A Theory of Evidence for Causal Claims

This paper develops an alternative account of evidence, relevant to the special sciences such as the biomedical and social sciences, that satisfies a number of plausible desiderata and that is helpful for advancing current debates in the philosophy of science. The account that emerges is contextualist in that whether or not a given fact is evidence depends on:

- material facts that pertain to the case at hand;
- what is known by the scientific community;
- what questions it purports to address.

The paper begins with a crucial but often overlooked distinction between two meanings of the word 'evidence': First, evidence as 'mark' or 'sign' or 'symptom' of the hypothesis' being true, '*piece* of evidence'; here this will be referred to as evidence-1. Second, evidence as 'proof' or 'warrant' or 'good reason to believe', '*body* of evidence'; here this will be referred to as evidence-2.

Desiderata. We want a theory of evidence that:

(A) tells us what kinds of data constitute evidence-1;
(B) tells us the conditions under which we can reasonably assert to be in the possession of evidence-2;
(C) allows evidence-2 to come in degrees;
(D) is informative about the modality of the supported claim.

None of the existing theories fulfil these desiderata. Bayesianism, for instance, only tells us how to revise our beliefs *given we accept a datum as evidence*; it doesn't tell us what data to look for to begin with (*i.e.*, it doesn't satisfy desideratum (A)). Mill's methods tell us what data to look for but are limited to ideal situations and thus not informative about the strength of evidence-2 we possess in non-ideal situations (*i.e.*, it doesn't satisfy desiderata (C) and (D)). Similar criticisms can be made with respect to all major theories, including Achinstein's, error-statistics and Roush's.

The alternative proposed in this paper can be regarded as a development of Sherlock Holmes' famous dictum 'Eliminate the impossible, and whatever remains, however improbable, must be the truth' (Bird 2007). Causal relations have certain typical markers. For instance, they may issue in regularities, successful interventions or probabilistic dependencies. But these markers have alternative explanations. A regularity between two variables C (for 'putative cause') and E (for 'putative effect'), say, can be explained by a direct causal relation but also by measurement error, Berkson's paradox, common-causal structures, reverse causation, fraud and so on. Evidence for a causal relation, then, is either a 'typical marker' or any datum that helps to rule out alternative explanations for the observation of the marker. More precisely, I propose: Evidence-1 for a causal hypothesis h is a set of data that records either:

- a primary indicator of causation, or:
- an n-ary fact helping to eliminate an alternative account for an $(n-1)$ary fact.

The 'primary indicators of causation' are given by a list (which comprises all the 'usual suspects'):

- A correlation between *C* and *E*;
- *E*'s changing after *C* has been intervened on (invariance);
- A continuous process between *C* and *E*;
- *C* being a necessary and/or sufficient condition for, or universally associated with *E*;
- A mechanism in which if *C* were to change, *E* would change.

What it means to eliminate an alternative account and which alternatives to consider are contextual matters. How context matters – and, in particular, where to stop the regress, will be illustrated by means of two case studies on smoking and lung cancer and lithium and mania, respectively.

I then define evidence-2 (evidence as proof) as follows: A scientific community is in the possession of evidence-2 for a causal hypothesis *h* whenever it is in the possession of evidence-1 for *h* ruling out all known alternative accounts of the primary, secondary *etc*. facts that constitute the evidence-1 for *h*.

The paper finishes by showing how this idea can be applied to define various degrees of the strength of evidence (*e.g.*, 'proof' vs 'strong' vs 'weak evidence') and how these grades of evidence support causal claims of different modalities (*e.g.*, '*C* causes *E*' vs '*C* likely causes *E*' vs '*C* might cause *E*').

Reference

Bird, Alexander 2007, 'Inference to the Only Explanation', *Philosophy and Phenomenological Research* 74(2): 424-32