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How Public Health Policy Is Created: Scientific Process and Political Reality

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The central premise of this symposium, that data can drive public policy, is both laudatory and even vaguely plausible. The historical record, however, is not encouraging. Galileo (1564–1642) recanted his solar-centric views rather than face the Inquisition. The Hungarian physician Ignaz Semmelweiss (1818–1865) was persecuted for blaming puerperal sepsis on his medical colleagues' lack of hygiene; he died in an insane asylum.

By definition, health policy is made in the public arena. The process is, therefore, subject to a complex array of considerations and influences, only some of which, sometimes none of which, have anything to do with data or with the public's health. How else can it be explained that our ban on lead paint came over half a century after Australia's? The most recent demise of national tobacco legislation had everything to do with manipulative powers of the tobacco industry and nothing to do with the 400,000 Americans who die prematurely each year from smoking. President Clinton's decision in 1998 not to fund needle-exchange programs was admittedly made in the face of reliable data demonstrating such programs can dramatically reduce the incidence of human immunodeficiency virus infection without increasing the prevalence of intravenous drug abuse (1).

More subtle than policy made in the face of existing data are research funding priorities and processes that determine whether data will exist at all. In an ideal world, health policy would be formulated in a rational, linear process, moving from data collection, to interpretation, to scientific consensus. These are the areas for which the epidemiologist is most responsible. Translating science to policy is far messier and convoluted, involving as it does societal priorities, resource allocation, opportunity costs, changing cultural mores, special interests, politics, prejudice, and pure greed.

DATA AND EVIDENCE

Study design and conduct provide epidemiologists the greatest opportunity and freedom for having an impact on health policy. It's the area in which we possess unrivaled expertise and yields the substrate upon which subsequent formulations and discussions are based. It is, therefore, critical that we get it right. While our freedom of action may be limited by ethical, political, and financial constraints, it is our obligation to ensure we employ the most definitive study designs and collect the most impeccable data. Sheer quantity and quality of data are not sufficient. We must be insightful and imaginative in building a web of complementary evidence, collaborating where appropriate with colleagues from other disciplines.

In pursuing the simple question of whether oral could replace parenteral vitamin A in the treatment of severe xerophthalmia, our randomized trial ultimately demonstrated clinical equivalency (2). It did confirm that parenteral dosing resulted in higher serum vitamin A levels, but it also showed that this was irrelevant. The response of holo-retinol-binding protein, the physiologically relevant biochemical parameter, was identical in the two groups. It had taken only a single "expert committee" to recommend use of oral or parenteral vitamin A. It took 10 years, however, to rid the official World Health Organization recommendation of the physiologically problematic and less practical parenteral option. With no new trials or data being offered in the interim, and our study being considered definitive, it was, therefore, the basis for policy formulation. Clearly, the data did not "speak for itself." The delay resulted solely from physicians' preference for injections in urgent clinical situations.

INTERPRETATIONS AND CONCLUSIONS

Perhaps the greatest impediment to epidemiologic influence is the general debasement of our coin of the realm. Unsought associations discovered in large observational expeditions are given a prominence and significance they don't deserve. Authors cagily insert a caveat or two ("further study is required before…"), but the damage is done. Conclusions extrapolated from associations caught in the web of epidemiologic fishing expeditions reverberate up the health-conscious food chain, their potential importance and purported policy implications magnified at every stage. Fortunately, the public has caught on. Several years ago the *International Herald Tribune* reported on its front page that coffee was dangerous; and on its back page, that coffee was harmless. A plaintive editorial asked the equivalent of what is the poor coffee drinker to think? Or, as an op-ed piece in the *New York Times* headlined: "It's good. No, it's bad. No, it's good, really. I think" (3).

As long as we build webs of meticulously crafted evidence, carefully interpreted, leading to warranted conclusions, we will retain our public credibility and be spared the

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outpourings of misplaced self-doubt that recently blanketed *Science* and the lay press (4).

SCIENTIFIC CONSENSUS

In the absence of scientific consensus, data-driven political action is unlikely. This is precisely the reason the tobacco industry has funded studies and publications that have muddled rather than clarified our understanding, and why they have steadfastly maintained that nicotine is not addictive and that tobacco smoke is harmless.

Reaching consensus, however, is not always easy, particularly among epidemiologists. Part of the problem is our innate skepticism, but a large part is the probabilistic nature of epidemiologic evidence. We readily accept the fact that observational studies are inherently less conclusive than randomized trials, but to what degree? All else being equal, do 10 observational studies equal one controlled trial? Should controlled trials, wherever practical, be mandatory? How many, and of what kind, do we require for conclusive evidence? What are our "stopping rules?" A recent issue of *The Scientist* noted that 21 of 32 observational studies "found a statistically significant association between low dietary or serum beta carotene and increased risk of cancer" (5), before the National Cancer Institute launched three (why three and not five?) prevention trials (which failed to support causality).

These are important methodological issues with which we have not adequately grappled. They are also complex. Consensus "trip wires" are variable and idiosyncratic, and depend in part on the existing bed of scientific opinion. One observational and two large trials suggested that vitamin A prophylaxis of deficient children reduced their subsequent mortality before other groups seriously considered studying the relation (6–8). But within months of publishing our small, hospital-based trial of vitamin A treatment of children with severe measles (9), the World Health Organization and the United Nations Children's Fund officially recommended large dose vitamin A for the routine treatment of measles (10, 11). This single, small study (later confirmed by other studies) had appeared at a fertile time in the evolution of scientific opinion concerning the role of vitamin A in infectious morbidity and mortality.

EXTRAPOLATION

I've been addressing issues of association and causality from data often collected on small and highly selected populations. The epidemiologist plays a major role in extrapolating from the data and quantifying its impact. One needs to define the size of the population to which the conclusions are relevant, and quantify absolute or attributable risk, not just relative estimates of risk. The policy implications of a fourfold increase in risk of a rare cancer are clearly different than a doubling in the risk of a common one. This point is usually missed, particularly by the lay media. A recent exception is an excellent *New York Times* article by Jane Brody explaining the real risks of breast cancer (12).

Extrapolations have a number of interesting policy implications, well illustrated by the recent study reporting that lovastatin reduces the risk of primary coronary events among individuals with "average" cholesterol levels (13). The lay press repeatedly announced it reduced risk among individuals with "normal" cholesterol, when total cholesterol averaged 221 mg/dl and high-density lipoprotein averaged 36 mg/dl (decidedly "abnormal"). This distinction did not bother physicians either. As reported in the *New York Times*, Dr. Alan Garber commented that half of all cardiologists were already taking statins, while "a good fraction of them had normal cholesterol values…" (14).

The epidemiologist also plays an important role in quantifying the collateral impact of any proposed intervention. A recent study of women aged 40–69 years revealed that annual mammography had a 10-year cumulative false positive rate of nearly 50 percent; and that 19 percent of all women without breast cancer would, after 10 screenings, undergo biopsy (15).

POLICY FORMULATION

The epidemiologist plays only a small but nonetheless critical role in translating science to policy, which entails providing objective testimony to the underlying truths and assisting policy makers in estimating the costs (financial and human) and benefits (health) of alternative policies and interventions. To play a useful role, the epidemiologist must be willing to become engaged in the process. Standing by the sidelines pursuing other interests and expecting published data to "speak for itself" is leaving a good deal to chance, particularly as it may need to speak in different languages to different audiences (16).

I've related how our small hospital-based trial of vitamin A therapy for measles resulted in a change in official international health policy. A nearly identical study had been published 50 years earlier, in the same journal (17); yet it never changed clinical practice and was long forgotten. If we wish to have an impact on the public's health, we can do so only by following our epidemiologic leads, refining the evidence, and engaging in policy formulation, regardless of how messy and convoluted the process.

Like the relation between war and generals, health policy is too important to be left to epidemiologists. Or, as Einstein reminded us, "Not everything that can be counted, counts; not everything that counts can be counted." Health policies have an impact on a host of issues which rarely yield to neat regressions. These must be explored by experts in relevant disciplines and solutions reached through a delicate political process. Last year's *Pfiesteria* outbreak in the Chesapeake Bay resulted in large fish kills. Clinical and epidemiologic studies suggested that human (recreational and occupational) exposure could result in profound, though usually transient, memory loss (18). Available evidence suggests the outbreak resulted from nutrification of the Bay from agricultural runoff consisting primarily of chicken manure. Over 300 million chickens are raised on Maryland's Eastern Shore each year. Chicken manure, a by-product, is spread on already overly enriched land, from which it is washed and leached into the Bay (19).

To this edpdemiologist (and the environmentalists) on the Governor's Commission, the obvious solution was to reduce chicken farming. Discussions with legislators representing

the Eastern Shore made it clear there were not only drastic financial consequences to such a plan, but environmental concerns as well. Eastern Shore agriculture is based on corn, grown to feed locally raised chickens. Eliminating intensive chicken raising would eliminate farming, open the Eastern Shore to greater development, greatly increase human density, and exacerbate nutrification of the Bay! The final, negotiated settlement was a phased-in reduction in chicken-derived fertilizer, with the State subsidizing its alternative use and shipment elsewhere.

Of course, good policy can come from poor evidence. The "*Pfiesteria* Hysteria," as it was dubbed, allowed the Governor to enact legislation that will greatly enhance the health of the Bay, even if the actual level of human risk was marginal (20).

CONCLUSIONS

A policy-maker giving advice to an epidemiologist in the policy formulation process might include the following:

1. Don't be a wild-eyed advocate. Epidemiologists are most useful to policy-makers when viewed as balanced, objective, and credible.
2. Don't suggest the need for public health policy until the evidence is solid and you've already begun to assess the impact of alternative interventions.
3. If asked for advice, don't shy away from providing the best estimates that existing data will allow, even if the data are wanting.
4. Don't expect to have all the answers a policy requires. Those with other perspectives will have something valuable (or at least necessary) to contribute. Health policy involves far more issues than epidemiologists and other scientists know or care about.

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