THE PHILOSOPHY OF CAUSATION, LECTURE 2

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Overview

I this lecture I will introduce and discuss some of the salient topics in the contemporary debate on causation (without taking much of a stance in this)

First, I’ll look at some of the problems in the special sciences:
- Physics: Russell and the (in)dispensability of causal laws
- Social and biomedical sciences: do we need (causal) mechanisms?
- Evidence-based policy and medicine: what is the role of RCTs?

Second, the first lecture and the first part of this lecture seem to make pluralism about causation an attractive option; but what do we mean by ‘causal pluralism’?
... was, at least at some point, highly sceptical of causation:

the reason why physics has ceased to look for causes is that, in fact, there are no such things. The law of causality, I believe, like much that passes muster among philosophers, is a relic of a bygone age, surviving, like the monarchy, only because it is erroneously supposed to do no harm ('On the Notion of Cause', 1912)
The laws of physics

According to Hartry Field (2003), Russell had two arguments against causality in physics:

- The **asymmetry argument**: causality is directional but nearly all laws of modern physics are time-symmetric (presupposes physicalism!)

- The **intervention** (or perhaps interference) **argument**: no matter how detailed the description of the cause (short of a description of everything that happens in the effect’s past light cone), there is always the possibility that something intervenes such that the effect is prevented from happening
Cartwright on causation

- Nancy Cartwright (1979), by contrast, has argued that neither science nor everyday life can make do without specifically causal laws: our successful strategies must be based on causal laws, not mere laws of association.

- Field thinks that this dilemma is ‘probably the central problem in the metaphysics of causation’.
The laws of physics

Russell’s point about *causal* laws can in fact be extended to all laws of physics; consider Newton’s law \( f = Gm_1m_2/d^2 \)

The predicted acceleration will only obtain when there are no interferences (such as a rigid rod keeping the masses apart, other masses...)

In the actual world there are no perfectly isolated systems – so do we need information about a cross-section of the past light cone?
There essentially are two responses to this conundrum:

- The laws of physics are not laws simpliciter but rather ceteris paribus laws – true only ‘other things being right’
- But c.p. clauses are notoriously hard to define if they are to have any content at all (e.g. Earman, Roberts and Smith 2002; Woodward 2002)
- The laws of physics describe not outcomes but influences or contributions to outcomes, i.e., capacities (Cartwright 1989, 1999, Corry 2006)
- But many philosophers are worried about the metaphysical baggage belief in capacities seems to commits us to
Mechanisms in the social and bio sciences

As far as I can see, mechanisms have been invoked in the philosophy of various special sciences for two main reasons:

- **We explain** phenomena by invoking mechanisms (e.g., Machamer et al. 2000: ‘In many fields of science what is taken to be a satisfactory explanation requires providing a description of a mechanism’)

- Mechanisms can be useful for **causal inference** (e.g., Steel 2004: ‘It is sometimes claimed that the consideration of social mechanisms can significantly ameliorate [the problem of confounders]’)

Mechanisms in the social and bio sciences

- We may thus ask, what is the role of (causal) mechanisms in scientific explanation and in causal inference?
- Before going into that, let us define ‘mechanism’
- There are about as many definitions of mechanism as there are contributors to the discussion
- Here’s my own (Reiss 2007): ‘A causal mechanism for a causal relationship between (aggregate...) variables X and Y is a set of entities and properties that are such that, if they were embedded in a stable structure, could operate unimpededly and, if X fired regularly, then Y would follow regularly’
Mechanisms in the social and bio sciences

- (This is similar to the definition in Machamer et al. but makes the importance of shielding explicit and does not take actual regularities to be essential)
- Metaphorically, we can say that investigating a mechanism means to ‘open the black box’, to examine what is behind and responsible for more readily observable phenomena
- That knowledge about mechanisms can sometimes be used for explanatory purposes should be uncontroversial
Are mechanisms necessary for explanation?

- But some philosophers make stronger claims than that: only models that represent genuine mechanisms are explanatory
- Social science: Jon Elster, critical realists
- Biomedical science: Carl Craver

- These claims seem too strong though:
  - Some explanations aren’t causal in nature (e.g., Kitcher)
  - Even genuinely causal explanations don’t have to involve a mechanism
    - Macro explanations
    - Explanations by counterfactual dependence without mechanistic connection
  - Requiring a mechanism sometimes seems arbitrary
Are mechanisms necessary for inference?

Arguably, causal inference is very hard in those domains where many causal factors interact in order to bring about an effect and where controlled experimentation is not possible or possible only to a very limited extent – such as the social and biomedical sciences.

In these sciences, then, the ‘problem of confounders’ is ubiquitous: any supposed causal hypothesis between some X and some Y can be challenged by an alternative hypothesis that invokes a common cause.

(Note: this is different from the usual underdetermination problem)

Mechanisms are thought to ameliorate the problem – and in fact to provide the only solution (Elster, Little, Hedström and Swedberg for social science; Russo and Williamson for biomedical science).
Are mechanisms necessary for inference?

- This claim, however, seems to be too strong, too:
  - It would entail that we can never know causal claims (Kincaid)
  - Tools for aggregate causal analysis are available – and sometimes work (Kincaid)
  - The inference rule ‘There is no plausible mechanism from X to Y, therefore X does not cause Y’ is virtually useless in social science (Steel)

- This does not mean that investigating mechanisms isn’t a fruitful research strategy – there may be cases where at the lower level controlled experimentation is possible or where the problem of confounders obtains to a lesser degree or where more background knowledge is available
Are mechanisms necessary for inference?

- Erik Weber (2007) has noticed another potentially interesting area for investigating mechanisms: external validity.
- External validity concerns of causal claims from test context to a context outside the test.
- Weber argues that while knowledge of mechanisms isn’t necessary for establishing that X causes Y, it is necessary for establishing that the causal relation continues to exist outside the context in which it was originally confirmed.
- But this claim also seems contentious (Steel 2007):
  - Unpractical as mechanisms are often hard to know.
  - Mechanisms vary too.
  - There are alternative ways for extrapolation.
Evidence-based policy and medicine

There are relatively recent movements in social policy and medical research that require policy and medical interventions to be ‘evidence-based’

(What the heck were they based on before???)

Thus, their general characterisation is uncontroversial; e.g.: ‘Evidence based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients’ (Sackett et al.)

Or: ‘we use [EBP] to refer to an approach to policy development and implementation which uses rigorous techniques to develop and maintain a robust evidence base from which to develop policy options. All policies are based on evidence - the question is more whether the evidence itself, and the processes through which this evidence is put to turn it into policy options, are of sufficiently high quality’ (defra.gov.uk)
What evidence in evidence-based X?

- So far, so good. So what’s the debate about?
- It is clear that in any area of science, evidence for causal claims comes in a multitude of forms, including:
  - Experiments
  - Statistical studies
  - Introspection
  - (Clinical etc.) Experience
  - Case study analysis
- So what do we do if evidence from each different source tells a different story?
What evidence in evidence-based X?

- If any method were completely reliable, evidence which has used that method in its production would beat every other piece of evidence.

- But if any method were completely reliable, we wouldn’t need the different kinds of evidence to begin with!

- This point seems to be forgotten by the evidence-based people; they construct so-called ‘hierarchies of evidence’.
A hierarchy of evidence

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>One</td>
<td>Strong evidence from at least one systematic review of well designed randomised controlled trials (RCTs)</td>
<td>Meta-analyses The Cochrane Collaboration</td>
</tr>
<tr>
<td>Two</td>
<td>Evidence from at least one properly designed RCT of appropriate size</td>
<td>Articles published in peer-reviewed journals</td>
</tr>
<tr>
<td>Three</td>
<td>Evidence from well designed trials without randomization: cohort, time series or matched case controlled studies</td>
<td>Articles published in peer-reviewed journals</td>
</tr>
<tr>
<td>Four</td>
<td>Evidence from well designed non-experimental studies from more than one centre or research group</td>
<td>Articles published in peer-reviewed journals</td>
</tr>
<tr>
<td>Five</td>
<td>Opinions from respected authorities, based on clinical evidence, descriptive studies or reports from committees</td>
<td>NICE guidelines Evidence-based local procedures and care pathways</td>
</tr>
<tr>
<td>Six</td>
<td>Views of colleagues/peers</td>
<td>Nursing colleagues or members of the multidisciplinary team</td>
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(Source: ebnp.co.uk)
Are RCTs infallible?

- On the positive side: one can prove that ideal RCTs lead to causally correct conclusions (Cartwright 2007)
- But the conditions for the proof are stringent indeed, probably hardly ever realised in practice and never known to be realised in practice
- What is worse, even an ideal RCT doesn’t give us the knowledge we want:
  - It yields knowledge about average effects, not individual effects
  - The average is over a certain kind of population that might not (and usually is not) the target population
  - RCTs are only good for short-term effects
  - Randomisation is a good blinding device; but that’s irrelevant in many areas (think of psychoanalysis or surgery techniques or education systems)
Are RCTs infallible?

- What is more, there is no guarantee that a second best is better than a completely different design (cf. Elster 2007)
- And practical problems abound, including:
  - Was randomisation successful?
  - Were eligibility decisions unbiased?
  - What about compliance?
  - Is the study **fully** blind?
- In sum, RCTs are powerful causal learning devices when applicable; but other methods have other advantages and disadvantages that are better suited to some situations (cf. Scriven 2008)
I take it that the lesson of the first lecture is that no (universal) theory of causation – whether it is reductive or whether it takes causal notions as analytically basic – is without problems.

One way out of this impasse that has been suggested is pluralism: causation is not one thing but rather many.

But ‘causal pluralism’ is itself far from clear an idea.
Christopher Hitchcock very usefully distinguishes nine types of pluralism about causation:

1. Pluralism about causes
2. Methodological pluralism
   a. scientific
   b. philosophical
3. Intramural pluralism
4. Wittgensteinian pluralism
5. Extramural pluralism
6. Disciplinary pluralism
7. Radical pluralism
8. Radical pluralism
Pluralism about causes

-* Least involving form of pluralism
-* Already espoused by Mill (Hitchcock calls his position monistic though)
-* Cf. Mackie’s INUS conditions: every effect requires a set of causal conditions to be brought about and there is different sets of conditions that are followed by the same type of effect
-* As we have seen, Mill also uses a principle of ‘plurality of causes’
-* Uncontroversial
Methodological pluralism

**Scientific version**: causal claims can be supported by different types of evidence

- e.g., evidence about mechanisms as well as evidence about correlations

**Philosophical version**: different theories bring out different aspects of causation (e.g., Campaner and Galavotti 2008) or are useful in different contexts
Intramural pluralism

Different causal concepts can be defined within the same framework:

- e.g., ‘positive cause’: $P(E | C.B_i) > P(E | B_i)$,
  ‘negative cause’: $P(E | C.B_i) < P(E | B_i)$

- or distinction between component and net cause:

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BC → P
     ↘
P ← T
     ↗
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Intramural pluralism

- Another important distinction one can make within the framework of probabilistic causation is that between an average cause and a unanimous cause (cf. Hitchcock 2005; Weber 2007).

- As we have seen, a probabilistic cause is one that raises the probability of its effects, holding fixed all other causes – but does it have to raise the probability in each cell or on average?

- Cartwright 1979 defended contextual unanimity:

  - C causes E iff \( P(E | C.K_j) > P(E | K_j) \) for all \( K_j \)

- By contrast, John Dupré and others defend the average conception:

  - C causes E iff \( P_C(E) > P_{\neg C}(E) \)
Wittgensteinian pluralism

- e.g., Cartwright 1999/2007, Psillos forthcoming, Longworth forthcoming, Godfrey-Smith forthcoming
- Cartwright/Anscombe: there are different causal concepts such as feed, suck, allow, push, eat, open, speed... that are sometimes usefully but abstractly described as ‘cause’ but the more concrete term is richer
- The different concepts bear no more than family resemblance to one another
Cartwright’s idea is that ‘cause’ is an abstract concept, which for her means two things:

- To have any meaning on a given occasion, the abstract concept must be fitted out with a more concrete concept.
- What the abstract concept means on that occasion is constituted by the meaning of the concrete concept.

Hitchcock agrees that concrete causal concepts have excess meaning over ‘cause’; but is this excess meaning causal in nature?

A further worry: what tells us whether a given concrete term falls under the abstract concept of cause?
Radical pragmatism

• cf. van Fraassen on explanation

• One can hold that the same is true of causation (as van Fraassen at times seems to think): causation is context dependent (because of its counterfactual nature), there is no single objective way to determine which causal claim is true of a given situation

• This may, but doesn’t have to, go along with other types of pluralism

• Hitchcock doesn’t really regard this as a form of pluralism; but we might argue that causation is both varied as well as context dependent
Radical pragmatism

E.g.,

- Regularity: c.p. clauses
- Counterfactual: backtracking
- Probabilistic: selection/definition of variables
- Process: framework (‘intersection’)
- Agency: intervention
Most (anti-reductionist) philosophers of causation are also realists or objectivists: they believe that there are causal relations in the world, independent of human cognition (Cartwright, Woodward, Armstrong etc.)

This contrasts with a view according to which humans contribute causality to the situation (most famously: Hume, Kant); this is a view that is gaining ground today (Williamson, Hitchcock, Weber, Reiss)

Analogously to pluralism about probability (Gillies), we could be pluralists in this sense too: some situations are understood well enough to have reason to believe that causation is really in the objects; others, we conceptualise causally without thinking that the entities of interest have real causal powers.