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Comment on "Causal inference, probability theory, and graphical insights" by Stuart G. Baker

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Modern causal inference owes much of its progress to a strict and crisp distinction between probabilistic and causal information. This distinction recognizes that probability theory is insufficient for posing causal questions, let alone answering them, and dictates that every exercise in causal inference must commence with some extra knowledge that cannot be expressed in probability alone.[1] The paper by Baker attempts to overturn this distinction and argues that "probability theory is a desirable and sufficient basis for many topics in causal inference." My comments will disprove Baker's claim, in the hope of convincing readers of the importance of keeping the boundaries between probabilistic and causal concepts crisp and visible.

Baker's argument begins with: "...besides explaining such causal graph topics as M-bias (adjusting for a collider) and bias amplification and attenuation (when adjusting for instrumental variable), probability theory is also the foundation of the paired availability design for historical control" (abstract). While I am not versed in the intricacies of "paired availability design" (Google Scholar lists only a handful of entries in this category), I doubt it can be based solely on probabilities. Indeed, Baker himself resorts to counterfactuals and other non-probabilistic notions[2] in explaining the research questions a "paired availability design" attempts to answer. I am quite familiar however with the concepts of "M-bias," "bias," "Simpson's paradox," and "instrumental variable" which I will show to have no interpretation in probability theory alone.

I will start with the concept of "instrumental variable" which should be familiar to most readers, and which is often mistaken to have probabilistic definition (see [2, pp.

[1] Cartwright [1] summarized this limitation in a well-known slogan: "no causes in, no causes out."

[2] By "non-probabilistic notions" I mean relations or parameters that cannot be defined in terms of joint distributions of observed variables. The restriction to observed variable is important for, otherwise, everything would become probabilistic, including Cinderella's age, horoscopic predictions, counterfactuals, latent variables, the answers to our research questions, and so on; we need merely hypothesize a distribution over such variables and turn every problem probabilistic. The distinction between causal in probability information would then lose its meaning and usefulness.

387–389]). Assume we have a joint distribution $P(x, y, z)$ defined on three variables X, Y, and Z. We ask: What condition should P satisfy in order for Z to qualify as an instrumental variable relative to the pair (X, Y). It is well known that, if X is continuous, no such condition exists. That is, for any causal model in which Z is an instrument there exists another model, indistinguishable from the first, in which Z is not an instrument.[3] Baker's Fig. 1(d), for example, where Z is in an instrument, cannot be distinguished (by measuring X, Y, and Z) from one to which an arrow $Z \to Y$ is added, thus rendering Z no longer an instrument. This demonstrates that an instrument, Z, cannot be defined probabilistically in terms of $P(X, Y, Z)$.

One may attempt to escape this demonstration by defining an instrument relative to a triplet (X, Y, U) rather than a pair, where U is a "confounder" (as in Baker's Fig. 1(d)). This, too, must fail. First, the concept of "confounder" is causal, and defies all definitions in probabilistic terms [2, Ch. 6; 3]. Second, consider Fig. 1(e) to which we add an arrow $Z \to Y$. The newly formed graph is "complete" (i.e., all pairs are adjacent) and, therefore, can generate any probability distribution $P(x, y, z, u)$ whatsoever. In other words, this complete graph, in which Z is not an instrument, can emulate any model that characterizes Z as an instrumental , such as the one in Fig. 1(d). We conclude, again, that an instrument cannot be defined in probabilistic terms, regardless of whether it is defined relative to a pair or a triplet.

Similar demonstrations can be used to show that "M-bias," and in fact any concept invoking the notion of "bias" cannot admit a definition in probability theory.[4] This raises the question why Baker's analysis may give one the impression that probability theory in itself can explain M-bias and bias amplification. The answer is, Baker's analysis merely re-validates (not "explains") known algebraically probabilistic aspects of these phenomena, while the conclusions rely crucially on causal information that is kept out of the analysis. Appendix A, for example, merely shows that, given a probability function compatible with the graph $X \leftarrow Q \to U \leftarrow R \to Y$, conditioning on U would create dependency between otherwise two independent variables, X and Y. This follows directly from d-separation, a probabilistic tool that has nothing to do with causation or with "bias." To show that conditioning on M creates bias one must give causal interpretation to the M-graph, and invoke the truncated product interpretation of interventions [2, pp. 22-24]; probabilistic interpretations in themselves remain insufficient.

The litmus test for classifying M-bias as "probabilistic" or "causal" is clear and crisp; given a joint distribution $P(X, Y, U, W, Q, R)$ can we determine (from P alone) whether adjustment for U produces bias on not? If the answer is yes, we have a probabilistic question on our hand; if the answer is no, and we find ourselves needing the causal graph to decide, we conclude that the question is not probabilistic but causal. In our case, the latter holds – we must examine the causal graph before deciding whether adjustment for M would introduce bias.

The same applies to Baker's treatment of Simpson's paradox; Appendix A does

[3] Weak inequality constraints may bound P when X is discrete [2, pp. 274–275] but, otherwise, the fact that Z is an instrument cannot be recognized in P.

[4] "Bias" is defined as the difference between the desired quantity, say the causal effect, and a quantity that is estimated from observed data. Since the former is causal, so is "bias."

not provide a proof that adjustment on U is not appropriate; no proof based solely on probability theory can deal with the question of "appropriateness" or decide that adjusting for U in Fig. 1(a) is appropriate while in Fig. 1(c) it is not appropriate. Such distinction comes from the causal information conveyed by the arrows, not the conditional independencies conveyed by those arrows; all attempts to explain the paradox without invoking causation have failed (see [2, Ch. 6; 4]).

Lastly, I am surprised by Baker's claim that the "paired availability design does not fit into a causal graph framework" (Baker, Section 5). The description of the "paired availability design" given in this paper, is formulated in terms of "principal strata" – a counterfactual framework that fit perfectly and, in fact, is subsumed by the causal graph framework (see [2, Ch. 8; 5–7]). A structural causal model represents *all* counterfactuals that may possibly be defined among the variables in the model [8] and, therefore, subsumes any design based on these counterfactuals.[5] The symbiosis between graphs and counterfactuals is much tighter than what Baker's paper presents, and has led to major advances in problem areas such as mediation analysis, external validity, heterogeneity and missing data (see http://bayes.cs.ucla.edu/csl_papers.html).

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[5] This subsumption was usually ignored in the potential outcome framework [9, 10] and is proven mathematically in [11, 12].

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