genetic component to obesity (14,15). However, there is also a strong environmental factor (16). Thus, the environment in our western technologically advanced society influences body weight. The variety, palatability, availability, and cheapness of food in America tends to enhance calorie intake, and the ever-increasing mechanical aids tend to decrease energy expenditure (17).

Obesity means an excess of body fat. Although how much fat is abnormal has been debated in the U.S., individuals who are >20% over their ideal or desirable body weights by actuarial tables, such as those of the Metropolitan Life Insurance Company, are considered obese (18). In cross-sectional surveys of the population, such as NHANES II, people above the 85th percentile of weight for height for sex have been defined as obese. With body mass index (BMI) as a measure (weight [kg]/height^2 [m^2]), a level of >28 is generally considered obese (18). The Canadian guidelines (19) state that a BMI between 25 and 27 may lead to health problems in some people and a BMI >27 is associated with increasing risk of developing health problems.

Obesity is the leading form of malnutrition in the U.S. Defining overweight as BMI ≥27.8 for men and ≥27.3 for women, the NHANES II of 1976–1980 found 26% of adults, or ~34 million people aged 20–74 yr, to be overweight (1).

Obesity in relation to race. The prevalence of obesity varies along racial lines and is greatly influenced by sex and age. American women as a group are fatter than American men, although both groups have problems. Figure 1 shows the prevalence of overweight among White and Black women, with data taken from NHANES II (1). The prevalence of overweight increases with each decade from 20 to 65 years of age. It is also evident that the prevalence of overweight is about double for Black women compared with White women, reaching values of >60% of the population for women >45 yr of age. Similar data were found in the Ten-State Survey (20), with the prevalence of obesity for Black women exceeding 50% in the 45- to 55-yr-old age-group. This was higher than the 40% of White women aged 45–55 yr who were obese.

In men, the prevalence of obesity increases to 30% at age 45 yr and then tends to plateau at that level, as shown in Fig. 1. After 45 yr, weight flattens out for a

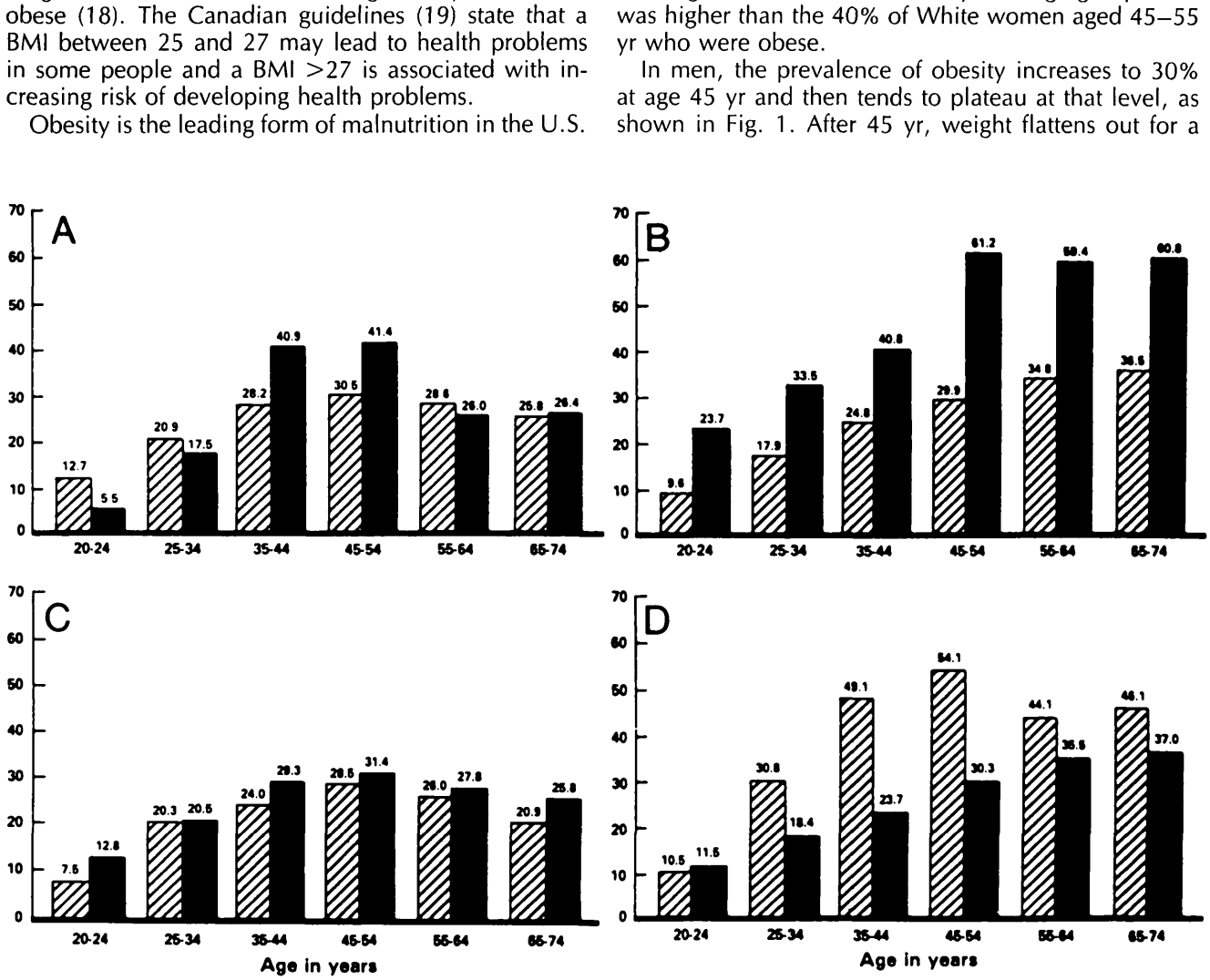

FIG. 1. *A*: percentage of men overweight by race and age. *Hatched bars*, Whites; *solid bars*, Blacks. *B*: percentage of nonpregnant women overweight by race and age. *Hatched bars*, Whites; *solid bars*, Blacks. *C*: percentage of men overweight by poverty status and age. *Hatched bars*, poverty; *solid bars*, nonpoverty. *D*: percentage of nonpregnant women overweight by poverty status and age. *Hatched bars*, poverty; *solid bars*, nonpoverty. From Van Itallie (1) with permission from the *Annals of Internal Medicine*.

decade and then after age 55 yr overweight declines. The data in Fig. 1 also show that Black men have similar patterns to White men except at 35–55 yr of age, when Black men have a clearly higher prevalence of obesity.

Weight gain. In a study of major weight gain over 10 yr in U.S. adults, Williamson et al. (21) showed that, among women 25–34 yr old, Black women gained on average 0.6 kg/m^2 more than White women and were 40% more likely to have experienced a major weight gain, defined as a 10-yr increase in BMI of >5.0 U (~20% weight gain). Among women aged 35–44 yr, Black women gained on average 0.4 kg/m^2 more than White women and were 30% more likely to have experienced a major weight gain. Among women who were not overweight at baseline, Black women were nearly twice as likely as White women to have become overweight during follow-up. Khoury et al. (22) also reported greater weight gain in Black than White women, and Kumanyika (23) reported similar findings extrapolated from NHANES II data. These three studies agree with the prevalence data described previously and point to the seriousness of the obesity problem in Black women.

Socioeconomic status. Obesity also varies according to socioeconomic status. In the data derived from NHANES II showing the prevalence of overweight in relation to poverty status, males under the poverty line (as defined by U.S. Bureau of the Census, 1985) have a somewhat lower mean weight at all ages. However, in women, those under the poverty line have a much higher prevalence of overweight than women in the nonpoverty groups. Stunkard (24) and Goldblatt et al. (25), in the mid-Manhattan study, reported that, at all ages, the poor are fatter and the affluent leaner. In this population sample, 30% of women of lower socioeconomic status were obese compared to 16% among those of middle-class status and no more than 5% in the upper-class status group. Among men, the differences were smaller but still present. Similar data were also reported by Silverstone et al. (26) in the United Kingdom. Because of the association of blackness and lower socioeconomic status, it has been difficult to determine how much of the increased incidence of obesity in different ethnic and racial groups is related to inherent genetic differences and how much to socioeconomic factors (24,25). Because 34.3% of Black men and women are below the poverty line, in contrast to 10.6% of White men and women (U.S. Bureau of the Census, 1985), poverty status may at least partly explain why Black people have higher rates of obesity.

Although poverty may explain part of the greater prevalence of obesity in Blacks, it cannot explain all of it. Multivariate analysis has demonstrated race to be an independent predictor of overweight status after adjustment for age, sex, and poverty status. The association of poverty status and overweight is different for men and women and is independent of race (1).

However, whatever the cause, the demographic data clearly show that the problem of obesity is greater in Black than White Americans and that it is a problem of epidemic proportions in Black women.

Regional fat distribution. Not only total body fat but where the fat is located is important. Regional fat cell distribution has a large influence with regard to the risk of diabetes. The risk of excess body fat can no longer be determined simply with BMI, the percentage of fat or the number of kilograms of excess body fat; the distribution of the fat is also extremely important. We must now think of fat not as one organ but as a number of organs, which may have differing impacts on health.

Android obesity is the propensity to deposit fat above the waist; gynecoid obesity is the propensity to deposit fat below the waist. Most men put on weight predominantly above the waist, i.e., around the middle, both subcutaneously and intra-abdominally. Women predominantly put on weight below the waist, in the thigh and gluteal areas. This regional deposition of fat has been defined with different criteria. Although some researchers have investigated upper-body obesity versus lower-body obesity, others have looked at central (truncal) fat versus peripheral (extremity) fat. Still others have suggested focusing on intra-abdominal fat. The three methods of measuring fat distribution correlate well with regard to establishing risk for diabetes mellitus, hypertension, and cardiovascular disease.

Several epidemiological studies have suggested that it is obesity in the upper body or central distribution that carries the greater risk not only for diabetes but for hypertension, coronary vascular disease, and stroke (27–30). Lower-body obesity is much less risky. This may explain why men, who proportionately are not as obese as women, have a higher mortality index related to obesity. They deposit their excess fat predominantly in an area that leads to more health risk.

The simplistic explanation for this phenomenon is that the differential fat distribution is related to circulating sex hormones and that higher androgen levels stimulate fat tissue to grow in the upper body or abdominal area, and lower androgens allow fat tissue to grow below the waist. Despite much work conducted on the binding of sex hormones to fat tissue in different body regions, it has not been possible to identify receptors to the sex hormones on adipocytes.

Another possibility is that cortisol secretion is high in individuals who preferentially put on fat in the abdominal area (31). Certainly, it is clear that people with marked hypercortisolism, e.g., Cushing's disease or adrenal carcinomas, have central (upper-body) fat distribution. This is an evolving field that will be important in further defining the causative factors behind the differential growth of fat depots.

However, because it is well known that the risk of diabetes is much greater for those with central obesity (32–34), a tendency to store more fat in the trunk could explain part of the greater prevalence of diabetes in Blacks. In fact, it has been reported that Blacks tend to

store more fat centrally than peripherally (35–37). For instance, Kumanyika (23) reported that, in the NHANES II data, obese Black women have a relative excess of truncal fat compared with extremity fat, as manifested by greater differences in subscapular than triceps skin folds (23).

Feldman et al. (33) also found increased diabetes with increased truncal fat in Blacks. Bonham and Brock (38), in a study in which the subjects were separated into quartiles of BMI, found an increased incidence of diabetes associated with increased truncal fat in a self-report survey conducted by the 1976 National Health Interview Survey.

However, Ama et al. (39), studying fat distribution and adipose tissue metabolism in Black Africans and Canadian Whites who were matched for sex (male), age (young), and weight (lean), could find no difference in fat distribution and total subcutaneous fat between the two groups. Thus, more specific cross-sectional and longitudinal data on the accretion of excess fat in Blacks is needed.

The study by Ama et al. (39) found that epinephrine-stimulated lipolysis and lipoprotein lipase activities were higher in the suprailiac adipocytes of Blacks than Whites. This suggests a greater ease of mobilization of free fatty acids out of triglyceride stores in Blacks, which could lead to higher lipid levels and greater insulin resistance. However, such data will have to be described in obese Blacks before any conclusions can be drawn about different lipolytic sensitivities.

DIABETES

What are the demographic figures for diabetes when Blacks in the U.S. are compared to Whites? At all ages, Blacks have a higher risk of developing diabetes than Whites. Since 1900, there has been an increase in the prevalence and mortality rate of diabetes in American Blacks. Both the Black-White ratio and the ratio of Black females to Black males have shifted from <1 to >1 (2). The National Diabetes Data Group reported that Black men have a prevalence of diabetes that is 16% higher than that of White men. Black women have rates 50% higher than their White counterparts (2). In a study by Harris et al. (40), Blacks had consistently higher rates than Whites, and again this was more so for women.

In addition to higher incidence and prevalence figures, Blacks also seem to suffer more from the complications of the disease. For instance, severe visual impairment from diabetes is 40% higher in Blacks. The age-standardized prevalence of blindness secondary to diabetic retinopathy was more than twice as high in non-Whites than Whites, with Black women having a rate three times higher than Black men (2).

The mortality rate of Black Americans is more than double that of Whites. Data from the National Vital Statistics System show that, although Whites have a diabetes mortality rate of 8.4/100,000, Blacks have a rate of 21.3/100,000. Therefore, the increase in risk is 2.5-fold (23).

What is the cause of these alarming statistics? Obviously, as discussed above, obesity is an important determinant. However, obesity cannot explain all of it. O'Brien et al. (41), in a recent study of a cohort of Army veterans, found a higher prevalence of diabetes among Blacks that could not be explained by differences in obesity. It could also not be explained by education or income. Within each age-group, adiposity, and socioeconomic stratum, Blacks were more likely to have diabetes than Whites.

GENETICS

Thus, it is clear that other facts also play a role. The most obvious is genetics. There is strong epidemiological evidence that certain racial groups are more prone to diabetes than others. North American Indians, with Pima Indians as a prime example (42), are one such group. Polynesians (43) and Australian aborigines (44) are two other well-documented groups with high diabetes rates. This suggests that the gene pool can have a large influence on the prevalence of diabetes in a specific population group. There are two other factors that make this a reasonable assumption: *1*) although some racial groups have a high prevalence of diabetes, other groups do not, e.g., the Japanese; and 2) in certain groups, as admixture occurs, the prevalence of diabetes drops, e.g., the Australian aborigines who have married Whites.

There is every possibility that American Blacks, descended from African Blacks, could have a gene pool that makes them more susceptible to diabetes and also more severely affected with it once they have it. If such is true of Polynesians and Australian aborigines, why could it not occur in peoples of the Black race?

However, the data from Africa do not seem to support this. Although there is diabetes in Africa, its prevalence has generally been reported as lower than in American Blacks (2). However, in more urban areas, some have reported similar prevalences (2). The lower prevalence generally found in Africa has been attributed to several factors, i.e., younger age, more rural living, less access to health care, greater activity, and lower weight. More accurate data are urgently needed, particularly from the tribal areas of West Africa, where American Blacks originated.

CONCLUSIONS

The problem of diabetes mellitus in the Black population of this country is disproportionately large and seems to be growing. In addition, obesity in this group is extraordinarily high, particularly among women. Whereas we have demographic de-

scriptive data on the scope of the problems, we require more knowledge of the causes of these two conditions and their interrelationships in Blacks so that adequate medical intervention and prevention programs can be instituted.

REFERENCES

1. Van Itallie TB: Health implications of overweight and obesity in the United States. *Ann Intern Med* 103:983–88, 1985
2. Roseman JM: Diabetes in black Americans. In *Diabetes in America. Diabetes Data Compiled 1984*. Harris MI, Hamman RF, Eds. National Diabetes Data Group. Washington, DC, U.S. Govt. Printing Office, 1985, p. 1–24 (DHHS publ. no. 85-1468)
3. West KM, Kalbfleisch JM: Influence of nutritional factors on prevalence of diabetes. *Diabetes* 20:99–108, 1971
4. *Report of the National Commission on Diabetes*. Vol. 3, pt. I. Washington, DC, U.S. Govt. Printing Office, 1976 (DHEW publ. no. 76-1021)
5. Kannel WB: *Health and Obesity*. Conn HL, DeFelice EA, Kuo P, Eds. New York, Raven, 1983, p. 1–19
6. Rimm AA, Werner L, Bernstein RA, Van Yserloo B: Disease and obesity in 73,532 women. *Obes Bariat Med* 1:77–84, 1972
7. Knowler WC, Pettit PJ, Savage PJ, Bennett PH: Diabetes incidence in Pima Indians: contributions of obesity and parental diabetes. *Am J Epidemiol* 113:144–56, 1981
8. Lew EA, Garfinkel L: Variations in mortality by weight among 750,000 men and women. *J Chronic Dis* 32:563–76, 1979
9. Westlund K, Nicolaysen R: Ten-year mortality and morbidity related to serum cholesterol. *Scand J Clin Lab Invest Suppl* 27:1–24, 1972
10. Larsson B, Bjorntorp P, Tibblin G: The health consequences of moderate obesity. *Int J Obes* 5:91–116, 1981
11. Medalie JH, Herman JB, Goldbourt U, Papier CM: Variations in the incidence of diabetes among 10,000 adult Israeli males and factors related to their development. In *Advances in Metabolic Disorders*. Vol. 9. Leveine R, Luft R, Eds. New York, Academic, 1978, p. 93–110
12. Van Itallie TB: Obesity: adverse effects on health and longevity. *Am J Clin Nutr* 32:2723–33, 1979
13. Burton BT, Foster, WR, Hirsch J, Van Itallie TB: Health implications of obesity: an NIH consensus conference. *Int J Obes* 9:155–70, 1985
14. Bouchard C: Genetics of body fat, energy expenditure, and adipose tissue metabolism. In *Recent Advances in Obesity Research*. Vol. 5. Berry EM, Blonheim SH, Eliahou HE, Eds. London, Libbey, 1987
15. Stunkard AJ, Sorenson TIA, Hanis C, Teasdale TW, Chakraborty R, Schull WJ, Schulsinger F: An adoption study of human obesity. *N Engl J Med* 314:193–201, 1986
16. Van Itallie TB: Bad news and good news about obesity. *N Engl J Med* 314:239–40, 1986
17. Segal KR, Pi-Sunyer FX: Exercise and obesity. *Med Clin North Am* 73:217–36, 1989
18. Pi-Sunyer FX: Obesity. In *Modern Nutrition in Health and Disease*. 7th ed. Shils ME, Young VR, Eds. Philadelphia, PA, Lea & Febiger, 1989, p. 795–816
19. *Promoting Healthy Weights: A Discussion Paper*. Health Services Promotion Branch, Health Welfare Canada, 1988, p. 7 (cat. no. H39-131/1988E)
20. *Ten-State Nutrition Survey 1968–1970*. Atlanta, GA, Centers for Disease Control, 1972 (DHEW publ. no. 72-8130)
21. Williamson DF, Kahn HS, Remington PL, Anda RF: The 10-year incidence of overweight and major weight gain in United States adults. *Arch Intern Med* 150:665–72, 1990
22. Khoury P, Morrison JA, Mellies MJ, Glueck CJ: Weight change since age 18 in 30- to 55-year old whites and blacks: associations with lipid values, lipoprotein levels, and blood pressure. *JAMA* 250:3179–87, 1983
23. Kumanyika S.: Obesity in black women. *Epidemiol Rev* 9:31–50, 1988
24. Stunkard AJ: From explanation to action in psychosomatic medicine: the case of obesity. *Psychosom Med* 37:195–236, 1975
25. Goldblatt PB, Moore ME, Stunkard AJ: Social factors in obesity. *JAMA* 192:1039–44, 1965
26. Silverstone JT, Gordon RP, Stunkard AJ: Social factors in obesity in London. *Practitioner* 202:682–88, 1969
27. Lapidus L, Bengtsson L, Larsson B, Pennert K, Rybo E, Sjostrom L: Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow-up of participants in the population study of women in Goteborg, Sweden. *Br Med J* 289:1261–63, 1984
28. Ducimetiere P, Richard J, Cambien F: The pattern of subcutaneous fat distribution in middle-aged men and the risk of coronary heart disease: the Paris prospective study. *Int J Obes* 10:229–40, 1956
29. Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, Adams PW: Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 54:254–60, 1982
30. Evans DJ, Hoffman RG, Kalkhoff RK, Kissebah AH: Relationship of body fat topography to insulin sensitivity and metabolic profiles in premenopausal women. *Metabolism* 33:68–75, 1984
31. Rebuffe-Scrive M, Nilsson A, Bronnegard M, Eldh J, Bjorntorp P: Regulation of steroid hormone effects on human adipose tissue metabolism and distribution. In *Obesity in Europe 88*. Bjorntorp P, Rossner S, Eds. London, Libbey, 1989, p. 219–22
32. Blair D, Habicht J-P, Sims EAH, Sylwester D, Abraham S: Evidence for an increased risk for hypertension with centrally located body fat and the effect of race and sex on this risk. *Am J Epidemiol* 119:526–40, 1984
33. Feldman R, Sender AJ, Siegelaub MS: Difference in diabetic and nondiabetic fat distribution patterns by skinfold measurements. *Diabetes* 18:478–86, 1969
34. Hartz AJ, Rupley DC Jr, Kalkhoff R, Rimm AA: Relationship of obesity to diabetes: influence of obesity level and body fat distribution. *Prev Med* 12:351–57, 1983
35. Davies CTM, Mbelwa D, Crokford GT, Weiner JS: Exercise tolerance and body composition of male and female Africans aged 18–30 years. *Hum Biol* 45:31–40, 1973
36. Garn SM, Clark DC: Trends in fatness and the origins of obesity. *Pediatrics* 57:443–56, 1976
37. Johnston FE, Hamill PV-V, Leneshow S: Skinfold thickness in a national probability sample of US males and females aged 6 through 17 years. *Am J Phys Anthropol* 40:321–24, 1974
38. Bonham GS, Brock DB: The relationship of diabetes with race, sex, and obesity. *Am J Clin Nutr* 41:776–83, 1985
39. Ama PFM, Poehlman ET, Simoneau JA, Boulay MR, Theriault G, Tremblay A, Bouchard C: Fat distribution and

adipose tissue metabolism in non-obese male black African and Caucasian subjects. *Int J Obes* 10:503–10, 1986
40. Harris MI, Hadden WC, Knowler WC, Bennett PH: Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in U.S. population aged 20–74 yr. *Diabetes* 36:523–34, 1987
41. O'Brien TR, Flanders WD, Decoufle P, Boyle CA, DeStefano F, Teutsch S: Are racial differences in the prevalence of diabetes in adults explained by differences in obesity? *JAMA* 262:1485–88, 1989
42. Bennett PH, Knowler WC: Increasing prevalence of diabetes in the Pima (American) Indians over a ten year period. In *Diabetes*. Waldhausl WK, Ed. Amsterdam, Excerpta Med., 1979, p. 507
43. Zimmet P, King H, Taylor R, Raper LR, Balkau B, Borger J, Heriot W, Thoma K: The high prevalence of diabetes mellitus, impaired glucose tolerance and diabetic retinopathy in Nauru—the 1982 survey. *Diabetes Res* 1:13–18, 1984
44. Wise PH, Edwards FM, Craig RT, Evans B, Murchland JB, Sutherland B, Thomas DW: Diabetes and associated variables in the South Australian aboriginal. *Aust NZ J Med* 6:191–96, 1976