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**EDITORIAL**


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# Deaths Attributable to Obesity

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**I**N THIS ISSUE OF *JAMA*, 2 STUDIES<sup>1,2</sup> PRESENT NEW ANALYSES on the subject of obesity. The study by Flegal et al<sup>1</sup> is likely to generate interest because it provides an estimate for deaths attributable to obesity that appears to strongly contradict prior estimates published in *JAMA*. Flegal et al<sup>1</sup> estimate that there were about 112 000 obesity-attributable deaths in the United States in 2000, far lower than the 414 000 estimated by Mokdad et al<sup>3,4</sup> for the same year and the 280 000 estimated by Allison et al.<sup>5</sup> for 1991. The magnitude of the differences cries out for explanation of the reasons behind these differences. Some might wonder: If well-intentioned efforts to calculate this number can result in such widely varying estimates, is it worth trying to do at all?

The underlying methods of all these studies involve the concept of population-attributable fraction. Population-attributable fraction (or attributable fraction or etiologic fraction) is the proportion of morbidity or mortality in a population that can be attributed to a particular cause or risk factor and is one of the empowering concepts of the public health perspective on health. The attributable fraction focuses attention not on a particular disease or risk of disease in the individual but on the health of populations. In its most basic equation, this quantity shows that the burden of disease caused by any risk factor is a function of the prevalence of

that risk factor and the magnitude of its causal association with disease, usually expressed as relative risk. The greater the prevalence of the risk factor and the greater the relative risk, the greater the population-attributable fraction. The calculation offers a perspective on health that crosses disciplines and specialties and attempts to focus attention on causes of disease that are most responsible for death and illness. Over the past 40 years, cigarette smoking, a common habit with highly elevated relative risks for several different causes of death, has always risen to the top of the list. The attention paid to the problem of cigarette smoking as a major cause of disease and death has been in part a result of the information and perspective provided by population-attributable fraction calculations.

Thus, it is natural, almost imperative, for public health care practitioners to apply this perspective to obesity, an issue perceived to be a growing public health problem. There is no doubt that the prevalence of obesity is increasing in the United States. However, for many reasons it is much more difficult to estimate the burden of disease due to obesity. Although weight is an easily measured characteristic, at a conceptual level attributing deaths to obesity requires many assumptions that are often not fully spelled out in most studies.

Consideration of the causal pathways through which obesity increases mortality is important. Body mass index itself is affected by dietary intake and physical activity lev-

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**See also pp 1861 and 1868.**

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els, which may affect health in ways mediated by body mass index or independent of it. According to the most recent clinical guidelines for cardiovascular risk reduction, most of the risk of cardiovascular disease caused by obesity is thought to be mediated through traditional risk factors such as diabetes, cholesterol, and hypertension.<sup>6</sup> Thus, the guidelines do not incorporate obesity as an independent factor in which to estimate risk of cardiovascular disease. Calculating obesity-related deaths without accounting for other cardiovascular risk factors, as has been done in previous studies,<sup>3-5</sup> assumes a consistent relationship between obesity and these mediating risk factors. However, in the study by Gregg et al<sup>2</sup> in this issue of *JAMA*, analysis of representative samples of Americans show that the association of obesity and other cardiovascular risk factors has changed over the past 40 years. Obese persons now smoke less and have lower cholesterol levels and lower blood pressure. Although mortality outcomes are not evaluated in the study by Gregg et al,<sup>2</sup> because of interactions between risk factors, this across-the-board decrease in risk factors across all weight groups could translate to a lowering of the elevated risk of death associated with obesity.

However, other gaps in current knowledge about health risks of obesity might temper this possibility. Most deaths occur among older persons, whose weight during old age may or may not reflect weight throughout their entire lifetime. Studies evaluating risks of obesity usually assess weight at a single point in time rather than throughout life. Knowledge is not complete regarding the health effects of differing lifetime trajectories of body weight. Many studies have shown that obesity among children and adolescents is increasing.<sup>7</sup> Younger age of onset of obesity may result in a longer duration of obesity throughout life, which may increase obesity-related mortality. Many other issues complicate the assessment of obesity-related disease, including choice of statistical techniques and choice of study populations in which to calculate mortality risks, to name a few issues.

Complicating the task even further is the basic issue of numerical uncertainty, caused by the need to estimate many numbers from so many different data sources. An important and possibly overlooked contribution by the study by Flegal et al<sup>1</sup> is the formal calculation of confidence intervals around the estimate of obesity-related deaths. When relative risk estimates are only modestly elevated, as in the case

of obesity, very small changes in the relative risk translate to large differences in the population-attributable fraction.<sup>8</sup> Thus, it should come as no surprise that the 95% confidence interval around the estimate of 112 000 deaths ranges from 54 000 to 170 000, greater than a 3-fold difference reflected within the range. Although the other studies<sup>3,5</sup> that previously estimated obesity-attributable death did not include confidence intervals, the estimates from those studies should be assumed to have underlying uncertainty at least as great.

These studies and their disparate findings highlight the importance of continuing to develop more rigorous approaches for estimating obesity-attributable deaths. Ultimately, though, it may be possible to gain a better and more realistic understanding of the preventable disease burden caused by obesity by evaluating public health and individual programs designed to both prevent and treat obesity, such as diet and exercise programs. Such programs should also be evaluated for their ability to reduce disease and morbidity in addition to effects on body weight, for there may be additional benefits (or possible risks). With sufficient knowledge of the effectiveness and required resources of these programs, it will be possible to make rational decisions regarding the best way to maintain and improve the health of the public.

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