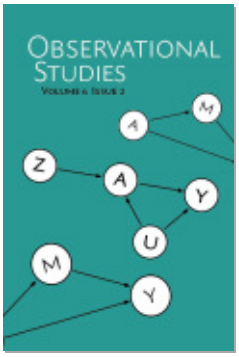
Causation in Action: Some Remarks Attendant to Re-reading Hill (1965)



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Causation in Action: Some Remarks Attendant to Re-reading Hill (1965)

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What a lovely paper! I use a number of the topics and points raised by Hill (1965) to reconsider aspects of the notion of causation in research in the social sciences and especially in sociology, the field with which I am most familiar. One could do more. The paper's intellectual concision and economy of expression are laudable. Each time I read it, I generate a new set of marginal comments.

I first read the paper many years ago. But why? The topical ambit is restricted: association or causation in epidemiology—occupational medicine in particular. It is very English in that it features chimney sweeps, although there is nothing Mary Poppins-y about them: These poor men were dying from scrotal cancer at a rate that was extraordinarily high relative to such deaths among other workers. This implicated as causes of their cancers the tars and oils characteristic of their trade (Hill 1965, p. 295).

I am a sociologist, not an epidemiologist; have not studied scrotal cancer or any of the other diseases and physical conditions discussed by Hill (1965); and until late in life had never been to England. I would only have known of the paper via Holland (1986, pp. 956-957), where it figures among the canonical disciplinary treatments of causation related to Rubin's (1974) model for causal inference. At that time I did not pick up much from it, because I was reading it with a whiggish cast of mind, as if it were evident that the diffuse treatments of causation in the past were noble-but-incomplete efforts on route to the precision of the present. Now I wonder, especially where the social sciences are concerned.

The mental discipline imposed by the potential outcomes framework (Rubin 2005) is very powerful. When I was first exposed to it (Holland 1986; Rosenbaum 1984), it was as though the scales fell from my eyes. I used this framework to first think (Smith 1990), then re-think (Smith 2013) all manner of studies in sociology, demography, criminology, and social epidemiology. Developments in causal thinking in the social sciences have been tremendous (e.g., Morgan 2013). But as I read Hill (1965) in retrospect, I think I see some threads of my own re-thinking of the situation, which is an admix of professional, scientific, and intellectual critique.

In brief, and without nuance: We have harnessed ourselves to a "game" in which the objective is to make a world of interconnected, purposive actors bound in historical time and changing social structures look something like a randomized experiment. This feeds into a reductionist, individualist view of social science—and of the world we live in. Causes become embodied in the subjects on whom we make measurements and do causal calculations. Researchers claim priority for the importance of causal analysis because of its

importance for action (in polite terms, "policy"), even if the action and active agents are at great remove from the assignment mechanisms (random or as-if-random) that constitute manipulation (action) in the experimental model. We find ourselves at the wrong level of analysis, justifying our claims about how the world works on the basis of a precision that is specious. It's not that we are stupid. It's the sociology of situation. The generalized scientific development of causal analysis for observational studies (because that's what most empirical social science is) feeds into a status hierarchy not just of ideas, but of individuals within the profession.

One or more versions of many of these points are elaborated in a more genteel and considered manner in Smith (2013). I stab further at a few here, drawing on topics suggested by Hill (1965), although I do not mean to implicate him anachronistically, which would be particularly unfair to someone who valued coherence (p. 298).

Those Lurking Confounds

Hill's (1965) first criterion with respect to causation is *strength* of an association. In addition to giving some trenchant examples of just how much damage certain environmental conditions induce in the humans who are exposed to them (pp. 295-296), he makes the important statistical point that for a strong observed association to be explained by some concomitant or antecedent factor, that factor must be very strongly associated in turn with the variable perhaps being mistaken for a cause. In particular:

> ...to explain the pronounced excess in cancer of the lung in any other environmental terms requires some feature of life so intimately linked with cigarette smoking and with the amount of smoking that such a feature should be easily detectable. (p. 296)

In sociology, it was once common practice to teach this kind of thinking with reference to cross-classified survey data (Rosenberg 1968). A large zero-order association was one with a large percentage difference, i.e., the percentage with some outcome characteristic under a potential treatment condition less the percentage with that same outcome characteristic conditional on an alternative (control). Before one spent a lot of time re-tabulating the data conditional on one (or two) control variables, it behooved one to create zero-order tables checking the association between a control variable and the original independent (or treatment) variable, and between the control variable and the dependent variable. If these zero-order percentage differences were not at least as large as the original effect (association) observed, then finding that that original association could be explained – in the sense of a substantial reduction in the association treatment/control (independent/dependent variable) conditional on re-categorization by the control variable – was just not going to happen.

This must have been the kind of knowledge people didn't really want to have, because the rise of high-speed computing and regression models with many variables led to a situation in which the vagueness of high-dimensional space gave license to all manner of seminar-room speculation and criticism of interpretations of strong observed associations as reflecting causal processes. Hope could spring, if not eternal, then at least more provisional than would be warranted given a tighter focus on elementary facts. With controlling and

partialling taking place through the calculation and inversion of ever-expanding variance-covariance matrices, it was easy to forget just how *big* an association a possible confounding or lurking variable needed to have to be the *real* explanation of an observed association. I thus sympathize with Hill's (1965) plaint:

> If we cannot detect it or reasonably infer a specific [confounding factor], then in such circumstances I think we are reasonably entitled to reject the vague contention of the armchair critic 'you can't prove it, there *may* be such a feature'. (p. 296)

Also, things improved: The statistical methods and vocabulary for addressing hidden bias in observational studies (Rosenbaum 1991) were a bracing antidote to "vague contention," since the establishment of bounds on estimated treatment effects entails a statement of just how strong selection into a treatment—how far a departure from random assignment, how "intimately linked" the confound and treatment– would need to be to gainsay the treatment effect as estimated conditional on observables and other aspects of study design.

These positive developments have been taken up by sociologists (e.g., DiPrete and Gangl 2004), but I would still like to quibble with the current state of scientific and scholarly affairs. Here I piggyback on Hill's (1965) perspicacious allusion to the idea that a strong confound "should be easily detectable" (p. 296). Our overriding concern with *hidden bias* in causal inference suggests to me (a) a level of social science so immature as to not yet have recognized the most powerful features of the explanatory environment; and/or (b) the human tendency to imagine that the forces that we cannot see are far larger than those that are in front of our eyes.

I suspect that there are non-sociologists and sociologists alike who would plump for the former characterization. I am not among them. Later I shall comment on some aspects of sociological explanation that run counter to the individual-level reductionism intrinsic to statistical and econometric causal analysis. Here I offer an example of the hoary sociological approach to causal analysis, in this case an investigation of whether a theoretically anticipated association is *suppressed* by *observable*, plausible confounds.

Davis (1982) presents findings "... [that] cast considerable doubt on the 'class culture' notion that occupational strata have vast and diffuse effects on the texture of our lives" (pp. 580-581). There is no evidence that an effect of occupation *cum* class culture on various dispositions is being *suppressed* by associations of occupation with race, age, and/or sex. *Pace* "'middle-class values,' 'the culture of poverty,' 'hard hat mentality,' 'working class authoritarianism'" (p. 582) and so on:

> ...[T]he association between race and [occupational] stratum, net of [e]ducation, is not all that strong... Since test variables must have stronger associations with X and Y than the associations they explain, race is not a promising [suppressor variable]. Sex, on the other hand, does have a healthy association with [o]ccupational stratum, net of [e]ducation..., but at the other end of the line, [s]ex is not, in general, a strong correlate of...attitude... Finally, we consider [a]ge. Very young workers do have lower prestige jobs... [and] there is a decent [association] for [a]ge and [o]ccupation net of [e]ducation, and numerous studies

> show younger people to be more 'liberal.' I suspect further analyses introducing [a]ge would allow more occupational effects to peep through. Nevertheless, [the partial association of age with occupation] is not a whopper vis-à-vis [other associations]. Consequently, age would have to show extraordinary effects on the [outcome] variables for it to suppress any but small effects of occupational stratum. Thus, I doubt that [a]ge has strong enough effects to change the broad picture. (pp. 582-583)

Davis (1982) concludes in a manner that is in line with Hill's (1965) thinking on confounding variables and causation:

> Similar mental exercises with other *obvious* test variables did not lead to any more promising ones... In sum, my conclusion is that occupational stratum simply does not have the diffuse and strong effects on our nonvocational attitudes and opinions that Sociologists have generally assumed. (p. 583; emphasis mine)

As for our penchant to credit the power of unseen forces for the acts of men: In the Western canon, we can date the imputation of reflexive action – of reasoning, of means-ends orientations consequent to thought and to choice – to the 5th century of the Athenian era (Romilly [1984] 1994). To be sure, prior to then people acted – Homer's heroes were nothing if not men of action – but this preceded the idea that a cause could and should attach itself to some purposive action, as in action after reflection. Before this, stuff just happens, as in the deterministic, inevitable cycles of revenge detailed by Herodotus (Romilly [1984] 1994, p. 177) or in consequence to exogenous shocks: storms, waves, and other accidental misfortunes (p. 34). But the most interesting causal attributions in the pre-psychological era were the forces that substituted for cognition in the minds of men. It was all about the gods, who hovered everywhere. They were primordial in Homer, for whom "action goes faster than reflection, brushing it away. So that, as necessary, a god can take it on himself to intervene and decide" (Romilly [1984] 1994, p. 34; my translation). In consequence

> divine causality often comes to remove much of the importance of human causation.... And above all, even when a man seems to be making a decision alone, one never knows if there isn't a god leading him along. Eschylus shows constantly this divine causation that doubles for, and puts the lie to, the free play of human motives. (pp. 36 and 65 [my translation])

Hill's (1965) skepticism regarding alternative explanations for the much higher rates of lung cancer among cigarette smokers than among non-smokers was restricted to "any other *environmental* terms" (p.296; emphasis mine). But what of non-environmental factors? Nobody would now claim that the gods of the ancients still exist, or that they meddle in the affairs of men. Fisher (1958, p. 163), the leading proponent for the view that the smoking-cancer relationship was spurious and not causal, instead emphasized genotype as the hidden variable ("common cause") behind the association between smoking and lung cancer. With a solicitude that would have been touching were it not so murderous, Fisher (1958) fulminated against telling people that something they were doing could be making them deadly ill, especially if their reasons for smoking were reasonable and, in the event, beyond their control. As with the gods, back in the day.

I'm kidding. Sort of. Genes exist. We can now observe them. They are correlated with all manner of things, many in the sociological realm (Bearman 2008). Let's set aside for the moment the fraught place of genetics in sociology and other social sciences—the difficulty in conversation between those for whom the science of the situation is so appealing as to make its desirability self-evident and those who smell yet another non-human-agency rationalization for social inequality. Are these the massive, powerful lurking variables, hitherto unobservable but now eminently so, that constitute the hidden bias in the estimation of causal effects in the social world? To date, the answer would seem to be "no," at least if we think about causation, especially in observational studies, with reference to a "fall from grace" relative to the experimental model. By this I mean genes that are non-randomly assigning folks to one social position or another while simultaneously determining some desirable social achievement, thereby creating the illusion that the social position is in some sense causing the social achievement. When really it is just the genes, doing their thing.

Instead, "genetic expression can only reveal itself through social structural change" (Bearman 2008, p. v). This remark derives from a collection of studies that find, *inter alia*, that all manner of gene-behavior associations are altered if not neutralized as social environments vary (e.g. school atmospherics [Guo, Tong, and Cai 2008], family life [Martin 2008], networks of social support [Pescosolido et al. 2008] , and national educational policies [Penner 2008]). The lurking variables, from a causative perspective, are less the gods and alleles—they are what they are—than all the social circumstances staring us in the face. The reference to *social structure*, as opposed to the socially meaningful attributes of individuals, may seem murky at this point. I address it further below. An overarching point is that our penchant for reductionism tends to steer us away from causation operating above the level of the individual (Smith 2013, pp. 65-69).

Causes with Many Effects

To help in adducing causation in observational studies, we can elaborate our theories to specify *unaffected units*, *essentially equivalent treatments*, and *unaffected responses* (Rosenbaum 1984, pp. 42-44). We gain confidence that something is a cause of something else if we find its effects where we anticipate them and not where we do not. A now common feature of econometrics, a *placebo* or *falsification* test (e.g., Rothstein 2010), is typically concerned with specifying unaffected responses.

Specificity was the third of Hill's (1965) criteria for establishing causation, although he was quick to add that "[w]e must not, however, overemphasize the importance of the characteristic" (p. 297; see, also, Holland 1986, pp. 956-957). Small wonder. The relationship between cigarette smoking and lung cancer is very strong (p. 296), but "the prospective investigations of smoking and cancer of the lung have been criticized for not showing specificity – in other words the death rate of smokers is higher than the death rate of non-smokers from many causes of death..." (p. 297). In the event, smoking is just flat-out bad for human health, even if its signature effect is on lung cancer. Hill (1965, p, 296) was quick to point out that "it does not follow...that [the] best measure of the effect upon mortality is also the best measure in relation to ætiology..." The case against smoking was best made with reference to differential mortality with respect to lung cancer, although the population prevalence of cardiac disease means that the weaker causal effects of smoking

are nonetheless associated with more excess deaths (e.g., Fenelon and Preston 2012). As Hill (1965, p. 296) noted,

> It does not, of course, follow that the differences revealed by ratios are of any practical importance. Maybe they are, maybe they are not; but that is another point altogether.

Analogies have their limits. Cigarettes and lung cancer may be an extreme causal relation. But the generalized noxiousness of smoking is not. In the social sciences there are several factors that are associated with all manner of outcomes. Education is the canonical example, and fortunately the effects of education are in general positive. Davis (1982) was combing the General Social Survey for predictors of morale (social life feelings), social attachment, political opinion, values and tastes, and stances on social issues. Again and again, education popped up as a strong predictor (if not a cause), even after netting out education's effect on occupational attainment, the indicator of the class cultures that Davis (1982) found wanting in explanatory power. Cutler and Lleras-Muney (2010) do a similar survey of the association between education and various health behaviors. Education is again an ever-present factor. It is hard to think of a domain of social life when it is not. When there is a factor whose effects are so ubiquitous, the precision of any one effect is of decreasing interest, in the sense that building an argument for action (or not) on the precise results in one domain may be a bit blind with respect to the sociology of the situation. Studies seeking to estimate *the* causal effect of education on this or that abound, and of course the estimated causal effects do not always look like the zero-order associations, or even the partial associations net of standard sets of observable antecedent variables. I don't mean to be a scientific Luddite: I am not arguing that we break our tools for determining what effects are and are not "causal." I do want to suggest that such results be taken in the larger context, where the larger context includes the mass of effects on all manner of outcomes. One counter-argument gets back to the gods and the genes: That once we uncover the hidden factors that are determining both education and everything else we do, we'll feel silly for having imagined that any of this was within our control—in the provision of education, for example.

Against this, I would argue that many social causative factors—especially those with many effects—are operating at a level above the inter-individual variability that tends to dominate our sense of what is causal and what is not. Education is in one sense embedded in the social structure; but changes in education—especially the stock of education—are also changing the social structure. Mass education and fertility is a good example. Education has long been associated with declines in fertility. But how exactly? Caldwell's (1982) theory of fertility and fertility decline brings into focus the *social structural* character of the factors first supporting high fertility, then precipitating its decline (Smith 1989, pp. 172-173):

> ...Patriarchy is a social institution subsuming large numbers of women within families, families within kin groups, and kin groups within communes or villages. The subordination of youth to their elders and of women to men is not a feature of particular households, families, or kin groups, but of the larger social structure. Individual variation (deviance) is of little account when arrayed

> against the larger forces militating for conformity to essential behaviors, including fertility (Caldwell, 1982:172). When change comes, it comes not through the collective exercise of individual choice, but through the collapse of a larger system that had heretofore constrained all choices of behavior open to individuals. Theories of modernization that concentrate on personality change are criticized, due to their implication that "individuals could always have lived different ways of life by opting to do so, whether or not the needed economic and social institutions for the new way of life had yet come into existence" (Caldwell, 1982:280). A primary source of fertility change is mass education. Mass education creates both educated (expensive, ungrateful, questioning) children and educated wives. Educated wives reduce the net wealth flow from wife to husband (and mother-in-law and father-in-law), strengthen the bonds between husband and wife (undermining the traditional family structure and its morality), and seek to avoid repeated pregnancies and periods with infants. Education is easily measured at the individual level, and its incorporation into micro models of fertility and fertility-related behaviors is on occasion justified with reference to Caldwell's (1982) emphasis on education as a source of fertility decline. But Caldwell unambiguously points to the macro properties of education: "[T]he education of only half the community does not have the same effect on that half of the population, nor half the effect on the whole population" (1982:329). When there remain many in a community who have not attended school, strong forces maintaining the traditional family morality still abound. "[T]he evidence suggests that the most potent force for change is the breadth of education (the proportion of the community receiving some schooling) rather than the depth (the average duration of schooling among those who have attended school)."

Causation and Action

If you are reading about John Snow and cholera, you are probably not studying cholera. More likely, you are being instructed regarding what causal inference in good observational science looks like (e.g., Fisher 1958, pp. 156-157; Freedman 1991, pp. 294-299). Ever willing to ape my betters, I have adduced Snow's admirable studies of cholera in support of a point that strikes me as basic but nevertheless goes against the grain of contemporary research habits: If you have a nice estimate of an effect based on random assignment or credible as-if-by-random assignment (Snow's case [Smith 2013, p. 50]), do you really need to understand the intervening process to have established *causation*? I think not (Smith 2013, pp. 60-63), and am buoyed in seeing that Hill (1965, p. 298) had also used Snow as an example in getting to the same point (albeit half a century earlier):

> Before deducing 'causation' and taking action we shall not invariably have to sit around awaiting the results of that research. The whole chain may have to be unraveled or a few links may suffice. It will depend upon circumstances. (p. 295)

Moreover, the social circumstances linking social conditions to specific outcomes, including health outcomes can be intransigent, in a way that is not well captured by their

elaboration in terms of individual-level intervening health behaviors and biological characteristics. "[F]undamental causes can defy efforts to eliminate their effects when attempts to do so focus solely on the mechanisms that happen to link them to a disease in a particular situation" (Link and Phelan 1995, p. 81). This is reflected in the enduring individual-level association between socioeconomic status, including education, and health. How does this happen?

> ...[T]he association between a fundamental cause can be preserved through changes either in the mechanisms or *in the outcomes*...[S]ome causes..."basic causes," have enduring effects on a dependent variable because, when the effect of one mechanism declines, the effect of another emerges or becomes more prominent. (Link and Phelan 1995, p. 87; my emphasis)

Why does this happen? Social structures are about nothing if not the differential allocation of scarce resources, both for their intrinsic value and for the maintenance of status hierarchies that will allow for differential allocation in the future, as new outcomes of interest arise. In the case of social status and health,

> ...[T]he essential feature of fundamental social causes...is that they involve access to resources that can be used to avoid risks or to minimize the consequences of disease once it occurs...[R]esources...include money, knowledge, power, prestige, and the kinds of interpersonal resources embodied in the concepts of social support and social networks. Variables like SES, social networks, and stigmatization are used...to directly assess these resources and are therefore especially obvious as potential fundamental causes. However, other variables...such as race/ethnicity and gender...are so closely tied to resources like money, power, prestige, and/or social connectedness that they should be considered as potential fundamental causes of disease as well (Link and Phelan 1995, p. 87)

Because the social structures that we create have an internal logic that transcends the temporal correlations that they create with so-called intervening variables, action oriented toward those intervening variables alone may not have the anticipated outcomes.

Knowing more is better, and elaborating the process leading from some factor to an outcome of interest is a hallmark of science. But it is not necessarily a hallmark of causation, especially inasmuch as causation is understood as what would or will happen if one were actually to *do* something (e.g. Hill 1965, p. 300). The distinction has been made by Holland (2008, p. 99):

> One of the problems of communication between social scientists and policy makers is related to the distinction I make between assessing effects and describing mechanisms. Understanding some aspect of a causal mechanism often advances science (i.e., theory), whereas the needs of public policy often require an answer that assesses the effects of an intervention, rather than reasons or speculations as to how these effects come about. If class size reduction results in better student learning, a policy maker might argue that it does not matter if this effect is due to more time for individualized instruction, fewer classroom disruptions,

> or something else. On the other hand, the mechanism might matter to the policy maker if other reform policies besides class size reduction are of interest. Knowledge of the causal mechanism could indicate that other policies would be supportive or possibly contraindicated when classes are small. My view is that both positions need to be clearly delineated and not confused with each other.

This confusion of purpose stares at us on the first pages of one of the best texts conveying the modern armamentarium for causal analysis within economics:

> A causal relationship is useful for making predictions about the consequences of changing circumstances or policies; it tells us what would happen in alternative (or "counterfactual") worlds. For example, as part of a research agenda investigating human productivity capacity—what labor economists call human capital—we have both investigated the causal effect of schooling on wages.... The causal effect of schooling on wages is the increment to wages an individual would receive if he or she got more schooling. A range of studies suggest the causal effect of a college degree is about 40 percent higher wages on average, quite a payoff. The causal effect of schooling on wages is useful for predicting the earnings consequences of, say, changing the costs of attending college, or strengthening compulsory attendance laws. (Angrist and Pischke 2009, pp. 3-4)

I am not contending that estimates of the "the causal effect of schooling on wages" are *useless* "for predicting the earnings consequences of, say, changing the costs of attending college." They are definitely useful, and it is possible to integrate formally the two perspectives. Todd and Wolpin (2006) is an admirable example, based on some serious theory (Wolpin 2013). On the ground, I think that we are far closer to the confusion that Holland (2008, p. 99) describes. In seminar rooms, researchers present and debate the fine points of *soi-disant* causal analysis. With their authority duly established by dint of having wrangled some micro-level observational data into the as-if-by-random-assignment computational frame, they soon weigh in on actions, which are virtually always policy prescriptions for altering choice sets, not the micro factors involved in the preceding causal estimation demonstration.

The focus on establishing "causation" as a form of legitimation (Holland 2008, p. 101) without keeping the action orientation in mind can lead us to strange places. For example: In the United States, lists of eligible voters are publicly available, so that one can observe who actually voted in a given election (if not for whom they voted, individually). In 2004 a team of political scientists obtained files of the electoral rolls in Illinois—more than seven million names—including information on their addresses, telephone numbers, and demographic characteristics including their sex and age, along with their histories of electoral participation (Arceneaux, Gerber, and Green 2010). They eliminated very large households and those without telephone numbers (talk about a bygone era!) This led to a file of 2.7 million households with at least one eligible voter (and less than five). They then took a random sample of 16,000 potential electors (only one per household) and tried to reach them by telephone to encourage them to vote in an upcoming election. Only 41% of the potential voters sampled could be reached by phone, and the researchers were concerned that the sort of folks who still pick up a telephone no matter who is calling are also the most likely to vote following a get-out-to-vote call.

They thus sought to estimate a treatment-on-treated effect, "the causal effect of a phone call among those who are reachable" (Arceneaux et al. 2010, p. 260). They were well aware that a control group—even one carefully selected from among the possible voters who were not called but who might be matched on observables (age, voting history, etc.) to those who were reached and hence had been encouraged to vote—would be mixing up the polite folks who feel obliged to pick up the phone when it rings with those who don't trust unsolicited phone calls, are rarely at home (remember: a bygone era!), and/or who didn't specify a home phone number. Therefore, to evaluate the effects of a get-out-the-vote phone call on those who did in fact receive one (they picked up the phone, they got the message), Arceneaux et al. (2010) did an analysis via two-stage least-squares, where the random selection for possible contact served as the instrumental variable in a regression of subsequent vote (went to the polls or did not) on whether contact was actually made (yes or no). It turns out that (a) the probability of going to vote increased by 2% as a result of receiving (implying answering) an encouragement-to-vote phone call; and as suspected, (b) the people who tended to answer such a phone call are also the kind of people who go out to vote, whether they are called or not.

Arceneaux et al. (2010) were interested in demonstrating the insufficiency of a different potential method—matching on observables, as described above—for estimating their preferred parameter, the effect of the treatment on the treated. Agreed: Matching doubtless does not control for an important unobservable, the tendency to pick up the telephone when it rings. In comparison, the two-stage estimator gives an unbiased estimate of the effect of calling among those who pick up the phone. In that sense it is a preferable causal estimator. But suppose you are a party worker or other campaign operative who is interested in finding more votes: The effect of a phone call on those who answer the phone is not your primary interest. You want to know what is going to happen when you inundate the registered voting population with telephone calls that will, in the main, go unanswered. The effect touted and precisely measured by Arceneaux et al. (2010) does not accord well with the intervention that one could imagine making. In which case I think the tendency to put causal estimation first and action not first is problematic, at least in the action-orientation sense of causation "What would happen if...?"

Social Structure as a Cause

A social structure is a relational system that exists not just in function of the individuals present in a society and their characteristics (psychological, genetic, social and otherwise), but of a set of social roles that tend to persist (or change) independent to great degree of the characteristics of the incumbents of roles. It includes power relations, norms, and habits of mind that weigh on opportunities and behavior.

This can sound imprecise and unscientific, certainly relative to measures of observables on individuals, such as alleles, levels of education, criminal records, etc. – maybe even people's sex and race – that figure in most statistics-based treatments of cause and effect. For example,

> At the class certification hearing in federal district court, Plaintiffs' sociological expert witness testified regarding his "social framework analysis" of Wal-Mart's "culture" and personnel practices, and concluded that the company was "vul-

> nerable" to gender discrimination. The reasoning here was from the general – that of Wal-Mart's "strong corporate culture" – to the specific – that Wal-Mart discriminated against its women employees as a consequence... (Dawid, Faigman, and Fienberg 2014, p. 380).

The many terms placed inside quotation marks speaks volumes: Dawid et al. (2014) were not fans of this form of analysis (nor was the court).

I cannot speak for the legal issues in general, nor about this case in particular, but I would not be so quick to dismiss the validity of the concepts—that is, their reality as depictions of essential aspects (yes, in the causative sense) of the organization in which these women were working. Imagine we were to describe the sort of gender system within which we are living, I as the writer, you as the readers. Unless the readership of *Observational Studies* is substantially different from what I imagine it to be, the rules of engagement, the expectations, the opportunities, the behaviors, and the relationships (in every sense of the word), not just between men and women, but between women and other women, men and other men—not to mention the creation of gender identities not bound to these binary criteria—well, I suspect that they would preclude if not sanction placing in our papers a homily of the form:

> If your wife ran off with the lodger last week you still have to take your perforated ulcer to hospital without delay. But with a hernia you might prefer to stay home for a while – to mourn (or celebrate) the event. (Hill 1965, p. 296)

By which I do not mean to impugn Sir Austin Bradford Hill. He, like most of us, was a man of his times with respect to many things, in addition to being farsighted intellectually in the ways we celebrate in revisiting his work.

I do, however, mean to illustrate why something along the lines of a "strong [XXXX] culture" might look like, what it might feel like, and how it might be perpetuated, even (especially) by those who mean no harm. The modern statistical and philosophical literatures on causation have long been stuck on how best to capture the "effects" of race and sex (or gender) – what sociologists call the *ascriptive* aspects of individuals. (Education and income, in contrast, are statuses that one putatively *achieves*). I would have thought that scholars would have moved past the idea that racial characteristics ascribed to individuals were in any sense a cause of what might or might not be happening to them (Smith 2003, p. 465), but I would have been wrong (cf. Marcellesi 2013). Yet even reframing the problem so that the subjects who are causing things are the units across whom race inputs are being measured (e.g. Pager 2003; Greiner and Rubin 2011) does not get at the social structure that is in a real sense causing the situation:

> [A]fter a society becomes racialized, racialization develops a life of its own. Although it interacts with class and gender structurations in the social system, it becomes an organizing principle of social relations in itself...Race, as most analysts suggest, is a social construct, but that construct, like class and gender, has independent effects in social life. After racial stratification is established, race becomes an independent criterion for vertical hierarchy in society. Therefore different races experience positions of subordination and superordination in society and develop different interests. (Bonilla-Silva 1997, p. 475)

The idea of racism without racists (Bonilla-Silva 2013) undercuts the continual focus on race as a causal property of individuals, because what one is dealing with are the pervasive, interrelated effects of a social structure with any number of taken-for-granted roles and social relations. Perhaps in another social world, the set of experiences would be very different:

> If race is not a causal variable, how do we analyze issues of social discrimination in causal terms, if at all? We certainly do think of racial discrimination in causal terms because many of us think racial discrimination is something that could be changed, reduced, or in some way altered. There are those who dream of a day when racial discrimination is a thing of the past and long forgotten. What is it that has to change? Certainly not the color of people's skin or some other physical characteristic. Clearly discrimination is a social phenomenon, one that is learned; it is taught and fostered by a social system in which it plays a complex part. When we envision a world without racial discrimination we thus envision it as a whole social system that must be different in a variety of ways from what we see before us. One almost has to envision a parallel world, so to speak, in which things are so different that what we recognize in our own world as racial discrimination does not exist in this other parallel world... (Holland 2008, p. 102)

Coda

I may be straying into dogmatism, which would be unfortunate, because this is precisely the point: Our interest in causation could use far less dogmatism, far less sense that there are some principles that are intrinsically more important than others in understanding one aspect of how the world works: i.e., what might we expect to happen if we did something or another? I read Hill (1965) as a reminder of what that sensibility might look like.

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