

Decrease in U.S. Deaths from Coronary Disease

TO THE EDITOR: Ford et al. (June 7 issue)¹ have developed a statistical model, called IMPACT, which explains almost 90% of the observed decrease in deaths from coronary heart disease in the United States in the 20 years from 1980 to 2000. This model, which has been validated and reproduced mainly in developed countries, is so robust that some of its findings are similar to those of the earlier Nurses' Health Study,² especially the proportional contributions of smoking and obesity to heart disease (13% and 8%, respectively). However, the model does not explain almost 10% of the observed decrease in deaths. The reduction in particulate air pollution, for example, explains from 18 to 76% of the decline in deaths from both coronary heart disease and coronary vascular disease.^{3,4} No doubt the U.S. outdoor air quality has improved substantially since 1980. An Irish study performed after the ban on burning coal showed a 10% decline in deaths from coronary vascular disease.⁵ Therefore, it would be worth considering air pollution in the IMPACT model, data that could be integrated into a comprehensive Chronic Disease Risk Model, thereby using such "gold standard" dynamic epidemiologic models as population-specific, evidence-based policy models.

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THE AUTHOR AND A COLLEAGUE REPLY: With respect to Kabir's suggestion that particulate air pollution might account for some of the decrease in deaths from coronary heart disease that were not explained by the risk factors in the IMPACT model: air pollution can have both short-term and long-term effects on outcomes in patients with coronary heart disease.^{1–3} Strong associations between peaks in air-pollution indexes and increased mortality and admissions for cardiac and respiratory disease have long been recognized.² Deaths from cardiovascular causes have also been associated with cumulative long-term exposure to air pollutants.³ However, quantifying the contribution of this factor remains problematic because of the difficulty in correcting for all potential confounders, particularly sex and socioeconomic status. Even after adjustment, residual confounding may persist.^{1,3} A long list of risk factors, including air pollution, could potentially account for the unquantified 10% of deaths in the IMPACT model. However, imprecision in the measurement and modeling of the major risk factors (cholesterol, smoking, and blood pressure) might also account for much of the gap.

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The Syndrome of Inappropriate Antidiuresis

TO THE EDITOR: In the article on the syndrome of inappropriate antidiuresis (SIAD), by Ellison and Berl (May 17 issue),¹ I disagree with the statement, in the discussion of areas of uncertainty, that "SIAD is characterized by a water excess, rather than a sodium deficit." In fact, salt deple-

tion occurs, as shown in elegant observations made by William B. Schwartz between 1950 and 1970. If one continuously administers vasopressin in dogs maintained on zero salt and normal water intake, volume expansion with urinary salt loss occurs initially. This period is followed by an