Economic Evaluation with Hormetic, Hockey-Stick, and Linear Response Functions: An Application to Radon in Drinking Water

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Abstract

Economic evaluation of policies to reduce exposure to environmental-health threats usually assumes that the exposure-response function is linear and exhibits no threshold exposure below which the incremental risk is zero. However, for many agents there is evidence that the exposure-response function has a threshold below which exposure produces no incremental risk, or is hormetic so that small exposures are beneficial and large exposures are harmful. We explore some of the practical issues involved in conducting economic evaluation using nonlinear exposure-response functions in the context of a case study: proposed regulation of radon in drinking water. This case study was chosen because of the availability of both an economic evaluation using a linear no-threshold model and an estimated nonlinear (hormetic) exposure-response function. We illustrate that economic evaluation can be conducted using nonlinear exposure-response functions, though the analysis requires more information than in the case of a linear no-threshold function because of the need to account for differences within the population in the benefits of exposure reduction that depend on initial exposure. The indicated stringency of regulation depends on the shape of the exposure-response function and on uncertainty about its shape, but it is not true that the linear function always suggests more (or less) stringent regulation than alternative non-linear functions.

Keywords: benefit-cost analysis, hormesis, linear no-threshold, radon

1. Introduction

Economic analysis is often used to evaluate government regulation of environmental and other health risks. In the U.S. federal government, such analysis is required for major rules under Executive Order 12866 and previous executive orders. In most cases, analysis assumes the relationship between exposure to an agent and health effect can be well described using a linear no-threshold model. Under this model, exposure to any level of the agent causes risk of harm and reductions in exposure reduce risk. Alternative, non-linear models include the hockey-stick or threshold model, in which exposure to an agent has no health effect unless exposure exceeds some non-zero threshold, and the hormetic model, in which low levels of exposure are beneficial and high levels are harmful, compared with zero exposure.

The threshold model has been widely recognized in risk assessment and risk management but rarely applied in economic evaluation. Threshold models underlie the use of "safe" levels such as reference dose. Economic evaluation with a reference dose is difficult because there is rarely any quantitative description of how the risk of adverse effects varies with exposure levels above the reference dose. Implicitly, there is no quantifiable benefit to reducing exposure from some level above the reference dose to another level that remains above the reference dose or to reducing exposure from some level below the reference dose to another level further below it. Usually, it is not even possible to quantify the benefit of reducing exposure from some level above the reference dose to a level below it, since the probability of harm when exposed above the reference dose is not specified.

In recent years, a substantial body of evidence has been developed which suggests that the relationship between exposure and effect for many agents may be characterized by a third, hormetic, model (e.g., Calabrese and Baldwin 2001a, 2001b, 2002). Under the hormetic model, the exposure-response function is J-shaped: exposures to small quantities of an agent are beneficial while exposures to larger quantities are harmful.

The theoretical implications for economically optimal regulation of an environmental health risk if one adopts a hormetic rather than a linear no-threshold model are examined by Hammitt (2004). In the current paper, the practical implications of

substituting a hormetic or hockey-stick model for the conventional linear no-threshold model in economic analysis are examined in the context of a case study. Specifically, we explore the implications of substituting threshold and hormetic exposure-response functions for the linear no-threshold model that was adopted in an existing economic evaluation of a hazardous agent. We identify issues that must be resolved in order to conduct the analysis using the alternative, non-linear exposure-response functions and examine how the optimal degree of regulatory stringency depends on the selected function. In addition, we consider the implications of uncertainty about the appropriate exposure-response function for optimal regulatory stringency.

As a case study, we require an environmental health risk for which an economic evaluation of a possible regulation is available and for which a possible hormetic exposure-response relationship has been estimated. Ionizing radiation was chosen as the agent, and lung cancer associated with radon in drinking water as the environmental health risk. Bogen and Layton (1998) also evaluate the appropriate management of radon exposure using a hormetic exposure-response function. We take no position on the strength of evidence supporting the linear no-threshold and alternative exposure-response functions for radon.

In the following section, we describe the regulatory alternatives evaluated by EPA and their estimated costs. Section 3 describes the linear no-threshold exposure-response function used in the RIA and the alternative hormetic and hockey-stick exposure-response functions we consider. Section 4 describes the methods for estimating risk and Section 5 presents estimates of the benefits and costs of alternative regulations using the three exposure-response functions and a case in which there is uncertainty about which exposure-response function is most accurate. Conclusions are in Section 6.

2. Regulatory Options and Estimation of Costs

Radon in indoor air is considered to be one of the major environmental health risks in the United States today. However, radon in residential indoor air is not subject to government regulation because neither EPA nor other agencies have the requisite legal authority. Radon is also present in domestic drinking water and EPA has authority to regulate this hazard under the Safe Drinking Water Act. EPA proposed regulations in

1999. As part of this action, EPA produced a regulatory impact analysis (RIA; EPA 1999) which estimates the economic value of the benefits and costs of regulations of varying stringency. The RIA estimates the benefits of reducing radon in drinking water using a linear no-threshold (LNT) exposure-response function.

EPA considered the possibility of imposing a maximum contaminant level (MCL) for radon in drinking water equal to 4,000, 2,000, 1,000, 700, 500, 300, or 100 pCi/l (picocuries per liter). Community water systems (CWS) having radon concentrations exceeding the MCL would be required to treat the water using aeration and other methods in order to achieve the MCL. Technologies for reducing radon concentration include aeration, granular activated carbon treatment, storage, and changing the mix of input to exclude higher-radon sources. EPA assumes each CWS would choose from a set of three, increasingly costly, alternative treatment systems with removal efficiencies of 50, 80, and 99 percent, respectively (EPA 1999, Table 3-3). Because a CWS chooses from this set of discrete alternatives, it cannot adopt the removal efficiency that would yield exact compliance with the MCL; rather, it will choose the least costly alternative that yields an after-treatment radon concentration that is less than or equal to the MCL. As a result, the concentration of radon in the treated water will typically be smaller than the MCL. A CWS with radon concentrations more than 100 times greater than the MCL would presumably install the treatment with 99 percent control efficiency but its after-treatment radon concentration would exceed the MCL. The nationwide cost of compliance depends primarily on the number of CWSs that would need to treat their water. Estimated costs are discussed below.

3. Linear No-Threshold and Alternative Exposure-Response Functions

The presence of radon in domestic drinking water presents risks of several types of cancer through multiple exposure pathways. The dominant risk is believed to be the risk of lung cancer due to inhalation of radon and its radioactive progeny that are volatilized from drinking water. Lung-cancer mortality (LCM) associated with the inhalation pathway is estimated to account for almost 90 percent of the total cancer risk associated with radon in drinking water (EPA, 1999; Table ES-1). Other risks include risk of stomach and other cancers associated with ingestion when drinking and dermal

absorption when bathing. Non-fatal lung and other cancers also result, but these contribute little to the economic value of limiting radon concentrations because there are many fewer cases of non-fatal than of fatal cancer and the economic value of reducing the risk of non-fatal cases is much smaller than that of reducing the fatal risk.

Benefits of reducing radon concentrations in drinking water are estimated in the RIA using a linear no-threshold model. First, the relationship between the concentration of radon in indoor air and radon in domestic water is modeled using a transfer coefficient of 1/10,000; i.e., a 1 pCi/l reduction of radon concentration in domestic water reduces the radon concentration in indoor air by 1E-4 pCi/l (EPA, 1999, p. 45). The lifetime risk of lung-cancer mortality is estimated using a linear no-threshold model with slope equal to 0.0067 (probability of cancer per pCi/l radon in indoor air; EPA 1999, p. 46), derived from National Academy of Sciences (1999).

We develop alternative hormetic and hockey-stick exposure-response functions using results of Bogen (1997, 2001). A hormetic relationship between radon exposure and lung-cancer mortality could result as the net of two competing effects: cell killing and mutation. Even if both effects are linear functions of exposure, the effect of killing premalignant cells could outweigh the effect of increasing mutation rates, leading to lower lung-cancer mortality risk, at low exposure levels. Bogen (1997) estimated a hormetic exposure-response function for radon using health data on white males in more than 1600 U.S. counties and white male Colorado Plateau uranium miners and compared his estimate with the LNT model from the BEIR IV report (National Research Council, 1988). In subsequent work, Bogen (2001) estimated an alternative hormetic exposure-response function using data on white females in more than 2800 U.S. counties and five cohorts of under-ground miners and compared this estimate with LNT models from the BEIR VI report (National Research Council, 1998).

We use the hormetic exposure response function from Bogen (1997), since this function yields the same estimated risk at high exposure levels (~ 20 pCi/l) as the comparable LNT model. In contrast, the Bogen (2001) function shows substantially smaller risk at all exposure levels than the corresponding LNT model. As is well known, uncertainty about the shape of an exposure-response function is most pertinent to risk analysis at low exposure levels at which the response cannot be measured (e.g., because

the risk is too small to distinguish from background). All plausible exposure-response functions must correspond at higher exposure levels where the response is measurable.

Bogen (1997) reports his estimated hormetic exposure-response function in the form of a graph plotting increased relative risk against residential indoor-air radon concentration for the hormetic function and the LNT function from BEIR IV. To maintain consistency with the RIA, we change the vertical axis to increased absolute risk (lifetime probability of fatal lung cancer) and rescale so that the LNT function has the same slope as the LNT function used in the RIA (0.0067 per pCi/l). For computational simplicity, we approximate the hormetic function as piecewise linear with nodes at each 1.25 pCi/l increment.

The resulting hormetic function and the LNT model used by EPA are illustrated in Fig. 1. As shown, the excess LCM risk at 20 pCi/l is the same for the LNT and hormetic exposure-response functions and equal to 0.134. The slope of the LNT function is constant and equal to 0.0067 per pCi/l. In contrast, the slope of the hormetic function varies with exposure. For exposure levels between 0 and 5 pCi/l the slope is negative. Over this range, increased exposure reduces LCM to a maximum risk reduction of about 0.032. As exposure increases, the incremental cancer risk increases at an increasing rate. The slope increases from zero at an exposure of 5 pCi/l to about 0.016 per pCi/l, more than twice the slope of the LNT function, at an exposure level of 20 pCi/l.

Debate about the shape of the exposure-response function for ionizing radiation has focused on the possibility of a hormetic relation rather than the standard LNT model; to our knowledge, the possibility of a hockey-stick or other threshold model has received less attention. For illustration, however, we consider the possibility that the relationship is characterized by a hockey-stick model. We base our hockey-stick exposure-response function on the hormetic model. Specifically, we set the threshold equal to 10 pCi/l, the exposure level at which excess risk under the hormetic model is zero. For exposures above the threshold, we assume a linear function such that the risk at high exposure (20 pCi/l) is the same as under the LNT and hormetic models. This implies the slope of the hockey-stick model for exposures greater than 10 pCi/l is twice the slope of the LNT model, 0.0134 per pCi/l. For exposure levels less than the threshold, the slope of the hockey-stick model is zero. The hockey-stick model is also shown in Fig. 1.

4. Risk Assessment

To evaluate the effect of substituting hormetic and hockey-stick exposure-response functions for the LNT model used in EPA (1999), we begin by developing a method for calculating the benefits under the LNT model and verifying that it reproduces the EPA estimates. We then estimate benefits under the alternative, nonlinear exposure-response functions using a modification of that approach.

Exposure Reduction

The RIA evaluates alternative regulations that would limit the Maximum Contaminant Level (MCL) for radon in drinking water to 4000, 2000, 1000, 700, 500, 300, and 100 pCi/l, respectively. Each community water system (CWS) with radon concentrations higher than the MCL would need to install remediation technology to reduce the radon level to the MCL. As described above, EPA assumes each CWS would choose from a set of treatment technologies with discrete removal efficiencies and so the typical CWS will over-comply, yielding treated water with a radon concentration less than the MCL. The RIA notes that the health benefit from the resulting over-compliance is small compared with the benefit of reducing radon concentrations from current levels to the MCL. In our analysis, we neglect this complication and simply assume that each CWS controls radon so that the concentration of radon in its treated water equals the MCL.

The change in population exposure for each alternative MCL can be calculated by taking account of the distribution of population by radon exposure. The number of people served by CWSs with drinking-water radon concentrations greater than each potential MCL is reported in the RIA (Table 3-3) and reproduced in Table I (column 2). From these figures, the number of people in each exposure bin (defined by adjacent potential MCLs) can be calculated. The average radon concentration within each bin is assumed to be equal to the midrange of the adjacent potential MCLs (for simplicity). For the bin including concentrations greater than 4000 pCi/l, an average concentration of 8000 pCi/l is chosen by calibration, as described below.

The average reduction in radon exposure for each MCL is the difference between the population-weighted average exposure for individuals with exposure exceeding the

MCL and the MCL. For example, if an MCL of 1000 pCi/l is imposed, mean radon exposure would fall: from 8000 to 1000 for 77.2 thousand people with exposure greater than 4000 pCi/l, from 3000 to 1000 for 303.8 thousand people with exposure between 2000 and 4000 pCi/l, and from 1500 to 1000 for 1314 thousand people with exposure between 1000 and 2000 pCi/l. In total, drinking-water exposure to 1695 thousand people would fall by an average of 1065 pCi/l.

Background Exposure

Recall that the dominant pathway by which drinking-water radon increases cancer risk is through inhalation of radon and its radioactive progeny that are volatilized. Under the LNT model, the effect of a change in indoor-air radon associated with radon in drinking water is independent of the background indoor-air radon concentration (i.e., the concentration in air that would exist with no contribution from drinking water). In contrast, under the hormetic and hockey-stick models, the effect of an incremental contribution to indoor-air radon depends on the background concentration.

The distribution of annual mean living-area residential indoor-air radon in the United States is approximately lognormal with geometric mean 0.67 pCi/l and geometric standard deviation 3.1 (Marcinowski et al., 1994; Lin et al., 1999). We assume this distribution is an adequate approximation for our analysis. The distribution implies that the great majority of households have background exposure levels such that reducing exposure is harmful under the hormetic model and has no effect under the hockey-stick model. Under the hormetic model, the slope of the exposure-response function is negative for indoor-air concentrations less than 5 pCi/l. This concentration is above the 96th percentile of the reported lognormal distribution, which implies that fewer than four percent of households would benefit from reducing radon concentration. Under the hockey-stick model, radon concentrations less than 10 pCi/l pose no risk. This concentration is above the 99th percentile of the distribution, so under this model fewer than one percent of households would benefit from reducing radon concentration.

In general, when calculating the effect of a reduction in exposure under a nonlinear exposure-response function, it may be necessary to account for the change in the slope of the function as exposure is reduced. In this analysis, however, the change in

indoor-air radon concentration associated with regulation of drinking-water radon is small compared with the curvature of the nonlinear exposure-response functions. Using the transfer coefficient of 1/10,000 relating indoor-air to drinking-water radon implies that the contribution of drinking-water radon is only 0.8 pCi/l for the subset of the population with the highest drinking-water radon (8000 pCi/l), who account for less than 0.1 percent of the population exposed to drinking-water radon. For most of the population, the effect of a change in drinking-water radon on indoor-air concentrations is much smaller. Hence when calculating the change in cancer risk associated with reducing drinking-water radon, beginning at any background concentration, the change in slope of the exposure-response function may be neglected.

LNT Model

Under the LNT model, the reduction in cancer risk is proportional to the number of people affected and the mean reduction in radon exposure. The reduction in annual lung-cancer mortality M associated with a particular MCL can be calculated as the product

$$M = PCqT / L \qquad (1)$$

where P is the population currently exposed to radon levels higher than the MCL, C is the mean reduction in drinking-water radon concentration to which these people are exposed (i.e., the mean drinking-water radon level conditional on exceeding the MCL reported in Table I minus the MCL), q is the slope of the exposure-response function (0.0067, the incremental lifetime probability of LCM per pCi/l radon in indoor air), T is the transfer coefficient describing the change in indoor-air concentration per unit reduction in domestic-water concentration (1/10,000), and L is the assumed lifetime used to convert lifetime risk to annual risk (70 years).

Table II reports the population distribution by radon level together with the annual reduction in LCM calculated using equation (1) and the annual reduction in LCM reported in the RIA. The conditional mean radon concentration for the population with exposure greater than 4000 pCi/l is chosen so that the calculated reduction in cancer cases

(column 5) closely approximates the reduction reported in the RIA (column 4) for the higher potential MCLs (4000 and 2000 pCi/l). The calculated values for all potential MCLs agree well with the values reported in the RIA, which implies that the simplified approach adopted here provides an adequate approximation of the more complex modeling undertaken for the RIA.

Hormetic Model

As noted above, under the nonlinear exposure-response models the change in LCM associated with a change in drinking-water radon depends on the background indoor-air radon concentration. For households with low indoor-air radon concentrations the reduction in exposure associated with reducing drinking-water radon will increase risk, and for households with high indoor-air radon it will decrease risk.

The change in population risk from reducing radon concentrations in drinking water can be calculated by dividing the affected population into groups having nearly the same background exposure, calculating the risk change for each group, and summing, i.e.,

$$M = P\left[\sum_i p_i C_i q_i\right] T / L \qquad (2)$$

where M, P, T, and L are defined as in equation (1), and p_i, C_i, and q_i denote the fraction of the population, change in drinking-water radon concentration, and slope of the hormetic exposure-response function for group i. (As noted above, the change in indoor-air radon is small compared with the curvature of the hormetic exposure-response function and so the change in slope as exposure is reduced for each group can be neglected without introducing significant error.)

The term in brackets is a weighted average of the slopes of the exposure-response function at the indoor-air radon concentrations faced by the groups (q_i) where the weights $p_i C_i$ depend on the share of the population and the reduction in drinking-water radon concentration in each group. These weights depend on the relationship between the concentrations of radon in drinking water and in indoor air. Lacking information on this

dependence, we assume the two concentrations are independent and calculate the change in risk as

$$M = PC\left[\sum_i p_i q_i\right]T / L \qquad (3)$$

where C is the mean reduction in drinking-water radon concentration as in equation (1).

The population-weighted-average slope of the hormetic exposure-response function, defined by the term in brackets in equation (3), is -0.0068. As noted above, approximately 96 percent of households have indoor-air radon concentrations less than 5 pCi/l and for these households the exposure-response function is negatively sloped. The calculated changes in fatal-cancer risk for each MCL under the hormetic model are reported in Table II (column 6). These values are almost exactly equal to the inverse of the changes in population risk calculated using the LNT model, because the population-weighted slope (-0.0068) is (coincidentally) almost the inverse of the slope of the LNT function (0.0067). As shown in Fig. 1, the average slope of the hormetic exposure-response function for indoor-air concentrations less than 5 pCi/l, where almost all the population is distributed, is nearly the inverse of the slope of the LNT function.

Hockey-stick Model

The change in population risk associated with each MCL under the hockey-stick model can also be calculated using equation (2) or (3) where q_i is redefined to be the slope of the hockey-stick exposure-response function for population group i. Assuming the change in drinking-water radon is independent of the indoor-air radon concentration and using equation (3), the population-weighted-average slope of the hockey-stick exposure function is 0.00011. The calculated changes in fatal-cancer risk under the hockey-stick model are reported in Table II (column 7). These are positive, because decreased exposure is never harmful under the hockey-stick model, but are much smaller than under either the LNT exposure-response function. This follows because more than 99 percent of the population has background exposure less than 10 pCi/l, and under the hockey-stick model reductions in radon exposure provide no benefit to these people.

Under this model, most of the population receives no benefit but the small number of people with background exposure above the threshold experience risk reductions twice as large as under the LNT model (recall the slope of the hockey-stick exposure-response function is twice that of the LNT function for exposures above the threshold of 10 pCi/l).

5. Benefit-Cost Analysis

The primary benefit of reducing exposure to radon is the decrease in the probability of fatal cancer. In the RIA, EPA valued this risk reduction using a value per statistical life (VSL) of $5.8 million. The costs of satisfying alternative MCLs were based on estimates of the capital and operating costs of aeration and other treatment technologies combined with estimates of the number of CWSs that would need to install such controls.

Estimates of the nationwide benefits and costs of alternative MCLs are presented in Table III. Costs of achieving each MCL are reported in column 2; these are reproduced from the RIA (Table ES-7). Benefits under each of the exposure-response functions (columns 3 – 5) are calculated by multiplying the corresponding reduction in annual lung-cancer mortality (reported in Table II) by the VSL ($5.8 million per case). Net benefits, the difference between benefits and costs of achieving each MCL, are calculated for each model and reported in columns 6 – 8.

As shown in Table III, the costs of achieving each potential MCL exceed the benefits calculated using any of the exposure-response functions and so the net benefits of each MCL are less than zero. This implies that regulating radon in drinking water is not a socially efficient use of resources as the value of the cancer-risk reduction is less than the compliance cost. Under the LNT model, the benefits and costs are of comparable magnitude and the benefits are more than 80 percent of the costs for MCLs of 1000 pCi/l and smaller. In contrast, the benefits under the hockey-stick model are never more than one percent of the costs and the benefits under the hormetic model are negative.

EPA also concluded that the costs exceed the benefits for each of the potential MCLs. In its proposed rule, EPA selected an MCL of 300 pCi/l. However, the proposed rule also allows CWSs to meet an alternative MCL of 4000 pCi/l if they are located in a state that adopts a state-wide multimedia mitigation program (MMM). EPA anticipates

that reducing indoor-air concentrations of radon through a state-wide MMM program is more cost-effective than reducing radon in drinking water. Hence it anticipates that almost all CWSs will be required to comply with only the alternative MCL of 4000 pCi/l and that the cost of the regulation will be much smaller than the cost of achieving an MCL of 300 pCi/l reported in Table III.

Alternative Scenarios

We supplement our main analysis, which finds that nationwide regulation of radon in drinking water is not justified on benefit-cost criteria under the LNT, hormetic, or hockey-stick models, to identify situations in which some degree of regulation may be economically justified. This exploration is motivated by an interest in exploring the ramifications of using alternative nonlinear exposure-response functions in economic evaluation, rather than an interest in regulation of radon in drinking water. In particular, we are interested in identifying contexts in which the efficient MCL depends on the exposure-response function.

We begin by noting that, under the LNT model, the benefits and costs are of comparable magnitude, and so a modest reduction in costs or increase in benefits could easily tip the balance toward positive net benefits for some potential MCLs. Compliance costs per individual served are highly sensitive to the size of the CWS. For example, the average compliance cost per household is on the order of $5 to $10 for large CWSs that serve 100,000 households or more, about $15 to $30 for small CWSs that serve 3,300 to 10,000 households, and on the order of $300 or more for the smallest CWSs that serve 25 to 100 households (EPA 1999, Table 10-9). Exempting small systems from the regulation could substantially reduce the average cost per household served. Moreover, the appropriate monetary valuation for mortality risk (the VSL) is subject to substantial uncertainty. The U.S. Office of Management and Budget has suggested that the appropriate VSL may fall between $1 million and $10 million (OMB, 2003). The appropriate value may also depend on the type of health risk, with larger values for fatal cancers than for fatal traumatic injuries (Sunstein 1997, 2004, Revesz 1999). Empirical evidence on this question is mixed: Van Houtven et al. (2008) estimate that the monetary value for valuing fatal cancer risks is 1.5 to 3 times larger than for fatal-injury risks

(depending on the latency of the cancer) but Magat et al. (1996) and Hammitt and Haninger (2010) find no difference in valuation.

Recognizing the significant uncertainties in the costs of compliance and the monetary value per avoided cancer fatality, we conduct a revised analysis in which we reduce the estimated compliance costs by 20 percent and continue to use the $5.8 million VSL. This analysis might correspond to a rule that exempted some of the small CWSs for which per capita control costs are particularly high. As shown in Table IV, with this revision in costs the net benefits under the LNT model are positive for MCLs of 1000 pCi/l and smaller and are maximized for the most stringent potential MCL, 100 pCi/l. In contrast, the net benefits under the hormetic and hockey-stick models remain negative for all MCLs. In this setting, benefit-cost analysis recommends the most stringent MCL when using the LNT model and recommends no regulation of radon in drinking water under the alternative models.

Under the nonlinear exposure-response functions, the effect of reducing radon concentrations in drinking water depends on the background concentration in indoor air. As noted above, most U.S. households have indoor-air radon concentrations small enough that reducing exposure through controlling drinking water has no effect (under the hockey-stick model) or is harmful (under the hormetic model). Hence, if regulation of radon in drinking water is to provide benefits greater than its costs, the exposure reductions must be targeted on homes with relatively high indoor-air radon levels.

We consider the possibility of regulating drinking-water radon only for homes that have indoor-air radon levels of 4 pCi/l and above. This level is chosen because it is the action level at which EPA recommends that homeowners remediate indoor-air radon. Although indoor-air radon varies significantly from home to home, depending on soil characteristics, presence of a basement, construction method, and other factors, we assume for simplicity that it would be possible to regulate CWSs that serve only homes with indoor-air radon greater than 4 pCi/l and to exempt CWSs that serve only homes with lower radon levels.

Results are shown in Table V. The fraction of households having indoor-air radon above 4 pCi/l is 5.7 percent (based on the assumed lognormal distribution of indoor-air radon levels). We assume that indoor-air radon is distributed independently of size of

CWS and hence the cost of achieving any MCL for this subset of households is equal to 5.7 percent of the cost of achieving that MCL for the full population (shown in Table IV; we continue to assume that control costs are 20 percent smaller than estimated in the RIA). Similarly, the benefits of each MCL under the LNT model are only 5.7 percent as large as when the full population is affected (Table IV).

In contrast, the benefits under the hockey-stick model are not affected by restricting coverage to households with radon levels exceeding 4 pCi/l (i.e., benefits under the hockey-stick model are the same in Table V as in Table IV). This follows because, under the hockey-stick model, only people living in households with indoor-air radon above the threshold value of 10 pCi/l incur additional risk from radon in drinking water. Benefits under the hormetic model are positive for each MCL. This occurs because the benefits to households with indoor-air radon concentrations greater than the nadir of the hormetic exposure-response function (5 pCi/l) exceed the harms accruing to households with indoor-air radon levels between 4 and 5 pCi/l, for whom reduced radon exposure is harmful under the hormetic exposure-response function. Of the population with indoor-air radon greater than 4 pCi/l, about one-third have radon levels less than 5 pCi/l (and are harmed by radon reductions) and two-thirds have greater radon exposure (and benefit from radon reduction).

The optimal MCL under each of the exposure-response functions remains the same when regulation is restricted to households with indoor-air radon levels above the EPA action level as when the regulation is applied to the full population. In both Table IV and Table V, the most stringent MCL (100 pCi/l) is optimal under the LNT model and no regulation is optimal under the nonlinear models. Even though targeting the regulation on households with higher indoor-air radon sharply reduces the costs of compliance without reducing the benefits under the nonlinear models, costs continue to exceed benefits at all candidate MCLs.

The per-capita benefits of regulation under the nonlinear exposure-response functions can be increased by targeting of the regulation on households with even higher indoor-air radon levels. If regulations are restricted to households with indoor-air radon levels greater than 5 pCi/l, then all households benefit under the hormetic exposure-response function. Under the assumed lognormal distribution of indoor-air radon, only

3.8 percent of households have radon levels above 5 pCi/l. If regulations are limited to these households, benefits exceed costs for MCLs of 1000 pCi/l and smaller under both LNT and hormetic models. Moreover, net benefits are maximized at the most stringent MCL(100 pCi/l) under both of these models (Table VI). In contrast, under the hockey-stick model net benefits remain negative at all MCLs. Under the hockey-stick model, only households with indoor-air radon concentrations greater than 10 pCi/l benefit from reduced radon concentration and these households represent less than one-quarter of the households with indoor-air radon levels greater than 5 pCi/l.

Finally, we consider restricting the drinking-water regulations to households having indoor-air radon levels greater than 7.5 pCi/l. These households constitute only 1.6 percent of the full population. As shown in Table VII, in this case net benefits are positive at all potential MCLs for all three exposure-response functions and are maximized at the most stringent MCL. When regulations are targeted to the households with the highest indoor-air radon levels, the smallest MCL is optimal under all three exposure-response functions. In this case, all affected households benefit from reduced radon exposure under the LNT and hormetic exposure-response functions, but only about half (those with indoor-air radon greater than 10 pCi/l) benefit under the hockey-stick exposure-response function.

Note that the relative benefits of regulation under the three exposure-response models depend on the subset of households to which regulation is targeted. It is not the case that one model always predicts greater benefits than another. For the full population, benefits are largest under the LNT model, intermediate under the hockey-stick model, and smallest (and negative) under the hormetic model (Table IV). When regulations are targeted to households with indoor-air radon greater than 4 pCi/l, benefits remain largest under the LNT model but are intermediate under the hormetic model and smallest under the hockey-stick model (Table V). When regulations are further restricted to households with indoor-air radon greater than 5 pCi/l, benefits are largest under the hormetic model, intermediate under the LNT model, and remain smallest under the hockey-stick model (Table VI). Finally, when regulations are restricted to households with indoor-air radon greater than 7.5 pCi/l, benefits are largest under the hormetic model, intermediate under the hockey-stick model, and smallest under the LNT model (Table VII). As illustrated in

Figure 1, the relative slopes of the exposure-response functions vary with background radon exposure, and so the relative benefits of reducing exposure under the three models depend on the background exposure of individuals who are affected.

Uncertainty about Exposure-ResponseModel

In practice, the most accurate exposure-response model is rarely if ever known and it is useful to represent this uncertainty by assigning probabilities to alternative models. For illustration, assume that linear and nonlinear models are judged to be equally likely and, conditional on the exposure-response function being nonlinear, the hormetic and hockey-stick models are judged to be equally likely. That is, the probability assigned to the LNT model is one-half and the probabilities assigned to the hormetic and hockey-stick functions are each one-quarter. In this case, the expected benefits at each MCL can be calculated as the probability-weighted benefits conditional on each exposure-response function, i.e., one-half times the benefits under the LNT model plus one-quarter times the benefits conditional on the hormetic model plus one-quarter times the benefits conditional on the hockey-stick model. Expected net benefits are calculated as expected benefits less costs (equivalently, as the probability-weighted average of the net benefits under each model).

The expected net benefits for each potential MCL are reported in Table VIII as a function of the subset of households on which regulations are targeted, assuming costs are 80 percent as large as reported in the RIA. For the full population, no regulation is optimal when there is model uncertainty, even though the most stringent MCL is optimal under the LNT model which is assumed to be the most likely. Similarly, no regulation is optimal with model uncertainty when the regulation is targeted alternatively to households with indoor-air radon levels greater than 4 pCi/l and greater than 5 pCi/l. Without model uncertainty, the most stringent MCL is optimal for households with indoor-air radon greater than 4 pCi/l under the LNT model, and for households with indoor-air radon greater than 5 pCi/l under both the LNT and hormetic models. However, the net benefits under the hockey-stick model are sufficiently unfavorable for these populations that even a one-fourth probability that the hockey-stick model is most accurate offsets the positive net benefits under LNT and hormetic models. Finally, if the

regulation is restricted to households with indoor-air radon greater than 7.5 pCi/l, then the most stringent MCL is optimal with model uncertainty. For this subpopulation, the most stringent MCL is also optimal under each of the alternative exposure-response functions.

When uncertainty about the most accurate exposure-response function is recognized, the most efficient level of regulation can be identified by maximizing the expected net benefits, calculated as the probability-weighted average of the net benefits under each of the alternative models. When the same decision is optimal under each of the possible models, it is also optimal when there is uncertainty about which model is most accurate. In the example illustrated in Table VIII, when regulations are restricted to households with indoor-air radon greater than 7.5 pCi/l, the most stringent MCL is optimal under each of the alternative exposure-response functions and also under the probability-weighted average. In contrast, when the optimal regulation depends on the exposure-response function, the optimal regulation when there is model uncertainty depends on both the probabilities assigned to each model and the sizes of the net benefits under each model. When regulations are targeted to households with indoor-air radon greater than 5 pCi/l, for example, the most stringent MCL is optimal under the LNT and hormetic models, but no regulation is optimal under model uncertainty. This is because the net loss associated with the most stringent MCL under the hockey-stick model, $12 million, is enough larger than the net gains under the LNT and hormetic models ($3.1 and $3.4 million, respectively) that the expected net benefits of regulation are negative, even when the probability that the hockey-stick model is most accurate is only one-fourth.

6. Conclusions

Admitting the possibility of nonlinear exposure-response functions for environmental-health risks does not preclude the possibility of economic evaluation of regulations. However, evaluation requires more information than when a linear no-threshold model is assumed, because the benefits of reducing exposure depend on the initial exposure. Under a hormetic exposure-response function, reducing exposure may even prove harmful. When exposure differs among a population, it is necessary to estimate the change in exposure and hence change in risk for subpopulations facing different initial exposure levels. The aggregate benefits of a regulation will then reflect

the differential benefits across the population, including the numbers of people who are benefited or harmed. Under a hockey-stick model, some individuals may benefit while others are unaffected; under a hormetic model, some individuals may benefit while others are harmed.

In the example case of regulating radon in drinking water, we find that substituting hormetic or hockey-stick exposure-response functions for the linear no-threshold model assumed in EPA's regulatory impact analysis yields no change to the initial conclusion that these regulations are not justified on a benefit-cost criterion. Indeed, social benefits of regulation under the hormetic and hockey-stick exposure-response functions are much smaller than under the LNT model. However, if regulations on drinking-water radon could be targeted to households having high indoor-air radon concentrations, then regulation could be economically justified. In this case, the estimated benefits of controlling radon concentrations in drinking water can be, but are not necessarily, larger under the alternative nonlinear exposure-response functions than under the standard linear no-threshold model.

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Fig. 1. Alternative exposure-response functions

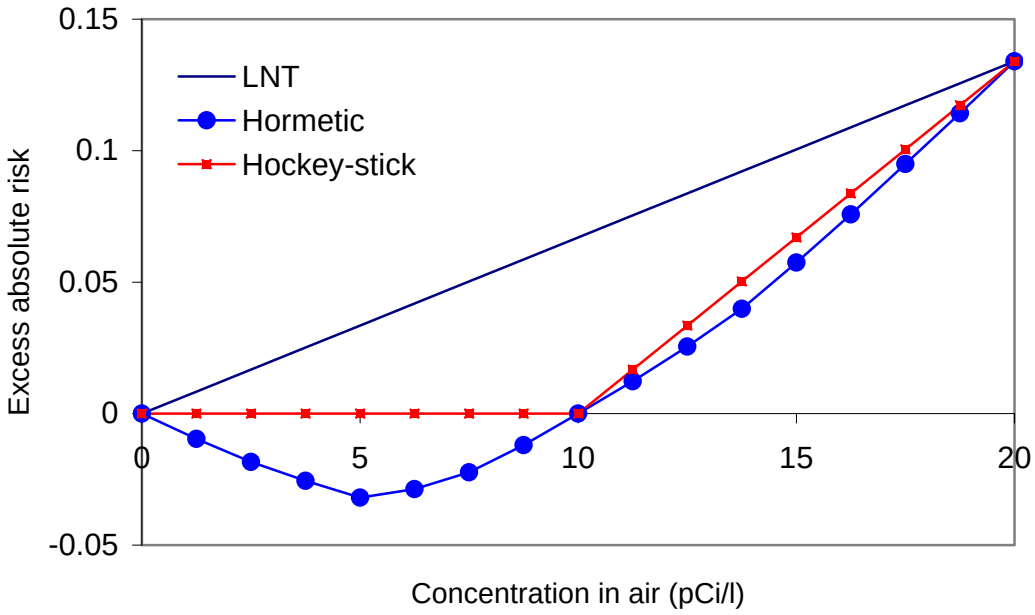

Table I. Reduction in Radon exposure for alternative MCLs

(1) Potential MCL (Rn in pCi/l)	(2) Total Population > MCL (thousands)	(3) Population in bin (thousands)	(4) Mean radon	(5) Population-weighted mean radon conditional on > MCL	(6) Reduction in population-weighted mean radon
4000	77.2	77.2	8000	8000	4000
2000	381	303.8	3000	4013	2013
1000	1695	1314	1500	2065	1065
700	3558	1863	850	1429	729
500	6893	3335	600	1028	528
300	16,641	9748	400	660	360
100	56,054	39,413	200	337	237
0	88,100	32,046	50	232	232

Notes: Column 2 from RIA Table 3-3.

Table II. Reduction in lung cancer mortality for under alternative models

(1)	(2)	(3)	(4)	(5)	(6)	(7)
				Reduction in cancers per year		
Potential MCL (Rn in pCi/l)	Total Population > MCL (thousands)	Reduction in population-weighted mean Rn	RIA	LNT	Hormetic	Hockey-stick
4000	77.2	4000	2.9	3.0	-3.0	0.05
2000	381	2013	7.3	7.3	-7.5	0.12
1000	1695	1065	17.8	17.3	-17.6	0.29
700	3558	729	26.1	24.8	-25.3	0.42
500	6893	528	37.6	34.8	-35.5	0.59
300	16,641	360	62.0	57.3	-58.5	0.97
100	56,054	237	120.0	126.9	-129.4	2.14
0	88,100	232		195.9	-199.7	3.31

Notes: Column 2 from RIA Table 3-3. Column 5 from RIA Table ES-6.

Table III. Benefits and costs of alternative MCLs under LNT and nonlinear models ($ millions)

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Benefits			Net benefits		
Potential MCL (Rn in pCi/l)	Costs	LNT	Hormetic	Hockey-stick	LNT	Hormetic	Hockey-stick
Baseline	0	0	0	0	0	0	0
4000	34.5	17.14	-17.48	0.29	-17.36	-51.98	-34.21
2000	61.1	42.58	-43.41	0.72	-18.52	-104.51	-60.38
1000	121.9	100.20	-102.16	1.69	-21.70	-224.06	-120.21
700	176.8	143.95	-146.76	2.43	-32.85	-323.56	-174.37
500	248.8	201.96	-205.91	3.41	-46.84	-454.71	-245.39
300	399.1	332.61	-339.11	5.62	-66.49	-738.21	-393.48
100	807.6	736.17	-750.56	12.43	-71.43	-1558.16	-795.17

Notes: Column 2 from RIA Table ES-7. Costs include annualized treatment, monitoring, and O&M costs, exclude record-keeping, reporting and administrative costs.

Table IV. Alternative analysis: 80% costs, full population ($ millions)

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Benefits			Net benefits		
Potential MCL (Rn in pCi/l)	Costs	LNT	Hormetic	Hockey-stick	LNT	Hormetic	Hockey-stick
Baseline	0	0	0	0	0	0	0
4000	27.6	17.14	-17.48	0.29	-10.5	-45.1	-27.3
2000	48.9	42.58	-43.41	0.72	-6.3	-92.3	-48.2
1000	97.5	100.20	-102.16	1.69	2.7	-199.7	-95.8
700	141.4	143.95	-146.76	2.43	2.5	-288.2	-139.0
500	199.0	201.96	-205.91	3.41	2.9	-405.0	-195.6
300	319.3	332.61	-339.11	5.62	13.3	-658.4	-313.7
100	646.1	736.17	-750.56	12.43	90.1	-1396.6	-633.6

Table V. Alternative analysis: 80% costs, population > 4 pCi/l (5.7 percent of households) ($ millions)

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Benefits			Net benefits		
Potential MCL (Rn in pCi/l)	Costs	LNT	Hormetic	Hockey-stick	LNT	Hormetic	Hockey-stick
Baseline	0	0	0	0	0	0	0
4000	1.58	0.98	0.39	0.29	-0.60	-1.19	-1.29
2000	2.79	2.43	0.97	0.72	-0.36	-1.83	-2.07
1000	5.57	5.73	2.28	1.69	0.15	-3.30	-3.88
700	8.08	8.22	3.27	2.43	0.14	-4.81	-5.65
500	11.37	11.54	4.59	3.41	0.17	-6.79	-7.96
300	18.24	19.01	7.55	5.62	0.76	-10.69	-12.63
100	36.92	42.06	16.71	12.43	5.15	-20.20	-24.48

Table VI. Alternative analysis: 80% costs, population > 5 pCi/l (3.8 percent of households) ($ millions)

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Benefits			Net benefits		
Potential MCL (Rn in pCi/l)	Costs	LNT	Hormetic	Hockey-stick	LNT	Hormetic	Hockey-stick
Baseline	0	0	0	0	0	0	0
4000	1.04	0.64	0.65	0.29	-0.40	-0.40	-0.75
2000	1.85	1.59	1.61	0.72	-0.26	-0.24	-1.13
1000	3.69	3.75	3.79	1.69	0.06	0.10	-2.00
700	5.35	5.39	5.45	2.43	0.04	0.09	-2.92
500	7.53	7.56	7.64	3.41	0.03	0.11	-4.12
300	12.08	12.45	12.58	5.62	0.37	0.50	-6.46
100	24.44	27.55	27.85	12.43	3.11	3.41	-12.01

Table VII. Alternative analysis: 80% costs, population > 7.5 pCi/l (1.6 percent of households) ($ millions)

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Benefits			Net benefits		
Potential MCL (Rn in pCi/l)	Costs	LNT	Hormetic	Hockey-stick	LNT	Hormetic	Hockey-stick
Baseline	0	0	0	0	0	0	0
4000	0.06	0.28	0.45	0.29	0.22	0.39	0.23
2000	0.11	0.70	1.12	0.72	0.59	1.01	0.61
1000	0.21	1.64	2.63	1.69	1.43	2.42	1.48
700	0.31	2.36	3.78	2.43	2.05	3.48	2.13
500	0.43	3.31	5.31	3.41	2.88	4.88	2.98
300	0.69	5.45	8.74	5.62	4.76	8.05	4.93
100	1.40	12.06	19.34	12.43	10.67	17.94	11.04

Table VIII. Expected net benefits with model uncertainty (80% costs) ($ millions)

(1)	(2)	(3)	(4)	(5)
Potential MCL (Rn in pCi/l)	Full population	Population > 4 pCi/l	Population > 5 pCi/l	Population > 7.5 pCi/l
Baseline	0	0	0	0
4000	-23.3	-0.92	-0.49	0.27
2000	-38.3	-1.16	-0.47	0.70
1000	-72.5	-1.72	-0.44	1.69
700	-105.5	-2.54	-0.69	2.43
500	-148.7	-3.60	-0.99	3.40
300	-236.3	-5.45	-1.30	5.62
100	-462.5	-8.60	-0.60	12.58

Note: probability (LNT) = 1/2, probability (hormesis) = 1/4, probability (hockey-stick) = 1/4