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For a (revised) PCA-analysis

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According to our PCA-analysis of causation (1996), c causes e iff c is a 'potentially complete ancestor' of e ; i.e. iff

(PCA) there is a (possibly empty) set of actual events, Σ , such that if c were to occur *without* any of the events in Σ , then e *might* occur as a descendant of c with only actual events mediating between c and e . (See our original paper for definitions, etc.)

Byrne and Hall (1998) have persuaded us that this is not quite right. But it is not far wrong. In this note we offer a modified version of our PCA-analysis which handles two of the three candidate counterexamples Byrne and Hall provide. The third example, we argue, does not in any case tell in favour of Lewis's quasi-dependence account (1986: 205–7) over ours – so we stand by our original conclusion that our approach is preferable to Lewis's.

Let's begin with their second counterexample (1998: 42, fig. 4). Event c comes out as a cause of e , contrary to intuition, because if c were to occur without a (fig. 5), e would occur as a descendant of c and the non-actual event d would not count as mediating between c and e since it still would not be a descendant of c . The problem is avoided if we simply replace the phrase 'with only actual events mediating between c and e ' in (PCA) with 'with only actual events as ancestors of e '. This revision is in line with our original intuition that effects might have been descendants of their causes without the 'aid' of any non-actual events (1996: 222).

Byrne and Hall's first counterexample (1998: 40, fig. 2) is avoided if, as they suggest (41, fn. 4), we allow non-actual events into Σ . But this move calls for a further reformulation of (PCA). For if Σ contains only non-actual events, the nearest world where c occurs without any of the events in Σ will be the actual world, where e occurs but is not a descendant of c (assuming c is a preempting cause of e). The revised version of PCA we propose, then, is given by the following definitions.

Def 1. For any events x and y , and any set of events Σ , y Σ -depends on x iff

- (i) if neither x nor any of the events in Σ were to occur, then y would not occur, and

(ii) if x were to occur without any of the events in Σ , then y might occur.

Def 2. For any events x and y , and any set of events Σ , x is a Σ -*ancestor* of y iff there is a chain of events, z_1, \dots, z_n , such that z_1 Σ -depends on x , ..., and e Σ -depends on z_n .

And now:

(PCA*) For any actual, distinct events c and e , c *causes* e iff there is a (possibly empty) set of possible events Σ such that

- (i) c is a Σ -ancestor of e , and
- (ii) every Σ -ancestor of e is an actual event.

In the fig. 2 example, c comes out as causing e as desired – take $\Sigma = \{b, f\}$ – and in the fig. 4 example, taking $\Sigma = \{a\}$ does not result in c coming out a cause of e , because d is a Σ -ancestor of e .

So far so good. As telegraphed, our response to the Byrne and Hall's third example (fig. 6) is more defensive. Our account delivers the verdict that both c and a are causes of e (take Σ to be $\{a\}$ in the first case and $\{c\}$ in the second). How does Lewis's quasi-dependence account fare? Byrne and Hall do not explicitly say why it should deliver the verdict that c is *not* a cause of e . But they do say:

Thanks to the occurrence of a , e does not depend on c . More to the point, c is not a cause of e : with the inhibitory connection [between d and e] severed, the occurrence of c is wholly irrelevant to e . (1998: 43)

However, a parallel point could be made about a : thanks to the occurrence of c , e does not depend on a – after all, a is relevant to the occurrence of e only if d fires, and this won't happen if c fires. So, their remarks do not at any rate capture a significant asymmetry between a and c . Nonetheless, it might be argued that the verdict that neither a nor c are causes of e is less counterintuitive than the PCA-verdict that both are; so if Lewis's theory does count neither as causes (for the reason they give), then there is a case for preferring it to ours.

This would be a good argument if it were not for the fact that there are similar examples (of overdetermined inhibition of an inhibitor) where the PCA-verdicts are intuitively preferable. Consider e.g. a set-up like fig. 6, but where c is an event that occurs at the same time as a and which *severs* the stimulatory connection between the neuron to the left of d and d . Again, the PCA-analysis would deem both a and c causes of e whereas Lewis's account, sticking with the same reasoning as above, would still count neither as causes. Yet this time, to our mind at any rate, the PCA-verdict seems less counterintuitive.

We are not persuaded, then, that Lewis's theory handles cases like the

present one better than the PCA-analysis. And the reasons we gave in our earlier paper (1997: 222 ff.) for preferring our approach to Lewis's remain good.^{1, 2}

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¹ The present account preserves Lewis's original view that any ancestor of an event is a cause of that event (take Σ to be the empty set); but there is growing resistance to this view – we are particularly impressed by Michael McDermott's dog-bite example (1995: 531 ff.). A simple way to avoid this result, without giving up our solution to the preemption problem or our responses to Byrne and Hall's examples, is to replace clause (i) of (PCA*) with: *e* Σ -depends on *c*.

² Thanks to Alex Byrne and Ned Hall for discussions on these matters.