Published in final edited form as:

Eur J Epidemiol. 2012 July ; 27(7): 489–497. doi:10.1007/s10654-012-9695-5.

Body mass index and mortality in an ethnically diverse population: the Multiethnic Cohort Study

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Abstract

Body mass index (BMI) has been strongly related to overall mortality, but the consistency of this association across diverse ethnic groups and the effects of early adult BMI versus BMI in later adulthood have not been adequately studied. A prospective analysis was performed using data from 183,211 adults aged 45–75 who enrolled the population-based Multiethnic Cohort Study by completing a questionnaire that included self-reported weight and height information in 1993–1996. Participants were African Americans, Native Hawaiians, Japanese Americans, Latinos, and whites living in Hawaii and California. During an average 12.5 years of follow-up, 35,664 deaths were identified. To control for confounding caused by conditions that lead to weight loss and mortality, we excluded participants with a history of cancer or heart disease, who ever smoked, and who died within the first 3 years of follow-up. An increased risk of mortality was observed in participants with a BMI ≥ 27.5 in both men and women compared with the reference category of BMI 23.0–24.9; a BMI ≥ 35.0 carried a greater risk of mortality in men than in women. Although the findings were generally similar across ethnic groups, the association of higher BMI with

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Conflict of interest The authors declare that they have no conflict of interest.

mortality in Latino men appeared to be weaker than in the other groups. A BMI of 25.0–34.9 at age 21 showed a stronger positive association, with no further increase in risk for a BMI ≥ 35.0, than did BMI in later adulthood. These results indicate that the association of BMI with mortality is generally consistent across sex and ethnic groups, with some variation in the strength of the effect. Most notably, the effect of overweight in young adulthood appears to be much stronger than that of overweight in later adulthood on mortality in later life. This emphasizes the importance of weight management in childhood and adolescence.

Keywords

Body mass index; Cohort studies; Mortality; Multiethnic population; Obesity

Introduction

Although body mass index (BMI) is based only on weight and height and thus is not a direct measure of body fat [1], it has been shown to be a reasonable proxy [2] and has long been used in epidemiological studies as a measure of general adiposity [3, 4]. Indeed, associations between overweight and obesity, as defined by BMI, and several diseases, including cardiovascular disease, type II diabetes, and certain cancers, are well-established [5, 6]. Large prospective studies in North America [7–15] and Europe [16–18] have found that BMI was strongly related to all-cause mortality, generally in the form of a U- or J-shaped curve. However, because the observed relationships may have been distorted by uncontrolled confounders or effect modifiers, such as smoking and weight loss due to preexisting illness [4, 19], some uncertainty remains.

To overcome some of these problems, a recent large pooling study limited the analysis to never smokers with no history of cancer or heart disease, and found a J-shape relationship between BMI and all-cause mortality [20]. However, the subjects were all non-Hispanic whites. Although some studies on the BMI-mortality association have been conducted in other racial/ethnic groups, most of these studies were in single homogeneous populations. The Multiethnic Cohort Study provides an unusual opportunity to extend this research using a large, diverse population sample of five racial/ethnic groups, in which data on potential confounders were collected uniformly.

Methods

Study population

The Multiethnic Cohort Study was established to investigate the association of dietary, lifestyle, and genetic factors with cancer and other chronic diseases [21]. In 1993–1996, over 215,000 participants aged 45–75 years at recruitment entered the cohort by completing a 26-page mailed questionnaire and consenting to be in the study. Participants were mostly African Americans, Native Hawaiians, Japanese Americans, Latinos, and whites living in Hawaii and California. The study was approved by the institutional review boards of the University of Hawaii and the University of Southern California. In the current analyses, we successively excluded participants who did not self-identify as one of the five principal

racial/ethnic groups (n = 13,989), who reported implausible diets (n = 8,263) based on total energy intake or its components, who were missing information on weight (n = 1,572) or height (n = 970), who had invalid BMI (<15 or >50 kg/m², n = 676), or who were missing information on smoking (n = 7,101) at cohort entry. A total of 183,211 participants (83,200 men and 100,011 women) remained.

Body mass index and covariates

BMI (kg/m²) was calculated from self-reported weight and height from the baseline questionnaire. Based on the principal and additional cut-off points defined by the World Health Organization [22], we categorized BMI as: >18.5 (underweight), 18.5–22.9 (low-normal), 23.0–24.9 (high-normal), 25.0–27.4 (low-overweight), 27.5–29.9 (high-overweight), 30.0–34.9 (obese class I), and 35.0 or more (obese class II+). A BMI of 23.0–24.9 was used as the reference category for the analyses on the basis of a preliminary analysis showing that this range of BMI was associated with the lowest mortality. Obese class III (BMI ≥ 40.0) was combined with obese class II, because the number of subjects in this group was too small to be analyzed separately, especially in Japanese Americans. BMI at baseline was calculated using the reported current weight and height, and BMI at age 21 was calculated using reported weight at age 21 and current height. On the baseline questionnaire, study participants also provided information on diet (including alcohol consumption), socio-demographic factors, personal behaviors (including smoking and physical activity), history of medical conditions, and use of medications.

Outcome ascertainment

Deaths of cohort members were identified by linkage to the death certificate files in Hawaii and California and to the U.S. National Death Index through December 31, 2007. During an average 12.5 years of follow-up, we identified 35,664 deaths (19,678 men and 15,986 women). Death from all causes was the primary endpoint in the analyses.

Data analysis

A Cox proportional hazards model, with age as the time metric, was used to estimate the hazard ratios (HR) and 95 % confidence intervals (CI) for mortality in relation to BMI. We first performed the analysis for all 183,211 eligible cohort members. To avoid confounding in the BMI-mortality association as much as possible, we then excluded 1) participants with a history of cancer (based on the baseline questionnaire and tumor registries) or heart disease (based on the baseline questionnaire); 2) ever smokers at baseline; and 3) participants who died within the first 3 years of follow-up [23]. All analytic models included adjustment for age at cohort entry (<50, 50–54, 55–59, 60–64, 65–69, 70–74, 75 years) and alcohol consumption (ethanol; 0, 1–<10, 10–<20, 20–<30, 30–<40, 40–<50, 50–<60, 60 g per day for men; 0, 1–<5, 5–<10, 10–<15, 15–<20, 20–<25, 25–<30, 30 g per day for women) as strata variables and race/ethnicity as a covariate. Although alcohol consumption was related to both BMI and mortality, adjustment for it did not affect substantially the BMI-mortality association in healthy never smokers. For the models including smokers, we additionally adjusted for smoking using a comprehensive model developed for lung cancer incidence, which included average number of cigarettes; average number of cigarettes squared; indicator variables for former and current smokers; number of

years smoked (time-dependent); number of years since quitting (time-dependent); and interactions of race/ethnicity with the following variables: average number of cigarettes, average number of cigarettes squared, smoking status, and number of years smoked [24]. Because BMI is a reflection of energy balance, factors such as energy intake and physical activity were not controlled for, as this would have resulted in over-adjustment. Educational level, as a marker of socioeconomic status, was considered as a potential confounding factor, but was not included in the final model because it did not change the results. Tests for heterogeneity between subgroups were based on Wald statistics. All analyses were performed using SAS statistical software version 9.2 (SAS Institute, Cary, NC, USA).

Results

Baseline characteristics are presented in Table 1, for all eligible members and according to sequentially applied exclusion criteria. After applying all exclusion criteria, 26 % of men and 48 % of women out of the full sample remained. Compared with the full sample, “healthy never smokers” (never smokers with no history of cancer or heart disease at baseline and living after 3 years of follow-up, presented in the last column of the table) were younger for men and consumed less alcohol for both men and women. In men, the ethnic proportions within the sample did not vary substantially with sequential exclusions except for African Americans whose percentage decreased. In women, the proportions of African Americans and whites decreased, while those of Japanese Americans and Latinos increased especially with exclusion of ever smokers. The exclusions had little effect on the proportions in each BMI category for either men or women.

The association between BMI at baseline and total mortality is shown in Table 2 and Fig. 1. In the full sample, the relation between BMI and mortality was U-shaped with similar HRs in both sexes. However, after applying all exclusion criteria, the relation was closer to a J-shape in men (P value for heterogeneity across the exclusion criteria = 0.0071 for the underweight, 0.063 for the obese I, and <0.001 for the obese II+ category). In women, the change in the association with exclusions was smaller (P value for heterogeneity across the exclusion criteria = 0.014 for the underweight, 0.22 for the obese I, and 0.049 for the obese II+ category), although it was in the same direction as in the men. In healthy never smokers, as presented in the last row of Table 2 for each sex, both underweight and over-weight carried an increased risk in men and women (P value for heterogeneity between sexes = 0.34 for the underweight and 0.80 for the high-overweight category), with a suggestion that obesity carried a higher risk in men than women (P value for heterogeneity between sexes = 0.016 for the obese II+ category), and that underweight carried a higher risk in women than men, where the HR was not statistically significant. In both men and women, the association of higher BMI with mortality showed a dose–response pattern. The pattern of risk was similar across age groups, although there were differences in strength of the association (Table 3). The middle age group (55–64 years) showed a stronger association between higher BMI and mortality in women than the younger and the older groups (P value for heterogeneity across the age groups = 0.049), while there was no significant heterogeneity across the age groups in men.

The ethnic-specific analyses for healthy never smokers are shown in Table 4. Among men, there was a monotonic positive association of mortality with increasing BMI above normal (P for trend based on log-transformed BMI above normal <0.001 for all ethnic groups) and the association in Latino men appeared to be weaker (P value for heterogeneity between Latinos and the other groups = 0.062 for low-overweight, 0.025 for high-overweight, 0.0011 for obese I, and 0.0030 for obese II+ groups). Among women, the associations of higher BMI showed very similar patterns across all ethnic groups, with generally monotonic positive trends and mostly statistically significant HRs in the highest two categories. For the underweight groups in Table 4, no statistically significant increases in risk were seen among men, and the point estimates did not show a consistent pattern. Among women, the point estimates were increased for the under-weight category in all groups except Latinos.

The association of BMI at age 21 with mortality among healthy never smokers was different from that of BMI at baseline (Fig. 1). At age 21, there was no apparent risk of underweight on subsequent mortality, in contrast to baseline, where there was an increased risk that was particularly pronounced in women. In fact, there was an inverse effect of underweight and low-normal weight at age 21 in women, suggesting a possible protective effect. In the overweight range (BMI 25.0–29.9), the relationship of BMI to mortality showed a much steeper slope for age 21 compared to baseline. For BMI at age 21, the effect showed a plateau at BMI above 30.0 in both sexes, whereas for BMI at baseline, the effect showed a continuous increase.

Discussion

In this large multiethnic cohort with follow-up for 12.5 years, the hazard ratios for mortality across BMI categories changed substantially after excluding persons with a history of cancer or heart disease, ever smokers, and persons who died within the first 3 years of follow-up period. Among healthy never smokers, the association between BMI and mortality was J-shaped in men and U-shaped in women. The obese II+ group showed a greater risk of mortality in men than in women. In ethnic-specific analyses, Latino men showed a less strong association for overweight and obese groups, while the relation of higher BMI to mortality was consistent across the ethnic groups among women. The increases in risk for overweight and obese I categories at age 21 were stronger than those based on BMI at cohort entry, with no further increase for the obese II + group. For women who were underweight and low-normal weight at age 21, we observed a decrease in risk among women.

Obesity, as reflected by BMI, is a well-recognized risk factor for several diseases, including diabetes, hypertension, dyslipidemia, coronary artery disease, and some cancers [6, 25]. However, the relation between BMI and all-cause mortality remains somewhat uncertain [4]. Reverse causality, in which diseases lead to both weight loss and higher mortality, could attenuate the apparent relationship of obesity to mortality, especially in studies with shorter follow-up periods and inadequate control for important confounders such as smoking [19, 26]. To address this issue, we applied the exclusionary approach that a number of large prospective studies have used [23]. We observed substantial changes in the HRs especially after excluding ever smokers, i.e., increases in risk for higher BMI and decreases in risk for

lower BMI. These changes were more marked in men than in women and suggest some residual confounding from smoking in the models that included ever smokers. This result is somewhat different from the findings of the pooled analyses of BMI and mortality involving whites [20] and Asians [27], where the results were more similar for men and women. For a given BMI, there are well-known body composition differences between the sexes including higher visceral and hepatic fat in men and higher peripheral or subcutaneous fat in women [28]. Also, body mass is more closely related to the visceral fat in men than in women, which may partly explain the stronger association with mortality for the higher BMI group in men than in women [18].

The association between overweight (i.e., BMI 25.0–29.9) and mortality is controversial. The National Health and Nutrition Examination Survey [11, 23] and a meta-analysis based on 26 observational studies [29] showed no significant association between overweight and mortality overall. A recent study from a national longitudinal study of Canadian adults observed a protective association of overweight with mortality [14], while two large cohorts in the US observed an increased risk of death associated with overweight [12, 13]. In the present study, the low-overweight range (BMI 25.0–27.5) was not associated with mortality, while the high-overweight range (BMI 27.5–29.9) carried an increased risk in both men and women. Methodological issues may mask the true relationship between overweight and all-cause mortality [30]. For example, the reference groups were not identical in the studies, some used the entire normal range (BMI 18.5–24.9) [11, 14, 23, 29], while others used a subset of this range [12, 13]. As pointed out in the pooling study of the National Cancer Institute Cohort Consortium [20], studies including smokers and persons with preexisting diseases may underestimate the association of overweight and obesity with mortality. In fact, after excluding these less healthy participants in the present study, the high-overweight groups showed about a 20 % increase in risk, compared with the reference group, for both men and women. In addition to a possible association with total mortality, overweight should be avoided as it is related to an increased risk of other health outcomes, and overweight often leads to obesity [30]. Furthermore, the current study showed that being overweight in early adulthood (age 21) was related to a significant increase in mortality risk in later life (about a 100 % increase in risk for being high-overweight in men). In contrast, we found that women in the low-normal weight category (BMI 18.5–22.9) at age 21 had a significant decrease in mortality risk. This interesting finding suggests that being very lean in early adulthood may be especially desirable, and offers support for the recommendation in the 2007 WCRF/AICR report [31] to “ensure that body weight through childhood and adolescent growth projects towards the lower end of the normal BMI range at age 21 (page 374).”

An important contribution of the present study is the comparison of findings across racial/ethnic groups. Only a few previous studies included more than one ethnic group, and in those studies, the majority of the subjects were white [7, 12]. The differences in findings across the different populations in our study are intriguing. Latino men showed a less strong association of higher BMI with mortality than did men from other ethnic groups. Among women, the HRs for higher BMI categories were not very different by ethnicity. Previous studies have shown differences in body fat pattern and accumulation by ethnicity, and possible ethnic-specific pathways to obesity-related disease [32–35]. Ethnic disparities in the

BMI-mortality relationship may be attributable to fundamental differences in adipose tissue biology, but this possibility has not been well studied [32].

Our study's strengths include the large sample size and population-based prospective design that allowed us to investigate the relation between BMI and mortality by sex and race/ethnicity, excluding people with conditions and exposures (notably smoking) that could lead to both weight loss and increased risk of mortality. In addition, we were able to consider other important confounding variables.

Nevertheless, our study had some limitations. Because we used self-reported weight and height, misclassification of BMI may have occurred. A pilot study with 60 female participants of the Multiethnic Cohort Study showed high correlations between self-reported and measured weight ($r = 0.98$) and height ($r = 0.78$) [36]. Although correlations between self-reported and measured values of these variables are usually strong, systematic errors according to sex, ethnicity, and overweight status have been reported [37–39]. One study in nationally representative U.S. samples assessed the BMI-mortality association using both self-reported and measured weight and height, and demonstrated that BMI based on self-reported values led to underestimation of the association for underweight and overestimation of the association for obesity [40]. Furthermore, long-term recall of weight at age 21 may be further error-prone.

The follow-up period (mean = 12.5 years) may not be long enough to fully reflect the association of obesity with long-term mortality. However, we were also able to examine the relationship of BMI in young adulthood with later mortality. Weight change from age 21 to cohort entry might help us to understand the differential effects of weight status in early versus later adulthood. Nevertheless, in the current paper, we did not include the results on weight change because we lacked information on weight change patterns (e.g., gradual/abrupt and fluctuating/stable) or on whether the weight change occurred intentionally or not, which would be needed to adequately explain the complex findings on weight change. Future research should explore these aspects of weight change over the adult lifespan.

Abdominal obesity, usually measured by waist/hip circumference, has been found to be strongly related to health outcomes independent of BMI [41] and has been suggested as a better predictor of mortality than BMI in older adults [8, 42]. Since we obtained waist and hip circumference measurement in a later follow-up survey on this cohort, we plan to explore the specific association of abdominal obesity specifically with mortality in the future.

In conclusion, among healthy never smokers, adult overweight and obesity were associated with an increased risk of mortality in men and women. Although the findings generally were consistent across the five racial/ethnic groups, there were some differences, most notably a weaker effect of overweight and obesity on mortality in Latino men. Finally, a BMI in the overweight range at age 21 showed stronger associations with mortality than did BMI in later adulthood. This emphasizes the importance of weight management in children and young adults. The findings of this study suggest that further research on the relationship of obesity to overall and disease-specific mortality are needed. In particular, studies that

examine differences in effects by ethnicity, sex, and lifecourse might provide useful insights towards future obesity prevention.

Acknowledgments

This work was supported in part by the National Cancer Institute at the National Institutes of Health [R37 CA54281].

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Fig. 1.

Risk for total mortality according to BMI at baseline and at age 21 among healthy never smokers. Healthy never smokers are never smokers who reported no previous cancer or heart disease at baseline and who did not die within the first 3 years. The models were adjusted for ethnicity, age at cohort entry, and alcohol consumption. *Vertical bars* indicate 95 % confidence intervals

Table 1

Characteristics of participants according to sequentially applied exclusion criteria in the Multiethnic Cohort Study, 1993–1996

Characteristics	Full sample	Sequential exclusions			
		Cancer and heart disease	Plus ever smokers	Plus deaths within 3 years	
Men					
Number of participants	83,200	68,332	21,893	21,540	
Age (years, mean)	60.2	59.1	58.5	58.4	
Ethnicity (%)					
African American	13.3	12.4	10.5	10.4	
Native Hawaiian	7.0	7.2	7.5	7.4	
Japanese American	30.4	31.1	29.7	29.8	
Latino	23.5	24.2	25.8	25.7	
White	25.7	25.1	26.6	26.7	
Alcohol intake (g/day, mean)	14.7	15.1	9.8	9.8	
BMI at baseline (%)					
<18.5	0.7	0.6	0.5	0.5	
18.5–22.9	15.5	15.5	15.8	15.7	
23.0–24.9	20.1	20.1	20.9	20.9	
25.0–27.4	29.5	29.7	29.9	30.0	
27.5–29.9	17.1	17.2	16.8	16.8	
30.0–34.9	13.3	13.1	12.3	12.3	
35.0	3.9	3.8	3.8	3.8	
BMI at age 21 (%)					
<18.5	4.6	4.6	4.5	4.5	
18.5–22.9	55.4	55.4	54.1	54.1	
23.0–24.9	20.9	21.0	21.5	21.5	
25.0–27.4	13.5	13.4	14.1	14.2	
27.5–29.9	3.5	3.5	3.6	3.6	
30.0–34.9	1.7	1.7	1.6	1.6	
35.0	0.5	0.5	0.5	0.5	
Women					
Number of participants	100,011	83,135	48,108	47,601	
Age (years, mean)	59.6	59.0	59.6	59.5	
Ethnicity (%)					
African American	19.2	18.2	15.1	15.0	
Native Hawaiian	7.4	7.4	6.0	6.0	
Japanese American	27.8	29.1	35.0	35.2	
Latino	20.7	20.8	24.3	24.3	
White	24.8	24.4	19.6	19.6	
Alcohol intake (g/day, mean)	4.3	4.3	2.3	2.3	

Characteristics	Full sample	Sequential exclusions		
		Cancer and heart disease	Plus ever smokers	Plus deaths within 3 years
BMI at baseline (%)				
<18.5	2.7	2.6	2.8	2.8
18.5–22.9	26.5	27.2	28.2	28.3
23.0–24.9	17.3	17.6	18.0	18.0
25.0–27.4	19.0	19.1	19.0	19.0
27.5–29.9	12.7	12.6	12.3	12.3
30.0–34.9	14.1	13.7	13.2	13.2
35.0	7.8	7.3	6.5	6.5
BMI at age 21 (%)				
<18.5	14.3	14.2	14.6	14.7
18.5–22.9	65.9	66.2	66.7	66.8
23.0–24.9	11.1	11.1	10.9	10.9
25.0–27.4	5.1	5.0	4.7	4.6
27.5–29.9	1.6	1.6	1.5	1.5
30.0–34.9	1.4	1.4	1.2	1.2
35.0	0.6	0.6	0.4	0.4

Table 2

Risk for total mortality according to BMI at baseline and sequentially applied exclusion criteria in the Multiethnic Cohort Study, 1993–2007

	No. of deaths	BMI at baseline						
		<18.5	18.5–22.9	23.0–24.9	25.0–27.4	27.5–29.9	30.0–34.9	35.0
Men								
Full sample	19,678	1.98 (1.75–2.23)	1.25 (1.20–1.31)	1 (Reference)	0.97 (0.93–1.01)	1.02 (0.97–1.07)	1.23 (1.17–1.29)	1.60 (1.48–1.72)
Sequential exclusions								
Cancer and heart disease	13,089	1.97 (1.70–2.30)	1.24 (1.17–1.31)	1 (Reference)	0.99 (0.94–1.04)	1.05 (0.99–1.12)	1.28 (1.20–1.36)	1.80 (1.64–1.97)
Ever smokers	2,972	1.42 (0.98–2.07)	1.06 (0.94–1.19)	1 (Reference)	0.94 (0.84–1.05)	1.14 (1.01–1.29)	1.45 (1.27–1.65)	2.37 (1.99–2.82)
Deaths within 3 years	2,619	1.25 (0.81–1.91)	1.06 (0.93–1.20)	1 (Reference)	0.97 (0.86–1.09)	1.20 (1.05–1.37)	1.56 (1.36–1.79)	2.71 (2.26–3.26)
Women								
Full sample	15,986	1.88 (1.72–2.04)	1.13 (1.07–1.19)	1 (Reference)	1.04 (0.98–1.10)	1.12 (1.06–1.19)	1.25 (1.18–1.33)	1.63 (1.53–1.74)
Sequential exclusions								
Cancer and heart disease	10,841	1.84 (1.66–2.04)	1.12 (1.05–1.19)	1 (Reference)	1.03 (0.97–1.11)	1.13 (1.05–1.22)	1.28 (1.19–1.37)	1.73 (1.59–1.87)
Ever smokers	5,152	1.71 (1.47–1.99)	1.07 (0.98–1.17)	1 (Reference)	1.09 (0.99–1.20)	1.20 (1.08–1.33)	1.39 (1.26–1.54)	1.94 (1.73–2.19)
Deaths within 3 years	4,645	1.66 (1.42–1.95)	1.07 (0.97–1.18)	1 (Reference)	1.10 (0.99–1.21)	1.21 (1.08–1.35)	1.40 (1.26–1.55)	1.97 (1.74–2.23)

Unless otherwise indicated, data are reported hazard ratios (95 % confidence intervals) adjusted for ethnicity, age at cohort entry, and alcohol consumption. Models including smokers for smoking status, average number of cigarettes, squared average number of cigarettes, number of years smoked (time-dependent), number of years since quitting (time-dependent), ethnicity and smoking status, average number of cigarettes, squared average number of cigarettes and number of years smoked

Table 3

Risk for total mortality according to BMI at baseline and age group among healthy never smokers^a in the Multiethnic Cohort Study, 1993–2007^b

		No. of deaths	BMI at baseline						
			<18.5	18.5–22.9	23.0–24.9	25.0–27.4	27.5–29.9	30.0–34.9	>35.0
Men									
45–54 years	321	Not applicable ^c	1.11 (0.72–1.71)	1 (Reference)	0.80 (0.55–1.17)	1.12 (0.75–1.66)	1.67 (1.15–2.42)	2.78 (1.82–4.25)	
55–64 years	638	1.80 (0.57–5.68)	0.95 (0.70–1.30)	1 (Reference)	0.89 (0.69–1.14)	1.19 (0.92–1.55)	1.62 (1.24–2.11)	2.99 (2.17–4.13)	
65–75 years	1,660	1.24 (0.78–1.97)	1.07 (0.92–1.24)	1 (Reference)	1.03 (0.89–1.18)	1.23 (1.04–1.46)	1.46 (1.21–1.75)	2.36 (1.78–3.13)	
Women									
45–54 years	440	1.23 (0.59–2.56)	0.98 (0.71–1.36)	1 (Reference)	0.94 (0.67–1.32)	1.09 (0.76–1.57)	1.20 (0.86–1.68)	1.87 (1.33–2.64)	
55–64 years	1,111	1.96 (1.27–3.03)	1.39 (1.11–1.73)	1 (Reference)	1.46 (1.17–1.82)	1.70 (1.35–2.14)	1.76 (1.40–2.21)	2.73 (2.14–3.48)	
65–75 years	3,094	1.59 (1.33–1.89)	1.01 (0.90–1.13)	1 (Reference)	1.03 (0.92–1.17)	1.11 (0.97–1.27)	1.36 (1.19–1.55)	1.75 (1.48–2.08)	

^aNever smokers who reported no previous cancer or heart disease at baseline and did not die within the first 3 years

^bUnless otherwise indicated, data are reported hazard ratios (95 % confidence intervals) adjusted for age at cohort entry and alcohol consumption

^cNo death occurred in this category

Risk for total mortality according to BMI at baseline and ethnicity among healthy never smokers^a in the Multiethnic Cohort Study, 1993–2007^b

	No. of deaths	BMI at baseline						
		<18.5	18.5–22.9	23.0–24.9	25.0–27.4	27.5–29.9	30.0–34.9	>35.0
Men								
African Americans	464	1.51 (0.20–11.17)	1.34 (0.92–1.96)	1 (Reference)	1.08 (0.78–1.48)	1.64 (1.18–2.28)	1.87 (1.36–2.58)	2.72 (1.81–4.08)
Native Hawaiians	190	0.70 (0.09–5.40)	1.27 (0.64–2.51)	1 (Reference)	1.16 (0.65–2.07)	1.18 (0.66–2.12)	1.67 (0.94–2.97)	3.05 (1.69–5.54)
Japanese Americans	744	1.31 (0.74–2.31)	1.08 (0.89–1.31)	1 (Reference)	1.01 (0.82–1.24)	1.30 (0.99–1.71)	1.89 (1.35–2.65)	3.97 (2.08–7.58)
Latinos	698	0.93 (0.29–2.95)	0.90 (0.66–1.22)	1 (Reference)	0.82 (0.65–1.03)	0.98 (0.77–1.25)	1.18 (0.91–1.52)	1.82 (1.27–2.62)
Whites	523	1.57 (0.57–4.30)	1.05 (0.79–1.39)	1 (Reference)	1.01 (0.78–1.30)	1.08 (0.79–1.46)	1.61 (1.18–2.18)	3.31 (2.26–4.85)
Women								
African Americans	1,095	1.88 (1.09–3.24)	1.22 (0.94–1.58)	1 (Reference)	1.06 (0.84–1.34)	1.23 (0.97–1.54)	1.45 (1.16–1.80)	1.74 (1.37–2.20)
Native Hawaiians	280	1.52 (0.53–4.34)	0.98 (0.63–1.52)	1 (Reference)	0.91 (0.59–1.41)	1.24 (0.79–1.93)	1.34 (0.89–2.01)	2.10 (1.36–3.24)
Japanese Americans	1,433	1.59 (1.30–1.94)	1.01 (0.88–1.17)	1 (Reference)	1.05 (0.87–1.25)	1.16 (0.92–1.47)	1.35 (1.02–1.79)	1.77 (0.96–3.24)
Latinos	1,090	1.05 (0.49–2.25)	1.34 (1.07–1.68)	1 (Reference)	1.32 (1.07–1.62)	1.34 (1.08–1.67)	1.53 (1.23–1.89)	2.27 (1.77–2.91)
Whites	747	1.84 (1.22–2.78)	0.93 (0.74–1.17)	1 (Reference)	1.06 (0.84–1.34)	1.16 (0.89–1.51)	1.29 (1.00–1.66)	2.26 (1.68–3.05)

^aNever smokers who reported no previous cancer or heart disease at baseline and did not die within the first 3 years

^bUnless otherwise indicated, data are reported hazard ratios (95 % confidence intervals) adjusted for age at cohort entry and alcohol consumption