Standard definitions of overweight and central adiposity for determining diabetes risk in Japanese Americans[1–3]

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ABSTRACT

Background: Despite having lower average body mass indexes (BMIs) than do whites, Asians are at high risk of type 2 diabetes, possibly because of their greater central adiposity. The criteria for identifying individuals at risk of obesity-related conditions are usually not population specific.

Objective: Our goal was to determine whether the National Heart, Lung, and Blood Institute (NHLBI) overweight and obesity guidelines are useful for identifying diabetes risk in Japanese Americans.

Design: This was a prospective, cohort study of 466 nondiabetic Japanese Americans [age: 52.2 ± 0.6 y; BMI (in kg/m^2): 24.1 ± 0.2; $\bar{x}$ ± SEM]. Diabetes status at a 5-y follow-up visit was assessed with an oral-glucose-tolerance test.

Results: Among 240 subjects aged ≤55 y, incident diabetes was strongly associated with overweight (BMI ≥ 25) at baseline [relative risk (RR): 22.4; 95% CI: 2.7, 183; adjusted for age, sex, smoking, and family history] and weight gain of >10 kg since the age of 20 y (adjusted RR: 4.5; 95% CI: 1.4, 14.5). NHLBI definitions of central obesity (waist circumference ≥ 88 cm for women and ≥ 102 cm for men) were unsuitable for this population because only 15 of 240 subjects met these criteria. A waist circumference greater than or equal to the third tertile was associated with diabetes (adjusted RR: 5.4; 95% CI: 1.7, 17.0). Among 226 subjects aged >55 y, incident diabetes was not associated with BMI, weight gain, or waist circumference.

Conclusions: NHLBI definitions are useful for identifying overweight Japanese Americans aged <55 y who are at high risk of diabetes. Although central adiposity is an important risk factor, the guidelines for waist circumference are insensitive predictors of diabetes risk in this population. *Am J Clin Nutr* 2001;74:101–7.

KEY WORDS Type 2 diabetes, Asian Americans, obesity, weight gain, body mass index, anthropometry, longitudinal study, central adiposity, National Heart, Lung, and Blood Institute guidelines

INTRODUCTION

The term *obesity* implies excess body fat, yet accurate measurement of body composition is not widely available in the clinical setting. Therefore, most clinical definitions of obesity rely on measures of body weight adjusted for height, such as the body mass index (BMI; in kg/m^2). National population averages were once widely used to determine reference ranges. However, as obesity becomes more prevalent worldwide, there is a recognized need for standard definitions that are applicable across countries and over time. An expert panel compiled by the National Heart, Lung, and Blood Institute (NHLBI) developed clinical guidelines that define overweight as a BMI of 25–29.9 and obesity as a BMI ≥30 (1, 2). These cutoffs are consistent with those used by the World Health Organization (3).

The risk of health problems, such as hypertension, dyslipidemia, coronary artery disease, and diabetes, increase incrementally above a BMI of 20–22 (2). The relation between increasing BMI and morbidity is observed across many racial and ethnic groups, although the absolute risk varies. Increased mortality is associated with BMIs ≥25 in whites and is the rationale for the current definition of overweight (2).

The clinical suitability of a single threshold definition of "normal" weight across ethnic groups remains unclear. High rates of obesity-related disorders have been noted in Asian populations, particularly in urban and westernized areas, despite the low average BMI of these populations relative to white populations (4–11). Variation in body fat distribution may account for some of this difference in risk, in that Asians may be more prone to central adiposity than are whites (12–14). The NHLBI guidelines include waist circumference cutoffs to identify high-risk individuals with central obesity, but the cutoffs are based on white populations (2, 15, 16) and may be inappropriate for Asians (11). Adulthood weight gain is another measure of excess adiposity that is associated with morbidity. Avoiding weight gain after reaching adult height was proposed as an appropriate health goal

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TABLE 1
Characteristics of the study subjects at baseline[1]

Baseline characteristic	Diabetes status at follow-up: Diabetic (n = 49)	Nondiabetic (n = 417)	Total (n = 466)	P[1]
Age (y)	58.8 ± 1.6[2]	51.4 ± 0.6	52.2 ± 0.6	<0.001
Male [n (%)]	23 (46.9)	221 (53.0)	244 (52.4)	0.422
Waist circumference (cm)				
Men[3]	92.5 ± 2.0	87.6 ± 0.5	88.0 ± 0.5	0.005
Women[4]	78.1 ± 1.3	75.2 ± 0.6	75.5 ± 0.6	0.111
BMI (kg/m^2)	24.9 ± 0.5	24.0 ± 0.2	24.1 ± 0.2	0.068
Reported weight at age 20 y (kg)	55.6 ± 1.5	57.0 ± 0.5	56.9 ± 0.5	0.357
Baseline weight (kg)	64.2 ± 2.1	63.1 ± 0.6	63.2 ± 0.6	0.562
Weight change				
(kg)	8.5 ± 1.1	6.1 ± 0.3	6.4 ± 0.3	0.017
(%)	15.5 ± 2.0	11.0 ± 0.6	11.5 ± 0.6	0.014
Smoking history [n (%)]				
Never smoked	22 (44.9)	117 (28.1)	139 (29.8)	0.015
Quit > 10 y ago	17 (34.7)	187 (44.8)	204 (43.8)	—
Quit < 10 y ago	5 (10.2)	52 (12.5)	57 (12.2)	—
Current smoker	5 (10.2)	61 (14.6)	66 (14.2)	0.401
Family history of diabetes [n (%)]	28 (57.1)	144 (34.5)	172 (36.9)	0.002

[1] Comparisons do not account for variation with age in the association between adiposity measures (BMI, weight change, and waist circumference) and diabetes risk. *See* text, Figure 1, and Tables 2–5 for details.
[2] $\bar{x}$ ± SEM.
[3] Data missing for 2 men.
[4] Data missing for 7 women.

for individuals (17), yet data on the health consequences of weight gain in Asians are sparse. To determine the applicability of current reference ranges for overweight and central obesity to a high-risk Asian population, we studied the relation between BMI, waist circumference, and weight gain since the age of 20 y and the risk of developing type 2 diabetes over the course of 5 y in Japanese Americans.

SUBJECTS AND METHODS

Subjects

Study subjects were second-generation (*Nisei*) and third-generation (*Sansei*) volunteers in the Japanese American Community Diabetes Study. Details on the recruitment and comparison of Nisei participants with nonparticipants residing in King County, Washington, were previously described (18). Subjects with known diabetes, or whose plasma glucose was ≥7.0 mmol/L (126 mg/dL) after a 10-h fast or ≥11.1 mmol/L (200 mg/dL) 2 h after a 75-g oral-glucose-tolerance test at baseline were excluded. This study was approved by the University of Washington Institutional Review Board, and all participants provided written, informed consent.

Measurements

Subjects were evaluated in the Clinical Research Center at the University of Washington at baseline and after 5 or 6 y of follow-up. A structured interview was used to obtain information about weight at the age of 20 y, medication use, smoking, and family history of diabetes. Subjects who reported a parent or sibling with diabetes were considered to have a positive family history of diabetes. Height and weight were measured while the subjects wore light clothing and no shoes. Waist circumference was measured at the level of the umbilicus in men and at the natural waistline (minimal waist) in women. Weight change was calculated as baseline weight minus weight at the age of 20 y. Percentage weight change was calculated as weight change divided by weight at the age of 20 y. BMI was calculated as weight divided by height squared (kg/m^2). Plasma glucose was assayed by an automated glucose oxidase method. Subjects were considered to have diabetes at follow-up if they were taking medication for diabetes or if their plasma glucose was ≥7.0 mmol/L (126 mg/dL) after a 10-h fast or ≥11.1 mmol/L (200 mg/dL) 2 h after a 75-g oral-glucose-tolerance test (19).

Statistical analysis

All means are presented as means ± SEMs. Baseline variables were compared between groups by using Student's t test or the chi-square test, except for age, which was analyzed by using Wilcoxon's rank-sum test because of its bimodal distribution. Sex-specific tertiles for waist circumference were calculated by using data pooled across age groups. Logistic regression models were used to estimate the relative odds of developing diabetes associated with BMI, waist circumference, or weight change, with adjustment for age, sex, smoking (ever versus never), and family history of diabetes. Given the infrequent occurrence of diabetes, the relative odds approximates the relative risk (RR). The likelihood ratio test was used to determine the statistical significance of variables and interaction terms in the logistic regression models. An age × BMI interaction with diabetes risk was observed (P = 0.004), so the results were stratified by age to simplify their interpretation. Statistics were calculated by using INTERCOOLED STATA software (version 5.0 for WINDOWS 95; Stata Corporation, College Station, TX).

RESULTS

Of the 466 subjects, 49 were diabetic at the 5-y follow-up. The baseline characteristics of the study subjects are shown in **Table 1**. Subjects ranged in age from 34 to 75 y. About one-half of the

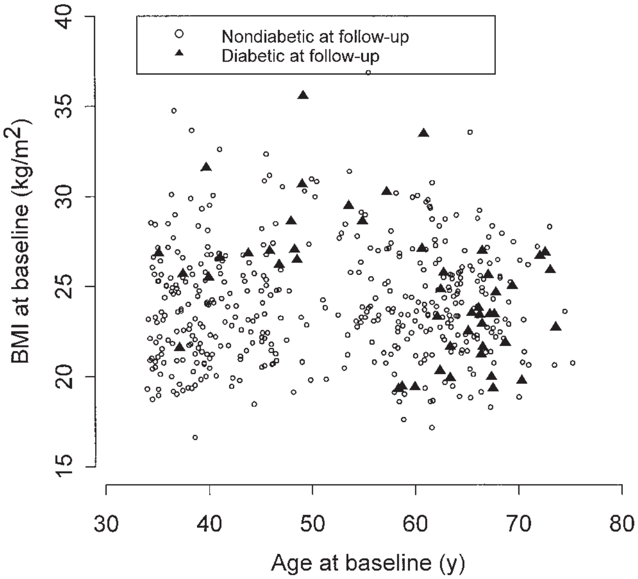

FIGURE 1. Scatter plot of baseline BMI versus age by diabetes status at the 5-y follow-up (*n* = 417 nondiabetic and 49 diabetic subjects).

subjects were male. The subjects' average BMI was 24.1 (range: 16.6–36.9). Weight change since the age of 20 y averaged 6.4 kg (11.5% gain), and the average weight gain was 2.4 kg (4.5%) higher in those who developed diabetes than in those who remained nondiabetic. About 30% of the subjects were lifelong nonsmokers, and 14.2% were actively smoking cigarettes at baseline. Most former smokers had quit smoking ≥10 y before baseline. Of those who developed diabetes, 57.1% reported a positive family history, compared with 34.5% of those who remained nondiabetic.

Younger subjects who developed diabetes tended to be above the median for BMI at baseline (23.8), whereas the BMIs of older subjects who developed diabetes were well dispersed throughout the BMI range (**Figure 1**). Scatter plots of baseline weight gain (kg) or percentage weight change versus age by 5-y diabetes status showed a similar pattern (data not shown). In a logit model of incident diabetes, BMI, and age, the coefficient of the first-order multiplicative interaction term between baseline BMI and age was significantly less than zero (coefficient: −0.013, $P = 0.004$). This result, together with the data shown in Figure 1, suggests that the association between BMI and incident diabetes was significantly greater with younger age.

Subjects aged ≤55 y who developed diabetes had higher baseline BMIs, weight gains, and waist circumferences than did subjects who remained nondiabetic, although the statistical power for detecting differences among younger women was poor (**Table 2**). Incident diabetes was not associated with baseline BMI, weight change since the age of 20 y, or waist circumference among men or women aged >55 y. Most of the younger subjects who developed diabetes were men, whereas most of the older subjects who developed diabetes were women. There were no significant interactions between sex and adiposity variables in modeling diabetes risk by logistic regression.

The proportion of subjects who developed diabetes by category of BMI and weight change is shown in **Table 3**. Most subjects were normal-weight according to NHLBI guidelines [144 of 240 (60%) of those aged ≤55 y and 147 of 226 (65%) of those aged >55 y]. Only 16 of 240 younger (6.7%) and 7 of 226 older (3.1%) subjects were obese. More than one-half of subjects who gained 5–10 kg since the age of 20 y were within the normal range of BMI at baseline, whereas only 14 of 68 (20.6%) younger and 15 of 58 (25.9%) older subjects who gained >10 kg since the age of 20 y had a normal BMI. Diabetes incidence increased incrementally with both weight gain since the age of 20 y and BMI at baseline among younger, but not older, subjects.

Substituting percentage weight change for absolute weight

TABLE 2
Adiposity variables at baseline by diabetes status at 5-y follow-up, stratified by age and sex

	Age ≤55 y		Age >55 y	
Baseline characteristic	Diabetic	Nondiabetic	Diabetic	Nondiabetic
No. of subjects [*n* (row %)]	16 (6.7)	224 (93.3)	33 (14.6)	193 (85.4)
Men	12 (9.3)	117 (90.7)	11 (9.6)	104 (90.4)
Women	4 (3.6)	107 (96.4)	22 (19.8)	89 (80.2)
Waist circumference (cm)				
Men[1]	94.4 ± 2.2[2]	87.5 ± 0.7[3]	88.3 ± 3.1	87.7 ± 0.7
Women[4]	80.7 ± 3.2	73.9 ± 0.8	77.6 1.4	76.7 ± 0.9
BMI (kg/m^2)	27.8 ± 0.8	24.0 ± 0.2[3]	23.6 ± 0.6	24.1 ± 0.2
Men	28.7 ± 0.8	25.1 ± 0.3[3]	24.9 ± 1.3	25.0 ± 0.3
Women	25.1 ± 1.2	22.8 ± 0.3	22.9 ± 0.5	23.0 ± 0.3
Weight change				
(kg)	13.5 ± 1.4	6.2 ± 0.4[3]	6.1 ± 1.3	6.0 ± 0.5
Men	14.3 ± 1.7	7.1 ± 0.6[3]	7.3 ± 2.9	7.3 ± 0.7
Women	10.9 ± 2.2	5.2 ± 0.6	5.6 ± 1.3	4.6 ± 0.7
(%)	22.0 ± 2.5	10.9 ± 0.7[3]	12.3 ± 2.5	11.1 ± 0.9
Men	21.8 ± 3.0	11.3 ± 0.9[3]	11.8 ± 4.7	12.2 ± 1.1
Women	22.5 ± 5.3	10.5 ± 1.2	12.5 ± 3.1	9.7 ± 1.4

[1] Data missing for 2 men ≤55 y old.
[2] $\bar{x}$ ± SEM.
[3] Significantly different from diabetic, $P < 0.001$ (Student's *t* test).
[4] Data missing for 7 women, 6 of whom were >55 y old.

TABLE 3
Proportion of subjects who developed diabetes by 5 year of follow-up by baseline BMI and weight change since the age of 20 y[1]

Baseline BMI (in kg/m^2)[3]	Weight change since the age of 20 y at baseline[2] <5 kg	5–10 kg	>10 kg	Total
Age ≤55 y				
<25 (normal weight)	0/88	1/42	0/14	1/144 (0.7)
25–30 (overweight)	0/8	3/29	9/43	12/80 (15.0)
≥30 (obese)	0/2	1/3	2/11	3/16 (18.8)
Total	0/98 (0)	5/74 (6.8)	11/68 (16.2)	16/240 (6.7)
Age >55 y				
<25 (normal weight)	16/88	4/44	3/15	23/147 (15.6)
25–30 (overweight)	0/13	5/23	3/36	8/72 (11.1)
≥30 (obese)	0/0	0/0	2/7	2/7 (28.6)
Total	16/101 (15.8)	9/67 (13.4)	8/58 (13.8)	33/226 (14.6)

[1] *n* with diabetes at follow-up/total *n* (%).
[2] Chi-square test of baseline weight-change category by diabetes status at follow-up: $P < 0.001$ for age ≤55 y; $P = 0.892$ for age >55 y.
[3] Chi-square test of baseline BMI category by diabetes status at follow-up: $P < 0.001$ for age ≤55 y; $P = 0.381$ for age >55 y.

change produced similar results, so these data are not presented in tabular form. Of the 240 younger subjects, diabetes incidence was 0% (0/67) among those with a weight gain of <5% of body weight, 5.8% (5/86) among those with a weight gain of 5–15%, and 12.6% (11/87) among those with a weight gain of >15%. Of the 144 younger subjects with a normal BMI, the single individual who developed diabetes was in the 5–15% weight gain category. Of the 12 overweight subjects aged ≤55 y who developed diabetes, 3 gained 5–15% and 9 gained >15% of their body weight since the age of 20 y. Among the 226 older subjects, diabetes incidence was 11.8% (9/76) among those with a weight gain of <5% of body weight, 15.6% (10/64) among those with a weight gain of 5–15%, and 16.3% (14/86) among those with a weight gain of >15%.

The proportions of subjects who developed diabetes by waist circumference tertile and BMI at baseline are shown in **Table 4**. Waist circumference data were missing for 9 subjects. Diabetes incidence increased incrementally with increasing waist circumference among younger, but not older, subjects. Only 15 of 237 younger and 13 of 220 older subjects met the NHLBI guidelines for increased waist circumference.

Among subjects ≤55 y at baseline, the RR for developing diabetes associated with a BMI ≥ 25 was 26.5 (95% CI: 3.4, 204) compared with normal-weight subjects (**Table 5**). Results were similar after adjustment for age, sex, smoking, and family history of diabetes. The mean BMI was 27.6 ± 0.2 for the group with a BMI ≥ 25 and 22.0 ± 0.1 for the normal-weight group. Because only 16 of 96 subjects with a BMI ≥ 25 were obese, results were similar for overweight subjects (BMI: 25–29.9; obese subjects excluded) compared with normal-weight subjects (adjusted RR: 23.5; 95% CI: 2.8, 196.2; $P = 0.004$; data not presented in table).

Weight change >10 kg was also a significant predictor of diabetes risk among younger subjects (adjusted RR: 4.5; 95% CI: 1.4, 14.5). Results were similar for percentage weight change >15% among subjects aged ≤55 y (RR: 4.3; 95% CI: 1.4, 12.8; $P = 0.0009$), although results were of borderline significance after adjustment for covariates (adjusted RR: 3.1; 95% CI: 1.0, 9.9; $P = 0.054$; data not presented in table). Younger subjects in the top sex-specific tertile for waist circumference were also at increased risk of diabetes (Table 5). Sample sizes were insufficient to analyze NHLBI criteria for increased waist circumference in younger subjects because only 15 of 240 younger

TABLE 4
Proportion of subjects who developed diabetes by 5 year of follow-up by baseline BMI and waist circumference[1]

Baseline BMI (in kg/m^2)[3]	Waist circumference tertile at baseline[2] 1	2	3	Total
Age ≤55 y				
<25 (normal weight)	0/95	1/40	0/6	1/141 (0.7)
25–30 (overweight)	0/2	4/33	8/45	12/80 (15.0)
≥30 (obese)	0/0	0/0	3/16	3/16 (18.8)
Total	0/97 (0)	5/73 (6.8)	11/67 (16.4)	16/237 (6.8)
Age >55 y				
<25 (normal weight)	7/70	12/57	2/14	21/141 (14.9)
25–30 (overweight)	1/4	1/26	6/42	8/72 (11.1)
≥30 (obese)	0/0	0/0	2/7	2/7 (28.6)
Total	8/74 (10.8)	13/83 (15.7)	10/63 (15.9)	31/220 (14.1)

[1] *n* with diabetes at follow-up/total *n* (%).
[2] Tertiles of waist circumference were as follows: <85.5, 85.5–91.5, and >91.5 cm for men and <71.6, 71.6–80.2, and >80.2 cm for women. Waist circumference was missing for 2 men and 7 women. Fifteen subjects aged ≤55 y (3 with diabetes) and 13 aged >55 y (2 with diabetes) had waist circumferences exceeding the National Heart, Lung, and Blood Institute guidelines (102 cm for men and 88 cm for women). Chi-square test of baseline waist circumference tertile by diabetes status at follow-up: $P < 0.001$ for age ≤55 y; $P = 0.609$ for age >55 y.
[3] Chi-square test of baseline BMI category by diabetes status at follow-up: $P < 0.001$ for age ≤55 y; $P = 0.381$ for age > 55 y.

TABLE 5
Five-year relative risk (and 95% CI) of diabetes associated with baseline BMI, weight change, and waist circumference in Japanese Americans

	BMI, unadjusted	BMI, adjusted for covariates	Weight change, unadjusted	Weight change, adjusted for covariates	Waist circumference, unadjusted	Waist circumference, adjusted for covariates
Age ≤55 y						
BMI ≥ 25[1]	26.5 (3.4, 204)[2]	22.4 (2.7, 183)[2]	—	—	—	—
Weight gain > 10 kg	—	—	6.4 (2.1, 19.3)[2]	4.5 (1.4, 14.5)[3]	—	—
Waist ≥ 3rd tertile[4]	—	—	—	—	6.5 (2.2, 19.5)[2]	5.4 (1.7, 17.0)[2]
Age (y)	—	1.1 (1.0, 1.2)[3]	—	1.1 (1.0, 1.2)	—	1.1 (1.0, 1.2)
Male sex	—	1.3 (0.4, 4.7)	—	2.4 (0.7, 8.3)	—	2.6 (0.8, 9.0)
Smoking (ever)	—	4.1 (0.5, 36.9)	—	2.9 (0.3, 25.6)	—	4.3 (0.5, 39.6)
Family history[5]	—	1.6 (0.5, 5.1)	—	2.1 (0.7, 6.3)	—	2.0 (0.7, 6.2)
Age > 55 y						
BMI ≥ 25[1]	0.8 (0.4, 1.7)	0.9 (0.4, 2.1)	—	—	—	—
Weight gain > 10 kg	—	—	0.9 (0.4, 2.2)	1.2 (0.5, 3.1)	—	—
Waist ≥ 3rd tertile[4]	—	—	—	—	1.2 (0.5, 2.8)	1.2 (0.5, 2.8)
Age (y)	—	1.1 (1.0, 1.3)[2]	—	1.1 (1.0, 1.3)[2]	—	1.1 (1.0, 1.2)[2]
Male sex	—	0.6 (0.2, 1.5)	—	0.5 (0.2, 1.4)	—	0.6 (0.2, 1.6)
Smoking (ever)	—	0.6 (0.3, 1.6)	—	0.6 (0.2, 1.6)	—	0.6 (0.2, 1.5)
Family history[5]	—	2.3 (1.1, 5.1)[3]	—	2.3 (1.1, 5.1)[3]	—	2.1 (1.0, 4.7)

[1] Mean (±SEM) BMI for those with a BMI <25: 22.0 ± 0.1 for those aged ≤55 y and 22.2 ± 0.1 for those aged >55 y. Mean (±SEM) BMI for those with a BMI ≥25: 27.6 ± 0.2 for those aged ≤55 y and 27.3 ± 0.2 for those aged >55 y.

[2] $P < 0.01$.

[3] $P < 0.05$.

[4] Third tertile of waist circumference: >91.5 cm for men and >80.2 cm for women. Waist circumference data were missing for 2 men and 7 women. Fifteen subjects aged ≤55 y (3 with diabetes) and 13 aged >55 y (2 with diabetes) had waist circumferences exceeding the National Heart, Lung, and Blood Institute guidelines (102 cm for men and 88 cm for women).

[5] Family history of diabetes in a parent or sibling.

subjects met these criteria.

In the older age group, BMI, weight gain, and waist circumference did not predict diabetes risk, although age and family history of diabetes were associated with increased risk (Table 5). Even when analyzed as continuous variables, however, BMI, weight change, and waist circumference were not significantly associated with incident diabetes (data not shown).

DISCUSSION

This study confirms that despite having a relatively low average BMI at baseline, a substantial proportion (10.5%) of Japanese Americans develop diabetes during 5 y of follow-up. This incidence rate is similar to that found by Hara et al (20) in a different population of Japanese Americans. We found that the association between diabetes risk and elevated BMI was strongest among adults aged ≤55 y, which is consistent with prior studies (21). Among younger Japanese Americans, a BMI ≥ 25 was strongly associated with diabetes incidence, with 15.6% of overweight compared with 0.7% of normal-weight (BMI < 25) subjects meeting the criteria for diabetes at follow-up. This was particularly striking because the mean BMI of the overweight group was only 27.6. Even when the 16.7% of overweight, younger subjects who met the criteria for obesity were excluded from this group, diabetes incidence remained significantly higher than in those with normal weights. This suggests that moderate increases above normal weight may substantially raise diabetes risk in younger Japanese Americans.

Our study does not explain why overweight is such a strong diabetes risk factor for younger Japanese Americans. There is some evidence that the relation between percentage body fat and BMI varies by ethnicity (22, 23). In these studies, Asians had a higher degree of adiposity for a given BMI than did whites. Another likely possibility is that Japanese Americans may be genetically predisposed to an unfavorable fat distribution pattern, namely, increased intraabdominal fat (24, 25). In our study, central adiposity measured as waist circumference was associated with diabetes risk in younger Japanese Americans, but only 6.3% of subjects in this group exceeded NHLBI sex-specific cutoffs. Recently, experts from several Asian and Pacific countries recommended lower thresholds for BMI and waist circumference for Asians than for whites (overweight, BMI ≥ 23; obese, BMI ≥ 25; high-risk waist circumference, ≥ 90 cm for men and ≥ 80 cm for women) (26). These proposed cutoffs for waist circumference are similar to the upper tertile cutoffs associated with increased diabetes risk in our study.

Among older Japanese Americans, we found no association between increased BMI or waist circumference and diabetes risk, possibly because subjects susceptible on the basis of increased overall adiposity already had diabetes at baseline. We previously reported that among older (Nisei) Japanese American men, intraabdominal fat was a significant predictor of diabetes risk at the 30-mo follow-up, whereas BMI did not predict diabetes (24). Thus, it appears that waist circumference and BMI are poor markers of intraabdominal fat in this older population. One possible explanation for these findings is that among older persons, increased intraabdominal fat is primarily associated with aging, whereas among younger persons, increased intraabdominal fat is due to excess adiposity (as measured by BMI and waist circumference). Thus, it may be that individuals with a propensity toward intraabdominal fat deposition who become overweight are those at highest risk of developing diabetes at a younger age. If an individual has not developed diabetes by the time of maximum adulthood weight gain (typically by the fifth decade of life) (27), then his or her major risk factor becomes age-related fat redistribution.

Our study confirms prior reports that adulthood weight gain is associated with an increased risk of diabetes (28–30). In these studies of white and African American populations, weight gain of >5 kg was associated with significant risk. We found no incident cases among younger adults with weight gains of <5 kg (probably because of the small sample size), but we found a significantly increased diabetes risk with a weight gain >10 kg. There are several advantages to using weight change as an adjunct to BMI for assessing disease risk in adults. From a clinical standpoint, weight on reaching adult height can be easily monitored and requires no calculations or tables to interpret. Focusing on adulthood weight change is a reasonable approach to both preventing and managing obesity. Because substantial weight loss is difficult to achieve, minimizing weight gain can also be expected to result in greater absolute risk reduction than can delaying interventions until after individuals become overweight. Larger studies may help to clarify whether adulthood weight gain cutoffs provide a practical alternative to population-specific BMI or waist circumference reference ranges for risk assessment of individuals in the clinical setting.

We found that a family history of diabetes predicted diabetes risk in older, but not in younger, Japanese Americans. Previously reported cross-sectional data from this population showed that diabetes was associated with increased BMI, waist circumference, and intraabdominal fat only among those without a family history of diabetes (31). The current study suggests that a family history of diabetes is associated with age-related derangements of glucose metabolism in older Japanese Americans, possibly as a result of a genetic predisposition to impaired islet β cell function with age. However, it appears that persons predisposed to adiposity-related diabetes, presumably via insulin resistance (32), cannot be identified on the basis of a family history of diabetes in this population.

There are several limitations to our study. We used self-reported weight at the age of 20 y to determine adulthood weight gain at baseline. Recalled weight is undoubtedly less precise than measured weight, and error may be accentuated in older subjects. However, BMI derived from adolescent height and weight as recalled by elderly subjects was shown to correlate well with measured BMI ($r = 0.75$) (33). Additionally, self-reported weight change was associated with morbidity in several other studies (28, 29, 34, 35). Another potential limitation of our study is the use of volunteers rather than a population-based sample. However, it is unlikely that our conclusions were substantially influenced by selection bias because all subjects were determined to be nondiabetic at baseline and adjustment for family history of diabetes did not significantly alter the results. Finally, our sample size was fairly small, particularly after stratification by age. It remains to be seen whether these results are confirmed by larger prospective studies and in other Asian populations.

In summary, Japanese Americans aged ≤55 y who are only moderately overweight (mean BMI of 27.6) are at high risk of developing diabetes. Although Japanese Americans may be especially prone to central obesity, the NHLBI guidelines for waist circumference were insensitive markers of diabetes risk in this population. These findings illustrate the potential limitations of applying uniform BMI and waist circumference cutoffs to assess the health risks of individuals in a diverse patient population. Additional research is needed to examine the feasibility of incorporating ethnic-specific cutoffs or dynamic measures (such as weight gain and waist circumference increase) into clinical guidelines.

REFERENCES

1. Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Arch Intern Med 1998;158:1855–67.
2. NHLBI Obesity Education Initiative Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Obes Res 1998;6:51S–209S.
3. WHO Expert Committee. Physical status: the use and interpretation of anthropometry. World Health Organ Tech Rep Ser 1995;854:1–452.
4. Wang SL, Pan WH, Hwu CM, et al. Incidence of NIDDM and the effects of gender, obesity and hyperinsulinaemia in Taiwan. Diabetologia 1997;40:1431–8.
5. Yamamoto A, Horibe H, Mabuchi H, et al. Analysis of serum lipid levels in Japanese men and women according to body mass index. Increase in risk of atherosclerosis in postmenopausal women. Research Group on Serum Lipid Survey 1990 in Japan. Atherosclerosis 1999;143:55–73.
6. He J, Klag MJ, Whelton PK, Chen JY, Qian MC, He GQ. Body mass and blood pressure in a lean population in southwestern China. Am J Epidemiol 1994;139:380–9.
7. Klatsky AL, Armstrong MA. Cardiovascular risk factors among Asian Americans living in northern California. Am J Public Health 1991;81:1423–8.
8. Masaki KH, Curb JD, Chiu D, Petrovitch H, Rodriguez BL. Association of body mass index with blood pressure in elderly Japanese American men. The Honolulu Heart Program. Hypertension 1997;29:673–7.
9. Davies MJ, Ammari F, Sherriff C, Burden ML, Gujral J, Burden AC. Screening for type 2 diabetes mellitus in the UK Indo-Asian population. Diabet Med 1999;16:131–7.
10. Whitty CJ, Brunner EJ, Shipley MJ, Hemingway H, Marmot MG. Differences in biological risk factors for cardiovascular disease between three ethnic groups in the Whitehall II study. Atherosclerosis 1999;142:279–86.
11. Ko GT, Chan JC, Cockram CS, Woo J. Prediction of hypertension, diabetes, dyslipidaemia or albuminuria using simple anthropometric indexes in Hong Kong Chinese. Int J Obes Relat Metab Disord 1999;23:1136–42.
12. Ramachandran A, Snehalatha C, Viswanathan V, Viswanathan M, Haffner SM. Risk of noninsulin dependent diabetes mellitus conferred by obesity and central adiposity in different ethnic groups: a comparative analysis between Asian Indians, Mexican Americans and whites. Diabetes Res Clin Pract 1997;36:121–5.
13. Potts J, Simmons D. Sex and ethnic group differences in fat distribution in young United Kingdom South Asians and Europids. J Clin Epidemiol 1994;47:837–41.
14. Unwin N, Harland J, White M, et al. Body mass index, waist circumference, waist-hip ratio, and glucose intolerance in Chinese and Europid adults in Newcastle, UK. J Epidemiol Community Health 1997;51:160–6.
15. Lean ME, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management. BMJ 1995;311:158–61.
16. Han TS, van Leer EM, Seidell JC, Lean ME. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. BMJ 1995;311:1401–5.
17. Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. N Engl J Med 1999;341:427–34.
18. Fujimoto WY, Leonetti DL, Kinyoun JL, et al. Prevalence of diabetes mellitus and impaired glucose tolerance among second-generation Japanese-American men. Diabetes 1987;36:721–9.
19. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, American Diabetes Association. Report of the Expert Committee on the Diagnosis and Classification of Diabetes

Mellitus. Diabetes Care 1997;20:1183–97.
20. Hara H, Egusa G, Yamakido M. Incidence of non-insulin-dependent diabetes mellitus and its risk factors in Japanese-Americans living in Hawaii and Los Angeles. Diabet Med 1996;13:S133–42.
21. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA 1999;282:1523–9.
22. Novotny R, Davis J, Ross P, Wasnich R. Adiposity and blood pressure in a multiethnic population of women in Hawaii. Ethn Health 1998;3:167–73.
23. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis among different ethnic groups. Int J Obes Relat Metab Disord 1998;22:1164–71.
24. Bergstrom RW, Newell-Morris LL, Leonetti DL, Shuman WP, Wahl PW, Fujimoto WY. Association of elevated fasting C-peptide level and increased intra-abdominal fat distribution with development of NIDDM in Japanese-American men. Diabetes 1990;39:104–11.
25. Fujimoto WY. Overview of non-insulin-dependent diabetes mellitus (NIDDM) in different population groups. Diabet Med 1996;13:S7–10.
26. Steering Committee of the Western Pacific Region of the World Health Organization, the International Association for the Study of Obesity, and the International Obesity Task Force. The Asia-Pacific perspective: redefining obesity and its treatment. Melbourne, Australia: Health Communications Australia Pty Limited, 2000. World Wide Web: http://www.diabetes.com.au/research/report_obesity.htm (accessed 11 April 2001).
27. Grinker JA, Tucker K, Vokonas PS, Rush D. Body habitus changes among adult males from the normative aging study: relations to aging, smoking history and alcohol intake. Obes Res 1995;3:435–46.
28. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med 1995;122:481–6.
29. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of US adults. Am J Epidemiol 1997;146:214–22.
30. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care 1994;17:961–9.
31. Fujimoto WY, Leonetti DL, Newell-Morris L, Shuman WP, Wahl PW. Relationship of absence or presence of a family history of diabetes to body weight and body fat distribution in type 2 diabetes. Int J Obes 1991;15:111–20.
32. Yoshinaga H, Kosaka K. Heterogeneous relationship of early insulin response and fasting insulin level with development of non-insulin-dependent diabetes mellitus in non-diabetic Japanese subjects with or without obesity. Diabetes Res Clin Pract 1999;44:129–36.
33. Must A, Willett WC, Dietz WH. Remote recall of childhood height, weight, and body build by elderly subjects. Am J Epidemiol 1993;138:56–64.
34. Brancati FL, Wang NY, Mead LA, Liang KY, Klag MJ. Body weight patterns from 20 to 49 years of age and subsequent risk for diabetes mellitus: the Johns Hopkins Precursors Study. Arch Intern Med 1999;159:957–63.
35. Rosengren A, Wedel H, Wilhelmsen L. Body weight and weight gain during adult life in men in relation to coronary heart disease