See corresponding editorial on page 759.

BMI and all-cause mortality in older adults: a meta-analysis[1–3]

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ABSTRACT

Background: Whether the association between body mass index (BMI) and all-cause mortality for older adults is the same as for younger adults is unclear.

Objective: The objective was to determine the association between BMI and all-cause mortality risk in adults ≥65 y of age.

Design: A 2-stage random-effects meta-analysis was performed of studies published from 1990 to 2013 that reported the RRs of all-cause mortality for community-based adults aged ≥65 y.

Results: Thirty-two studies met the inclusion criteria; these studies included 197,940 individuals with an average follow-up of 12 y. With the use of a BMI (in kg/m^2) of 23.0–23.9 as the reference, there was a 12% greater risk of mortality for a BMI range of 21.0–21.9 and a 19% greater risk for a range of 20.0–20.9 [BMI of 21.0–21.9; HR (95% CI): 1.12 (1.10, 1.13); BMI of 20.0–20.9; HR (95% CI): 1.19 (1.17, 1.22)]. Mortality risk began to increase for BMI >33.0 [BMI of 33.0–33.9; HR (95% CI): 1.08 (1.00, 1.15)]. Self-reported anthropometric measurements, adjustment for intermediary factors, and exclusion of early deaths or preexisting disease did not markedly alter the associations, although there was a slight attenuation of the association in never-smokers.

Conclusions: For older populations, being overweight was not found to be associated with an increased risk of mortality; however, there was an increased risk for those at the lower end of the recommended BMI range for adults. Because the risk of mortality increased in older people with a BMI <23.0, it would seem appropriate to monitor weight status in this group to address any modifiable causes of weight loss promptly with due consideration of individual comorbidities. *Am J Clin Nutr* 2014;99:875–90.

INTRODUCTION

The WHO defines a healthy body weight range for adults as a BMI (in kg/m^2) between 18.5 and 24.9 on the basis of reduced mortality risk (1). However, this range has been based primarily on studies in younger adults, for whom the risks of diabetes, cardiovascular disease, certain cancers, and mortality associated with increased body weight are well documented (2).

Because of multiple factors such as physiologic changes associated with aging, chronic disease, polypharmacy, and psychosocial changes, older adults have an increased risk of undernutrition (3), which is associated both with increased mortality (4, 5) and morbidity (6–8). Undernutrition often goes unrecognized because nutrition assessment is limited to one measure of BMI or weight. In westernized countries, it is estimated that more than two-thirds of adults aged >65 y have a BMI of ≥25 (9–11). Therefore, it is important to understand the association between BMI and mortality in the older population.

Previous reviews of weight and mortality outcomes in older adults have concluded that individuals with a BMI in the overweight range (ie, 25–29.9) had a similar or lower risk of all-cause mortality than did those in the normal-weight range (12, 13). These reviews, however, were focused on the risks associated with overweight or obesity and were less concerned with the risks associated with a BMI at the lower end of the normal-weight range. There is some evidence to suggest that not only is the upper end of the normal-weight range overly restrictive for older adults but being within the normal range may actually be associated with greater mortality (14). Therefore, we conducted a meta-analysis of longitudinal studies in community-based populations to determine all-cause mortality risk associated with BMI in those aged 65 y or older.

SUBJECTS AND METHODS

Search strategy

Relevant articles were identified through electronic searches, suggestions from colleagues based on knowledge of relevant literature, as well as hand searching of review articles. We conducted an electronic search of MEDLINE (www.ebscohost.com/academic/medline-complete) and CINAHL (www.ebscohost.com/academic/cinahl) databases as well as the Cochrane Library (www.thecochranelibrary.com/0/index.html) from 1990 to September 2013. Search term combinations were "body mass index" OR "BMI" OR "weight" and "mortality" and "old*" OR "geriatr*" OR "senior". In addition, we conducted a search of MEDLINE for review articles published between 2010 and 2013 by using search terms of "body mass index" OR obesity AND mortality NOT institute* OR hospital* or "nursing home". References from these were reviewed for potential relevant citations. Limits of age ≥65 y and English-language article were applied.

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References from reviews and selected articles were also reviewed for potential relevant citations. Only articles published in their full length were considered.

Inclusion and exclusion criteria

Studies identified were prospective cohort studies in community-living adults aged ≥65 y. Included studies reported RRs (relative risk ratios or risk ratios) or HRs and corresponding 95% CIs of all-cause mortality, had a minimum follow-up period of 5 y, and had ascertained baseline BMI and smoking status. Studies were excluded if HRs were reported only for weight in kilograms, or weight change (rather than BMI), and if they reported <3 quantitative categories of BMI. Studies in wholly nonwhite populations were also excluded. Where multiple published reports from the same study population were available, only the one with the most detailed information was included, or if similar, the most recent report. Studies were deemed suitable only if they included full details of statistical models, including the confounding factors.

Data collection

The search was conducted independently by 2 of the reviewers (JEW and NW) in April 2011, and differences were resolved by discussion with a third reviewer (CAN). The search was repeated in October 2013 to identify any additional studies meeting the inclusion criteria. Authors were contacted by e-mail if required to obtain further details of articles that met inclusion criteria. Results for each study were extracted for maximally adjusted models. The mean or median value within each category was typically not provided. For such reports, we used the midpoint as a proxy for the median for closed categories. For the open-ended categories, we estimated the median values of BMI by using data

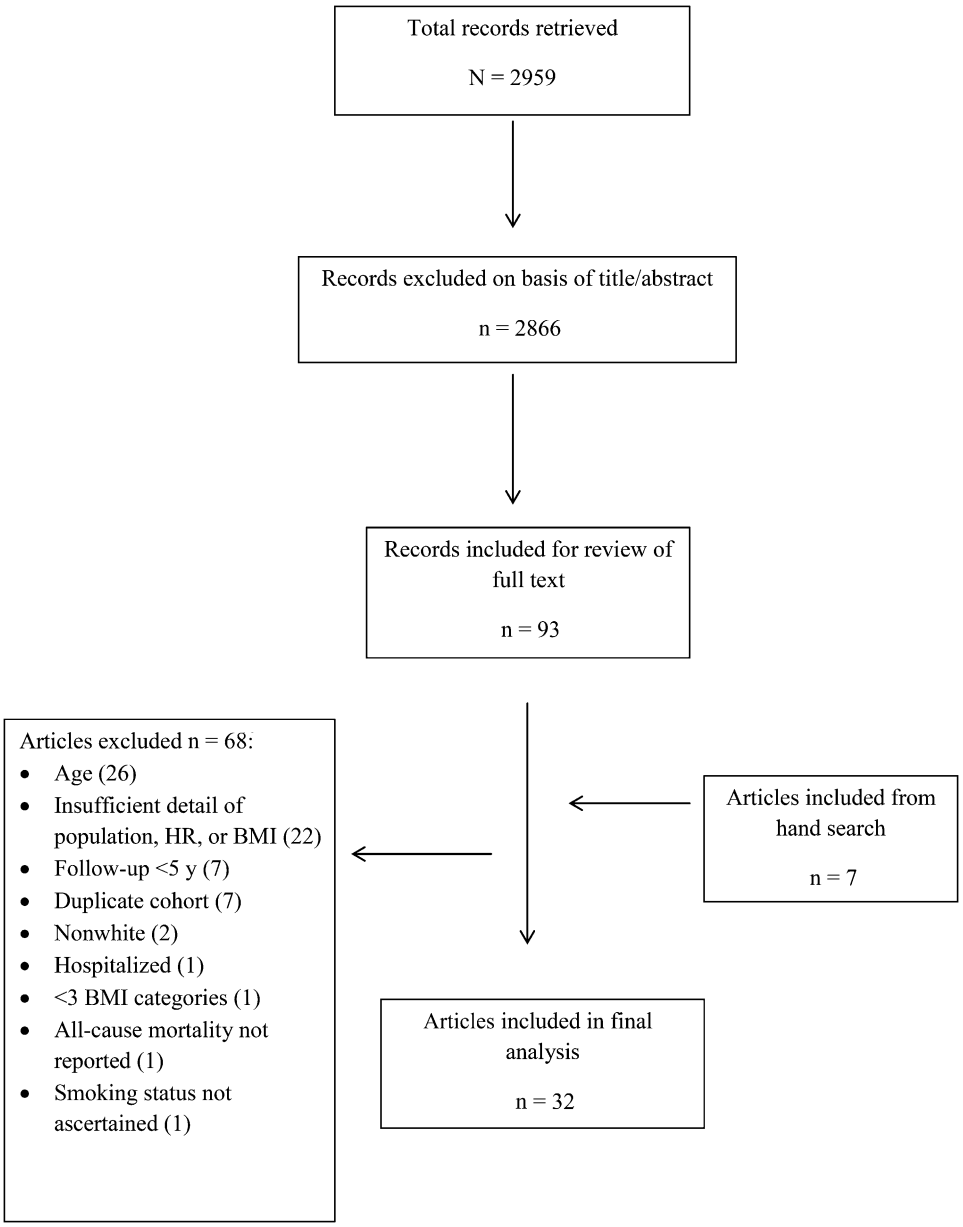

FIGURE 1. Flow diagram of the study selection and exclusion process.

TABLE 1
Details of 32 studies assessed as suitable for inclusion[1]

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
				y	*kg/m²*		
Al Snih, 2007 (21)	EPESE study, USA; cohorts from 1982–1989, 1986–1993, 1993–2000	12,725 (2019)	7 (max)	≥65	<18.5	1.53 (1.31, 1.80)	Age, sex, race, marital status, education, smoking status, comorbidity, EPESE site
					18.5–24.9	1.00	
					25.0–29.9	0.78 (0.72, 0.85)	
					30.0–34.9	0.80 (0.72, 0.90)	
					35.0–39.9	1.02 (0.84, 1.24)	
					≥40.0	1.13 (0.79, 1.60)	
Atlantis, 2010 (22)	MELSHA, Australia, 1994–2006	1000 (409)	12 (max)	≥65	<18.5	2.15 (1.15, 4.02)	Sex, age, current smoker, instrumental ADLs, timed up and go, social activity, cognitive impairment, CVD
					18.5–24.9	1.00	
					25.0–29.9	0.96 (0.77, 1.20)	
					≥30	1.04 (0.76, 1.42)	
Berraho, 2010 (23)	PAQUID cohort study, France, 1988–2001	3646 (1973)	13 (max)	≥65	<18.5	1.45 (1.17, 1.78)	Sex, age, physical activity, smoking status, comorbidity (diabetes, dyspnea, hypertension, ischemic heart disease, antecedent, stroke antecedent, number of medications)
					18.5–21.9	1.27 (1.12, 1.43)	
					22.0–24.9	1.00	
					25.0–29-9	0.98 (0.88, 1.10)	
					≥30	1.06 (0.89, 1.27)	
Berrington de Gonzalez, 2010 (24)	Cohort consortium (19 pooled cohort studies), baseline year 1970 or later	~28,466 (5160)	10 (median)	19–84; subgroup analysis: 70–84	15.0–18.4	1.65 (1.39, 1.95)	Sex, alcohol, education, marital status, physical activity (healthy nonsmokers)
					18.5–19.9	1.32 (1.15, 1.51)	
					20.0–22.4	1.06 (0.97, 1.16)	
					22.5–24.9	1.00	
					25.0–27.4	1.04 (0.96, 1.13)	
					27.5–29.9	1.15 (1.04, 1.26)	
					30.0–34.9	1.24 (1.12, 1.38)	
					35.0–39.9	1.59 (1.33, 1.90)	
					40.0–49.9	1.91 (1.44, 2.52)	
Blain, 2010 (25)	EPIDOS, France (women), 1992/1993–2001	1300 (410)	8 (max)	≥75	<18.0	1.33 (0.62, 2.83)	Age
					18–25	Ref	
					25–30	0.88 (0.71, 1.09)	
					≥30	1.18 (0.89, 1.55)	
Breeze, 2006 (26)	Male civil servants, UK, 1997–2002	4862 (1075)	5 (max)	67–96.4	<22.7	1.22 (1.00, 1.40)	Age, marital status, employment grade, smoking, alcohol, unable to do at least 1 ADL, poor physical performance, exertional symptoms
					22.7 to <24.3	1.00	
					24.3 to <26.1	0.92 (0.80, 1.10)	
					≥26.1	1.09 (0.90, 1.30)	

(Continued)

TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Corrada, 2006 (27)	Leisure World Cohort Study, USA, 1981/1982/1983/1985–2004	13,451 (11,203)	23 (max)	44–101	<18.5	1.53 (1.40, 1.67)	Age at entry, sex, smoking, "active activities," history of hypertension, angina, myocardial infarction, stroke, diabetes, arthritis, and cancer
					18.5–24.9	1.00	
					25.0–29.9	0.97 (0.93, 1.01)	
					≥30.0	1.12 (1.01, 1.24)	
Dahl, 2013 (28)	OCTO-twin, GENDER, NONA, Sweden	882 (667)	18 (max)	70–95	<25.0	Ref	Age, education, multimorbidity
					25.0–29.9	0.80 (0.67, 0.95)	
					≥30.0	0.93 (0.71, 1.22)	
de Hollander, 2012 (29)	SENECA study, 1988/1989–1998/1999	1970 (751)	10 (max)	70–75	<20.0	1.06 (0.73, 1.55)	Sex, smoking status, educational level, age at baseline
					20.0–25.0	1.00	
					25.0–30.0	0.92 (0.78, 1.09)	
					≥30.0	1.05 (0.89, 1.29)	
Dey, 2001 (30)	Sweden cohorts from 1971/1972–1986/1987, 1976/1977–1991/1992, 1981/1982–1996/1997	2590 (1333)	15 (max)	70–75	Men:		Birth cohort, smoking habits at age 70 y
					14.0–22.6	1.20 (0.96, 1.51)	
					22.7–24.6	1.07 (0.85, 1.34)	
					24.7–26.4	1.00	
					26.5–28.5	1.01 (0.81, 1.26)	
					28.6–39.2	1.19 (0.95, 1.49)	
					Women:		
					14.1–22.5	1.49 (1.14, 1.96)	
					22.6–24.5	1.16 (0.88, 1.53)	
					24.6–26.5	1.00	
					26.6–29.2	1.16 (0.88, 1.52)	
					29.3–39.8	1.25 (0.95, 1.64)	
Dolan, 2007 (31)	Study of osteoporotic fractures, USA women, 1986/1988–1994/1996	8029 (945)	8 (max)	≥65	≤22.4	1.00	Age, smoking, self-reported health, grip strength, non–thiazide diuretic use, femoral neck bone mineral density
					>22.4–24.6	0.80 (0.65, 0.96)	
					>24.6–26.7	0.70 (0.57, 0.87)	
					>26.7–29.8	0.72 (0.58, 0.89)	
					>29.8	0.89 (0.72, 1.10)	
Flicker, 2010 (32)	Health in Men Study and Australian Longitudinal Study of Women's Health, Australia, 1996–2006	9240 (2308)	10 (max)	70–75	<18.5	1.76 (1.39, 2.22)	Age, sex
					18.5–24.9	1.00	
					25.0–29.9	0.87 (0.78, 0.94)	
					≥30	0.98 (0.85, 1.11)	

(*Continued*)

TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Freedman, 2006 (33)	Radiologic technologists, USA; baseline: 1983–1989 until 2002	4572 (743) Never-smokers	19 (max)	≥65	Men:		Race-ethnicity, education, alcohol behavior, year first worked as a radiologic technologist
					18.0–24.9	1.00	
					25.0–29.9	0.77 (0.57, 1.03)	
					30.0–34.9	1.15 (0.70, 1.89)	
					≥35.0	3.16 (1.27, 7.84)	
					Women:		
					18.0–24.9	1.00	
					25.0–29.9	1.09 (0.89, 1.33)	
					30.0–34.9	1.44 (1.08, 1.92)	
					≥35.0	2.57 (1.68, 3.95)	
Gale, 2007 (34)	Department of Health and Social Security Survey, UK women, 1973/1974–1998	348 (315)	24 (max)	≥65	<18.5	1.41 (0.90, 2.38)	Age, height, smoking, social class, physical activity, diagnosed disease at baseline, calorie intake, reported weight loss, measures of body composition
					18.5–24.9	Ref	
					25.0–29.9	1.00 (0.74, 1.34)	
					≥30.0	1.14 (0.76, 1.71)	
Grabowski, 2001 (35)	Longitudinal Study of Aging, USA, 1984–1991	7527 (∼2860)	8 (max)	≥70	Men:		Age, health care use, functional limitations, sex, race, private health insurance, region of country, education, self-rated health, lives alone, need for proxy, married
					<19.0	2.00 (1.20, 3.20)	
					19.0–21.9	1.00	
					22.0–24.9	0.90 (0.70, 1.10)	
					25.0–26.9	0.90 (0.70, 1.20)	
					27.0–28.9	0.70 (0.50, 1.00)	
					29.0–31.9	0.80 (0.50, 1.10)	
					≥32.0	0.80 (0.40, 1.20)	
					Women:		
					<19.0	1.40 (1.10, 1.80)	
					19.0–21.9	1.00	
					22.0–24.9	0.90 (0.70, 1.10)	
					25.0–26-9	0.70 (0.50, 1.00)	
					27.0–28.9	0.90 (0.70, 1.20)	
					29.0–31.9	0.60 (0.40, 0.80)	
					≥32.0	0.80 (0.60, 1.10)	
Gulsvik, 2009 (36)	Bergen Clinical Blood Pressure Study, Norway, 1965–2007	788 (∼231)	Mean: 29 (women); 25 (men)	20–75; subgroup analysis: 65–74	<22.0	1.58 (1.11, 2.25)	Sex, hypertension, cholesterol, smoking, activity, CVD, diabetes, pulmonary disease, socioeconomic status
					22.0–24.9	1.00	
					25.0–27.9	0.86 (0.64, 1.16)	
					≥28.0	1.10 (0.83, 1.46)	

(*Continued*)

TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Janssen, 2007 (37)	Cardiovascular Health Study, USA, 1989–1998	4968 (1464)	9 (max)	≥65	Men:		Sex, age, race, socioeconomic status, smoking, activity, prevalent disease (diabetes, coronary heart disease, congestive heart failure, stroke, cancer)
					20.0–24.9	1.00	
					25.0–29.9	0.91 (0.78, 1.06)	
					≥30.0	0.77 (0.61, 0.97)	
					Women:		
					20.0–24.9	1.00	
					25.0–29.9	0.85 (0.71, 1.01)	
					≥30.0	0.88 (0.71, 1.10)	
Janssen, 2008 (13)	Framingham Heart Study, USA, initiated 1948	4982 (3224)	Median: 10.8 (70s), 4.8 (80s)	70–89	70s:		Sex, age, smoking, alcohol
					≤24.9	1.00	
					25.0–29.9	0.99 (0.90, 1.09)	
					≥30.0	1.22 (1.08, 1.38)	
					80s:		
					≤24.9	1.00	
					25.0–29.9	0.90 (0.78, 1.04)	
					≥30.0	0.96 (0.79, 1.15)	
Keller, 2005 (38)	Canadian Study of Health & Aging Canada, 1991–2001	539 (207)	10 (max)	≥65	<18.5	1.95 (0.61, 6.31)	Age, sex, smoking, educational level, marital status, cognitive impairment at first follow-up
					18.5–24.9	1.00	
					25.0–29.9	0.70 (0.44, 1.11)	
					≥30	0.91 (0.45, 1.86)	
Kulminski, 2008 (39)	National Long Term Care Survey, USA, 1994–2003	4791 (2956)	9 (max)	≥65	Men:		Sex, age, alcohol, smoking, heart attack, other heart problems, stroke, cancer, race, weight change
					<18.5	2.24 (1.74, 2.88)	
					18.5–21.9	1.51 (1.26, 1.80)	
					22.0–24.9	1.00	
					25.0–29.9	0.80 (0.68, 0.93)	
					30.0–34.9	0.85 (0.66, 1.08)	
					≥35.0	1.15 (0.79, 1.67)	
					Women:		
					<18.5	1.70 (1.44, 2.02)	
					18.5–21.9	1.15 (1.00, 1.31)	
					22.0–24.9	1.00	
					25.0–29.9	0.84 (0.73, 0.95)	
					30.0–34.9	0.75 (0.62, 0.89)	
					≥35.0	0.99 (0.80, 1.23)	

(Continued)

TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Kvamme, 2012 (14)	Fourth Tromso Study and HUNT study, Norway; cohorts from 1994/1995–2007, 1995/1997–2007	16,711 (7474)	9.3 (mean)	≥65	Men:		Age, study site, smoking, educational level, and marital status
					<18.5	2.32 (1.75, 3.07)	
					18.5–19.9	1.28 (1.03, 1.60)	
					20.0–22.4	1.23 (1.09, 1.38)	
					22.5–24.9	1.12 (1.02, 1.22)	
					25.0–27.4	1.00	
					27.5–29.9	1.05 (0.95, 1.15)	
					30.0–32.4	1.19 (1.05, 1.34)	
					32.5–34.9	1.31 (1.09, 1.56)	
					≥35.0	1.53 (1.21, 1.95)	
					Women:		
					<18.5	1.78 (1.37, 2.31)	
					18.5–19.9	1.57 (1.28, 1.92)	
					20.0–22.4	1.32 (1.17, 1.49)	
					22.5–24.9	1.10 (0.99, 1.22)	
					25.0–27.4	1.00	
					27.5–29.9	1.06 (0.96, 1.18)	
					30.0–32.4	1.00 (0.89, 1.13)	
					32.5–34.9	1.19 (1.03, 1.38)	
					≥35.0	1.45 (1.25, 1.67)	
Mazza, 2007 (40)	CASTEL, Italy, 1979–1991	1275 (713)	12 (max)	65–95	Men:		Age, pulmonary disease, smoking, alcohol consumption, serum total cholesterol, history of coronary heart disease, arterial hypertension, diabetes, serum creatinine
					<22.7	1.63 (1.23, 2.71)	
					22.7–24.9	1.28 (0.92, 1.73)	
					25.0–26.5	1.20 (0.85, 1.67)	
					26.6–29.0	1.16 (0.82, 1.32)	
					>29.0	1.00	
McAuley, 2009 (41)	US Veterans Exercise Testing Study, US men, 1987–2004	981 (208)	6.9 (mean)	≥65	<20.0	2.51 (1.26, 4.98)	Age, ethnicity, hypertension, cholesterol, smoking, physical activity
					20.0–24.9	1.00	
					25.0–29.9	0.66 (0.48, 0.90)	
					30.0–34.9	0.50 (0.31, 0.78)	
					≥35.0	0.44 (0.20, 0.97)	
McTigue, 2006 (42)	Women's Health Initiative Study, US women, 1993–2004	18,651 (1876)	9.9 (max), 7 y (mean)	50–79; subgroup analysis: 70–79	18.5–24.9	1.00	Age, tobacco use, education, US region, physical activity level
					25.0–29.9	0.86 (0.77, 0.96)	
					30.0–34.9	1.00 (0.87, 1.15)	
					35.0–39.9	1.16 (0.94, 1.44)	
					≥40.0	1.26 (0.92, 1.72)	

(*Continued*)

TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Miller, 2002 (43)	ALSA, Australia, 1992/1993–2000/2001	1396 (579)	8 (max)	≥70	<20	1.36 (0.94, 1.99)	Sex, age group, marital status, smoking status, self-rated health, comorbid conditions, cognitive performance, ADLs, depression
					20–25	1.00	
					>25–30	0.99 (0.82, 1.21)	
					>30	1.13 (0.86, 1.49)	
Price, 2006 (44)	UK adults; baseline data: 1995–1998	9984 (4077)	5.9 (median)	≥75	Men (nonsmoking):		Serious illness in loved one in past year, depression, cognitive impairment, unexplained recent weight loss >3.2 kg, housing type, alcohol use, former smoking
					15.9–23.0	1.00	
					>23.0–25.0	0.81 (0.69, 0.94)	
					>25.0–26.7	0.73 (0.63, 0.85)	
					>26.7–29.0	0.67 (0.57, 0.78)	
					>29.0–40.4	0.64 (0.55, 0.75)	
					Women (nonsmoking):		
					14.7–22.3	1.00	
					>22.3–24.6	0.94 (0.84, 1.05)	
					>24.6–26.8	0.74 (0.65, 0.83)	
					>26.8–29.7	0.75 (0.65, 0.87)	
					>29.7–45.2	0.72 (0.62, 0.84)	
Reis, 2009 (45)	NHANES III, USA, 1988–1994	3748 (1593)	12 (max)	30–102; subgroup analysis: ≥65	Men:		Age, race, education, smoking status, alcohol use, heart disease, stroke, respiratory disease, cancer
					<18.5	2.46 (1.39, 4.37)	
					18.5–24.9	1.00	
					25.0–29.9	0.88 (0.70, 1.11)	
					30.0–34.9	0.74 (0.54, 1.02)	
					≥35.0	0.70 (0.37, 1.34)	
					Women:		
					<18.5	2.32 (1.49, 3.62)	
					18.5–24.9	1.00	
					25.0–29.9	0.88 (0.70, 1.11)	
					30.0–34.9	0.91 (0.68, 1.21)	
					≥35.0	0.87 (0.58, 1.30)	
Stessman, 2009 (46)	Jerusalem Longitudinal Study, Israel; recruitment waves: 1990, 1998, 2005	2408 (733)	18 (max)	≥70	Men:		Perceived economic hardship, self-rated health, physical activity, smoking, ADL dependency, diagnoses of hypertension, diabetes mellitus, ischemic heart disease, and cancer
					<25.0	1.00	
					25.0–29.9	0.93 (0.63, 1.70)	
					≥30.0	1.03 (0.59, 1.79)	
					Women:		
					<25.0	1.00	
					25.0–29.9	0.55 (0.33, 0.92)	
					≥30.0	0.45 (0.25, 0.83)	

(*Continued*)

TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Tayback, 1990 (47)	NHANES I and NHEFS, USA; baseline data: 1971–1975; follow-up data: 1982–1984	2568 (792)	12 (max)	55–74; subgroup analysis: 65–74	Men:		Smoking, elevated blood pressure, age, and poverty
					<22.0	1.30 (1.00, 1.60)	
					22.0–30.0	1.00	
					>30.0	1.10 (1.00, 1.20)	
					Women:		
					<22.0	1.30 (1.00, 1.70)	
					22.0–30.0	1.00	
					>30.0	1.00 (1.00, 1.10)	
Visscher, 2004 (48)	Finland; baseline: 1973–1977	1559 (731) Never-smokers	15 (max)	65–92	Men:		Age, educational level, geographic region, alcohol use
					18.5–24.9	1.00	
					25.0–29.9	0.9 (0.7, 1.2)	
					≥30.0	1.4 (1.0, 2.0)	
					Women:		
					18.5–24.9	1.00	
					25.0–29.9	0.9 (0.7, 1.1)	
					≥30.0	0.9 (0.8, 1.2)	
Wee, 2011 (49)	Medicare Current Beneficiary Survey, USA; baseline data: 1994–2000; follow-up data: 2008	20,975 (11,093)	14 (max)	≥65	Men (white):		Baseline age, smoking status, education, proxy response, chronic health conditions
					<18.5	2.37 (1.84, 3.07)	
					18.5–21.9	1.24 (1.12, 1.37)	
					22.0–24.9	1.00	
					25.0–27.4	0.84 (0.77, 0.92)	
					27.5–29.9	0.83 (0.74, 0.92)	
					30.0–34.9	0.91 (0.82, 1.02)	
					≥35.0	1.53 (1.20, 1.95)	
					Women (white):		
					<18.5	1.75 (1.51, 2.02)	
					18.5–21.9	1.13 (1.03, 1.25)	
					22.0–24.9	1.00	
					25.0–27.4	0.88 (0.80, 0.97)	
					27.5–29.9	0.82 (0.73, 0.92)	
					30.0–34.9	0.99 (0.89, 1.10)	
					≥35.0	1.21 (1.04, 1.42)	

(Continued)

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TABLE 1 (*Continued*)

First author, year (ref)	Cohort, country (if applicable), and study period	Sample size (no. of deaths)	Years of follow-up	Age	BMI groups	HR (95% CI)	Adjusted variables
Zunzunegui, 2012 (50)	Aging in Leganes, Spain, 1993–2009	1008 (672)	16 (max), 11.6 (median)	65–101	Men:		Age, sex, education, chronic conditions, ADL disability, smoking, physical activity
					<18.5	4.79 (2.27, 10.11)	
					18.5–24.9	1.52 (1.07, 2.18)	
					25.0–29.9	1.39 (0.98, 1.96)	
					30.0–34.9	1.00	
					≥35	1.16 (0.41, 3.27)	
					Women:		
					<18.5	1.55 (0.56, 4.31)	
					18.5–24.9	0.90 (0.63, 1.27)	
					25.0–29.9	0.81 (0.59, 1.11)	
					30.0–34.9	1.00	
					≥35	1.09 (0.71, 1.68)	

[1] ADL, activity of daily living; ALSA, Australian Longitudinal Study of Ageing; CASTEL, Cardiovascular Study in the Elderly; CVD, cardiovascular disease; EPESE, Established Populations for Epidemiologic Studies of the Elderly; EPIDOS, Epidemiology of Osteoporosis Study; HUNT, North Trøndelag Health Study; max, maximum; MELSHA, Melbourne Longitudinal Studies on Healthy Ageing; NHEFS, National Health Examination Epidemiologic Follow Up Survey; OCTO-twin, octogenarian twins; PAQUID, Personnes Agées QUID; ref/Ref, reference; SENECA, Survey in Europe on Nutrition and the Elderly: a concerted action.

from NHANES I participants who were aged ≥65 y. In articles in which numbers of deaths were not specified, an estimate was determined from the number of deaths for each BMI category that would equate to the HRs and CIs stated in the minimally adjusted model.

Statistical analysis

Extracted HRs were recalibrated if the reference range did not include a BMI of 23.5. For example, if an article reported results for only 3 different categories—eg, BMI <25, 25–29.9, and ≥30—and their reference category was 25–29.9, then the HRs would be recalibrated so that the <25 group would be the reference category, as this range includes 23.5, which was our reference range value and the most common reference-range midpoint across all studies. A 2-stage random-effects meta-analysis was used to examine a potential nonlinear relation between BMI and all-cause mortality risk (15, 16). In the first stage, naturally conducted by authors of the original studies, the HRs for death were estimated and compared for various BMI groups within each trial. In the second stage, the authors of the meta-analysis combined the reported estimates of the HRs from all of the trials by using a random-effects analysis in which the trials were assumed to be a random sample from a population of trials (both actual and potential). Consequently, the resulting combined estimates of the HRs for the BMI groups take into account both the variation within trials and the variation between trials. BMI was modeled by using restricted cubic splines with 3 knots chosen at the 5th, 50th, and 95th percentiles of the distribution (15). Restricted cubic spline models were initially computed for each study taking into account the within-study correlation; afterward, a random-effects meta-analysis was performed by using the regression coefficients and the variance-covariance matrix from each individual study (17, 18). This approach allowed assessment of potential nonlinear relationships as well as statistical heterogeneity of the dose-response relation across studies. Pooled HRs for each 1-unit increment of BMI were then reported. A BMI of 23.0–23.9 was chosen as the reference. For the full analysis, studies that reported results only by subgroups of age or sex were combined by using a within-study fixed-effects meta-analysis to derive common risk estimates. Separate meta-analyses were performed stratified by sex, geographical region (North America compared with Europe), measured compared with self-reported anthropometric variables, never-smokers, exclusion of early deaths (deaths within the first 1 to 5 y of follow-up), exclusion of adjustment for intermediary factors in the obesity-mortality causal pathway (eg, hypertension, diabetes, or hyperlipidemia), and absence of preexisting disease. Nonlinearity of the meta-analysis was assessed by testing the null hypothesis that the coefficient of the second spline was equal to zero. Statistical heterogeneity was assessed by using multivariate generalization of the I^2 statistic (2 cutoff points) (18, 19).

In a separate analysis, we pooled the HRs for each study into 5 broadly defined categories of BMI (<21.0, 21.0–24.9, 25.0–29.9, 30.0–34.9, and ≥35.0) on the basis of the midpoint of the range reported, with 21.0–24.9 being chosen as the reference category because this incorporated the reference category used in the above nonlinear analysis.

Publication bias was evaluated by using funnel plots and Egger's regression test (20). All statistical analyses were performed and graphs created by using Stata version 11.2 (StataCorp).

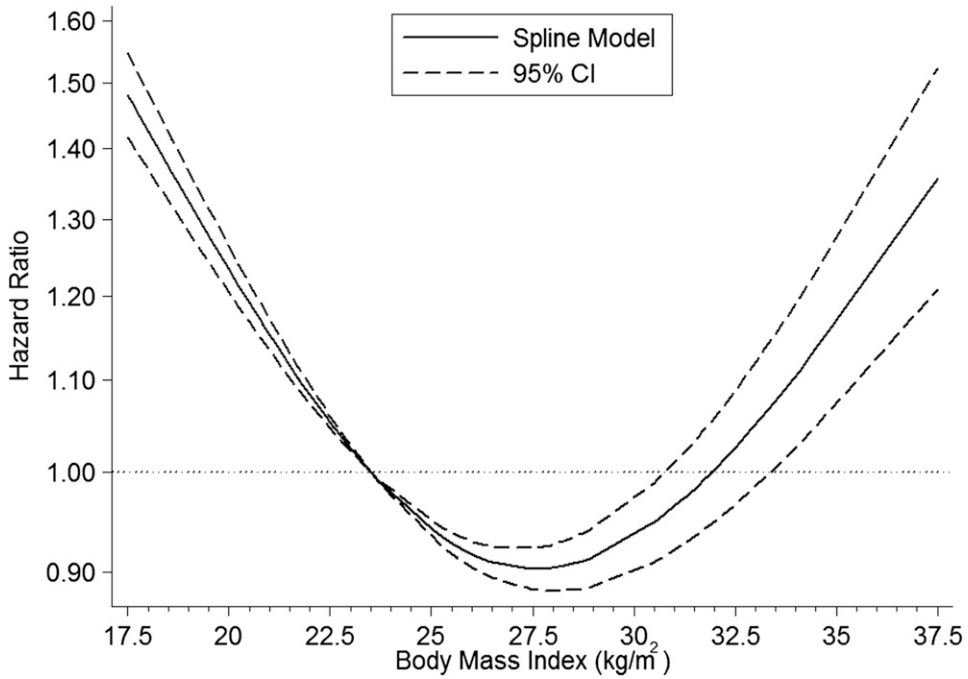

FIGURE 2. HRs (95% CIs) of all-cause mortality according to BMI for men and women aged ≥65 y. BMI was modeled with restricted cubic splines in a random-effects dose-response model. A BMI (in kg/m^2) of 23.5 (most common midpoint for the reference BMI category) was used as the reference to estimate all HRs. The vertical axis is on a log scale.

RESULTS

Study selection

The literature search identified 2959 records, 93 of which were reviewed for inclusion. After further exclusions based on our selection criteria, 32 provided sufficient information for data extraction and analysis and were deemed suitable for inclusion in the final analysis (**Figure 1**). The included studies are summarized in **Table 1** (13, 14, 21–50). In total, these studies contributed 197,940 individuals (72,469 deaths) with an average duration of follow-up of 12 y. All were population-based cohorts, which included participants from Europe, North America,

TABLE 2
Overall and subgroup HRs (95% CIs) of all-cause mortality according to BMI for adults aged ≥65 y[1]

BMI (kg/m^2)	All (32 studies)	Men (16 studies[2])	Women (17 studies[3])	Never-smokers (9 studies[4])
17.0–17.9	1.48 (1.42, 1.55)	1.66 (1.51, 1.82)	1.48 (1.41, 1.54)	1.36 (1.26, 1.45)
18.0–18.9	1.38 (1.33, 1.43)	1.51 (1.40, 1.63)	1.38 (1.33, 1.43)	1.28 (1.21, 1.36)
19.0–19.9	1.28 (1.24, 1.32)	1.38 (1.30, 1.46)	1.28 (1.24, 1.32)	1.21 (1.16, 1.27)
20.0–20.9	1.19 (1.17, 1.22)	1.26 (1.21, 1.31)	1.19 (1.17, 1.22)	1.15 (1.11, 1.19)
21.0–21.9	1.12 (1.10, 1.13)	1.16 (1.13, 1.19)	1.12 (1.10, 1.13)	1.09 (1.07, 1.12)
22.0–22.9	1.05 (1.05, 1.06)	1.07 (1.06, 1.08)	1.05 (1.05, 1.06)	1.04 (1.03, 1.05)
23.0–23.9	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
24.0–24.9	0.96 (0.96, 0.97)	0.95 (0.94, 0.96)	0.96 (0.95, 0.97)	0.97 (0.96, 0.98)
25.0–25.9	0.93 (0.92, 0.94)	0.91 (0.89, 0.92)	0.92 (0.91, 0.94)	0.95 (0.93, 0.96)
26.0–26.9	0.91 (0.90, 0.92)	0.88 (0.86, 0.91)	0.90 (0.89, 0.92)	0.94 (0.92, 0.96)
27.0–27.9	0.90 (0.88, 0.92)	0.87 (0.84, 0.90)	0.89 (0.87, 0.91)	0.94 (0.91, 0.97)
28.0–28.9	0.91 (0.88, 0.93)	0.87 (0.83, 0.91)	0.89 (0.86, 0.92)	0.96 (0.92, 1.00)
29.0–29.9	0.93 (0.90, 0.96)	0.89 (0.84, 0.94)	0.90 (0.87, 0.94)	0.99 (0.93, 1.05)
30.0–30.9	0.95 (0.91, 0.99)	0.91 (0.84, 0.97)	0.92 (0.88, 0.96)	1.02 (0.95, 1.10)
31.0–31.9	0.98 (0.93, 1.03)	0.94 (0.86, 1.02)	0.95 (0.90, 1.00)	1.06 (0.97, 1.17)
32.0–32.9	1.03 (0.97, 1.09)	0.98 (0.88, 1.09)	0.99 (0.93, 1.05)	1.12 (0.99, 1.26)
33.0–33.9	1.08 (1.00, 1.15)	1.04 (0.91, 1.18)	1.03 (0.96, 1.12)	1.20 (1.03, 1.40)
34.0–34.9	1.13 (1.05, 1.23)	1.10 (0.95, 1.28)	1.09 (1.00, 1.19)	1.28 (1.07, 1.54)
35.0–35.9	1.21 (1.10, 1.33)	1.18 (0.98, 1.41)	1.16 (1.05, 1.28)	1.36 (1.11, 1.69)
36.0–36.9	1.28 (1.16, 1.43)	1.25 (1.02, 1.54)	1.23 (1.10, 1.37)	1.45 (1.15, 1.83)
37.0–37.9	1.36 (1.21, 1.52)	1.33 (1.06, 1.66)	1.30 (1.15, 1.46)	1.53 (1.19, 1.97)

[1] BMI was modeled with restricted cubic splines in a random-effects dose-response model. A BMI (in kg/m^2) of 23.0–23.9 was used as the reference to estimate all HRs.

[2–4] Studies included in the subgroup analyses: [2]14, 26, 30, 33, 35, 37, 39–41, 44–50; [3]14, 25, 30, 31, 33–35, 37, 39, 42, 44–50; [4]24, 26, 27, 30, 31, 33, 37, 40, 48.

Canada, and Australia. One article was a pooled analysis of 19 prospective studies, including community-based populations of predominantly white adults, and reported HRs for all-cause mortality according to age (70–84 y) and BMI (24).

The authors of 3 studies were contacted to request further information; however, responses from authors indicated that these studies did not meet inclusion criteria or the required information could not be provided (51–53). Of the included studies, 22 used measured height and weight to calculate BMI, 8 used self-reports, 1 used a mix (depending on the study site), and 1 used self-reported weight and measured height. In 12 US studies with a mix of ethnicity, the majority of the subjects were white, non-Hispanic. Population cohorts of 6 studies included younger adults; subgroup analyses were presented for adults aged ≥65 y. We included one study that had a small proportion of adults aged <65 y at study entry (3% of person-years) because the authors reported that results "excluding persons under age 65 years at entry (not shown) were essentially unchanged" (27).

Association between BMI and all-cause mortality

The association between all-cause mortality and BMI was found to be U-shaped with a broad base (**Figure 2**) (P-nonlinearity < 0.001). The nadir of the curve for BMI and mortality was between 24.0 and 30.9, with the lowest risk being between 27.0 and 27.9 (HR: 0.90; 95% CI: 0.88, 0.92). Accordingly, a BMI of 21.0–21.9 had a 12% greater risk of mortality during the follow-up period compared with the reference BMI (23.0–23.9). Those individuals with a BMI of ≤20 had at least a 28% greater mortality risk than did those with a BMI between 23.0 and 23.9.

Results of analyses restricted to the subset of never-smokers (n = 51,514) shifted the mortality curve to the left, with the lowest mortality risk moving from a BMI of 27.0–27.9 to a BMI of 26.0–26.9 (HR: 0.94; 95% CI: 0.91, 0.97). The increased mortality risk at a BMI of <23.0 remained (**Table 2**). There were no notable differences in results between men and women (Table 2). Analyses of studies using only measured BMIs, no adjustment for intermediary factors, exclusion of early deaths, or populations with no preexisting disease confirmed the association of an increased risk of mortality at a BMI <23.0 and >33.0 compared with the reference of 23.0–23.9 (**Table 3**). Results stratified by geographic region were similar to those from the full analysis (results not shown).

When we pooled the HRs for broadly defined categories of BMI, the results were in concordance with those shown from the dose-response curves. Compared with the reference category (BMI of 21.0–24.9), there was a 37% increase in mortality risk associated with a BMI <21.0 (HR: 1.37; 95% CI: 1.30, 1.46) (**Figure 3**). The overall HR for the BMI range of 25.0–29.9 was 0.90 (95% CI: 0.87, 0.93), for a BMI of 30.0–34.9 the HR was 0.96 (95% CI: 0.90, 1.02), and for a BMI >35.0 the HR was 1.18 (95% CI: 1.00, 1.39).

There was substantial between-study heterogeneity for study-specific trends, defined by coefficients of the first (I^2 = 69%) and second (I^2 = 63%) spline transformations of BMI. There was little evidence of funnel plot asymmetry for each of the measures

TABLE 3
HRs (95% CIs) of subgroup analyses for all-cause mortality according to BMI[1]

				BMI	
BMI (kg/m^2)	No preexisting disease (8 studies[2])	Exclusion of early deaths: 1–5 y (14 studies[3])	No adjustment for intermediary factors (20 studies[4])	Self-reported (10 studies[5])	Measured (22 studies[6])
17.0–17.9	1.56 (1.23, 1.98)	1.43 (1.29, 1.58)	1.49 (1.41, 1.56)	1.56 (1.45, 1.68)	1.43 (1.37, 1.48)
18.0–18.9	1.44 (1.18, 1.74)	1.44 (1.23, 1.45)	1.38 (1.32, 1.44)	1.44 (1.35, 1.53)	1.34 (1.29, 1.38)
19.0–19.9	1.32 (1.13, 1.54)	1.25 (1.17, 1.34)	1.28 (1.24, 1.32)	1.32 (1.26, 1.39)	1.25 (1.22, 1.28)
20.0–20.9	1.22 (1.09, 1.36)	1.17 (1.12, 1.23)	1.19 (1.17, 1.22)	1.22 (1.18, 1.26)	1.17 (1.15, 1.20)
21.0–21.9	1.13 (1.05, 1.22)	1.10 (1.07, 1.14)	1.12 (1.10, 1.13)	1.13 (1.11, 1.16)	1.11 (1.09, 1.12)
22.0–22.9	1.06 (1.02, 1.10)	1.05 (1.03, 1.06)	1.05 (1.05, 1.06)	1.06 (1.05, 1.07)	1.05 (1.04, 1.05)
23.0–23.9	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
24.0–24.9	0.96 (0.93, 0.99)	0.97 (0.94, 0.98)	0.96 (0.96, 0.97)	0.96 (0.95, 0.97)	0.96 (0.96, 0.97)
25.0–25.9	0.93 (0.87, 0.98)	0.94 (0.91, 0.97)	0.93 (0.92, 0.94)	0.92 (0.91, 0.94)	0.93 (0.92, 0.95)
26.0–26.9	0.91 (0.84, 0.99)	0.93 (0.89, 0.96)	0.92 (0.90, 0.93)	0.90 (0.87, 0.92)	0.92 (0.90, 0.93)
27.0–27.9	0.91 (0.82, 1.01)	0.92 (0.87, 0.97)	0.91 (0.89, 0.93)	0.89 (0.85, 0.92)	0.91 (0.88, 0.94)
28.0–28.9	0.92 (0.81, 1.04)	0.93 (0.87, 0.99)	0.92 (0.89, 0.95)	0.89 (0.85, 0.94)	0.91 (0.88, 0.95)
29.0–29.9	0.95 (0.83, 1.09)	0.95 (0.88, 1.03)	0.95 (0.91, 0.98)	0.91 (0.86, 0.96)	0.93 (0.89, 0.98)
30.0–30.9	0.99 (0.85, 1.15)	0.98 (0.90, 1.07)	0.97 (0.93, 1.02)	0.93 (0.87, 1.00)	0.95 (0.90, 1.01)
31.0–31.9	1.03 (0.88, 1.22)	1.01 (0.92, 1.11)	1.01 (0.96, 1.07)	0.97 (0.89, 1.05)	0.98 (0.92, 1.06)
32.0–32.9	1.09 (0.92, 1.31)	1.05 (0.95, 1.17)	1.07 (1.00, 1.14)	1.01 (0.92, 1.11)	1.02 (0.94, 1.12)
33.0–33.9	1.19 (0.98, 1.45)	1.11 (0.99, 1.24)	1.13 (1.04, 1.22)	1.07 (0.96, 1.19)	1.08 (0.97, 1.19)
34.0–34.9	1.29 (1.05, 1.59)	1.17 (1.04, 1.32)	1.20 (1.10, 1.32)	1.13 (1.00, 1.28)	1.14 (1.01, 1.28)
35.0–35.9	1.39 (1.11, 1.73)	1.24 (1.09, 1.42)	1.29 (1.16, 1.43)	1.21 (1.05, 1.39)	1.21 (1.06, 1.38)
36.0–36.9	1.49 (1.18, 1.88)	1.31 (1.14, 1.51)	1.38 (1.22, 1.55)	1.29 (1.10, 1.50)	1.27 (1.10, 1.48)
37.0–37.9	1.59 (1.24, 2.02)	1.38 (1.19, 1.60)	1.46 (1.28, 1.67)	1.36 (1.15, 1.61)	1.34 (1.14, 1.58)

[1] BMI was modeled with restricted cubic splines in a random-effects dose-response model. A BMI (in kg/m^2) of 23.0–23.9 was used as the reference to estimate all HRs.

[2–6] Studies included in the subgroup analyses: [2]24, 26, 30, 33, 35, 39, 41, 50; [3]14, 23–25, 27, 30, 35, 38–40, 46, 47, 49, 50; [4]13, 14, 24–27, 29–33, 35, 38, 39, 42, 44, 45, 48–50; [5]21, 23, 24, 27, 32, 33, 35, 39, 49, 50; [6]13, 14, 22, 25, 26, 28–31, 34, 36–38, 40–48.

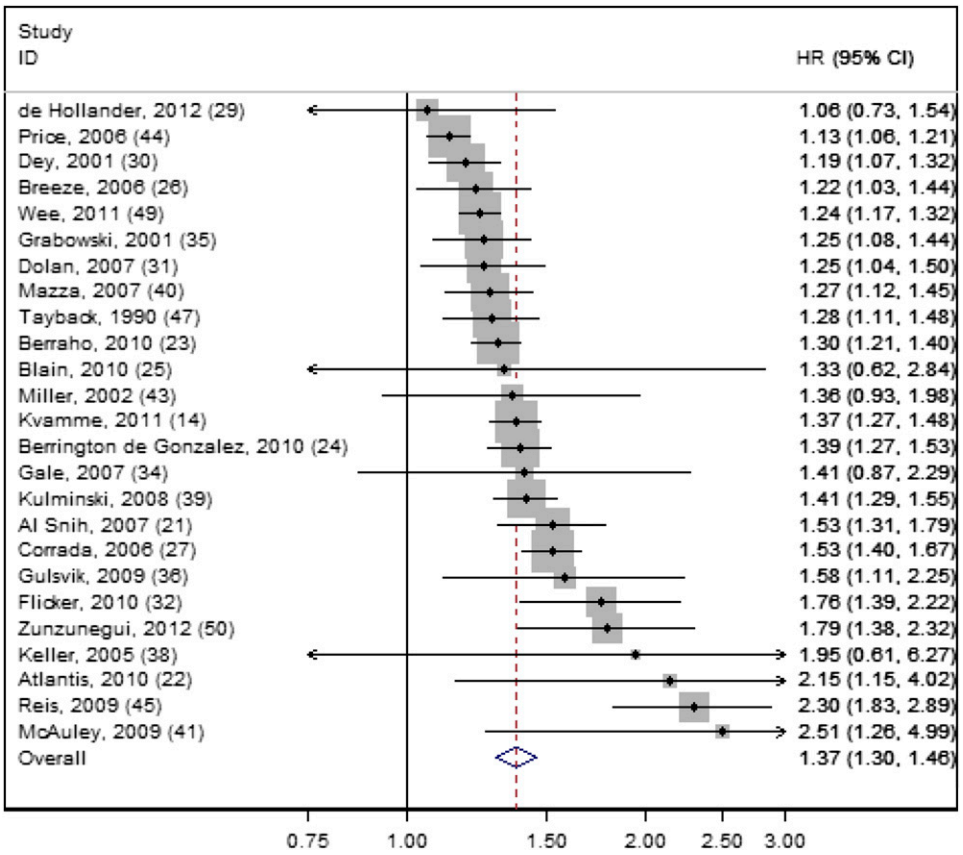

FIGURE 3. HRs (95% CIs) for a BMI (in kg/m^2) <21.0 compared with a BMI of 21.0–24.9 in relation to all-cause mortality for men and women aged ≥65 y. HRs were combined by using a random-effects model. Squares represent study-specific HR estimates (the size of the square reflects the study-specific statistical weight), horizontal lines represent 95% CIs, and the diamond represents the combined HR (95% CI). ID, identifier.

except for the BMI <21.0 category (**Figure 4**), where smaller studies tended to show stronger positive associations than did larger studies (Egger's test P = 0.03). Removal of individual studies one at a time from the analysis did not materially alter the results (data not shown).

DISCUSSION

The association between all-cause mortality and BMI for adults aged ≥65 y was found to be U-shaped with the nadir of the curve between 24.0 and 30.9. In the past, longitudinal data have shown varied results regarding BMI and mortality in older adults. In contrast to our findings, a recent large analysis of 57 studies including nearly 900,000 adults by the Prospective Studies Collaboration found that each 5-unit increase in BMI above 22.5–25 was associated with a 30% increase in mortality risk, an increase that persisted in all age groups, although the magnitude was reduced in the older age groups (54). However, <2% of the study population was aged ≥70 y. Similarly, results published from the National Cancer Institute Cohort Consortium showed that adults who were never-smokers and had a BMI of >25 had an increased risk of mortality at all ages, although for those aged ≥70 y (<7% of their population) who had a BMI of 25–27.4, the increase in mortality was <5% (24). We found a 4–10% lower mortality risk for participants in the overweight range (BMI of 25.0–29.9), with a 21% increase in mortality risk for the BMI range of 35.0–35.9. Our finding of an overweight BMI range being associated with lower mortality risk is consistent with a number of other systematic reviews and analyses. In a meta-analysis of 32 studies including individuals aged ≥65 y, Janssen and Mark (55) found that a BMI in the overweight range was not associated with an increased all-cause mortality risk, whereas a BMI in the obese range was associated with only a 10% increase in mortality risk (HR: 1.10; 95% CI: 1.06, 1.13).

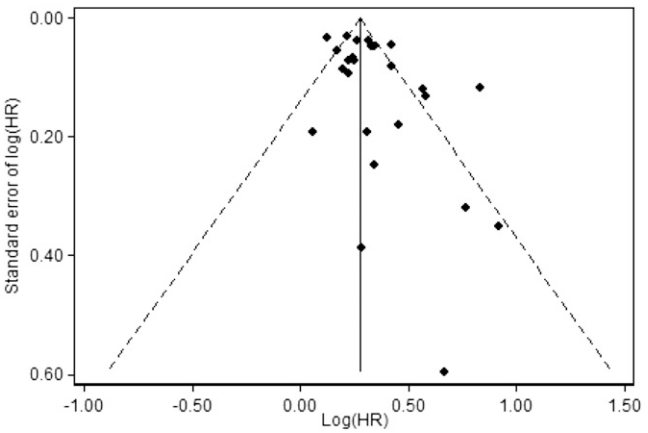

FIGURE 4. Funnel plot of studies of BMI (in kg/m^2) <21.0 compared with a BMI of 21.0–24.9 in relation to all-cause mortality for men and women aged ≥65 y. Dotted lines are pseudo 95% CIs. The large studies at the top of the plot were somewhat more symmetrically distributed than the small studies at the bottom. This indicates publication bias favoring studies with significant results.

Heiat et al (12) in an earlier systematic review found that in only 3 of the 13 articles included, a BMI of >27 was a significant prognostic factor for mortality. Similarly, the recent large meta-analysis by Flegal et al (56) showed a significant decrease in all-cause mortality for the overweight >65-y age group, although these findings have been questioned on the basis that comparison with a heterogeneous reference group may have underestimated the RRs associated with higher BMIs.

Results of cohort studies and meta-analyses that have indicated reduced mortality risk at higher BMIs and increased risk at lower BMIs among older people have been challenged on the basis of selective survival, inappropriate adjustment for intermediary factors in the causal pathway between BMI and mortality, or inadequate consideration of preexisting illness or smoking status, all of which can modify the association between BMI and mortality risk (57, 58). However, we found that none of our subgroup analyses altered the overall BMI mortality association.

The focus of most studies on the BMI-mortality relation in older adults has been on the RRs of overweight and obesity; however, our interest was on the "healthy weight range" and its suitability to older people. Importantly, we found that all-cause mortality risk started to increase at a BMI of <23.0, which falls within the WHO healthy weight range for adults (BMI: 18.5–24.9). Although slightly attenuated, this increased risk persisted when the analysis was restricted to never-smokers.

A strength of our study is the large number of individuals included with a follow-up of at least 5 y. The use of BMI as a continuous variable and exploring nonlinear associations allowed an assessment of risks at all BMI points rather than just in broad groupings and allowed us to specifically look at lower BMI points. The majority of included studies were large longitudinal cohort studies with well-documented measures and outcomes. Although some used self-reported rather than measured weight and height, this did not affect the results and death is a readily quantifiable outcome. There was some indication of publication bias in the lowest category of BMI (<21.0) where it could be that smaller studies are more likely to be published when the effect sizes are larger, studies are conducted and analyzed with less methodologic rigor, or that smaller trials were conducted in patients at higher risk. However, the only difference we observed was that in a few studies that used a wide BMI reference range in their analysis (eg, 18.5–24.9) and had a relatively large proportion of participants at the lower end of the <21.0 BMI distribution.

Our analysis has limitations in that we assessed only mortality risk associated with BMI rather than weight change or body composition, and weight change may be more important for older adults in terms of health risks. Some studies have reported that annual weight loss or gain increased the risk of all-cause mortality in older adults (30, 59, 60), and even in the presence of low or high BMI, weight stability was associated with reduced mortality risk (61). Significant body composition changes occur with age that are not captured by BMI, specifically a reduction in lean body mass and an increase in fat mass with altered distribution, and increased abdominal fat deposition may increase mortality risk. Waist circumference and waist-to-hip ratio have both been shown to have strong associations with mortality in older populations (62). We limited our analysis to all-cause mortality rather than to morbidity or cause-specific mortality, which may have different associations with BMI. We also pooled all of the published results together to determine mortality risk for adults aged ≥65 y. It is likely that for the younger age groups within this range, the risks of higher BMI are greater than those for the older age groups (>75 y). We included only predominantly white populations because the BMI mortality relation may differ according to race or ethnicity (63); however, similar results have been found in Asian populations (64). Articles generally did not include standardized assessments of physical activity, and it may be that a mix of activity levels of individuals in the BMI categories influenced our results, but few studies provided details of levels of physical activity. We have not analyzed the relation between BMI and morbidity but recognize that carrying significant excess body weight can reduce mobility in older adults, which may compromise functional capabilities. Further research is required to understand the relations between morbidity, functionality, and BMI in older adults because these outcomes are more likely to be associated with quality of life. Although there are limitations in using BMI as a measure of body composition, BMI remains the most commonly used measure of weight status across the spectrum of health care settings from acute through primary care; therefore, understanding how to interpret BMI as it relates to older adults is important to ensure that appropriate monitoring of health and nutritional risk is implemented. Dietary restrictions in older adults have been shown to be associated with an increased risk of malnutrition (65), suggesting that, in this population, imposing restrictions purely on the basis of an elevated BMI is potentially detrimental.

Our meta-analysis included only older adults living in the community, and the relation between BMI and mortality may be different for those in institutionalized or residential care who are sicker and frailer (66). However, we were interested in understanding the mortality risks associated with BMI among the "independent"- living older population, because these are the likely recipients of dietary advice based on weight status.

Overall, we found a greater mortality risk for those with a BMI of <23.0, which was not observed for the BMI in the WHO overweight range. The increased risk associated with a lower BMI persisted among never-smokers but was attenuated. The WHO healthy weight range may not be suitable for older adults, and the interpretation of BMI for this group should be in the context of other existing comorbidities and functional capacity. Monitoring weight status in those individuals with a BMI <23.0 would seem appropriate to detect weight loss promptly and address modifiable causes.

The authors' responsibilities were as follows—JEW and CAN: created the study concept and design; JEW, NW, and CAN: performed the literature search, study selection, and data extraction; RJM: performed statistical analysis; JEW: wrote the original manuscript; and CAN: had full access to all the data in the study and had final responsibility for the decision to submit for publication. All of the authors were involved in and contributed to analysis and interpretation of data and the critical revision of the final manuscript. JEW is an employee of Nestlé Health Science, Australia. None of the other authors had a conflict of interest.

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