

## Being Precise about Precision and One-to-One Specificity

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**Abstract:** Following from my criticism of Calcott's analysis of the permissive/instructive distinction, I rebut his claims that 1) he clarifies my measure of one-to-one specificity; 2) for all intents and purposes of his analysis, his notion of precision is different from my measure of one-to-one specificity; and 3) the Waddington box is a better and different model than the extension of Woodward's radio I propose.

### Main Text

In Bourrat (2019a), I showed two things. First, I demonstrated that Calcott (2017) did not link his analysis to Woodward's (2010) notion of one-to-one specificity. Instead, Calcott argued that his notion of precision is not captured by Woodward's analysis of specificity. This claim surprised me as I saw a clear link between precision and one-to-one specificity, one of Woodward's two dimensions of causal specificity. This led me to confront Calcott's claim that the notion of specificity cannot be used to discriminate some cases where a cause has a precise effect from other situations where a cause can have a range of effects. Based on one of Calcott's own examples, I showed, *contra* his claim, that if by 'specificity' one means 'one-to-one specificity,' these cases can be distinguished when an adequate information-theoretic measure known as 'variation of information' is used. In light of this, I then proposed that the permissive/instructive distinction can be explained away by considering the two dimensions of specificity proposed by Woodward: (1) the degree of fine control a cause exerts on its effect, or INF—measured by mutual-causal information—and (2) the extent to which to one causal value corresponds exactly to one causal effect, or one-to-one specificity—measured by variation of causal information.<sup>1</sup>

In his response to my criticism, Calcott (2019) argues three things: (1) that my measure of one-to-one specificity 'goes awry'; (2) that, contrary to what I claim, the two dimensions of causal specificity proposed by Woodward do not permit capturing the permissive/instructive distinction; and (3) that extending Woodward's radio to show that it is equivalent to the Waddington box is inadequate.

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<sup>1</sup> As a side note, Calcott claims that I have used not two but three measures of specificity (2019, p. 4). I never proposed a third measure but rather claimed multiple times that there are two dimensions for the notion of causal specificity and that, to obtain a fuller picture of a given situation, it is interesting to measure specificity in both dimensions. I take this to be different from proposing a third measure.

With respect to the first point, Calcott spends more than a third of his response showing that by subtracting my measure of one-to-one specificity to 1, one can link it to mutual-causal information and that this allows us to discuss ‘specificity’ rather than ‘(un)specificity,’ a term I used to link my measure to one-to-one specificity. This is quite disconcerting—I myself drew, albeit in a different way, the direct link between the two measures as it is explicitly stated in equation (1) and (2) in Bourrat (2019a) and Bourrat (2019b), which Calcott himself cites. The relationship between mutual information and variation of information is stated anywhere the definition of variation of information is presented. However, Calcott presents his ‘alternative’ measure as if I did not draw this link, omitting any mutual information term when he defines variation of information, while they were present in my definitions.

Numerous measures related to mutual information can be produced. The real question is whether anything is clarified by Calcott’s alternative measure. The answer is a resounding ‘no,’ unless stating that a glass is half empty clarifies the statement that the same glass is half full. Thus, although I gladly accept that Calcott’s proposition is more elegant than mine, in no way does his analysis clarify, correct, or represent an alternative to mine.

With respect to the second point, I will not repeat my analysis here; instead, I will make several remarks clarifying why one-to-one specificity amounts to precision in Calcott’s examples. First, let me note that Calcott proposes that a permissive cause may be seen as a ‘canalised switch’ (2017, p.495). I certainly agree that *some* permissive causes can be regarded as such and that Calcott’s analysis might be targeted at those type of causes, but this does not represent the consensus on what a permissive cause is. This should be obvious from the different quotations I provided in Bourrat (2019a, Table 1). For the most part, the biologists I cite use the distinction in the way I do, where ‘permissive’ can be equated with ‘background condition.’ This is perhaps a hint that the permissive/instructive distinction has more than one meaning and is, thus, more elusive than Calcott seems to believe. This remark aside, I clearly stated in my criticism that my main target was Calcott’s claim that the notion of precision is not captured by Woodward’s analysis—when in fact it is. One crucial point to note is that Calcott’s notion of precision lies at the core of his analysis of the permissive/instructive distinction. Surely, showing that his notion of precision is captured by specificity would show that Calcott’s analysis is partly mistaken. Importantly, Calcott claims that his measure of precision and my measure of specificity are related but different. If, by ‘different,’ Calcott means that his measure is a component of mine but that this difference plays no role in the examples he uses, then we agree—but, once again, this is stretching what the word ‘different’ means. Indeed, there is a straightforward way to show why, for all intents and purposes, in Calcott (2017), the two measures are equivalent.

In using his Waddington box, Calcott (2017, p. 493-495) contrasts two settings,  $p_3$  and  $p_4$ , and claims that a measure of specificity cannot account for their difference. The outcome is indeterministic (or fuzzy) in  $p_3$  while it is deterministic (or precise) in  $p_4$ . Calcott (2017, fn. 7) claims what is distinct about  $p_4$  is that the conditional entropy of the effect on the cause is nil or, more formally, that  $H(E|\hat{C}) = 0$ . I recall here that one condition for maximal one-to-one specificity I propose in Bourrat (2019a, 2019b), using variation of causal information, is  $VI(E; \hat{C}) = H(E|\hat{C}) + H(\hat{C}|E) = 0$ . Thus, Calcott’s measure is a component of mine.

Now, in his examples  $p_3$  and  $p_4$ , Calcott assumes that the number of states for the cause (his  $S$  variable) is lower than the number of states for the effect (his  $B$  variable) *and* that one state of the effect cannot be produced by more than one state of the cause. This necessarily implies that  $H(\hat{C}|E) = 0$ . Thus, for the purpose of comparing the *only* situations proposed by Calcott—which

are supposed to present biological situations—our conditions are perfectly equivalent. However, in some biologically realistic situations, such as when the same effect can be produced by two distinct causes that produce no other effects, his measure of precision would only tell us that each cause has a precise effect. Based on this criterion alone, using Calcott's measure,  $p_3$  would be indistinguishable from  $p_4$ . Conversely, with the measure I proposed, the two would be distinguishable, which is the only reason I claimed that it is more general.<sup>2</sup>

To summarise, Calcott (2017) claimed to have found an alternative to specificity; however, it was simply one component of Woodward's one-to-one specificity. In his response to my criticism, he timidly recognises that he did not see the link between the two in his original paper and presents links between different measures—which I acknowledged and are well known. Crucially, however, what Calcott claimed was not possible regarding specificity and Woodward's analysis—namely, discriminating  $p_3$  from  $p_4$  (one important exemplar that motivated Calcott to present his analysis)—is perfectly possible when one refers to one-to-one specificity. To my mind, this demonstrates that, at least in part, his analysis went astray.

Finally, Calcott claims that extending Woodward's radio provides no benefits for the analysis and, worse, it is inadequate. In Bourrat (2019a), I showed that by *merely* adding one more dial, which would make the radio indeterministic—one does not hear the same thing every time the second dial is in one given position and the other variables are the same—the radio becomes equivalent to the Waddington box. Calcott claims that by adding a second dial, we do not have a radio anymore and that the metaphor is lost. This is supposed to persuade us that the Waddington box is more relevant. Yes, we do not have a radio anymore, but it connects to the intuitive example of the radio. We *almost* have a radio—and this is the crucial point. Citing more than three million possible layouts certainly looks impressive, but one could argue that it adds unnecessary complications and asks the reader to switch examples and learn new variables. And all of this for which benefit? A Waddington box that refers to nothing in the world but itself. In the end, the same analysis can be conducted with a second dial on the radio. Is a Waddington box really closer to a real biological example than a radio with a second dial? I remain sceptical. Two slots, a ball, four buckets, and different layouts of pins are intuitively not much closer to real biology than a radio with two dials and one switch. Further, the Waddington box's complexity—or, more accurately, intricacy—seems to go against Calcott's own stated aim, which is 'to construct a simple model which generates, as best we can, the same intuitions as the biological cases' (2019, p. 8)

While I have rebutted Calcott's main affirmations against my analysis, showing that they detract from the main points I made, I would like to finish on a more positive note. Following Woodward's analysis permits us to discriminate situations that Calcott claimed one could not; however, this does not mean that Calcott's analysis has no value. Clearly, examining what occurs when controlling for a variable—which is analogous to conditioning on a probability distribution—is an important aspect of experimental sciences. It permits us to learn more details about the interactions between different variables than if one were to look at things more globally—that is, without conditioning. My main target, however, was Calcott's claim that precision captures something that is not captured in Woodward's analysis.

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<sup>2</sup> Calcott confuses a one-to-one *relationship* with a *measure* of one-to-one specificity when he claims: 'Bourrat goes on to claim that his measure is "more general" than my own, but this cannot be correct. If something is one-to-one, then it is precise, but not vice-versa. This makes the one-to-one relation more restrictive, rather than more general.' I obviously agree that a one-to-one relationship is more restrictive than one that can be many-to-one. However, this tells us nothing about the measure of one-to-one specificity. Many-to-many relationships score a certain value on one-to-one specificity. By 'more general,' I meant that my measure can deal with a greater range of biological situations.

## References

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