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Research Article

Obesity in Older People With and Without Conditions Associated With Weight Loss: Follow-up of 955,000 Primary Care Patients

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Abstract

Background: Moderate obesity in later life may improve survival, prompting calls to revise obesity control policies. However, this obesity paradox may be due to confounding from smoking, diseases causing weight-loss, plus varying follow-up periods. We aimed to estimate body mass index (BMI) associations with mortality, incident type 2 diabetes, and coronary heart disease in older people with and without the above confounders.
Methods: Cohort analysis in Clinical Practice Research Datalink primary care, hospital and death certificate electronic medical records in England for ages 60 to more than 85 years. Models were adjusted for age, gender, alcohol use, smoking, calendar year, and socioeconomic status.
Results: Overall, BMI 30–34.9 (obesity class 1) was associated with lower overall death rates in all age groups. However, after excluding the specific confounders and follow-up less than 4 years, BMI mortality risk curves at age 65–69 were *U*-shaped, with raised risks at lower BMIs, a nadir between 23 and 26.9 and steeply rising risks above. In older age groups, mortality nadirs were at modestly higher BMIs (all <30) and risk slopes at higher BMIs were less marked, becoming nonsignificant at age 85 and older. Incidence of diabetes was raised for obesity-1 at all ages and for coronary heart disease to age 84.
Conclusions: Obesity is associated with shorter survival plus higher incidence of coronary heart disease and type 2 diabetes in older populations after accounting for the studied confounders, at least to age 84. These results cast doubt on calls to revise obesity control policies based on the claimed risk paradox at older ages.

Keywords: Paradox—Mortality—Overweight—BMI

From childhood to midlife, there is little question about the enormous health burden associated with obesity. However, less severe obesity (body mass index "BMI" obese-1 = 30–34.9) in older people is reportedly associated with paradoxical outcomes in those aged ≥65 years: for example, a 97 study meta-analysis found the obese-1 group had similar mortality to normal weight (BMI 18.5–24.9) and that overweight (BMI 25–29.9) groups had lower mortality ([1]). Similarly, in 3.3 million older American veterans, the overweight (60 to <70 years; hazard ratio [HR] 3.63; 95% confidence interval [CI] 3.15–4.18) and obese-1 groups (HR 3.21; 95% CI 2.79–3.69)

had a mortality advantage relative to the normal weight group (HR 5.96; 95% CI 5.18–6.86, BMI 20 to <25) (2). Dixon and colleagues (3) have argued that this "obesity risk paradox" in older people is counter to "decades of advice to avoid even modest weight gain", and that current weight control policies may be doing harm in older groups. There is therefore some urgency to clarify whether overweight or obese older people are or are not at greater risk of death and diseases related to adiposity.

BMI is the most widely used and practical clinical measure of adiposity in adults and children. In people aged 65 and older, BMI is correlated with body fat percentage measured by dual-energy x-ray absorptiometry (correlation 0.81 and 0.71 in women and men, respectively) (4). Conventional groupings of BMI were set by the U.S. National Heart, Lung, and Blood Institute (5) and the World Health Organization (6). However, these cut-points are disputed especially for those of South Asian origin (7), and older groups (8–11).

Various explanations have been offered for the obesity paradox in older people, including potential biological mechanisms (12). Smoking is associated with lower weight and markedly raised health risks and can therefore distort regression estimates, even when used to adjust models (13). At older ages, several diseases cause both weight loss and increased mortality, introducing reverse causation confounding into models (14).

To clarify obesity risks in later life, large sample sizes are needed to provide separate estimates for different subgroups of older people. We analyzed Clinical Practice Research Datalink (CPRD) data of near-complete older populations in England registered with primary care. We estimated associations in age group–specific cohorts between baseline BMI and incident type 2 diabetes, coronary heart disease (CHD), and all-cause mortality during follow-up in older people with and without the potential confounding effects of smoking and diseases associated with weight loss.

Methods

Study Design

Deidentified electronic medical record data were from CPRD (15), and included General Practitioner (GP) records linked to Hospital Episode Statistics (HES) data for admissions (available for England only) and Office for National Statistics death certificate data. Registration with GPs is nearly complete and includes patients in institutional settings. The CPRD database includes essentially all patients who are registered with CPRD participating general practices with very few patients withdrawing their data during the study period. CPRD diagnostic and outcome coding has generally high validity (16), improved further with hospital and death certificate data. CPRD has Multiple Research Ethics Committee approval (05/MRE04/87) to undertake purely observational studies, with external data linkages including HES and Office for National Statistics mortality data. The work of CPRD is also covered by NIGB-ECC approval ECC 5-05 (a) 2012. This study was approved by the Independent Scientific Advisory Committee for MHRA database research under protocol number 14_135 (2014).

All patients with BMI (usually measured) records since January 1, 2000 and registered with a CPRD practice were included (*n* = 955,031; 62% of patients), with GP record inclusion to November 17, 2014. Those with BMI measures had better survival (age, sex, and deprivation-adjusted Cox regression HR = 0.46; 95% CI 0.45–0.46) and were more likely to have chronic kidney disease, diabetes, or hypertension, but less likely to have heart failure or dementia (Supplementary Table S1).

We excluded outlier values of BMI (<14.0 and >56.5) (*n* = 6,431). We used the earliest recorded BMI within each age group 60–64, 65–69, 70–74, 75–84, and ≥85 years (termed "index" BMI for analysis), but did not include patient duplications within age-specific models. For instance, a person could be included in the 60–64 age-group model and the 70–74 age-group model if they met all the inclusion criteria. Excluding people from subsequent age-group models could result in a disproportionate number of patients who joined practices later or avoided contact with practices in the older age groups. BMI was grouped by World Health Organization cut-points but we combined obese class 2 and 3 in the 85 and older group as there were less than 200 patients.

In patients with more than one BMI measure, weight stability was derived (data available for 53.1% patients aged 60–64; 58.0% aged 65–69; 61.0% aged 70–74; 57.4% aged 75–84; and 62.7% ≥85 years). Patients were classified as substantial weight losers (lost ≥5 kg), weight gainers (gained ≥5 kg), or weight stable (loss or gain <5 kg), using the weight difference between the study index weight and the mean of all weight records recorded over the preceding 4 years. Smaller fluctuations in weight could reflect measurement errors, acute events, or minor changes due to dieting. The confounder effect of substantial weight loss was estimated in sensitivity analyses for patients with repeat BMI measures. The sample was predominantly "white" ethnicity, for example, in 65–69-year olds, 81.8% had ethnicity data of whom 95.0% were "white" and 2.3% were South Asian.

Empirical models for exclusions

To identify groups most susceptible to prior weight loss, we tested associations with 15 major diagnoses ascertained before BMI measures (see Supplementary Table S1 for details). We also identified patients having greater than 6 of 36 Rockwood frailty index conditions (17), using ResearchOne Electronic Frailty Index coding rules. In age- and gender-adjusted regression models, recent cancer (within 5 years, excluding nonmelanoma skin cancer), dementia, heart failure, and multimorbidity all yielded statistically significant odds ratios ≥1.5 for weight loss, with other conditions having ORs less than 1.4. We therefore excluded the named conditions from models, to identify a "healthier ager" subgroup. We plotted hazards for mortality for 2-year follow-up periods to guide how many years to exclude to limit reverse causality. There is no agreement on how many years of follow-up period should be excluded, for instance, some studies have used all the follow-up period (12), whereas, others have excluded up to 5 years (18).

Covariates

Smoking status was based on GP-recorded Read terms in the previous 10 years. Patients were classified as current (or recent) smokers, ex-smokers, never smokers, and not recorded. Alcohol status was based on GP-recorded Read terms and units of alcohol per week (where available) in the previous 10 years (heavy drinkers were defined as >35 units for women and >50 units for men). Patients were classified as heavy drinkers, nondrinkers, current drinkers, former drinkers, and not recorded. Relative socioeconomical status was measured by the Index of Multiple Deprivation 2007 (19), calculated on each patient's residential postal code and incorporating seven deprivation domains (income, employment, education, health, crime, barriers to housing and services, and living environment) and categorized by quintiles (1 least deprived). Calendar year was included as a covariate to account for changing trends in BMI recording and medical care during baseline selection. Physical activity was recorded in CPRD as inactive,

gentle activity, moderate activity, vigorous activity, or not recorded (most recent data preceding index BMI but up to 10 years before).

Outcomes

Outcomes were incident angina or myocardial infarction diagnoses from ICD10-coded hospital inpatient records, incident type 2 diabetes (from GP or hospital records), and mortality (from Office for National Statistics death certificate data).

Statistical Analysis

For BMI category mortality analyses, we used Cox Proportional hazards models with follow-up years as the timescale. We used spline models with four knots to estimate nonlinear associations between BMI as a continuous measure and mortality, using the exclusions previously detailed. For subsequent analyses, we had preplanned to revise the BMI groupings based on the spline point curves. We used competing risks models (accounting for mortality) for the CHD and diabetes events. The proportional hazards assumption was tested for each model using Schoenfeld residuals. Missing values for smoking and alcohol intake were multiply imputed using the chained (mlogit) multinomial logistic regression approach. We used the rate advancement periods approach to estimate the effective age of BMI exposure group [20], essentially the number of additional years of aging in the control group that would result in equivalent mortality risks to those experienced by the exposed group. Analyses were carried out using Stata statistical software (version 13.1) and R statistical software (version 3.1.2.) with packages "pspline" (version 2.37–7) and "survival" (version 1.0–16).

Results

There were 955,031 patients in analyses (n = 822,811 with complete data for covariates), with 1,540,553 patient follow-ups from some contributing in more than one age-specific analysis. The maximum follow-up was 14.9 years (mean 5.97 years, SD = 4.02).

Mean BMI was 28.2 kg/m^2 in the 60–64-year group and 24.8 kg/m^2 in the 85 and older group (Table 1, Table 2 and Supplementary Table S2). Covariate distributions also showed age-group trends with, for example, current or recent smoking declining from 34.2% in the 60–64 group to 19.7% in the 85 and older group. Substantial measured weight loss was present in 5.2% and 11.5% of the youngest and oldest groups, respectively.

Overall, 13.2% (n = 48,442) of the 65–69-year olds died during follow-up, with rates rising to 56.9% (n = 67,814) in the 85 and older age group (Supplementary Table S3). Group mean BMI decline was modestly lower with advancing age, and it was also progressively lower over the 13 years before death, especially in the final few years (Figure 1): for example, in those aged 65–69 at baseline, 34% were obese-1 13 years before death but only 24% in the year of death, while the "normal" weights increased from 23% to 39% over the same period.

Using models similar to those producing paradoxical associations, we computed Cox proportional hazards for all-cause mortality for all subjects with complete data, adjusting for age, gender, alcohol intake, smoking status, calendar year, and a measure of relative socioeconomic position. We included all follow-up data from baseline to 14.9 years (Supplementary Table S4). In the 65–69-year age group (n = 312,352 with 40,815 deaths), the obese-1 group had a lower mortality hazard than the normal BMI group (obese-1 HR = 0.91; 95% CI 0.88–0.93), and estimates were even more paradoxical for the overweight group (HR = 0.79; 95% CI 0.77–0.81). The

mortality hazards were raised (nonparadoxical) in the obese-2 group (HR = 1.07; 95% CI 1.02–1.11) and obese-3 groups (HR = 1.54; 95% CI 1.46–1.63). Paradoxical hazards were present across the other age groups studied. Incidentally, the highest HRs were for the underweight category (HR = 2.54; 95% CI 2.40–2.69 at age 65–69).

We then excluded current smokers plus patients with multimorbidity, heart failure, dementia, or recent cancer at baseline (ie, conditions most strongly associated with prior weight loss: see Methods and Supplementary Table S1) to provide estimates for the remaining group, termed "healthier agers." We plotted hazards for 2-year follow-up periods to examine the stability of estimates (Supplementary Figure S1 and Supplementary Table S5). In the 65–74 age group, the obese-1 group had a survival advantage during the first 2 years of follow-up only, but this reversed rapidly thereafter. Hazards for the other BMI categories showed similar patterns, all reaching stability after 4 years (except for the underweight group which stabilized after 6 years). In subsequent healthier ager modeling, we therefore excluded the 0–3.9 years follow-up data, to estimate stable longer-term hazards.

Healthier ager only models showed raised (nonparadoxical) mortality hazards for the obese-1 group to age 74 (Supplementary Table S4): in the 65–69 age-group HR = 1.17 (95% CI 1.11–1.23) compared to the normal BMI category. In the overweight group at age 65–69, the apparent protective effect also reversed to yield a nonsignificant difference from the conventional normal group (HR = 0.96; 95% CI 0.92–1.01). For continuous BMI, using the spline point regression (Figure 2 and Supplementary Figure S2), in the lower end of normal BMI range (18.5–22.9) hazards rise very sharply with reducing BMI. The lowest relative hazards were approximately between BMI 23 and 26.9 at ages 60–69, although a little higher in older groups. Hazards increased moderately from BMI 27–29.9 and steeply at higher BMIs in the obese range. We therefore estimated healthier ager model hazards with a comparison group of BMI 23–26.9, the lowest part of the risk curve, which produced slightly larger effect sizes than with the conventional BMI categories (Supplementary Table S6). Applying the rate advancement period approach, the healthier ager obese-1 group have mortality rates equivalent to being 1.96 years older than their chronological age (compared to lowest mortality BMI 23–26.9), with bigger age accelerations in the obese-2 (3.51 years) and obese-3 groups (7.38 years) (Supplementary Table S7). Age acceleration in the BMI 27 to 29.9 group was 0.52 of a year.

Type 2 Diabetes and CHD Events

Healthier ager models showed competing hazards for diabetes in both the obese-1 and BMI 27–29.9 overweight group were raised in all age groups (Table 3). The hazards for the 65–69 age group for the overweight and obese-1 group were HR 1.79 (1.67–1.93) and 2.68 (2.49–2.88), respectively. For CHD, competing hazards were raised in BMI 27–29.9 overweight groups in all age groups, with the obese-1 group having raised hazards to age 84 and a nonsignificant estimate in the 85 and older group (Table 3). The hazards for the 65–69 age group for the overweight and obese-1 group were HR 1.14 (1.07–1.22) and HR 1.26 (1.17–1.35), respectively.

Sensitivity Analyses

We carried out a series of sensitivity analyses for gender differences (among the several analyses, only the 70–74 age group had a significant interaction and the CIs overlapped for men and women within each BMI category), adjustment for physical activity measures (Supplementary Table S8), restricting estimates to the "white" group (Supplementary Table S9), with measured weight change only

Table 1. Characteristics of the Sample (complete cases with no missing data on model covariates)

	Age Group (y)				
	60–64	65–69	70–75	75–84	85 and Older
N	340,753	312,352	265,912	278,422	96,498
Follow-up years, mean (SD)	5.7 (3.9)	5.5 (3.9)	5.5 (3.8)	5.3 (3.6)	3.6 (2.7)
Age years, mean (SD)	61.8 (1.4)	66.6 (1.4)	71.6 (1.4)	77.7 (2.7)	87.1 (2.6)
Women, n (%)	173,747 (51.0)	156,075 (50.0)	136,522 (51.3)	153,563 (55.2)	60,713 (62.9)
BMI (kg/m²), mean (SD)	28.2 (5.4)	28.0 (5.3)	27.6 (5.1)	26.7 (4.9)	24.8 (4.5)
Alcohol status, n (%)					
Nondrinker	40,021 (11.7)	40,431 (12.9)	39,304 (14.8)	49,391 (17.7)	20,462 (21.2)
Current drinker	220,992 (64.9)	201,070 (64.4)	171,597 (64.5)	180,801 (64.9)	60,220 (62.4)
Ex-drinker	11,428 (3.4)	12,223 (3.9)	11,878 (4.5)	13,417 (4.8)	6,520 (6.8)
Heavy drinker	68,312 (20.1)	58,628 (18.8)	43,133 (16.2)	34,813 (12.5)	9,296 (9.6)
Smoking status, n (%)					
Never	143,886 (42.2)	128,225 (41.1)	110,662 (41.6)	123,469 (44.4)	45,502 (47.2)
Current smoker	116,548 (34.2)	100,612 (32.2)	78,108 (29.4)	68,180 (24.5)	19,026 (19.7)
Ex-smoker	80,319 (23.6)	83,515 (26.7)	77,142 (29.0)	86,773 (31.2)	31,970 (33.1)
Index of multiple deprivation					
Quintile 5 (most deprived), n (%)	43,081 (12.6)	38,751 (12.4)	33,356 (12.5)	35,816 (12.9)	12,216 (12.7)
Diagnoses at baseline, n (%)					
Recent cancer (<5 years)	13,153 (3.9)	16,156 (5.2)	17,418 (6.6)	21,681 (7.8)	8,999 (9.3)
Dementia	685 (0.2)	1,177 (0.4)	2,271 (0.9)	7,190 (2.6)	8,076 (8.4)
Heart failure	5,699 (1.7)	8,782 (2.8)	11,979 (4.5)	21,139 (7.6)	14,021 (14.5)
Diabetes	39,879 (11.7)	45,137 (14.5)	44,521 (16.7)	45,671 (16.4)	16,558 (17.2)
Coronary heart disease	20,457 (6.0)	25,471 (8.2)	27,818 (10.5)	34,572 (12.4)	16,296 (16.9)
Electronic frailty index (score ≥ 6)	15,072 (4.4)	23,702 (7.6)	32,649 (12.3)	52,477 (18.9)	34,940 (36.2)
Weight stable* (weight loss or gain of 0 to <5.0 kg)	135,740/180,926 (75.0)	139,237/181,302 (76.8)	128,079/162,158 (79.0)	125,986/159,693 (78.9)	45,470/60,463 (75.2)
Weight loss of ≥5 kg*	17,699/180,926 (9.8)	18,248/181,302 (10.1)	16,869/162,158 (10.4)	19,345/159,693 (12.1)	11,063/60,463 (18.3)
Weight gain of ≥5 kg*	27,487/180,926 (15.2)	23,817/181,302 (13.1)	17,210/162,158 (10.6)	14,362/159,693 (9.0)	3,930/60,463 (6.5)

BMI = body mass index.

*Cell contents: number/subgroup %. Weight stability measures available for 53.1% of the 60–64 age group, 58.0% for the 65–69 age group, 61.0% for the 70–74 age group, 57.4% of the 75–79 age group, and 62.7% for the 85 and older age group.

Table 2. BMI Category Distribution by Age Group

BMI (kg/m², n (%))	Age Group (y)				
	60–64	65–69	70–75	75–84	85 and Older
Underweight: 14.0 to <18.5	3,882 (1.1)	3,917 (1.3)	4,443 (1.7)	7,692 (2.8)	6,260 (6.5)
Normal weight: 18.5 to <25.0	93,428 (27.4)	86,315 (27.6)	78,498 (29.5)	100,107 (36.0)	46,396 (48.1)
Overweight: 25.0 to <30.0	135,154 (39.7)	126,911 (40·6)	109,126 (41.0)	109,431 (39.3)	31,720 (32.9)
Obese-1: 30.0 to <35.0	72,102 (21.2)	64,981 (20.8)	52,292 (19.7)	45,264 (16.3)	9,823 (10.2)
Obese-2: 35.0 to <40.0	24,888 (7.3)	21,247 (6.8)	15,827 (6.0)	12,201 (4.4)	1,891 (2.0)
Obese-3: ≥40.0	11,299 (3.3)	8,981 (2.9)	5,726 (2.2)	3,727 (1.3)	408 (0.4)

BMI = body mass index.
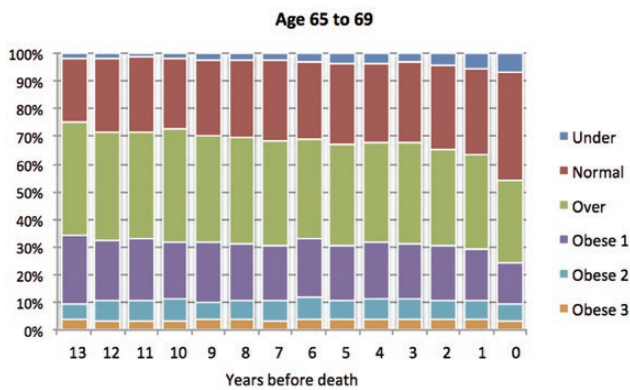
Figure 1. Cross-sectional estimates of the percentage of subjects by conventional body mass index category and number of years to death, for the 65–69 age group.
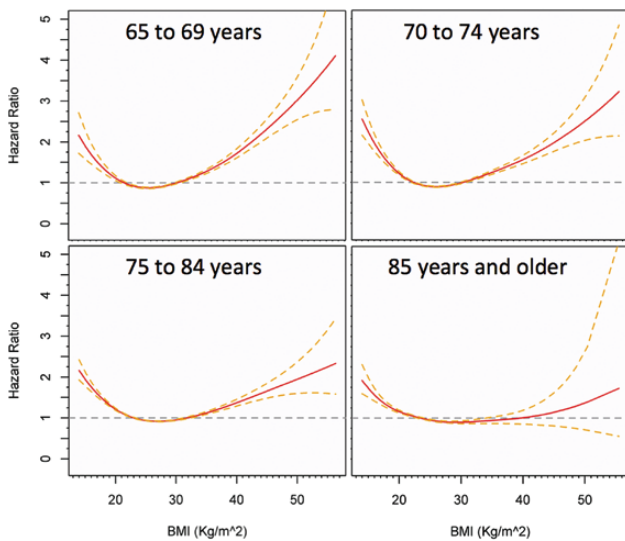

Figure 2. Spline point estimates for continuous body mass index by age group for the "healthier agers."

(Supplementary Table S10), excluding measured weight losses of ≥2.5 kg (rather than >5 kg) (Supplementary Table S10), and multiple imputation for smoking and alcohol intake (Supplementary Table S11). Results were little changed. Estimated hazard for mortality for smokers plus patients with multimorbidity, heart failure, dementia, or recent cancer (Supplementary Table S4) were markedly paradoxical for overweight and obese-1 groups. Paradoxical associations for the obese-1 (age group 65–69 HR 0.77; 95% CI 0.74–0.81) and obese-2 groups (age group 65–69 HR 0.86; 95% CI 0.81–0.92) for mortality were found in current smokers only models. There were lower risks for mortality for the obese-1 (age group 65–69 HR 0.68; 95% CI 0.64–0.71) and obese-2 groups (age group 65–69 HR 0.75; 95% CI 0.70–0.81) in models that included only patients with conditions associated with weight loss.

Discussion

Several analyses have reported that older overweight and moderately obese subjects have better or similar survival to normal BMI groups, apparently undermining the scientific rationale for some responses to the global obesity epidemic. In models ignoring suggested confounding, we obtained similar paradoxical estimates. However, in models focused on healthier agers (ie, nonsmoking and free of disease-associated weight loss) obesity class-1 was associated with increased hazards for all-cause mortality, CHD, and diabetes compared to risk nadir, at least to age 85 and older. For healthier agers, therefore, our results do not support calls to revise policies to reflect the claimed obesity risk paradox in the general older population. At age 65, healthy agers have long life expectancies (women 21.0 years, men 18.5, for England (21)) during which gains from optimized weight control could be enjoyed. Our evidence on being overweight at older ages is mixed, but BMI >27 was associated with modestly increased mortality at the younger studied ages compared to the lowest risk BMI range of 23–26.9.

Analyzing clinical records offers advantages (eg, large samples, near-complete population inclusion, clinically recorded diagnoses plus no loss to follow-up in outcome ascertainment) but recording of risk factors can be incomplete or triggered by clinical events. This problem is somewhat reduced here as GPs were offered financial incentives to record cardiovascular risk measures in the time period included in our analyses.

There are no data on whether weight loss was intentional or not, but a 1996 study in British primary care found that 18% of 56 to 75-year olds experienced any perceived weight loss in the previous 4 years, with only 4% citing personal reasons unrelated to health concerns or physician advice (22). Our exclusion of diseases empirically most strongly associated with measured weight loss is systematic but incomplete: in the weight change subgroup (age 65–69), 25.1% of the patients with greater than 5 kg weight loss would remain in the analysis despite the healthier ager model disease exclusions. This residual confounding may explain the paradoxical estimates in the

Table 3. Competing Sub-Hazard Ratios for Incident Coronary Heart Disease and Type 2 Diabetes for "Healthier Agers"

	Age Group (y)				
	60–64*	65–69*	70–74*	75–84*	85 and Older*,†
Coronary heart disease					
Underweight: 14.0 to <18.5	0.65 (0.38–1.09)	0.96 (0.68–1.37)	0.85 (0.64–1.12)	0.79 (0.65–0.95)	0.60 (0.43–0.85)
Low-normal‡,§: 18.5 to <23.0	0.79 (0.70–0.89)	0.84 (0.76–0.93)	0.91 (0.83–0.99)	0.89 (0.83–0.95)	0.97 (0.85–1.11)
Reference: 23.0 to <27.0	1	1	1	1	1
Overweight‡,§: 27.0 to <30.0	1.16 (1.07–1.26)	1.14 (1.07–1.22)	1.12 (1.05–1.19)	1.10 (1.03–1.16)	1.22 (1.05–1.42)
Obese-1: 30.0 to <35.0	1.25 (1.15–1.36)	1.26 (1.17–1.35)	1.19 (1.11–1.28)	1.11 (1.04–1.19)	1.03 (0.84–1.26)
Obese-2: 35.0 to <40.0	1.44 (1.28–1.62)	1.30 (1.16–1.45)	1.17 (1.04–1.32)	1.10 (0.97–1.25)	1.12 (0.74–1.67)
Obese-3: ≥40.0	1.49 (1.26–1.77)	1.21 (1.02–1.45)	0.97 (0.77–1.21)	1.22 (0.97–1.55)	_
Type 2 diabetes					
Underweight: 14.0 to <18.5	0.19 (0.07–0.51)	0.27 (0.13–0.56)	0.32 (0.18–0.55)	0.45 (0.32–0.64)	0.68 (0.39–1.18)
Low-normal‡,§: 18.5 to <23.0	0.50 (0.44–0.59)	0.56 (0.49–0.64)	0.61 (0.54–0.69)	0.65 (0.58–0.72)	0.77 (0.60–0.98)
Reference: 23.0 to <27.0	1	1	1	1	1
Overweight‡,§: 27.0 to <30.0	1.83 (1.70–1.98)	1.79 (1.67–1.93)	1.55 (1.43–1.67)	1.48 (1.36–1.60)	1.53 (1.20–1.97)
Obese-1: 30.0 to <35.0	3.05 (2.83–3.28)	2.68 (2.49–2.88)	2.16 (2.00–2.34)	1.98 (1.81–2.16)	1.41 (1.02–1.95)
Obese-2: 35.0 to <40.0	4.43 (4.04–4.87)	3.66 (3.32–4.05)	3.18 (2.84–3.56)	2.55 (2.20–2.94)	3.88 (2.47–6.08)
Obese-3: ≥40.0	5.59 (4.92–6.34)	4.68 (4.07–5.38)	3.18 (2.61–3.87)	2.19 (1.62–2.95)	_

*Cell contents: events/number, Sub-Hazard Ratios (SHR) (95% CI).

†In the 85+ group, obese-2 and obese-3 are combined.

‡Revised low-normal§ BMI: 18.5 to <23.0, reference = 23.0 to <27.0, revised-overweight§ = 27.0 to <30.0.

oldest old and in the first years of follow-up, and may have resulted in some underestimation of the risks of being overweight.

Our results are difficult to compare with previous work, as most reports were based on smaller samples of older volunteers, with varying groupings of BMI and varying follow-ups. Also, most reports relate to patients who were less exposed to modern cardiovascular and diabetes interventions. Lu and colleagues (2) recently reported an analysis of 3.3 million patients admitted to Veterans Administration hospitals, and those aged 60–69 years old in obese class 1 had markedly lower mortality compared to normal BMI, but no subgroup analysis excluding smokers and prior weight loss was reported. A recent meta-analysis in older people reported increasing mortality hazards at BMIs greater than 33 kg/m² for a pooled 65 and older age group, a substantially higher threshold for increased hazards than in our estimates (11). Berrington and colleagues pooled 19 studies and excluded smokers and those with cancer, heart disease or aged ≥85 years, yielding relatively small numbers of deaths to analyze (2,754 and 546 deaths in obese-1 aged 60–69 and 70–84, respectively), but reported similarly raised hazards for mortality in their obese-1 older group (23). Our result are also broadly similar to an earlier meta-analysis of 57 studies (median recruitment year 1979, mean baseline age 46 years, 2% of the subjects aged ≥70 at baseline) although this reported that the lowest mortality risk, after excluding the first 5 years of follow-up, was within the BMI range 22.5–25 kg/m² (18).

We have shown that there are substantially raised absolute death rates in later life in obese groups at least to age 84, and also raised risks of diabetes and CHD. In addition, obesity is associated with substantial excess disability and mobility limitations (24,25). Stenholm and colleagues reported that obese (BMI ≥ 30 kg/m²) men and women aged 70–79 years from the Health, Aging and Body Composition Study had an increased risk of mobility limitation during a 6.5-year follow-up period (26). Obese men and women aged 65 years and older from the English Longitudinal Study of Ageing were reported to have an increased risk of self-reported difficulties with activities of daily living and with a measure of functional impairment during a 5-year follow-up period (27). Basing calls for revising current obesity control policies on the claimed obesity risk paradox in the general older population is therefore inappropriate. Clinical advocacy of weight control for general health risk reduction was never claimed to be relevant to those already suffering from conditions associated with weight loss.

Further work is needed to clarify whether the apparently protective effects of being obese in smokers and those with diseases causing weight loss represents real protective effects (sometimes referred to as the obesity paradox in chronic disease) or whether BMI in such groups is a measure of disease severity. Further research is required into dynamic changes in BMI with mortality, especially in the oldest age groups. Further work is also needed on whether more specific measures of adipose tissue mass in older people add significantly to risk estimation for targeting of interventions. Revision of normal ranges for BMI in later life would be useful, as the classification includes groups at BMIs below 23, which are associated with substantially increased mortality in older groups.

Conclusions

In this large population-based older cohort studying longer-term outcomes, our results show that obesity is associated with shorter survival in older people who do not have the studied confounding factors, at least to age 84. These results cast doubt on calls to revise obesity control policies to reflect the claimed obesity risk paradox in the general older population. The conventional normal BMI category appears too broad for older people as it includes BMIs below 23, which are associated with higher mortality.

Supplementary Material

Please visit the article online at http://gerontologist.oxfordjournals.org/ to view supplementary material.

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Conflict of Interest

The authors' have no conflicts of interest to declare.

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