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Evidentiary Standards and Animal Data

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ABSTRACT

Those who wish to deny some instance of environmental injustice often attempt to place inappropriate evidentiary burdens on scientists who show disproportionate pollution effects on vulnerable populations. One such evidentiary standard is the epidemiological-evidence rule (EER). According to EER, legitimate causal inferences about pollution-related harm (and actions to reduce probable environmental injustice) require human-epidemiological data, not merely good animal or laboratory data. This article summarizes the grounds for supporting EER, evaluates central scientific problems with EER, assesses key ethical difficulties with EER, then concludes that EER ought not be used either to deny otherwise-probable environmental injustice or to delay possible action to correct well-documented pollution-related harms.

BECAUSE SCIENCE IS the gold standard for evaluating pollution-related health effects, those skeptical of environmental-injustice (EIJ) charges often employ a number of questionable scientific strategies.[1] One such strategy is to demand inappropriately high evidentiary standards of those who assert EIJ—who claim some pollutant disproportionately affects vulnerable populations. One evidentiary standard is the epidemiological-evidence rule (EER), according to which causal inferences about harm (and actions to reduce probable EIJ) require human-epidemiological data, not merely animal or laboratory data. For instance, although the US National Academy of Sciences says children are not adequately protected by current pesticide standards,[2] chemical-industry groups used EER to reject the 10-fold safety factor (for children's pesticide-exposure standards) mandated by the 1996 US Food Quality Protection Act.[3] A key argument (against this more-protective standard) was that, despite abundant animal data and children's known higher sensitivity to toxins, scientists also must provide human-epidemiological data in order to confirm pesticide-related EIJ.

To evaluate the soundness of EER, the article (1) summarizes the grounds for supporting EER, (2) evaluates central scientific problems with EER, and (3) assesses key ethical difficulties with EER. The article concludes that EER ought not be used to deny otherwise-probable EIJ or to delay possible action to correct it.

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GROUNDS FOR SUPPORTING EER

EER supporters include University of California geneticist Bruce Ames and many industry-funded scientists like Michael Gough or Elizabeth Whelan (President of the industry front group, the American Council on Science and Health). They claim animal testing of pollutants often is unreliable,[4] largely "speculative," and therefore that regulators should use "epidemiological evidence in humans."[5]

On one hand, these industry scientists are partly right. While results of animal tests often are not precise predictors of human harm, well-designed, sufficiently-sensitive, human-epidemiological studies frequently provide the most direct evidence of pollutant-related human harm. As a prominent US National Academy of Sciences panel put it, "uncertainty in extrapolating among different species of mature animals is appreciable. . . . [Because of] interspecies maturation patterns . . . choice of an appropriate animal model for pesticide toxicity of neonates, infants, and children becomes even more complex."[6] Echoing this complexity, risk assessors routinely apply an uncertainty factor of 10 to animal results—to account for interspecies variation—and another factor of 10 to account for intraspecies variation.[7] Both applications suggest imprecise animal tests are one reason the American Cancer Society has argued that laboratory and animal

data provide insufficient evidence for carcinogenicity, that only human-epidemiological studies are sufficient to reject the null hypothesis.[8] A quarter-century ago, Irwin Bross (then-Director of Biostatistics at New York's Roswell Park Memorial Institute) said lack of knowledge about cancer has arisen partly because of misleading animal studies and scientists' not demanding human-epidemiological data.[9] He quoted Marvin Pollard, former American Cancer Society president, who claimed many cancer-research failures have arisen because of reliance on animal studies that are inapplicable to humans. Many courts likewise require EER in order to establish harm in toxic-torts, such as the Agent Orange, Bendectin, and other cases.[10]

On the other hand, EER proponents appear partly wrong in demanding human-epidemiological data before warning about (or regulating) apparent EIJ. Their adherence to EER raises at least 8 scientific and 6 ethical problems.[11]

SCIENTIFIC PROBLEMS WITH SUPPORTING EER

On the scientific side, those who support EER typically ignore at least two major problems that make human-epidemiological data almost always inferior to good animal data, namely (1) errors in gathering human-exposure data and (2) selection biases such as the healthy-worker survivor effect. EER proponents also often wrongly support human, over animal, tests because they (3) confuse the precision of exposure-disease relations with their strength; (4) reject classical accounts of scientific explanation; (5) erroneously privilege human-epidemiological data despite ignoring weight-of-evidence rules and committing fallacious appeals to ignorance; (6) demand infallible, rather than highly probable, scientific evidence and assume that merely probable evidence is no evidence; (7) ignore past inductive evidence for using animal data, rather than EER; and (8) ignore dominant scientific practices regarding using animal evidence for causal claims about humans. Consider each of these 8 scientific problems with EER.

EER proponents' *first* scientific problem is their overestimating difficulties with getting accurate animal-exposure data, yet underestimating difficulties with getting accurate human-exposure data. As compared to human data, good animal-exposure data have at least five important scientific merits. They usually result from (1) *intended or controlled exposures*; (2) *direct, large-sample observation* of exposures to thousands of subjects; and (3) *direct, long-term observation* of exposures that often capture effects over entire lifetimes or multiple generations.[12] Animal-exposure data also are typically from (4) *consistent or constant exposures* over time, and from (5) *empirically-confirmed exposures* obtained through frequent measurements of differences between target-exposures and actual delivered doses.[13]

Human-exposure data, by contrast, usually are less reliable because they typically arise from (1) *unintended exposures*. Because good human-epidemiological studies are difficult and expensive, human studies often rely instead on (2) *indirect (rather than controlled), small-sample observations* of scores to hundreds of subjects, and (3) *indirect, short-term observation* of exposures. Consequently most human studies are prone to confounding and bias; miss many legitimate effects,[14] e.g., being conducted too late to detect all effects, as with workers exposed to benzene;[15] less able (than animal-exposure studies) to take account of interindividual variability;[16] and likely to underestimate effects, given long latencies of many cancers (up to 40 years). Likewise, typical human data are from (4) *variable exposures* over time, and from (5) *merely estimated exposures* (often after-the-fact) from occasional measurements of subject exposures, or of others who are thought to have had similar exposures. For all five reasons, because human-exposure studies typically have less ability to control quality (than animal-exposure studies), in all the ways necessary to produce good science, human studies usually have greater exposure-related uncertainties. These uncertainties often lead to distortions in central estimates and potency estimates—distortions that require discussion/interpretation, and perhaps carefully adjusting estimation procedures for various slope factors and for "fitting" dose-response models. Although biostatisticians use various modeling techniques to compensate for these "errors in variables," how to compensate is frequently unclear because of the dearth of good quantitative analyses of likely errors in exposure-estimates.[17]

A *second* scientific error of EER proponents is their ignoring massive selection-biases in human studies. These biases are minimal/nonexistent in animal studies, yet can complicate mortality-data comparisons between human-study populations and the general population. Some of these selection-biases include "healthy-worker" and "healthy-worker-survivor" effects.[18] All other things being equal, the "healthy-worker effect" occurs because, despite their higher exposures to occupational hazards, workers nevertheless represent healthier segments of the population (as compared to children, the sick, or the elderly) and have lower mortality rates. Consequently, because human-epidemiological tests often are done in occupational settings, they usually underestimate health effects on the general population, especially effects on sensitive subpopulations like children.[19]

The "healthy-worker-survivor" effect occurs because, all other things being equal, those who survive various health threats tend to be healthier than the average population and overrepresented in longer-term pollution-exposure groups because they have lower mortality than those having shorter-term exposures to environmental-health threats. Consequently the healthy-worker survivor effect—adjustment for which is both very difficult and at the cutting edge of current epidemiology—typically produces distortions in relationships between measured cumulative exposure and measured risks—because shorter-term-exposure subjects suffer greater mortality than longer-term-exposure subjects.[20] Indeed, for diesel particulates, the relative-risk-versus-cumulative-dose curve even has a negative, rather than a positive, slope.[21]

Apart from the two preceding weaknesses in human-exposure data, other scientific problems arise because

EER proponents make several epistemological and logical errors in preferring human-epidemiological, to good animal, test results. A *third* error is erroneously confusing two different things: the *precision* with which animal-based relations between exposure and disease can be measured, and the *strength* of those relations. While imprecise animal data (with its 10-fold uncertainty in animal-to-human extrapolation) may motivate scientists to accept EER, imprecise animal data are nevertheless compatible with strong exposure-harm associations. Similarly, precise data on animal-human responses (theoretically at least) could reflect a weak exposure-harm association.[22] Yet the strength of these exposure-harm relations, not their precision, is more important to regulation and to preventing EIJ.

A *fourth* scientific weakness in the stance of EER proponents is their rejecting classical accounts of scientific explanation. According to these accounts, when scientists have experimentally established that a certain class of chemicals is of type X, they have explained something. They know that because chemicals X have certain structures and functions, they are likely to have particular properties.[23] Whenever they investigate a new chemical in this same class X, they do not assume they know nothing about it. Nor do they demand all new tests (analogous to demanding human tests) on this new chemical, before drawing conclusions about its potential harmfulness. Instead, they rely on their earlier experimentally-established explanation about the structure, functions, and effects of chemicals in this class—at least until those earlier explanations are proved wrong. EER proponents, who require human- epidemiological studies for causal inferences about harm/EIJ, are like those who require all new tests for some chemical—already known to be in class X. If EER proponents were right, science would be reduced to case-by-case "bean counting," not principled explanation.

A *fifth* scientific problem is EER proponents' discounting relevant evidence from good animal or laboratory tests, thus privileging only human-epidemiological data, and thereby rejecting weight-of-evidence rules for harm—rules dictating that the hypothesis supported by the most evidence (not necessarily human-epidemiological evidence) is the preferred hypothesis. Yet EER proponents ignore weight-of-evidence rules and instead demand satisfying EER as a precondition of showing EIJ. Yet if good animal tests suggest an agent causes disease, to demand epidemiological studies, before positing risk of harm, is to ignore existing evidence and privilege only another type of evidence.[24] Such a stance is implausible because airplane, auto, or space-shuttle investigators, for instance, make causal claims about harms, but without human-epidemiological data.[25] In the face of massive scientific evidence, (albeit not human-epidemiological evidence), privileging human-epidemiological data thus requires scientists to reject a weight-of-evidence rule for assessing toxins and potential EIJ.[26] If all existing, non-epidemiological evidence suggests some agent can cause disease, weight-of-evidence considerations at least create a presumption in favor of causality, a fact EER proponents forget. As a consequence, they often commit a fallacious appeal to ignorance. That is, they confuse the absence of human-epidemiological evidence for harm—with evidence for the absence of human-epidemiological harm.

A *sixth* problem of EER proponents is that they require near-infallible evidence (from human–epidemiological studies), yet ignore highly probable evidence (animal studies), and thus behave in unscientific ways. Science requires only probable evidence for positing harm because, apart from purely abstract logic, no science is infallible, given that it cannot overcome the problem of induction. Rather, science is what reasonable and careful people would agree to accept. In life-and-death cases, reasonable people do not demand only infallible or flawless evidence for harm (or only the most difficult-to-obtain evidence, as long-term human epidemiological studies are), before acting. Reasonable people don't wait until they see flames to call the Fire Department. They call when they smell smoke. Reasonable people don't go to the doctor only when they are ill. Instead they get annual check-ups. Reasonable people don't carry umbrellas only when it is raining. Instead they carry them even when it looks like rain. Reasonable people do not do nothing, merely because their evidence is not infallible. For all these reasons, reasonable people do not reject animal testing as inadequate for all public-health and EIJ-related decision-making.[27]

A *seventh* scientific problem is that EER proponents commit an inductive fallacy by tending to ignore previous scientific history that shows most agents, established as harmful to animals, also have been confirmed as harmful to humans. Human and animal tests are "highly correlated," as Princeton University risk assessor Adam Finkel notes.[28] He confirms that there are no legitimate scientific tests showing that rodent carcinogens are not also human carcinogens; he also points out that most human tests, used to deny human carcinogenicity, employ small samples (from which no reliable conclusions can be drawn), as with most industry tests of pesticides.[29] If anything, says Finkel (and as the first reason above shows), typical high-powered animal tests are more reliable than typical low-powered, human-epidemiological tests that often underestimate human-cancer risks because they are performed on less-sensitive, adolescent-to-late-middle-age animals, rather than on more-sensitive neonatal animals.

An *eighth* scientific problem with EER is that most reputable scientific programs do not follow it. Both the US National Toxicology Program and the International Agency for Research on Cancer (IARC) have classified many agents as possible or probable human carcinogens, even though there are no, or inadequate, human-epidemiological studies.[30]

ETHICAL PROBLEMS WITH SUPPORTING EER

On the ethical side, those who support EER err in at least 6 different ways. *First*, they demand data that often are unethical to obtain. Insofar as EER proponents demand human-epidemiological data on substances that are

already known to be risky or harmful, they ignore classical bioethics prohibitions against experimenting on humans in ways that are likely to harm them. For instance, because it is ethically/legally questionable to dose humans with pesticides so as to obtain epidemiological data,[31] these studies could/ought not be done. Consequently, using EER begs the question against following bioethics, against rejecting the null hypothesis, and against protective regulation. If EER were right, medical ethics would be reduced to a guinea-pig approach, doing nothing until dead bodies started to appear.[32] A *second* ethical problem is that EER proponents demand data that also are impractical and expensive to obtain, given the large sample sizes and long time-frames required for human-epidemiological studies. These exorbitantly high transaction costs for human-epidemiological studies help explain why less than one percent of all hazardous substances have been tested epidemiologically. Instead government relies mainly on controlled laboratory testing of animals. By rejecting this testing as inadequate, EER proponents use economics in a way that again begs the ethical question against regulation and against protection of EIJ victims.

A *third* ethical problem is that EER proponents ignore the classical ethics rule to protect the vulnerable. Instead they place the heaviest evidentiary and health burdens on the most vulnerable potential EIJ victims. When pollutant harm is controversial, there is no reason potential victims should bear the evidentiary burden, especially because they typically have fewer intellectual, financial, and political resources than other members of society. Moreover, EER proponents also unfairly assume that highly vulnerable potential EIJ victims must meet a scientific standard that polluters themselves never meet. Polluters have never funded (or at least have never made public) a multiple-decades-long epidemiological study, with thousands of human subjects, to assess full health effects of their products or pollutants. Because of both EIJ victims' vulnerability and the probable harm from pollutants, ethics requires protecting the vulnerable and thus having the "deep-pocket" polluters bear the heavier evidentiary burden.[33]

A fourth ethical problem is that EER proponents behave expediently when they reject the classical ethics default rule that, in the face of probable farm, one should take precautions and not ignore good animal or laboratory evidence for probable EIJ. Virtue ethics, in particular, recognizes precaution, benevolence, and care as necessary for being a moral person. It is neither benevolent nor virtuous to claim polluters can pollute, ignore good animal or laboratory evidence for harm, yet require satisfying EER as sufficient evidence for pollution harm or EIJ. This stance is like allowing hunters to shoot anywhere, at will, without reasonable assurance that no people are nearby. If hunters ought not ignore evidence for possible human risks (like movement in nearby brush), polluters ought not ignore evidence (like good animal or laboratory data) before seeking protection against EIJ.[34]

EER proponents also ignore a fifth ethical rule, to take responsibility for risks and harms caused by one's actions. In ignoring both ethical responsibility, and the fact that polluters are the main economic beneficiaries of pollution, EER proponents assume that polluters have no duty to ensure the safety of their activities and the safety of those they put at risk. Yet virtually all ethics holds that *rights* (to pollution-related economic *benefits)* presuppose corresponding *responsibilities* (for pollution-related *costs* imposed on innocent others, especially EIJ victims). This rights-responsibilities principle is fundamental to all accounts of human rights, from deontological (or duty-based) ethics, to contractarian ethics (based on contracts, promises, and treating people consistently). All law likewise is premised on equal treatment, equal rights, and corresponding equal responsibilities. That is why people have rights to their property, provided they use it in responsible, rather than harmful, ways. Their property rights end where other people's equal rights begin. Ignoring these responsibility principles, EER proponents forget that if good animal data suggest some pollutant threatens EIJ, polluters and scientists have ethical responsibilities either to accept these data (and reduce pollutants) or show why the animal data are wrong.[35]

A sixth ethical problem is that EER supporters risk harmful consequences when they wait to obtain human-epidemiological data, before taking action against potential pollution harms. Consequently they ignore the utilitarian ethical principle to minimize harm and to maximize desirable consequences. If animal or laboratory evidence shows some pollutant is probably harmful, this utilitarian principle requires this probability to be taken into account. Utilitarian ethics requires people to calculate the expected utility of their acts—the *magnitude* of harmful or beneficial consequences, times the *probability* that each of those consequences may occur. In demanding human studies but rejecting good animal and laboratory studies before regulating pollutants, Ames and others violate utilitarian ethics because they ignore the probabilities of human harm suggested by animal harm.[36]

CONCLUSION

Where do the preceding considerations leave us? In the absence of any compelling arguments to the contrary, the previous scientific and ethical considerations argue against EER. All other things being equal, EER ought not be used to deny either EIJ, or pollution-related health effects, or possible action to correct these apparent problems.

ENDNOTES

1. Critics of such strategies include Carl Cranor. *Toxic Torts* (Cambridge University Press, 2006), hereafter cited as TT; Paul and Anne Ehrlich. *Betrayal of Science and Reason* (Island Press, 1996); Thomas McGarity and Wendy Wagner. *Bending Science* (Harvard University Press, 2008); and Kristin Shrader-Frechette. *Taking Action, Saving Lives: Our Duties to Protect Public and Environmental Health* (Oxford University Press, 2007), hereafter cited as TASL.

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5. Ames and Gold 1989, p. 757; 1997. Elizabeth Whelan claims animal tests should be used to regulate chemicals more strictly only when two or more species exhibit "highly lethal" cancers that do not also occur spontaneously, that have short latency, and that arise at low doses. Whelan. Stop banning products at the drop of a rat. *Insight* 10 (December 12, 1994): 18–20.
6. Note 2 above, p. 51.
7. Note 2 above, p. 288.
8. S.S. Epstein. *Cancer-Gate* (Baywood Press, 2005), p. 83; M. Cohen. The Impact of medical censorship on patient care, *Townsend Letter for Doctors and Patients* (December 2004); available at <http://www.findarticles.com/p/articles/mi_m0ISW/is_257/ai_n7638036>; accessed July 16, 2006.
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10. Cranor, TT, pp. 224–225, 248–254.
11. Several of these scientific and ethical difficulties are discussed in Shrader-Frechette, TASL, ch. 3.
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13. See (but do not cite, quote, or take as policy) Stephen Roberts and US Environmental Protection Agency (EPA) Science Advisory Board (SAB) Ethylene Oxide Review Panel. *Draft Advisory Report* (US EPA SAB, 2007), pp. 36–37l available at <http://www.epa.gov/sab/pdf/ethylene_oxide_final_review_draft_report_8-30-07.pdf>; accessed July 31, 2008.
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15. S. R. Silver, R. A. Rinsky, S. P. Cooper, R. W. Hornung, and D. Lai. Effect of follow-up time on risk estimates. *American Journal of Industrial Medicine* 42 (2002): 481–489.
16. Dale Hattis and K. Silver. Human interindividual variability—a major source of uncertainty in assessing risks for non-cancer health effects. *Risk Analysis* 14 (1994): 421–431.
17. See note 13. Exceptions to the dearth of good quantitative analyses of exposure-estimate errors include, for instance, S.C. Brown, M.F. Schonbeck, D. McClure, A.E. Baron, W.C. Navidi, T. Byers, A.J. Ruttenber. Lung cancer and internal lung doses among plutonium workers at the Rocky Flats Plant: A case-control study. *American Journal of Epidemiology* 160 (2004): 163–172; D. Richardson, S. Wing, K. Steenland, W. McKelvey. Time-related aspects of the healthy worker survivor effect. *Annals of Epidemiology* 14 (2004): 633–9; and Dale Hattis, Illustration of a simple approach for approximately assessing the effect of measurement/estimation uncertainties for individual worker exposures on estimates of dose-response slopes, Appendix B, in note 13 reference above.
18. See, e.g., L. Stayner, K. Steenland, M. Dosemeci, I. Hertz-Picciotto. Attenuation of exposure-response curves in occupational cohort studies at high-exposure levels. *Scandinavian Journal of Worker and Environmental Health*. 29 (2003): 317–24; and K. Steenland, J. Deddens, A. Salvan, L. Stayner. Negative bias in exposure-response trends in occupational studies: Modeling the healthy worker survivor effect. *American Journal of Epidemiology* 143 (1996): 202–210.
19. See previous note.
20. See note 18.
21. See, e.g., E. Garshick, F. Laden, J.E. Hart, B. Rosner, T.J. Smith, D.W. Dockery, F.E. Speizer. Lung cancer in railroad workers exposed to diesel exhaust. *Environmental Health Perspectives* 112 (2004): 1539–43. See note 13.
22. K. J. Rothman. *Epidemiology* (Oxford University Press, 2002), p. 117.
23. National Research Council and Institute of Medicine. *Dietary Supplements* (National Academy Press, 2005), pp. 205–206.
24. S. Jasanoff. *Science at the Bar* (Harvard University Press, 1995), p. 125.
25. Cranor TT, p. 267.
26. D. Weed. Weight of evidence: a review of concept and methods. *Risk Analysis* 25 (2005): 1545–1557.
27. See Alan Gewirth. The rationality of reasonableness. *Synthese* 57 (1983): 225–47; Virginia Held. Rationality and reasonable cooperation. *Social Research* 44 (1977) 708–744; John Rawls, Kantian constructivism in moral theory. *Journal of Philosophy* 77 (1980): 515–572; David Richards, *A Theory of Reasons for Actions* (Clarendon Press, 1971).
28. See note 12.
29. See notes 12, 14.
30. D. P. Rall, M. D. Hogan, J. E. Huff, B. A. Schwetz, and R. W. Tennant. Alternatives to using human experience in assessing health risks. *Annual Review of Public Health* 8 (1987): 356; Cranor, TT, p. 250.
31. See note 14.
32. See Thomas Beauchamp and James Childress. *Principles of Biomedical Ethics* (Oxford University Press, 1989); Charles Culver and Bernard Gert. *Philosophy in Medicine* (Oxford University Press, 1982); Norman Daniels. *Just Health Care* (Oxford University Press, 1985); Robert Veatch, *A Theory of Medical Ethics* (Basic, 1981).
33. Regarding ethics and burdens of proof, see Kristin Shrader-Frechette. *Risk and Rationality* (University of California Press, 1991), pp. 100–145 (hereafter cited as RR); Carl Cranor. *Regulating Toxic Substances* (Oxford University Press, 1993).
34. Regarding virtue and protecting the vulnerable, see Philippa Foot. *Virtues and Vices and Other Essays in Moral Philosophy* (University of California Press, 1978); Peter

Geach. *The Virtues* (Cambridge University Press, 1977); Robert Kruschwitz and Robert Roberts (eds.). *The Virtues* (Wadsworth, 1987); Edmund Pincoffs. *Quandaries and Virtues* (University of Kansas Press, 1986); G.H. von Wright. *The Varieties of Goodness* (Routledge, 1963).

35. Regarding ethical responsibility, see Aristotle. *Nicomachean Ethics*, esp. books I–II; Joel Feinberg. *Doing and Deserving* (Princeton University Press, 1970); Jonathan Glover. *Responsibility* (Routledge, 1970); and Michael Zimmerman. *An Essay on Moral Responsibility* (Rowman and Littlefield, 1988). Regarding deontological and contractarian ethics, see Ernest Barker (ed.). *Social Contract* (Greenwood, 1980); James Buchanan and Gordon Tullock. *The Calculus of Consent* (University of Chicago Press, 1975); Ronald Dworkin. *Taking Rights Seriously* (Harvard University Press, 1977); John Locke. *Two Treatises of Government* (Cambridge University Press, 1977); John Rawls. *A Theory of Justice* (Harvard University Press, 1971); and John Simmons. *Moral Principle and Political Obligations* (Princeton University Press, 1979).
36. Regarding utilitarianism, see Shrader-Frechette, RR, pp. 100–130; Richard Brandt. *Ethical Theory* (Prentice-Hall, 1959), esp. chs. 12–19; John Stuart Mill. *Utilitarianism*, ed. J. M. Robson (University of Toronto Press, 1969); Samuel Scheffler (ed.). *Consequentialism and its Critics* (Oxford University Press, 1988); J.J.C. Smart. *Utilitarianism* (Cambridge University Press, 1973); Amartya Sen and Bernard Williams (eds.). *Utilitarianism and Beyond* (Cambridge University Press, 1982).

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