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ANALYSIS & COMMENTARY

The Contributions Of Prevention And Treatment To The Decline In Cardiovascular Mortality: Lessons From A Forty-Year Debate

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ABSTRACT Mortality from coronary heart disease in the United States has fallen 60 percent from its peak in the mid-1960s. Cardiologists and epidemiologists have debated whether this decline reflects better control of risk factors, including lifestyle interventions to reduce smoking or intake of dietary fats, or the power of medical interventions, including defibrillators and therapeutics such as statins. Attempts to resolve this debate and guide health policy have generated sophisticated data sets and techniques for modeling cardiovascular mortality. Neither effort has provided specific guidance for health policy. Historical analysis of the debate over the causes of the decline, concomitant with development of cardiovascular modeling, offers valuable policy lessons about tensions among medical and public health strategies, the changing meanings of *disease prevention*, and the ability of evidence-based research and models to guide health policy. Policy makers must learn to open up the "black box" of epidemiological models—and of their own decision-making processes—to produce the best evidence-informed policy.

At some unnoticed moment in the mid-1960s, mortality from coronary heart disease in the United States peaked and then began to decline. Even though coronary heart disease remains the leading cause of death worldwide, mortality from it has fallen 60 percent from its peak.[1] Although this achievement may represent the greatest public health accomplishment of the twentieth century, it is not clear who or what deserves credit. Does the decline demonstrate the therapeutic power of modern medicine or the impact of lifestyle change and management of risk factors?

Cardiologists and epidemiologists have struggled for nearly forty years to resolve this question. They have developed sophisticated data sets and quantitative models of the factors that cause or mitigate cardiovascular mortality. They hoped that their analyses would help policy makers decide whether to invest in treatment or prevention. The analyses, instead, have consistently demonstrated the value of both.

Much can be learned by studying the history of the intersection of cardiovascular epidemiology and health policy in this debate. As researchers' methods evolved from anecdote to back-of-the-envelope calculations to complex models, increasing precision came at the cost of increasing opacity. Few readers will understand the subtle mechanisms and assumptions on which such models rely. The models, however, have become more versatile and ubiquitous. It is essential for policy makers to understand the promise and limitations of the models, the changing meanings of *prevention*, and the likelihood that empir-

ical research might shape policy. Just as the decline itself provided a "natural experiment" for cardiovascular epidemiologists, the debate over the sources of the decline provides an opportunity to analyze the dynamic interplay between knowledge production and health care policy.

Rise And Fall Of Coronary Heart Disease

Heart disease devastated postwar America. By 1960 it killed one-third of all Americans.[2] Led by the American Heart Association and the National Heart Institute (subsequently renamed the National Heart, Lung, and Blood Institute), the country mobilized unprecedented resources against the scourge. The first evidence of changing fortune came in 1964, when state health officials reported a decline in coronary heart disease mortality in California,[3] but this news received little attention. Well into the 1970s, cardiologists and the national media sounded the alarm about the inexorable rise of coronary heart disease.

In March 1974, however, a "sign of spring" emerged.[4] Cardiologist Weldon Walker reported that age-adjusted coronary heart disease mortality rates had actually been declining in the United States since 1963.[5] His announcement was met with guarded enthusiasm: Not everyone was certain whether the decline was real or not.[6,7] Part of the problem was the delicacy of epidemiology as a historical science, dependent upon cause of death reporting with disease taxonomies or naming conventions that are constantly in flux. Depending on how epidemiologists parsed mortality data, coronary heart disease reached its peak in either 1963 or 1968.[5–8]

Eager to reach consensus about the reality and causes of decline, Robert Levy, director of the National Heart, Lung, and Blood Institute, called leading researchers to the institute in October 1978 for what became known as the "Decline Conference." Epidemiologists and clinicians concluded that the decline—a 20 percent drop between 1968 and 1978—was "real."[9] But debate continued on the second question: what had caused the decline? The timing of the decline had coincided with too many relevant changes: vigorous efforts to educate Americans about smoking, diet, and other coronary heart disease risk factors; changes in medical care, including aggressive control of hypertension, specialized coronary care units, beta-blockers, and bypass surgery; and the enactment of Medicare and Medicaid.

Determining the causes of the decline was not just an academic question. Everyone present felt the "urgent need" to answer the question and allow "intelligent decisions about the allocation of scarce resources [among] competing programs."[10(p639)] Clinicians, in particular, were on the defensive. Critiques by Thomas McKeown and Ivan Illich inspired raging debates about whether medicine made substantial contributions to the health of society and whether its contributions justified its growing cost. McKeown was a physician and demographic historian who attributed the growth of Western societies and their residents' life spans to improved standards of living arising from economic growth rather than specific medical and public health interventions.[11] Illich was a late-twentieth-century philosopher and social critic of the medicalization of Western society.

The critiques of these two experts cast a long shadow at the Decline Conference. Speakers at the conference invoked Illich as a warning to those who would take the value of medicine for granted.[9] Proponents of both medical care and disease prevention knew that they had to make their case carefully.

Quantifying The Value Of Prevention And Treatment

The first decade of research into the causes of the decline saw arguments based simply on temporal association. Walker, who noted that the onset of the decline coincided with the 1964 *Surgeon General's Report on Smoking and Health* and with the American Heart Association's campaigns against risk factors, favored prevention.[5] In an editorial, "Signs of Spring?," R.H. Moser emphasized the impact of coronary care units.[4] Although concordance might suggest cause and effect, critics emphasized that the evidence was circumstantial. For instance, as preventive cardiologist Jeremiah Stamler complained in 1978, "When such multiple socio-medical trends evolve over the years, it is virtually impossible to make a definitive scientific assessment as to the role of each of them singly, and all of them together in causing the decline in mortality rates."[12] Amid this uncertainty, an ecumenical solution appeared necessary. As the National Heart, Lung, and Blood Institute's Levy concluded, "both primary prevention through lifestyle changes and improved treatment regimes have played a role in the decline."[8(p312)]

Frustrated by their initial inability to solve "the case of the disappearing epidemic," the researchers sought more rigorous, quantitative analyses of the impact of specific interventions.[13] The Framingham Heart Study had identified specific risk factors that correlated with coronary heart disease mortality. One team of researchers used Framingham data to argue that the observed

5 mg/dL drop in cholesterol levels since the 1960s would predict a 4.3 percent decline in coronary heart disease mortality.[14] Epidemiologist Michael Stern used Framingham algorithms to calculate the cumulative impact of changes in several risk factors. He concluded that these changes accounted for 50 percent of the decline in men.[10] Stern, however, admitted that it was "not possible at present to quantify definitively" the relative impact of lifestyle changes and improved medical care, stating that "both have played a role."[10(p638)]

Researchers realized that they needed to distinguish between two effects: the extent to which prevention campaigns reduced the incidence of coronary heart disease and the extent to which medical care reduced fatality rates.[14] Making this distinction required data on both incidence and mortality.

Motivated by the Decline Conference, researchers undertook community-based surveillance projects. The World Health Organization orchestrated the largest such project: the Multinational Monitoring of Trends and Determinants in Cardiovascular Disease Project. Researchers at thirty-nine centers in twenty-six countries collected data about risk factors, medical care, event rates, case-fatality rates, and mortality from more than 100,000 people.[15]

These efforts did not yield decisive answers. To obtain high-resolution data, researchers had to focus on specific sites that might not be representative of entire populations. They struggled to ensure consistent data collection and analysis. Their analyses of aggregate population data (for example, correlating changes in average risk-factor levels and event rates) impeded their ability to decipher causal relationships. Finally, they realized that their basic assumptions—that risk factors determined event rates while health care influenced case fatality—were too simplistic.

Emergence Of Models And Simulations

Other cardiologists and epidemiologists took up the challenge from the Decline Conference and went in a different direction. They adapted analytic techniques from systems engineering to produce more precise and integrated assessments of specific preventive and therapeutic interventions.

In 1984 Lee Goldman, a cardiologist, and Francis Cook, an epidemiologist, published a model that recapitulated the passage of a coronary heart disease patient through the health care system, including emergency medical services, coronary care units, and surgical and medical treatment.[16] Each domain was divided into specific interventions that could be quantified with data from observational studies and then reassembled using simple arithmetic to calculate the number of lives saved.

For example, to enumerate the value of coronary care units, the authors estimated that 500,000 patients were hospitalized for heart attacks each year. Among them, 4.5 percent suffered ventricular fibrillation, and 88 percent of that group were successfully resuscitated: $500,000 \times 0.045 \times 0.88 = 19,800$ lives saved annually. Similar calculations revealed the contributions of lifestyle interventions against dietary fat, cholesterol, smoking, obesity, and exercise.

The authors acknowledged the subjective assessments, approximations, and potential errors in their model. But they celebrated when the interventions they modeled—four therapeutic, four preventive—combined to account for 90 percent of the decline between 1968 and 1976. Lifestyle changes accounted for 54 percent of the total, a finding they confessed "may be as serendipitous as it is accurate."[16(p832)] Goldman and Cook's model, intelligible to multiple audiences, coupled rigorous literature review with explicit assumptions and transparent calculations. It remains the most-cited reference in the decline literature.

To take this analysis further, Goldman teamed up with Milton Weinstein to form the Coronary Heart Disease Policy Model research group. In 1987 they developed the first computer model to forecast coronary heart disease mortality.[17] Their "state-transition" model simulated patient trajectories over time, considering the impact of primary prevention, the transition from health to coronary heart disease, and the impact of treatment and secondary prevention. The model could be run to follow a simulated population as it aged and as it did or did not develop coronary heart disease.

These simulations could be compared against historical data to determine how much of the actual observed decline had been captured by the model.[18] With no adjustment for improving risk factors or treatments, the model overestimated mortality between 1980 and 1990 by 34 percent. When it took these interventions into consideration, the model came within 2.8 percent of the actual data. The team concluded that a substantial portion of the decline must have come from these interventions.

Modeling has now become a popular tool in cardiovascular epidemiology, applied both to explain past declines and to predict future possibilities. A 2006 review found seventy-five articles that used forty-two different models to inform coronary heart disease policy.[19] But models, by

their nature, are imperfect representations of reality. They make simplifying assumptions to facilitate methodical analysis. Goldman and Cook, for instance, cautioned that their model "must be considered approximate at best," as "a perspective rather than a definitive explanation."[16(p832)] This is not a problem as long as readers understand a model's limits.

The challenge for readers is twofold. First, many different types of models exist. Some are static, calculating effects based on the prevalence and impact of specific interventions in a population. Others are simulations that analyze a computer-generated cohort of "individuals" as they age over time. Second, the quality of models varies considerably. Are the assumptions explicit? Are the mechanisms transparent? Have sensitivity analyses been done (for example, to test the effect of different assumptions)? Has the model been validated (for example, tested against existing data sets)? The 2006 review found that few of the forty-two models met these quality criteria.[19]

Consider several prominent examples. Researchers working with the World Health Organization's Disease Control Priorities Project developed a model of the global burden of disease. They compiled data on morbidity and mortality of more than 130 diseases and calculated what share of this burden could be attributed to specific risk factors. Using assumptions about socioeconomic development and risk-factor trends, they forecast the burden of disease in 2030. Coronary heart disease, increased by tobacco use, hypertension, and inactivity and decreased by alcohol use, will remain the leading cause of death worldwide.[20] Such projections might help countries determine where to invest health resources.

A different model, the IMPACT mortality model, focused on the decline of coronary heart disease. Developed by Scottish cardiologist and epidemiologist Simon Capewell, IMPACT, like Goldman's initial analysis, quantified the use and impact of interventions to calculate the number of deaths prevented by each.[21] Capewell demonstrated his model on data from Scotland and found that of the deaths prevented between 1975 and 1994, 10 percent came from acute coronary care, 9 percent from treatment of hypertension, 8 percent from secondary risk-factor management, 8 percent from management of heart failure, 2 percent from bypass surgery, 2 percent from aspirin, and 0.1 percent from angioplasty. Meanwhile, of the risk factors, smoking contributed 36 percent, cholesterol 6 percent, blood pressure 6 percent, and socioeconomic deprivation 3 percent. Taken together, "risk factor reductions and modern treatments contributed almost equally," 40 percent treatment and 51 percent prevention.[21(p385)]

Capewell shared IMPACT widely, developing a website for the model and collaborating with researchers in many countries to run analyses on New Zealand, England and Wales, Finland, Ireland, the United States, Sweden, Canada, Italy, Iceland, China, Spain, and Northern Ireland. Although results varied, the analyses almost always shared responsibility with near-equality between reductions in risk factors and improved treatments: 42 percent and 58 percent in England and Wales; 47 percent and 44 percent in the United States.[22,23] Only in Scandinavian countries, such as Finland, where aggressive public health campaigns reduced consumption of dairy fat, did this balance shift (72 percent prevention and 23 percent treatment).[24] A microcosm of decline literature, IMPACT produced remarkably consistent results across time and place: half credit each to prevention and treatment.

IMPACT also has been adapted to predict the impact of interventions and "bring together public health professionals, clinicians, and service commissioners in interactive scenario planning activities to inform policy decisions."[25] For instance, an additional 372,000 deaths could be prevented if Americans achieved "ideal risk-factor levels."[26] IMPACT can also detect disquieting trends. Improvements in cholesterol, blood pressure, and smoking in the United States have been offset by worsening obesity and diabetes.[23] Decline has slowed and even plateaued for younger adults, "potential warning signs" that hard-fought gains might soon be lost.[27]

The most ambitious model, Archimedes, has been developed by David Eddy and Leonard Schlessinger at Kaiser Permanente. Archimedes attempts a "full-scale simulation model of human physiology, diseases, behaviors, interventions, and health care systems." It offers researchers, administrators, and policy makers the chance to "run clinically realistic virtual trials on any population and create compelling evidence to make decisions."[28]

For instance, Eddy's team simulated the impact of eleven prevention activities over thirty years in a representative population ages 20–80.[29] The team found that the interventions could prevent 63 percent of all heart attacks (or 36 percent, using more realistic assumptions about treatment uptake). Aspirin (in high-risk patients), diabetes prevention, and weight loss had the biggest impact. Only one intervention, smoking cessation, was cost saving. The lowest value came from cholesterol reduction in low-risk populations, a finding with "important policy and clinical implications, as it is currently one

of the most heavily promoted of all the prevention activities."[29(p1694)]

Models And Their Discontents

The evolution of the decline debate has been animated by the prospect that historical modeling would inform health care policy by enumerating the relative contributions of risk-factor management and medical interventions. Researchers' ambitions developed in parallel, from explaining the past to predicting the future. But as researchers' models have grown more powerful, they have become less intelligible.

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Archimedes, for instance, has been critiqued for being "extraordinarily opaque."[30] This is a problem for anyone who believes that models are useful only if their inner workings can be understood. Eddy, in response, has argued that transparency is a poor criterion for judging a model. What matters is not how a model works, but how well it works.[31] And even the critics of Archimedes acknowledge that its "results are astounding."[30] Eddy and Schlessinger ran simulations of seventy-four randomized clinical trials of diabetes interventions and then compared their simulated results to the results obtained in the actual trials. They found a correlation coefficient of 0.99. It remains to be seen whether researchers and policy makers will be more swayed by transparency or by accuracy.

The inner workings of IMPACT, in contrast, are accessible to motivated readers. Close analysis reveals several important features.

First, its outcome and implications are malleable: IMPACT can be run with different endpoints that yield different assessments. Capewell developed IMPACT to analyze deaths prevented. Beginning in the 2000s, his team ran parallel analyses of "life-years gained." When IMPACT analyzed deaths prevented in the United States, it allocated more credit to treatment than to changes in risk factors (47 percent versus 44 percent). However, when IMPACT analyzed life-years gained, it allocated treatment less credit (35 percent versus 65 percent).[23,32] In England the shift was even starker: from 42 percent and 58 percent to 21 percent and 79 percent.[22,33]

Why the difference? IMPACT calculated life-years gained by multiplying the number of deaths prevented by the median survival after the intervention. Because prevention targets younger and healthier patients, they have longer median survival after the intervention—which accentuates the benefit of prevention.[34] Researchers can conduct either analysis, aware of the diverging policy implications.

Second, such models apply a thin veneer of specificity atop a messy foundation. IMPACT requires researchers to reduce the efficacy of each treatment to a single coefficient.[35] When conflicting data exist, researchers seek the most recent, least biased, and most representative estimates.[23] Researchers also estimate compliance rates, with a range from 50 percent among asymptomatic outpatients to 100 percent in hospitalized patients. Both sets of estimates are, themselves, uncertain.[21]

Estimating the contribution of risk-factor reductions remains an even "less precise science."[35(p30)] In Finland, for instance, risk factors explained either 53 percent or 71.8 percent of the decline, depending on whether the researchers derived their coefficients from Finnish or international studies. Latitude in the estimates of these parameters—efficacy, compliance, and all the others—raises an important question. Since the 1970s, analyses of coronary heart disease decline have generally assigned equal credit to prevention and medical care. Does this mean that the analyses have reliably revealed a correct answer? It might also mean that researchers' expectations have subtly influenced their methods and produced expedient results.

Third, these models can sweep uncertainty under the carpet. Because of the potential variation in parameter estimates, IMPACT analyses include an analysis of extremes, using "maximum and minimum feasible values" to produce a range of estimates of deaths prevented.[21(p385)] For the United States, sensitivity analysis showed that the modeled parameters could explain anywhere from 51 percent to 160 percent of the decline, not just the 91 percent advertised in the abstract.[23] Thus, the model could explain nearly all of the decline, or half of the decline, or substantial decline that had not actually happened.

Fourth, ambiguity about what IMPACT does or does not leave unexplained focuses attention on another issue: IMPACT and the other models can analyze only those factors that have been quantified and measured. It is no accident that they focus on the usual suspects, including smoking,

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blood pressure, body mass index, and the impact of specific medical interventions. Variables that are less easily measured, such as stress or social context, get left out. Social epidemiologist Michael Marmot conceded this point in his own foray into the debate in 1984: "'Stress' is excluded from discussions of trends in mortality because of conceptual, definitional and measurement difficulties."[36]

Because social factors remain unmodeled, IMPACT researchers can attribute shortfalls to these "other, unmeasured risk factors."[22(p1106)] The magnitude of this "other" could be as high as 24 percent, as it is in Finland.[24] Analyses of life-years gained erased the "other" altogether.[32,33] When one epidemiologist pointed out that the analysis of the United States had ignored the important role played by reduced air pollution,[37] Capewell and his coauthor offered a complex response. Although air pollution and other risk factors might account for the 9 percent unexplained, it was also possible that "imprecision in the measurement and modeling of the major risk factors (cholesterol, smoking, and blood pressure) might also account for much of the gap."[38] How likely was this? That depends on the model's robustness. With the sensitivity analysis revealing that IMPACT accounted for anywhere between 51 percent and 160 percent of the actual decline, the "other" might be responsible for as much as 49 percent.

The role of nontraditional risk factors remains controversial. As cardiovascular epidemiologists developed their models, social epidemiologists sought different causal explanations. Marmot's Whitehall Study demonstrated that coronary heart disease mortality correlated powerfully with occupational grade within the British civil service.[39] Subsequent work linked mortality to relative position in any status hierarchy, whether of education, income, or control.[40]

The potential importance of social factors can be seen in the United States, where decline followed different trajectories in different parts of the country. Decline began in California, and then in other regions in the West and Northeast, before spreading from the coasts to the interior and from cities to rural areas.[7,8] Speakers at the Decline Conference recognized that these disparities held clues to causes of the decline, including socioeconomic status and lifestyle.[9]

Other researchers have downplayed the significance of social variables. A 2001 review concluded that 75 percent of all coronary heart disease deaths could be attributed to the three major risk factors: cholesterol, blood pressure, and cigarettes.[41] One team reanalyzed the Whitehall results and argued that Marmot's mortality gradients were substantially explained by risk-factor gradients along the occupational hierarchy.[42]

Capewell's team, aware of the potential role of diet, stress, or poverty, has modeled those factors when adequate data exist. They found, for instance, that decreased deprivation (that is, economic development) accounted for 3.4 percent of the coronary heart disease decline in England and Wales.[22]

Such modeling remains a work in progress.[26] In the meantime, researchers must balance the appeal of the quantitative models against awareness of the potentially important factors that they exclude.

One last point deserves mention. Researchers have validated their models by testing how well they match the observed historical changes in risk factors, health care, and outcomes. However, the ability of a model to explain the past is not a perfect marker of its ability to predict the future. This will be especially true if the models are used in attempts to understand the emerging epidemic of coronary heart disease in developing countries.

Conclusion

Protagonists in the decline debate have long sought answers that would guide policy choices between prevention campaigns, such as education targeting populations to bring about lifestyle change and reduce risk factors, and medical care.

Sometimes researchers have been ecumenical. The final report of the Decline Conference assumed that changes in risk factors and improved medical care had both contributed.[9] Sometimes they have been oppositional. Earl Ford and Capewell subtitled their 2011 review "Public Health Versus Clinical Care."[26] The debate, as a result, has perpetuated long-standing tensions between medicine and public health,[43] even as it offered an olive branch by crediting both with

substantial contributions to past decline and by offering each a substantial role in future policy.

Lost in the debate is recognition of how much the categories of "medical care" and "prevention" have changed. Prevention once meant ensuring the healthiness of environments: clean air, clean water, and clean food. In the closing decades of the twentieth century, prevention has metamorphosed and been integrated into biomedical regimes of surveillance and control. Future health is increasingly ensured through compliance with pharmaceutical regimens, whether for diabetes, hypertension, or high cholesterol. The persistent debate between treatment and prevention polices is a boundary that becomes less meaningful each year. The role of social forces in producing and ameliorating disease, in contrast, has largely been left without a voice in the debates.

Is it likely, in the end, that the decline debate and its models will provide useful guidance to policy makers? When the debate began, no one doubted whether particular preventive or therapeutic interventions had potential value: Nearly all of them do. The question was whether researchers could demonstrate a large enough differential value to justify difficult decisions about resource investment. This has not happened.

Instead of favoring prevention or treatment, researchers' findings have motivated calls for increased investment in both. Better use of evidence-based therapies could save even more lives. More aggressive campaigns against risk factors could prevent even more deaths.[44] The United States needed a "comprehensive strategy," as did Finland and England and Wales.[22–24,26]

Given the powerful interests at stake, it is no surprise that expedient results—half credit each to medicine and public health—emerged time and time again. As Lewis Carroll's Dodo bird observed to Alice, "*Everybody* has won, and *all* must have prizes." Such conclusions provide little guidance to policy makers. But even if a definitive answer did emerge for or against a particular strategy, should health policy necessarily follow suit? The Archimedes analysis of coronary heart disease, for instance, did make a strong critique of statins and cholesterol-lowering medications.[29]

Confronted with such findings, policy makers face a difficult challenge. First, they must decide whether they can trust the result. Models, as do randomized clinical trials, need to be read critically. Whether policy makers scrutinize a model's inner workings (for example, IMPACT) or check its validation studies (for example, Archimedes), they must make the effort to understand the quality of the result. If the model passes muster, then policy makers must weigh its results against other factors that inevitably influence decisions.

When the IMPACT team examined this process in 2011, its members were dismayed by what they found.[45] Policy makers felt that existing research was too uncertain, had poor local applicability, paid too little attention to social determinants, and was poorly communicated. They often gave more weight to their own intuitions, expert consensus, public opinion, stakeholder pressure, financial sustainability, and political viability.

Researchers and policy makers face a delicate situation. As the Patient-Centered Outcomes Research Institute, created by the Affordable Care Act, takes shape amid increasing pressure to improve the efficiency and quality of health services, stakeholders will demand that research findings actually guide health policy. But it is unlikely that any single model or research study will produce findings that are clear and reliable enough to justify transformative policy.

Capewell's team has emphasized that both researchers and policy makers must be aware of how "the concept of evidence is negotiated and socially constructed by and between individuals."[45] Policy makers must work to understand what kinds of knowledge are produced as well as obscured by researchers' analyses. They must learn what lessons can be drawn from a particular study despite its limitations, and they must be consciously aware of what else they consider (and the many limits of these considerations) when they formulate policy. Just as they must open the "black box" of medical research and modeling, they must open the "black box" of their own decision-making processes. This will not guarantee perfect policy, but it will at least make clear how and why the policy was made. ■

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NOTES

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In this month's *Health Affairs*, David Jones and Jeremy Greene chronicle the forty-year debate over the relative contributions of prevention and treatment to the decline in cardiovascular disease mortality. Despite the advent of sophisticated data sets and modeling techniques, they observe that a definitive resolution to the debate hasn't emerged, and thus there is no specific guidance for health policy. The authors' multilayered analysis leads them to call on policy makers to open up the "black box" of epidemiological models in formulating policy based on the best available evidence.

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