Causal inference without the consistency rule, definition, assumption, theorem, axiom, or whatever

In 2009 Cole and Frangakis published an article (titled Commentary) in which they concluded that "the consistency rule" is actually an assumption [1]. VanderWeele, in a follow-up article (strangely titled Brief Report), concurred and elaborated [2]. A year later, Pearl published a counter viewpoint (also titled Brief Report) in which he concluded that the consistency assumption is a theorem [3]. All four authors claim that consistency is required to draw causal inference.

I argue here that the consistency thingamajig (rule, definition, theorem, whatever) is neither an assumption, nor a theorem, nor a relevant idea. Everything that is captured by the so-called consistency and inconsistency can be stated as theories about effect modification [4], effect-modification bias [5], thought bias [6], or information bias [6]. Not surprisingly, some of these ideas don't dwell well with authors who subscribe to deterministic causation.

For readers who are not familiar with the consistency-thingamajig, here are some of the explanations that were offered by those authors. No, it is not the intuitive sense of the word.

Cole and Frangakis wrote:

> "The consistency assumption is often stated such that an individual's potential outcome under her observed exposure history is precisely her observed outcome.[4]"

Citing Cole and Frangakis, VanderWeele wrote:

> "Cole and Frangakis define $Y_j(x, k)$ as the potential outcome for individual j if exposure X is set to value x by means k. They let X_j and Y_j^{obs} denote, respectively, the observed values of X and Y for individual j. The consistency assumption is then reformulated as the assumption that "$Y_j^{obs} = Y_j(x, k)$ if $x = X_j$, no matter the value of k."

Pearl wrote:

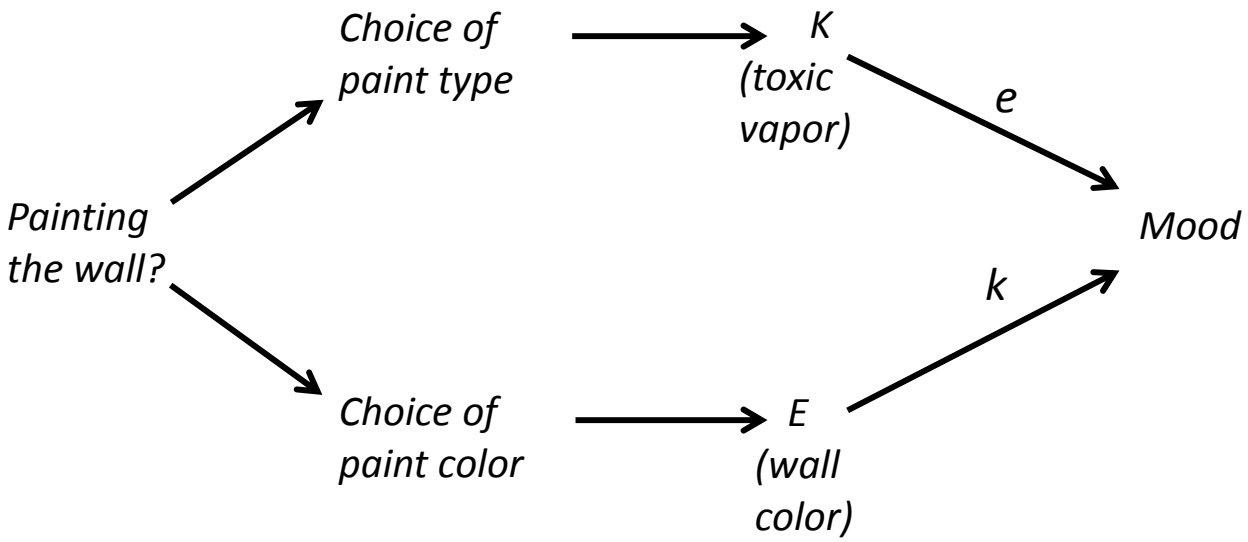
> "Informally, the consistency rule states that an individual's potential outcome under a hypothetical condition that happened to materialize is precisely the outcome experienced by that individual. When expressed formally, this rule reads[1]:
>
> $$X(u) = x \Rightarrow Y_x(u) = Y(u)$$
>
> where, $X(u)$ stands for the exposure that individual u actually experienced; $Y_x(u)$, the potential outcome of individual u had the exposure been at level $X = x$; and $Y(u)$ is the outcome actually realized by u."

Why might the observed outcome not be consistent with the potential outcome? Abstract ideas are best explained by examples, and Pearl offered one. He considered the statement "If we paint the wall red my uncle will be cheerful", or its equivalence in counterfactual terms: "If the wall were red, my uncle would be cheerful." The expected cheerful mood of the uncle is the potential outcome above, $Y_x(u)$, whereas the actual mood of the uncle is the realized (observed) outcome, $Y(u)$. The uncle might not be cheerful at all if the paint discharged toxic vapor, for example, which means that the consistency-thingamajig does not hold: $Y_x(u) \neq Y(u)$.

Pearl drew a causal diagram in which he showed that the so-called consistency problem resulted from failure to display the effect of toxic vapor on mood—hardly surprising.

Here is my version of Pearl's diagram:

Figure 1. A causal diagram: the effect of wall color and toxic vapor on mood.



(You may add an arrow from "choice of paint color" to "toxic vapor", or from "choice of paint type" to "wall color".)

The lower case letters above two of the arrows convey a theory of effect modification between two causes of the outcome [4]. The effect of wall color (*E*) on mood depends on the value (*k*) of toxic vapor. Given reciprocity of effect modification [4], the effect of toxic vapor (*K*) on mood depends on the value (*e*) of wall color. (It's not that funny: people can feel elated by a sight, even when they are physically uncomfortable. Ask hikers.)

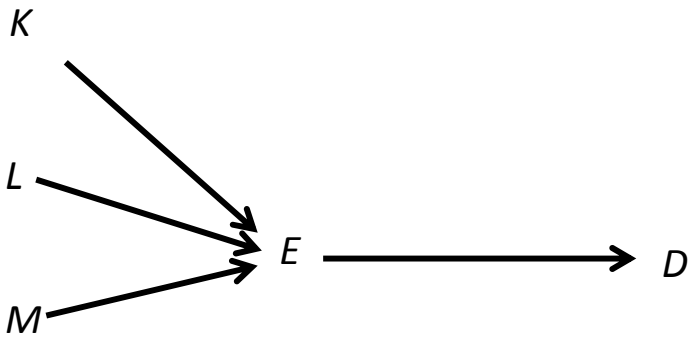
If the diagram depicts causal reality, wall color does not have a single effect on mood, and anyone who infers a single effect ("my uncle will be cheerful") is committing two mistakes: First, such a claim cannot be made under indeterminism. Second, the estimator contains effect-modification bias [5]—an idea that deterministic authors choose to ignore.

Cole and Frangakis wrote:

"Consistency is plausible in observational studies of medical treatments, because one can imagine how to manipulate hypothetically an individual's treatment status. However, consistency is problematic in observational studies with exposures for which manipulation is difficult to conceive. Consistency is especially difficult when the exposure is a biologic feature, such as body weight, insulin resistance, or CD4 cell count.[5,6] For example, there are many competing ways to assign (hypothetically) a body mass index of 25 kg/m^2 to an individual, and each of them may have a different causal effect on the outcome."

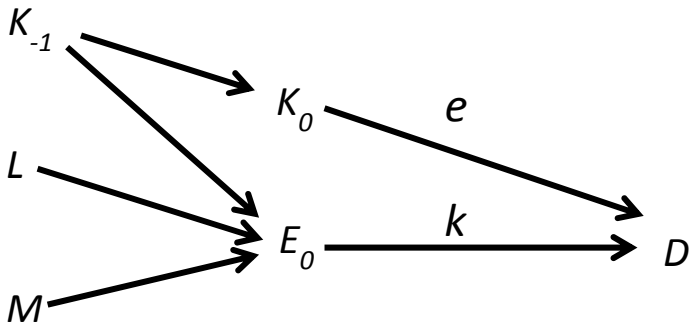
Why does it matter that "there are many competing ways to assign [the value of the exposure variable]"? Ways to influence the value of the exposure are simply causes of the exposure variable, as the figure below shows. And why are they "competing"? Are causes mutually exclusive?

Figure 2. "Competing ways" to assign the value of the exposure: a causal structure



Under this causal structure *K*, *L*, and *M* might have different effects on *D*, but the effect of *E* on *D* is indifferent to the causes of *E*. To claim that the effect *E*→*D* depends on *K*, for instance, is to propose the following structure:

Figure 3. "Competing ways" to assign the value of the exposure variable: a different causal structure



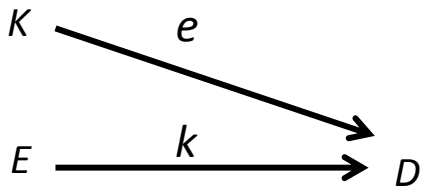
But this structure depicts confounding, which is violation of exchangeability (another requirement for unbiased estimation). Once confounding is removed (by conditioning on K_{-1}), the problem of consistency translates, again, to a theory of effect modification between E_0 and K_0 (Figure 3).

Cole and Frangakis provide an example of "departures from consistency".

> "Many examples of departures from consistency can be viewed from the perspectives of study design or exposure measurement error. For instance, the causally relevant window for postmenopausal hormone therapy exposure to provide protection against cardiovascular disease[22] may be viewed as a consistency issue."

Not at all. The so-called example of departure from consistency is an example of effect modification. The effect of hormones (*E*) might vary by age (*K*), or whatever post-menopausal age is measuring, as shown in Figure 4. It is as simple as that.

Figure 4. The effect of hormones (*E*) varies by age (*K*)



As described so far, consistency is no more than a claim that the study is free from effect-modification bias [5]. Rather than willingness to condition on modifiers, it is strangely assumed that no modifiers are left after some conditioning that is needed to satisfy exchangeability.

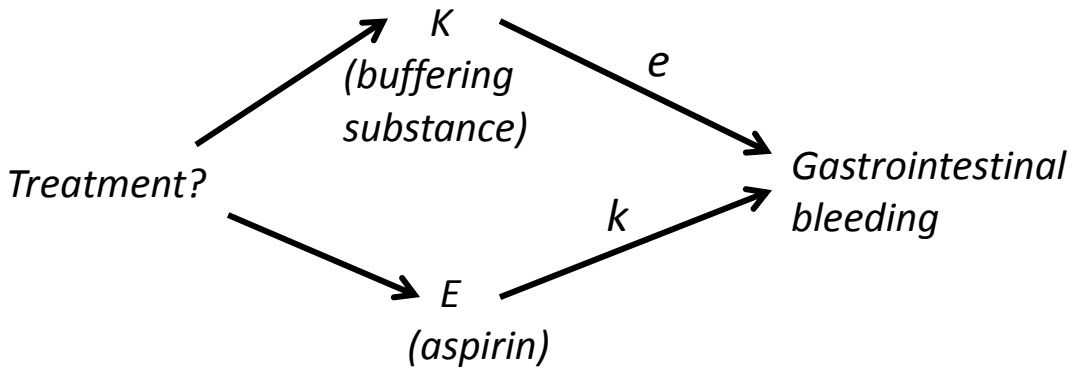
Cole and Frangakis wrote:

> An informal definition of consistency is "have I defined exposure to include the causally relevant features?

Question: What do definitions have to do with the study of causal reality?
Answer: Human-made definitions of variables illustrate thought bias [6]. To make up a variable from "causally-relevant features" (i.e., causal variables) is thought bias [6].

Cole and Frangakis present an example. Discussing the effect of aspirin on the gastrointestinal tract (say, on bleeding), they argue that the consistency thingamajig has to do with explicit "definitions" of the exposure (e.g., is it plain aspirin or buffered aspirin?). They fell into the trap of replacing causal reality with human definitions. You don't "define" the cause as the combination of the values of *E* and *K*. This would be analogous to "treatment regime"—another example of thought bias [7]. Their example corresponds to a theory of effect modification (Figure 5): the effect of aspirin (variable *E*) on gastrointestinal bleeding depends on the amount of the buffering substance (variable *K*).

Figure 5. A causal diagram: aspirin and buffering substance



Finally, Cole and Frangakis also consider the dose of a drug as part of the consistency problem. They wrote:

"For example, x may be use of aspirin or not. Then k includes the number of doses/day, milligram/dose, and whether the aspirin was buffered or not. Such variations can be included in the "technical errors" discussed by Rubin.[7]"

Indeed, the error in question is technical, not some problem of consistency. The technicality has to do with information bias. If the causal variable is the dose of aspirin, for example, then imputing the dose by a binary variable (use of aspirin or not) is a trivial example of information bias.

In summary, the consistency thingamajig is a bundle of biases rolled into one. First, it is the assumption that effect-modification is absent (which is strange by itself because effect modification plays a bizarre role in determinism.) Second, the claim that exposure needs to be "defined" either leads to thought bias or illustrates misunderstanding of information bias.

The consistency-thingamajig? Another much ado about (almost) nothing.

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References:

1. Cole SR, Frangakis CE. The consistency statement in causal inference: a definition or an assumption? *Epidemiology* 2009 Jan;20(1):3-5.

2. VanderWeele TJ. Concerning the consistency assumption in causal inference. *Epidemiology* 2009 Nov;20(6):880-3.

3. Pearl J. On the consistency rule in causal inference: axiom, definition, assumption, or theorem? *Epidemiology* 2010 Nov; 21(6): 872-5.

4. Shahar E, Shahar DJ. On the definition of effect modification. *Epidemiology* 2010 Jul;21(4):587.

5. Shahar E, Shahar DJ. 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, 2012:pp. 31-62.

6. Shahar E, Shahar DJ. Causal diagrams, information bias, and thought bias. *Pragmatic and Observational Research* 2010;1:33–47.

7. Shahar E, Shahar DJ. Marginal structural models: much ado about (almost) nothing. *Jornal of Evaluation in Clinical Practice* 2013 Feb;19(1):214-22.