On the Origin of Hill’s Causal Criteria

Alfredo Morabia

The rules to assess causation formulated by the eighteenth century Scottish philosopher David Hume are compared to Sir Austin Bradford Hill’s causal criteria. The strength of the analogy between Hume’s rules and Hill’s causal criteria suggests that, irrespective of whether Hume’s work was known to Hill or Hill’s predecessors, Hume’s thinking expresses a point of view still widely shared by contemporary epidemiologists. The lack of systematic experimental proof to causal inferences in epidemiology may explain the analogy of Hume’s and Hill’s, as opposed to Popper’s, logic. (Epidemiology 1991;2;367–369)

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“The Environment and Disease: Association or Causation,” by Sir A. Bradford Hill,1 is a classic reference in epidemiologic research on assessing the causal nature of an observed association. As recently noted in a review by Susser,7 Hill codified in his paper a set of criteria that had been elaborated by several epidemiologists since 1955.1,2 Part of these criteria was applied in assessing the causal relation between cigarette smoking and lung cancer in the 1964 Report of the Advisory Committee to the US Surgeon General, Smoking and Health.7 The rationale was that a finding satisfying several criteria was more likely to be causal than one that satisfied only a few or none.

While reading David Hume’s A Treatise of Human Nature,6 I came upon the “rules by which to judge of causes and effects” (pp. 173–176) and was struck by their similarities to Hill’s criteria. To find analogous approaches to causality in contemporary epidemiologists and in an eighteenth century philosopher was puzzling. It raised the question of the philosophic roots of Hill’s criteria that, to my knowledge, has not been addressed heretofore. In addition, a potential link between Hume and Hill (read: between Hume and the generation of epidemiologists who contributed to the elaboration of the criteria efficiently summarized by Hill) suggested that there could be a parallelism between the Popper–Hume and the Popper–Hill controversies. The first controversy is now well-known to epidemiologists.9 The second controversy, initiated 15 years ago by Carol Buck,10 has generated a variety of criticisms to Hill’s criteria, ranging from rejection of the criteria reflecting inductive logic11 to rejection of the method itself.12

Here I compare the causal criteria formulated by Hume and by Hill. A first contrast is of historical nature. In 1740, empirical sciences were emerging, but the experimental method was not yet a system of research. Hume believed that observations were fragile bases for causal inferences and that the discovery of causes required an intellectual process going “beyond the impression of our senses.” He therefore proposed a set of rules to structure such a process. In contrast, Hill and his contemporary epidemiologist colleagues, concerned by the biases resulting from the “method of observation” in studying human populations,11 tried to define the circumstances in which we can infer causation from nonexperimental observation.

Methods
I have tried to match Hill’s and Hume’s criteria as they are expressed in their two publications.1,6 Because Hume’s treatise was published 225 years before Hill’s report, it is obviously impossible to get a perfect match. For example, Hume presented his rules as universal statements, whereas Hill’s criteria are worded specifically for preventive medicine. My goal, however, was to shed light on a potential philosophical kinship rather than on the formulation itself.

Results
The comparison of Hume’s and Hill’s criteria is summarized in Table 1.

The conceptual identity is striking for what Hill has called “temporality,” “biologic gradient,” and “consistency.” The temporality criterion is stated explicitly by both Hume and Hill: “The temporal relationship of the association—which is the cart and which is the horse?” Hume: “The cause and effect must be contiguous in space and time. The cause must be prior to the effect” (Rules 1 and 2).

The concept of gradient as a support for causal associations is also mentioned by both authors, but for

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Hume this is a sufficient causal criterion. Hill: "If the association is one which can reveal a biological gradient, or dose–response curve, then we should look most carefully for such evidence. . . . The clear dose–response curve admits of a simple explanation and obviously puts the case in a clearer light." Hume: "When any object increases or diminishes with the increase or diminution of its cause, 'tis to be regarded as a compounded effect, deriv'd from the union of the several different effects, which arise from the several different parts of the cause. The absence or presence of one part of the cause is here suppos'd to be always attended with the absence or presence of a proportionable part of the effect. This constant conjunction sufficiently proves that the one part is the cause of the other" (Rule 7).

Hill's second criterion relates to whether "the association [has] been repeatedly observed by different persons, in different places, circumstances and time?" The counterpart is Hume's concept of "multiplicity of resembling instances [that] constitutes the very essence of power or connexion" (not a specific rule but in the premises of the catalog, p. 163).

Strength of the association, as a measure of relative effect, does not have an exact complement in Hume's rules. Nevertheless, Hume's constant-conjunction formula is, just like the relative risk, a measure of association rather than of causation: "There must be a constant union betwixt the cause and effect. 'Tis chiefly this quality, that constitutes the relation" (Rule 3).

There is some resemblance between Hill's analogy criterion and Hume's sixth rule. Hill: "In some circumstances it would be fair to judge by analogy. With the effects of thalidomide and rubella before us we should surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy," Hume: "Where several different objects produce the same effect, it must be by means of some quality, which we discover to be common amongst them" (Rule 5). "Like effects imply like causes" (Rule 6).

It is possible to find in Hume's writing formulas that correspond to Hill's criteria of specificity and coherence. Again, the importance lies in the similarity of the intellectual approach rather than in the exact formulation. For example, Hume's "same cause always produces the same effect, and the same effect never arises but from the same cause" (Rule 4). "Like effects imply like causes" (Rule 6).

For historical reasons, two remaining criteria listed by Hill (biological plausibility and experiment) have no counterpart in Hume's set of rules. It would be an anachronism to find the concept of biological plausibility in the writing of a seventeenth century philosopher. Also, although a contemporary of Newton, Hume (1711–1776) was a preexperimental thinker. Experimental method became a system of research during the nineteenth century, after the works of Lavoisier and others. Indeed, John Stuart Mill, who agreed with Hume's approach on causation, emphasized the role of experiment: "Observation without experiment cannot ascertain sequences and co-existences, but cannot prove causation."14

Discussion
The strength of the analogy between Hume's and Hill's causal criteria suggests that, irrespective of whether Hume's work was known to Hill or Hill's predecessors,3–7 Hume's thinking expresses a point of view still widely shared by contemporary epidemiologists. The point is that Hume's criteria sound reasonable to us, and it is likely that Hill's criteria would have sounded reasonable to Hume. Does this conclusion illuminate the debate between Popperian and non-Popperian epidemiologists?

For Hume, there is no proof of causation. Cause-to-effect relations can be inferred from past experience, but such inferences must rely on the assumption that the course of nature will not vary. Since there is no proof that causally related events will still be related in the future in

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TABLE 1. Hill's Criteria* and Corresponding Hume's "Rules by which to Judge of Causes and Effects"†

<table>
<thead>
<tr>
<th>Hill's Criteria</th>
<th>Hume's Rules</th>
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<tbody>
<tr>
<td>1. Temporality</td>
<td>1. &quot;The cause must be prior to the effect&quot; (Rules 1 and 2).</td>
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<tr>
<td>2. Dose–response</td>
<td>2. &quot;When any object increases or diminishes with the increase or diminution of its cause&quot; (Rule 7).</td>
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<tr>
<td>3. Consistency</td>
<td>3. &quot;Multiplicity of resembling instances constitutes the very essence of power or connexion&quot; (not a specific rule but in the premises of the catalog, p. 163).</td>
</tr>
<tr>
<td>4. Strength of association</td>
<td>4. &quot;There must be a constant union betwixt the cause and effect&quot; (Rule 3).</td>
</tr>
<tr>
<td>5. Analogy</td>
<td>5. &quot;Like effects imply like causes&quot; (Rule 6).</td>
</tr>
<tr>
<td>6. Specificity</td>
<td>6. &quot;Same cause always produces the same effect, and the same effect never arises but from the same cause&quot; (Rule 4).</td>
</tr>
<tr>
<td>7. Biological plausibility</td>
<td>7. Not applicable.‡</td>
</tr>
<tr>
<td>8. Experiment</td>
<td>8. Not applicable.‡</td>
</tr>
</tbody>
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*Source: Reference 1.
†Source: Reference 8, pp. 173–176.
‡See Results section.

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the same ways, the validity of an inference from cause to effect can never be demonstrated.19

Popper has argued against Hume that the scientific community could temporarily consider as valid theories that had resisted repeated tests. By tests, Popper means experiments that can be reproduced—in principle—by anyone.16 As a matter of fact, there is historical evidence that such successful theories are likely to remain valid within the conditions of their discovery. A classic example is Newton’s laws of motion.17

Epidemiologists may agree with Popper that experiments are important tools to identify causal laws. Lind was a pioneer of the experimental method.18 Textbooks of epidemiology refer to Lind, Louis,19 and so on20 to highlight the historical roots of experiments in epidemiologic research. But, epidemiologists can hardly share Popper’s central thesis that only testable theories are scientific.11 Epidemiology deals with the characteristics of human populations and therefore is more an observational than an experimental discipline.15 Epidemiologists often have to infer causation without being able to bring objective proofs. For example, the smoking–lung cancer connection was elucidated although it was not amenable to controlled experimentation.

I suggest that Popper’s philosophy appears too restrictive for epidemiologists because it does not recognize the scientific character of theories that do not have potential experimental proof. Hume’s philosophy that proofs are elusive is also rejected by most contemporary scientists, but when proof is not available, pragmatic epidemiologists simply acknowledge that there is yet no alternative to the causal criteria logic that says, in substance: before inferring causation, it is imperative to check for illogicalities and rule out gross contradictions between what has been found and what we think we know. Hence, the analogy of Hume’s and Hill’s, as opposed to Popper’s, logic.

References


