Edmonton Obesity Staging System: association with weight history and mortality risk

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Abstract: We sought to determine whether the Edmonton Obesity Staging System (EOSS), a newly proposed tool using obesity-related comorbidities, can help identify obese individuals who are at greater mortality risk. Data from the Aerobics Center Longitudinal Study ($n = 29\,533$) were used to assess mortality risk in obese individuals by EOSS stage (follow-up (SD), 16.2 (7.5) years). The effect of weight history and lifestyle factors on EOSS classification was explored. Obese participants were categorized, using a modified EOSS definition, as stages 0 to 3, based on the severity of their risk profile and conditions (stage 0, no risk factors or comorbidities; stage 1, mild conditions; and stages 2 and 3, moderate to severe conditions). Compared with normal-weight individuals, obese individuals in stage 2 or 3 had a greater risk of all-cause mortality (stage 2 hazards ratio (HR) (95% CI), 1.6 (1.3–2.0); stage 3 HR, 1.7 (1.4–2.0)) and cardiovascular-related mortality (stage 2 HR, 2.1 (1.6–2.8); stage 3 HR. 2.1 (1.6–2.8)). Stage 0/1 was not associated with higher mortality risk. Lower self-ascribed preferred weight, weight at age 21, cardiorespiratory fitness, reported dieting, and fruit and vegetable intake were each associated with an elevated risk for stage 2 or 3. Thus, EOSS offers clinicians a useful approach to identify obese individuals at elevated risk of mortality who may benefit from more attention to weight management. Further research is necessary to determine what EOSS factors are most predictive of mortality risk, and whether these findings can be generalized to other obese populations.

Key words: obesity treatment, risk stratification, weight cycling, weight loss.

Résumé : Cette étude se propose de vérifier si le nouvel instrument incorporant les comorbidités associées à l'obésité, soit le système edmontonien de stadification de l'obésité (EOSS), facilite l'identification des personnes obèses à plus haut risque de décès. On utilise les données de l'étude longitudinale du *Aerobics Center* ($n = 29\,533$) pour évaluer le risque de décès des personnes obèses en fonction du stade EOSS (suivi sur 16,2 ± 7,5 ans). On analyse en outre l'effet des antécédents de problème de poids et des facteurs associés au mode de vie sur la classification de l'EOSS. Les participants obèses sont classés au moyen d'une définition modifiée de l'EOSS en trois stades définis par la gravité du risque et d'autres conditions : stade 0 (aucun facteur de risque ni comorbidité), stade 1 (conditions légères) et stades 2 et 3 (conditions modérées à graves). Comparativement à des personnes de poids normal, les obèses aux stades 2 et 3 ont un plus haut risque de décès, toutes causes confondues (stade 2 : ratio de risques (HR, I de C de 95 % = 1,6 (1,3–2,0); stade 3 : HR = 1,7 (1,4–2,0)) et décès d'origine cardiovasculaire (stade 2: HR = 2,1 (1,6–2,8); stade 3: HR = 2,1 (1,6–2,8)). Les stades 0 et 1 ne sont pas associés à un plus haut risque de décès. Les variables suivantes sont toutes associées à un risque élevé aux stades 2 et 3 : poids souhaité et autoattribué moindre, poids à l'âge de 21 ans, condition physique cardiorespiratoire, diète antérieure autorapportée, consommation de fruits et légumes. En conclusion, l'EOSS procure aux cliniciens une approche utile pour identifier les personnes obèses présentant un risque élevé de décès et qui ont potentiellement besoin de plus d'attention sur le plan de la gestion du poids. Il faut faire d'autres études pour identifier les facteurs de l'EOSS davantage associés au risque de décès et pour généraliser, le cas échéant, ces observations à d'autres populations d'obèses.

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Introduction

Body mass index (BMI) is the most widely used measure of adiposity, but variation exists in terms of the comorbidities present and the mortality risk observed at a given BMI (Brochu et al. 2001; Calle et al. 1999; Dvorak et al. 1999; Sims 2001). In light of this, it has been suggested that not all obese individuals are at increased health risk (Brochu et al.

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2001; Dvorak et al. 1999; Sims 2001), and therefore may not require weight loss. This is in contrast to a recent observation demonstrating that obese individuals with or without the metabolic syndrome are at a similarly elevated level of mortality risk (Kuk and Ardern 2009). This discrepancy highlights the fact that using metabolic risk factors alone as a risk-stratification system may not be sufficient to identify obese individuals who are at elevated mortality risk, and that a unique risk-stratification system specifically for obese individuals is required.

The Edmonton Obesity Staging System (EOSS) is a novel risk-stratification system that classifies obese individuals into 5 graded categories, based on their morbidity and health-risk profile. All patients can be provided weight-management advice; however, patients in the first 2 stages (EOSS stages 0 and 1) may not necessarily require weight loss, as they represent an obese phenotype with relatively minor health problems (Sharma and Kushner 2009). This is in contrast to the typical obese phenotype that is associated with several clinical metabolic, mental, and physiological aberrations (EOSS stages 2 to 4) (Sharma and Kushner 2009).

It is currently unclear why some obese individuals do not experience any (or very minor) metabolic aberrations, despite high levels of adiposity. Weight history, as in the length of obesity (Brochu et al. 2001; Janssen et al. 2004; Wannamethee and Shaper 1999), and weight cycling (Blair et al. 1993) may alter one's health profile. Lifestyle and behavioral factors, such as diet (Brunner et al. 2008; Yusuf et al. 2004) and physical activity (Yusuf et al. 2004) habits, may also explain these discrepancies in metabolic aberrations across the spectrum of obesity. This information will provide valuable insight to clinicians so that they may better advise and treat their obese patients.

Therefore, the primary purpose of this study was to examine the association between EOSS and mortality risk. Second, we aimed to examine whether weight history and lifestyle factors differentiate risk among those with elevated EOSS scores.

Materials and methods

Participants

This study consisted of data from the Aerobics Center Longitudinal Study, a cohort of participants who attended the Cooper Clinic (Dallas, Tex.) for periodic self- or physician-referred medical examinations between 1987 and 2001. All study procedures and medical and behavioral questionnaires used in this analysis were consistent from 1987 to 2001. Participants were predominantly non-Hispanic white (>99%), well educated, and of middle and higher socioeconomic status (Blair et al. 1989). Inclusion criteria for the EOSS analysis required participants to have a body mass index (BMI) of 30 kg·m^{-2} or higher and available data for at least half of the EOSS criteria for each stage listed in Table 1 (3 of 6 stage 1 criteria; 9 of 18 stage 2 criteria; and 6 of 11 stage 3 criteria). EOSS stage 0 was defined as the absence of stage 1, 2, and 3 criteria. This resulted in 5 453 men and 771 women being available for EOSS grading. To determine whether individuals in EOSS stages 0 to 3 were at elevated risk for all-cause mortality, compared with a cohort that does not require weight loss, an additional 23 309 normal-weight individuals

(BMI, 18.5 to 24.9 kg·m^{-2}) were included as a comparison group; overweight individuals were excluded from the analyses. All study participants gave their informed written consent prior to participation in the examination, and the study was reviewed and approved annually by The Cooper Institute Institutional Review Board.

Mortality ascertainment

The National Death Index was used to identify deaths and cause of death, and official death certificates were cross-referenced with the participant's clinical record to confirm a match. Mortality follow-up was conducted up to December 31, 2003. Cardiovascular disease (CVD) mortality was defined by International Classification of Diseases, Ninth Revision (ICD-9) codes 390 to 448 before 1999, and Tenth Revision (ICD-10) codes I00 to I78 from 1999 to 2003. Coronary heart disease (CHD) mortality was defined by ICD-9 codes 410 to 414 and 429.2 and ICD-10 codes I20 to I25. Cancer mortality was defined by ICD-9 codes 140 to 208 and ICD-10 codes C00 to C97.

Clinical data

Participants completed self-reported personal health histories (doctor-diagnosed hypercholesterolemia and hypertriglyceridemia), a standardized medical examination by a physician, and a maximal Balke treadmill exercise test. Resting blood pressure was measured with the participant in the seated position, and was recorded as the first and fifth Korotkoff sounds, using auscultatory methods, after at least 5 min of sitting quietly. Two or more readings separated by 2 min were averaged. If the first 2 readings differed by more than 5 mm Hg, additional readings were obtained and averaged. Fasting blood was taken after an overnight fast. Cardiorespiratory fitness was categorized into sex- and age-specific quintiles, as described elsewhere (Blair et al. 1995). Participants were categorized as low fit if they were in the lowest 20% for their age- and sex-specific maximal oxygen consumption (mL·kg^{-1}min^{-1}). BMI was calculated from measured weight and height. Smoking habits were categorized as current smoker, previous smoker, or never smoker, based on self-reported smoking history.

EOSS

EOSS level was categorized on the basis of the highest-stage risk factor present for each individual, according to the operational criteria defined in Table 1. For example, an individual with normal fasting glucose, normal lipids, no psychopathology, no functional limitations (stage 0), but with diagnosed hypertension (stage 2), would be categorized as EOSS stage 2. Similarly, an individual with borderline hypertension, impaired fasting glucose (stage 1), osteoarthritis, and anxiety disorder (stage 2) would also be categorized as EOSS stage 2.

Questionnaires — weight history and diet

Participants completed a comprehensive behavioural and health history assessment questionnaire that included questions on weight history (the number of 5, 10, 20, 30, 50, and 100 lb (2.3, 4.5, 9.1, 13.6, 22.7, and 45.4 kg) weight losses over their lifetime), maximum adult weight, age at maximum weight, lowest adult weight, weight at age 21, preferred body weight ("What do you consider a good weight for your-

Table 1. Edmonton Obesity Staging System (EOSS) definition.

Stage	Conceptual description (Sharma and Kushner 2009)	Modified study definition*
0	No apparent obesity-related risk factors (e.g., BP, serum lipids, fasting glucose, etc., within normal range), no physical symptoms, no psychopathology, no functional limitations and (or) impairment of well being	No reported EOSS factors
1	• Presence of obesity-related subclinical risk factors (e.g., borderline hypertension, impaired fasting glucose, elevated liver enzymes, etc.) • Mild physical symptoms (e.g., dyspnea on moderate exertion, occasional aches and pains, fatigue, etc.) • Mild functional limitations • Mild psychopathology and (or) • Mild impairment of well being	• BP $\geq$130/85 and (or) <125/75 mm Hg for individuals with T2D • Fasting glucose $\geq$100 and <125 mg·dL^{-1} • Cholesterol $\geq$200 and <240 mg·dL^{-1} • Triglycerides $\geq$150 and <200 mg·dL^{-1} • HDL <60 mg·dL^{-1} • Shortness of breath during physical activity
2	• Presence of established obesity-related chronic disease (e.g., hypertension, type 2 diabetes, sleep apnea, osteoarthritis, reflux disease, polycystic ovary syndrome, anxiety disorder, etc.) • Moderate limitations in activities of daily living and (or) • Moderate impairment of well being	• Diagnosed hypertension or hypertension medication • BP $\geq$140/90 mm Hg or 130 /80 for individuals with T2D • T2D • Fasting glucose $\geq$125 mg·dL^{-1} • Diagnosed hypercholesterolemia • Cholesterol $\geq$240 mg·dL^{-1} • Diagnosed hypertriglyceridemia • Triglycerides $\geq$200 mg·dL^{-1} • HDL < 40 mg·dL^{-1} • Gout • Depression • Fatigue • Urinary leakage • Low back pain • Joint stiffness • Reported emotional outlook of "generally sad," or • Self-reported health of "fair"
3	• Established end-organ damage (e.g., myocardial infarction, heart failure, diabetic complications, incapacitating osteoarthritis, etc.) • Significant psychopathology • Significant functional limitations and (or) • Significant impairment of well being	• Reported chest pain • Chest pain during exercise • Heart attack • Calf pain during exercise • Stroke • Shortness of breath when sleeping • Shortness of breath when sitting • Psychiatric or psychological counseling, or • Moderate or severe cardiomegaly • Reported emotional outlook of "often depressed", or • Self-reported health of "poor"
4	• Severe (potentially end-stage) disabilities from obesity-related chronic diseases • Severe disabling psychopathology • Severe functional limitations and (or) • Severe impairment of well being	This stage was not examined as these factors were not available in the ACLS database

Note: BP, blood pressure; HDL, high-density lipoprotein, T2D, type 2 diabetes.
*Based on the availability of data in the Aerobics Center Longitudinal Study (ACLS) dataset.

self?"), and intention to lose weight (yes or no). The total amount of self-reported weight lost was calculated as the product of the number of times weight was lost and the magnitude of each reported weight loss. Dietary habits (i.e., "Are you currently on a diet?," and the number of fruit and vegetable servings consumed per week) were also obtained from a standardized questionnaire. Fruit and vegetable consumption was dichotomized (men >35 per week; women >25 per week) to identify participants who met current dietary guidelines (Health Canada 2007).

Statistical analysis

Data are presented as mean (SD) or prevalences. One-way analysis of variance was used to determine group differences between normal-weight and obese participants by EOSS stage with Tukey's post hoc comparisons. Chi-square tests were used to determine differences in prevalence by EOSS stage with least-squared differences post hoc comparisons. Cox proportional hazards regression was used to assess the relative risk of all-cause mortality by EOSS stage, compared with normal-weight participants, after adjustment for age,

sex, smoking status, and exam year, and additional adjustment for fitness and dietary habits in the subsequent model. The proportional hazards assumption was examined by comparing the log–log survival plots grouped on exposure categories; no appreciable violations were noted. Individuals with a follow-up of less than 1 year were excluded from the mortality analyses. There were no significant sex–EOSS stage interaction effects; thus, all analyses were conducted for men and women combined ($p > 0.10$).

In the obese cohort, logistic regression was used to compare the factors (BMI, weight history, low fitness, reported dieting, and inadequate fruit and vegetable consumption)associated with severe EOSS (stage 2 or 3) with those associated with stage 0 or 1 EOSS. To facilitate comparison, odds ratios were expressed per standard deviation increase for all continuous variables after adjustment for age, sex, smoking status, BMI, and exam year.

Statistical analyses were performed using SAS version 9.1 (Cary, N.C.), with statistical significance set at alpha < 0.05.

Results

Characteristics of participants by EOSS stage are shown in Table 2. The average follow-up time (SD) was 16.2 (7.5) years (median follow-up time and number of deaths for normal-weight participants was 17.6 years and 1 297 deaths; for EOSS stage 0 or 1 participants was 18.4 years and 254 deaths; for stage 2 participants was 10.2 years and 102 deaths; and for stage 3 participants was 11.9 years and 141 deaths). The prevalence of EOSS was 37.2% for stage 0 or 1, 31.2% for stage 2, and 31.6% for stage 3. Participants with more severe EOSS stages were slightly older, had modestly higher BMIs, were more likely to be former smokers, lost more weight over their lifetime, lost weight more frequently, weighed less at age 21, and were older when they attained their maximum adult weight ($p < 0.05$) (Table 2). Nearly all obese individuals were intending to lose weight (>99%).

Compared with normal-weight individuals, patients in stage 2 or 3 were at elevated relative risk of all-cause, CVD (Fig. 1), and CHD mortality (stage 0/1 HR (95% CI), 0.72 (0.56–0.93); stage 2 HR, 1.58 (0.99–2.52), $p = 0.053$; stage 3 HR, 2.13 (1.48–3.05)); however, patients in stage 0/1 were not. In fact, those in stage 0/1 were at lower risk for CVD and CHD mortality than normal-weight individuals ($p < 0.05$). All obese patients, regardless of EOSS stage, were at elevated risk for cancer mortality, but were not at elevated risk for non-CVD or noncancer mortality.

After additional adjustment for fitness and dietary factors, only EOSS stage 3, and not stage 2, remained significantly associated with elevated all-cause (stage 2 HR, 1.21 (0.97–1.51); stage 3 HR, 2.19 (1.00–1.48)) and CVD mortality risk (stage 2 HR, 1.37 (0.97–1.94); stage 3 HR, 1.38 (1.03–1.86)). EOSS stage 0/1 remained significantly associated with lower risk for CVD mortality (HR, 0.80 (0.66–0.96)), but not all-cause mortality (HR, 1.00 (0.90–1.11)). EOSS stage 0/1 (HR, 1.27(1.06–1.52)), and stage 2 (HR, 1.48 (1.01–2.16)), but not stage 3 (HR, 1.23(0.86–1.77)), remained significantly associated with cancer mortality, and EOSS was not associated with non-CVD or noncancer deaths.

In the obese cohort, BMI a significant predictor of all-cause (HR, 1.03 (1.01–1.05)), CVD (HR, 1.04 (1.01–1.07)), and non-CVD or noncancer (HR, 1.04 (1.01–1.07)) mortality, but not CHD (HR, 1.04 (1.00–1.08)) or cancer mortality (HR, 0.98(0.93–1.04)), with adjustment for EOSS stage. In the obese cohort, EOSS stage was not associated with mortality, with or without adjustment for BMI.

To identify factors that were associated with more severe EOSS in the obese cohort, we examined the independent associations between weight history and EOSS stage 2 or 3 (Table 3). Lifetime weight lost, preferred body weight, lowest adult body weight, and weight at age 21 years were predictive of EOSS stage 2 or 3 after adjustment for age, BMI, sex, smoking status, and exam year. Similarly, weight cycling, dieting, inadequate fruit and vegetable consumption, and low cardiorespiratory fitness were associated with prevalent EOSS stages 2 and 3 ($p < 0.05$; Table 3).

Discussion

This study clearly demonstrates that the EOSS can be used to help differentiate individuals at increased mortality risk. Independent of BMI, weight history and lifestyle factors were shown to influence the severity of EOSS and may, therefore, help physicians identify high-risk individuals who are most in need of weight-management interventions.

Overweight and obesity are associated with several metabolic aberrations that increase risk for CVD, CHD, type 2 diabetes, several cancers, and many other conditions (National Institutes of Health 2000). Although BMI is associated with increased mortality risk, there was considerable variation in the health risk profile observed in the obese population, and results from this analysis suggest that EOSS may be useful for clinicians in the identification of patients at higher mortality risk, beyond BMI alone. The finding that there were no differences in all-cause mortality risk between obese individuals in EOSS stage 0/1 and normal-weight individuals brings into question whether weight loss is beneficial for reducing health risk in this unique obese population. This is in contrast to the current U.S. obesity treatment guidelines, which suggest that obese individuals should be treated for their obesity, regardless of their overall risk profile (National Institutes of Health 1998). However, this is in line with the EOSS treatment algorithm that promotes weight management to prevent further weight gain for the first 2 stages, as opposed to weight loss that is prescribed for the more advanced stages. Interestingly, only EOSS stages 2 and 3 were associated with increased all-cause, CVD, and CHD mortality risk, compared with normal-weight adults. This algorithm represents an improvement on other risk-stratification systems, such as the metabolic syndrome or insulin sensitivity, that do not appear to differentiate mortality risk in obese populations (Kuk and Ardern 2009).

Lifestyle behaviours clearly have an effect on health, and individuals in EOSS stages 2 and 3 were less likely to be dieting, to eat adequate fruits and vegetables, and to be previous smokers. Indeed, adjustment for fitness and dietary factors abolished many of the associations between EOSS and mortality. First, smoking is associated with a lower body weight and several deleterious health outcomes. Although

Table 2. Characteristics of ACLS participants, 1987 to 2001.

Variables	Normal weight	EOSS stage 0/1	EOSS stage 2	EOSS stage 3
n (% of obese)	23 309	2315 (37.2%)	1944 (31.2%)	1965 (31.6%)
Age (y)	42.7 (10.2)[a]	43.3 (9.5)[b]	46.5 (9.4)[c]	45.1 (9.4)[d]
Weight (kg)	68.7 (10.1)[b]	104.8 (13.8)[a]	104.8 (15.4)[a]	104.3 (14.7)[a]
BMI (kg·m^{-2})	22.6 (1.7)[a]	33.0 (3.2)[b]	33.4 (3.9)[c]	33.5 (3.9)[c]
Sex (% male)	63.4[a]	91.8[b]	87.3[c]	83.1[d]
Smoking (%)				
Nonsmoker	74.8[a]	70.8[b]	61.9[c]	62.0[d]
Current smoker	13.9	20.3	14.2	17.9
Former smoker	11.3	8.9	23.9	20.2
Currently dieting (%)	30.1[a]	22.8[b]	23.0[b]	29.9[a]
Low CRF (%)	8.2[a]	46.83[b]	37.7[c]	50.0[d]
Weight history				
Lifetime weight loss (kg)	12.5 (39.0)[a]	26.3 (78.7)[b]	70.2 (113.6)[c]	68.0 (116.8)[c]
Frequency of weight loss	3.6 (11.3)[a]	5.3 (16.6)[b]	14.5 (26.2)[c]	13.9 (25.9)[c]
Weight at age 21 y (kg)	65.9 (11.7)[a]	86.2 (14.6)[b]	83.5 (15.4)[c]	81.8 (15.4)[d]
Preferred weight (kg) *	66.1 (11.2)[a]	87.7 (11.5)[b]	86.6 (12.4)[b]	83.5 (13.1)[c]
Minimum adult weight (kg)[†]	61.1 (10.8)[a]	80.2 (11.5)[b]	79.0 (13.2)[b]	76.0 (13.4)[c]
Maximum adult weight (kg)[‡]	75.6 (12.1)[a]	109.6 (15.2)[b]	108.9 (16.7)[b]	108.7 (16.7)[b]
Age at maximum weight (y)[§]	32.6 (10.8)[a]	39.6 (10.4)[b]	42.7 (10.6)[c]	42.2 (10.3)[c]
Weight loss intention (%)	81.6[a]	99.3[b]	99.0[b]	99.5[b]

Note: Different superscript letters indicate significant group differences ($p < 0.05$). CRF, cardiorespiratory fitness.

*n = 13 223.

[†]n = 12 465.

[‡]n = 27 799.

[§]n = 18 728.

Fig. 1. Association between Edmonton Obesity Staging System (EOSS) stage and risk of all-cause (A), cardiovascular disease (CVD) (B), cancer (C), and non-CVD or noncancer mortality (D) in men and women. Adjusted for age, sex, smoking status, exam year, low fitness, dieting, and adequate fruit and vegetable consumption. *$p < 0.05$. HR, hazard ratio; NW, normal weight.
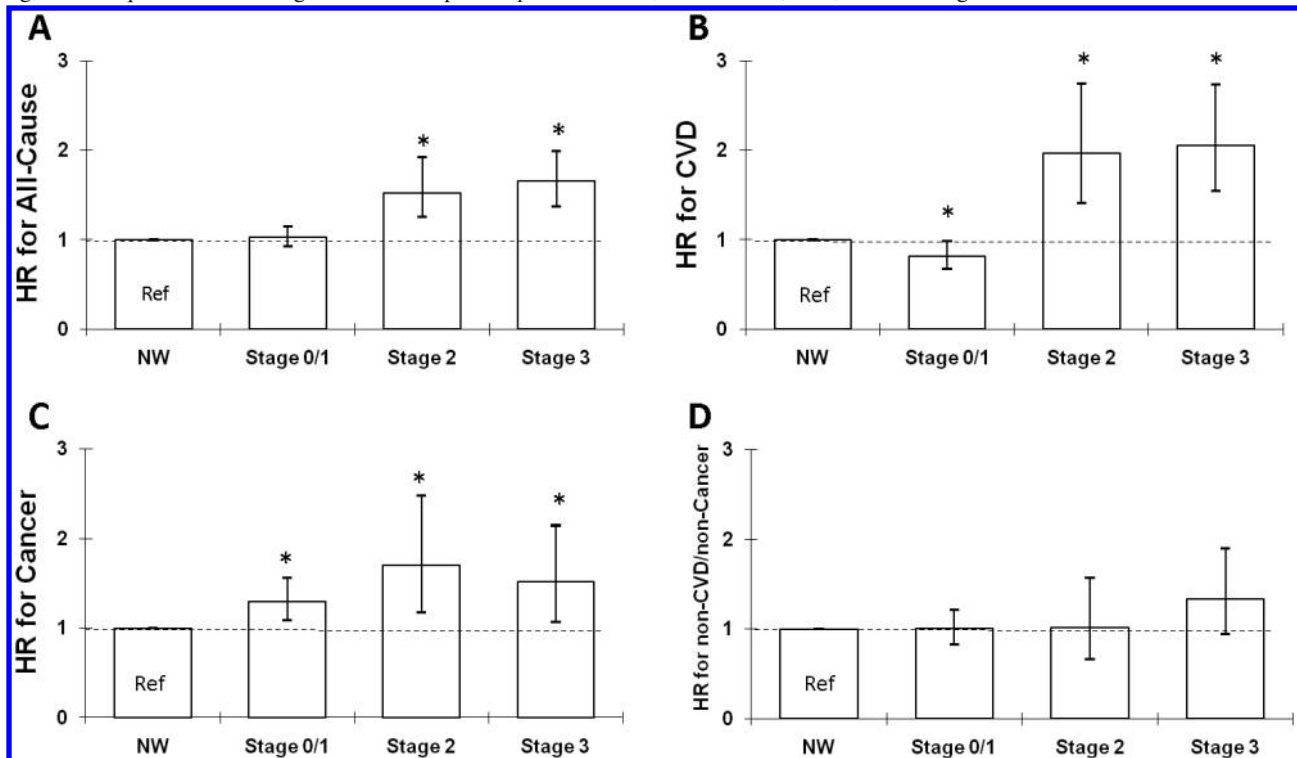
Table 3. Weight history and lifestyle factors associated with EOSS stage 2 or 3 in obese men and women.

OR of EOSS stage 2 or 3	Standardized OR (95% CI)[†]
Body mass index[*,‡]	1.11 (1.05–1.18)[†]
Weight at age 21 y[‡]	0.86 (0.80–0.93)[†]
Age of maximum weight[‡]	0.98 (0.87–1.11)
Maximum weight difference[‡]	1.10 (0.98–1.23)
Lowest adult weight[‡]	0.89 (0.79–0.99)[†]
Maximum adult weight[‡]	0.94 (0.87–1.02)
Preferred body weight[‡]	0.83 (0.74–0.93)[†]
Intenting to lose weight	1.09 (0.36–3.29)
Lifetime weight loss[‡]	1.22 (1.12–1.32)[†]
Weight cycling frequency[‡]	1.20 (1.11–1.30)
Low fitness	1.32 (1.17–1.50)[†]
Reported dieting	0.70 (0.64–0.77)[†]
Adequate fruit and vegetable intake	0.80 (0.74–0.86)[†]

Note: OR, odds ratio; CI, confidence interval.

*Adjusted for age, sex, smoking status, and exam year.

[†]OR significant at $p < 0.05$.

[‡]OR are expressed per SD for continuous variables.

smoking cessation is unlikely to be the direct cause of negative disease outcomes, it is often associated with weight gain (Reas et al. 2009). Second, higher EOSS stage was associated with lower minimum adult body weight, but similar maximum adult weights. Third, lifestyle factors, such as diet and physical activity, can influence health risk, independent of BMI (Yusuf et al. 2004), and form the foundation of weight management (National Institutes of Health 2000).

However, for the vast majority of obese individuals, lifestyle-based weight loss is not maintained over the long term (Wing et al. 1995). This is particularly concerning, given that weight cycling is associated with greater weight gain over time (Van Wye et al. 2007) and potentially worse health outcomes, compared with individuals who may have maintained a stable body weight (Blair et al. 1993; Wannamethee et al. 2002). Although we observed that greater reported weight loss was associated with worse EOSS scores, it is unclear whether individuals with more severe EOSS staging had attempted to lose more weight *because* of their poor health, or whether they had poorer health because they had weight cycled. Furthermore, it is unclear whether obese individuals without existing comorbidities will develop metabolic abnormalities if they remain at a stable BMI; the literature is currently divided over the importance of obesity duration on metabolic risk. It has been proposed that the length of obesity predicts metabolic aberrations, and that adequate time is needed for metabolic abnormalities to develop in response to an obese state (Janssen et al. 2004; Wannamethee and Shaper 1999). Somewhat paradoxically, results from our study and other studies (Brochu et al. 2001) suggest that higher body weights earlier in life are associated with *lower* EOSS stages. Though speculative, it may be that individuals who develop obesity earlier in life exhibit an adaptive capacity, or perhaps these individuals are naturally predisposed to higher body weights because of higher insulin sensitivity — a factor associated with greater weight gain (Swinburn et al. 1991). Nevertheless, these factors, together, indicate that obese patients, particularly in EOSS stages 0 and 1, may be better

served if physicians promoted weight maintenance, as opposed to weight loss, as it remains to be seen whether individuals in EOSS stages 2 and 3 will benefit from weight loss.

Obesity in the absence of metabolic aberrations has been termed, in the literature, "metabolically normal but obese," and it has been suggested that these individuals may not require weight loss per se (Brochu et al. 2001; Sims 2001). This is in contrast to a recent observation that obese individuals are at similarly increased mortality risk, regardless of their metabolic profile (Kuk and Ardern 2009); however, it stands to reason that these differences can be explained by the inclusion of other nonmetabolic diagnostic criteria in EOSS. Indeed, the health risks associated with overweight and obesity are not limited to metabolic conditions, as obese individuals are more likely to be diagnosed with cancer at more advanced stages (Hahn et al. 2007) and to die from traumatic injuries (Viano et al. 2008) than their normal-weight counterparts. Further, the weight bias of some health professionals results in greater reluctance to provide health care, a problem that is compounded by the fact that obese individuals are more likely to avoid seeking health care (Puhl and Brownell 2001). In this study, cancer and non-CVD and noncancer causes of death were not associated with EOSS staging, and suggest that normal-weight and obese individuals are at a similar risk for these causes of death. However, further research is required to examine the factors within EOSS that are most predictive of mortality risk.

Strengths and limitations

The Aerobics Center Longitudinal Study is a unique clinical dataset that provides mortality follow-up, in addition to rich information on several clinical measures and factors related to diet, cardiorespiratory fitness, and weight history in a large sample of predominantly white, middle- to upper-class men and women, and may not be representative of the general U.S. population. The EOSS definition used was a modified version because of restrictions in the available data. To maintain a reasonable sample size, some individuals included in the analyses did not have complete data for all the EOSS variables used. However, this likely reflects a real-world scenario, as these data were collected in a clinical visit and physicians may not assess their patients for all potential EOSS factors. Furthermore, this error should have only diminished the observed association between EOSS and mortality risk, as we may have underestimated EOSS stage. In our analysis, we were unable to evaluate the effect of changes in body weight over time on mortality outcomes. It is possible that some individuals may have lost weight while others gained weight. Such misclassification of exposure would likely underestimate of the magnitude of the association we observed. Future prospective analyses with multiple measures of EOSS and weight will aid in our understanding of the relationships between health risk and weight change. Now that we have demonstrated the prognostic ability of EOSS, future studies should evaluate EOSS against other currently used algorithms, such as Framingham, metabolic syndrome, and the National Institutes of Health clinical guidelines for overweight and obese individuals.

In conclusion, the EOSS can be used to help clinicians identify overweight and obese patients who are at higher mortality risk. Further research is necessary to evaluate the

potential role of weight history and lifestyle factors in determining EOSS risk and the importance of individual EOSS components in the overall risk assessment.

Acknowledgements

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