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Adolescent Overweight and Coronary Heart Disease

TO THE EDITOR: The Centers for Disease Control and Prevention (CDC) recommends using the body-mass index (BMI) for age as a marker in assessing patients between the ages of 2 and 20 years for obesity.¹ In the article by Bibbins-Domingo et al. (Dec. 6 issue),² weight alone was used as a marker for obesity in adolescents, which may not reflect the true underlying value. Was this approach justified? Furthermore, the assumption that a high BMI does not directly increase the risk of coronary heart disease (CHD) may be unwarranted, since there is enough evidence to the contrary.³

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TO THE EDITOR: The results of the study by Bibbins-Domingo et al. are consistent with our recent analyses of trends in CHD in the United States.^{1,2} Between 1980 and 2000, the age-adjusted rate of death from coronary heart disease was halved, with 341,745 fewer such deaths in 2000. Approximately 47% of the decrease was attributable to treatments and approximately 44% to changes in major risk factors. Crucially, these

gains were offset by increases in diabetes and obesity. Between 1980 and 2000, BMI (defined as the weight in kilograms divided by the square of the height in meters) increased from 25.5 to 28.2, accounting for approximately 25,900 additional deaths.²

Our model used the large random-effects meta-analysis (with a total of 302,296 subjects) by Bogers et al.³ A five-unit increase in BMI generated a 29% increase in deaths from coronary heart disease, or, crucially, a 16% increase after adjustment for cholesterol level and blood pressure.³ Might Bibbins-Domingo et al. have underestimated the mortality effects?

Trends toward increased mortality from coronary heart disease are already detectable in the U.S. population.⁴ From 1997 through 2002, the mortality rate leveled off among men who were 35 to 44 years of age and actually increased by 1.3% annually among women in the same age group.⁴ Perhaps the party is already over.

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THE AUTHORS REPLY: Kumar et al. incorrectly state that we used weight alone as a marker for obesity. Our article states that we used BMI for adults and CDC growth charts for adolescents. Contrary to the suggestion by Kumar et al. and by Capewell and Critchley that an elevated BMI is an established independent risk factor for CHD, we find the evidence mixed and the possible independent relationship of BMI with the risk of CHD controversial. Bogers et al., cited by Capewell and Critchley, showed that half of the effect of obesity on CHD was attributable to cholesterol and hypertension but that study did not adjust for changes in glucose metabolism; the authors themselves acknowledged that the residual risk may be due to diabetes. Lawler et al., the study cited by Kumar et al. as evidence that obesity has a direct effect on CHD, actually concluded that childhood BMI was not associated with the subsequent risk of CHD. We modeled the well-established association of increases in BMI with changes in diastolic blood pressure, changes in levels of high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol, and diabetes. We did not model an independent association of BMI with the risk of CHD after accounting for these measured factors on the basis of our analysis of Framingham data and observations that the relationship between BMI and the risk of CHD is substantially diluted after blood pressure, cholesterol levels, and the presence or absence of diabetes are considered.^{1,2} In fact, we could not find data confirming an independent relationship between BMI and the risk of CHD after adjusting for measured blood pressure, measured HDL and LDL cholesterol, and confirmed presence or absence of diabetes.

Capewell and Critchley and their colleagues,³ like our group,⁴ used epidemiologic data to explain previous trends in death from CHD, and they have recently presented data showing a slowing of the declines in mortality from CHD among men between the ages of 35 and 54 years and an insignificant increase among women in the same age group.⁵ By comparison, our recent analysis estimated future CHD. Even if, as we acknowledge in our article, we may have underestimated the effect of obesity to avoid overstating the case, we agree that obesity-induced changes in risk factors for CHD represent a potential time bomb that could offset much of the progress made over the past four decades.

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Cochlear Implants

TO THE EDITOR: The review article by Papsin and Gordon (Dec. 6, 2007, issue)¹ provides useful information about cochlear implantation. However, additional practical points should be noted. The majority of patients with hearing impairment live in countries where the cost of a cochlear implant is the most influential factor. For example, in Iran, two thirds of the price of a \$20,000 implant is

covered by the government, and the perioperative costs would not exceed \$2,000, but fewer than 2000 implants have been inserted in the past 10 years, whereas this figure reaches more than 40,000 in the United States.² The difference in the gross domestic product per capita between Iran and the United States (\$8,900 vs. \$43,800) explains this difference.³ Genetics, first through