THE ECONOMICS OF CHRONIC ENVIRONMENTAL HEALTH RISKS

Erik Lichtenberg

This research was supported in part by the Economic Research Service, U.S. Department of Agriculture, under Cooperative Agreement 58-3AEM-8-0065,1. The comments of John Horowitz are gratefully acknowledged.

Department of Agricultural and Resource Economics
University of Maryland at College Park

Working Paper No. 89-32

November, 1989

Environmental health risks have emerged as a driving force in a wide range of public policy areas, including pesticides and other agricultural chemicals, ground and surface water pollution, air pollution, occupational safety and food safety. The public has exhibited special concern over risks that are chronic in nature, that is, caused by cumulative exposure to toxic elements, for example, cancers, birth defects and genetic damage. Because the extent of risk is a function of cumulative exposure, these issues are inherently dynamic. Yet policy discussions of these problems have tended to be static, focusing on long run costs and benefits and all-or-nothing policy options. For example, debates over policies aimed at curbing exposure to environmental carcinogens have been dominated by discussions of whether or not dose-response estimates are sufficiently "conservative", and whether or not suspected carcinogens should be banned in light of the estimated eventual number of cancer cases. Dynamic issues such as how quickly such substances should be phased out, the appropriateness of phase-outs versus outright bans or at what point in time remediation should be initiated have received little attention.

This paper focuses on the implications of alternative dose-response behaviors for risk reduction policy. We show that the shape of the dose-response curve influences the <u>type</u> of policy that should be undertaken (e.g., an immediate ban versus gradual reductions in pollutant levels) as well as the rate at which policies should be phased in and time at which remediation should be initiated. An immediate ban on use of suspected carcinogens is likely to be desirable if the dose-response curve is concave. If the dose-response curve is linear, an immediate reduction to

the equilibrium usage level will be optimal, while gradual reductions in use will be optimal if the dose-response function is S-shaped.

These results have important implications for risk assessment procedures. Chronic toxicity at low environmental or occupational exposures is estimated using data on toxicity at high doses, extrapolated using a specific functional form chosen for "conservatism". Such a procedure does more than generate more stringent standards, it biases policy choices against gradual measures in favor of more drastic ones.

The paper is organized as follows. We begin with a model of social welfare optimization when health risk is a function of cumulative pollution. We then compare optimal pollutant use over time and pollutant use in a steady state in situations where the incidence of adverse health effects is an S-shaped, linear or concave function of cumulative exposure.

Optimal Pollution under Chronic Environmental Risk

Some implications of treating pollution as a dynamic phenomenon were addressed by Keeler, Spence and Zeckhauser in the context of several models of optimal economic growth involving cumulative pollution. Of particular relevance here is their analysis of a model involving optimal use of an input (labor) that increases social welfare via the production of consumables but simultaneously adds to the stock of pollution, a bad. The utility from consumption and disutility from accumulated pollution were assumed additively separable and known with certainty. They showed that under their assumptions about the shapes of the utility and disutility functions, there exists a unique equilibrium pollution level. If the stock of pollution is initially low (high), the use of the input should start high (low) and gradually be reduced (increased). If initial pollution is

very high, a temporary ban on polluting activity may be appropriate.

Cropper investigated a case where the impact of accumulated pollution is stochastic, i.e., where accumulated pollution poses a risk. She restricted her attention to catastrophic risks, that is, the risk that the decision maker (an individual or society) is eradicated. In her model, social welfare is the expected utility of consumption, defined as the utility of consumption times the probability that the catastrophe does not occur. When the probability distribution of the catastrophe is unimodal, there may be no, one or many equilibria. In the multiple equilibrium case, the appropriate policy given an initially very high stock of pollution may be to allow increases in polluting activity.

While chronic health effects such as cancer may be catastrophic to the individuals contracting them, they are certainly not so on a social level, especially very low incidence health effects such as environmentally induced ones tend to be. Public policy debates focus on the appropriate tradeoffs between the expected number of cases of illness or statistical deaths and national income. We generalize Cropper's model to allow for such tradeoffs. In contrast to Cropper, who assumed that the relationship between exposure and the risk of an adverse health effect could be represented by a unimodal probability distribution, we derive possible shapes for the dose-response function from the quantitative risk assessment literature, permitting examination of a broader range of cases. Like Keeler, Spence and Zeckhauser, we assume that the benefits and costs of toxic pollution are additively separable. We conceive of the cost of accumulated pollution, or cumulative exposure to a toxic pollutant, as a function of an increased incidence of chronic health effects such as cancer in a given population.

Let X denote the usage level of a polluting input and assume that production can be expressed as a neoclassical function of X, f(X), so that $f_X > 0$ and $f_{XX} < 0$. (Subscripts denote derivatives.) Let the unit cost of the polluting input be w, so that national income, normalized by the price of output, is f(X) - wX. Let G(S) denote the increased risk of adverse health effects in the populations given cumulative exposure S, where $G_S \geq 0$. Let v denote the social value of an increase in adverse health effects relative to the social value of national income, for example, the value of saving a life times the size of the population or the social willingness to pay to avoid birth defects times the size of the population, both normalized by the output price. Social welfare in any period is thus

$$W = f(X) - wX - vG(S). \tag{1}$$

Assume that the rate of change of cumulative exposure over time is

$$\dot{S} = h(X) - \delta S \tag{2}$$

where δ represents the breakdown of pollutants by natural factors in the environment and the human body and $h_X > 0$. If there is a threshold due to the existence of natural capacity for breaking down the pollutant, then $h(X) = 0$ for values of X less than a threshold value of pollution $\hat{X} > 0$. If no threshold exists, then $h(0) = 0$. Clearly $h(X) \leq X$, the addition to cumulative exposure, cannot exceed the amount of the pollutant used in production. This suggests that, while $h_{XX} > 0$ is possible for some X, eventually $h_{XX} \leq 0$, i.e., $h(\cdot)$ is most likely an S-shaped curve.

Leaching of nitrates from chemical fertilizer into groundwater provides a case in point. At low fertilizer application rates, the crop will take up essentially all the nutrients supplied, and the groundwater nitrate level will remain unchanged. As the fertilizer application rate is increased beyond the crop's nutritional requirements, the groundwater

nitrate level will increase, conceivably at an increasing rate. The groundwater nitrate level will always increase by less than the amount of fertilizer applied due to crop uptake and bacterial denitrification. Because crop uptake and bacterial denitrification capacity are limited, the increase in the groundwater nitrate level will eventually differ from the fertilizer application rate roughly by a constant. Thus one would expect h(X) to increase first at an increasing rate, then at a decreasing rate until it is parallel with a 45 degree line through the origin, as depicted in Figure 1.

The social optimization problem involves choosing pollution X to

(3) $\max \int_0^\infty \{f(X) - wX - vG(S)\}e^{-rt}dt$

subject to (2) and the constraints that $X \geq \hat{X}$ and S be non-negative, where r is the periodic interest rate. The Hamiltonian is

(4) $\mathcal{H} = f(X) - wX - vG(S) - \mu[h(X) - \delta S] + \lambda(\hat{X} - X)$,

where $\mu \geq 0$ is the negative of the current value costate variable, expressing the absolute value of the decrease in social welfare caused by in increase in cumulative exposure, i.e., the marginal cost of exposure. The necessary conditions for a maximum are

(5a) $f_X - w - \mu h_X - \lambda = 0$

(5b) $(r+\delta)\mu - vG_S = \dot{\mu}$

plus equation (2).

The necessary conditions will be sufficient if the Hamiltonian, equation (4), is jointly concave in X and S. This requires that

(6a) $f_{XX} - \mu h_{XX} \leq 0$

and

(6b) $-vG_{SS} \leq 0$.

The inequality in (6a) will always hold if $h_{XX} \geq 0$. If $h_{XX} < 0$, the value

of the marginal product of the pollutant must decrease more rapidly than the social disutility of the increase in cumulative exposure. The inequality in (6b) holds when the dose-response function is linear or for low levels of S when the dose-response function is S-shaped, but not for high levels of S when the dose-response function is S-shaped or when the dose-response function is concave. The implications of this nonconcavity will be discussed later.

Equation (5a) implies that pollutant use in any time period will be no greater (and would typically be expected to be less) than the myopic usage level X^m defined by $f_X(X^m) - w = 0$. This myopic usage level X^m is assumed to be greater than the threshold $\hat{X}$ so that a pollution problem exists.

From equations (2) and (5a), along $\dot{S} = 0$,

$$(7) \qquad \left.\frac{d\mu}{dS}\right|_{\dot{S}=0} = \delta(f_{XX}-\mu h_{XX})/h_X^2 < 0,$$

i.e., $\dot{S} = 0$ is downward sloping in the (S,μ) plane. If a threshold $\hat{X}$ exists, then $\dot{S} = 0$ passes through a point $(0,\hat{\mu})$ where $\hat{\mu} = [f_X(\hat{X})-w]/h_X(\hat{X})$. If no threshold exists, then $\dot{S} = 0$ passes through a point $(0,\hat{\mu})$ where $\hat{\mu} = [f_X(0)-w]/h_X(0)$. From equation (2), $X \to \infty$ along $\dot{S} = 0$ as $S \to \infty$; since $f_X - w < 0$ for some $X > 0$, $\mu < 0$ along $\dot{S} = 0$ for some $S > 0$ as well. The preceding discussion of h(X) suggests that h_X is likely to be low for low levels of X (and therefore of S along $\dot{S} = 0$), increasing and then decreasing to a constant as X (and therefore S) increases, and that h_{XX} is likely to be increasing (although less than h_X) and then decreasing toward zero. For a neoclassical production function $f_{XX} \to 0$ as $X \to \infty$, which, along with the assumption that h_X eventually becomes constant, suggests that $d\mu/dS \to 0$ along $\dot{S} = 0$ as S becomes large. This suggests that $|d\mu/dS|$ decreases, then increases, then decreases again toward zero as S becomes

large, as depicted in Figure 2. Equation (2) and the fact that μ and X are inversely related implies that $\dot{S} < 0$ for (S,μ) to the right of $\dot{S} = 0$ and vice versa.

From equation (5b), $\dot{\mu} = 0$ whenever

$$(8) \qquad (r+\delta)\mu = vG_S,$$

so that

$$(9) \qquad \left.\frac{d\mu}{dS}\right|_{\dot{\mu}=0} = \frac{vG_{SS}}{(r+\delta)},$$

the sign of $d\mu/dS$ along $\dot{\mu} = 0$ will be the same as the sign of G_{SS}, i.e., the optimal time path of pollution depends on the shape of the dose-response function.

S-shaped Dose-Response Function

Consider first the optimal time path and steady state levels of pollution when the probability of an adverse health effect is an S-shaped function of cumulative exposure. In this case there will be a level of cumulative exposure $\hat{S}$ such that $G_{SS} > (<)\ (=)\ 0$ when $S < (>)\ (=)\ \hat{S}$. $\dot{\mu} = 0$ will thus be upward sloping when $S < \hat{S}$, flat when $S = \hat{S}$ and downward sloping when $S > \hat{S}$, i.e., it will be a bell-shaped curve as shown in Figure 1. From equation (5b), $\dot{\mu} < (>)\ 0$ below (above) the $\dot{\mu} = 0$ locus.

In a typical S-shaped specification such as a normal, Weibull, logistic or gamma distribution, $G_S(0) = 0$, so that $\mu = 0$ and $X = X^m$ when $S = 0$. At least one non-zero equilibrium thus exists as long as $\hat{\mu} > 0$. If the equilibrium is unique, it will also be stable, as can be seen from Figure 2. Three equilibria are also possible, as shown in Figure 3. Two of these are stable while the third is unstable. This makes intuitive

sense, since the unstable equilibrium involves a relatively high level of pollution S and relatively high marginal health damage G_S. Decreasing the use of the pollutant results in relatively large reductions in the risk of adverse health effects and therefore has a high payoff if the social value of health damage v is high. If the social value of health damage is low, increasing the use of the pollutant results in relatively small increases in the risk of adverse health effects and therefore makes sense if the social value of health damage is low.

If a unique equilibrium exists, the optimal policy depends on whether equilibrium exposure is greater or less than $\hat{S}$. If equilibrium exposure is less than $\hat{S}$ and initial exposure is less than equilibrium exposure, $S_0 < S^* < \hat{S}$, (see Figure 2a), regulation will initially be lax and will become more stringent as time goes on. Exposure S and the marginal cost of exposure μ will build up to equilibrium levels and pollutant use X will decline. If initial exposure exceeds equilibrium exposure, $S^* < S_0 < \hat{S}$, the opposite will occur. Pollutant use will be kept at a sufficiently low level to allow decumulation of the stock of pollution, so that exposure will gradually fall to the equilibrium level. As exposure falls, the marginal cost of exposure will decline and pollutant use will expand.

If equilibrium exposure S* exceeds $\hat{S}$, the optimal policy will be the mirror image of that just described (see Figure 2b). If initial exposure is less than the equilibrium level, regulation will initially be relatively stringent and will become more lax over time. Pollutant use will be restricted initially, but not enough to prevent increased exposure. As exposure increases, the marginal cost of exposure will fall because $G_{SS} < 0$ and thus pollutant use will gradually increase. If initial exposure exceeds the equilibrium level, pollutant use will initially be relatively

unrestricted. As exposure declines, the marginal cost of exposure will rise and pollutant use will gradually be reduced to the equilibrium level. (Recall that $h(X^m) < \delta S$ is possible for large S.)

Note though that when $S > \hat{S}$, the Hamiltonian is nonconcave in S and equations (2) and (5a,b) are likely to indicate a minimum rather than a maximum solution. Such high initial exposure levels indicate an extraordinarily high incidence of the adverse health effect in the population, e.g., over 50 percent. If the willingness to pay to avoid the adverse health effect is high (the adverse health effect is serious, e.g., cancer a opposed to minor respiratory discomfort) and/or the exposed population is large, the constraint on X will be binding and it will be optimal to restrict pollutant use to the threshold level immediately and allow exposure to decumulate until production at $X > \hat{X}$ is worth more than the value of health damage. If there is no threshold, this amounts to a temporary (albeit possibly long-lasting) ban on the use of the pollutant. In this case the optimal policy may be cyclical, with period of restricting pollutant use to the threshold level alternating with periods of gradual relaxation of pollutant use restrictions.

If the willingness to pay to avoid the adverse health effect is low and/or the exposed population is small, though, equations (2) and (5a,b) will give a maximum and the optimal policy will be that described above.

When there are multiple equilibria, the optimal policy depends on the initial exposure level S_0 and the relative social value of health damage v. If initial exposure is less than $\hat{S}$, equilibrium exposure S* will also be less than $\hat{S}$ and the optimal policy will be the same as shown in Figure 2a. If initial exposure is greater than the unstable middle equilibrium level, $\hat{S} < S^{**} < S_0$, the system will tend either toward the stable higher

equilibrium S*** or a corner solution $X = \hat{X}$ and the optimal policy will be the same as shown in Figure 2b. If initial exposure lies between $\hat{S}$ and the unstable middle equilibrium level, $\hat{S} < S_0 < S^{**}$, the relevant equilibrium and optimal policy depend on the relative social value of health damage v. If v is high, the system will tend toward the low exposure equilibrium and the optimal policy involves initially restrictions on pollutant use severe enough to cause reductions in exposure (possibly involving restriction of pollutant use to the threshold level as well), with gradual reductions in stringency as exposure declines. If v is low, pollutant use will not be restricted sufficiently to produced reductions in exposure; pollutant use will be allowed to increase over time as exposure increases (and thus the marginal cost of exposure falls).

The qualitative behavior of the system in equilibrium depends on the sign of $\Delta = (f_{XX} - \mu h_{XX})\delta(r+\delta) - vG_{SS}h_X^2$. Clearly $\Delta < 0$ when $G_{SS} \geq 0$. Since G_{SS} is small in absolute value in any stable equilibrium with high levels of S, we assume that $\Delta < 0$ in these cases also. Equilibria thus have the following properties:

(1) The equilibrium use of the pollutant is decreasing in its cost ($\partial X/\partial w = \delta(r+\delta)/\Delta < 0$) and in the relative social value of health damage ($\partial X/\partial v = h_X \delta G_S/\Delta < 0$) and increasing in the decay rate of the pollution stock ($\partial X/\partial \delta = -h_X(\mu\delta + vSG_{SS})/\Delta > 0$) and the interest rate ($\partial X/\partial r = -\mu\delta h_X/\Delta > 0$).

(2) Equilibrium exposure is decreasing in the cost of the pollutant ($\partial S/\partial w = -h_X(r+\delta)/\Delta < 0$) and the relative social value of health damage ($\partial S/\partial v = \delta G_S(f_{XX} - \mu h_{XX})/\Delta < 0$) and increasing in the interest rate ($\partial S/\partial r = -\mu h_X/\Delta > 0$). It can be increasing or decreasing in the

decay rate ($\partial S/\partial \delta = [-S(r+\delta)(f_{xx}-\mu h_{xx})-\mu h_x^2]/\Delta$).

(3) The marginal cost of exposure is decreasing in the interest rate ($\partial \mu/\partial r = -\mu\delta(f_{xx}-\mu h_{xx})/\Delta < 0$), in the decay rate of the pollution stock if the equilibrium stock level is less than $\hat{S}$ ($\partial \mu/\partial \delta = -(f_{xx}-\mu h_{xx})(\mu\delta+vG_{ss})/\Delta < 0$ for $S < \hat{S}$) and in the cost of the pollutant if the equilibrium stock level is greater than $\hat{S}$ ($\partial \mu/\partial w = -h_x vG_{ss}/\Delta <$ 0 for $S > \hat{S}$). It is increasing in the relative social value of health damage ($\partial \mu/\partial v = \delta G_s(f_{xx}-\mu h_{xx})/\Delta > 0$) and in the cost of the pollutant if the equilibrium stock level is less than $\hat{S}$. It may be increasing in the decay rate of the pollution stock of the equilibrium stock level is greater than $\hat{S}$.

Linear Dose-Response Function

When the probability of the adverse health effect is linear in cumulative exposure to pollution, G_s is constant and $\dot{\mu} = 0$ only when the marginal cost of exposure is at its equilibrium level, given by

$$(11)\quad \mu^* = \frac{vG_s}{(r+\delta)},$$

as shown in Figure 4.

If $\mu^* < \hat{\mu}$, there will be a unique non-zero equilibrium. From equation (5b), $\dot{\mu} > 0$ above the $\dot{\mu} = 0$ locus and $\dot{\mu} < 0$ below it. The equilibrium is a saddle point and therefore stable. The optimal policy is to set pollutant use equal to the equilibrium level X* given by

$$(12)\quad f_x(X^*) - w - vG_s h_x/(r+\delta) = 0$$

regardless of the initial exposure level S_0, i.e., it is optimal to restrict pollutant use to the long run equilibrium level immediately. If

$S_0 < S^*$, the equilibrium exposure level, then exposure will increase over time until it reaches the equilibrium level. If $S_0 > S^*$, the opposite will occur.

Since G_S is constant, $G_{SS} = 0$ and therefore $\Delta < 0$. Equilibrium pollutant use X^*, exposure S^* and marginal cost of exposure μ^* will thus have the same properties as in the case described above.

If $\tilde{\mu} > \hat{\mu}$, the social value of health damage outweighs the social value of production at any exposure level and it is thus optimal to restrict use of the pollutant to the threshold level $\hat{X}$. Since most environmental health risks are small at typical exposure levels (i.e., on the order of 1 in 10,000 or less), this outcome is unlikely, however.

Concave Dose-Response Function

The third type of specification possible for the dose-response function is the concave one, obtained for example from the one-hit model of carcinogenesis $G(S) = 1-\exp\{-\gamma S\}$ or often from the the multi-stage model $G(S) = 1-\exp\{\gamma_0+\gamma_1 S+\gamma_2 S^2+\ldots+\gamma_n S^n\}$. Animal toxicity data indicate that some carcinogens do have concave dose-response curves; this appears to be the case for vinyl chloride and a number of other chemicals, for example (Bailar, Crouch, Shaikh and Spiegelman).

When the dose-response curve is concave, G_S is decreasing in S ($G_{SS} < 0$) and the Hamiltonian is nonconcave in S. The most likely outcome is that it will be optimal to restrict pollutant use to the threshold level immediately.

Otherwise, there will be a unique equilibrium as shown in Figure 5. From equation (5a), μ is increasing above $\dot{\mu} = 0$ and decreasing below it. The equilibrium is a saddle point and therefore stable. Because the

marginal cost of exposure decreases as exposure increases, the optimal policy will be as follows. Initial exposure less (greater) than the equilibrium level implies increasingly lax (stringent) regulation over time with a gradual increase (reduction) in pollutant use as exposure increases (decreases) toward the equilibrium level.

Implications for Risk Assessment Procedure

The preceding analysis indicates that the functional form of the dose-response function affects not only the rate at which exposures should be curtailed, but also the goals and type of policy chosen. When the dose-response curve is S-shaped, the optimal policy is likely to be a gradual phase-down of pollutant use, with some exposure allowed in the long run. When the dose-response curve is linear, the optimal policy is likely to be to restrict pollutant use to the equilibrium level immediately; equilibrium exposure will be positive but less than if the dose-response curve were assumed to be S-shaped. When the dose-response curve is concave it is more likely that all-or-nothing restrictions on pollutant use will appear justified; when all-or-nothing restrictions are not justified, the appropriate policy involves initial curtailment of pollutant use, followed by a gradual phase-in, the opposite of the S-shaped case.

One of the most contentious issues in chronic risk policy has been the appropriate estimation of dose-response functions. Chronic toxicity is generally evaluated using animal (usually mouse or rat) bioassays. To ensure detection of toxic effects while keeping the number of test animals (and thus testing costs) reasonable, the animals are given high doses of the substance under investigation. Estimating toxicity at the low exposure levels typical of environmental contamination situations

requires using a specific functional form. While S-shaped curves are believed to have the greatest general biological validity, linear and concave functions (e.g., the one-hit and multi-stage models of carcinogenesis) are often advocated (and adopted) as ways of correcting risk estimates for the uncertainties that arise in the process of risk assessment, that is, as mechanisms for giving a margin of safety to risk estimates. Debate over the choice of functional form typically revolves around the validity of the resulting point estimate of risk at average lifetime exposure.

Choosing a more "conservative" functional form obviously affects the choice of pollutant use and exposure levels. Imposing a linear form on the dose-response function means that estimates of the value of G_s will be higher for low exposures (typical of environmental and occupational situations) than with an S-shaped dose-response function. This implies that the marginal cost of exposure associated with the initial exposure level S_0 will be higher under a linear health damage process than a S-shaped one, since $\mu(0) = \int_0^T vG_s e^{-rt}dt$. From equation (10), it is evident that the equilibrium marginal cost of exposure will be higher. The choice of a linear specification thus implies tighter restriction of the use of the pollutant. Similarly, a concave dose-response model has higher values of G_s in the relevant range of exposures than either the linear or S-shaped specifications, so that the marginal cost of exposure associated with the initial exposure level and the equilibrium marginal cost of exposure will be higher than under a linear or S-shaped specification. Moreover, for low exposure levels such as those typical of environmental contaminants, the marginal cost of exposure is monotonically decreasing along the optimal trajectory under a concave specification, while it is constant under a

linear specification and monotonically increasing under a S-shaped specification. This implies that pollutant use and long run exposure will be lower at all times under a concave specification than under a linear or an S-shaped alternative.

This bias in policy toward more restrictive standards is intended. But the preceding analysis shows that using dose-response specifications to build "conservatism" into risk estimates has an additional, unintended effect, namely that it biases policy outcomes toward "all-or nothing" approaches such as bans or restrictions of pollutant use to fixed levels and away from more gradualist approaches. The more "conservative" the specification used, the more likely it is that a ban on use of a pollutant will be found to be desirable and that a more gradual approach will be ruled out.

Biasing the choice of type of policy approach seems to stretch the notion of adding a margin of safety rather far. A more satisfactory alternative might be to conduct a probabilistic risk assessment (that is, a risk assessment that estimates the error and variability associated with each point estimate of risk) that maintains the most plausible specification based on the data and biological concerns. The concern for a margin of safety can be addressed statistically by using the upper 95 or 99 percent confidence limit estimate of risk. Such a procedure accounts for uncertainty while preserving the shape of the dose-response curve, leaving the choice of the type of policy to be determined by the data rather than by arbitrary assumptions.

Conclusion

Risks of adverse health effects associated with environmental or

occupational exposure have become a growing source of public concern. While many of these risks are believed to be functions of cumulative exposure, and are therefore inherently dynamic, the bulk of policy discussion has centered on static issues such as long run costs and benefits and all-or-nothing policies such as permanent restrictions on pollutant use.

This paper analyzes optimal policy toward such risks in a dynamic context, focusing on the implications of alternative dose-response mechanisms. The shape of the dose-response curve is shown to affect policy in the short and long run. A concave dose-response curve likely implies the desirability of an immediate ban on use of suspected carcinogens, while a linear dose-response curve implies an immediate reduction to the equilibrium usage level and an S-shaped dose-response curve implies gradual reductions to a non-zero equilibrium use level. In many cases, the shape of the dose-response curve is a matter of choice for regulatory agencies, because data on chronic toxicity are typically collected at exposures much higher than those found in the environment. The choice of a functional form for the dose-response curve is typically made on the basis of "conservatism" to account for error in the estimation process, rather than for biological plausibility. Such a procedure generates more stringent standards, as intended, but also biases policy choices against gradual measures in favor of more drastic ones. For this reason, it seems preferable to find alternative ways of adjusting for uncertainty that preserve the most plausible shape of the dose-response curve.

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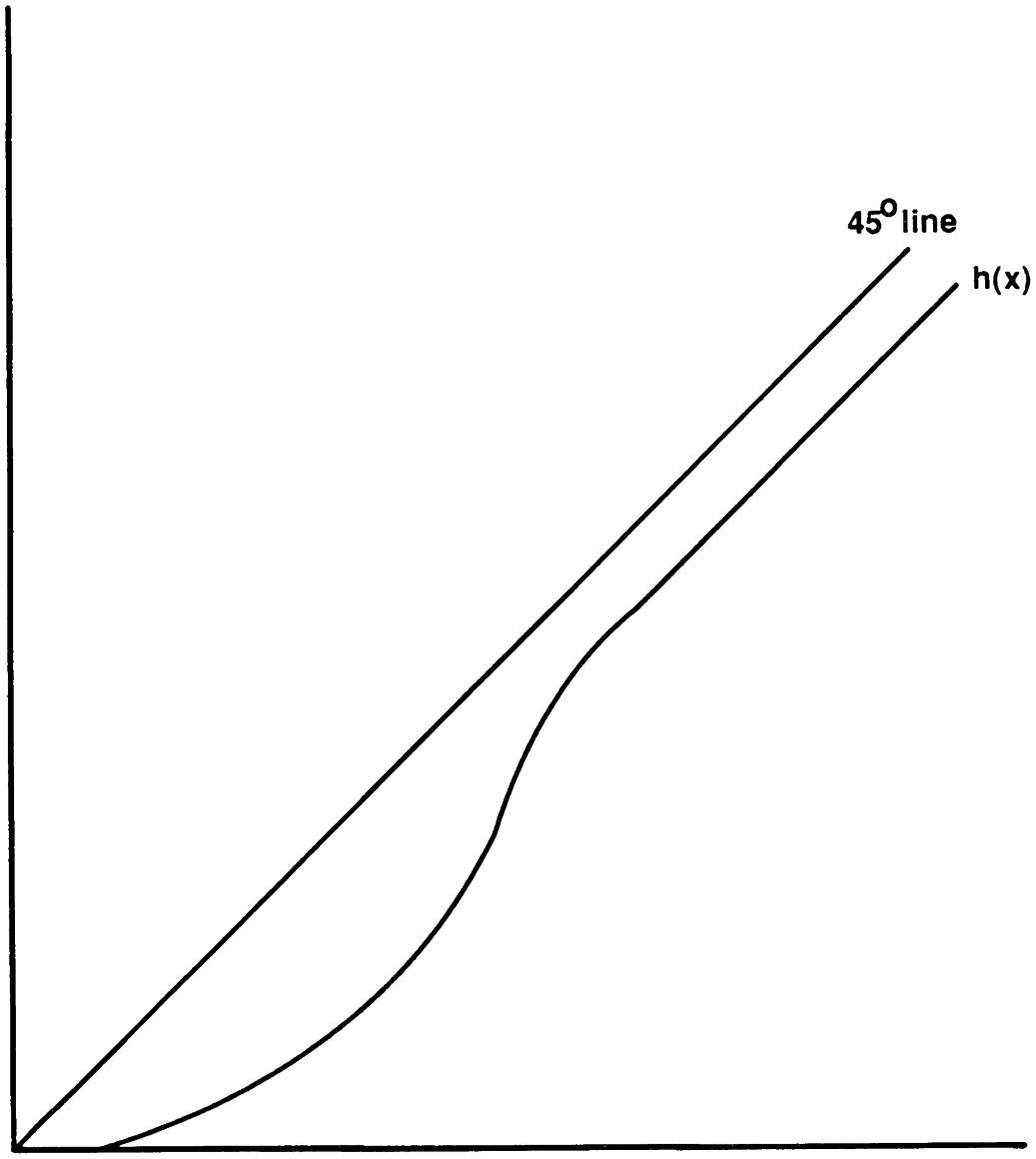

Figure 1: Change in cumulative exposure as a function of pollutant use (x).

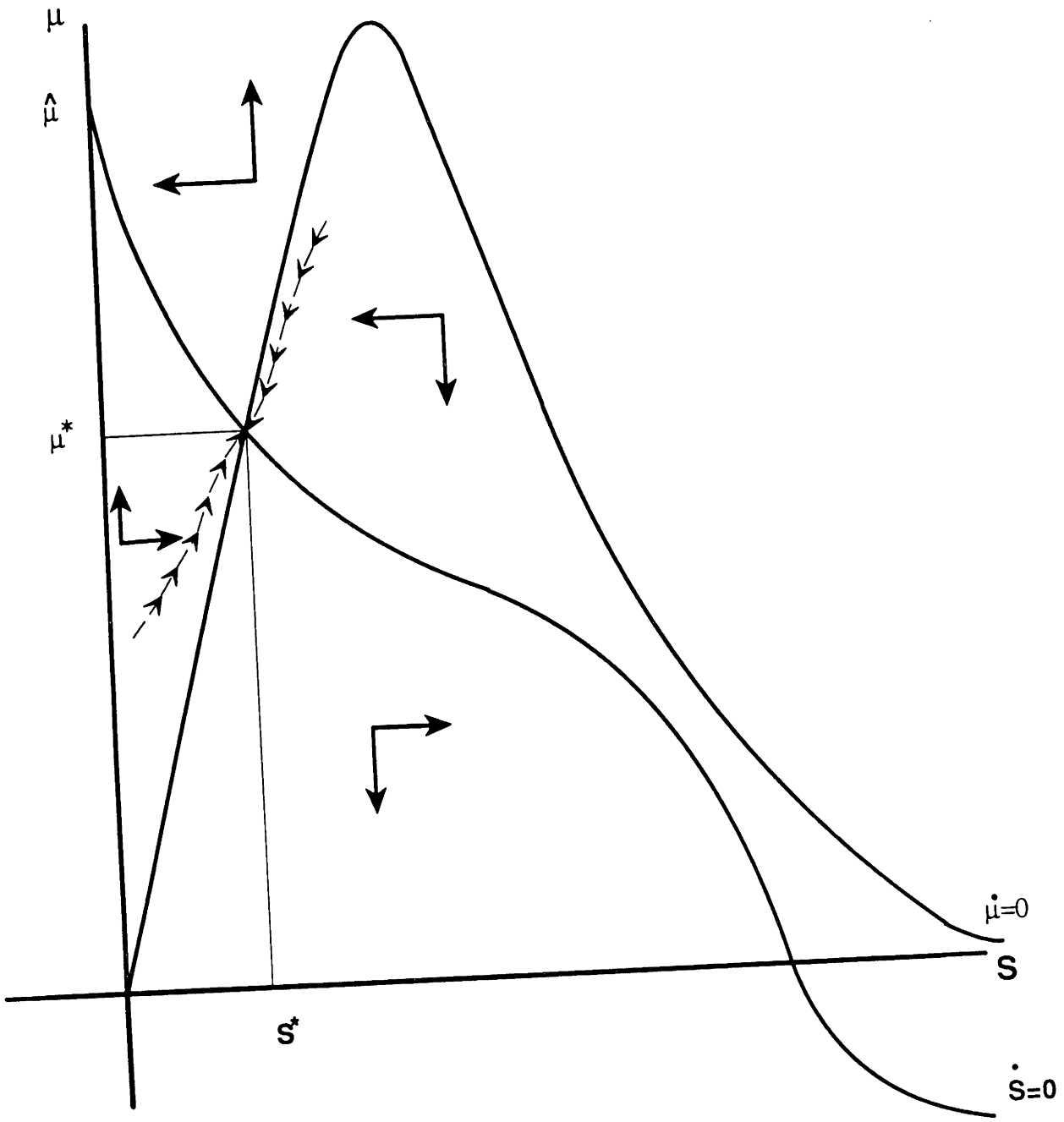

Figure 2a: Unique (low) equilibrium, S-shaped dose-response curve

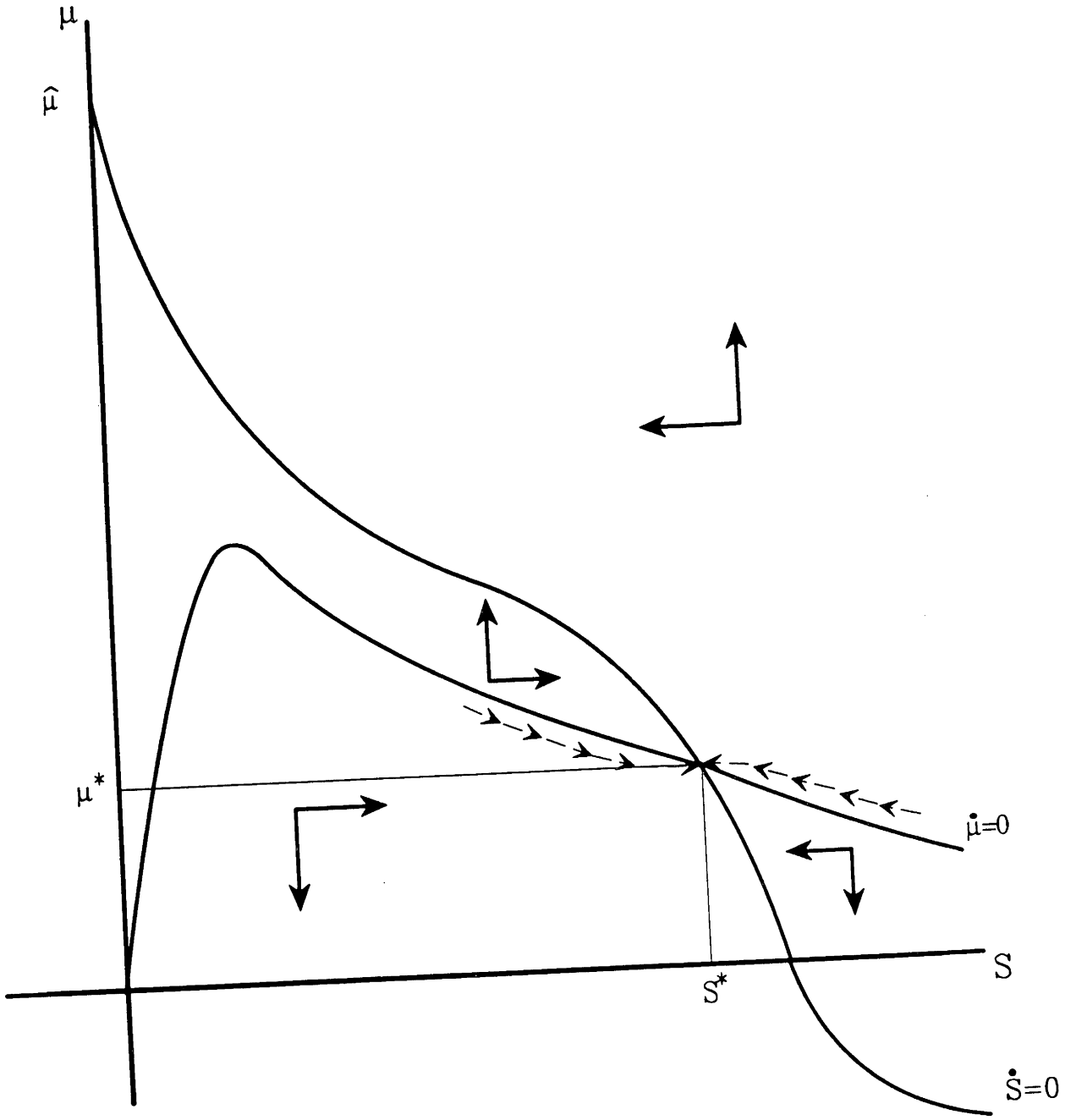

Figure 2b: Unique (high) equilibrium, S-shaped dose-response curve

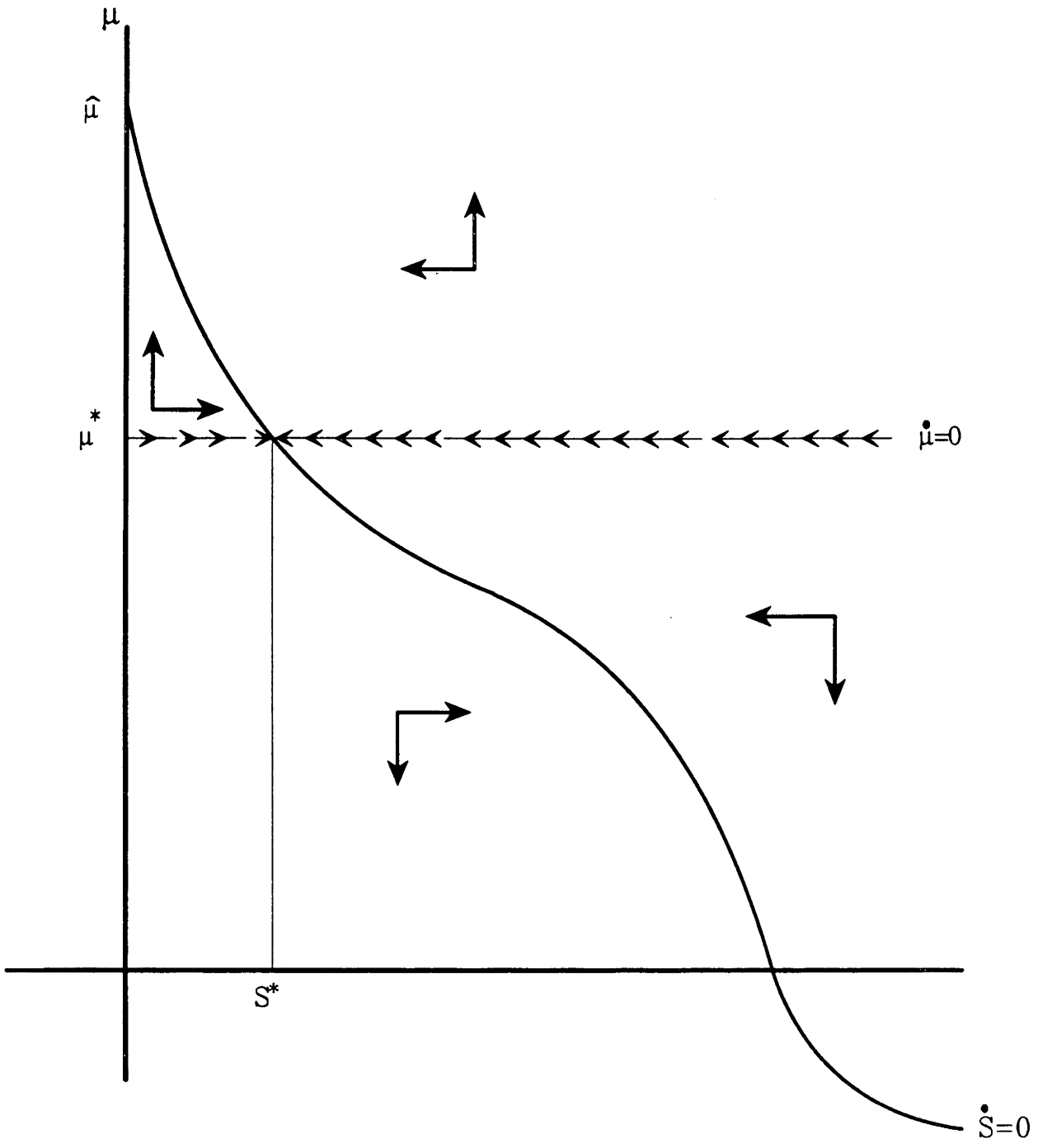

Figure 4: Equilibrium, linear dose-response curve

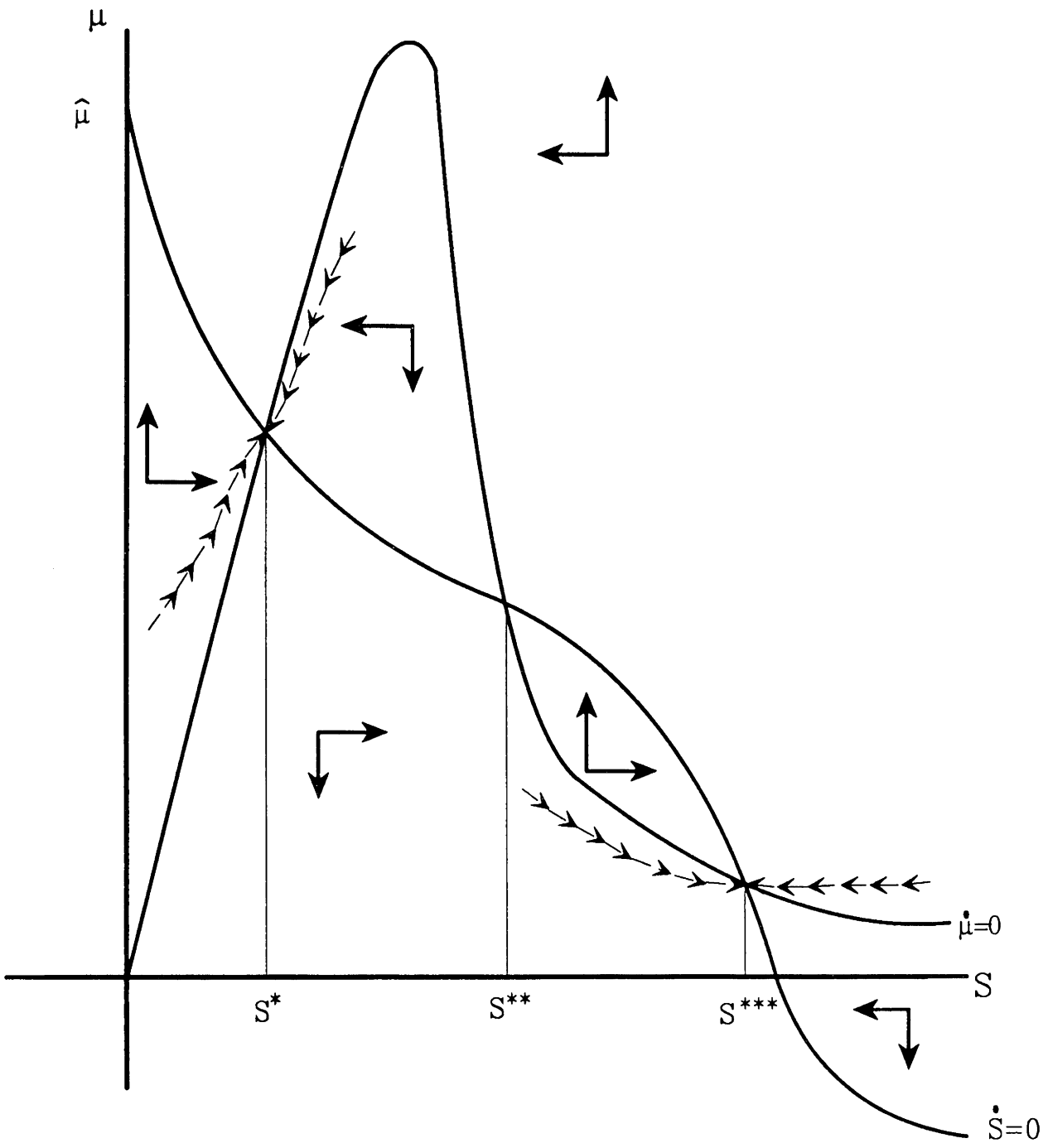

Figure 3: Multiple equilibria, S-shaped dose-response curve

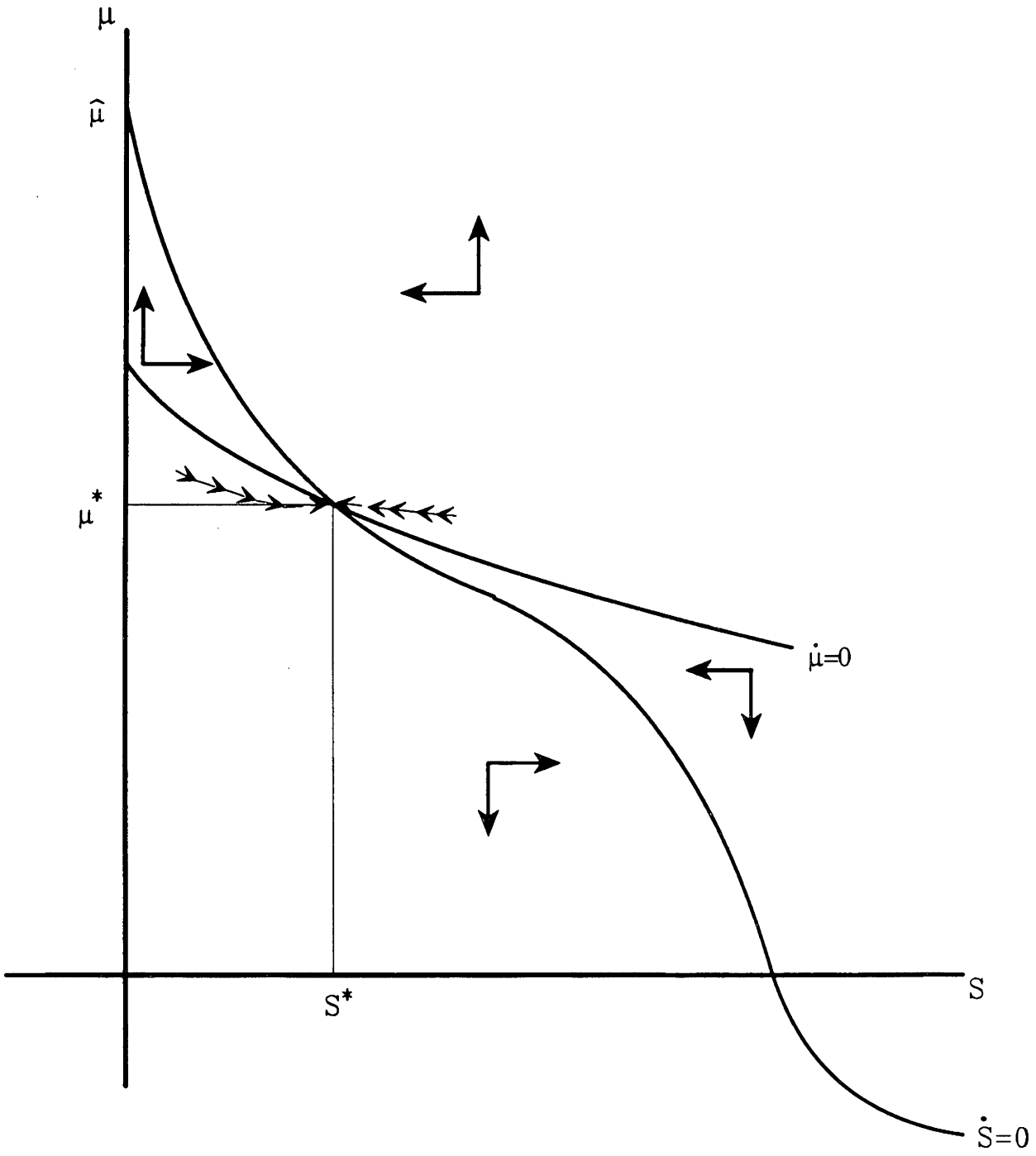

Figure 5: Equilibrium, concave dose-response curve