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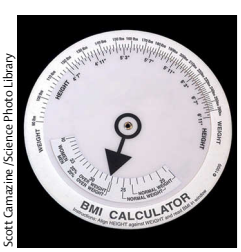
The association between BMI and mortality: implications for obesity prevention



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Excess adiposity is an established risk factor for all-cause mortality, likely mediated mainly through its effects on a wide range of chronic diseases, including several types of cancer, type 2 diabetes, and cardiovascular disease. In their population-based cohort study published in *The Lancet Diabetes & Endocrinology*, Krishnan Bhaskaran and colleagues[1] found a J-shaped association between BMI and overall mortality among 3·6 million adults in the UK. This study provides further support for the strong association between increasing BMI and excess mortality at BMI of 25 kg/m² or higher (estimated hazard ratio per 5 kg/m² increase 1·21 [95% CI 1·20–1·22]). These observations are consistent with several recent studies, collectively representing tens of millions of participants globally.[2–4] The authors also report the association between BMI and deaths from specific causes, with optimal BMI in the range of 21–25 kg/m² for cancer, cardiovascular, and respiratory deaths. Causes of death with weak or no biological link to excess bodyweight, such as mental health, behavioural, neurological and accidental causes, and suicide, were not associated with increased BMI, but with underweight (<18·5 kg/m²). With a large sample size, the authors analytically controlled for biases that often underestimate the magnitude of the association of BMI with mortality in their primary analyses, by limiting their analyses to never smokers and excluding deaths occurring less than 5 years from BMI ascertainment. Participants with prevalent chronic diseases that affect bodyweight and subsequent mortality risk (eg, cancer, neurological diseases, and cardiovascular disease) were included in the main analyses, although they were excluded in sensitivity analyses that confirmed the main results.

In this study, the J-shaped association between BMI and all-cause mortality was partly driven by the associations between lower BMI or underweight and increased mortality from mental and behavioural, neurological, and external causes. There is a high likelihood that these associations reflect methodological issues such as reverse causation (ie, low BMI is the consequence rather than the cause of these conditions), but further research is needed to disentangle complex associations between these mental health and neurological conditions and bodyweight.

This study also quantified the population attributable fraction (PAF), an estimate of the contribution of overweight and obesity to total mortality, or the potential population-level reduction in mortality given the hypothetical scenario in which everyone had been of optimal bodyweight. Because this metric accounts for the prevalence of exposure in the population, it arguably better reflects the disproportionate burden for underweight versus overweight and obese categories than the J-shaped association. As such, the authors estimated that, assuming causality, overweight and

obesity (BMI ≥25 kg/m²) contributed to 5·5% of total deaths, whereas underweight contributed to 0·7%, in their UK-based study population. The estimate for excess bodyweight with mortality is similar to that of the 2015 Global Burden of Disease Study,[3] which estimated that, globally, overweight and obesity contributed to approximately 7·1% (95% uncertainty interval 4·9–9·6) of total deaths. PAF estimates are expected to vary across study populations according to the time period of data collection, socioeconomic development, and the ranking of common causes of mortality. Estimates of PAF for excess bodyweight with mortality will also continue to be a moving target as the population distribution of BMI shifts. Regardless, this metric provides a useful tool for health-care providers and stakeholders to appreciate the importance of overweight and obesity as an important driver of excess mortality.

This study adds to the overwhelming evidence about the public health importance of the obesity epidemic to overall and cause-specific mortality. It also has important clinical and public health implications for obesity prevention, especially for the prevention of further increase in bodyweight and waist size among moderately overweight individuals. Although the debate might persist as to the precise point at which the association between continuous BMI with excess mortality becomes statistically significant, it is important to note that most people will gain weight throughout midlife, which is associated with increased subsequent risk of chronic diseases and mortality.[5] Many individuals with a BMI in the range of overweight (25·0–29·9 kg/m²) are already on a trajectory of gaining more weight that will transition them into the BMI range of obesity (≥30·0 kg/m²). Therefore, although the excess mortality associated with overweight is relatively small, it is important for overweight individuals to prevent further weight gain. Even among older populations, a plateau or decline in bodyweight often masks a trajectory of fat mass gain, offset by losses in lean body mass (ie, decreases in muscle tissue and bone density).[6] Therefore, it is important for older individuals to prevent an increase in waist size, a marker of abdominal obesity, while minimising loss of muscle mass. Additional studies might shed more light on the role of body fat distributions and different fat depots in chronic disease morbidity and mortality. Meanwhile, current efforts need to be intensified to identify more effective and impactful strategies for prevention of weight gain and obesity-related comorbidities.

*Deirdre K Tobias, *Frank B Hu*
Division of Preventive Medicine (DKT) and Channing Division of Network Medicine (FBH), Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA; and Department of Nutrition (DKT, FBH) and Department of Epidemiology (FBH), Harvard TH Chan School of Public Health, Boston, MA, USA
frank.hu@channing.harvard.edu

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Oral health: a neglected aspect of diabetes care

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Periodontitis and diabetes are well known to have a bidirectional association.[1] Patients with uncontrolled diabetes are prone to periodontitis, probably due to their hyper-responsiveness to the oral dysbiosis and weakened healing processes, collectively causing periodontal breakdown.[2] Meanwhile, periodontitis associates with poor glycaemic control, leading to microvascular complications.[2] Furthermore, periodontitis can also contribute to macrovascular complications through systemic inflammation and its associated dyslipidaemia;