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Discussion

Causes and Logic in Epidemiological Psychiatry

The paper by Paul Bebbington 'Causal Models and Logical Inference in Epidemiological Psychiatry', a revised form of an essay awarded the Bronze Medal of the Royal College of Psychiatrists and published in the *Journal* (April, 1980, **136,** 317–25) led to criticisms, invited and otherwise. A selection is published here.

1. THE NOTION OF CAUSALITY

By Rom Harré *(Fellow, Linacre College, Oxford)*

There are considerable difficulties in assessing the force of Dr Bebbington's argument, occasioned by problems with his way of understanding causality and its relation to the structure of sentences used to describe causal relations.

He demonstrates, correctly, that the classical truth-functional definition of the conjunction 'if . . . then . . .' is inadequate to represent the sense of a sentence in which the relation expressed in that form is supposed to represent a causal relation. That relation should hold between whatever is referred to in the phrase following 'if' as the cause and whatever is referred to by the phrase following 'then'. If I follow Dr Bebbington, he seems to want to explain the inadequacy by reference to some extra-logical feature of statements describing causal relations. The problem for traditional logic is posed by the existence of sentences such as his 'If Stalin was a communist then sugar is sweet'. This has to be admitted as a true statement since both of its components are true. But it certainly is not a causal statement. What features do causal statements have over and above statements like the one just cited?

According to Dr Bebbington causal statements must also somehow capture in words two features of real world causal relations, namely that causes must precede their effects, that is the asymmetry of the relation, and that causes must be sufficient (and sometimes necessary) conditions for the coming into being of anything that purports to be among their effects. But he leaves out the most important feature of all, that causes must produce their effects. This last condition is of the greatest importance since it forces us to treat causality as a dual criterion relation. The reasoning to this conclusion goes as follows: to satisfy ourselves that the item we are taking to be a cause is sufficient to produce the required effect we must be able to find many cases where items of that kind have preceded effects of that kind, and be able to explain away cases where they have not by reference to some plausible interferences from outside. From these considerations we can derive a *prima facie* criterion:

(i) An item of kind A is likely to be a cause of an item of kind B if whenever an item of kind A occurs it is followed by an item of kind B, *ceteris paribus*. (Notice that there is no need for us to demand that whenever an item of kind B occurred it must have been preceded by an item of kind A. There are more ways of killing a cat than choking it with cream, but if you choke it with cream it will surely die).

(ii) But to rule out coincidence or the possibility that both A-type items and B-type items are joint products of a third kind of item C, we

need a stronger, a definitive criterion. In natural science (and so too I believe in good psychological, medical and social science) this criterion is met by demanding a demonstration of or plausible hypothesis concerning a generative influence from items of kind A to items of kind B, usually through some generative mechanism. In fundamental science causation occurs through some basic system of influences such as electro-magnetic fields.

A simple example should make this dual criterion demand clear. To establish that smoking *causes* lung cancer one must not only establish a good statistical relation, that is satisfy the *prima facie* criterion, but also establish how smoking generates or produces cancers, that is satisfy the definitive criterion. The first criterion demands surveys and statistics and is based on the extensive design, the second demands biochemistry and physiology and is based on the intensive design, that is on the deep and detailed investigation of single cases treated as typical of the class of cases to be studied.

Before I turn to examine his case against Brown *et al* with a more powerful conception of causality to hand than that Bebbington wields in his discussion, I need to draw attention to a terminological inexactitude that Dr Bebbington is not alone in employing. He speaks in several places of causal 'models'. Now a model is a simulacrum of something; sometimes this takes the form of an idealization of its subject, sometimes it is an analogue. I am unable to identify either of these senses of 'model' with Bebbington's usage. He seems to me to mean 'hypothesis' when he speaks of 'model', for those things he calls causal models are various hypotheses as to what is causally related to what. I shall proceed then as if he is talking about hypotheses and not about models, since his remarks are meaningless if taken literally.

All of Dr Bebbington's arguments concern variations of the *prima facie* criterion, that is whether one should admit other concomitances into the story. So far as I can see his reasoning is impeccable. So we are driven to consider a variety of additional putative causal factors. And the one to pay attention to is an alleged 'vulnerability' factor. Bebbington points out, correctly I think, that the method of testing the hypothesis (not the model!) associated with this factor is defective. But then he and I must part company—for, instead of now condemning Brown *et al* for continuing to occupy themselves only with the *prima facie* criterion and the extensive design, he plunges into a further exercise in statistical analysis. Ironically he reaches the same conclusion that could be reached directly by an attempt to design an intensive test of the vulnerability hypothesis—'it remains unclear' he says, 'how the factors (of the vulnerability components) could operate as provoking agents'. But it remains unclear only if we shy away from the intensive step.

The next stage in a truly scientific programme of research would be to take two or three cases as types, each representative of the possibility that a vulnerability factor could be a provoking agent, and get down to brass tacks to see how in each particular case it worked. Then classes of cases could be reconstructed by defining each as the set of cases sufficiently like the case taken as the centre piece of an intensive design.

2. CAUSAL MODELS AND LOGICAL INFERENCE

By Neil Tennant *(Department of Philosophy, Edinburgh)* ***and Ian E. Thompson*** *(Research Fellow, Edinburgh Medical School)*

Bebbington's paper is an example of all that is bad when a scientist is over-theoretical about a common-sense discipline. The prefatorial excursus into propositional logic is both unnecessary and ill-informed. It is quite unclear how it improves understanding of epidemiological data or theory. It is historically inaccurate and philosophically muddled. Indeed, it threatens to take the 'logical' out of 'epidemiological'. It will have nasty effects on logicians who read it. At least one has, and is concerned to prevent further outbreaks.

Consider the breathtaking claim in the first paragraph that "(the) nature of epidemio-

logical data makes it impossible to draw inferences according to classical logic". Now classical logic has to do with inferences that preserve truth from premises to conclusions by virtue of the forms of the statements involved, and regardless of their subject matter—be it epidemiological or otherwise. So Bebbington's claim raises the immediate suspicion that he has misconstrued the logical operators when translating sentences of epidemiological English into logical notation.

And indeed he has. "Let p be a cause and q be an effect. If the causal connection can be represented in the form p→q, this implies that q *inevitably* follows from p . . ." (our emphasis). It certainly doesn't. That this is simply a howler can be read off from the truth table for p→q:

		q	
	→	t	f
p	t	t	f
	f	t	t

But more serious is the category mistake involved in letting event descriptions p and q flank the arrow. The arrow is in fact a connective, joining two sentences to make a new one. Later on, Bebbington construes it as a relation between statements (p. 319 col. 1)—which, with his talk of inevitability, leads one to wonder whether what he is after is not after all logical implication rather than material implication.

One hopes not. Since Hume we read no logical necessity into the causal relation. But in playing with the weaker material implication Bebbington burns his fingers with two howlers on p. 319. In both Example 2 and Example 3 he has the arrow going the wrong way. Also, for some bizarre reason he distorts 2×2 contingency tables such as

	Life event	No life event
Ill	30	10
Well	0	100

into structural analogy with the truth table by claiming that "the value 'zero' is the formal equivalent of 'false', and other values are the formal equivalent of 'true' ". This is just silly, and bears no relation at all to the computation of truth values for statements given the data.

How, then, *is* logic relevant to causality? Insofar as one cleaves to a notion of cause—and indeed there are some philosophers who maintain that it dissolves and drops out of any fully explanatory physical theory—there are two ways one might go about it. Both are ignored by Bebbington. The first is to analyse conditional statements such as

> If you light that match the gas will explode
>
> If you had lit that match the gas would have exploded

by means of more sophisticated arrows. These will be supplied with their own semantics and governed by their own axioms and rules of inference. There is now a large literature on this subject of indicative and subjunctive conditionals.

The second way is to treat causality as a non-logical relation between events:

> (His lighting the match) *caused* (the gas explosion)

and then seek an axiomatization of this relation like the mathematical axiomatizations of certain ordering relations, or the set-membership relation.

Both ways (*pace* Bebbington) leave classical logic untouched. In neither do we confuse, as Bebbington does, necessary and sufficient truth conditions of statements with necessary and sufficient causal conditions of events.

All that Bebbington's contingency tables boil down to is the atomic diagram of a finite data base. He uses certain one-place predicates: x is ill, x has had life event, x is a worker, . . . and so on, with their negations. So let the sample population be as large as one likes—the larger the better. Characterize each individual with the appropriate predicates or their negations:

> Smith is ill Smith has had a life event . . .
>
> Jones is ill Jones has not had a life event . . .
>
> .
>
> .
>
> .
>
> .

Then see what universal or statistical generalizations are true. The table tells all. Common

sense can do the rest. And classical logic will govern the unfolding of the story.

One wonders whether Bebbington has seriously examined Mill's *Methods of Experimental Enquiry* which he treats so dismissively (p. 319 col. 1). There is an obvious irony in the fact that he attempts to do with his contingency tables what Mill arguably did better with the Joint Method of Agreement and Difference, Method of Residues and the Method of Concomitant Variations. Mill attempts to provide us with practical criteria for determining necessary and sufficient conditions for given events and these are arguably more relevant to epidemiological psychiatry than Bebbington's dubious logical schemata and misleading formulations of the relation between causal and material implication.

It is not clear from his paper whether he regards the relation of causation as transitive, by analogy with material implication, or not. His remarks about weak implication (a notion which is foreign to logic) are confusing (p. 319 col. 2). He says on the one hand "If we consider the situation of three variables linked thus: $p \rightarrow q$ and $q \rightarrow r$, we may logically infer $p \rightarrow r$", but on the other hand he has previously suggested that '$p \rightarrow q$' be read as 'p causes q' (where p is cause and q effect) (p. 318 col. 1). Thus his previous statement might be understood as signifying 'p causes q, and q causes r, therefore p causes r', which is obviously an invalid inference. He does not indicate sufficiently clearly whether the relation of causation is to be understood as a 'weak implication' or not for in terms of his definition of 'weak implication' some may be transitive and others not.

There is a similar confusion in his discussion of causality in terms of necessary and sufficient conditions (p. 318 col. 2). It is logically valid to assert that p states a *sufficient condition* for another proposition q if "p implies q" is true, and that p states a *necessary condition* for another proposition q if "not-p implies not-q" is true (or, what is the same thing, if "q implies p" is true). However, it is incorrect to assert as Bebbington does that "it is possible to conceive a cause as being logically both necessary and sufficient, that is both $p \rightarrow q$ and $q \rightarrow p$, or as it is sometimes written $p \leftrightarrow q$". We cannot say that p causes q and q causes p without doing violence to the semantics of cause and effect which distinguish the former from the latter in terms of temporal priority and efficacy or agency. Causal relations cannot be symmetrical even if under certain conditions relations of implication or necessary conditions may be. They do not translate into each other. There is incidentally no evidence that the founding fathers of logic whom Bebbington mentions confused causality with implication, and the main one, Frege, (conspicuously absent from Bebbington's list) certainly did not confuse them.

Bebbington fails to distinguish clearly enough between the Law of Universal Causation, particular causal laws, and instances of particular causal connections which could be said to fall under those laws. Which "concept of causality is partly one of imposed meaning"? Does "the nature of causality as a metaphysical proposition" embrace all three types of causal statement? His appeal to the authority of Popper does not save his argument from confusion, it only compounds it, for Popper dismisses the possibility of legitimate inferences from particular causal statements to more universal ones, while Bebbington's approach tends to demand such inferences.

Finally it may be questioned whether the present stage of data-collection and statistical analysis of results in psychiatric epidemiology is correctly to be seen in terms of causal models. In the absence of agreed causal laws it is doubtful whether deductions of causal connections can be inferred. In the scientific sense of cause one can only say that an event A causes an event B if there is a law, L, such that from the conjunction of L and a description of A the occurrence of B is logically deducible. It would seem that argument in psychiatric epidemiology is at a stage preliminary to this where researchers are still in the process of trying to formulate particular causal laws, as psychiatrists are busy seeking to define psychiatric illnesses more precisely. Definitions of disease are not true or false, but more or less useful, and consequently disease classifications should not be seen as causal systems, but as predictive systems. Given the present state of knowledge and agreed theory in psychiatry it may well be more appropriate to

see the attempt to arrive at an analysis of significant correlations between psychiatric illness, social class, life events etc. in terms of traditional inductive and statistical procedures.

3. CAUSAL MODELING WITH CONTINGENCY TABLES

By D. J. Cooke *(Psychology Department, Gartnavel Royal Hospital, Glasgow)*

Bebbington's recent paper (1980) is a valuable contribution towards unravelling some of the problems of causal models in epidemiology. Bebbington contends that two-value logic may be applied to elaborate causal hypotheses in epidemiological psychiatry. He argues that the 2×2 contingency tables, commonly used in psychiatric epidemiology, represent the relationship between two variables in a manner analogous to the truth tables of formal logic. Through the examination of multi-dimensional contingency tables he highlights the difficulties in describing causal processes in psychiatric epidemiology in general, and life-event work in particular. Despite these difficulties, he argues that the method may lead to the corroboration of one causal model rather than another.

Nowak (1960, 1976) indicated that causal analysis of empirical data, based on the principles of truth tables, is fraught with difficulties. Truth tables depend on dichotomous classification. Pearson (1957) argued that dichotomization is often merely a crude procedure for handling continuous variables. Blalock (1964) indicated that while it is technically possible to think always in terms of dichotomies difficulties may arise because they are frequently the result of an essentially artificial cutpoint in some quantitative variable.

If dichotomies are based on essentially arbitrary divisions of continuously distributed variables then problems may occur, firstly in the assignment of entries to the truth table, and secondly in the elaboration of causal inferences from these entries.

Blalock (1964) illustrated these difficulties. Given two continuous variables, one a hypothetical causal agent and the other its postulated effect then the best fitting curve, be it linear or non-linear, represents a continuous causal function linking the postulated cause and effect.

For the purpose of illustration the regression line is plotted in Fig 1, sample fluctuation being ignored. Examination of Fig 1 illustrates that our decision regarding the arbitrary point at which to dichotomize the variables dramatically influences the entries in the truth table and the resulting causal inferences made.

If fortuitously the intersection of our dichotomizing lines falls precisely on the regression line then Fig 1a results. When the cause is absent the effect is also absent, when the cause is present the effect is present. The resulting truth table implies that the cause is both necessary and sufficient.

If, however, the intersection of the dichotomizing lines crosses above the regression line then different entries appear in the truth table (Fig 1b). In this case when the cause is absent, the effect is absent, when the cause is present, however, the effect only appears spasmodically. The resulting truth table suggests that the cause is necessary but not sufficient. When the intersection of the dichotomizing lines falls below the regression line a new set of entries is placed in the truth table. When the cause is absent the effect appears spasmodically, however, when the cause is present the effect is always present. This truth table implies that the cause is sufficient but not necessary.

Sampling fluctuations and other factors entailed in any empirical distribution will tend to result in truth tables of the form 1d. This table, the most common form in psychiatric epidemiology (Susser, 1973), suggests that causes are neither necessary nor sufficient.

It must be contended, therefore, that the entries in tables and the influences made from them are critically influenced by essentially arbitrary decisions regarding the point of dichotomization of continuous cause and effect variables. Even given that empirical data will inevitably result in truth tables without empty cells, the relative proportion of the entries in the

ILLUSTRATION OF EFFECT THAT ARBITRARY DICHOTOMISATION OF CONTINUOUS VARIABLES HAS ON CAUSAL INFERENCE

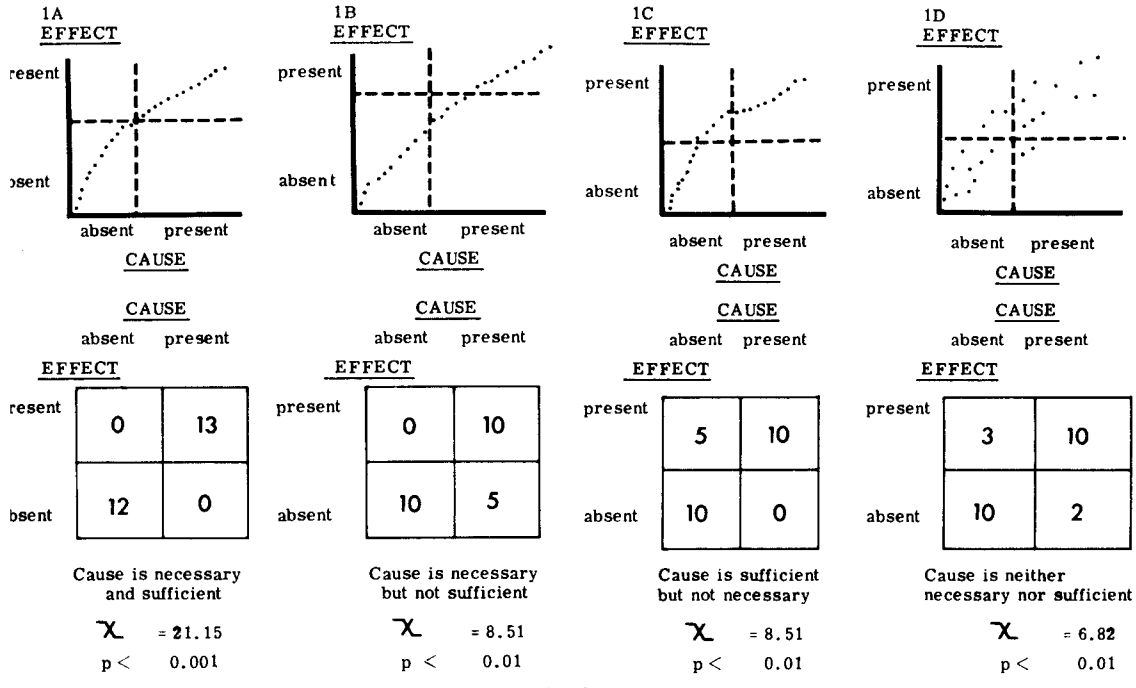

FIG 1.

cells critically affects inferences made about causal processes.

The above argument clearly entails the assumption that cause and effect are best conceptualized as continuous variates and not attributes. The validity of this assumption in the context of the example used by Bebbington will be considered.

Bebbington applied his principles of analysis to the data published by Brown *et al* (1975). Brown *et al* (1975) initially considered the link between the experience of life-events (postulated cause) and caseness (postulated effect).

Brown and his colleagues dichotomize their subjects into those who have experienced no severe life events and those who have experienced one or more severe life event. The relevant question is whether the effect of life events is additive, that is, is the risk of becoming a case greater for a subject experiencing three events rather than for one experiencing one event? Brown and Harris (1978), commenting on this issue, suggest that their data support a modest additive effect even although, "our method of measuring threat probably rules out finding an 'additive effect' should one exist" (p. 109).

Miller and Ingham (1979) and Surtees and Ingham (1980), using similar threat measures, on different populations, identified modest additive effects. This implies that the hypothetical causal variable is continuous in nature. Dichotomizing event frequencies into zero and non-zero, while it may be convenient, is probably an essentially arbitary division of a continuous variable.

Brown and his colleagues used the Present State Examination (Wing *et al*, 1977), as their case defining instrument. Using this instrument they defined 'cases', 'borderline cases', and 'non-cases'. In their analysis, however, they used a dichotomy including the borderline cases with the non-cases. They noted that "There is evidently an arbitary element in choosing a cut

off point between a case and a borderline" (p. 229) (Brown *et al*, 1975). The use of the 'borderline' category and the admission that the cut-off is essentially arbitary implies that Brown and his colleagues construe 'caseness' as being to some extent continuous. Williams *et al* (1980), in their review of case definition problems in psychiatric epidemiology, contended that caseness should be viewed as a continuum: psychiatric patients acting as the caseness criterion, with the case-finding instrument measuring the subject's closeness to this criterion. Duncan-Jones and Henderson (1978) provided empirical support for this continuum view by demonstrating that G.H.Q. scores are related by a sigmoid curve to probability of "caseness". Caseness measure would appear, therefore, to be underpinned by a continuous variable. The event frequency and caseness measures used by Brown and his colleagues are clearly dichotomized at arbitrary points. It could be that their inferences are stable for different cut-off points. This, however, remains an empirical question.

Bebbington's paper provides a valuable theoretical description of a vulnerability factor. The use of contingency and truth tables, however, does not appear to provide a reliable way of modelling this concept. As Blalock (1964) notes "The simplicity and other obvious advantages of 2×2 tables should not blind us to its defects" (p. 124). Multivariate techniques may provide more flexible procedures for modelling the concept of a vulnerability factor.

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4. FURTHER COMMENTS ON THE VULNERABILITY MODEL

By G. W. Brown and T. Harris *(Department of Sociology, Bedford College, London)*

(Abridged: A fuller version available from the authors)

Dr Bebbington earlier criticized our causal model (Brown and Harris, 1978b); as we assumed an additive (not multiplicative) interaction our model was statistically fully justified (Everitt and Smith, 1979). He now argues that three predictions must be met for our model to be confirmed and that the third is not met, i.e. that "There will be an association between life events and vulnerability factors *only* in the cases"; this is an unnecessary stipulation. A critical consideration is that there is such an overwhelming association between the onset of depression and provoking agents that there are only four instances in the community survey in Camberwell where there is depression without such an agent. It is therefore understandable that a statistically significant association is not achieved in the left hand tabulation of his Table 4.

The error of his assertion can be seen in the

situation where provoking agents are a necessary condition for depression. This would mean that there could be no association between vulnerability factors and provoking agents on the left of Table 4 as no one would be without a provoking agent. However this situation is consistent with our model. Bebbington also claims that, for normal women, there should be *no* association. In the example he gives from our data this holds, but there is no valid logical basis to this claim when dealing with one vulnerability factor *on its own*. Although it happens that there is no association for the vulnerability factor of early loss, he would have found such an association if he had carried out a calculation on the vulnerability factor of intimacy. The Table below (taken from Table I, p. 177 of our book *Social Origins of Depression*) repeats his crucial test of the third prediction using this variable; although the small number of cases without a provoking agent militates against a significant result for the cases, one does emerge for the normal women, and it is in no way far-fetched to relate this to the class differences we know about in Camberwell. Working class women tend to experience an intimate relationship with a husband or boyfriend less often especially when they have young children, and they also have a higher rate of severe events and major difficulties.

This result is possible because there are *other* vulnerability factors not taken into account. It is quite possible to explain why at least some of the 52 non-cases with a provoking agent who are also low on intimacy did not become cases, by hypothesizing that they did not have other factors which could have summated to increase vulnerability. Indeed, something like this appears to occur, because the association in the normal group is non-significant when vulnerability is scored in terms of an index accounting for *all* our vulnerability factors.

	Intimacy of cases		Intimacy of normals	
	Low	High	Low	High
Provoking agent	24	6	52	79
No-provoking agent	2	2	60	191
	$\chi^2 = 0.88$, ns gamma .45		$\chi^2 = 10.36$, $P < .01$ gamma .35	

To summarize, our model has no logical implications at all for significance patterns for the left or right hand side of the Table. In the end it is only in combination with the size of the marginal totals that these patterns of significance have implications for the relevance of the vulnerability-factor model. We are therefore puzzled by his assertion that, although our interpretation is intuitively the most satisfactory model, "it can only be salvaged by adducing arguments which do not arise in the analysis". Wider considerations must be taken into account. It is always necessary to include considerations outside any logical or mathematical system one might be utilizing. Stephen Toulmin has discussed the cult of systemacity: that is the belief that concepts best form 'logical systems'. As we understand his argument there has been a long and largely misguided tradition in science and philosophy that has sought to solve arguments in terms of self-validating logical schemes —the prime examples of such an outlook are, of course, the universal and authoritative systems found in logic and geometry. The danger of such an approach is that one may draw conclusions which are of little relevance for the real world, where probability is as important as the dichotomy between truth and falsehood.

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