11.3 ESTIMATING CAUSAL EFFECTS

11.3.1 The Intuition behind the Back-Door Criterion (Chapter 3, p. 79)

Question to Author:

In the definition of the back-door condition (p. 79, Definition 3.3.1), the exclusion of X's descendants (Condition (i)) seems to be introduced as an after fact, just because we get into trouble if we don't. Why can't we get it from first principles; first define sufficiency of Z in terms of the goal of removing bias, and then show that, to achieve this goal, we neither want nor need descendants of X in Z.

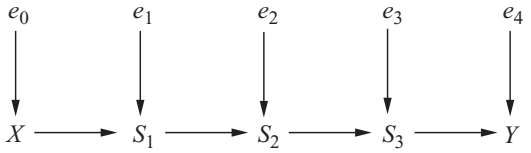

Figure 11.4 Showing the noise factors on the path from X to Y.

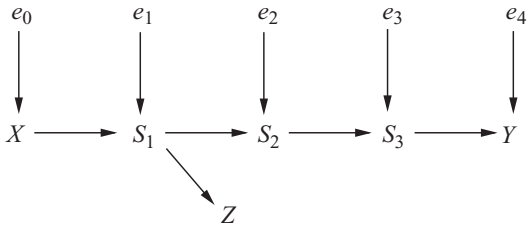

Figure 11.5 Conditioning on Z creates dependence between X and e_1, which biases the estimated effect of X on Y.

Author's Answer:

The exclusion of descendants from the back-door criterion is indeed based on first principles, in terms of the goal of removing bias. The principles are as follows: We wish to measure a certain quantity (causal effect) and, instead, we measure a dependency $P(y \mid x)$ that results from all the paths in the diagram; some are spurious (the back-door paths), and some are genuinely causal (the directed paths from X to Y). Thus, to remove bias, we need to modify the measured dependency and make it equal to the desired quantity. To do this systematically, we condition on a set Z of variables while ensuring that:

1. We block all spurious paths from X to Y,
2. We leave all directed paths unperturbed,
3. We create no new spurious paths.

Principles 1 and 2 are accomplished by blocking all back-door paths and only those paths, as articulated in condition (ii). Principle 3 requires that we do not condition on descendants of X, even those that do not block directed paths, because such descendants may create new spurious paths between X and Y. To see why, consider the graph

$$X \rightarrow S_1 \rightarrow S_2 \rightarrow S_3 \rightarrow Y.$$

The intermediate variables, S_1, S_2,…, (as well as Y) are affected by noise factors e_0, e_1, e_2,. . . which are not shown explicitly in the diagram. However, under magnification, the chain unfolds into the graph in Figure 11.4.

Now imagine that we condition on a descendant Z of S_1 as shown in Figure 11.5. Since S_1 is a collider, this creates dependency between X and e_1 which is equivalent to a back-door path

$$X \leftrightarrow e_1 \rightarrow S_1 \rightarrow S_2 \rightarrow S_3 \rightarrow Y.$$

By principle 3, such paths should not be created, for it introduces spurious dependence between X and Y.

Note that a descendant Z of X that is not also a descendant of some S_i escapes this exclusion; it can safely be conditioned on without introducing bias (though it may decrease the efficiency of the associated estimator of the causal effect of X on Y). Section

11.3.3 provides an alternative proof of the back-door criterion where the need to exclude descendants of X is even more transparent.

It is also important to note that the danger of creating new bias by adjusting for wrong variables can threaten randomized trials as well. In such trials, investigators may wish to adjust for covariates despite the fact that, asymptotically, randomization neutralizes both measured and unmeasured confounders. Adjustment may be sought either to improve precision (Cox 1958, pp. 48–55), or to match imbalanced samples, or to obtain covariate-specific causal effects. Randomized trials are immune to adjustment-induced bias when adjustment is restricted to pre-treatment covariates, but adjustment for post-treatment variables may induce bias by the mechanism shown in Figure 11.5 or, more severely, when correlation exists between the adjusted variable Z and some factor that affects outcome (e.g., e_4 in Figure 11.5).

As an example, suppose treatment has a side effect (e.g., headache) in patients who are predisposed to disease Y. If we wish to adjust for disposition and adjust instead for its proxy, headache, a bias would emerge through the spurious path: treatment $\rightarrow$ headache $\leftarrow$ predisposition $\rightarrow$ disease. However, if we are careful never to adjust for any consequence of treatment (not only those that are on the causal pathway to disease), no bias will emerge in randomized trials.

Further Questions from This Reader:

This explanation for excluding descendants of X is reasonable, but it has two shortcomings:

1. It does not address cases such as

   $$X \leftarrow C \rightarrow Y \rightarrow F,$$

   which occur frequently in epidemiology, and where tradition permits the adjustment for $Z = \{C, F\}$.

2. The explanation seems to redefine confounding and sufficiency to represent something different from what they have meant to epidemiologists in the past few decades. Can we find something in graph theory that is closer to their traditional meaning?

Author's Answer

1. Epidemiological tradition permits the adjustment for $Z = (C, F)$ for the task of testing whether X has a causal effect on Y, but not for estimating the magnitude of that effect. In the former case, while conditioning on F creates a spurious path between C and the noise factor affecting Y, that path is blocked upon conditioning on C. Thus, conditioning on $Z = \{C, F\}$ leaves X and Y independent. If we happen to measure such dependence in any stratum of Z, it must be that the model is wrong, i.e., either there is a direct causal effect of X on Y, or some other paths exist that are not shown in the graph.

   Thus, if we wish to test the (null) hypothesis that there is no causal effect of X on Y, adjusting for $Z = \{C, F\}$ is perfectly legitimate, and the graph shows it (i.e., C and F are nondescendant of X). However, adjusting for Z is not legitimate for assessing the causal effect of X on Y when such effect is suspected,

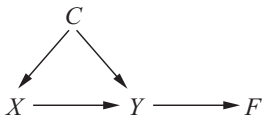

Figure 11.6 Graph applicable for accessing the effect of X on Y.

because the graph applicable for this task is given in Figure 11.6; F becomes a descendant of X, and is excluded by the back-door criterion.

2. If the explanation of confounding and sufficiency sounds at variance with traditional epidemiology, it is only because traditional epidemiologists did not have proper means of expressing the operations of blocking or creating dependencies. They might have had a healthy intuition about dependencies, but graphs translate this intuition into a formal system of closing and opening paths.

   We should also note that before 1985, causal analysis in epidemiology was in a state of confusion, because the healthy intuitions of leading epidemiologists had to be expressed in the language of associations – an impossible task. Even the idea that confounding stands for "bias," namely, a "difference between two dependencies, one that we wish to measure, the other that we do measure," was resisted by many (see Chapter 6), because they could not express the former mathematically.[3]

   Therefore, instead of finding "something in graph language that is closer to traditional meaning," we can do better: explicate what that "traditional meaning" ought to have been.

   In other words, traditional meaning was informal and occasionally misguided, while graphical criteria are formal and mathematically proven.

   Chapter 6 (pp. 183, 194) records a long history of epidemiological intuitions, some by prominent epidemiologists, that have gone astray when confronted with questions of confounding and adjustment (see Greenland and Robins 1986; Wickramaratne and Holford 1987; Weinberg 1993). Although most leading epidemiologists today are keenly attuned to modern developments in causal analysis, (e.g., Glymour and Greenland 2008), epidemiological folklore is still permeated with traditional intuitions that are highly suspect. (See Section 6.5.2.)

   In summary, graphical criteria, as well as principles 1–3 above, give us a sensible, friendly, and unambiguous interpretation of the "traditional meaning of epidemiological concepts."