The Similarity of Causal Inference in Experimental and Non-Experimental Studies*

Abstract

For nearly as long as the word “correlation” has bee

1. Introduction

Philosophers, statisticians, and computer scientists, at least those who have abandoned the goal of producing a reductive account of causation, have come to largely agree on how to represent qualitative causal claims and how to connect such claims to statistical evidence through probabilistic independence and dependence (Glymour and Cooper 1999; Pearl 2000; Spirtes, Glymour and Scheines 2000; Woodward 2003).[1] Included in this scheme is a method for representing experimental interventions, and for clarifying what sorts of assumptions we must make about interventions in order to consider them "ideal." With this appar 0 0 rg32000 1 Tf12 0m72(h0 62P5TjET6 6363.2764 Tm[;BDC Qq0 6592 0m7)3( )]

instrumental variable that stands in the same relationship to X and Y in the observational study as does the ideal intervention on X in the experimental study. In what follows, I briefly sketch the key ideas behind the representational system, I show how an experimental causal inference works in this system, how the typical observational causal inference works involving detectible instrumental variables, and the parallel between detectible instrumental variables and experimental interventions.

2. Representing Causation

2.1 Causal Graphs, Probability Distributions, and the Causal Markov Axiom

Recently from computer science, but as far back as Sewall Wright in the early 20th century (Wright 1934), the fundamental representational device for causal systems is the directed graph. A directed graph is simply a collection of vertices and directed edges over pairs of these vertices. In a directed graph interpreted as a causal graph, each directed edge (or arrow) from one vertex X to another Y is taken to assert that X is a direct cause of Y relative to the set of vertices in the graph. For example, Figure 1 represents a graph **G** = <**V**,**E**>, with vertices **V** = {Exposure, Infection, Symptoms}, and 19mpt3{1,i1.80 659

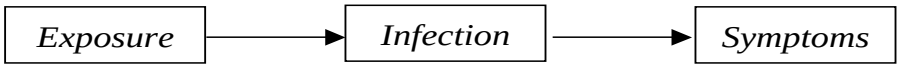
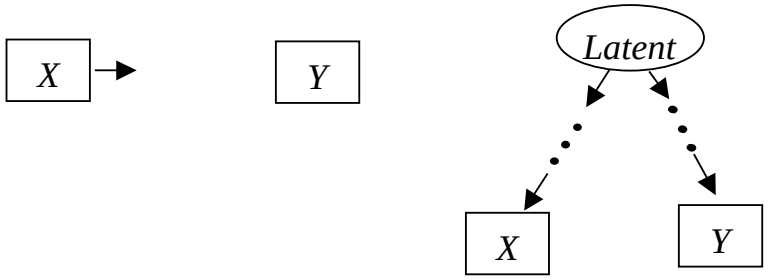


Figure 1: Causal Graph

A causal graph is assumed to be representationally complete in the following sense: if two variables in the graph are effects of a common cause C, then C is included in the graph. This does *not* require us to include all the causes of a variable in the graph, it only requires that we include all the common causes. To be clear, this is a representational assumption, not one concerning which variables we will measure when the goal is inference. The key assumption connecting causal graphs to probability distrubtions is an axiom that constrains the set of probability distributions that a given causal graph can generate (Spirtes, et. al. 2000):

> **Causal Markov Axiom**: In any probability distribution **P** generated by a given causal graph **G**, each variable X is probabilistically independent of the set **Y** consisting of all variables that are not effects of X, conditional on the direct causes of X. That is, $\forall X \in$

3. A pair of paths, one from some third variable C (possibly latent) to X and one from C to Y.



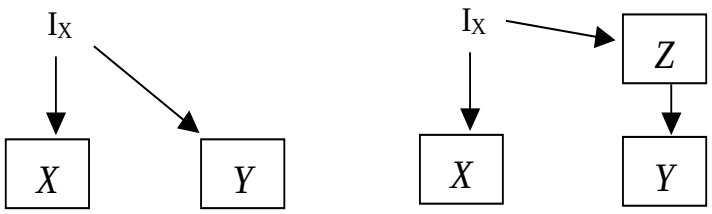
of association can be used to estimate the size of the effect of X on Y (Pearl, 2000). The key to this type of simple experimental inference is that the intervention is:

i) a direct cause of X, and

ii) not adjacent to Y, and

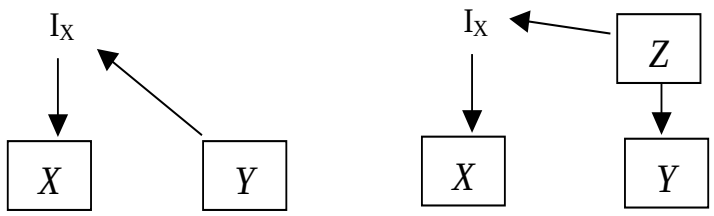
iii) ideal.

Consider why it is desirable that it satisfy these conditions. First, if the intervention is a direct cause of X, but also of Y or some other cause of Y,[5] then X and Y will be associated in virtue of the intervention, not in virtue of the effect of X on Y (Figure 6).



(a)

after an intervention, but not because of an effect of X on Y. Again, we could handle the second form of treatment-bias (Figure 7-b) by conditioning on Z, but the first form (Figure 7-a) is fatal.



(a) **(b)**

Figure 7: Treatment-Bias Interventions

Generally then, an intervention on X can be fat-hand or treatment-biased without making causal inference impossible, but I_X *cannot be adjacent*[6] *to Y* in the causal graph. Need the intervention be ideal? Is causal inference still possible in cases in which the intervention on X does not fully determine X's probability distribution and thereby x-out the influence of all other direct causes on X? For the argument as I have sketched it above, clearly yes, but in general the answer is no.

In cases where we know something about the parametric form of the dependence of effects on their causes, for example linear structural equation models (Bollen 1989), interventions need not be ideal. In linear structural equation models each effect is a linear combination of its direct causes plus Gaussian noise, and in certain such models instrumental variable estimators (Bowden and Turkington 1974) can be used to estimate the strength of causal influence even in the presence of latent common causes. In Figure 8, for example, I_X is an instrumental variable for X

$\alpha\beta / \alpha = \beta$ is a consistent estimator of the effect of X on Y, even though X and Y are confounded by a latent common cause.

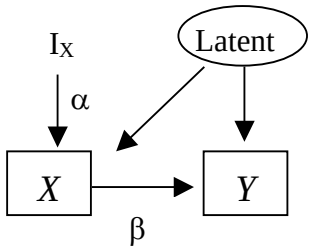


Figure 8: Instrumental Variable I_X

4. Causal Inference in Non-Experimental Studies

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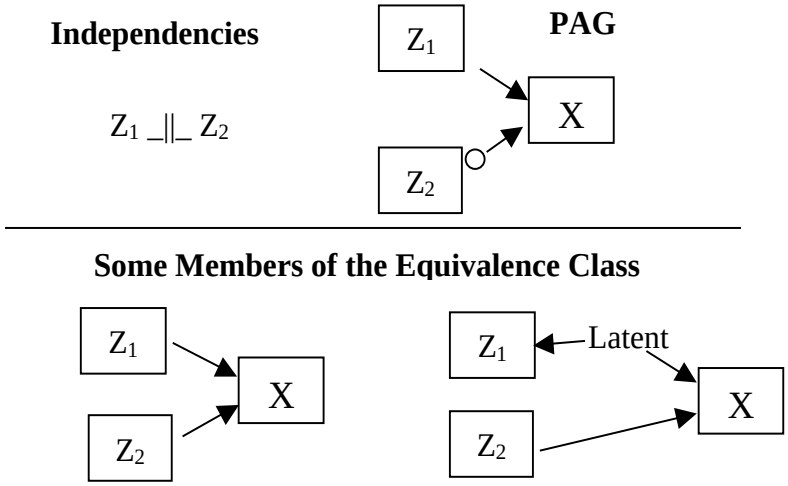
unconditionally independent, then the PAG and some of the members of the equivalence class it represents are pictured in Figure 9.

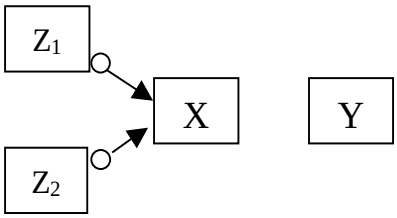


Independencies

$Z_1 \perp\!\!\!\perp Z_2$

$Z_1 \perp\!\!\!\perp Y \mid X$ RG

PAG



Some Members of the Equivalence Class

Latent

After going through all the triples and orienting them with the collider rule, we go through them again, this time looking for triples in which B was oriented as a non-collider from the triple A,B,C, but as collider from a triple A,B,D, that is Figure 11.

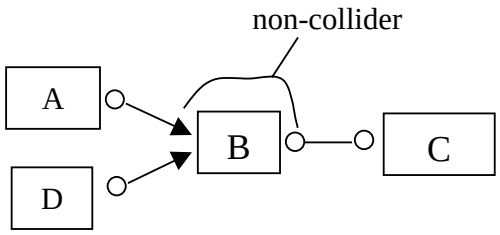


Figure 11

We can then combine these orientations to fully orient the Bo-oC adjacency as B C, which is the only way to orient Bo-oC in order to avoid making B a collider in the A,B,C triple. We call this the "**away-from-collider rule**."[10]

Consider a concrete empirical case to illustrate. Sewall and Shah (1968) collected data on over 10,000 Wisconsin high school seniors in order to study the relationship between parental encouragement (PE) and college plans (CP). They also measured socio

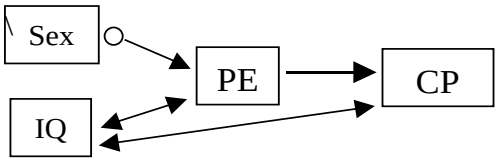


Figure T30.5 29. d0.793451 65 2 1 2 (o 644 C)2(ol)-2(l)-2(6.31g6.3164-10(P

X or there is a latent common cause of V and X. By intervening on X with I_X, we ensure that the adjacency between I_X and X is *into* X, but for the causal inference it is not strictly necessary.

I use the word "detectible" to highlight the fact that, in a non-experimental study, the issue is finding a variable that detectibly satisfies the same basic conditions that we believe are satisfied in an experimental study. For example, in the case study involving college plans and parental encouragement above, we managed to detect that the adjacency between Sex and PE was *into* PE because Sex and IQ are independent, thus giving us a collider oriented triple: Sex o PE o IQ.

Suppose, however, that Sewall and Shah had not thought to measure IQ. Just measuring Sex, PE, and CP, and finding only

question that does not permit, for ethical or practical reasons, an experimental intervention, a good causal scientist should not throw up his hands and proclaim that "only experimental studies can support causal conclusions." Rather she should seek to systematically combine background knowledge and statistical analysis to find detectible instruments for causal inference.

Wright, S. (1934). The method of path coefficients. *Ann. Math. Stat*. 5, 161-215.

[11] We do *not* need to condition on PE to make Sex and IQ independent.

[12] We *do* need to condition on PE to make Sex and CP independent.

[13] The orientation of the IQ - CP and IQ - PE adjacencies results from applying another rule I will not explain. It is not relevant to orienting the PE CP adjacency.

[14] which means they are associated no matter what set we condition on.