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

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Abstract

For nearly as long as the word “correlation” has been part of statistical parlance, students have been warned that correlation does not prove causation, and that only experimental studies, e.g., randomized clinical trials, can establish the existence of a causal relationship. Over the last few decades, somewhat of a consensus has emerged between statisticians, computer scientists, and philosophers on how to represent causal claims and connect them to probabilistic relations. One strand of this work studies the conditions under which evidence accumulated from non-experimental (observational) studies *can* be used to infer a causal relationship. In this paper, I compare the typical conditions required to infer that one variable is a direct cause of another in observational and experimental studies. I argue that they are essentially the same.

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 apparatus, it is easy to show that if we have ideally intervened experimentally upon (X), then an association between X and Y entails that X is a cause of Y. Inferring that one variable is a cause of another is what I call "causal inference."

With this apparatus, Spirtes, Glymour, and Scheines (2000), Pearl (1988, 2000) and others have developed algorithms for determining the *set* of causal structures that are consistent with the independence relations assumed to hold over a set of measured variables in a non-experimental, or observational study, even causal structures that include latent, or unmeasured variables. In some instances all the causal structures in an equivalence class agree on some subset of the causal relations, and in some cases they all agree that one variable X is a cause or direct cause of another Y. In these cases we can, using basically the same assumptions as are made in experimental studies, infer that X is a cause of Y.

Although we have no general characterization of the conditions under which a causal inference to X → Y can be made in observational studies, it turns out that when the inference is possible it is often driven by the existence of what I call a *detectible*

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 edges **E** = {<Exposure, Infection>, <Infection, Symptoms>}. We further assume that the vertices in such a graph can be interpreted as random variables with some probability distribution P, and that causal processes situated in some definite background context generate probability distributions over these varables.

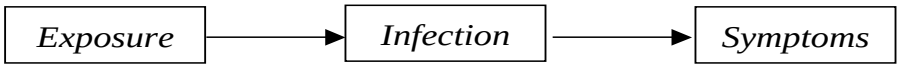


Figure 1: Causal Graph

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, $\forall X \in$ **G**, X _||_ **Non-effects of X** | **Direct Causes of X**, in **P**.

This entails, for example, that Exposure is independent of Symptoms conditional on Infection in any probability distribution that the causal graph in Figure 1 can generate. In acyclic causal graphs, the Causal Markov Axiom is equivalent to a graphical relation called d-separation (Pearl 1988). If **X** and **Y** are d-separated by **Z**, then the Causal Markov Axiom entails that **X** and **Y** are independent conditional on **Z**.

2.2 Interventions

To model experimental interventions on a causal system, we add a new variable representing the intervention, connect it to the graph in a particular way, and make an assumption about how ideal interventions change the causal graph (Spirtes, et. al 2000; Pearl 2000).

For simplicity we restrict interventions to act only on one variable at a time. Let an intervention on X be called I_X, and model this by adding I_X to the graph as a direct cause of only X, and the effect of no variable. For example, to model an experimental intervention on Infection in the causal graph in Figure 1, we build the graph in Figure 2.

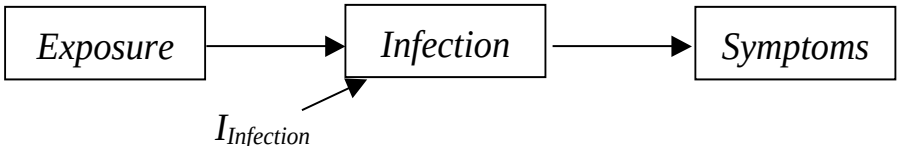


Figure 2: Modeling an Intervention

Modeling an intervention as a direct cause makes explicit the fact that this account of causation does not attempt to reduce causation to intervention. It goes to the other extreme, it reduces intervention to the idea of a direct cause. Direct cause is left undefined, but connected to probability through the Causal Markov Axiom.

Finally, we say an intervention is "ideal" if it totally determines the probability distribution of its target. For example, a medical trial assessing the effect of St. John's Wort on depression might assign either of two treatments: St. John's Wort or placebo, by flipping a fair coin to randomly assign subjects to one of these two treatments. This

intervention is ideal if the coin flip totally determines the treatment, leaving no influence from the subject's disposition to take alternative medicines, etc.

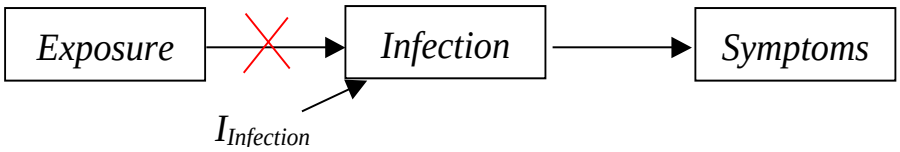


Figure 3: Modeling an Ideal Intervention

If I_X is an *ideal* intervention on X, then we eliminate, or "x-out" any arrows in the original causal graph that point into X (Figure 3). This represents the idea that our ideal intervention has taken over X's probability distribution, overwriting any influence its direct causes might have had prior to our intervention. In contrast, we do not x-out arrows coming *out of* X. So ideal interventions on X annihilate the relationship between X and its direct causes, but leave intact the relationship between X and its direct effects.[2]

3. Causal Inference in Experimental Studies

The problem of causal inference from association is underdetermination. If two variables X and Y are associated in a non-experimental study, then many different causal graphs can explain the association. In general, three kinds of causal connection[3] (Figure 4) can produce an association (probabilistic dependence) between two variables X and Y:

1. A path[4] from X to Y
2. A path from Y to X

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

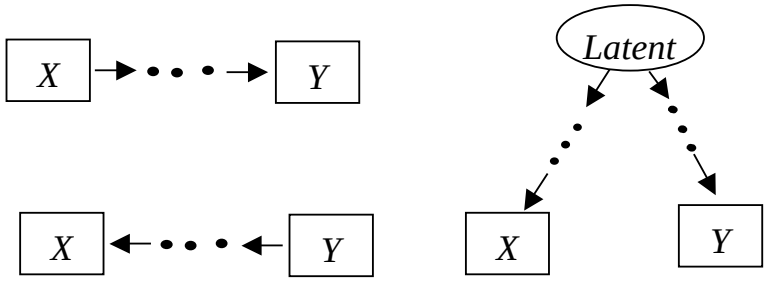


Figure 4: Causal connections which explain an association between X and Y

Consider the same problem after an ideal intervention on X, however. The intervention eliminates the influence of all the direct causes of X in the original graph, thus all causal connections save paths from X to Y are destroyed (Figure 5).

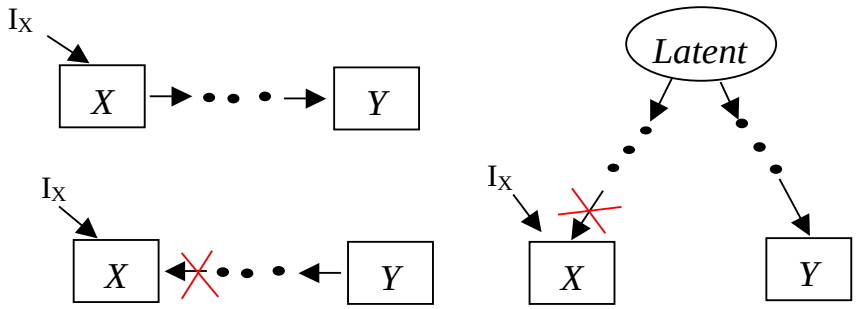


Figure 5: Causal connections after an ideal intervention on X

The result is incredibly simple, but incredibly powerful. If X and Y are associated after an ideal intervention on X, then X is a cause of Y. Further, the quantitative degree

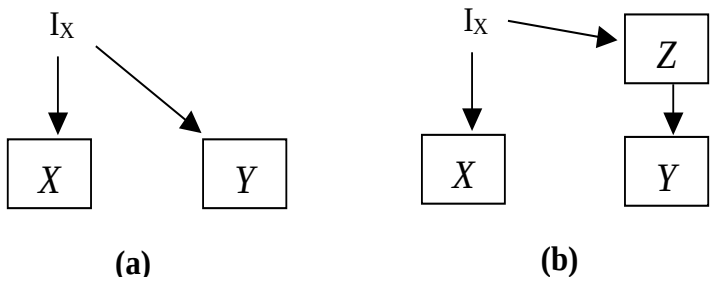
of association can be used to estimate the size of the effect of X on Y (Pearl, 2000). The key to this type of simple experimental inference is that the intervention is:

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).



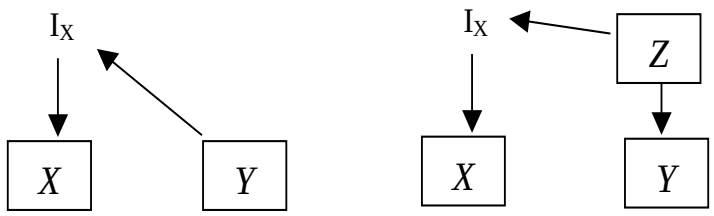
(a) **(b)**

Figure 6: Fat-hand Interventions

It is possible to handle the second form of fat-hand intervention (Figure 6-b) by looking not just at whether X and Y are associated but also at whether X and Y are associated *conditional* on Z. The first type of fat-hand intervention (Figure 6-a), in which I_X is a direct cause of Y is fatal to causal inference.

Similarly, the intervention must itself not be an effect of any other variable, a problem I will call treatment-bias (Figure 7). Again, in such cases X and Y would be associated

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 → Y, and the quantity $\rho_{IX,Y} / \rho_{IX,X}$ =

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

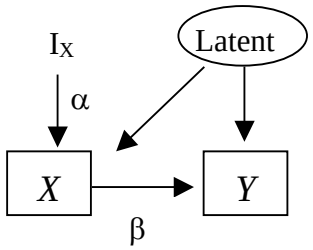


Figure 8: Instrumental Variable I_X

If I_X is an intervention, but not an ideal intervention, as in Figure 8, and the dependencies are linear, then the instrumental variable estimator can be used to do causal inference; we simply statistically test whether $\beta = 0$. Whether or not the instrumental variable Z is an intervention,

i) Z must be adjacent to X but not an effect of X,[7] and

ii) Z may not be adjacent to Y

To summarize, in experimental settings causal inferences concerning whether X is a cause of Y are driven by interventions on X which are:

i) direct causes of X, and

ii) not adjacent to Y, and

iii) ideal (they totally determine the probability of X)

4. Causal Inference in Non-Experimental Studies

In non-experimental studies, we have no intervention variable with known relationships to the variables under study. We have only a set of measured variables governed by some causal structure that we assume satisfies the Causal Markov Axiom, but which might include unmeasured common causes of the variables we have measured. To set aside statistical difficulties, we will assume that we can determine which probabilistic independence relations hold over the measured variables with perfect reliability. Finally, we will assume that the probability distribution **P** is Faithful[8] to the causal graph (Spirtes, et. al 2000). That is, we assume that all independence relations that hold in **P** are entailed by the Causal Markov Axiom, thereby ruling out independence relations holding in **P** in virtue of particular settings for the probabilities in **P**.

In this setting, the underdetermination of causation can be characterized with an equivalence class of causal graphs, each member of which is a causal graph that entails all and only those independence relations observed to hold in some distribution $\mathbf{P_O}$ over the set of observed variables **O**. As you would expect, when we allow the equivalence class to include graphs that have unobserved common causes, that is, variables not in **O**, then the class is infinite. Spirtes and Richardson (1996) describe a graphical object called a Partial Ancestral Graph or PAG, to compactly represent equivalence classes that include latent common causes.

Fortunately, it is sometimes the case that all members of the equivalence class represented by a PAG share features of a causal relationship between two variables in **O**. For example, if we have measured three variables, Z_1, Z_2, and X, and find Z_1 and Z_2 to be

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

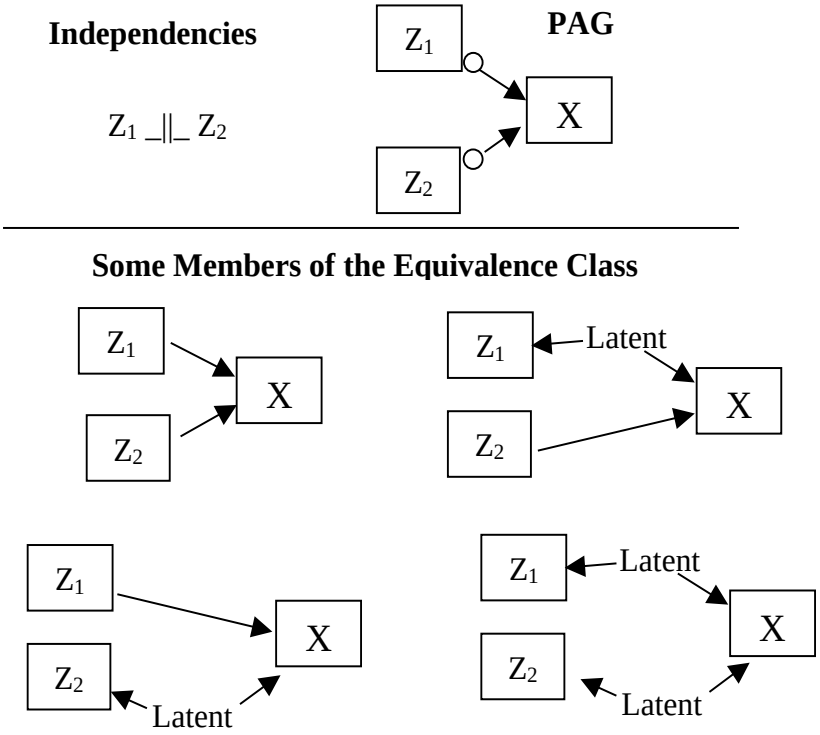


Figure 9: Equivalence Class

Even though X is associated with both Z_1 and Z_2, in no member of this equivalence class is X a cause of either Z_1 or Z_2. We can't, from three variables and no extra background knowledge make a positive causal inference, but we can make two negative ones: X is not a cause of Z_1 nor is it a cause of Z_2. With four variables we can actually make a positive causal inference. Adding Y, and assuming that Y is unconditionally associated with all the other variables but independent of Z_1 and Z_2 conditional on X, the PAG in Figure 10 represents the set of causal graphs that entail all and only these independencies.

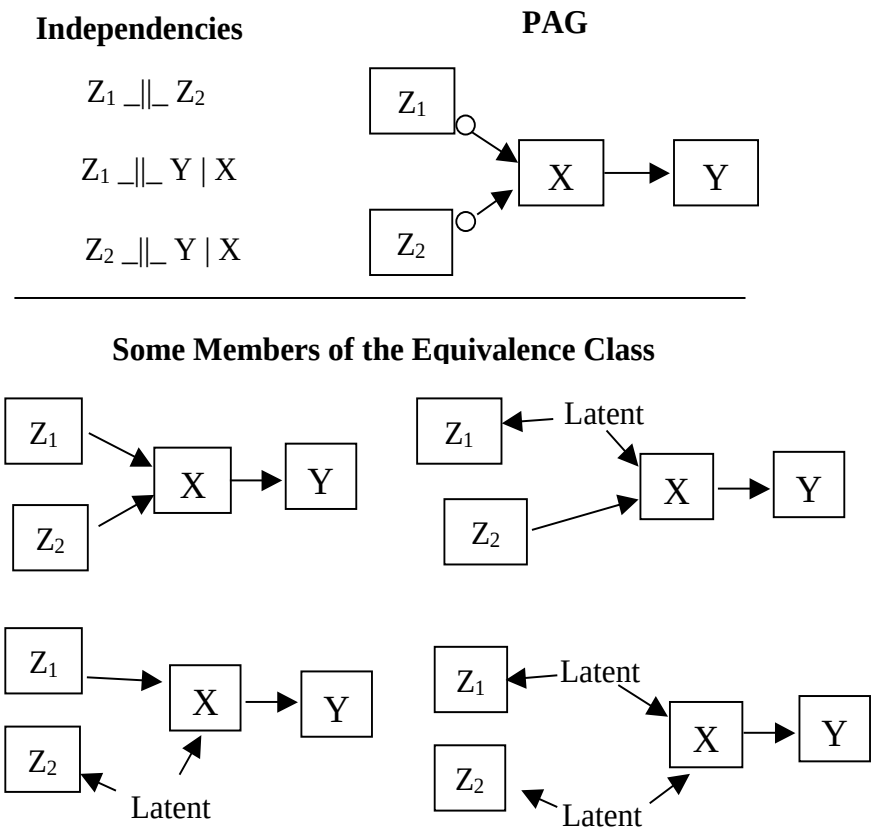


Figure 10: PAG that gives a Causal Inference

In every member of the equivalence class represented by the PAG in Figure 10, X is a cause of Y, and in no member of the equivalence class is there a latent common cause of X and Y, exactly the same conclusion we can reach by finding an association between X and Y in an experimental study in which we have ideally intervened upon X.

The FCI algorithm (Spirtes, et. al 2000) computes PAGs from given independencies, assuming the Causal Markov Axiom, Faithfulness, and that there is a graph that generated the independencies given. By examining one set of conditions required by the algorithm in order to determine that i) X is cause of Y and ii) there is no latent common cause of X and Y in every member of the equivalence class, I will try to illuminate the similarity between causal inference in experimental and non-experimental settings.

The FCI algorithm proceeds in two stages. In the first it identifies the adjacencies in the PAG, and in the second it orients any of these adjacencies it can. Any pair of variables that are not adjacent after the adjacency phase are not adjacent in any causal graph in the final equivalence class (Spirtes, et al. 2000). In a PAG, an unoriented adjacency is represented as Ao-oB. The possible orientations and what they represent about the members of the equivalence class are:

- A o→ B means that B is not a cause of A in any member of the class, but either A is a cause of B or they have a latent common cause.
- A ←→ B means that A and B have a latent common cause in every member of the class.
- A → B means that A is a cause of B with no latent common cause in every member, and
- A o-oBo-o C means that either i) B is a cause of A and there is no latent common cause of A and B, *or* ii) B a cause of C with no latent common cause.

In the orientation phase, the algorithm looks for triples A,B,C such that A and B are adjacent, B and C are adjacent, but A and C are *not* adjacent, i.e., A o-o B o-o C. If B *is* included in the set that made A and C independent,[9] then we orient this triple as a "non-collider" at B: A o-oBo-o C, else we orient the triple as a "collider" at B: A o→ B ←o C. We call this orientation step the "**collider rule**."

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.

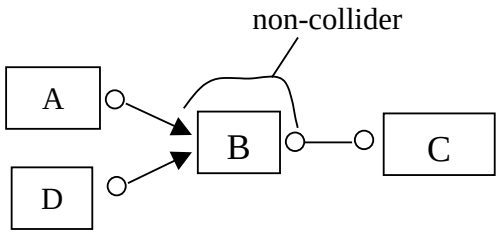


Figure 11

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-economic status (SES), Sex, and IQ. For simplicity I omit SES. The independence relations that hold statistically among PE, CP, Sex, and IQ are: Sex _||_ IQ, Sex _||_ CP | PE. After the adjacency phase of FCI, we have Figure 12.

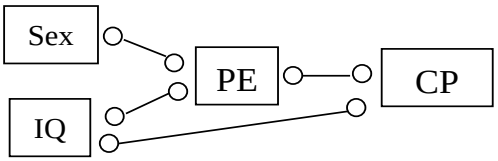


Figure 12: After Adjacency Phase

There are two triples that satisfy the requirements for orientation: 1) Sex o-o PE o-o IQ, and 2) Sex o-o PE o-o CP. In 1, we can orient PE as a collider,[11] and in 2, we can orient PE as a non-collider.[12] So after applying the collider rule we have the orientation in Figure 13.

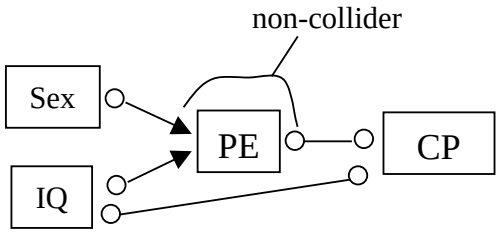


Figure 13: After Applying the Collider Rule

Going back through these two triples, we can apply the away-from-collider rule to the Sex, PE, CP triple, giving us a PAG[13] in which parental encouragement is an unconfounded cause of college plans:

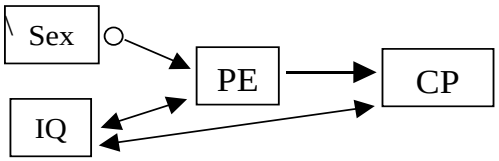


Figure 14: PAG for College Plans

5. The Similarities

Consider the inference to X → Y in a non-experimental setting with the away-from-collider rule. We need a triple Z o→ X o-o Y. Besides knowing that X and Y are adjacent,[14] this construction involves a *detectible instrument* Z that is:

i) adjacent to X but not an effect of X,

ii) not adjacent to Y

These conditions match precisely with those required of an instrumental variable as used commonly in econometrics (see section 3 above), and they also match quite closely to the two conditions I specified for experimental studies, which I repeat here. Interventions I_X on X should at a minimum:

i) be direct causes of X,

ii) not be adjacent to Y.

A variable V that satisfies the first condition will also satisfy the first condition for a detectible instrument Z, but structurally it is not necessary for V to directly cause X. It is enough that the adjacency between V and X be *into* X, that is, either V is a direct cause of

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 that Sex _||_ CP | PE, we would have the following PAG: Sex o-o PE o-o CP, which does not support a causal inference between PE and CP. Why? Because we do not know that the Sex o-o PE adjacency is *into* PE. If, however, we add the perfectly plausible background knowledge that parental encouragement cannot be a cause of one's gender, then the variable Sex *would* satisfy the conditions for a detectible instrument: Sex is adjacent to PE but not an effect of PE (Sex o→ PE), and Sex is not adjacent to CP. In that case would have a detectible instrument and could, from the Causal Markov Axiom and Faithfulness assumptions, infer that parental encouragement is indeed a cause of college plans with no latent common cause between them.

The parallels between these forms of experimental and non-experimental inference are not complete, nor are they necessary. They do suggest, however, that faced with a causal

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.

REFERENCES

Bowden, R. and Turkington, D. (1984). *Instrumental variables*. Cambridge University Press, NY

Cartwright, Nancy (2003). What is Wrong with Bayes Nets? in *Probability is the Very Guide of Life*, Henry Kyburg and Mariam Thalos (eds), Open Court Press, 253-276.

Glymour, C., and Cooper, G. (1999). *Computation, Causation, and Discovery*. AAAI Press and MIT Press.

Hausman, D. (1998). *Causal Asymmetries*. Cambridge University Press.

Pearl, J. (1988). *Probabilistic Reasoning in Intelligent Systems***.** Morgan Kaufmann, San Mateo.

Pearl, J. (2000). *Causality: Models, Reasoning, and Inference*. Cambridge University Press.

Sewall, W., and Shah, V. (1968). Social class, parental encouragement, and educational aspirations. *American Journal of Sociology*. 73: 559-572.

Spirtes, P., Glymour, C., and Scheines, R. (2000) *Causation, Prediction, and Search*, 2nd Edition. MIT Press, Cambridge MA.

Spirtes, P., and Richardson, T. (1996), A Polynomial Time Algorithm For Determining DAG Equivalence in the Presence of Latent Variables and Selection Bias, *Proceedings of the 6th International Workshop on Artificial Intelligence and Statistics*.

Woodward, James (2003). *Making Things Happen: A Theory of Causal Explanation*. Oxford University Press.

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

FOOTNOTES

[1] Of course there are many important exceptions. For example, I do not believe Nancy Cartwright, who more than anyone showed the hopelessness of reducing causation to probability, would endorse this sentence (Cartwright, 2003).

[2] Woodward (2003) calls the latter "invariance," and argues at length that it is the asymmetry of invariance between 1) a variable and its causes and 2) a variable and its effects that captures the asymmetry of causation.

[3] Hausmann (1998) defines a causal connection between X and Y as either X causes Y, Y causes X, or a third variable causes both, or some combination.

[4] A path is just a sequence of arrows all pointing in the same direction.

[5] Thanks to Kevin Kelly, such interventions are called "fat-hand," the analogy being reaching to move a particular piece in chess but knocking over others because of a fat hand.

[6] X and Y are adjacent if X is a direct cause of Y or Y a direct cause of X.

[7] Clearly if I_X is adjacent to Latent or to Y, then $\rho_{IX,Y} \neq \alpha\beta$. Similarly, if I_X is an effect of X, then again $\rho_{IX,Y} \neq \alpha\beta$, this time because there is a causal connection of I_X and Y from the Latent common cause which is *not* present when I_X is a cause of X.

[8] Pearl (1988) uses the word Stable, but means the same thing.

[9] which was necessary to eliminate the adjacency between A and C

[10]There are other orientation rules that can support a positive causal inference, e.g., the definite-discriminating-path rule, but they are too complicated to explain or characterize in the space I have here.

[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.