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Effects of Causes and Causes of Effects: Some Remarks From the Sociological Side

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Abstract

Sociology is pluralist in subject matter, theory, and method, and thus a good place to entertain ideas about causation associated with their use under the law. I focus on two themes of their article: (1) the legal lens on causation that “considers populations in order to make statements about individuals” and (2) the importance of distinguishing between effects of causes and causes of effects.

Keywords

causes; effects; potential outcomes; causal inference; probability of cause

Sociology is rarely practiced clinically, so the idea of inference from populations to individuals seems foreign. American sociology is, historically, nomothetic (Kim 1997), concerned with generalizations at the aggregate or population level. Individual deviation from group central tendencies is expected, tolerated, and attributed (if only tacitly) to some combination of measurement error, theoretical imprecision, resolution of scale, heterogeneity, and/or stochastic variability. There is also much idiographic research in sociology that emphasizes the context, particularities, and contingent nature of specific subjects or events, including causal interpretations of outcomes that are *sui generis*. However, the practice of both nomothetic *and* idiographic sociology comports with Fienberg, Faigman, and Dawid’s (In press:2) observation that “science [sociology] studies individuals in order to make statements about populations.” Even researchers in the idiographic tradition, including ethnographers, are inclined to generalize their observations to larger populations (e.g., Anderson 1990). Some practitioners of case study analysis within sociology make explicit their presumed (nonsampling) warrant (Ragin and Becker 1992; cf. Lieberman 1992) and offer a deterministic account, drawn from “small *n*” settings, of causal inference (Ragin 1987; cf. Sobel 1995:9-10).

Fienberg, Faigman, and Dawid (In press:11-16) outline the legal logic of “differential etiology,” or the practice of pronouncing on specific causes in specific cases given causal

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relations adduced at a more general level, and suggest what might be required to make this legal precept accord with scientific practice. Griffin (1993) gives a sociological account of the lynching of a man, in 1930, in the Mississippi Delta. Event structure analysis (Heise 1988, 1989) is used to query a narrative version of events—to make explicit events that are causally related (in the sense of being essential and required, and to admitting to a counterfactual nonevent)—and to link the sociologically informed rendering of this lynching to what is known about lynchings in general (“How can comparative knowledge about other lynchings be employed in the analysis of this particular lynching?. . . . How can the historical and structural *contexts* of the lynching be used to account for the *actions* in the lynching? [Griffin 1993:1096 (emphasis in original)]”). Whether event structure analysis could furnish the logical framework for integrating general scientific knowledge with attribution of cause in a discrete set of events, *in a legal setting*, is beyond my ken, although the parallels are evident. The problem is that “[e]vent-structure analysis is time consuming, highlights ignorance, and is humbling. It offers no ready answers to the questions it poses or to the difficulties induced by the tension between the general and particular. What it does do is make users extraordinarily self-conscious about what we know in general and particular . . . ” (Griffin 1993:1128). Humility and self-awareness are praiseworthy in intellectual work but may be less valued in court.

Holland’s (1986) canonical treatment of statistics and causation begins “[t]he emphasis here will be on measuring the effects of causes because this seems to be a place where statistics, which is concerned with measurement, has contributions to make” (p. 945) and concludes “that looking for causes of effects is a worthwhile scientific endeavor, but it is not the proper perspective in a theoretical analysis of causation” (p. 959). Dawid (2000:408) introduces as exemplars, the “headache questions” (“I have a headache. Will it help if I take aspirin?” “My headache is gone. Is it because I took aspirin?”) that feature in Fienberg, Faigman, and Dawid (In press), along with their correspondence to, respectively, “problems of *general* and *singular* causation.” The distinction between effects of causes (EoC) and causes of effects (CoE) is important conceptually and oft overlooked by social scientists. In practice, the distinction is one of degree rather than of kind (Smith 2013a:53-56). Fienberg, Faigman, and Dawid’s (In press) focus on *probability of causation* (PC) is helpful in identifying when the problems are similar and when they are not. The application of Fienberg, Faigman, and Dawid’s (In press) perspective to sociological research can be encouraged in downplaying the connection between CoE and inference to individuals that dominates in legal settings.

Figure 1 locates these points conceptually. The scale of observation, or number of units, is the left vertical axis. The same scale also appears on the right, but now refers to the scale of inference. Most studies in sociology, regardless of where they are situated on the observational scale, are attempting to push their findings, on the inferential scale, toward the base of the picture. In contrast, in courtroom settings, the problem is moving from a general observational scale, as per a randomized experiment or statistical manipulation of data from an observational study, to an inference about a specific case (Fienberg, Faigman, and Dawid In press). This is indicated in Figure 1 by the dotted arrow moving upward from the question regarding the effects of taking aspirin to the question regarding whether the cessation of a headache can be attributed to the aspirin that one took.

The horizontal dimension in Figure 1 reflects the CoE/EoC distinction. Sometimes the two issues are quite distinct from one another in a way that is mainly a function of epistemology, or warrant for inference. Figure 1 is “warmest” where inference is most secure, as per studies with appropriate coverage and low sampling variability (i.e., population studies on the observational scale) plus close adhesion to the experimental model, the kernel of the potential outcomes framework for the estimation of the effects of a cause (Rubin 1974; Rosenbaum 1984). Thus, one researcher interested in divorce in the United States studies the effect on divorce of the adoption of nofault provisions for marital dissolution. This is an EoC question; the problem can be framed in terms of alternative exposures to treatments and a comparison of differential outcomes. A second researcher asks why divorce increased in the United States in the second half of the twentieth century. Changes in state laws may (Rodgers, Nakonezny, and Shull 1997, 1999) or may not (Glenn 1997, 1999) have had something to do with this, but so might a host of other factors (Cherlin 2004). This is a CoE problem, as it typically appears in sociology where causes are many (Marini and Singer 1988:354-56; Blalock 1991:330) and types of causes (Freese and Kevern 2013) need to be delimited outside of the inferential frame. Reduction of unexplained variance is a typical criterion for adding a factor to the list of causes. This can make sense for articulating mechanisms and/or giving compositional accounts of a distribution of an outcome (Smith 2013a:56) but is largely incoherent otherwise (Sobel 2000:250).

“In the social sciences our interest focuses on scientific theories pertaining to classes of events of things. We are therefore usually interested in the identification of what we might call a causal structure, as reflected in the disjunctive plurality of causes that may produce an effect” (Marini and Singer 1988:356). Autism is increasing in many populations for many possible reasons (Jick and Kaye 2003), although vaccinations containing Thimerosal do not appear to be among them (Fienberg, Faigman, and Dawid In press:28). Elsewhere (Smith 2013a:54-56), I outline a suite of demographic studies accounting for changes in prevalence of autism in California in function of, inter alia, age of parents at the birth of a child (King et al. 2009), social networks (Liu, King, and Bearman 2010), and changes in immigration laws (Fountain and Bearman 2011). These are classed in Figure 1 as strong population-based studies adumbrating the CoE in the demographic or sociological sense of accounts of the distribution of observed outcomes. Immigration laws may need changing and people could be advised to have children earlier, but the causal effects of these possible manipulations are not the focus of the studies. In contrast, a good observational case-control study of the link between childhood vaccinations and autism is informative regarding the effect of a cause. If there were a causal link, vaccinations might be abandoned or reengineered. But given the “plurality of causes” at work in the changing prevalence of autism, it is doubtful that this link alone would suffice for understanding the causes of the epidemic.

There are occasions, however, when establishing the effect of a cause is tantamount to establishing the cause of an effect—a “twofer” (Smith 2013a:53). The complicated situation with respect to autism can be contrasted with Snow’s classic work on the epidemiology of cholera (e.g., Freedman 1991:293-300), where an ingenious natural experiment implicated drinking water fouled by human waste as the fundamental cause. Why is unsanitary drinking water (and a bacillus) a fundamental cause of cholera and parental age not a fundamental cause of autism, notwithstanding good side evidence on a genetic mechanism in the latter

case (Liu, Zerubavel, and Bearman 2010)? Having an older parent raise the prospects of a de novo mutation, but older parents rarely give rise to the de novo mutations implicated in autism, and autism exists even without mutations or older parents. Not everyone who drinks unsanitary water gets cholera, but—to a close approximation—avoiding unsanitary water (and food washed in same) made it highly unlikely that one would contract cholera. This is related to Fienberg, Faigman, and Dawid's (In press) PC, which is, in so many words, the extent to which a “positive” outcome can be attributed to a specific treatment.

In a double-blind randomized controlled trial, headache sufferers are offered a pill containing either chalk or aspirin. Those who take the aspirin are over 3 times more likely to report recovery. This is clearly an effect of a cause. It is *an* effect because aspirin is being compared with chalk. The same aspirin, if compared with acetaminophen, might have yielded a different effect. There may be other, measurable factors implicated in recovery, but these are largely irrelevant, first because the emphasis is on the effect of aspirin and not on the effect of other causes, second because in an experiment of reasonable scale, randomization will likely have balanced these other factors as between chalk- and aspirin takers, so that the measured effect of aspirin is not confounded with other causal effects. Aspirin has some chemical properties that chalk does not, and it is quite possible that stochastic aspects linking these properties to biological variation within the subject population may explain why some people recovered and others did not, but an understanding of the biomedical *mechanisms* linking aspirin to headache relief is not required to estimate aspirin's causal effect.

Fienberg, Faigman, and Dawid (In press:24) distinguish R_1 , the observed outcome given exposure to aspirin, from R_0 , the potential outcome of the same individuals had they been given chalk instead. PC is a bounded probability solution to the question of whether an aspirin taker who recovered would have recovered had they taken chalk alone. It is a *statistical* solution (Holland 1986:947) since, as Fienberg, Faigman, and Dawid (In press:24) note, “we have not observed—indeed, we have no way of observing— R_0 .” The algebraic solution ($1.0 \quad PC \quad 0.6$) is identical to that obtained by randomly matching, post hoc, the aspirin takers with the chalk takers: At least 18 of the recovering aspirin takers must find a still suffering chalk taker, and perhaps all 30 will. “[E]ven if we had perfect information from the experiment, with no uncertainty, we can say little more about PC (unless we have some additional relevant scientific information)” (Fienberg, Faigman, and Dawid In press: 25). What constitutes relevant scientific information will vary according to the problem. In the social sciences, it is common to meld scientific solutions with statistical ones. For example, fixed effects models with repeated observations on units are given a causal interpretation (Allison 2005:1-2) by assuming, at least implicitly, *temporal stability* and *causal transience* (Holland 1986:948). In the experimental setting, if units could be matched a priori on some compelling scientific grounds, with subsequent random assignment to treatment within pairs, then PC could be known precisely (sampling error aside) by conditioning on $R_1 = 1$ and counting the pairs where $R_0 = 0$.

If we change aspirin and chalk to Head Start program or no Head Start program, and observe not the absence of a headache but the absence of grade retardation, then the statement that at least 60 percent of the children who are in the normative grade for age would have been held

back in the absence of participation in Head Start is a very useful piece of information. Whether PC is only loosely bounded or more precisely so, its interpretation appears to have little to do with whether one is interested in inference to a single case or to a larger aggregate. Even in *Perry Mason*, most defendants do not have an identical twin, so one is rarely if ever making inference to a particular case within a particular study. Fienberg, Faigman, and Dawid (In press:15) refer to the courtroom circumstance in which “a miracle occurs and some expert offers the opinion that the case at hand *is* an instance of that general phenomenon.” Sociology and other social sciences are fields of everyday miracles since a great deal of standard practice is devoted to establishing the generalizability (external validity) of a study, sample, observational setting, and so on.

Fienberg, Faigman, and Dawid’s (In press) articulation of the notions of EoC and CoE in legal contexts is a valuable one, even for those of us not preoccupied with the issue of adducing cause in particular cases. This is because the primitives behind causal argumentation can never adjudicate “causal analysis” alone. Methods and interpretations depend, to put it in the vernacular, on what it is we think we are talking about. I was long ago attracted to the power of the potential outcomes model for clarifying problems in sociology, especially with respect to understanding the EoC (Smith 1990). This led me in the direction, in sociology and related fields, of thinking about *meaningful* causes as operating at a higher level of analysis and intervention than the unit level at which most measurement obtains (Smith 2003, 2005, 2009, 2013a, 2013b). I thus have no problem with the idea that a “strong corporate culture” (Fienberg, Faigman, and Dawid In press:27) might have a demonstrable effect on the wages of women, relative to what they might expect in the presence of an alternative workforce organization. Well-designed comparisons of cities with different gun laws provide good estimates of the comparative probability that an inhabitant of the city will be murdered by one (Sloan et al. 1988). In the modern U.S. military, where rules, promotion systems, and the like are transparent and closely monitored, African Americans have marriage prospects and patterns that look like their white peers; in the rest of U.S. society, they do not (Lundquist 2004, 2006). This may sound like typical sociological dreaminess, but one can also get to the same point from a statistical perspective (Holland 2008:102-3).

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Biography

Herbert L. Smith is a professor of sociology and the director of the Population Studies Center at the University of Pennsylvania. Current research focuses on the role of cohorts in demographic forecasting, including the relationship between deterministic demographic and stochastic econometric forecasting models. Thoughts on the role of the potential outcomes model of causation in, respectively, sociology and demography appear as chapters in the Springer Verlag volumes *Handbook of Causal Analysis for Social Research*, edited by Stephen L. Morgan, 2013, and *Causal Analysis in Population Studies: Concepts, Methods, Applications*, edited by Henriette Engelhardt-Woelfler, Hans-Peter Kohler, and Alexia Fuernkranz-Prskawetz, 2009.

Figure 1.
Heuristic depiction of the relationship of causes of effects to effects of causes, with reference to observational and inferential scale.