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ARTICLES

Facts vs. Passion: The Debate over Science-Based Regulation

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On May 4, 2018, Peter Wood, on behalf of the National Association of Scholars, submitted a letter to Scott Pruitt, administrator of the U.S. Environmental Protection Agency, in support of "strengthening transparency in regulatory science," new rules about the quality of science that would be an acceptable basis for EPA regulations. Wood emphasized NAS's recent report on the irreproducibility crisis in social and biomedical science and its implications for new and existing environmental regulations.[1]

On May 22, 2018, *Nature*, one of the two most prestigious general science journals, published an op-ed titled "Beware: Transparency Rule is a Trojan Horse."[2] The piece was very critical both of the EPA proposal and NAS's support of it.

Its author was historian Naomi Oreskes, a vigorous defender of environmental causes such as global warming, and one who, apparently, is not afraid to impugn the motives of those with whom she disagrees. In her *Nature* piece, for example, Oreskes refers to "climate-change deniers" who, she alleges, use a tactic that "exploits the idea of scientific uncertainty to imply there is no scientific consensus," as if scientific truth were to be decided by opinion poll. The point is

[1] *The Irreproducibility Crisis of Modern Science: Causes, Consequences, and the Road to Reform*, National Association of Scholars, New York, N.Y., April, 2018, https://www.nas.org/storage/app/media/Reports/Irreproducibility%20Crisis%20Report/NAS_irreproducibilityReport.pdf

[2] Naomi Oreskes, "Beware: transparency rule is a Trojan Horse," *Nature,* May 22, 2018, https://www.nature.com/articles/d41586-018-05207-9

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that if on any specific scientific subject there exists such a "consensus," it is often wrong, and Oreskes, an historian of science, should know this.[3]

Oreskes argues forcefully against the proposed rule, which simply requires that EPA regulations be based on transparent and replicable science. She judges this not as a welcome improvement in rigor, but as a sly attempt to weaken necessary restraints and put human health at risk. In Oreskes's view, the real crisis is not that published science is sometimes questionable, but that critics "attempt to discredit scientific findings that threaten powerful corporate interests."

In one paragraph that seems almost irrelevant to her main point, Oreskes takes aim at the NAS for its report *The Irreproducibility Crisis of Modern Science,* but offers no actual criticisms. She has seized an opportunity to belittle an organization she dislikes, perhaps because it has "an acronym easily confused with that of the prestigious National Academy of Sciences." "I will not call it the NAS!" she exclaims. But it's not really the acronym. It's because the NAS report "dwells frequently on" (i.e. is skeptical of) climate science. Oreskes's passion is unmistakable; her scientific judgement somewhat less so.

An unprovoked attack deserves a response. NAS (I use the acronym without embarrassment!) was permitted a short rebuttal.[4] But since Naomi Oreskes is influential enough to be published in *Nature*, it may be worthwhile to examine her *oeuvre* more closely, so as to better judge her qualifications as a critic.

In 2011 Oreskes and co-author Erik Conway published a book, *Merchants of Doubt* (MoD), which is a full-throated attack on critics of the science underlying a number of controversial environmental issues: acid rain, the ozone hole, global warming and—the topic on which I will expand—secondhand (environmental) tobacco smoke (ETS). Their treatment of ETS does little to validate Oreskes as a science critic.

Secondhand Smoke

Any issue related to health risks generates a huge controversial and tendentious literature of "research" (some real, some faux) and opinion. Smoke,

[3]Or perhaps just forgotten, since in 2007 she wrote "If the history of science teaches anything, it's humility. There are numerous historical examples where expert opinion turned out to be wrong . . . Moreover, in any scientific community, there are always some individuals who depart from generally accepted views, and occasionally they turn out to be right. At present, there is a scientific consensus on global warming, but how do we know it's not wrong?" Naomi Oreskes, "The Scientific Consensus on Climate Change: How Do We Know We're Not Wrong?" in Joseph F. DiMento, Pamela Doughman, *Climate Change* (Cambridge, MA: MIT Press, 2007).

[4]Peter Wood, "Justification for the EPA's transparency rule," *Nature,* July 11, 2018, https://www.nature.com/articles/d41586-018-05677-x

with its links to a large industry whose motives will always be suspect, is no exception. A review of manageable proportions must select what seem to the writer to be the key issues and studies. This is what I have tried to do.

Detecting a meaningful health effect of environmental tobacco smoke is bound to be very difficult. Any effect on mortality is likely to be modest (only a small fraction of the exposed population will be affected), and the effect will be delayed. If people are going to get sick or die after exposure to ETS it will only be after a lapse of time, and not of hours or days but years. Proving an effect of ETS is tough.

Experiment, the only way to establish causation beyond a reasonable doubt, is impossible, for both ethical and practical reasons: we cannot expose people to things that may make them sick, and even if we could, there will be few researchers eager to take on a project which offers an uncertain outcome and only after a delay of decades.

Nevertheless, Oreskes and Conway feel that a harmful effect of a low concentration of tobacco smoke is "common sense": "The U.S. Department of Health and Human Services tells us that 'there is no risk-free level of exposure to second-hand smoke: even small amounts . . . can be harmful to people's health." (quoted from the 2006 Surgeon General's report.) They, and (probably) a "consensus" of medical people, have no doubt "that secondhand smoke can kill."

But is it true in any meaningful sense? After all, everyone dies, with no intervention at all, and the effects of ETS are delayed, so what we are looking for is not immediate lethality, but some shortening of life.

Since experiments are ruled out, we are left with epidemiology as our only guide. Epidemiology has many limitations, the most obvious being that it can only detect correlations, which may be causal but may not. The great statistician R. A. Fisher famously quoted his colleague Udny Yule, who pointed out "that in the years in which a large number of apples were imported into Great Britain, there were also a large number of divorces. The correlation was large, statistically significant at a high level of significance, unmistakable. But no one, fortunately, drew the conclusion that the apples caused the divorces or that the divorces caused the apples to be imported." Yet "correlation is not causation" is a truism as often ignored as acknowledged.

Oreskes and Conway accept epidemiological data without demur. They begin their discussion with a confident conclusion: secondhand smoke can kill. But it is soon clear that their target is not so much tobacco as the tobacco companies: "just as the tobacco industry knew that smoking could cause cancer long before the rest of us did, they knew that secondhand

smoke could cause cancer, too." These statements refer to a RICO judgment which concluded that the tobacco companies were guilty of racketeering for claiming that ETS and low-tar-and-nicotine cigarettes have smaller toxic effects on human health than primary smoke from regular cigarettes.[5] As for the tobacco companies knowing about the dangers of cigarettes in advance of the public, that is surely nonsense: the definitive publication was the research by British epidemiologists Richard Doll and Bradford Hill in 1950, and even before that time cigarettes were called "coffin nails" and soon after "cancer sticks."

The politicization of the science related to ETS is quite extraordinary. As we will see, the anti-smoking side has simply ignored substantial data on the other side.

I begin with MoD's account. The weight of their claim that ETS is lethal is a 1981 Japanese study, by Tekeshi Hirayama. A 2011 summary of his work by Smith and Beh cites a 1979 paper by Enstrom (remember that name), which includes a comment on "a large relative increase in lung cancer cases in non-smokers in the United States that was observed between 1914 and 1965." Enstrom's observation apparently alerted Hirayama to the problem. Hirayama's study looked at the wives of smokers and non-smokers in Japan, hypothesizing that the wives of non-smokers, free of chronic exposure to ETS, would have lower lung cancer rates than the wives of smokers, long exposed to ETS.

Ideally, one should focus on mortality rates, because these are more reliable than, say, diagnoses of emphysema or cancer. Unfortunately, Hirayama looked not at death *per se*, but death from lung cancer. He studied a large population: "These cases occurred among 91,540 non-smoking married women whose husbands' smoking habits were studied," a sub-population selected from a total of 265,118 adults. (For some reason MoD refers to "540 women," suggesting that Oreskes and Conway misunderstood the study—which is easy to do because the paper is heavy on statistical tests and the raw data are hard to tease out. Indeed, the Hirayama study itself is an argument for the proposed new EPA rule.)

The aggregate figure for lung cancer deaths among the wives of smokers appears in the methods section. Apparently 245 married women in the sample died from lung cancer, 174 married to non-smokers died, so 71 non-smoking wives married to smokers died from lung cancer. Table 1 in the study shows more details: sorted by husband's age and the amount he smoked, from zero to

[5]For a fuller account of this sorry episode in U.S. law see John Staddon, *Unlucky Strike: Private Health and the Science, Law and Politics of Smoking* (Buckingham, UK: University of Buckingham Press, 2014).

1-19/day to > 20, there is a clear trend: the more the husband smoked, the greater the wife's cancer risk.

Statistical objections[6] were raised to this study, and the Smith and Beh (2011) paper admits that the aggregate data do not pass the chi-square test of significance. But, by parceling out the data by age and considering three variables (husband's age, cancer status, smoking frequency) rather than just two, these authors are able to get a highly significant result. (Why the husband's age rather than the wife's is critical is not explained.) Smith and Beh conclude that Hirayama was, after all, correct in claiming an increased cancer-death risk from secondhand smoke. As to the detailed assumptions underlying this now more complicated statistical model, little is said. Yet, as I have pointed out elsewhere, these assumptions are as important to the conclusions as the actual data, even in studies much simpler than Hirayama's. Nevertheless. Hirayama's results were, and are, generally accepted.

There is a 2003 study, not mentioned either by MoD or by Smith and Beh, that might have settled the issue, not by proving that ETS is harmless (which is impossible), but by showing that a large-scale attempt to find harm failed to do so. The study is by the aforementioned J.E. Enstrom and co-author G.C. Kabat in the *British Medical Journal*. (This reference appears in the citations for Chapter 7 of the 2006 Surgeon General's 709-page report but, curiously, is not discussed in the text. It has vanished from the 727-page 2010 Report.) The authors looked at 35,000 never-smokers in California with spouses with known smoking habits. The participants were selected from 118,000 adults enrolled in late 1959 in a cancer-prevention study. The numbers are large and the researchers asked a simple question: are Californians who are married to smokers likely to die sooner than those married to non-smokers? Their answer is unequivocal:

> No significant associations were found for current or former exposure to environmental tobacco smoke before or after adjusting for seven confounders and before or after excluding participants with preexisting disease. No significant associations were found during the shorter follow up periods of 1960-5, 1966-72, 1973-85, and 1973-98 . . . *The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect.* The association between

[6]Statistician Nathan Mantel concluded: "Much more careful analysis of the data would be needed before it can be claimed that a passive effect of smoking has been clearly established." https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1507117/pdf/bmjcred00679-0044b.pdf

> exposure to environmental tobacco smoke and coronary heart disease and lung cancer may be considerably weaker than generally believed. [emphasis added]

Enstrom and Kabat found no, or little, effect of secondhand smoke. The study received some criticism, but more about its sources of funding than scientific details. Yet this study—which seems to be relatively definitive—is completely ignored not just by the Surgeon General, but by Oreskes and Conway.

There are strong feelings on both sides of any smoking-related issue, although the anti-smokers seem to have the edge. Nevertheless it is hard to conclude from Enstrom and Kabat and other studies—on things like the "natural experiments" provided by before and after analyses of cancer rate following smoking bans—that the effect of ETS is anything other than trivial.[7] It is also hard not to conclude that there is considerable bias against anything that seems to minimize the dangers posed by smoking or concedes anything positive about the tobacco industry. Figures on the wrong side of the anti-smoking argument such as respected science writer Gina Kolata and scientists Fred Singer and Fred Seitz are routinely demonized. Research sponsored by the tobacco industry (e.g., Rodgman and Perfetti's book *The Chemical Components of Tobacco and Tobacco Smoke*, 2009 and related papers) is ignored.[8]

It is tough to give a balanced picture of the smoking landscape. But Oreskes and Conway don't even try.

No Threshold?

There is one more arrow in the anti-tobacco advocates' quiver: something called the "linear no-threshold" (LNT) assumption. The argument goes like this: there is little doubt that heavy cigarette smokers are at risk of lung cancer and related diseases like COPD (Chronic Obstructive Pulmonary Disease). We "know" that the effect of a toxin is strictly proportional to its concentration, with no lower bound, no threshold. Hence the lethality of even low doses of tobacco smoke is proved. "There is no risk-free level of exposure," as the Surgeon General said. Which opens the door wide, justifying restrictive regulation and confiscatory taxation on smoking and smokers. Pubs in England, and the social space they provide, have

[7] "Changes in U.S. Hospitalization and Mortality Rates Following Smoking Bans," NBER Working Paper, March 17, 2009, https://papers.ssrn.com/sol3/papers.cfm?abstract_id=1359506

[8] A. Rodgman, "Environmental tobacco smoke," *Regul Toxicol Pharmacol* 16, no. 3 (December 1992): 223-44.

been decimated by bans on all smoking in interior public areas, an unmeasured social downside. Many UK citizens now spend their time smoking and watching a screen at home, rather than discussing the issues of the day with their friends over a pint. This may please purveyors of media propaganda; it is hardly a benefit to democracy. The human costs of regulatory impositions do not figure in the calculations of Oreskes and Conway and other anti-smoking advocates.[9]

Is the LNT correct? Is there really no lower bound to the damage caused by toxins like ETS? Could LNT be true of all toxins, as its adherents claim? Or is it possible that each toxin has its own dose-response curve? The no-threshold assumption does after all go against the (admittedly self-serving) saw "the solution to pollution is dilution" and the old adage quoted in MoD "the dose makes the poison."

Well, yes, *this* does seem like common sense. Many substances, salt or alcohol, say, are toxic in high doses but harmless or even beneficial at low. Is ETS different? Even if true for some toxins, LNT surely cannot be true of all. And the human body—all bodies—is what Nassim Taleb has called "antifragile," they respond to stress by becoming stronger. The immune system, after all, requires some exposure to infectious agents to develop a resistance to them. Too much may be lethal, but no exposure at all can have harmful effects also.

An early EPA paper points out that if we understood the "mode of action" of a potential pollutant, the way in which it affects the body's biochemistry, there would be no doubt about its effect at any dose. [10] Unfortunately, even today, we rarely do. The way that ETS interacts with the body's physiology and biochemistry is still largely unknown.

There was much criticism of the no-threshold idea. The LNT assumption is false as a general truth. Research on radiation risk, where the idea originated, has come under increasing attack. Clear no-harm radiation thresholds have been found in animal and plant preparations. The checkered history of honest fact in

[9]They are happy to accept a 10 percent significance level, even though current thinking about irreproducibility suggests at least the 1 percent level is necessary (but probably not sufficient) to ensure replicability. "Think of it this way," they write, "If you were nine-tenths sure about a crossword puzzle answer, wouldn't you write it in?" There is no cost to making a mistake in a crossword puzzle. Public policy is different, but apparently not to the authors of MoD, where the costs of regulation simply do not warrant consideration.

[10]J. Michael Davis and William H. Farland, "Biological Effects of Low-level Exposures: A Perspective from U.S. EPA Scientists," *Environmental Health Perspectives* 106, Supplement 1, (February 1998).

"Mode of action" is an EPA term of art, "defined as a sequence of key events and processes, starting with interaction of an agent with a cell, proceeding through operational and anatomical changes, and resulting in cancer formation. A 'key event' is an empirically observable precursor step that is itself a necessary element of the mode of action or is a biologically based marker for such an element. Mode of action is contrasted with 'mechanism of action,' which implies a more detailed understanding and description of events, often at the molecular level." (*Guidelines*, footnote 2).

private letters and dishonest denial proclaimed in public is recounted in detail by E.J. Calabrese, who concludes that the no-threshold hypothesis is invalid for radiation risk.[11]

The bad effects of radiation are relatively rapid, and the use of non-human subjects allows for experiment. Comparable research on ETS with human subjects is prohibited by ethical constraints. In the absence of conclusive human data, the convenient LNT assumption has been applied by fiat to all potential carcinogens, including tobacco smoke where it is almost certainly wrong.

The LNT assumption has a sad history in which its inherent implausibility and weak empirical basis have given way before the regulatory imperatives of the precautionary principle. Despite the problems and criticism, the no-threshold assumption has become an EPA rule, at least as far as potential carcinogens are concerned. With the LNT as a super-weapon, there are few limits to the controls that may be imposed in the interests of "health and safety." Petrified by claims that there is "No Safe Level of Smoking: Even low-intensity smokers are at increased risk of earlier death," the populace wilts before regulations based not on a solid foundation but on a sea of scientific sand.

What to Do When the Science Is Uncertain

Controversies in this area typically arise at the margin. No one these days argues about the cancer risk to which heavy cigarette smokers are exposed. But when the science is ambiguous, when the health effects of a toxin, especially one like ETS that is experienced at low levels, when its effects have not, and perhaps cannot, be established with certainty, then the issue changes. It becomes a matter to be decided not by science, but by ethics and the political process. In partial recognition of this shift, the EPA years ago decided to treat cancer and non-cancer risks differently. In its *Risk Assessment for Toxic Air Pollutants: A Citizen's Guide* (1991), they say the following about cancer risks:

> In the absence of clear evidence to the contrary, EPA assumes that there are no exposures that have "zero risk"—even a very low exposure to a cancer-causing pollutant can increase the risk of cancer . . . EPA also assumes that the relationship between dose and response is a straight

[11]Edward J. Calabrese, "Societal Threats from Ideologically Driven Science," *Academic Questions* 30, no. 4 (Winter, 2017); Peter Bonilla, "James Enstrom vs. UCLA: Terminating Environmental Debate," *Academic Questions* 30, no. 4 (Winter, 2017).

> line—for each unit of increase in exposure (dose), there is an increase in cancer response.

Yet for non-cancer risks they say this:

> A dose may exist below the minimum health effect level for which no adverse effects occur. EPA typically assumes that at low doses the body's natural protective mechanisms repair any damage caused by the pollutant, so there is no ill effect at low doses . . . The dose-response relationship . . . varies with pollutant, individual sensitivity, and type of health effect.

I have been unable to find a scientific justification for this distinction between cancer and non-cancer risk. In 2005 the EPA published *Guidelines for Carcinogen Risk Assessment,* which should say something about the issue, but says only "the Agency's more current guidelines for these effects [i.e., thresholds] . . . however, do not use this assumption." The 166-page document reads like a "how-to" manual on scientific research. It attempts to anticipate every problem and confound for a wide range of toxins, tumor types and subject populations—an impossible task. Science doesn't work like that. In practice each problem must be tackled with an open-ended ingenuity that defies detailed advance specification. The attempt to do so leads, as *Guidelines for Carcinogen Risk Assessment* does, to a tree with endlessly proliferating branches—to confusion.

This approach almost guarantees omissions and even errors. The document says little about the problems caused by delayed effects, for example, and seems hesitant even about the meaning of causation: "A causal interpretation is strengthened when exposure is known to precede development of the disease." Just "strengthened"? Since when is a cause *following* its effect even a possibility?[12] These cancer guidelines say nothing about why the threshold issue is different for cancer and non-cancer risk. The distinction seems to derive simply from the special scariness of cancer.

Conclusion

The Oreskes *Nature* op-ed is an embarrassment. It is a testament to political passion rather than the legitimate scientific criticism it pretends to be. The fact that a scientific journal of the highest rank agreed to publish it shows how

[12]It is possible that what the report meant is "Causation cannot be claimed unless exposure to the toxin precedes development of the disease."

widespread that passion is and how deeply it has become embedded in the scientific establishment.

The EPA seems to have set itself an impossible task: to understand the science of toxicity while coming up with rules and regulations to control it. Many people seem to have difficulty separating facts from the emotion they generate. Perhaps, therefore, the scientific mission of the EPA should be severed from its regulatory function: actions separated from science, passion from fact. The proposed new guidelines may tighten up the factual basis for regulation, but the way the regulations are written and vetted should also be looked at afresh.