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Evidence, Inference, and Purpose

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All univocal analyses of causation face counterexamples. An attractive response to this situation is to become a pluralist about causal relationships. "Causal pluralism" is itself, however, a pluralistic notion. In this article, I argue in favor of pluralism about concepts of cause in the social sciences. The article will show that evidence for, inference from, and the purpose of causal claims are very closely linked.

Keywords: *causation; pluralism; evidence; methodology*

I

In recent years, philosophers have slowly come to realize that the marginal benefit of continuing the quest for a monistic account of causation, an account that provides a characterization of a single set of features that distinguishes all causal from noncausal relations, is very low indeed. Many have responded by becoming pluralists about causation in one way or another (versions of causal pluralism are defended by Campaner and Galavotti 2007; Cartwright 1999, 2007; De Vreese 2006; Godfrey-Smith forthcoming; Hall 2004; Hitchcock 2003; Psillos forthcoming; Russo and Williamson 2007; and Weber 2007, among others).

Causal pluralism is, however, itself a pluralistic notion: there are many different kinds of it, and different versions differ greatly with respect to plausibility (for a classification, see Hitchcock 2007). In this article, I argue in favor of pluralism about concepts of cause in the social sciences. The argument proceeds by showing, first, that counterexamples to the different accounts of

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causation have led a number of researchers to become pluralists about *evidence* for causal claims. This is a position that social scientists should find very attractive because of the wealth of alternative methods used to establish causal claims throughout the social sciences. I then show how evidential pluralism leads to pluralism about the concept of cause, at least prima facie. Next, I consider a possible rescue for the conceptual monist, namely, to claim that possessing causal knowledge of one type allows the *inference* to other types of causal knowledge, thereby unifying prima facie different concepts. I reject this attempt. Last, I show that social scientists' different *purposes* require different types of causal knowledge. In sum, evidence for, inference from, and purpose of causal claims are tied together very closely.

II

A large variety of accounts of causation, each aspiring to be a candidate for the one true theory, can be found in the philosophical literature. The starting point for this article is the observation of what I take to be a fact: every account of causation, when offered as a universal theory of what causation consists in or what we mean by the word *cause*, is false because it is subject to counterexamples. In this section I give the reader a flavor of this fact.

I will present kinds of counterexample for both the necessity and the sufficiency of definition provided by each account. While it is certainly the case that each type of theory can be improved such that it ceases to be subject to many specific counterexamples I list here, I claim on inductive grounds that one can reformulate the counterexample in such a way that the new theory fails. For more detail on each type of theory and the recalcitrance of the associated counterexample, the reader is referred to the pertinent literature.

Counterfactual accounts. Both historians interested in singular causal analysis and many legal theorists frequently identify causation with some form of counterfactual dependence. For example, in his famous essay "Objective Possibility and Adequate Causation in Historical Explanation," Max Weber (1905/1949, 171) wrote,

> Rather, does the attribution of effects to causes take place through a process of thought which includes a series of abstractions. The first and decisive one occurs when we conceive of one or a few of the actual causal components as modified in a certain direction and then ask ourselves whether under the conditions which have been thus changed, the same effect . . . or some other effect "would be expected."

Weber's essay is no historical anomaly. In recent years, one could witness an explosion of contributions to so-called virtual, alternate, or "what if?" history (e.g., Tetlock, Lebow, and Parker 2006; Cowley 1999), and while some of these are certainly written mostly for entertainment, others (see in particular the collection of Tetlock and Belkin [1996], which includes a thorough methodological discussion of this technique) have genuine cognitive purposes: historians construct counterfactual scenarios, such as a United Kingdom in 1938 without appeasement policy, a Cuba crisis in which Kennedy shows greater resolve, or a Persian victory at Salamis, to identify the cause or causes of certain events of interest (in these cases, the Second World War, the Soviet deployment of missiles, the rise of the West). Likewise, *economic* historians sometimes identify the causes of singular events (e.g., 19th-century American economic growth) by counterfactually removing a potential causal factor (e.g., the introduction of the railroad) and examine whether the outcome would have been different (Fogel 1964). In some cases, the set of qualitative causes of an outcome of interest is uncontroversial, and the real issue lies with which of that set has more (quantitative) explanatory relevance. Robert Northcott's (2008) analysis of "weighted causal explanations" also employs a counterfactual criterion.

In many areas of the law, similarly, causes of events are identified using the "but for" or *sine qua non* criterion: the claimant has to prove that but for the alleged conduct of the defendant, the harm would not have occurred (for criminal law, see for example Card [2006]; for tort law, McBride and Bagshaw [2005]; for a detailed criticism of this criterion, see Hart and Honoré [1985, chap. 5]).

However, some well-known cases show that causation cannot be identified with counterfactual dependence. Cases of so-called redundant causation in which a number of potential causes compete in bringing about an effect show that counterfactual dependence is not necessary for causation. If some factor C would have brought about Y in the absence of X but due to X's presence was prevented from doing so, X can be a cause of Y, but Y would have happened even in the absence of X.

To show that counterfactual dependence is not sufficient for causation is more subtle. Counterfactual statements are ambiguous in at least one important respect. Consider an example David Lewis (1979, 456) discusses:

> Jim and Jack quarreled yesterday, and Jack is still hopping mad. We conclude that if Jim asked Jack for help today, Jack would not help him. But wait: Jim is a prideful fellow. He never would ask for help after such a quarrel; if Jim were to ask Jack for help today, there would have to have been no quarrel

yesterday. In that case Jack would be his usual generous self. So if Jim asked Jack for help today, Jack would help him after all.

Lewis, and many others after him, resolve this tension by demanding that counterfactuals be *nonbacktracking*—evaluated by inserting a small miracle just before the cause obtains and changing nothing but the occurrence of the cause and its effects. But as I argue in a series of papers, this is only one way of evaluating a counterfactual claim, and not necessarily the best for all purposes (see Reiss and Cartwright 2004; Reiss forthcoming). In particular, it seems that historians, when addressing questions of the kind "What would have happened to *Y*, had *X* not happened?" often ask what conditions would have had to be present for *X* not to obtain and thus evaluate a backtracking counterfactual. Evaluating counterfactuals in this way, however, creates a series of counterexamples (Reiss forthcoming).

Regularity accounts. Qualitative comparative analysis (QCA) is a method that uses Boolean algebra for the qualitative study of macro-social phenomena and has been applied to fields as wide-ranging as sociology, political science, economics, and criminology (for a full list of applications, see the bibliographical database at www.compasss.org). It identifies causes of phenomena of interest (e.g., ethnic political mobilization among Western European minorities; see Ragin 1998) by first arranging all observed instances (in this case, minorities) in a table and determining whether the phenomenon is present. Then a list of factors (in this case, size, linguistic ability, wealth relative to core region, and population growth) is constructed, and it is noted whether each factor is present or absent. A factor is judged to be a cause whenever it is a member of a group such that that group of factors is always associated with the phenomenon of interest and no subgroup is always associated with the phenomenon. In other words, a factor is judged to be a cause whenever it is an INUS condition, that is, an insufficient but nonredundant part of an unnecessary but sufficient condition (Mackie 1974).

That regularities are not sufficient for causation is demonstrated by Mackie himself. It is easy to verify that the sounding of the Manchester hooters at 5.00 p.m. is an INUS condition for the Londoners to leave work shortly thereafter, but of course the Londoners do not leave the factory because of the sound of the Manchester hooters (see Mackie 1974, 81-84). That regularities, even those of the complex INUS type, are not necessary for causation can be shown by considering indeterministic cases of causation of the kind one finds in quantum-mechanical phenomena, at least according to some interpretations.

Probabilistic accounts. Methods aimed at inferring causal relations from statistical analyses of data abound in quantitative social sciences such as econometrics, quantitative sociology, and political science. Many of the applications are based on the classical linear regression model or extensions of it:

$$y_i = \alpha + \beta x_i + u_i,$$

where y is the dependent variable, α and β are regression parameters, u is an error term, and the subscript $i = (1, 2, 3 \ . \ . \ . \ )$ denotes the observation number.

The main idea behind it is that causes should be correlated with their effects (x and y are correlated if and only if β differs from zero in the regression): if money is a genuine cause of nominal income, or the availability of drug addiction rehabilitation a genuine preventer of recidivism, the observation of one of these variables should be informative about the likely value of the other. It is clear, however, that not all correlated variables are also related as cause and effect. To recite a philosophers' stock example, a change in the barometer reading is informative about the occurrence of a storm, but both are in fact caused by a change in atmospheric pressure. The standard solution to this problem is to "hold fixed"—condition upon—certain background factors that may affect the probability of the putative effect. In the regression model, it means to include these background factors as additional independent variables.

Nevertheless, counterexamples are not difficult to find. Even though many genuine causes will be correlated with their effects, some are not. If a factor affects another via two independent routes, the individual causal influences can cancel such that there is causation without correlation. Two monotonically increasing time series can be correlated, even conditional on putative common causes, when there is no causal relationship between them (see Sober 1987, 2001). Monotonically increasing time series are said to be "nonstationary"; there are many other sources of nonstationarity, and the bulk of time series in the social sciences has this property so that correlations are seldom indicative of a causal connection alone, if at all (see Reiss 2007b). Correlation is therefore neither necessary nor sufficient for causation.

Mechanistic accounts. Another connotation of causation social scientists employ for a variety of purposes is mechanism. If X causes Y, we would expect there to be a mechanism from X to Y such as the transmission mechanism from changes in the money stock to nominal income or the "self-fulfilling prophecy" mechanism by which bad news may cause bank

runs. The main idea is that causal relations between social variables can be decomposed into parts such that it can be shown how the causal message is transmitted from cause to effect. A related method of causal inference in the social sciences has been called "process tracing." Daniel Steel (2004, 67) summarizes it as follows:

> Process tracing consists in presenting evidence for the existence of several prevalent social practices that, when linked together, produce a chain of causation from one variable to another. A successful instance of process tracing, then, demonstrates the existence of a social mechanism connecting the variables of interest.

The trouble is that there are ranges of cases of apparent causation in which no such mechanism can be found. In an example due to Ned Hall (2004), a villain poses a threat to an air traffic controller who was about to send a signal to two planes on a crash course. As it happens, the planes crash, because of the threat. But no process or mechanism connects the two events. In cases of omissions, such as the failure of a government to protect its population against floods, there is no connecting mechanism either—because there is no event of the right kind to begin with. Such cases are of great importance in the law, especially tort law and criminal law (see for instance Pundik 2007).

Depending on how precisely to cash out the meaning of "mechanism of the appropriate kind," there are various problems with sufficiency too. According to one understanding, a mechanism is merely a series of (spatiotemporally contiguous) events X, C_1, C_2, . . . C_n, Y such that the transition from each element to the next is governed by one or more laws (see Little 1991, 14). Here one may encounter problems due to the lack of transitivity of some such relations. A stock philosophical example is as follows: the falling boulder causes me to duck, the ducking causes my survival, but the falling boulder does not cause my survival. In the social sciences, threshold effects can pose problems of this kind. For example, it may happen that X causes Y in the sense that some changes in X affect Y, and Y causes Z in that sense, but X does not cause Z because the changes that X induces in Y are not large enough (i.e., remain below the threshold) for Y to affect Z.

Interventionist accounts. A final intuition about causation I want to discuss here is the idea that one can often use causal relationships as recipes for change (e.g., Gasking 1955; Woodward 2003). If, say, money really does cause nominal income, it should in principle be possible to use that relationship to stabilize the economy; or if addiction programs really do prevent recidivism, governments should be able to reduce the latter by investing in the former.

Many social scientists think of this meaning when thinking about causation. In their influential textbook, Thomas Cook and Donald Campbell (1979, 36) write,

> *The paradigmatic assertion in causal relationships is that manipulation of a cause will result in the manipulation of an effect.* This concept of cause has been implicit in all the foregoing examples, and philosophers claim that it reflects the way causation is understood in everyday language. Causation implies that by varying one factor I can make another vary.

The econometricians' notion of superexogeneity is based on this conception (see Engle, Hendry, and Richard 1983), and so is Kevin Hoover's *Causality in Macroeconomics* (2001).

But not all causal relationships are manipulable by us to effect change in this way. Especially relationships in the social world can be fragile in the sense that no matter how "surgical" the intervention is, it will break after the intervention. The history of the Phillips curve (on a causal reading of it) illustrates this issue: the inverse relationship between unemployment and inflation, which had been more or less stable for over a century and at the time was understood as causal rather than epiphenomenal, broke down after attempts to exploit it for policy. Of course, one can always argue that the type of intervention used in this case was not "of the appropriate kind." But as long as our causal knowledge is supposed to help with the cognitive and practical purposes we pursue, this response has little bite. In the social sciences, we require real rather than ideal interventions (pace, in particular, Woodward 2003).

Similar problems beset the sufficiency of the condition. Though it can be proved that invariance under an ideal intervention identifies causal relationships in certain kinds of system (see Cartwright 2007, chap. 10), such systems are rare (at any rate, not all systems are of the right kind); in other kinds of system, we will always have to make do with real rather than ideal interventions, and these may lead to spurious results. Clearly, for instance, if the intervention affects the putative effect via a route that does not go through the putative cause, a joint change in the two variables is not necessarily indicative of a causal relationship.

III

The problem the mentioned counterexamples point to is a difficulty for these accounts of causation to the extent that they are thought of as universal

theories of causation, that is, as providing necessary and sufficient conditions for causation. One possible and straightforward response is to loosen the relationship between causation and what one might call the "manifestations" of causation such as counterfactual dependence, correlation, stability under intervention, and so on. The manifestations of causation, according to this response, are not regarded as *defining* causation or as expressing *characteristics universally associated with* causal relationships but rather as providing *evidence* or *test conditions* for the existence of causal relationships. The relation between causation and its manifestations can thus be regarded as roughly equivalent to the relation between theoretical entities in science and their observable counterparts. Few philosophers today would hold that theoretical entities are defined in terms of their observable manifestations. Nevertheless, observations can be evidence on the basis of which we infer the existence of and facts about the unobservable theoretical entity.

And of course, there are different sources of evidence for theoretical claims, just as there are a number of different kinds of evidence for causal relations. Some philosophers and social scientists are thus led to what one might call evidential pluralism about causation (this term seems to be due to Russo [2006]; however, I would also list John Gerring [2005], Paul Thagard [1999], and Jon Williamson [Russo and Williamson 2007] as holding this view).

The idea behind evidential pluralism is that evidence of a variety of kinds—say, probabilistic, mechanistic, regularity—can bear on a causal hypothesis and strengthen it. Especially when evidence from two or more different sources speaks in favor of the hypothesis, our confidence in the hypothesis should be boosted. Given what was said above, the rationale behind this kind of thinking is straightforward. Since any given method is fallible—as shown by the counterexamples to the various accounts—the epistemically responsible strategy is to bring as much evidence as possible to bear on the hypothesis at stake, and confirmation from a number of independent methods is one and perhaps the only way to be reasonably confident about the truth of the hypothesis. The idea, then, is pretty much like the idea of "triangulation" in other parts of science. One way to deal with the problem of unreliable measurement instruments is to try to use a number of physically different instruments such that if the result persists it cannot be an artifact of any of the instruments used as it would be highly unlikely that two or more physically different instruments produce the same kinds of artifacts.

IV

While a lot is to be said about this form of pluralism about causal relations it seems to involve an important presupposition that I will scrutinize in this section. The presupposition is that the connection between the concept of cause and its manifestations or test conditions must be *loose enough* for evidential pluralism to work. Evidential pluralism could not work if every evidential method defined its own concept because when moving from method to method we would in fact change the hypothesis to be tested. If (say) "*X* causes *Y* (as supported by probabilistic evidence)" means something different from "*X* causes *Y* (as supported by mechanistic evidence)," evidential pluralism does not get off the ground because instead of having one hypothesis that is being supported by two sources of evidence, we in fact have two separately supported hypotheses. In other words, we cannot be operationalists about the concept of cause. Rather, we require an independent concept of cause that, nevertheless, bears some systematic relationship with different evidential methods.

A version of this type of position is defended by Jon Williamson (2006a; but see also Russo and Williamson 2007; Russo 2006; Gerring 2005). Williamson believes that there is a single, independently understood concept of cause that can be employed in hypotheses scientists confirm on the basis of the different evidential methods. He defends an epistemic theory of causation that takes an epistemology of rational belief as its starting point. Evidence determines which causal beliefs the agent should adopt. The causal relation is then given by the set of causal beliefs that an agent with total evidence should adopt (Russo and Williamson 2007; cf. Williamson 2005, chap. 9, 2006a, 2006b, 2007). Thus, for example, an agent might initially believe that two variables are causally connected because of an observed correlation; however, she later learns that there is no possible mechanism in between the two variables and thereby is led to revise her earlier belief and so forth.

Unfortunately, there is a problem with the combination of conceptual monism and evidential pluralism: there are ranges of cases where it does not work. To see this, consider the causal hypothesis "Watching violent TV programs causes violence" (the example is entirely fictional; I use it to make a conceptual, not an empirical, point). Suppose, then, that we follow the strategy described above and first look for probabilistic evidence. Let us assume that the consumption of violent TV programs (*X*) and violence (*Y*) are indeed correlated and that all noncausal sources of correlation (such as nonstationarity) can be controlled for. For simplicity, let us further assume

that there are good reasons to believe that causation does not run from *Y* to *X*. However, as is common in social science, not all common causes are known or measurable, and thus we cannot distinguish between "*X* causes *Y*" and "*C* affects both *X* and *Y*, and *X* does not cause *Y* directly," where *C* is a common cause, on the basis of probabilistic evidence alone. The evidential pluralist now has us turn to a different kind of evidence, such as mechanistic evidence, for help.

Now, suppose we find such evidence. For instance, it may be possible to study some individuals with enough detail such that a psychological mechanism, according to which, say, consumers identify with aggressive characters and come to think of the depicted scenarios as realistic, which then results in more violent behavior in real-life situations, can be established. Does this confirm the initial hypothesis?

In some sense, yes. But only if the meaning of the word *cause* in our hypothesis is as ambiguous as "cause in some sense or other." This is because what has been said so far about the case is entirely compatible with the existence of a second psychological mechanism, present in other individuals (say), such that in these individuals TV consumption acts as a deterrent, resulting in *lower* violence. In the relevant population these two mechanisms might just cancel so that in that population the two variables are uncorrelated.

Of course, we still need to account for the correlation in the overall population. In the example, this may be due to an unobserved common cause such as, say, socioeconomic status. Within each socioeconomic stratum, TV consumption and violence are uncorrelated. This is because within each stratum the influences from the positive and the negative mechanism cancel. The correlation in the total population is brought about by a common cause, but we cannot learn this from the statistics because the common cause is not measurable (or not measured).

It may be argued that the situation described in the example is unlikely to happen outside a philosopher's armchair because a fair amount of exact canceling has to occur, and the chances for that to take place are very low. Now this may well be so. But the point I am making here is conceptual, not empirical. It may be an *empirical* truth that normally when mechanisms operate in a certain way, their operation will show up in statistical data, so that the two kinds of causation go together. This is, however, an empirical truth that has to be discovered a posteriori, not a truth we should build into the *concept* of causation.

To repeat this point, suppose we start out with a vague idea of what "causes" in the hypothesis "Watching violent TV programs causes violence" means—nothing more than, say, "brings about," "affects," "is responsible for," and its other cognates. Once we turn to evidential support for the hypothesis, the term acquires a more determinate meaning such as "in a population that is causally homogeneous with respect to violence, the variables 'TV consumption' and 'violence' are correlated." Another method defines another concept: establishing that there is a mechanism from TV consumption to (greater) violence establishes just that: for some individuals, TV consumption and violent behavior are connected by a psychological mechanism. Of course, the two are not entirely unrelated: if this mechanism is the only one that connects the two variables, we would expect the variables to be correlated as well. Likewise, if (in the relevant population) this type of mechanism can be found in many more individuals than countervailing mechanisms, we would expect a correlation. But these are statistical arguments, pertaining to populations, not individuals and have little to do with the mechanistic understanding of "cause."

To summarize, evidential pluralism of the kind defended by Williamson and others presupposes that evidence produced by different methods can be brought to bear on the same causal claim. But this does not always seem possible. In our example, the hypothesis we can hope to establish or reject on the basis of statistical evidence is a probabilistic one: in a causally homogeneous population, is violence correlated with the consumption of violent TV programs? (Answer in the example: no.) Using mechanistic evidence, by contrast, we can hope to establish or reject a mechanistic hypothesis: is there, in some individuals, a continuous mechanism from "input variable"—TV consumption—to "output variable"—violence? (Answer in the example: yes.) Conceptual monism is therefore, at least prima facie, false.

V

Causal claims are associated with certain inference rules that the competent user of the claim is licensed to make. What I mean by "licensed to make an inference" is that there are good reasons to believe that the inference rules are reliable for the purposes envisaged by the user. If, for example, a user competently claims that a certain training program causes a certain educational achievement in the probabilistic sense, say, he is entitled to infer that the claim holds not only in the population studied but also

in populations that differ in no causally relevant detail but that have not, thus far, been studied.[1]

As has been argued in the previous section, the meaning of causal claims is constrained by the type of evidence put forward in their favor. Perhaps it is possible to lift that constraint to some extent by showing that although a given causal claim was initially established on the basis of one type of evidence, the inference rules a competent user is licensed to make are more encompassing. For instance, it may be the case that if *X* is an INUS condition for *Y*, then if a user claims that *X* causes *Y* in that sense, he is licensed to infer that there is also a mechanism between *X* and *Y* or that one can intervene on *X* to change *Y*. If what was said in section II is correct, however, then this is not so. Here are some examples of inferences that are *not* automatically licensed:

- Knowing that *X* makes a difference to *Y* does not automatically allow the inference that there is a continuous process between *X* and *Y*.
- Knowing that *X* and *Y* are connected by some causal process does not automatically allow the inference that *X* raises the probability of *Y*.
- Knowing that *X* raises the probability of *Y* does not automatically allow the inference that we can control *Y* via *X*.

Let us go through these examples in slightly greater detail to show the relevance for causation in the social sciences.

1. Does knowing that X is a difference-maker to Y allow the inference that there is a continuous process between the two variables? In a great variety of legal contexts as well as in many analyses of social phenomena, certain kinds of omission are regarded as causes. Negligence in civil law, for instance, requires the defendant to have caused the harm that happened to the plaintiff and typically consists in a failure to act. There is, typically, no continuous process (under any reasonable understanding of the term) between an omission and its effect. In such legal inquiries, the same counterfactual concept of cause is at work as in historical contexts and analyses of world politics. Some U.S. Democrats, for instance, accuse the Bush

1. There is a danger to understand the qualifier "differs in no causally relevant detail" as excusing any apparent violation of the claim and therefore rendering it empty—"*X* causes *Y* unless it doesn't." But causal claims have intended applications and purposes, and therefore scientists normally know what counts as a legitimate application and as causally relevant detail (Lange 2000). Importantly, if in the new population the correlation does not hold, there must be a good reason to believe that that factor is itself a cause of the putative effect (Cartwright 2002).

administration of having ignored early terrorism warnings and thereby causing the 9/11 attacks. Even if they were right, it would be foolish to try to find the mechanism that led from the ignoring to the attacks.

2. Does knowing that X and Y are connected by a causal process allow the inference that X and Y are correlated? Although Steel (2004, 71-72) recommends "process tracing" as an aid to ameliorate the "problem of confounders" (the problem of distinguishing alternative causal hypotheses by statistical means), he expresses some doubts about the practical usefulness of the method:

> It is also important to recognize how modest the accomplishments claimed by process tracing actually are. Without the aid of statistical data, the best one can hope to establish by means of process tracing is purely qualitative causal claims. For instance, in Malinowski's example, all we can conclude is that there is at least one path through which the number of wives exerts a positive influence on wealth among Trobriand chiefs. Not only does this conclusion fail to specify anything about the strength of the influence generated by this mechanism, it does not even entail that the overall effect of the number of wives on wealth is positive. One would naturally presume that having more wives would mean having more members of the household to provide for, which would be expected to exert a downward influence on wealth. *Clearly, statistical data concerning the average cost-benefit ratio in yams of acquiring additional wives would be needed to decide which of these two conflicting influences was predominant* [italics added], and no such data are provided by Malinowski.

The overall influence can thus be positive or negative—but also nil.

3. Does knowing that X is a probabilistic cause of Y allow the inference that we can manipulate X to control Y? The denial of this question is precisely the essence of the Lucas critique. A way to paraphrase Lucas is to say that the prevailing large-scale econometric models (of the 1960s) at best provide evidence for historical causal relations that are subject to change when the system is tampered with. Since the aggregate relations depend for their existence partly on the economic agents' expectations, and policy interventions may change the expectations, the aggregate relations may be disrupted by policy. This is, of course, just what happened historically.

We therefore have at least four concepts of cause at work here: "difference making," "connecting by means of a continuous process," "probability raising," and "remaining invariant under intervention." This is not to say that

there are no cases in which the different concepts coincide. Even if economists disagree about their understanding of the mechanism underlying the liquidity effect, say, there will be *some* mechanism that transports the causal message from increases in the money supply to the reduction in nominal interest rates. In such cases, a broader range of inferences is licensed by applying the concept.

Perhaps the attitude advocated here is too cautious. Is it not the case that the different meanings of cause *typically* coincide, that they come apart only in special situations, often constructed by philosophers? Williamson seems to hold this view. In his defense of conceptual monism, he distinguishes between an "inferential" and an "explanatory" use of "the" causal relation and argues,

> There is also the rather general use of beliefs to systematise one's evidence: an agent's beliefs should typically be able to offer some kind of explanation of her experience and evidence. For example, if the agent discovers that two events are probabilistically dependent, and she knows of no non-causal explanation of this dependence (the events are not known to be overlapping, for instance) then she should (tentatively) believe that some causal connection between the events gives rise to the dependence, because dependencies between physical events are typically explained causally. This sketch involves a lot of "typically"s, because none of these features of causality hold invariably; if they did, a more straightforward analysis of causality in terms of one or more of these features might be possible; yet "typically" is quite enough for causal beliefs to be useful from an inferential and explanatory point of view. (Williamson 2006a, 75)

He thus seems to be saying that although there are cases where there is probabilistic raising but no mechanism and vice versa, typically the two go together, and therefore we are licensed to expect one if we have evidence for the other.

I would put the matter differently. At the level of semantics, there are various concepts of cause such as probability raising, mechanism and so forth. It may well be that different concepts apply to a given situation, but if they do so, this is a matter of empirical truth, not a matter of conceptual truth. On the basis of experience, we discover that in a certain domain all or most probabilistic dependencies can be explained by reference to an underlying causal mechanism (say). Discovering this empirical fact is much like discovering that various symptoms of a disease typically co-occur (such as nasal stuffiness, sore throat, hoarseness, and cough typically accompany the common cold). Making such discoveries is enormously useful. But we cannot stop short of empirical investigation to make them.

Importantly, having evidence in favor of a causal claim of one type does not, pace Williamson, entitle the bearer of the evidence to the belief in another type of causal claim, even tentatively. Using terminology I developed in a different context (see Reiss 2008, chap. 1), I would say that establishing a causal claim of one type at best provides prima facie evidence in favor of the related claim that uses a different causal concept. Prima facie evidence is only then valid evidence if all alternative explanations of its existence can be ruled out. For instance, a correlation between *X* and *Y* is prima facie evidence in favor of the claim that *X* causes *Y*. But it is valid evidence only insofar as noncausal explanations of the correlation as well as alternative causal accounts can be ruled out.

The alternative account in the case at hand is simply that we face a case of causation where the different concepts do not coapply. And this can only be ruled out by testing the alternative causal claim in its own right, using evidence tailored to that alternative claim. The upshot is, prima facie evidence gives merely a license to *investigate*; for a license to *believe*, valid evidence is required.

VI

The value of investigating the truth of causal hypotheses lies in the degree to which these claims help in realizing scientists' purposes and in the value of realizing these purposes. About the latter, I have nothing to say in this article. But I do want to make some remarks about how causal claims help to attain social scientists' cognitive and practical purposes.[2]

Social scientists pursue a variety of different purposes such as predicting events of interest, explaining individual events or general phenomena, and controlling outcomes for policy. It is interesting to note that the language of "cause" is employed in all these contexts. Consider the following examples from econometrics, statistics, history, and sociology.

In econometrics, the notion of Granger causality, which is closely related to probabilistic accounts of causation, cashes out whether a time series helps to predict another. In a standard textbook, the following is said about it:

2. A fascinating story could be told about why, at certain times and places, certain purposes seem to dominate at the expense of others and when, why, and how these preferences are revised. The current passion in social science to investigate explanatory mechanisms, for example, is probably in large part due to the field's frustration with earlier strong positivist tendencies. Unfortunately, there is no space here to pursue these matters. For the sake of this article, I just take some salient purposes social scientists pursue as given and examine their connections with causation. For a rudimentary defense of pluralism about the purposes of social science, see Reiss (2007a).

> Granger causality (a kind of statistical feedback) is absent when $f(\mathbf{x}_t \mid \mathbf{x}_{t-1}, \mathbf{y}_{t-1})$ equals $f(\mathbf{x}_t \mid \mathbf{x}_{t-1})$. The definition states that in the conditional distribution, lagged values of $\mathbf{y}_t$ add no information to explanation of movements of $\mathbf{x}_t$ beyond that provided by lagged values of $\mathbf{x}_t$ itself. *This concept is useful in the construction of forecasting models* [italics added] (Greene 2000, 657)

That this notion relates to prediction rather than one of the many other senses of causation is made plain by the following example, also taken from an econometrics textbook:

> The study uses annual data on two variables: total U.S. production of eggs (EGGS) from 1930 to 1983 and total U.S. production of chickens (CHICKENS) for the same period. The test is simple. EGGS is regressed on lagged EGGS and lagged CHICKENS; if the coefficients on lagged CHICKENS are significant as a group, then chickens cause eggs. A symmetric regression is then used to test whether eggs cause chickens. To conclude that one of the two "came first," it is necessary to find unidirectional causality, i.e., to reject the noncausality of one to the other and at the same time fail to reject the noncausality of the other to the one.
>
> Thurman and Fisher's test results were dramatic. Using lags ranging from 1 to 4 years, they obtained a clear rejection of the hypothesis that eggs do not cause chickens, but were unable to reject the hypothesis that chickens do not cause eggs. Thus they were able to conclude that the egg came first! (Pindyck and Rubinfeld 1991, 218-19)

Of course, this story is told partially tongue-in-cheek. But it does illustrate a serious point: econometricians use the notion of cause often to mark out predictive relations, quite independently of whether or not other kinds of causal assertions (for example, about connecting mechanisms) are supported as well. In this case, we would expect mechanisms to run both ways but only eggs help to predict chickens.

Econometricians and statisticians also use another notion of cause. This notion picks out those relations that are stable under intervention or "autonomous" in econometricians' jargon. The statistician David Freedman (1997, 62) distinguishes three uses of regressions:

- to summarize data,
- to predict values of the dependent variable, and
- to predict the results of interventions.

He then reserves the notion of cause to the third:

> Causal inference is different, because a change in the system is contemplated; for example, there will be an intervention. Descriptive statistics tell you about the correlations that happen to hold in the data; causal models claim to tell you what will happen to *Y* if you change *X*.

Patterns in the data are deemed causal because they are useful for the prediction of the results of policy interventions.

A further important purpose across the social sciences is explanation. Explanation, to be sure, is itself not a monolithic concept, and different scientists pursue different explanatory ideals. Two major approaches characterize the historical sciences: the idiographic and the nomothetic. Historians leaning toward idiographic analysis focus on the explanation of singular events and regard those conditions as causes (often significant decisions of rulers), without which the event of interest would not have happened. Such a decision explains the event of interest in just this sense: the event would not have happened but for the decision. As mentioned above, this "but-for" conception is also at work in the law.

By contrast, nomothetically leaning historians focus on generalizations and think of explanation as subsumption under covering law. These historians consequently hold a regularity view of causation (for the two conceptions of *cause* in history, see Goertz and Levy 2007).

In other social sciences, most notably economics and sociology, an event or pattern of events is sometimes regarded as explained only if the mechanism that generates the event or pattern is understood (for economics, see for instance Elster 2007, chap. 2; for sociology, Hedström and Swedberg 1999). Here we therefore find a mechanistic conception of cause.

What kind of causal hypothesis should be investigated (and, in tandem, what kind of evidence should be sought) therefore is to be determined on the basis of purpose pursued in the given context. For certain kinds of prediction, Granger causation is appropriate and thus probabilistic evidence. Explanation is itself a multifaceted concept, and different notions of explanation require counterfactual, regularity, or mechanistic concepts of cause and the associated kind of evidence. Some kinds of policy require a concept of cause as invariant under intervention and, again, evidence able to support this kind of relation.

VII

If the analysis provided in this article is correct, the news is not altogether that good. Although there are different kinds of evidence for causal

relationships, different kinds of evidence tend to support different types of causal claim, a fact that ties evidence and type of causal claim together very tightly. This is unfortunate as we pursue many different purposes and it would be nice if we could establish that *X* causes *Y* and thereby be helped in realizing all our purposes. For instance, it would be nice if we could base policies on probabilistic evidence or if we found a mechanism between *X* and *Y* infer that *X* makes a difference to *Y*. As a general rule, this will not work. To be sure, the different kinds of causal claim are sometimes true of the same system, but whether that is so is an empirical question that has to be addressed, and answered supported by evidence, in its own right.[3]

Perhaps there does remain an open issue. Why do we call all these different relationships causal, and if they are really different, can one not at least describe systematic connections between them? Perhaps this does stand in need of explanation, but I cannot see systematic connections between them save being useful in the light of certain types of purposes. And why we have come to call the different kinds of relationships causal is a matter of historical, not philosophical, inquiry.

What about Williamson's observation that neither scientists nor ordinary folk usually distinguish between the different senses of "cause" by qualifying "*X* probabilistically causes *Y*," "*Z* mechanistically causes *W*," and so on? I do agree with the observation. Unlike Williamson, however, I would not take it as evidence for conceptual monism. Rather, I think that the equivocation has often proved to be a hindrance to successful social science and policy. It is pretty much as Francis Bacon said more than 400 years ago:

> Although we think we govern our words, . . . certain it is that words, as a Tartar's bow, do shoot back upon the understanding of the wisest, and mightily entangle and pervert the judgment. So that it is almost necessary, in all controversies and disputations, to imitate the wisdom of the mathematicians, in setting down in the very beginning the definitions of our words and terms, that others may know how we accept and understand them, and whether they concur with us or no. For it cometh to pass, for want of this, that we are sure to end there where we ought to have begun—in questions and differences about words. (Bacon 1605/2001, 126)

3. Nancy Cartwright makes a related point by lamenting the fact that we do not have a "theory of causality," by which she means a systematization of the connections between the different concepts of cause (see Cartwright 2007, chap. 4).

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