Colliding bias (part 2): missing theorems and missing proofs

The heuristic argument

In part 1, we saw a heuristic example for uni-path colliding. Let's see the heuristic argument for bi-path colliding. To simplify, I will use deterministic jargon.

Suppose there are only two causes, *A* and *B*, of some outcome, *C*, and all three variables are binary. Assume that *A* and *B* are independent – that is, knowing the value of one tells us nothing about the value of the other. This state of affairs is perfectly encoded by the diagram $A \rightarrow C \leftarrow B$, in which *C* is a collider. According to this diagram, *A* and *B* are not associated because neither is a cause of the other, and they don't share any cause.

Assume we code the values of the three variables such that a value of "1" for *A* or *B* (the causes) will result in a value of 1 for *C* as well (the effect). For example: if *A* is trauma status, *B* is cancer status, and *C* is vital status, then *A*=1 denotes "trauma", *B*=1 denotes "cancer", and *C*=1 denotes "dead". Obviously, if *A*=1 (trauma) or *B*=1 (cancer), then *C*=1 (dead). The table below shows all permutations of the values of *A*, *B*, and *C*, which may be observed under these terms.

Values of the causes	Value of the effect
A=0	*C*=1
B=1	
A=1	*C*=1
B=0	
A=1	*C*=1
B=1	
A=0	*C*=0
B=0	

Recall that in our hypothetical story, *A* and *B* are independent (not associated). What will happen, however, after explicit conditioning on *C*? What happens to that independence, if we know, for example, that *C*=1 (restriction)? Well, things change. Now, knowing the value of one cause of *C*=1 might inform us about the value of the other cause. For instance, if we know that *A*=0, then *B*=1. Otherwise, what has caused *C* to take the value *C*=1? (If both *A*=0 and *B*=0, *C* must take the value *C*=0). Reverting to associational language, we see that a null association between *A* and *B* before conditioning, $\Pr(B=1|A=0)/\Pr(B=1|A=1)=1$, has turned into a non-null association after conditioning on *C*: $\Pr(B=1|A=0, C=1)=1$ whereas $\Pr(B=1|A=1, C=1)\neq 1$ (implying: probability ratio ≠ 1). To sum up, explicit conditioning on *C* (that is, knowing the value of *C*) resulted in dependence between *A* and *B*. The two causes of *C* are associated – conditional on *C*.

I used the triad "trauma-cancer-dead" as an example for A=1, B=1, and C=1, but anyone can make up other examples, provided they satisfy the diagram $A \rightarrow C \leftarrow B$. The next table shows several other triads that you can find in the literature on colliding variables. Pick some of them and tell yourself the story that I told you in the previous paragraph. Convince yourself that you can make some guesses about the value of B from the value of A and vice versa, provided you know the value of C. It is fairly simple.

Causes (A, B)	Effect (C=1)	Effect (C=0)
Sprinkler *Rain*	*Wet lawn*	*Dry lawn*
Burglary *Earthquake*	*Alarm sounds*	*Alarm doesn't sound*
Battery *Fuel*	*Car starts*	*Car doesn't start*
Aspirin *Statin*	*Alive*	*Dead*
Talent *Beauty*	*Success in Hollywood*	*Failure in Hollywood*
Grades *Musical talent*	*Admitted to graduate school*	*Not admitted to graduate school*

In search of theorems and proofs (or: the unsolvable citation maze)

I have a long list of questions, which might have crossed your mind, too: Heuristic arguments aside, where is the formal proof that conditioning on a collider creates or alter an association between the colliding variables? Are there any necessary or sufficient conditions for that statistical phenomenon? What happens if C takes three values, rather than two? Does it matter which measure of association is used? What is the relevance of the heuristic argument above to the consequences, for example, of adding a continuous collider to a linear regression model? In short, are there theorems on colliding bias that have been stated and proved?

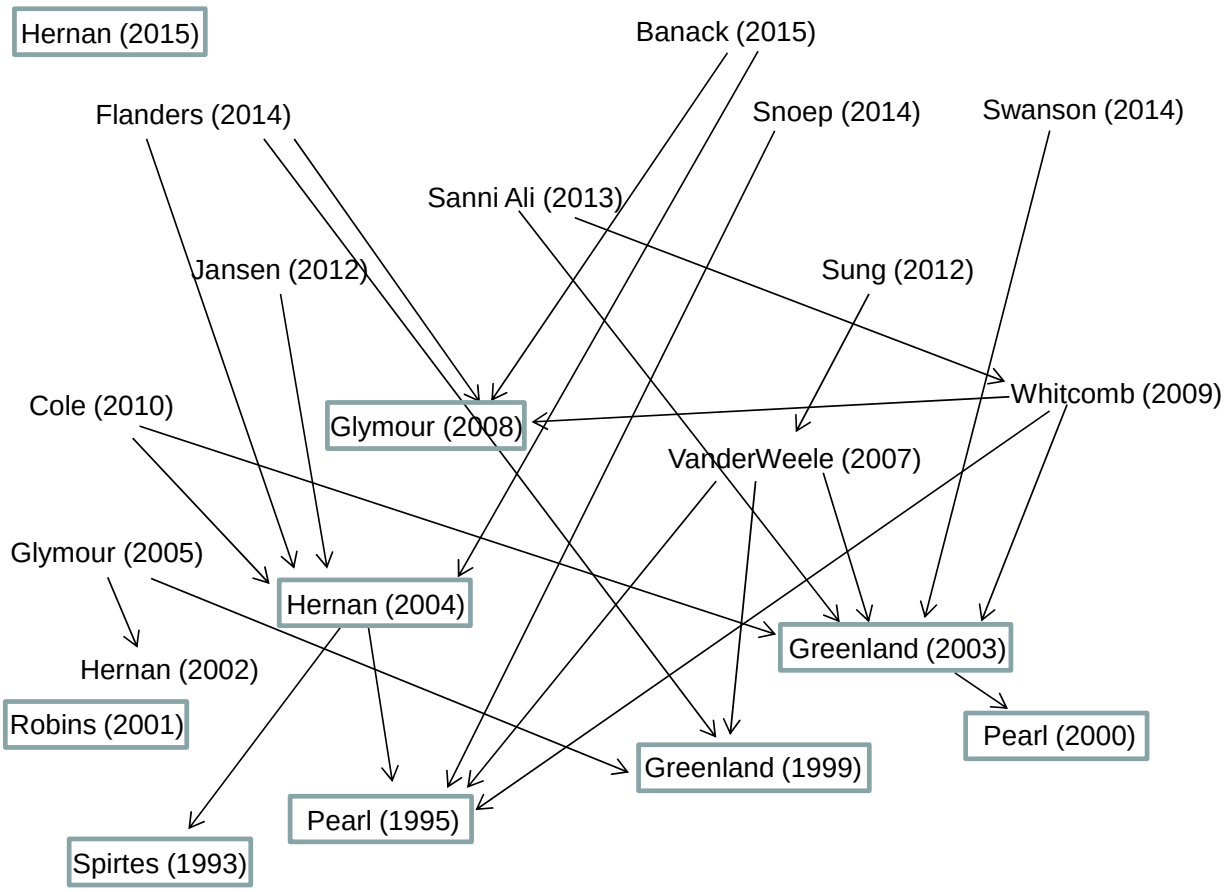
You are about to be surprised by the answers.

Theorems and proofs, in any domain, may be traced by a backward search of the literature. And that's what I did. Starting with recent publications (2012-2015) on colliding bias, in any of its names, I looked for key statements on the phenomenon, retrieved the associated citation(s), and repeated the process as needed. I also added some publications of which I was aware independently. Although the starting list

was neither exhaustive nor methodically sampled, some citation paths should have led to theorems and proofs. Sounds reasonable?

Figure 1 shows the maze of citations (causality works in reverse to the direction of the arrows). Most of the paths end after one generation or two; some publications hang alone. Publications in a box seem to be promising roots, so I will examine them closely: what they say and what they cite, if anything.

Figure 1.



Hernan (2015)

Hernan MA, Robins JM (2015): *Causal Inference* (August 27, 2015)

https://cdn1.sph.harvard.edu/wp-content/uploads/sites/1268/2015/08/hernanrobins_v1.10.30.pdf

(Accessed on January 14, 2016)

Page 75:

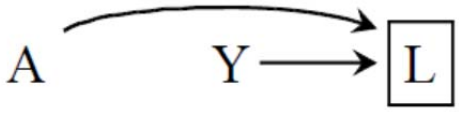


Figure 6.7

would perfectly predict the presence of the other. Graph theory shows that indeed conditioning on a collider like L opens the path $A \rightarrow L \leftarrow Y$, which was blocked when the collider was not conditioned on. Intuitively, whether

And again, a few lines below "graph theory shows":

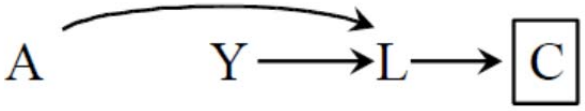


Figure 6.8

effect of A and Y. Graph theory shows that conditioning on a variable C affected by a collider L also opens the path $A \rightarrow L \leftarrow Y$. This path is blocked in the absence of conditioning on either the collider L or its consequence C.

Page 95:

their (conditioned on) common effect C. An analysis conditioned on C will generally result in an association between A and Y. We refer to this induced

Page 96:

According to the rules of d-separation, conditioning on the collider C opens the path $A \rightarrow C \leftarrow L \leftarrow U \rightarrow Y$ and thus association flows from treatment A

Page 106:

because L is a collider on that path. Thus, even if the causal effect of A on Y is null, the conditional (on L) risk ratio would be generally different from 1.

In summary,

- "graph theory shows" (page 75). No reference.
- "will generally result" (page 95). Only generally? No graph theory?
- "according to the rules of d-separation" (page 96). Graph theory has turned into rules
- "would be generally different" (page 106). Back to "generally".

So which is it? A proved theorem in graph theory? A rule made by an anonymous ruler? Or just wishy-washy "generally"?

Glymour (2008)

Glymour MM, Greenland S. Chapter 12: causal diagrams. In *Modern Epidemiology* (3rd edition), 2008

On page 187, we find a section heading on "rules":

RULES LINKING ABSENCE OF OPEN PATHS TO STATISTICAL INDEPENDENCIES

And in that section we find the following rule:

Page 188:

In contrast, conditioning on a collider requires reverse reasoning. If two variables X and Y are marginally independent, we expect them to become associated upon conditioning (stratifying) on a shared effect W. In particular, suppose we are tracing a path from X to Y and reach a segment on the path with a collider, $X \rightarrow W \leftarrow Y$. The path is blocked at W, so no association between X and Y passes through W. Nonetheless, conditioning on W or any descendant of W opens the path at W. In other words, we expect conditioning on W or any descendant to create an X–Y association via W. We thus come to the second criterion for blocking paths by conditioning on covariates:

- Conditioning on a collider W on a path, or any descendant of W, or any combination of W or its descendants, opens the path at W.

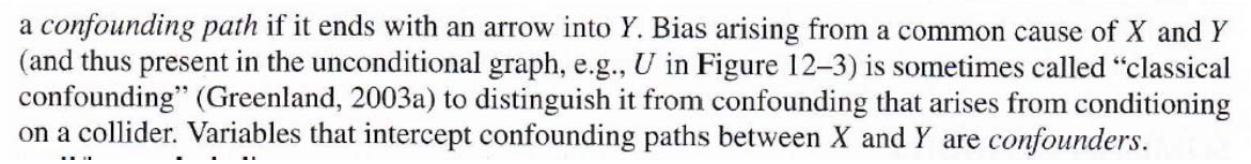
So, which is it? "conditioning…opens the path at *W*", or we only "expect conditioning on *W*…to create an *X*–*Y* association via *W*"? The first sounds like a theorem, whereas the second sounds like another version of "generally". No reference is cited here, but at the very beginning of the chapter (Introduction) we are told that the rules are "grounded in mathematics".

The graphical rules linking causal relations to statistical associations are grounded in mathematics. Hence, one way to think of causal diagrams is that they allow nonmathematicians to draw

And at the end of the introduction (page 184), we are referred to two references that provide "Full technical details of causal diagrams and their relation to causal inference": Pearl (2000) and Spirtes et al

(2001). Both books indeed contain lots of technical details, including theorems and proofs. Do they also contain theorems and proofs on the consequences of conditioning on a collider in every (or any) form of conditioning? If you find any, let me know the page number.

Unrelated, but noteworthy, is the pedagogical mistake of mixing colliding bias with confounding bias. Read the next two (complete) sentences in that chapter (page 193), starting at "Bias arising":


a *confounding path* if it ends with an arrow into Y. Bias arising from a common cause of X and Y (and thus present in the unconditional graph, e.g., U in Figure 12–3) is sometimes called "classical confounding" (Greenland, 2003a) to distinguish it from confounding that arises from conditioning on a collider. Variables that intercept confounding paths between X and Y are *confounders*.

"…confounding that arises from conditioning on a collider"? But the authors themselves called the phenomenon – in this chapter – "collider bias" (or "selection bias"), not "confounding". Where is the internal consistency? And then we also encounter – in the second sentence – a new definition of a confounder. No longer are confounders shared causes of the exposure and the disease; they are "Variables that intercept confounding paths". If you are confused, let me clarify what I think the authors are proposing:

In their terminology, there are two types of confounding bias: classical (e.g., *X*←*A*←*C*→*B*→*Y*) and "non-classical" (e.g., *X*←*A*---*B*→*Y*). In the first structure, the variables *A*, *B*, and *C* are all confounders. In the second structure, the variables *A* and *B* are confounders.

Do these definitions sound helpful, or might you prefer the following?

Confounding bias arises by a confounding path (e.g., *X*←*A*←*C*→*B*→*Y*). The variable *C*, a shared cause of *X* and *Y*, is a *confounder* – the culprit. A confounding path can be blocked by conditioning on the confounder (here, *C*), or on any other intermediary variable along the path (here, *A* or *B*).

Colliding bias arises by an induced, open path (e.g., *X*←*A*---*B*→*Y*). The path can be blocked by conditioning on any intermediary variable along the path (here, *A* or *B*).

Hernan (2004)

Hernan MA, Hernandez-Diaz S, Robins JM. A structural approach to selection bias. *Epidemiology* 2004;15:615-625

Four arrows in my maze of citations enter this publication. Here is what we find there:

Page 615:

causal structure in epidemiologic settings.[6–12] In fact, the structure of bias resulting from selection was first described in the DAG literature by Pearl[13] and by Spirtes et al.[14] A DAG is

Page 616:

We now provide intuition for why structure (3) induces a conditional association. (For a formal justification, see references 13 and 14.) In Figure 3, the genetic haplotype E and

FIGURE 3. Conditioning on a common effect C of exposure E and outcome D.

13. Pearl J. Causal diagrams for empirical research. *Biometrika*. 1995;82: 669–710.
14. Spirtes P, Glymour C, Scheines R. *Causation, Prediction, and Search. Lecture Notes in Statistics 81*. New York: Springer-Verlag; 1993.

The phrase "formal justification" should mean "theorems and proofs". If not, I fail to grasp its precise meaning. References 13 and 14 (shown above) are promising roots in my maze of citations, so they will be examined later.

Greenland (2003)

Greenland S. Quantifying biases in causal models: classical confounding vs collider-stratification bias. *Epidemiology* 2003;14:300-306

example, if X and Y are marginally independent (*ie*, unassociated before stratification), then they will be associated within at least one stratum of a variable that they both affect.[1(pp17)]

1. Pearl J. *Causality*. New York: Oxford, 2000.

That's a statement that sounds exactly like a theorem – actually, a strong theorem ("within at least one stratum"). So reference "1(pp17)" should provide a proof. What do we find there? That's coming up next.

Pearl (2000)

Pearl J. *Causality: models, reasoning, and inference*. Cambridge University Press, New York, 2000

Here is the relevant text on page 17 to which we are referred in Greenland (2003):

given m. Inverted forks $i \rightarrow m \leftarrow j$, representing two causes having a common effect, act the opposite way; if the two extreme variables are (marginally) independent, they will become dependent (i.e., connected through unblocked path) once we condition on the middle variable (i.e., the common effect) or any of its descendants. This can be confirmed in the context of Figure 1.2. Once we know the season, X_3 and X_2 are independent (assuming that sprinklers are set in advance, according to the season); whereas finding that the pavement is wet or slippery renders X_2 and X_3 dependent, because refuting one of these explanations increases the probability of the other.

That's the familiar heuristic argument. And here it is again:

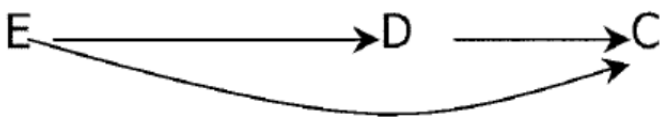
At first glance, readers might find it a bit odd that conditioning on a node not lying on a blocked path may unblock the path. However, this corresponds to a general pattern of causal relationships: observations on a common consequence of two independent causes tend to render those causes dependent, because information about one of the causes tends to make the other more or less likely, given that the consequence has occurred. This pattern is known as *selection bias* or *Berkson's paradox* in the statistical literature (Berkson 1946) and as the *explaining away effect* in artificial intelligence (Kim and Pearl 1983). For example, if the admission criteria to a certain graduate school call for either high grades as an undergraduate or special musical talents, then these two attributes will be found to be correlated (negatively) in the student population of that school, even if these attributes are uncorrelated in the population at large. Indeed, students with low grades are likely to be exceptionally gifted in music, which explains their admission to graduate school.

In short, no shred of proof on page 17 of reference 1. Just the usual heuristic explanation. And if you find a relevant proof elsewhere in the book, let me know the page number.

Robins (2001)

Robins JM. Data, design, and background knowledge in etiologic inference. *Epidemiology* 2001;11:313-320.

Page 315



DAG 4

Page 316 (referring to DAG 4)

intercept C. This example tells us that if we condition on a common effect C of two independent causes *E* and *D*, we "usually" render those causes conditionally dependent. For instance, if we know a subject has the outcome C (that is, we condition on that fact) but does not have the disease *D*, then it usually becomes more likely that the subject has the exposure *E* (because we require some explanation for his or her having C). That is, among subjects with the outcome C, *E* and *D* are "usually" negatively associated (have an odds ratio less than 1).

The reason we included the word "usually" in the above is that although CMA allows one to deduce that d-separation implies statistical independence, it does not allow one to deduce that d-connection implies statistical dependence. However, d-connected variables will gen-

By the way, the explanation here for the word "usually"(in "usually negatively associated") has nothing to do with the case of explicit conditioning on *C* (restricting *C* to one value).

Page 317 (referring again to the previous diagram)

> Consider next DAG 4. There are no unmeasured common causes of E and D. As discussed in Example 2 above, under the causal null hypothesis of no arrow from E to D, E and D will be independent. It follows that the marginal association OR_{ED} is unconfounded and represents the causal effect of E on D. In contrast, the conditional association $OR_{ED|C}$ will be confounded and thus will not be equal to the causal effect of E on D within strata of C, because we showed in Example 2 that, under the causal null of no arrow from E to D, E and D will be conditionally associated within strata of C. This example shows that conditioning on a common effect C of E and D introduces confounding within levels of C. This example also shows why, to check for

Four points may be made on the two paragraphs:

First, the example is the heuristic argument again. No proof. No reference.

Second, the theorem-like claim has been extended. It is not "within at least one stratum" (Greenland 2003), but "within levels of *C*" (plural), which means "at least two strata" or perhaps "in all strata"?

Third, "This example tells us" and "This example shows" must be a new method of proving a theorem in statistics. I used to think that an example can only logically disprove a general claim (the so-called counter-example).

And lastly, "… conditioning on a common effect *C* of *E* and *D* introduces confounding within levels of *C*" is another example of mixing the terms "colliding bias" and "confounding bias".

Greenland (1999)

Greenland S, Pearl J, Robins JM. Causal diagrams for epidemiologic research. *Epidemiology* 1999;10:37-48

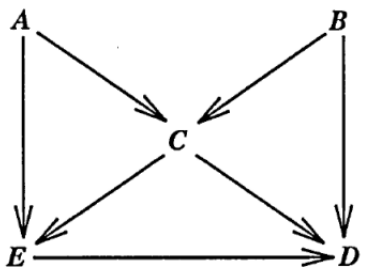


FIGURE 1.

Page 41 (referring to Figure 1 above):

There is, however, a chain of reasoning that would have allowed us to answer the question correctly. First, A and B have no marginal (crude) association under Figure 1, because A and B share no common ancestor that would produce covariation between them. Second, A would have to be associated with C given B because A affects C; similarly, B would have to be associated with C given A. Thus, when we stratify on C, the association of A and B within at least one stratum of C will almost certainly differ from the crude A-B association; in particular, it will almost certainly not be null in all strata of C, or in an overall summary across strata of C. (By "almost certainly," we mean that exceptions can occur in special examples involving perfect cancellations across strata of a polytomous C.[18])

18. Whittemore AS. Collapsibility in multidimensional contingency tables. J R Stat Soc B 1978;40:328–340.

First, I fail to understand the chain of reasoning. How do the associations of C with A and B lead to "Thus, when we stratify on C, the association of A and B within at least one stratum of C will almost certainly differ from the crude A-B association"?

Second, it is not clear to me why "almost certainly" was placed before "not be null in all strata pf C", when it is explained (in parentheses) that "almost certainly" refers to "an overall summary across strata of C". Reference 18, by the way, predates the literature on causal diagrams and was not motivated by stratification on a shared effect. At any rate, the collapsibility theorems in reference 18, although generic, are not theorems about the consequences of restricting a collider to a single value.

Next comes a numerical example and then, the following text:

Page 41:

The preceding example corresponds to a general rule about causal relations: Suppose two variables A and B both affect a third variable C (that is, C is a descendant of A and B). Then the association of A and B within strata of C will almost certainly differ from the marginal association of A and B. This rule has long been recognized in epidemiologic problems in which the effect of A on B is of interest[7,13,15]; it is known as the "explaining away" effect in artificial-intelligence research.[19] Apply-

So now "the association of A and B within strata of C will almost certainly differ from the marginal association of A and B". Is it "almost certainly within strata", or "almost certainly within at least one stratum"? Or just "certainly within at least one stratum"? What is the presumed theorem? Or maybe it is just "a rule" that "has long been recognized in epidemiologic problems"? One thing is certain: all of these imprecise statements could not have originated from any published, proven theorem(s). Moreover, if there was a proven theorem, we would undoubtedly have seen a reference.

Pearl (1995)

Pearl J. Causal diagrams for empirical research. *Biometrika* 1995;82:669-710

No text is shown here, simply because there is no relevant text to show. There is a lot on confounding bias in that article (e.g., section 3 "Controlling Confounding Bias"), and the theorems for a sufficient set for conditioning inevitably take into account the phenomenon of colliding bias. But there is nothing explicit on colliding bias (or "selection bias", or "collider bias", or "explaining away effect"). In fact, none of these terms is even mentioned. Since the key theme of the article is "identifiability" – conditioning that allows bias-free estimation of an effect, given a graph – it is not surprising that there are no theorems on the consequences of conditioning on a collider. The word "collider" is nowhere to be found in that article (although colliders are shown in Section 5.3, Figure 7, where non-identifiable models are described.)

Spirtes (1993)

Spirtes P, Glymour C, Scheines R. *Causation, Prediction, and Search*. 1993. (online PDF; accessed on January 29, 2016)

https://www.cs.cmu.edu/afs/cs.cmu.edu/project/learn-43/lib/photoz/.g/scottd/fullbook.pdf

On page 68 we find the basic diagram of a collider (essentially $A \rightarrow B \leftarrow C$) followed by the text below (section 3.5.2): "

> Judea Pearl (1988) offers a Bayesian example that illustrates why, when a causal structure like that in graph (ii) obtains, one should expect that A and C, though independent, are dependent conditional on B: Whether or not your car starts depends on whether or not the battery is charged and also on whether or not there is fuel in the tank, but these conditions are independent of one another. Suppose you find that your car won't start, and you hold in that case that there is some probability that the fuel tank is empty and some probability that the battery is dead. Suppose next you find that the battery is not dead. Doesn't the probability that the fuel tank is empty change when that information is added?

" That's a citation of the heuristic argument in a 1988 publication. No theorem, no proof.

And then on page 72, we find reference to the text above as follows:

"That conditioning on a collider makes it active was noted in section 3.5.2 above."

The book contains many proofs of theorems and lemmas on causal diagrams, with special emphasis on the faithfulness condition. Is there also a direct proof of a theorem on colliding bias? The notation is heavy, but I did not recognize one.

My writing

A small surprise at the end. I used to belong to the majority that believes that "brilliant minds must have proven the fundamentals". So, I also contributed my share to the claims about theorems. Here are two examples:

Shahar E, Shahar DJ. Causal diagrams and the logic of matched case-control studies. *Clinical Epidemiology* 2012;4:137-144 [Corrigendum: *Clinical Epidemiology* 2014;6:59]

Page 138:

The theorems of causal diagrams build a solid bridge between a causal structure and expected associations. Both

Yes, there are quite a few theorems on causal diagrams and expected dependence/independence between displayed variables. But are there stated theorems on the consequences of conditioning on a collider? If so, why do we encounter repeated heuristic explanations? Why does the maze of citations not end in a common reference of a theorem and a proof?

Page 138

over each arrow. But more might happen: under certain conditions,[2] new associations (denoted by dashed lines) will be created between variables that collide at S (that is, between causes of S). As a result, we observe new

And here is reference 2, followed by the relevant text:

Shahar E, Shahar DJ (2012): Causal diagrams and three pairs of biases. In: *Epidemiology – Current Perspectives on Research and Practice* (Lunet N, Editor). www.intechopen.com/books/epidemiology-current-perspectives-on-research-and-practice, pp. 31-62

Pages 37-38

As we have seen, conditioning on a confounder removes confounding bias. In contrast, conditioning on a collider sometimes *adds* bias. After conditioning on C (Figure 5, Diagram B), the colliding variables E and D will be associated not only due to the effect $E \rightarrow D$, but also due to a newly-formed association between them (E---D). Consequently, the estimator contains a new type of bias—colliding bias. How much bias will be added, and whether bias will be added at all, depends on several factors: the value to which C is restricted; effect-modification between E and D on the probability ratio scale; and the type of post-conditioning analysis (a weighted average, regression, or none). Unlike confounding bias, some rules that govern colliding bias depend on the scale and analysis by which effects are estimated.

Why does conditioning on a collider sometimes create an association between the colliding variables? (And why does a confounder create an association between its effects?) Formal proofs are available, but we will just provide intuitive explanations. Considering

"Formal proofs are available" on colliding bias? I assumed so – carelessly accepting published statements such as "For a formal justification, see references…" and "graphical rules…grounded in mathematics". In my defense (or rather, to my discredit), I did not cite any external reference.

In part 3, I hope to revisit the topic of effect modification (mentioned above) as a prerequisite for a newly formed association after conditioning on a collider. That's quite interesting (and possibly a theorem for some types of conditioning).

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Colliding bias (part 3): what theorems should we look for? (forthcoming)