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On Calcott's permissive and instructive cause distinction

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Abstract
I argue that Calcott (in Biol Philos 32(4):481–505, Calcott 2017) mischaracterizes in an important way the notion of causal specificity proposed by Woodward (in Biol Philos 25(3):287–318, Woodward 2010). This leads him to (1) rely too heavily on one single aspect of Woodward's analysis on causal specificity; (2) propose an information-theoretic measure he calls 'precision' which is partly redundant with, but less general than one of the dimensions in Woodward's analysis of specificity, without acknowledging Woodward's analysis; and (3) claim that comparing the specificities of two or more causes under what he calls a competitive analysis of causes, does not permit to capture the distinction between permissive and instructive causes. After having restated Woodward's analysis of causal specificity, I present an information-theoretic measure (variation of causal information) which, although related to Calcott's measure, is more general than his and corresponds to the notion of specificity he missed in Woodward's analysis. I then show how this measure can be used, together with mutual causal information (which captures another dimension of specificity in Woodward's analysis), to distinguish permissive from instructive causes in a competitive analysis of causes.

Keywords Causation · Interventionist account · Causal specificity · Information theory · Mutual information · Variation of information

The recent literature on causation has seen a burgeoning number of contributions discussing the concept of causal specificity, especially in the context of biology.[1] Calcott (2017) attempts to extend this work by providing some substance

[1] See for instance Woodward (2010), Griffiths and Stotz (2013), Griffiths et al. (2015), Pocheville et al. (2017), Weber (in press), Weber (2006), Waters (2007).

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Table 1 Quotes from biologists using the permissive/instructive distinction

Authors	Quote
Holtzer (1968, 152)	"Embryonic induction is generally described as an instructive event. The problem itself is often posed in terms implying the transmission of informational molecules from one cell to another cell. [...] I shall suggest that 1) induction cannot be reduced to a didactic inducer molecule transforming naive cells, but more often involves a sequence of permissive events involving covertly differentiated cells..."
Crair (1999, 88)	"What remains uncertain is whether the role of neural activity in development is instructive or permissive. In other words, it is not clear whether the pattern of neural activity molds directly the development of the neural connections or whether it is simply the presence of neural activity that enables other developmental cues, such as molecular factors, to guide appropriate neural connections"
Lemmon et al. (1992, 818)	"Such findings suggest that some substrate molecules may be permissive, inducing neurite elongation, but not instructive, in the sense of directing growth in a particular direction along a pathway or indicating which way to go at an intersection between two pathways"
Hunter and Hatten (1995, 2061)	"Instructive signals would commit a precursor cell to a particular cell lineage at the expense of production of other classes of cells, while in a permissive system the generation of different classes of cells would follow a stochastic mechanism, with differentiation factors permitting the survival of particular, partially committed progenitor cells"

to the distinction between 'permissive' and 'instructive' causes which Woodward (2010, 317) qualifies as being "elusive." Gilbert (2010, 81–82) attributes the origin of this distinction to Holtzer (1968, see Table 1 for a specific quote) within the context of induction. Induction is a type of close range interaction by which a tissue or cell (inducer) produces a signal that changes the state of another tissue or cell (responder) during development. For induction to occur, the production of a signal or signals with the ability to change the state of the responder is obviously necessary. Yet, for this to happen there must be a context or environment permitting it. In that sense, the environment has a *permissive* role for induction, while the signal has an *instructive* one. An example of an instructive factor given by Gilbert is the signal sent by an optic vesicle to induce the development of the ectoderm into a lens. When the vesicle is not present, a lens does not develop. Examples of permissive factors in this case are any of the molecules composing the extra cellular matrix that supports cells in tissues. A number of biologists have used the distinction in a similar way (see Table 1 for quotes).

The permissive/instructive distinction points towards a more abstract distinction in the philosophy of causation between 'background' or 'enabling' conditions, on the one hand, and 'triggering' conditions, on the other hand, with background conditions being classically regarded as less specific than triggering conditions. Following this analogy, a permissive cause corresponds to a background condition while an instructive cause corresponds to a triggering condition. The lighting of a match for instance involves at least two factors, namely striking the match and the

presence of oxygen surrounding the match. In practice, however, only the striking of the match is classically considered to be *the* cause or the triggering condition for the match lighting, while the oxygen is regarded as a background condition. One reason to regard it as a background condition might be because it is a less specific cause. In fact, the presence of oxygen permits many other events besides the lighting of the match, while striking the match seems to be specific to its lighting. To be sure, Gilbert (2010, 82) draws a similar link between permissive factors and background conditions, on the one hand, and instructive factors and triggering conditions, on the other hand, when he writes:[2]

> It is easy to distinguish permissive and instructive interactions by an analogy with a more familiar situation. This textbook is made possible by both permissive and instructive interactions. The reviewers can convince me to change the material in the chapters. This is an instructive interaction, as the information in the book is changed from what it would have been. However, the information in the book could not be expressed without permissive interactions with the publisher and printer.

The reviewers in the example of the book play a similar role to striking the match in the match example, and the publisher a similar role to the oxygen. That said, one difference between triggering conditions and instructive causes, is that 'instructing' evokes a program or sequence of steps to be followed, while 'triggering' does not. Besides this difference there is a strong overlap between the two notions.[3] Although the distinction between background (permissive) and triggering (instructive) conditions (cause) is intuitive, is there a way to justify the claim that striking the match (reviewers) rather than the presence of oxygen (publisher) is the triggering (instructive) cause of the match lighting (information in the book), or at least a cause that should be regarded as more important? This problem has been called the 'problem of causal selection' which can be traced back at least to Mill (see Franklin-Hall 2015).

One classical response to the problem of causal selection is to be a pragmatist and argue that what counts as triggering and background conditions will depend on the context or background in which a question is asked (Schaffer 2016). However, assuming a certain setting, there might be a more objective way to rank causes as being more or less important because of some of their properties. Calcott (2017, 488) proposes a dichotomy between what he calls a "competitive" and a "hierarchical" analysis of causes, which map respectively, at least partly, with a pragmatic and an objective approach to the problem of causal selection. In a competitive analysis, the causes are compared with all possible states of each cause considered while in a hierarchical analysis the relationship between one cause and its effect is assessed while the value for other (background) causes producing the effect is held constant. The measure obtained with this latter approach can then be compared with measures

[2] The same is true of Woodward (2010, 317).

[3] The difference between 'triggering' and 'instructing' might itself breakdown since triggers can lead to a succession of very complex events such as with Rube Goldberg machines.

of the same relationship when background causes are held constant with different values.[4] Of these two approaches, Calcott (2017, 483) argues that it is best to follow the hierarchical approach. He then asserts that the quantitative measure of causal specificity proposed by Griffiths et al. (2015) can give substance to the distinction between permissive and instructive factors under the hierarchical approach. I have several contentions with Calcott's claims.

First and foremost, he makes some claims about Woodward's analysis on causal specificity which are inaccurate. Second, because of this, the information theoretic measure of causal specificity he uses does not permit one to fully capture the notion of causal specificity relevant for distinguishing between triggering/instructive causes, on the one hand, and background/permissive conditions, on the other hand. I will take the opportunity to provide such a measure. Finally, with a correct measure of causal specificity, a competitive or objective analysis of causes permits, contrary to what Calcott (2017, 481) claims, the separation between permissive and instructive causes.

As was just mentioned, to quantify causal specificity, Calcott draws from the work of Griffiths et al. (2015) within Woodward's (2003) interventionist account of causation. Griffiths et al. (2015) intend to provide a measure of causal specificity which is one of the dimensions of causal relationships Woodward (2010) argues is important in biology. Woodward (2010) proposes two notions of causal specificity. One he calls "INF" for influence, the origin of which he credits to Lewis (2000). This is the notion of specificity which is the target of Griffiths et al. (2015) who argue that INF is captured by the amount of mutual information transferred from a causal variable to an effect variable (hereafter 'mutual causal information'). This measure is obtained by calculating the mutual information between two variables, to one of which, the causal variable, is applied Pearl's (2009) do(.) operator, which represents intervening on the variable.[5]

The other notion of causal specificity proposed by Woodward (2010, 310) is what he calls "one cause-one effect" specificity (hereafter 'one-to-one specificity'), which corresponds to the specificity of a kind of cause for a given effect. Woodward takes the example of asbestos and smoking as causes of lung cancer. When considering the effect 'lung cancer', the specificity of causes is low, as this effect has many different causes. When considering the cause 'smoking', the specificity is also low, as smoking has many different effects. Although Woodward makes the case for this second sense of specificity in contexts in which the terms *causes* and *effects* refer to variables, he clearly entertains the possibility that they could refer to states of variables. To be sure, this is quite explicit when he claims: "[t]hus, one may think of each state of the cause-variable as causing one and only one state of the effect-variable, so that (in this sense) the one–one requirement is satisfied with respect to

[4] Calcott's hierarchical approach is actually more subtle than a pure pragmatic approach, but this does not matter for my purpose here.

[5] Intervening on a variable can be defined as setting the state of that variable independently of the states of other variables that would otherwise influence its state. For details see Griffiths et al. (2015, Appendix B).

states" (313). This move is further justified from the fact that, in many situations, multiple variables can be aggregated into a single new variable, resulting in what were considered distinct variables becoming states or values of the aggregated variable. In the remainder, by 'one-to-one specificity', I will refer to specificity of the states rather than the variables.[6]

In spite of Woodward being clear that there are two different notions of causal specificity, throughout his article, Calcott develops his argument as if causal specificity had only been characterized by Woodward as INF. Thus, his claim that "[t]he notion of a cause precisely determining its effects is not captured by any of Woodward's three properties, specificity, proportionality, or stability, though it is clearly an important characteristic of a causal relationship"(2017, 495) is incorrect.[7] 'Precision' is Calcott's proposed notion to account for what he claims is missing in Woodward's analysis. Yet, a cause precisely determining its effect, that is when every state of the causal variable determines a single state of the effect variable, is one of two conditions for a relationship to be maximally one-to-one specific, following Woodward's analysis. The other condition is that every state of the effect variable is determined by a single state of the causal variable. When the two conditions are satisfied, the mapping is bijective, that is maximally one-to-one specific. The notion of precision proposed by Calcott, because it only deals with the first of the two conditions, is thus less general than Woodward's notion of one-to-one specificity. In a biological context, it might be interesting to know to what extent multiple causes can lead to the same outcome (e.g., multiple chemicals lead to the same developmental outcome), but equally interesting to know to what extent the same cause can lead to different outcomes (e.g., the same chemical can lead to multiple developmental outcomes), something Calcott's measure does not account for. To be fair, Calcott recognizes in the last section of his paper that the term 'specific', which he claims is not captured by Woodward's dimensions of causation, "can also mean "precise"" (2017, 501). However, he never links his notion of precision with the measure he provides to Woodward's notion of one-to-one specificity.

Having drawn the distinction between the range of causal influence and the one-to-one notions of causal specificity, what does this mean for the permissive/instructive cause distinction? My contention is that a formal measure of one-to-one specificity, coupled with the INF and Woodward's notion of specificity, as formalized by Griffiths et al. (2015), can perfectly account for the notions of permissive and instructive cause within an objective or competitive analysis of causes. In a competitive analysis of causes, following Calcott's terminology, causes are compared on some dimension(s) while taking all the possible values of the other causes into consideration. In a hierarchical analysis, the values of causal dimension(s) for a cause are assessed while conditioning on some value for other causes (Calcott 2017, 488, 497). To provide a competitive approach we will first require a formal measure that maps with one-to-one specificity. To do so, assuming a particular grain

[6] Woodward (2010, 312) talks about "splitting" and "lumping" variables.

[7] Note also that proportionality is not a characteristic of causal relationships, as claimed by Calcott, but a feature of the description we give of them.

of description,[8] one can exploit the notion of variation of information, a measure related to mutual information.[9]

The variation of information between two variables C and E ($VI(E; C)$) is defined as

$$\begin{aligned} VI(E;C) &= H(E,C) - I(E;C) \\ &= H(E) + H(C) - 2I(E;C) \\ &= H(E|C) + H(C|E) \end{aligned} \tag{1}$$

where $H(E)$ and $H(C)$ are the Shannon entropies (see Shannon 1948) of E and C respectively, $H(E, C)$ is the joint entropy of E and C, $I(E; C)$ is the mutual information between E and C, and $H(E|C)$ and $H(C|E)$ are the conditional entropies of E knowing C and of C knowing E respectively.[10] Using the do(.) operator, represented with the symbol $\widehat{\ }$ applied to the variable on which an intervention is made, as done by Griffiths et al. (2015) and Calcott (2017), one can define a causal version of variation of information (thereafter 'variation of causal information') as

$$\begin{aligned} VI(E;\widehat{C}) &= H(E,\widehat{C}) - I(E;\widehat{C}) \\ &= H(E) + H(\widehat{C}) - 2I(E;\widehat{C}) \\ &= H(E|\widehat{C}) + H(\widehat{C}|E) \end{aligned} \tag{2}$$

We can see from Eq. (2) that when the variation of causal information from $\widehat{C}$ to E is zero, not only does this imply that all the entropy present in C is transferred to E, but also that it does so without any loss of information. In other words, it tells us that the mapping between C and E is bijective, which means that to one value of C corresponds exactly one value of E. When the variation of causal information is non-zero, this implies either that part of the entropy of C is not transferred to E, which means that some values of E are multiply realized (which maps with Calcott's measure of precision), and/or that the entropy of E is higher than the entropy of C in which case the relationship $C \rightarrow E$ is to some extent indeterministic. Stated more formally, Calcott's requirement for maximum precision is that $H(E) = I(E, \widehat{C})$, while the requirement for one-to-one specificity ($VI(E;\widehat{C})$) to be maximal is that $H(E) = H(\widehat{C}) = I(E, \widehat{C})$. In other words Calcott's measure cannot account for cases where $H(E) > I(E;\widehat{C})$, while mine can. The measure proposed in Eq. (2) represents thus a general measure of causal one-to-one (un)specificity between C and E. A longer version of the argument for the appropriateness of variation of causal information as mapping with one-to-one specificity, as well as some exploration of the relation between the one-to-one and the INF notions of specificity are proposed in Bourrat (2019). Griffiths et al. (2015, 538), although they do not clearly make the

[8] For a method to choose the appropriate grain of description see Pocheville et al. (2017) and Bourrat (2019).

[9] For more on this measure see Meilă (2003).

[10] For more on these notions see Griffiths et al. (2015).

distinction between range of influence and one-to-one specificity, are careful in their claim about specificity when they write:

> [B]oth the conditional entropy $H(E|\widehat{C})$ and the mutual information $I(E;\widehat{C})$ capture aspects of the intuition that causes differ in 'specificity'. Because the prior uncertainty $H(E,\widehat{C})$ is not constant—it depends in particular on the size of the repertoire of effects—both measures are needed. The mutual information $I(E;\widehat{C})$ measures how much a cause specifies an effect. The conditional entropy $H(E|\widehat{C})$ measures how much an effect is determined when knowing the value set for the cause.

Recall that $H(E|\widehat{C})$ is one term of the two terms of $VI(E;\widehat{C})$ (see Eq. 2). It corresponds effectively to Calcott's precision since when $H(E|\widehat{C}) = 0$ we have $H(E) = I(E,\widehat{C})$. In light of this, it is even more surprising for Calcott to claim that none of Woodward's properties can account for the precision of a variable in determining its effect.

With a measure of one-to-one specificity as variation of causal information in hand, we can now propose a competitive analysis of causes that captures the distinction between permissive and instructive causes. To do so, I will use a version of the toy example proposed by Calcott, namely what he calls 'Woodward's radio' so that my account can be compared to his. Calcott uses a second example that he calls the 'Waddington box' which, one might argue, is more biologically realistic. In fact, it allows 1) an effect to be indeterministic while in Woodward's radio only a single state of the effect variable is obtained in a given background, and 2) it permits to change the mapping that links states of a cause to the states of its effect. Although interesting, presenting this example would detract us from the main points made in this manuscript.[11] Note however that Woodward's radio is formally equivalent to the bimolecular example of transcription presented in Griffiths et al. (2015). As such, Woodward's radio also has some biological relevance. In Woodward's radio example, the dial of a radio (D) is contrasted with its power switch (S) which has two states $\{s_{\text{OFF}}, s_{\text{ON}}\}$. Assuming the dial permits to select one of eight possible positions $\{d_1, d_2, \ldots, d_8\}$, one can hear nine different sounds (A for 'audio') $\{a_1, a_2, \ldots, a_9\}$ if each channel selects for a different station and that 'no sound', for s_{OFF}, is one state of A. In such a setting, represented in Fig. 1, both D and S are causes for A. Yet, how can we capture the extent to which a single state of the variables D and S leads to a single state of A? In other words, how can one measure the extent to which S and D

[11] For the interested reader, note that Woodward's radio can easily be tweaked to give the same sort of phenomena as a Waddington box. Since there are only two differences between a Waddington box and Woodward's radio, namely the possibility of indeterministic outcomes and the ability to change the mapping between a cause and its effect in the Waddington box, Woodward's radio could be modified to be equivalent to a Waddington box by (1) allowing the dial to have an indeterministic effect on what we hear (sometimes one station, other times another or even nothing), which is something Calcott (2017, p. 495) himself alludes to, to make a point more intuitive; (2) adding a second dial that would change the mapping of the first dial with what we hear. *Mutatis mutandis* with these two modifications we would effectively have Woodward's radio behaving like a Waddington box.

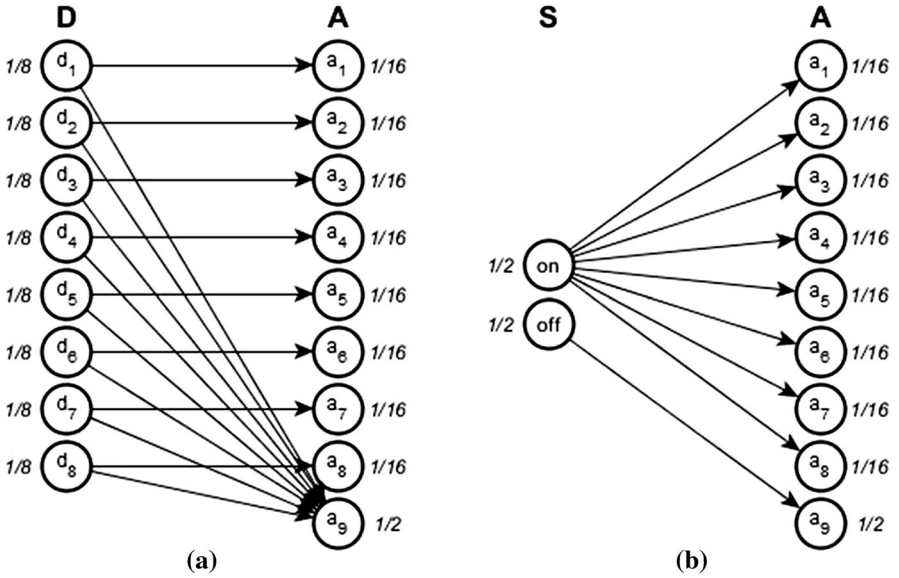

Fig. 1 Causal diagrams of Woodward's radio. **a** Causal diagram representing the causal relationship between D and A, assuming S is in the background and each of its eight possible states is equiprobable. **b** Causal diagram representing the causal relationship between S and A, assuming D is in the background and each of its two possible states is equiprobable

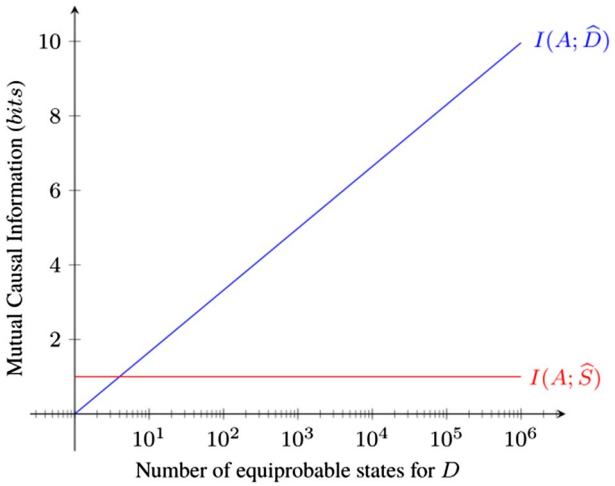

Fig. 2 Mutual causal information from D to A and S to A for Woodward's radio when the number of equiprobable states for D varies

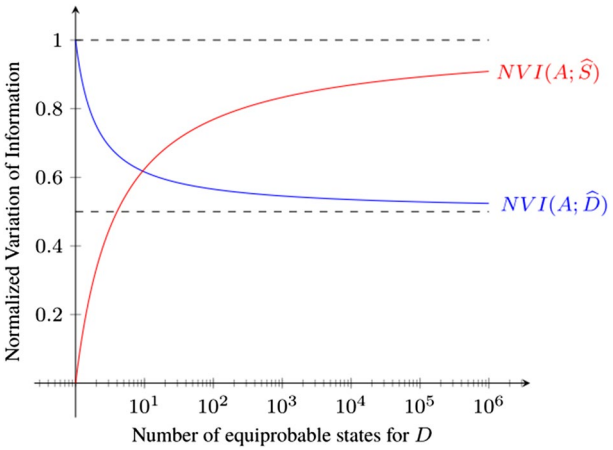

Fig. 3 Normalized variation of causal information from D to A and S to A for Woodward's radio when the number of equiprobable D states varies

are one-to-one specific causes of A. As argued above, variation of causal information permits this.

Assuming, as Calcott does, equiprobability of states for the variable D and S, as well as an equiprobable distribution for each state of D and of S mapping to the states of A when more than one state of A is caused by one state of D or S, we can calculate the range of causal influence and one-to-one causal specificity for the pair of causal relationships $D \rightarrow A$ (Fig. 1a) and $S \rightarrow A$ (Fig. 1b). Starting with range of causal influence, we obtain $MI(A;\widehat{S}) = 1$ bit and $MI(A;\widehat{D}) = 1.5$ bits. As claimed by Calcott (2017, 487), the difference between the two results is modest. Decreasing the number of states of the dial to four gets the result $MI(A;\widehat{D}) = 1$ bit, while multiplying by two gets $MI(A;\widehat{D} = 2$ bits), multiplying by four gets $MI(A;\widehat{D}) = 2.5$ bits, and so forth (see Fig. 2). $MI(A;\widehat{S})$ is always 1 bit in these cases, as shown in Fig. 2, because the maximum mutual information between two variables cannot be higher than the maximum entropy of the variable with the lowest entropy, which is 1 bit in the case of S. This result is not particularly surprising. It simply indicates that the range of causal influence of a variable with a higher number of equiprobable states than that of a switch (two states) will typically (but not necessarily) be higher than that of the switch. More importantly, from this result alone, that is without knowing how many possible states for the causal and effect variable there are, it cannot be deduced how far from bijective the mapping is. In fact, although a bijective mapping with a mutual causal information of 1 bit necessarily implies that the causal variable has two states, a non bijective mapping between a cause and an effect can also have a mutual causal information of 1 bit.

If we now calculate the variation of causal information of these relationships, we obtain $VI(A;\widehat{S}) = 1.5$ bits and $VI(A;\widehat{D}) = 2.5$ bits. Although this seems to indicate that $\widehat{S}$ is more one-to-one specific than $\widehat{D}$, this conclusion cannot be reached until each value is normalized over the respective joint entropy of the pair of variables. This is necessary because variation of information is an *absolute* measure which

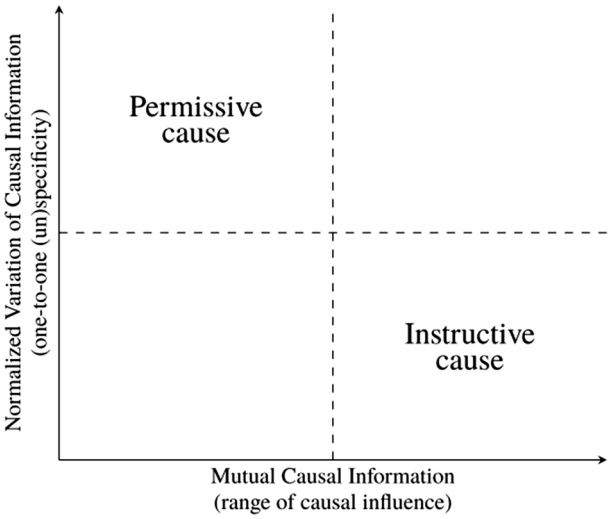

Fig. 4 Relationship in a relative state space between permissive and instructive causes in terms of mutual causal information and normalized variation of causal information

does not take into account possible differences in entropies and range of causal influences of the different causes. Normalizing over the joint entropy permits us to overcome this problem. The range of values for the normalized version of variation of information (*NVI*) is between 0 and 1. Once applied, we get $NVI(A;\widehat{S}) = 0.6$ and $NVI(A;\widehat{D}) = 0.625$. This indicates that in the Woodward's radio setting proposed by Calcott, D is less one-to-one specific than S. In other words, the relationship $S \rightarrow A$ is more bijective than $D \rightarrow A$. This is even more the case when the number of states for D is decreased to four and two states as shown in Fig. 3. For four states we get $NVI(A;\widehat{S}) = 0.5$ and $NVI(A;\widehat{D}) = \frac{2}{3}$, while for two $NVI(A;\widehat{S}) = \frac{1}{3}$ and $VNI(A;\widehat{D}) = 0.75$. Considering cases in which the number of states of D increases to 16 equiprobable states we have $NVI(A;\widehat{S}) = \frac{2}{3}$ and $VI(A;\widehat{D}) = 0.6$, indicating that D becomes more one-to-one specific than S when there are between eight and 16 states for D. When there are 32 states $NVI(A;\widehat{S}) \approx 0.71$ and $NVI(A;\widehat{D}) \approx 0.58$, and so forth (see Fig. 3).[12] This indicates that the larger the number of possible states for D the more the relationship $D \rightarrow A$ is one-to-one specific. As can been seen in Fig. 3, as the number of states of D increases, the value of normalized variation of causal information from $\widehat{D}$ to A tends toward 0.5, which is its lower limit under the assumption of equiprobability. This is because even if the mapping between $\widehat{D}$ and A is bijective when $\widehat{S} = s_{\mathrm{ON}}$, it is considered that $\widehat{S} = s_{\mathrm{OFF}}$ half of the time ($P(\widehat{S} = s_{\mathrm{OFF}}) = 0.5$). It can be shown that by changing the probability distribution of $\widehat{S}$ and making $P(\widehat{S} = s_{\mathrm{OFF}})$ a much rarer event, the lower limit of $NVI(A;\widehat{D})$ is decreased so that $\lim_{P(\widehat{S}=s_{\mathrm{OFF}})\rightarrow 0} NVI(A;\widehat{D}) = 0$. Similarly, as $NVI(A;\widehat{D})$ decreases,

[12] $NVI(A;\widehat{S})$ would be equal to $NVI(A;\widehat{D})$ in a setting with D having around 9.4 states.

$NVI(A;\widehat{S})$ increases toward the maximum value, namely 1 so that $\lim_{P(S=s_{OFF})\to 0} NVI(A;\widehat{S}) = 1$, indicating that $\widehat{S}$ becomes less one-to-one specific (maximally so in the limit).

With this example in mind, the permissive/instructive distinction can be drawn as follows. A permissive cause has both a lower causal range of influence (ideally two possible states, to act as a switch) and a lower one-to-one specificity (higher normalized variation of causal information) than the other causal variables producing the same effect. These other causal variables are instructive relatively to the first cause for they have a high(er) mutual causal information which indicates they are not a switch and a high(er) one-to-one specificity, which in itself indicates that they direct a particular outcome. These properties match the definitions of permissive and instructive cause used by biologists (see Gilbert's quote above and those of Table 1). Thus, in a relative state-space with the two dimensions of causal specificity, the permissive and instructive causes corresponds to a situation in which the two causes have scores on the two dimensions matching those depicted in Fig. 4 (the higher left and the lower right corners of the relative space respectively). When the two causes cannot be separated as such in the relative state space presented in Fig. 4, the distinction between permissive and instructive causes loses its appeal, as is the case, for instance, when the number of positions for the dial is lower than 10 in Woodward's radio. A situation in which a causal relationship scores both lower for normalized variation of causal information and for mutual causal variation of information relative to other relationships, would necessarily imply that this cause, in spite of being more of a switch than other causes, sends on average more 'instructions' to the effect variable than other causes do. In fact, in virtue of a low normalized variation of causal information, the mapping between this cause and the effect would be more bijective relative to other relationships. Second, a situation in which a causal relationship scores both higher for normalized variation of causal information and mutual causal information relative to other relationships, would necessarily imply that the cause, in spite of being less of a switch than other variables, sends nevertheless less instructions to the effect variable (the mapping is less bijective).

To sum up, a large part of Calcott's claims about causal specificity seems to have come from an incomplete reading of Woodward's analysis on causal specificity. Once one-to-one specificity is taken into consideration and measured by variation of causal information, pace Calcott (2017, 481) who claims that "the permissive–instructive distinction cannot be captured by simply contrasting the specificity of two causes as Woodward proposes", a competitive analysis of causes with the two dimensions of causal specificity, that is one precisely contrasting the specificity of two or more causes based on Woodward's analysis and over all possible states for the causal variables, as I showed, can be given straightforwardly. The less a cause is one-to-one specific and the lower its range of influence, when compared to other causes, the more it is permissive. The more a cause is one-to-one specific and the higher its range of influence, when compared to other causes, the more it is instructive.

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