The effect of decision rules on the choice of a body mass index cutoff for obesity: examples from African American and white women[1–3]

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ABSTRACT

Background: Ethnic differences in the relation of body mass index (BMI; in kg/m^2) to morbidity and mortality have led investigators to question whether a single cutoff for obesity should be applied to all ethnic groups.

Objective: The effects of using 4 different outcomes and 3 different measures of effect as criteria for comparing BMI cutoffs were shown with the use of data from 45- to 64-y-old African American and white women.

Design: Data were from the Cancer Prevention Study I (CPS-I) and the Atherosclerosis Risk in Communities (ARIC) Study. The outcomes were mortality (9211 deaths), diabetes (757 cases), hypertension (1518 cases), and hypertriglyceridemia (1264 cases). The measures of effect were incidence rate, rate ratio, and rate difference. The BMI in African American women that was associated with a risk equivalent to that of white women with a BMI of 30 was estimated.

Results: There was no significant association between BMI and mortality in African American women. The BMI in African American women that was associated with a risk of diabetes equivalent to that of white women with a BMI of 30 was 28.0–34.5, depending on the measure of effect. For hypertension, the equivalent risk in African American women occurred at a BMI of <18–38, depending on the measure of effect. There was no BMI at which African American women had an incidence rate or rate ratio for hypertriglyceridemia that was as high as that of white women with a BMI of 30.

Conclusion: BMI cutoffs associated with equivalent risk across ethnic groups differ widely depending on the outcome and the risk estimate. *Am J Clin Nutr* 2002;75:986–92.

KEY WORDS Epidemiology, African American women, mortality, diabetes, hypertension, hypertriglyceridemia, body weight, body mass index, obesity, Cancer Prevention Study I, Atherosclerosis Risk in Communities Study

INTRODUCTION

Within the past 5 y, the National Institutes of Health (1) and the World Health Organization (2) have provided guidelines for the definition of overweight and obesity that use body mass index (BMI; in kg/m^2) as the criterion measure. BMI cutoffs of 25 for overweight and 30 for obesity have been promoted by both organizations, and these definitions have been rapidly adopted by many clinicians and researchers in the United States and Europe (3). In February 2000 a report was issued jointly by the Regional Office for the Western Pacific World Health Organization, the International Association for the Study of Obesity, and the International Obesity Task Force that addressed the definitions of overweight and obesity in Asian populations (4). The report states that "in Pacific Island populations e.g. Samoa, the recommended BMI standards should be higher than those recommended by WHO . . . , whereas in certain other Asian populations such as Chinese and Japanese it is likely that they should be lower."

Although no such statements were made in regard to other ethnic groups, many studies have shown that the risk of obesity in other ethnic groups, including African Americans (5), Mexican Americans (6), and Native Americans (7, 8), may differ from that in whites. The issue of whether different cutoffs should be used for different ethnic groups is a complex one. The World Health Organization report, the National Institutes of Health Evidence Report, and the more recent report that was focused on Asia all relied heavily on published results on the associations between BMI and mortality and morbidity. The reports show results with several outcomes and various types of risk estimates. Yet, nowhere in these reports were the criteria for determining the BMI cutoffs for overweight or obesity explicitly listed, and a decision rule for determining whether different BMI cutoffs are needed in different ethnic groups was not stated.

The purpose of this study was to compare the results obtained when different decision rules were used to evaluate BMI cutoffs across ethnic groups. By way of illustration, we show analyses of data from African American and white women. We calculated the BMI value in African American women that was associated with a risk equivalent to that of white women with a BMI of 30, the currently accepted cutoff for obesity (3). Four different outcomes and 3 different measures of effect were compared.

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SUBJECTS AND METHODS

Cancer Prevention Study I

Data for analyses of mortality were from the Cancer Prevention Study I (CPS-I). In 1960 CPS-I baseline data were collected in 26 states from >1 million men and women aged ≥30 y (9). American Cancer Society volunteers recruited participants, who completed a questionnaire concerning their personal health and medical history. The subjects' ethnicity was determined by self-identification with the use of a checklist, and no attempts were made to assess admixture. The subjects' self-reported height (without shoes) and weight (in indoor clothing) were collected, and their vital status was determined annually from October 1960 through 1965 and thereafter in 1971 and 1972 and was confirmed by death certificates. The study was approved by the relevant institutional review boards.

We restricted this analysis to African American and white women who were between 45 and 64 y of age (n = 352 171). Participants who responded by proxy (n = 13 239), were missing data for pertinent variables (n = 33 643), or were pregnant at baseline (n = 69) were excluded. To avoid confounding by smoking, we excluded both current and former smokers (n = 106 496). To avoid confounding by preexisting illness, we excluded those who had involuntarily lost ≥4.5 kg (10 lb) body weight over the previous 2 y, those who died within the first year of follow-up, and those who reported heart disease, stroke, or cancer other than skin cancer (n = 61 107). The final sample included 193 135 white women and 3160 African American women.

Atherosclerosis Risk in Communities Study

The associations between BMI and incident diabetes, hypertension, and hypertriglyceridemia were examined by using data from the Atherosclerosis Risk in Communities (ARIC) Study, a prospective, multicenter investigation of atherosclerosis and cardiovascular disease (10). Between 1987 and 1989, 15 792 men and women aged 45–64 y were examined in 4 US communities. The cohort was reexamined in 3 additional clinic visits, with an average of 3 y between visits. The study protocol was approved by the institutional review committee for human protection at each of the 5 participating universities.

For these analyses, participants who were neither white nor African American were excluded and only women were studied (n = 8685). Ethnicity was determined by self-identification with the use of a checklist, and no attempts were made to assess admixture. Twenty-six African American women in the Minneapolis and Washington County field centers were excluded because their number was too small to permit modeling associations within the African American women in those centers. We also excluded cohort members who were not reexamined after the baseline visit (n = 570) and participants who were missing data for pertinent variables (n = 51) or who had values out of the quality-control range (n = 19).

Participants were classified as having diabetes if they had a glucose value ≥6.99 mmol/L (126 mg/dL) after fasting for ≥8 h, had one nonfasting glucose value ≥11.1 mmol/L (200 mg/dL), reported that a physician had told them they had diabetes, or reported taking medication for diabetes. Participants were classified as having hypertension if their systolic blood pressure was ≥140 mm Hg, their diastolic blood pressure was ≥90 mm Hg, or they reported having taken an antihypertensive drug in the past 2 wk. Finally, participants were classified as having hypertriglyceridemia if they had a fasting triacylglycerol concentration >2.26 mmol/L (200 mg/dL).

Participants were instructed to fast for ≥12 h before the clinic appointment and to bring all prescription and nonprescription drugs used in the 2 wk preceding the examination. Details of the laboratory methods (11, 12) and repeatability estimates (13, 14) are available elsewhere.

Height was measured to the nearest centimeter with the use of a metal rule attached to a wall and a standard triangular headboard. Weight was measured in pounds with the use of a beam balance while the subject wore a scrub suit and no shoes.

Statistical methods

Associations between BMI and incidence rates of mortality and morbidity were examined with the use of Poisson regressions (15). These analyses were performed with the use of the SAS procedure PROC GENMOD (16). We used the quadratic form of BMI with BMI centered to the ethnic-specific mean. On the basis of these models, we estimated the expected incidence rates for a range of BMI values. Incidence rates are expressed as the number of events (death, diabetes, hypertension, or hypertriglyceridemia) per 1000 person-years (the sum of the number of event-free years observed for each subject divided by 1000). Using expected incidence rates estimated from the Poisson regressions, we calculated rate ratios and rate differences by using a BMI of 21.0 as the reference. The risk associated with a BMI of 30 in white women was estimated, and the BMI value associated with the same level of risk in African American women was calculated. Here the term *risk* is used as a general term that includes the 3 measures of effect studied here, ie, incidence rate, rate ratio, and rate difference.

The baseline characteristics included in the models as covariates for analyses of mortality in the CPS-I were age, education, physical activity, and alcohol consumption. The confounding effects of smoking and preexisting illness on the association between BMI and mortality were handled by exclusions. For analyses of the associations between BMI and morbidity in the ARIC data set, we used smoking status (current, former, and never) and study center as covariates in addition to the covariates used in the CPS-I analysis. Because it was not our intention to adjust for social or economic factors that contribute to ethnic differences (17), separate models were run for each ethnic group.

RESULTS

The mean ages of the white and African American women studied in the CPS-I sample were 53.6 and 53.0 y, respectively (**Table 1**). The African American women were heavier and less well educated than were the white women. The African American women were more likely to report heavy physical activity and to abstain from alcoholic beverages than were the white women.

Baseline information is shown in **Table 2** for the women in the ARIC cohort after the standard exclusions but before exclusions for the specific conditions studied. The mean ages of the white and African American women (54.0 and 53.2 y, respectively) were similar to those of the women in the CPS-I cohort. The African American women had higher mean BMI values and less education than did the white women. The percentages of women who were current smokers were similar by ethnicity, but the African American women were more likely to have never smoked. A higher percentage of the African American women than of the white women reported low levels of physical activity,

TABLE 1
Baseline characteristics of the white and African American women in the analysis sample from the Cancer Prevention Study I cohort[1]

	White (n = 193 135)	African American (n = 3160)
Age (y)	53.6 ± 5.5[2]	53.0 ± 5.4
BMI (kg/m^2)	25.0 ± 4.2	28.0 ± 5.5
Education (%)		
Less than high school graduate	38.4	54.9
High school graduate	23.8	11.9
More than high school graduate	37.8	33.2
Physical activity (%)		
None	1.2	2.7
Slight	12.4	13.6
Moderate	76.7	66.5
Heavy	9.7	17.2
Alcohol consumption (%)		
None	81.2	86.7
1 drink/d	3.6	2.5
2 to <4 drinks/d	1.8	1.3
≥4 drinks/d	0.4	0.3
Occasional drink (<1 drink/d)	5.7	3.0
Irregular drink (moderate to heavy and sporadic)	7.5	6.3

[1] Data from reference 9.
[2] $\bar{x}$ ± SD.

and the African American women were more likely to abstain from alcoholic beverages than were the white women. Descriptive information on the women excluded from some analyses because they had diabetes, hypertension, or hypertriglyceridemia at baseline is also shown in Table 2. The data are displayed in this way to highlight the characteristics of these women.

Over the follow-up period there were 253 deaths among the African American women and 8958 deaths among the white women. The incidence rates, rate ratios, and rate differences for mortality by BMI value in the African American and white women are shown in **Figure 1**. The association between BMI and mortality in the African American women was only a trend; it did not meet the criterion for significance ($P < 0.05$). The mortality rate of the white women was lower than that of the African American women throughout most of the range of BMI values. The mortality rate of the white women with a BMI of 30 was estimated to be 8.04/1000 person-years. The corresponding BMI in the African American women associated with the same mortality rate was 18.

Because the death rate at a BMI of 21 was used as the reference for the calculation of the mortality rate ratios, the rate ratio curves for the African American and white women cross at that point. With increasing BMI, the rate ratio increased in the white women and was estimated to be 1.44 at a BMI of 30. The rate ratio tended to increase in the African American women with increasing BMI. At a BMI of 40.5, the African American women tended to have a rate ratio equivalent to that in the white women who had a BMI of 30.

A BMI of 21 was also used as the reference in the evaluation of mortality rate differences, and therefore the rate difference was zero in both the African American and white women at that BMI. With increasing BMI, the rate difference increased in the white women and tended to increase in the African American women. For the white women with a BMI of 30, the rate difference was 3/1000 person-years. An equivalent difference was calculated for the African American women with a BMI of 35.5.

Overall there were 319 cases of diabetes in the African American women and 438 cases in the white women. In contrast with the results for mortality, BMI and diabetes were significantly associated in both the African American and the white women. As shown in **Figure 2**, the incidence rates increased similarly in both groups

TABLE 2
Baseline characteristics of the white and African American women in the analysis sample from the Atherosclerosis Risk in Communities Study cohort[1]

	All participants after standard exclusions		Diabetic participants excluded		Hypertensive participants excluded		Hypertriglyceridemic participants excluded	
	White (n = 5715)	African American (n = 2304)	White (n = 438)	African American (n = 442)	White (n = 1452)	African American (n = 1286)	White (n = 683)	African American (n = 124)
Age (y)	54.0 ± 5.7[2]	53.2 ± 5.7	55.6 ± 5.9	55.3 ± 5.8	56.0 ± 5.7	54.2 ± 5.7	53.7 ± 5.8	53.2 ± 5.9
BMI (kg/m^2)	26.6 ± 5.1	30.8 ± 6.1	31.2 ± 5.1	33.2 ± 6.2	28.9 ± 5.2	31.8 ± 6.1	26.2 ± 5.2	30.7 ± 6.3
Education (%)								
Less than high school graduate	15.5	39.2	25.8	53.6	21.7	45.0	25.0	47.6
High school graduate	43.0	23.0	45.4	22.9	47.3	23.3	45.2	29.8
More than high school graduate	41.5	37.9	28.8	23.5	31.0	31.7	29.7	22.6
Smoking (%)								
Current	23.8	23.5	21.2	18.6	19.5	22.2	27.7	27.4
Former	24.8	17.7	20.3	21.3	25.5	17.4	24.2	17.7
Never	51.4	58.9	58.5	60.2	55.0	60.3	48.2	54.8
Physical activity (%)								
Low	39.9	61.2	47.5	64.5	42.6	64.2	46.8	59.7
Moderate	47.8	34.2	45.0	31.9	47.3	32.2	44.1	35.5
High	12.3	4.6	7.5	3.6	10.1	3.6	9.1	4.8
Alcohol consumption (%)								
Current	61.1	20.6	40.6	10.9	54.3	18.4	48.6	19.3
Former	13.8	19.4	22.4	23.1	15.5	21.3	16.1	16.1
Never	25.0	60.1	37.0	66.1	30.2	60.3	35.3	64.5

[1] Data from reference 10.
[2] $\bar{x}$ ± SD.

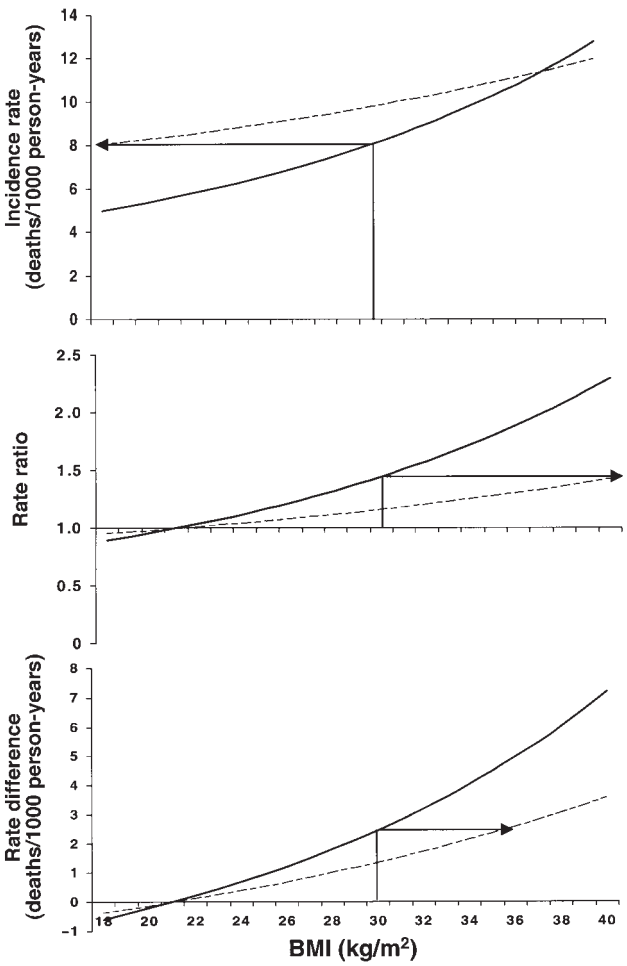

FIGURE 1. The incidence rate, rate ratio, and rate difference for mortality by BMI value in the white (solid line) and African American (dashed line) women in the Cancer Prevention Study I cohort. A BMI of 21 was used as the reference.

with increasing BMI. The differences in the incidence rates were consistent across a wide range of BMI values. Nevertheless, the rate ratios were considerably lower in the African American women than in the white women at high BMI values. This divergence was driven by the higher rate of diabetes in the reference BMI group in the African American women (10/1000 person-years) than in the white women (5/1000 person-years).

There were 407 cases of hypertension in the African American women and 1111 cases in the white women. The incidence rate was higher in the African American women than in the white women in the normal range of BMI (**Figure 3**). The effect of BMI on the rate ratio was greater in the white women than in the African American women, but the effect of BMI on the rate difference was greater in the African American women.

There were 179 cases of hypertriglyceridemia in the African American women and 1085 cases in the white women. The incidence rate was consistently and markedly higher in the white women than in the African American women (**Figure 4**). The association between BMI and hypertriglyceridemia peaked at a BMI of <35 and the incidence declined at very high BMI values (P for quadratic BMI term < 0.001 in both ethnic groups). As shown in Figure 4, there was no BMI at which the incidence rate or the rate difference was as high in the African American women as it was in the white women with a BMI of 30. For the rate ratio, a risk equivalent to that of white women with a BMI of 30 was estimated in the African American women at a BMI of 27.0.

The estimated incidence rates, rate ratios, and rate differences for mortality, diabetes, hypertension, and hypertriglyceridemia in the white women with a BMI of 30 are summarized in **Table 3**. The last column contains the BMI values in the African American women that were associated with risks equivalent to those estimated in the white women with a BMI of 30. The equivalent BMI varied widely for different outcomes and risk estimates. For the incidence rate and rate difference of hypertriglyceridemia, a BMI with equivalent risk could not be estimated from our models.

DISCUSSION

This study shows that a BMI cutoff for obesity in African American women could be recommended that is higher, lower, or similar to that in white women, depending on the outcome and the measure of effect examined. The effect of these choices is not subtle; in our examples, the BMI values in the African American

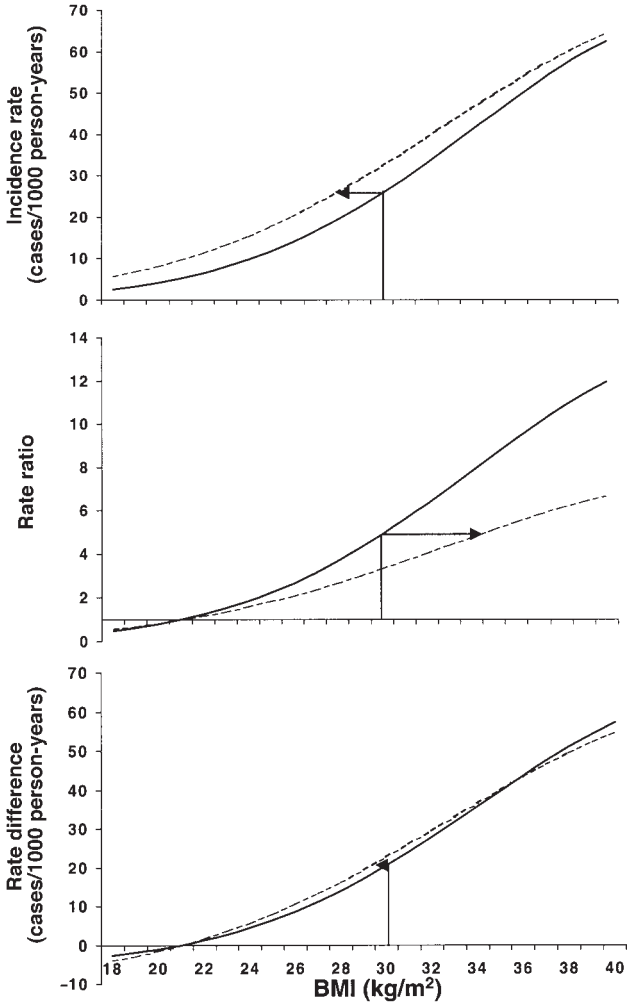

FIGURE 2. The incidence rate, rate ratio, and rate difference for diabetes by BMI value in the white (solid line) and African American (dashed line) women in the Atherosclerosis Risk in Communities Study cohort. A BMI of 21 was used as the reference.

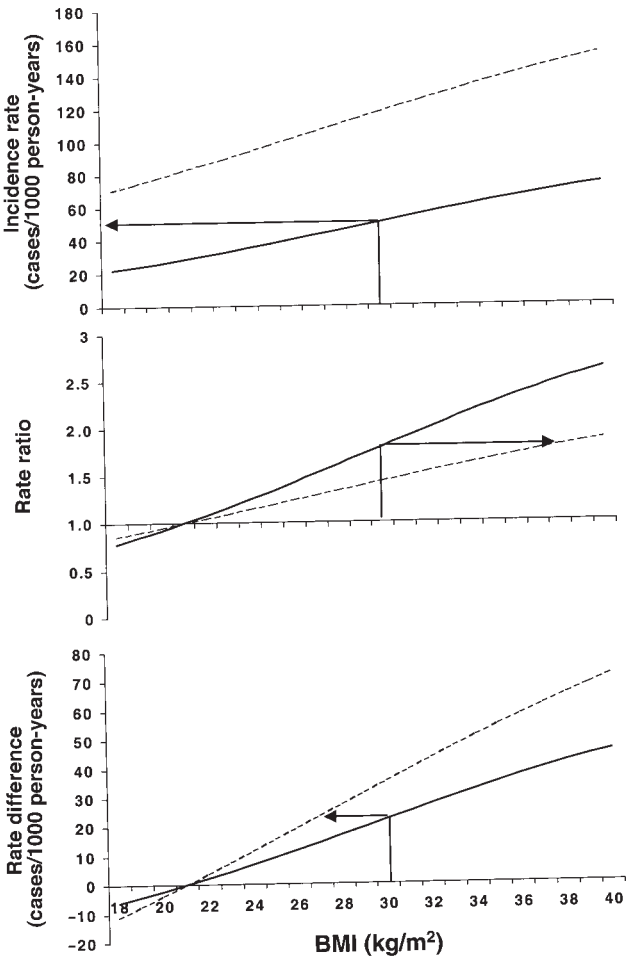

FIGURE 3. The incidence rate, rate ratio, and rate difference for hypertension by BMI value in the white (solid line) and African American (dashed line) women in the Atherosclerosis Risk in Communities Study cohort. A BMI of 21 was used as the reference.

women that were associated with risks equivalent to those of the white women with a BMI of 30 ranged from very low to as high as 40.5, depending on the criteria used. In fact, there was essentially no BMI value in the African American women at which the incidence of death was as low as it was in the white women with a BMI of 30, and there was no BMI value in the African American women at which the incidence of hypertriglyceridemia was as high as it was in the white women with a BMI of 30. Factors other than body weight obviously have powerful effects on these outcomes in African American and white women.

The influence of these other factors on the risks experienced by African American and white women argues against the use of incidence rates as a criterion for setting BMI cutoffs. For all the outcomes examined here, there were important differences in the incidence rates of the 2 ethnic groups at the BMI reference value. The choice of 21.0 as the BMI reference value in the present study was arbitrary, and any BMI within the normal range (18.5–25.0) would have been equally applicable to this example.

Although assigning BMI cutoffs to produce equivalence in the overall burden of disease has some appeal, if the goal is to identify a BMI value at which risk is higher because of an elevation in BMI, then some type of measure that considers differences in baseline risk is needed. Both rate ratios and rate differences incorporate the level of risk at BMI reference values. However, we find the rate ratio the less attractive of the 2 measures of effect because equivalent multiplicative increases in risk can be associated with very different increases in the number of cases. The increase in the number of cases above that in persons with reference weight seems to be the most meaningful estimate for public health and policy decisions.

Among the outcomes that could be used to set a BMI cutoff for obesity, mortality has several appealing qualities. Mortality is unique, extreme, and easily and precisely assessed. However, in the present study [as in some other studies (5)], no significant association between BMI and mortality was detected in the African American women. This implies that no BMI is better than another in terms of promoting longevity. This finding is so counterintuitive that it is difficult to accept. An association may not have been found in the present study because of unidentified sources of confounding or limitations in statistical power.

It would be difficult to defend the use of any one disease or risk factor over that of any other to set standards for obesity. A

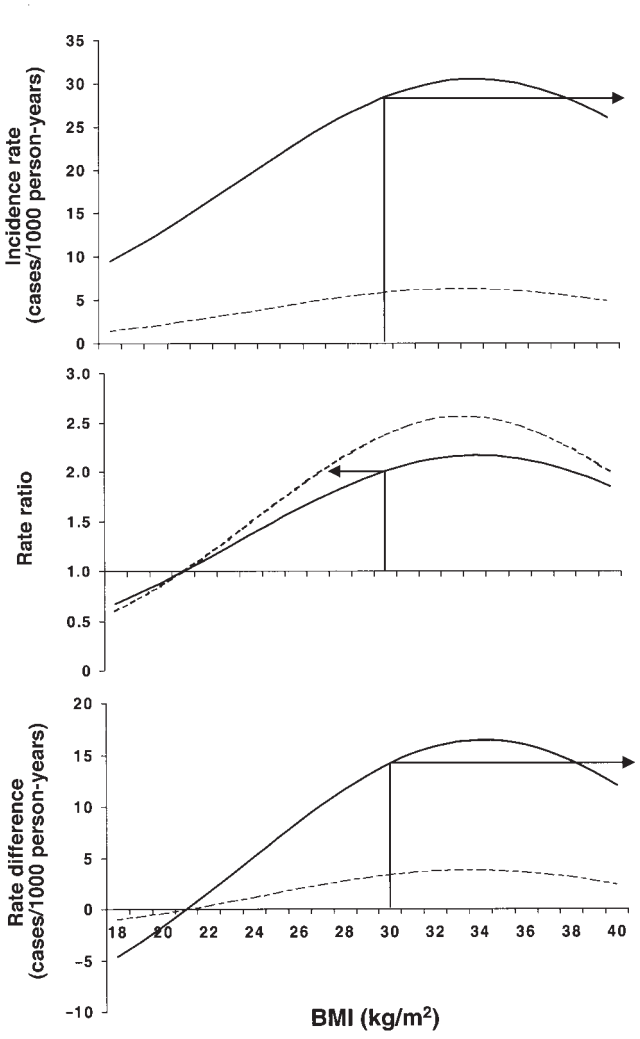

FIGURE 4. The incidence rate, rate ratio, and rate difference for hypertriglyceridemia by BMI value in the white (solid line) and African American (dashed line) women in the Atherosclerosis Risk in Communities Study cohort. A BMI of 21 was used as the reference.

TABLE 3
Risks associated with a BMI (in kg/m^2) of 30 in the white women and the BMI values associated with equivalent risks in the African American women[1]

	White women with a BMI of 30	Equivalent BMI in African American women
		kg/m^2
All-cause mortality		
Incidence rate (deaths/1000 person-years)	8	18.0
Rate ratio	1.44	40.5
Rate difference (deaths/1000 person-years)	3	35.5
Diabetes		
Incidence rate (cases/1000 person-years)	26	28.0
Rate ratio	4.9	34.5
Rate difference (cases/1000 person-years)	21	29.5
Hypertension		
Incidence rate (cases/1000 person-years)	50	Very low
Rate ratio	1.79	38.0
Rate difference (cases/1000 person-years)	23	26.5
Hypertriglyceridemia		
Incidence rate (cases/1000 person-years)	29	None
Rate ratio	2.0	27.0
Rate difference (cases/1000 person-years)	15	None

[1] Values for all-cause mortality are derived from data from the Cancer Prevention Study I (9). Values for diabetes, hypertension, and hypertriglyceridemia are derived from data from the Atherosclerosis Risk in Communities Study (10). For rate ratios and rate differences, a BMI of 21 was used as the reference.

measure such as disability-adjusted life years offers a logical option that could be used as an outcome to set obesity standards (18). This measure is the sum of the years of life lost and the years lived with a disability, adjusted for the severity of the disability. Of course, numerous subjective decisions must be made to determine what constitutes a disability and to establish scores for the severity of different disabilities.

Some of the differences in BMI-associated risk noted in the present study between the African American and white women could have been because of differences in body composition. Wagner and Heyward (19) recently reviewed biological differences in blacks and whites. They noted that blacks have greater bone mineral density and body protein content than do whites, resulting in a greater fat-free body density. They also pointed out differences in the distribution of subcutaneous and visceral fat and in the length of the limbs relative to the trunk. Deurenberg et al (20) showed that for the same body fat, age, and sex, African Americans have a BMI that is 1.3 units lower than that of whites. If excess body fat is the critical variable that conveys obesity-associated risk, then this would imply that the BMI standard in African Americans should be 1.3 units lower than that of whites. The present study clearly shows that the discrepancies in different disease risks associated with BMI cannot be entirely accounted for by this adjustment.

One strength of the present study is that all outcomes were examined prospectively rather than in cross-section. Therefore, the antecedent-consequence uncertainty was satisfied, and BMI was examined as a predictor of future risk. Another strength is the relatively large number of African American women studied and the availability of data on fasting glucose, lipids, and blood pressure.

A limitation of the CPS-I data was that the baseline BMI measurements for the mortality analyses were collected ≈40 y ago. Nevertheless, a more recent report from the CPS-II, in which the baseline data were collected in 1982 (21), showed patterns between BMI and all-cause mortality that were similar to those seen in the CPS-I. In both studies, there was no significant effect of BMI on mortality in African American women, whereas there were significant effects of similar magnitude in white women. A limitation common to both the CPS-I and the CPS-II cohorts is that height and weight data were from self-reports. Although self-reported height and weight are in general highly correlated with measured height and weight, with correlation coefficients above 0.9, heavier persons tend to underreport their weight more than leaner persons do (22–24). This bias could affect the results of an analysis of BMI and mortality.

Explicitly stating the measures of effect and the outcomes used to determine a BMI cutoff for obesity would aid the quantitative evaluation of cutoffs for different ethnic groups. However, other issues must also be considered in decisions related to policy. In both the CPS-I and ARIC cohorts, ethnicity was determined by self-identification with the use of a checklist. This variable names the cultural, social, and familial group with which the participants identify themselves and should not be overinterpreted. In a multiethnic society such as that in the United States, it is likely that many persons would have difficulty classifying themselves as belonging to only one ethnic group. This ambiguity, together with our lack of insight into what specific components or aspects of ethnicity lead to observed differences in risks, make the justification of different BMI cutoffs more difficult. Because of the enormous stigma attached to obesity and the sensitivity of ethnic issues, it seems unlikely that formal policies setting different cutoffs for different ethnic groups in the United States are feasible.

The adoption of different cutoffs for all persons within different countries seems more feasible. BMI cutoffs can affect policy because they are used to evaluate the health of populations and the need for health promotion activities. Risks for many disease outcomes, however expressed, are generally higher at higher BMI values, and most health professionals view excess adiposity negatively. Therefore, even in groups such as Samoans, in which the scientific evidence may indicate that a higher cutoff could be justified, a policy to set a higher BMI cutoff for obesity may not gain strong support. For advocates of health promotion, evidence indicating the need for a lower BMI cutoff for obesity is of more concern. Thus, a BMI cutoff for obesity that is lower than that accepted in Western countries may become accepted in some Asian countries. Whether the criteria used are quantitative, qualitative, political, or pragmatic, careful examination of the decision rules used to set the definition of obesity in diverse populations is merited.

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REFERENCES

1. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. Obes Res 1998;6(suppl):51S–209S.
2. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation on obesity, Geneva, 3–5 June 1997. Geneva: World Health Organization, 1998:9.

3. Kuczmarski RJ, Flegal KM. Criteria for definition of overweight in transition: background and recommendations for the United States. Am J Clin Nutr 2000;72:1074–81.
4. International Diabetes Institute. The Asia-Pacific perspective: redefining obesity and its treatment. Geneva: World Health Organization, 2000:1–55.
5. Stevens J. Obesity and mortality in African Americans. Nutr Rev 2000;58:346–58.
6. Stern M, Patterson J, Mitchell B, Haffner S, Hazuda H. Overweight and mortality in Mexican Americans. Int J Obes 1990;14:623–9.
7. Mendlein JM, Freedman DS, Peter DG, et al. Risk factors for coronary heart disease among Navajo Indians: findings from the Navajo Health and Nutrition Survey. J Nutr 1997;127(suppl):2099S–105S.
8. Welty TK, Lee ET, Yeh J, et al. Cardiovascular disease risk factors among American Indians. The Strong Heart Study. Am J Epidemiol 1995;142:269–87.
9. Lew E, Garfinkel L. Variations in mortality by weight among 750,000 men and women. J Chronic Dis 1979;32:563–76.
10. The ARIC Investigators. The Atherosclerosis Risk in Communities (ARIC) Study: design and objectives. Am J Epidemiol 1989;129: 687–702.
11. National Heart Lung and Blood Institute. Atherosclerosis Risk in Communities (ARIC) Study. Operations manual no. 10: clinical chemistry determinations. Version 1.0. Chapel Hill, NC: ARIC Coordinating Center, School of Public Health, University of North Carolina, 1987.
12. National Heart Lung and Blood Institute. Atherosclerosis Risk in Communities (ARIC) Study. Operations manual no. 8: lipid and lipoprotein determinations. Version 1.0. Chapel Hill, NC: ARIC Coordinating Center, School of Public Health, University of North Carolina, 1987.
13. Chambless LE, McMahon RP, Brown SA, Patsch W, Heiss G, Shen Y-L. Short-term intraindividual variability in lipoprotein measurements: the Atherosclerosis Risk in Communities (ARIC) Study. Am J Epidemiol 1992;136:1069–81.
14. Chambless L, McMahon R, Wu K, Folsom A, Finch A, Shen Y-L. Short-term intraindividual variability in hemostasis factors: the ARIC Study. Ann Epidemiol 1992;2:723–33.
15. Kleinbaum D, Kupper L, Muller K, Nizam A. Applied regression analysis and other multivariable methods. Pacific Grove, CA: Duxbury Press, 1998.
16. SAS Institute Inc. SAS/STAT user's guide, version 6. Vol 1. 4th ed. Cary, NC: SAS Institute Inc, 1997.
17. Kaufman JS, Cooper RS, McGee DL. Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. Epidemiology 1997;8:621–8.
18. Murray C. Quantifying the burden of disease: the technical basis for disability-adjusted life years. Bull World Health Organ 1994;72: 429–45.
19. Wagner DR, Heyward VH. Measures of body composition in blacks and whites: a comparative review. Am J Clin Nutr 2000;71:1392–402.
20. 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.
21. Calle E, Thun M, Petrelli J, Rodriguez C, Heath W Jr. Body-mass index and mortality in a prospective cohort of US adults. N Engl J Med 1999;341:1097–105.
22. Stevens J, Keil J, Waid R, Gazes P. Accuracy of current, 4-year, and 28-year self-reported body weight in an elderly population. Am J Epidemiol 1990;132:1156–63.
23. Plankey M, Stevens J, Flegal K, Rust P. Prediction equations do not eliminate systematic error in self-reported BMI. Obes Res 1997; 5:308–14.
24. Rowland ML. Self-reported weight and height. Am J Clin Nutr 1990;52:1125–33.

Erratum

Lund EK, Wharf SG, Fairweather-Tait SJ, Johnson IT. Oral ferrous sulfate supplements increase the free radical–generating capacity of feces from healthy volunteers. Am J Clin Nutr 1999;69:250–5.

On page 253, the unit on the *y* axis of Figure 2 should read (μmol/g wet wt).

Erratum

Stevens J, Juhaeri, Cai J, Jones DW. The effect of decision rules on the choice of a body mass index cutoff for obesity: examples from African American and white women. Am J Clin Nutr 2002;75:986-92.

On page 991, column 1 (paragraph 2, lines 9-15), the text should read as follows: Deurenberg et al (20) showed that for the same body fat, age, and sex, African Americans have a BMI that is 1.3 units higher than that of whites. If excess body fat is the critical variable that conveys obesity-associated risk, then this would imply that the BMI standard in African Americans should be 1.3 units higher than that in whites.

We had repeated an error in the abstract of the publication by Deurenberg et al referred to above.

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