Death by Regulation

How Regulations Can Increase Mortality Risk

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Abstract

This paper updates the cost-per-life-saved cutoff, which is a cost-effectiveness threshold for life-saving regulations, whereby regulations costing more per life saved than this threshold level are expected to increase mortality risk on net. Two competing methods of deriving the cutoff exist: a direct approach based on empirical observation and an indirect approach grounded in economic theory. Both methods build from the assumption that changes in income lead to changes in mortality risk. The likely mechanisms driving this relationship are discussed, with support from recent empirical studies. The indirect approach is preferable in that it avoids the problems of endogeneity of health status and income found with the direct approach. The cost-per-life-saved cutoff value at which regulations increase mortality risk is estimated to have a lower bound value of $75.4 million and an upper bound value of $123.2 million, with a midpoint value of $99.3 million. This cutoff value range is compared with cost-effectiveness estimates for a series of recent policies, including several state expansions of the Medicaid public insurance program in the first few years of the 21st century, an early version of the "travel ban" executive order that restricted refugee admissions into the United States, and nine recent air pollution regulations from the Environmental Protection Agency. The paper concludes that the mortality risk test is an important and underutilized tool in the policy analyst's toolkit, both as an overall test of regulatory efficacy and as an integral component of calculations of net risk effects of policies.

Keywords: cost-per-life-saved cutoff, value of a statistical life, benefit-cost analysis, cost-effectiveness analysis, health-health analysis, welfare analysis

JEL codes: D6, I1, K2, K3

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1. Introduction

Regulations of various kinds seek to reduce mortality risk. Typically, such rules relate to health, safety, security, and the environment. While it is obvious how regulations can reduce the risk of death, since reducing risk is often the primary aim of regulations, it is less obvious how rules might also increase mortality risk. Nonetheless, many regulations result in unintended consequences that increase mortality risk in various ways. These adverse repercussions are often the result of regulatory impacts that compete with the intended goal of the regulation, or they are direct behavioral responses to regulation.

As examples, fuel efficiency regulations can encourage automobile manufacturers to produce smaller cars that are more dangerous in an accident (Crandall and Graham 1989). Increased airport security measures after 9/11 made air travel more inconvenient, which has led to increases in estimated car accident deaths as individuals substituted driving for flying (Blalock, Kadiyali, and Simon 2007). And construction activities or handling of equipment when engaged in compliance activities can lead to accidents (Viscusi and Zeckhauser 1994). There are also negative health consequences associated with unemployment (Burgard, Brand, and House 2007; Eliason and Storrie 2009; Sullivan and Wachter 2009; Strully 2009), which can be the result of regulatory policies. Finally, regulatory efforts reduce individual expenditures on health, both because risk reduction achieved through regulation is a substitute for private risk reduction and because the costs incurred by regulations reduce private health-related expenditures.

It is this last item that has been the focus of *health-health analysis* (HHA), the analytic tool used to evaluate the mortality risk effects of regulations (Lutter and Morrall 1994). *Health-health analysis* is something of a misnomer, since it implies that the analysis is used to assess the total net health effects of regulatory policies. In practice, however, HHA has been used exclusively to evaluate the effects on one health risk in particular—mortality risk.

While in principle HHA could be extended to focus on all health-related risks, there are some compelling reasons for focusing exclusively on mortality risk. Mortality risk reduction benefits constitute the largest component of most risk-reducing regulatory policies. Mortality risks are easier to measure than other health risks, which makes estimates of mortality risk effects of regulations more reliable, at least with present data. In many instances, agencies do not attempt to quantify health impacts other than those pertaining to mortality risks. Additionally, given the statutory goals guiding regulatory policies, one would expect regulations to reduce mortality risks on net, not increase them.

Despite a robust academic literature that spans decades, HHA has not become widely used by policymakers, perhaps in part because its earliest uses by policymakers generated controversy. HHA's use was controversial largely because of misunderstandings surrounding the analytic tool. For example, in 1992, the Office of Management and Budget (OMB) suspended review of a proposed regulation from the Occupational Safety and Health Administration (OSHA) on the grounds that OSHA's regulation might increase mortality risk. The rule targeted air contaminants from construction and maritime activities, and in a return letter from the OMB to OSHA, OMB analysts produced a back-of-the-envelope HHA.[1] OMB's analysts suggested

[1] See letter of James MacRae, Acting Administrator and Deputy Administrator, Office of Information and Regulatory Affairs, OMB, to Nancy Risque-Rohrbach, Assistant Secretary for Policy, Department of Labor, March 10, 1992.

that the rule might increase mortality risk, which resulted in a firestorm of criticism for making the seemingly bizarre claim that a regulation might increase rather than reduce risk. Two OMB analysts were called to testify before Congress and a US General Accounting Office (GAO) study was requested by the Chairman of the Committee on Government Affairs to explore the matter further.

The GAO study, published later in 1992, would be highly critical of OMB's use of HHA (GAO 1992) on the grounds that the income-mortality relationship, upon which early versions of HHA were based, is an observable correlation, but that causation had not yet been established. GAO also claimed that HHA is a form of benefit-cost analysis (BCA), which OSHA was precluded from using in the specific case at hand. As will be shown, however, both of these critiques involve a fundamental misunderstanding of health-health analysis.

A principal impetus for using HHA is that it does not involve benefit-cost balancing. While the approach does require *estimation* of costs, it does not require any consideration of costs relative to benefits. Furthermore, the final decision metric in HHA is not presented in dollar terms, like estimates of cost. Rather, HHA is an assessment of whether on balance a regulatory effort increases mortality risk or decreases mortality risk, which is a consideration that is consistent with agencies' statutory mandates.

One need not rely on correlations between income and mortality to conduct HHA either. HHA relies on an estimate of what is known as the cost-per-life-saved cutoff (the "cutoff"), which is a threshold cost-effectiveness level beyond which life-saving regulations will be counterproductive in that they can be expected to induce more fatalities than they prevent. This cutoff also establishes the rate at which expenditures lead to mortality risks, thus making it possible to assess the net mortality risk effects of policies generally.

There are two competing ways of identifying the cutoff, a direct approach based on empirical observation and an indirect approach grounded in economic theory. The direct approach relies on correlations between income and mortality and attempts to root out problems of endogeneity by controlling for variables such as initial health status and education. It is the direct approach that has led to misunderstandings about whether correlation is being confused with causation. Criticisms of the direct approach have merit in that studies employing the approach have likely underestimated the cutoff value. However, such criticisms do not justify abandoning the use of HHA or imply that the cutoff value is infinite.

The indirect approach, which is our preferred method, relies on a theoretical model of the income-mortality relationship that is calibrated using data on the value of a statistical life (VSL) and the marginal propensity to spend on health (MPSH). The indirect approach avoids problems of endogeneity found in the direct approach and is closely linked to the VSL concept, a well-accepted and widely used measure of risk valuation in BCA.

Studies using the direct approach have estimated the cutoff at between $8 million and $26 million (2015 dollars) for the United States; however, these estimates are likely to be too low, given the nature of the simultaneity bias. Employing the indirect approach has led to a cost-per-life-saved cutoff value closer to $85 million for the United States. We employ the indirect approach here as well, estimating a cutoff range from $75.4 million to $123.2 million (2015 dollars). A reasonable rule of thumb might be to assume that regulations costing more than $100 million per life saved will be counterproductive in that they can be expected to increase mortality risk on net.

To put this number in perspective, in 2014 there were 135,928 unintentional injury deaths in the United States (US Centers for Disease Control 2016). US GDP in 2014 was $17.68

trillion (2015 dollars), according to the US Bureau of Economic Analysis. Thus, the GDP divided by the total number of accidental deaths is approximately $130 million per death, which is close to the $100 million figure and just above the high end of our range of estimates for the cost-per-life-saved cutoff. This finding implies that devoting the nation's entire GDP to preventing accidental deaths would not only exhaust all of society's resources, but would also be expected to induce slightly more fatalities via health-health tradeoffs than would be saved directly through the effort.

We compare our cutoff value range with cost-effectiveness estimates for several recent policies that aim to reduce mortality risk. Several state expansions of the Medicaid public insurance program in the first few years of the 21st century appear to have reduced mortality risk. By contrast, an early version of President Trump's controversial "travel ban" executive order, which cut refugee admissions into the United States, may have counterproductive mortality risk effects, according to one back-of-the-envelope estimate.

Comparing the cutoff value range with mean cost-effectiveness estimates for nine recent air pollution regulations from the Environmental Protection Agency suggests mortality risk is decreased on net from these regulations. There are reasons to doubt these mean estimates, however. Expert elicitation surveys reveal that uncertainty is greater for these rules than is typically acknowledged, and benefits estimates are based on a series of assumptions of uncertain validity, which tend to err on the side of increasing benefits. The possibility that mortality risk is increased from these rules lies within reasonable bounds of uncertainty.

This paper is organized as follows. Section 2 discusses the channels by which income losses are likely to increase mortality risk and provides empirical support for these mechanisms. Section 3 reviews estimates of the cutoff employing the direct approach. Section 4 reviews some

criticisms of the direct approach and provides responses to these criticisms. Section 5 presents the alternative indirect approach. Section 6 calibrates the theoretical model of the income-mortality relationship with updated estimates of the VSL and the MPSH. The updated cost-per-life-saved cutoff is then compared to estimates of the cost-effectiveness of recent policies aimed at saving lives. Section 7 concludes by discussing the relative merits of HHA both as a component of BCA and as an alternative to BCA.

2. Channels Linking Income and Health

The most direct mechanism by which changes in income will lead to changes in mortality risk are the changes in health-related expenditures that follow from changes in income. Several recent studies find that expanding health insurance coverage to uninsured populations is associated with reduced mortality risk (Sommers 2017; Sommers, Long, and Baicker 2014; Sommers, Baicker, and Epstein 2012). Presumably the mechanism by which this occurs is increased health-related expenditures arising from individuals gaining health insurance coverage. Outside of mortality risk, expanding insurance tends to produce more mixed effects with respect to health generally (e.g., Baicker et al. 2013; Marmot et al. 1991; Newhouse 1993). Nonetheless, these findings are broadly consistent with increases in income reducing mortality risk through the effect on health-related expenditures.

Another literature attempts to identify the causal impact of income on general health. The literature offers important insights as to the mechanisms by which income losses increase health-related risks and, by extension, likely increase the risk of death and reduce life expectancy. Most of these studies are instructive in that they document different linkages that establish a positive causal relationship between income and health status, but they do not necessarily yield a specific estimate of the overall income-mortality risk relationship. Although health risks are not exactly

the same thing as mortality risk (which is the focus of HHA), the two concepts are clearly related, meaning this literature is relevant to the debate about the income-mortality gradient.

There are two mechanisms in particular identified as ways in which income changes likely translate into health outcomes: mental health and early childhood socioeconomic status. With regard to mental health, the psychological stress that often follows negative income shocks can lead to behavioral responses, such as increases in risky behavior. Prolonged periods of stress can also lead to elevated use of the body's physiological systems, which leads to health problems (J. P. Smith 1999).

Currie (2009) provides a literature review that supports the general conclusion that parental economic status influences child health and other long-run outcomes, such as adult income and education levels, that can determine adult health. Fetal health may be a particularly important predictor of these future outcomes. Case et al. (2005) show how childhood health and economic circumstances predict future educational attainment levels, earnings, and health, and a myriad of other studies also link childhood socioeconomic status to long-run health outcomes (e.g., Case, Lee, and Paxson 2008; Case, Lubotsky, and Paxson 2002; Currie and Moretti 2003; Currie and Rossin-Slater 2015; Currie and Stabile 2003; Khanam, Nghiem, and Connelly 2009; Propper, Rigg, and Burgess 2007).

These two possible drivers of the income-health relationship also share some common characteristics. They are both long-run in nature, suggesting the health impacts of income shocks are often not experienced immediately. Both mechanisms are likely to be hard to identify in studies relying on relatively few years of data for this reason. And disentangling the effects of education from the underlying circumstances of one's childhood upbringing is likely to be difficult.

Despite these challenges, a variety of studies do find that income shocks influence health outcomes. One relatively recent example looks at health outcomes for Native Americans on tribal land where casinos have been built (Wolfe et al. 2012). Since casino revenue is often used to supplement tribe members' incomes or to build public infrastructure on tribal lands, a casino opening acts as an instrument for an exogenous income shock. The study reports a reduction in health problems such as obesity, hypertension, diabetes, and anxiety, as well as lower rates of many risky behaviors, such as smoking and heavy drinking, following the building of a casino. Similarly, Evans and Topoleski (2002) find a 2 percent reduction in mortality four or more years after a casino opening in a county, with additional mortality declines of about half this magnitude in neighboring counties. Costello et al. (2003) look at a Native American casino opening in North Carolina and find significant reductions in some types of childhood psychological disorders, suggesting a link between income and children's mental health.

Some studies use lottery winners to identify the effect of exogenous income shocks on health. For example, a study of Swedish lottery players found that winning SEK 100,000 increases general health and decreases the probability of dying (Lindahl 2005). Although not all studies make a distinction between physical and mental health, those that do often find that winning the lottery has a greater impact on mental health than it does on physical health. For example, Cesarini et al. (2016) look at Swedish lottery winners, finding no evidence that wealth impacts adult mortality or healthcare utilization, though the authors find evidence of improvements in mental health. Similarly, Apouey and Clark (2015) look at lottery winners in the United Kingdom and find no effect of income on self-reported measures of general health, but the authors do find positive effects with respect to mental health. Gardner and Oswald (2007)

used surveys in Britain to assess whether mental well-being is improved for lottery winners and find a significant positive effect of income on psychological health.

Other studies use exogenous events or employ novel statistical techniques to identify the causal influence of income and wealth on health. For example, Frijters, Haisken-DeNew, and Shields (2005) used the reunification of East and West Germany as a natural experiment to test the effect of income on measures of self-reported health. A significant and positive effect is found, though the authors note the magnitude of the change is small.

A study by Snyder and Evans (2002) exploits an exogenous change in US Social Security law that led to higher payments for individuals born before January 1, 1917—the so-called "Social Security notch"—relative to those born after this date. Strangely, higher-income individuals actually experienced *higher* mortality rates compared to the lower-income group, as well as lower rates of employment. This is consistent with health becoming more resistant to income shocks with age, reinforcing the idea that income shocks may matter more for children than for adults. Adda, von Gaudecker, and Banks (2009) created a synthetic cohort using longitudinal data over a 25-year period and find no effect of income shocks on a wide range of health measures; but interestingly, their study does find a significant effect on mortality.

Ettner (1996) employed an instrumental variables approach to estimate the effect of income on several measures of physical and mental health. The author used four different instruments, finding significant improvements in both physical and mental health associated with higher income. Several of the instruments used involve measures of education, so to some degree, the study may blur the effects of income, education, and early childhood socioeconomic status.

Some studies have looked at how income is related to health in developing countries. Pritchett and Summers (1996) used an instrumental variables approach in a cross-country context

and find that income reduces child and infant mortality and increases life expectancy. Case (2001) looks at outcomes from changes in a government pension program in South Africa. In households that pooled income, the author finds significant improvements in physical health for those who received the income as well as for all other household members, suggesting those who receive funds directly are not be the only beneficiaries of those funds.

Fernald, Gertler, and Neufeld (2008) used a randomized control trial experiment conducted in Mexico, where low-income communities were randomly assigned to participate in a cash transfer program. The authors find significant improvements in children's height and body-mass index, a lower prevalence of being overweight, and improvements in motor and cognitive development. Gertler (2004) finds similar improvements in child health from the same program, noting significant declines in child illnesses.

A more speculative channel linking income and health relates to inequality in relative social rank. Such social inequality has been found to raise levels of what is known as "psychosocial stress," which is "the wear and tear that comes from subordinate status and from having little control over one's own life" (Cutler, Deaton, and Lleras-Muney 2006, 114). Psychosocial stress may negatively impact endocrine levels and immunological processes (J. P. Smith 1999), and some researchers believe this kind of stress is a critical determinant of public health outcomes (Wilkinson 1996). For example, psychosocial stress might explain why health inequalities persist across social classes, such as across the ranks of British civil servants (Marmot et al. 1991), even after dramatic expansion of access to healthcare services.

While some studies find little or no effect of income on health or mortality, these studies typically look at short-run impacts only, focus on adults rather than children, or do not assess mental health outcomes. For example, Elesh and Lefcowitz's (1977) study involved a randomized

control trial experiment in New Jersey and Pennsylvania where low-income families were randomly assigned to different negative income tax plans. The authors find no effect of higher income on several measures of health or on utilization of healthcare services. However, the experiment was limited to three years and did not assess mental health outcomes. Similarly, J. P. Smith (2004) used stock market data to study the effects of exogenous wealth increases on health, finding shocks to wealth do not predict significant health changes over a time span of eight years, but he does not distinguish between mental and physical health. An unconditional cash transfer program in Ecuador also found no improvement in children's health outcomes, although there were some modest improvements among the poorest children (Paxson and Schady 2010).

There is also mixed evidence from inheritances, which is sometimes used as an instrument for income shocks. Limited findings in this area could be explained in part because inheritances are often anticipated, thereby leading to an underestimation of the effect of income or wealth shocks on health. Meer, Miller, and Rosen (2003) look at inheritance data and find no significant short-run effect of wealth shocks on self-reported physical health; the study does not look at mental health. Kim and Ruhm (2012) find that bequests have no significant impact on overall health or mortality, despite increases in healthcare spending and utilization of medical services, though they do find some positive effects with respect to quality of life and rates of depression.

Despite the lack of a significant effect found in some studies, most findings are consistent with a long-run relationship between income and mortality. Many studies find a positive effect of income on physical health, mental health, or health of children. Of those studies that don't find an effect, most look at only a few years of data, focus only on adults older individuals, or do not distinguish between mental and physical health. Furthermore, a

relatively new literature on the expansion of health insurance to uninsured populations finds significant impacts with respect to mortality.

The complex and long-run nature of the relationship between income and mortality is one reason why estimating the magnitude of the effect based on empirical data alone is likely to be insufficient, and an approach grounded in consensus economic theory, supplemented with empirical data, may be superior. While no theoretical model is perfect, over time and with gradual iterative improvements, future research and advances in knowledge will help add more dimensions and realism to any model of the income-mortality relationship.

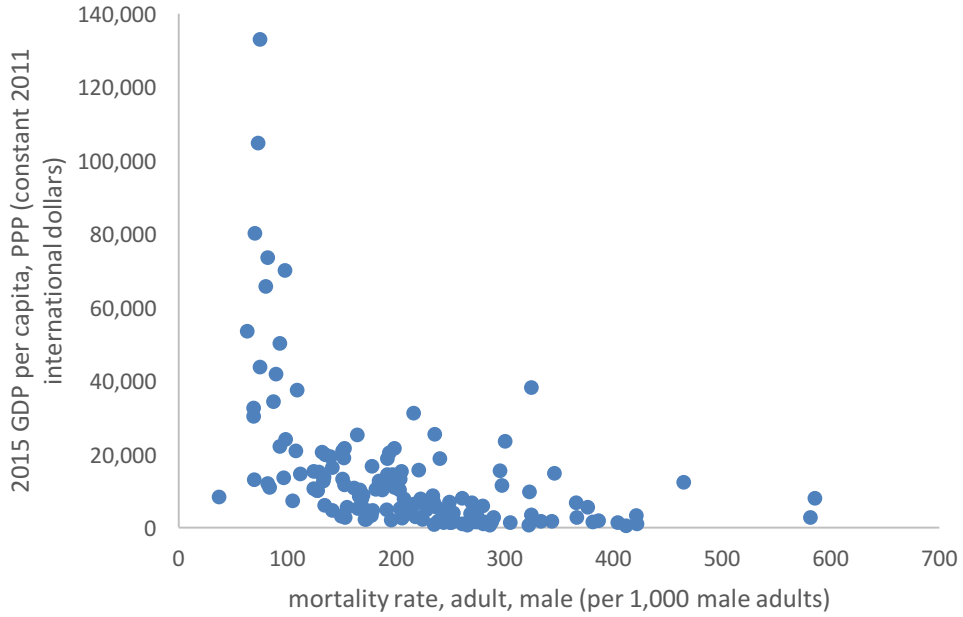
3. A Review of Studies Employing the Direct Approach to Estimate the Cutoff

The idea underlying HHA is that public policies save lives but corresponding reductions in private spending to reduce risks also induce losses of life. This relationship has come to be known as the "richer is safer" hypothesis and is often associated with Berkeley political scientist Aaron Wildavsky (Wildavsky 1981). Although the idea that regulatory interventions increase mortality risk may be controversial, it should not be. Reductions in personal income will inevitably increase mortality risk so long as some nonzero fraction of income is spent on risk reduction. Only in the extreme case where no income at all is spent on risk reduction, or when expenditures are completely ineffective, will regulatory interventions have zero negative consequences with respect to mortality risk.

This is no doubt one reason why there is a strong correlation between income and mortality. Figure 1 plots the relationship globally between income per capita and the mortality rate of adult men in 2015. Though this is a cross-country relationship examined at a moment in time, income is also negatively correlated with mortality in the United States (Dowd et al. 2011),

is positively associated with life expectancy in the United States (Chetty et al. 2016), and is negatively associated with accident rates across the US states (Viscusi 1983).

Figure 1. Income and Mortality (1953–2013): Deaths per 1,000 People per Year



Source: World Bank Development Indicators.

The key question is not whether income causes better health and reduced mortality risks—there can be little doubt that higher incomes are used to offset health and mortality risks of numerous kinds. Rather, the key question regards the magnitude of the effect and the degree of uncertainty surrounding estimates of the magnitude.

Decision scientist Ralph Keeney developed the first formal model for estimating fatalities induced by income losses, finding that for every $7.25 million (1980 dollars) in costs, one statistical fatality will be induced (Keeney 1990). Chapman and Hariharan's (1994) study, published in a special issue of the *Journal of Risk and Uncertainty* devoted to HHA, develops a similar empirical model but controls for initial health status as a means to account for reverse

causality (i.e., poor health causing lower income). The study's authors estimate the cutoff at $12.2 million (1990 dollars).

Keeney provided an update of his model in 1997, estimating the cutoff at between $5 million and $14 million (1991 dollars), depending on the distribution of costs. The study notes that the cutoff is likely to be lower for low-income individuals and for African Americans (Keeney 1997). This emphasis on distributional impacts would show up in other studies as well. For example, Chapman and Hariharan (1996) estimate that the cutoff is about twice as high for the richest 20 percent of the population as for the poorest 20 percent.

Finding different cutoff values at different income levels should not be surprising given the likely diminishing marginal efficacy of income in improving individual health. A loss of income represents a larger share of a poor person's budget. Additionally, those who live in poorer areas face higher levels of risk across numerous aspects of their lives.[2]

Kuchler et al. (1999) produced one the few studies that attempts to conduct an original health-health analysis for a regulation, exploring the repercussions of a potential oyster harvesting ban in the Gulf of Mexico. Using the Keeney (1997) estimate of the cutoff, and combining this figure with projections of income losses to fisherman that would result from a ban, these authors estimated that the ban, if implemented, would induce 8–12 fatalities annually, while preventing roughly 17 premature deaths annually from food poisoning. However, application of HHA to other regulatory policies finds that the risk reduction balance is often less favorable. Hahn et al. (2000) conducted HHA for 24 federal regulations, estimating that a majority of the regulations in their sample increase mortality risk, though aggregate mortality risk was estimated to fall for all regulations together.

[2] See table 4 in Thomas (2011, 27) for examples of risks that are higher in low-income counties relative to high-income counties.

Although relatively little has been written on health tradeoffs by American scholars after the year 2000, a new literature began to emerge around the turn of the century from scholars in other countries, most notably in Scandinavia. Elvik (1999) is a Norwegian study that estimated the cutoff in Norway at between 25 million and 317 million NOK (1995 prices), which translates to US$3.8 million to US$47.5 million (1995 US dollars). Gerdtham and Johannesson (2002) used longitudinal data (tracking individuals for between 10 and 17 years) for a sample of randomly selected Swedes. After controlling for initial health status, they estimated the cutoff at between US$6.8 million and US$9.8 million (1996 US dollars), depending on how costs are distributed.

More recently, Ashe et al. (2012) examined fire prevention regulations in Australia. These authors estimate the cutoff at between AU$20 million and AU$50 million (2010 Australian dollars), again depending on how costs are distributed across the population. They did not conduct a formal analysis of the net mortality effects of fire prevention efforts. However, the authors compare estimates of induced deaths (90 to 225 fatalities per year) to the 114 lives lost on average to fires each year in Australia, suggesting the costs incurred by fire prevention efforts may be excessive given the current size of the problem. However, because the number of prevented deaths is unknown, it is unclear whether fire prevention efforts in Australia pass a mortality risk test.

4. Criticisms of the Direct Approach

The models and data used to estimate the cost-per-life-saved cutoff under the direct approach have been subjected to a number of criticisms. Among the first to criticize these models was Sinsheimer (1991), who criticized the Keeney (1990) model on two grounds: ecological fallacy and confounding bias. Ecological fallacy refers to making inferences about individual

phenomena on the basis of the observations of groups. The Keeney study used census tract data but inferred a causal relationship between individual income and mortality based on this group data. This issue would later be corrected in Keeney (1997), which used individual-level data rather than census tract data.

Confounding bias, on the other hand, which is more commonly referred to as omitted variables bias, refers to the possibility that an unobserved variable or variables influence both income and mortality, leading either to a spurious correlation or to an overestimation of the impact of income on mortality. A similar criticism relates to the degree to which the correlation between income and health can be explained by the causal effect of health on income (i.e., reverse causality).

The 1994 special issue of the *Journal of Risk and Uncertainty* included other criticisms of the direct approach and HHA more generally. Portney and Stavins (1994), for example, argue that Congress already implicitly considers health tradeoffs when legislation is crafted, implying HHA is redundant. These authors further assert that negative health outcomes as a result of regulations are only likely in cases of large income losses (e.g., due to unemployment).

Contrary to these claims, the evidence suggests that the kinds of tradeoffs considered in HHA are often ignored, even in the rare cases where significant data and analysis are available to legislators. For example, Congress appears to have ignored a relevant benefit-cost analysis produced by the Department of Transportation when it mandated positive train control technology to improve rail safety (Ellig and Horney 2016). Furthermore, even small per-capita income losses are likely to result in expected fatalities when those losses are spread across large populations. Keeney (1997) found little difference in estimated fatalities when comparing a scenario in which costs are concentrated on an industry to a scenario in which costs are spread out across society.

Portney and Stavins also stress that most versions of HHA ignore morbidity risk and focus exclusively on mortality risk. This is no doubt true. HHA as presently practiced is a mortality risk analysis, not a broader health risk analysis (despite the name "health-health analysis"). However, many government policy analyses also only quantify and monetize mortality risk reductions so that the failure to incorporate morbidity risks in HHA may not be a pertinent oversight. So long as mortality risk reduction benefits comprise the dominant health benefit estimate for government policies, any such neglect of morbidity risk offsets will be of subsidiary importance. Moreover, there is no reason why morbidity risks couldn't become more central to HHA. One way to do this would be to convert estimates of morbidity risk into mortality risk equivalents using implicit labor market estimates of the value of injuries, as was done in the early 1980s in the analysis of the OSHA hazard communication regulation (Viscusi 1992).

Even if other health risks are ignored, it is important for policymakers to know whether a policy on net increases mortality risk. Armed with such information, policymakers could use HHA as a screening device to weed out particularly ineffective regulations. It is hard to imagine that many would defend regulations that, on net, increase rather than reduce mortality risk, even if there might be other coincidental health-related benefits of the regulation. As a practical matter, few such situations are likely to arise to the extent that regulations have correlated impacts on different health risks.

V. K. Smith et al. (1994) criticize the use of cross-country data in some HHA studies (a variation of the ecological fallacy argument). Using the same data as Lutter and Morrall (1994), a study discussed in more detail below, Smith and coauthors find that by adding additional controls, the relationship between income and health is no longer significant and

falls to one-tenth the magnitude in the Lutter and Morrall study, suggesting omitted variables bias may be influential.

J. P. Smith (1999) is perhaps the most conclusive study establishing that adverse health causes lower income as well as lower wealth. This study demonstrates that health-induced income shocks explain a significant portion of the income-health gradient. In fact, the author notes that health is relatively insensitive to income shocks, especially for older individuals. There is also considerable evidence that the onset of adverse health reduces household wealth and leads individuals to dip into savings or into bequests to pay health expenses. Nonetheless, J. P. Smith (1999, 165) is careful to note, "economic resources also appear to impact health outcomes, [and] this may be most acute during childhood and early adulthood when health levels and trajectories are being established."

Cutler et al. (2006) argue that by adding additional controls, especially education, most of the effect of income on health disappears, seeming to affirm the relevance of omitted variables bias. These authors go so far as to say, "Conditional on education, which acts as a form of protection against new episodes of illness, changes in income do not predict changes in health, and lagged income does not predict future incidence of ill health."

This conclusion is likely overstated, however. For one thing, that income may not be correlated with mortality after controlling for education only highlights the difficulty in isolating short- and long-run effects. Given that individuals growing up in wealthier families are likelier to obtain higher levels of education, it is unclear whether the coefficient on education is confounded by other factors, such as early childhood socioeconomic status. In fact, educational attainment may be a critical pathway linking income and wealth in childhood to better health and lower mortality rates in adulthood.

These criticisms of the direct approach seem to confirm that the effect of income on mortality is complicated; the effect is likely to be difficult to identify in the data and income is likely to have different impacts on health over the lifecycle. There may also be differences between transient income losses and permanent income losses (Graham, Chang, and Evans 1992).

Despite these empirical complications, there is no compelling evidence that the causal effect of income on mortality is zero. On the contrary, there is considerable evidence that the effect is negative and that the effect will be hard to estimate in statistical analyses looking at only short periods of time. These difficulties are not an argument for abandoning health-health analysis; rather, they suggest that an approach grounded in economic theory may be superior to a purely empirical approach, at least with present data and knowledge.

5. The Indirect Approach

Viscusi (1994) developed a theoretical model to estimate the cutoff without estimating correlations between income and mortality. A desirable feature of this alternative to direct estimation is that it incorporates the VSL, which is already a well-accepted and widely used concept in regulatory analysis, as a component of the model.

A person's VSL will guide the level of health-related investments that will be desirable. A person with a higher VSL will find it desirable to expend more funds on health-enhancing efforts than a person with a lower VSL. If the income elasticity of the VSL is positive, that will establish an economic mechanism by which decreases in income will adversely affect health-related expenditures by the individual.

The positive impact of income on the VSL is well established. Estimates of the income elasticity of the VSL are consistently positive. Viscusi and Aldy (2003) estimated a variety of

specifications that have been used in the literature and found income elasticity estimates of the VSL ranging from 0.46 to 0.61. Based on a longer-term historical analysis, Costa and Kahn (2004) estimated an elasticity range from 1.5 to 1.7. Bellavance, Dionne, and Lebeau (2009) estimated an elasticity range from 0.4 to 0.75. Kniesner, Viscusi, and Ziliak (2010) estimated the VSL across the wage distribution, yielding a mean VSL income elasticity across quantiles of 1.44. The OECD's (2012) analysis of stated preference studies yielded an elasticity range from 0.7 to 0.9. The meta-analysis of previous meta-analyses by Doucouliagos, Stanley, and Viscusi (2014) found an income elasticity range from 0.5 to 0.63. Viscusi and Masterman (2017) estimated a US income elasticity of the VSL of 0.5, an international non-US income elasticity of 1.1, and a variation of the income elasticity that increases at lower income levels as in Kniesner, Viscusi, and Ziliak (2012).

Thus, the empirical evidence from the United States and other countries, as well as the evidence from labor market estimates of the VSL and revealed preference studies, indicate a positive income elasticity of the VSL and a greater income elasticity at lower income levels. This economic mechanism is also consistent with the common conjecture that the mortality effects of regulatory expenditures will be greatest for the poorest members of society.

Using the VSL in the model also potentially addresses a puzzling aspect of many of the direct estimates of the cost-per-life-saved cutoff, namely that estimates were surprisingly close to the VSL. If true, this would imply that risk-reducing expenditures observed in the marketplace (such as in the studies estimating the VSL) were almost breakeven efforts that generally led to little net marginal risk reduction. In other words, the costs people incur by spending on risk reduction increase risk levels by a comparable amount to the risk being addressed through spending. This seems implausible.

The cost-per-life-saved cutoff can be estimated using the model originally developed in Viscusi (1994), which begins with an individual who chooses the level of health-enhancing expenditures and the level of job risk (for which the individual is paid a compensating differential), but the analysis generalizes to product risks as well. The optimal decision will equalize the implied marginal value of a statistical life across different risk domains.

When a binding government regulation affects risk levels, there will be two effects. First, because health expenditures and job safety levels are substitutes, regulation will decrease the private incentive to invest in health. Second, because the individual bears regulatory costs, there will be decreased investment in health. Whether a regulation reduces risks on balance depends on the sum of three components: the direct effect of the regulation on safety, the indirect effect on risk through a substitution toward safety achieved through regulation and away from personal health expenditures, and the indirect effect on risk as personal health expenditures fall from reduced income as a result of compliance with regulations.

The key equation to describe this relationship, found in Viscusi (1994, 102), is equation (1):

$$\Delta q = \frac{\partial q}{\partial s}\Delta s + \frac{\partial q}{\partial h}\frac{\partial h}{\partial s}\Delta s + \frac{\partial q}{\partial h}\frac{\partial h}{\partial y}\Delta y. \qquad (1)$$

Here, q represents the worker's probability of survival, s is the level of safety in the workplace, which is set by government regulation, h is the level of health expenditures made by the worker, and y is the worker's income.

The first term on the right-hand side of equation (1) is the direct effect that changing safety standards has on mortality risk. The second term is a substitution effect term that describes how health expenditures and the probability of survival change as a result of changing the safety level. Again, this effect follows from the fact that health expenditures and safety standards set by

government regulation are close substitutes.[3] The final term is an income effect term that describes how health expenditures and the probability of survival change as income changes. If a regulation is to pass a health-health test, then $\Delta q > 0$. In other words, the sum of the three terms on the right-hand side of equation (1) is positive.

At first glance, it may seem odd that healthcare spending is the primary means that the agent in the model invests in mortality risk reduction. Many other forms of spending also address mortality risk, including spending on food, housing, and transportation. What is pleasing about using measures of healthcare spending as a proxy for spending on mortality risk generally is that the primary goal of all health expenditures is to improve health, and presumably by extension to extend life and reduce the risk of death. Unlike health spending, the primary objective of other forms of spending is usually something else (e.g., nourishment, a roof over one's head, travel), even if some fraction of that spending is related to risk. For practical purposes, it will be difficult to identify what fraction of these other forms of spending relates to risk reduction, whereas almost all forms of health spending are presumably aimed at achieving better health and, by extension, reduced risk of death.

Interestingly, equation (1) also highlights how the marginal value of life, $\frac{1}{\frac{\partial q}{\partial h}}$, is endogenous to policy interventions. In other words, what individuals are willing to pay to reduce mortality risk will itself depend on the level of safety set by regulations. This is an example of how insights from HHA can be used to improve estimates of benefits in standard BCA.

After some algebra, Viscusi (1994) derives the cost-per-life-saved cutoff value as equal to the ratio VSL/MPSH. Several studies follow the indirect approach developed by Viscusi

[3] In theory, it is possible that public health expenditures could increase the effectiveness of private spending on risk reduction, making public and private expenditures on risk reduction complements, rather than substitutes. To account for this possibility, one could add an additional term to equation (1) to account for a positive interaction effect between public and private risk mitigation efforts.

(1994), but they modify the core model (and hence the final ratio) in one manner or another, for example by incorporating elasticities of risky behavior. Lutter, Morrall, and Viscusi (1999) supplement the model with income elasticities for cigarette smoking, activity-related illnesses, and alcohol abuse and estimate the cutoff to be roughly $15 million (1990 dollars).

It is possible that behavioral changes might reduce risks more effectively than changes in health expenditures (although the two are related). This likely influenced Randall Lutter and his coauthors (1999), who began with the observation that most deaths are related to lifestyle choices, including overeating, lack of exercise, smoking, and excess alcohol consumption. Yet the link between income and these risky behaviors is complicated. Some risky behaviors rise with income. Dobkin and Puller (2007) looked at the timing of monthly government transfer program payments and found increases in the consumption of illegal drugs, as measured by increases in drug-related hospitalizations and deaths, around the beginning of every month when payments are received. Donald Kenkel and his coauthors (2014) used variation in the earned income tax credit (EITC) as an instrument for income, finding that increases in income are associated with increases in smoking and decreases in smoking cessation. Schmeiser (2009) used longitudinal data in combination with exogenous variation in the EITC to find income increases body mass index and the prevalence of obesity in women.

For their study, Van Kippersluis and Galama (2014) developed a theoretical model (one different from the Viscusi model) that allows wealth shocks to influence health in either positive or negative directions. These authors calibrated their model using data from lotteries and inheritances, finding changes in health as well as changes in risky behaviors that vary by groups, depending on initial income and health levels before a wealth shock. Specifically, these authors found that the least healthy members of society change unhealthy behavior very little in response

to positive wealth shocks. The authors found that, on average, risky activity increases in response to positive wealth shocks, but there are differences across risks. There is a higher likelihood of engaging in risky activity when the relative risk from the consumption good is low, which could explain why moderate drinking often rises in response to wealth shocks (a common finding in some of the studies in section 2).

Given these findings, we have chosen to forgo including elasticities of risky behaviors in our model, although with more information the model could certainly be expanded in the future to account for the different effects of income on risky behaviors. Oddly, there are only two estimates of the cutoff using the indirect approach that do not supplement the model with elasticities of various types. One is Viscusi (1994), which estimates the cutoff to be $50 million (1990 dollars). The other is Hjalte et al. (2003), which uses Swedish survey data. The latter study estimates the cutoff to be 116 million (1999 SEK), which translates to US$13.3 million (1999 US dollars).

Table 1 in the appendix lists the estimates of the cutoff from the studies reviewed here. The vast majority of studies rely on the direct approach. Two studies employ the indirect approach, and two studies employ a modified indirect approach. In other words, these studies adapt the indirect approach to include elasticities from micro- or macroeconomic correlations between income and health or income and risky behavior.[4] Since the studies in table 1 were conducted across many years, estimates of the cutoff have been adjusted for inflation and presented in 2015 US dollars, using PPP currency conversions where necessary. Estimates fall in the range of $5.6 to $84.5 million (2015 dollars). If one excludes estimates from outside the United States, the range is $8.1 million to $84.5 million.

[4] One example of a study employing macroeconomic correlations is Lutter and Morrall (1994), which employs a version of the indirect approach but which also relies on cross-country correlations between GDP per capita and mortality. The study estimates the cutoff value at between $9 million and $12 million for the United States (1991 dollars).

The only study that strictly employs the indirect approach for the United States, without further modifications, is Viscusi (1994), with a cutoff estimate of $84.5 million (2015 dollars). The next highest estimate is $26 million for the United States, which is the high end in the range of estimates found in Chapman and Hariharan (1996). The Viscusi estimate is notably higher than any of the other estimates appearing in table 1. While this makes the estimate an outlier, we believe this higher cutoff value avoids the simultaneity concerns involved in estimating the income-mortality relationship with the direct approach. A higher cutoff value is also consistent with the reasonable suspicion that some, but not all, of the observed correlation between income and mortality is due to reverse causality and omitted variables. A higher cutoff is also appealing because intuitively it makes little sense for the VSL and the cutoff value to be roughly the same number.

6. Updating the Cost-per-Life-Saved Cutoff

Only two values are required to calibrate the model described in section 5: the VSL and the MPSH. For the VSL, we use a recent estimate from the US Department of Transportation (2016). The department's internal guidance recommended a VSL of $9.4 million for 2015. The revealed preference values are based on recent labor market estimates of the VSL using the Census of Fatal Occupational Injuries data. Similar values are used by other agencies, such as the US Environmental Protection Agency (2016), which recommended a VSL of $9.7 million (2013 dollars) and the US Department of Health and Human Services (2016), which recommended a figure of $9.6 million (2014 dollars).

The marginal propensity to spend on health, dh/dy, is equal to the fraction of income spent on healthcare, h/y, multiplied by the income elasticity of the demand for healthcare, which is denoted η in equation (2). This is the approach taken by Viscusi (1994, 105).

$$\frac{dh}{dy} = \frac{h}{y}\eta, \tag{2}$$

One way to estimate the fraction of income spent on healthcare is using national data. According to the Centers for Medicaid and Medicare Services, national health expenditures in the United States amounted to $3.2 trillion in 2015, or 17.8 percent of GDP.[5] These expenditures include both public and private spending on healthcare. If public spending is less effective in enhancing health than private expenditures, use of the total expenditure value will overstate the healthcare share of income that is pertinent to this calculation.

To establish a floor on the healthcare share of income, we examine expenditures on healthcare at the consumer level. According to the 2015 Consumer Expenditure Survey from the US Bureau of Labor Statistics, average pre-tax income for a consumer unit that year was $69,629, while spending on healthcare was $4,342 on average.[6] This represents 6.2 percent of consumer income.[7] This number includes $2,977 in spending on health insurance but excludes employer spending on premiums as part of employer-provided health insurance. According to the BLS, the average share of premiums paid by the employer was 81 percent for single coverage and 68 percent for family coverage in March of 2016 (BLS 2016a). A Kaiser Family Foundation survey finds a similar breakdown. For family coverage, the average annual health insurance premium for employer-based coverage in 2015 was $17,545, of which $4,955 (28 percent) was paid by the worker on average, and $12,591 (72 percent) was paid by the employer (Claxton et al. 2015).

[5] NHE Fact Sheet, available at https://www.cms.gov/research-statistics-data-and-systems/statistics-trends-and-reports/nationalhealthexpenddata/nhe-fact-sheet.html, accessed October 17, 2017.
[6] US Bureau of Labor Statistics, "Consumer Expenditures—2015," released August 30, 2016, accessed October 17, 2017, https://www.bls.gov/news.release/archives/cesan_08302016.htm.
[7] A consumer unit contains 2.5 people on average, and 0.6 of these people are children under the age of 18 on average, those for whom spending may be most effective.

Since a BLS consumer unit contains 2.5 people on average, we use the ratio for family coverage; assuming a split where 70 percent is paid by the employer and 30 percent is paid by the employee.[8] Assuming the $2,977 spent on health insurance is all spent on premiums, then $9,923 would be spent in total ($6,946 by the employer and $2,977 by the employee). If we add the $6,946 paid by the employer as a contribution to a consumer unit's total compensation, then total income rises to $76,575 in 2015. Spending on healthcare would then represent 14.7 percent of total income ((4,342+6,946)/76,575).

However, according to the US Bureau of Labor Statistics, only about 52 percent of civilian workers participated in employer-provided health insurance (BLS 2016b). This number likely underestimates participation generally since many individuals participate through a spouse. However, the 2015 Current Population Survey from the US Census Bureau estimates that 55.7 percent of individuals in the United States are covered by employment-based health plans (Barnett and Vornovitsky 2016), which is only slightly higher.[9] We assume 45 percent of the population spends the unadjusted fraction of income spent on healthcare for a consumer unit (6.2 percent), and the other 55 percent spends the higher fraction estimated for consumer units covered by employer-provided health insurance (14.7 percent). The weighted average of these values, 10.9 percent, is our lower bound value used for h/y in equation (2).

There is considerable debate surrounding the income elasticity η of health spending. At the high end is Fogel (2009), which uses an elasticity of 1.6. Most elasticity estimates come from aggregated data as well, which creates difficulties in making inferences about individual behavior. Studies using national data tend to find an elasticity over 1, for example, while those

[8] This is the average of the BLS breakdown and the breakdown in the Kaiser Family Foundation report.
[9] The Census Bureau estimates that 289,903,000 individuals were covered by any health plan in 2015 and that 28,966,000 were uninsured, for a total of 318,869,000 indidividuals in the survey. Of these, 177,540,000 were estimated to have employer-based plans.

using regional data typically estimate the elasticity as below 1 (Costa-Font, Gemmill, and Rubert 2011). This is further complicated by the fact that much of public health expenditures in the United States show up at the national level but not the individual level or regional level.

We believe the estimates in the lower range may be more accurate since these include studies that attempt to correct for problems related to endogeneity and publication bias. A recent meta-regression analysis found the income elasticity for health expenditures to be in the range of 0.4 to 0.8 (Costa-Font, Gemmill, and Rubert 2011). Research by Acemoglu, Finkelstein, and Notowidigdo (2012), which uses oil shocks as an instrument for exogenous income increases, estimates the income elasticity of health spending to be around 0.72. We choose to use 0.7, which lies in the range estimated in the recent meta-regression analysis and is also close to the value estimated by Acemoglu and his coauthors.

If healthcare expenditures range from 10.9–17.8 percent of income and the income elasticity of healthcare spending is 0.7, this implies a marginal propensity to spend on health of about 0.0763 to 0.1246.[10] With a VSL of $9.4 million, the cutoff estimate range is from $75.4 million to $123.2 million (2015 dollars) with a midpoint of $99.3 million, which clearly exceeds all of the estimates in table 1.

The $123.2 million figure is almost certainly an upper bound on the cutoff, since spending on risk reduction is not limited to just expenditures on healthcare. Income elasticities are likely to vary by income level as well, so the cutoff value will vary by income level. In a cross-country study, Di Matteo (2003) notes that income elasticities are higher at low income levels and lower at high income levels. The same may hold for low-income individuals. Use of higher elasticities would raise the MPSH, which in turn would lower the cost-per-life-saved

[10] Viscusi (1994) estimated an MPSH of about 0.1. According to our updated estimates, this remains a reasonable rule of thumb. The midpoint of our range for the MPSH is 0.10045.

cutoff. This is consistent with a Swedish study, which estimated that the lowest income quintile had an average MPSH of 0.20, while the highest quintile had an average MPSH of 0.14 (Hjalte et al. 2003).

Although income- or age-varying VSLs are not widely used by government analysts, the value of a statistical life year (VSLY) is a metric that is sometimes used, for example by the Food and Drug Administration. There is a corollary to the VSLY with the cutoff value. Much as one can divide the VSL by the expected remaining life expectancy, one can also divide the cutoff value by the number of years life is expected to be shortened. This value could then be used in health-health tests relying on cost-effectiveness values measured in life-years saved.

Tengs et al. (1995), for example, estimated life-years saved for over 500 regulations. By contrast, Morrall (1986, 2003) included "league tables" listing the cost-per-life-saved of a suite of regulations. Because these studies are rather old and the results of health-health analysis in this regulatory era has been examined by Hahn, Lutter, and Viscusi (2000), we instead choose to compare our cutoff estimate to cost-effectiveness estimates from more recent studies. One recent study estimates that state expansions of the Medicaid public insurance program in the first few years of the 21st century reduced mortality at a cost of $327,000 to $867,000 per life saved (2007 dollars) (Sommers 2017). This estimate range comes in well below the $99.3 million mortality risk threshold estimated in this paper and is also well below the $9.4 million efficiency breakeven threshold for life-saving regulations.

A study by Krutilla, Good, and Graham (2015) focuses on the cost-per-life-saved of a suite of Environmental Protection Agency (EPA) air pollution regulations. Although the sample is limited to environmental regulations, the study has some desirable features over previous studies. First, it is more recent. Next, the study presents 90 percent confidence intervals rather

than just point estimates of cost-effectiveness, and given the highly uncertain nature of the benefits of many life-saving regulations, confidence intervals are preferable.

Table 2 in the appendix presents cost-effectiveness estimates from Krutilla, Good, and Graham (2015) for nine environmental regulations. Mean cost-effectiveness estimates based on an American Cancer Society study, and used by the EPA in its regulatory impact analyses, range from $200,000 to $24 million (2013 dollars) per life saved. Even without adjusting for inflation, it is clear these numbers do not come close to the estimate of the lower bound value of the cost-per-life-saved cutoff of $75.4 million. The Krutilla, Good, and Graham study also presents mean cost-effectiveness estimates that are adjusted based on expert elicitations. A benefit of this approach is that a range of uncertainty is evaluated. Mean cost effectiveness estimates for the nine regulations range from $100,000 to $14 million per life saved (2013 dollars), lower than the American Cancer Society study estimates, and again well below the cutoff values estimated here. Taken at face value, the mean benefit assessments do not indicate any counterproductive regulations.

However, the degree of uncertainty surrounding these regulations is large. In each case, an infinite value of the cost-per-life-saved lies within a 90 percent confidence interval. An infinite cost-effectiveness estimate implies that zero lives are saved by an intervention. Therefore, statistically speaking, it cannot be ruled out that these regulations on net increase mortality risk, even with an upper bound estimate of the cost-per-life-saved cutoff of $123.2 million.

The enormous uncertainty surrounding these benefit estimates is likely related to a series of critical assumptions about the relationship between mortality risk and one specific pollutant, particulate matter (or PM2.5). An annual report published by the Office of Management and Budget notes that the PM-related health benefits calculated in EPA regulatory impact analyses rely on a series of "six key assumptions and uncertainties." These include that "inhalation of fine

particles is causally associated with premature death," that the "concentration-response function for fine particles and premature mortality is approximately linear," and that "all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality," as well as several other factors (US Office of Management and Budget 2016). While a complete review of these assumptions is beyond the scope of this study, they tend to all operate in the direction of increasing benefits estimates.

Moreover, recent regulatory analyses have been subject to a variety of critiques, which, if valid, would imply that agency-estimated benefits are generally overestimates. These critiques include the reliance on upper bound conservative risk assumptions (Hamilton and Viscusi 1999), incorporation of co-benefits in calculation of environmental policy benefits (Dudley 2012), use of a global social cost of carbon rather than the benefit pertinent to US citizens (Gayer and Viscusi 2016), and undocumented assumptions of market failures involving consumer energy-related decisions (Gayer and Viscusi 2013). Notwithstanding these possible critiques, it may also be the case that the cost-effectiveness of regulatory policies has improved over time due to the influence of regulatory oversight efforts.

A final area where the benefits of life-saving regulations are highly uncertain relates to national security and counter-terrorism efforts. In many cases, since the benefits of these policies are unknown, analysts rely on breakeven analysis rather than benefit-cost analysis.[11] Breakeven analysis identifies the number of terrorism-related events that would need to occur in absence of an intervention in order to justify a regulation of a specified cost. But breakeven analysis cannot guarantee that terrorist attacks are actually prevented. Presumably some anti-terrorism

[11] For some examples of breakeven analysis for anti-terrorism regulations, see Mueller and Stewart (2013, 2011), as well as regulatory impact analyses from the Department of Homeland Security.

regulations would prevent fewer acts of terrorism than this breakeven level, or even zero acts of terrorism, and by extension increase mortality risk on balance in some cases.

An analyst at the Cato Institute produced a back-of-the-envelope estimate of the cost-effectiveness of a January 2017 executive order that restricted refugee admissions into the United States (Nowrasteh 2017). The policy was estimated to have a cost-effectiveness of $525.5 million per life saved, on the basis of historical data about the number of acts of refugee-caused terrorism on US soil, as well as an estimate of forgone output from refugee contributions to the US economy. If accurate, this figure implies the order would increase mortality risk on the basis of the cutoff values estimated in this paper.

7. Conclusion

Health-health analysis is a relatively simple analytic tool that offers useful information to decision makers. It can be used as a form of screening analysis to weed out the most cost-ineffective regulations. It may also be desirable because there is no need to apply dollar values to health-related risks, a practice that remains controversial to some.

HHA can also be used in cases where BCA is expressly prohibited by law. Such is the case for certain pollution exposure standards set by the US Environmental Protection Agency and by the Occupational Safety and Health Administration. Although some have argued that HHA is actually a form of BCA (e.g., GAO (1992) made this claim), this criticism is mistaken. The units in a BCA are dollars, while the units in an HHA are estimates of risk. Furthermore, while it is true that dollar estimates of compliance costs are an input used to estimate mortality risks, decision makers can easily consider the headline number (net risk reduction) without considering any underlying costs.

One might even argue that in cases where consideration of costs must be superseded by considerations of health, this provides a legal basis for a *mandate* that an agency *must* conduct a health-health analysis. Harvard Law School professor Cass Sunstein has argued, for example, that agency decisions that increase aggregate risks should be considered "arbitrary and capricious" under the Administrative Procedure Act (Sunstein 1996).

Another common misconception about HHA is that it necessarily relies on correlations between income and mortality. As has been demonstrated here, however, it is possible to estimate the cutoff value, a critical input in HHA, indirectly without relying on such correlations. Furthermore, an overlooked benefit of HHA is that the theoretical model used to estimate the cutoff value can also be used to improve estimates of the value of a statistical life. The VSL is endogenous to regulatory interventions since what individuals are willing to pay to reduce mortality risk will change in response to policy. The cost-per-life-saved cutoff can also be used to set an upper bound on estimates of VSL, since individuals should not be willing to pay for incremental risk reductions that increase countervailing risks by an even greater extent.

Finally, HHA can be used to improve estimates in risk-risk and benefit-cost analyses. For example, suppose the lower-bound cost-per-life-saved value of $75.4 million is used. If the regulation imposes a cost of $754 million, then there will be an increase in 10 expected deaths from the policy, and these risk effects could be included in the assessment of the net risk effects and the net benefits of the regulation. HHA could eventually be a standard component of regulatory analyses.

According to our estimates, the cost-per-life-saved cutoff is in the range of $75.4 million to $123.2 million (2015 dollars). Any regulation with a cost-per-life-saved that exceeds this

range can be expected to increase mortality risk on net. There is a great deal of uncertainty surrounding a number of factors that produce this estimate, however, including the fraction of income spent on risk reduction, the income elasticity of risk-reducing expenditures, and the VSL. Despite this uncertainty, it can be said with significant confidence that any regulation costing more per life saved than $123.2 million will have counterproductive mortality effects.

We have reviewed three sets of policies to assess their mortality risk consequences. First, several state expansions of the Medicaid public insurance program in the first few years of the 21st century appear to have reduced mortality risk. Second, a suite of environmental regulations from the EPA have point estimates of cost-effectiveness far below the cutoff value. But there is so much uncertainty surrounding these figures that, statistically, one cannot rule out the possibility that these regulations increase mortality risk. Third, an assessment of recent refugee restrictions suggests they may have counterproductive mortality risk effects.

In conclusion, HHA estimates serve a number of different potential policy roles. First, they can be used in cases where BCA is prohibited by agency statutes. Second, the cost-per-life-saved cutoff value can be used in calculating the net mortality reduction, as opposed to the gross reduction usually appearing in regulatory analyses. Finally, for particularly ineffective regulations, HHA can identify situations in which a proposed regulation has net adverse effects on mortality, allowing policymakers to screen out ineffective policies. Some possible examples of counterproductive policies have been identified here, and future research should help to identify more such examples.

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Appendix: Tables

Table 1. Studies Estimating the Cost-per-Life-Saved Cutoff

Study	Cost-per-life-saved cutoff (millions of US dollars)	Dollar year	Direct or indirect approach	MPSH	Inflation-adjusted cutoff (adjusted to millions of 2015 US dollars)	Comments
Keeney (1990)	3.14–7.25	1980	Direct	N/A	8.1–18.8	First model to formally estimate the cutoff; mortality risk is a function of income with no additional controls used; cutoff varies depending on distribution of regulatory costs; study is open to critiques of ecological bias and confounding bias.
Viscusi (1994)	50	1990	Indirect	0.10	84.5	First study to employ the indirect approach; builds a structural model of the income/mortality risk relationship; incorporates the VSL and MPSH; avoids problems of endogeneity and reverse causation.
Lutter and Morrall (1994)	9–12	1991	Indirect	0.275	14.7–19.7	Coined the term "health-health analysis"; works with the Viscusi (1994) model but incorporates income elasticities of various health measures from cross-country studies; these adjustments introduce the possibility of confounding and ecological bias.
Chapman and Hariharan (1994)	12.2	1990	Direct	N/A	20.6	Controls for initial health status to account for possibility of reverse causation; Social Security data are used for males around retirement-age.
Chapman and Hariharan (1996)	6.7–15.4	1990	Direct	N/A	11.3–26.0	Controls for initial health, marital status, age, a quandratic income variable, assets (as a measure of patience), and time-varying (fixed) effects.
Keeney (1997)	5–14	1991	Direct	N/A	8.2–22.9	Uses individual-level data rather than census-tract data to correct for ecological bias but does not control for other confounding variables; study finds little difference in fatalities from concentrated versus dispersed costs; estimates of fatalities vary significantly across income and racial groups.

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Study	Cost-per-life-saved cutoff (millions of US dollars)	Dollar year	Direct or indirect approach	MPSH	Inflation-adjusted cutoff (adjusted to millions of 2015 US dollars)	Comments
Lutter et al. (1999)	15	1990	Indirect	0.10 (same as Viscusi [1994])	25.4	Begins from Viscusi (1994) model; incorporates income elasticities of various risky behaviors into the model, which could lead to confounding and reverse causation biases and an underestimation of the cutoff.
Elvik (1999)	3.8–47.5	1995	Direct	N/A	5.6–70.6	Uses Norwegian data; cutoff varies depending on controls used in regression analysis; controls include healthcare spending, age, and sex.
Gerdtham and Johannesson (2002)	6.8–9.8	1996	Direct	N/A	9.9–14.2	Uses Swedish data; controls for initial health status and various personal characteristics.
Hjalte et al. (2003)	13.3	1999	Indirect	0.179	18.3	Calibrates Viscusi (1994) model with Swedish data; estimates new MPSH based on surveys; finds MPSH varies by income level (MPSH is 0.20 for individuals in lowest quintile of household income and 0.14 for inidividuals in top quartile for household income).
Ashe et al. (2012)	20–50	2010 AU$	Direct	N/A	14.5–36.2*	Builds on Keeney (1997) model; assumes American correlations between income and mortality hold for Austrialia, which may be incorrect.

Sources: Authors' calculations, based on work by Ashe and coauthors (2012); Chapman and Hariharan (1994); Elvik (1999); Gerdtham and Johannesson (2002); Hjalte and coauthors (2003); Keeney (1990, 1997); Lutter and Morrall (1994); Lutter and coauthors (1999); and Viscusi (1994).

Data note: Figures converted to 2015 dollars using the Trimmed Mean PCE Inflation Rate available from the Federal Reserve Bank of Dallas.

* Converted to 2010 US dollars using the World Bank PPP conversion factor for Australia in 2010 and then adjusted for inflation from 2010 US dollars to 2015 US dollars. World Bank, "PPP Conversion Factor, GDP," International Comparison Program database, http://data.worldbank.org/indicator/PA.NUS.PPP?page=1.

Table 2. Gross Cost per Discounted Life Saved of EPA Air Pollution Regulations

Rule	American Cancer Society Study mean estimate, 3 percent discount rate (US$2013 in millions)	Simulated distribution from integrated expert elicitations, at 3 percent discount rate (US$2013 in millions)		
		0.05	Mean	0.95
Mercury and Air Toxic Standards (MATS)	$2.80	$0.70	$1.60	∞
Cross State Air Pollution Rule (CSAPR)	$0.20	$0.10	$0.10	∞
National Emissions Standards for Hazardous Air Pollutants (NESHAP) for Major Boilers ("Boiler MACT")	$0.60	$0.20	$0.40	∞
National Emissions Standards for Hazardous Air Pollutants (NESHAP) for area source boilers	$24.00	$6.00	$14.00	∞
NESHAP for commercial and industrial solid waste incinerators	$6.60	$1.70	$3.90	∞
NESHAP for the Portland cement manufacturing industry and performance standards for Portland cement plants	$1.30	$0.30	$0.80	∞
NESHAP for Compression Ignited Reciprocating Internal Combustion Engines (CIRICE)	$5.10	$1.30	$3.00	∞
NESHAP for Spark-Ignited Reciprocating Internal Combustion Engines (SIRICE)	$19.00	$4.80	$11.00	∞
New Source Performance Standards (NSPS) for petroleum refineries	$4.60	$1.20	$2.70	∞

Source: Krutilla, Good, and Graham (2015).

Data note: Estimates rounded to two significant figures; ∞ corresponds to no lives saved.

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