Causal Mechanisms: Yes, But…

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Abstract

In recent years, the importance of mechanism-centered explanation has become an article of faith within the social sciences, uniting researchers from a wide variety of methodological traditions—quantitative and qualitative, experimental and nonexperimental, nomothetic and idiographic, formal models and narrative prose. Despite its many virtues, there are reasons to be skeptical of social science's newfound infatuation with causal mechanisms. First, the concept of a mechanism-centered ("mechanismic") explanation is fundamentally ambiguous, meaning different things to different people. Second, the minimal objectives associated with the turn to mechanisms—to specify causal mechanisms and engage in detailed causal reasoning—are not at variance with traditional practices in the social sciences and thus hardly qualify as a distinct approach to causal assessment. Finally, the more demanding goal of rigorously testing causal mechanisms in causal arguments is admirable but often unrealistic. To clarify, this is not a polemic *against* mechanisms. It is a polemic against a dogmatic interpretation of the mechanismic mission. Causal mechanisms are rightly regarded as an important, but secondary, element of causal assessment—by no means a necessary condition.

Keywords

causality, mechanisms, methodology, microfoundations

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In recent years, the importance of mechanism-centered explanation and analysis has become an article of faith within the social sciences.[1] The turn toward mechanisms unites researchers practicing a wide array of methodologies—quantitative and qualitative, experimental and nonexperimental, cross-case and case study, formal models and narrative prose. Indeed, perhaps the *only* thing that practitioners of these diverse approaches share is an appreciation for the value of causal mechanisms and a corresponding suspicion of "covariational" (aka "correlational" or "associational") arguments. Leverage on causal questions, it is argued, can be gained only by scaling down. We need to get inside the box of causation, toward microfoundations.[2]

The turn to mechanisms offers a helpful corrective to a naïve—"positivistic"—view of causality, according to which causality is understood simply as a constant conjunction (Hume) or a probabilistic association between X and Y. Researchers schooled in the maxim that "correlation is not causation" now think carefully about the generative component of an argument—the pathway(s) through which X might affect Y. Careful attention to mechanisms has helped to expand our theoretical knowledge of the world as well as to guard against spurious causal arguments.

Yet despite its many virtues there are reasons to be skeptical of social science's current infatuation with causal mechanisms. I show, first, that the notion of a mechanism-centered ("mechanismic") explanation is often ambiguous, meaning different things to different people. I argue, second, that the minimal objectives associated with the turn to mechanisms (ones agreed to by all partisans of this school)—to specify causal mechanisms, as much as is possible—are not at variance with traditional practices in the social sciences and thus do not qualify as a truly distinctive approach to causal assessment. I argue, finally, that the more demanding goal of rigorously testing causal mechanisms in causal arguments is admirable but often unrealistic.

To clarify, this is not a polemic *against* mechanisms. It is a polemic against a dogmatic interpretation of the mechanismic mission. I argue that the analysis of causal mechanisms is best regarded as an important, but secondary, element of causal assessment—not a necessary condition.

The Many Meanings of "Mechanism"

The first difficulty with the current vogue of mechanism-centered causal analysis is that the key concept contains a plethora of meanings. In prior work, I demonstrate that "causal mechanism" may refer to (a) the pathway or process by which an effect is produced, (b) a micro-level (microfoundational) explanation for a causal phenomenon, (c) a difficult-to-observe causal

factor, (d) an easy-to-observe causal factor, (e) a context dependent (tightly bounded or middle-range) explanation, (f) a universal (i.e., highly general) explanation, (g) an explanation that presumes probabilistic, and perhaps highly contingent, causal relations, (h) an explanation built on phenomena that exhibit law-like regularities, (i) a technique of analysis based on qualitative or case study evidence, and/or (j) a theory couched in formal mathematical models (Gerring, 2008; also see Hedstrom, 2005, p. 25; Mahoney, 2003; Mayntz, 2004; Norkus, 2004).

Some of these attributes are reconcilable and might therefore be regarded as attributes of a single coherent concept. Others are patently contradictory. Evidently, a mechanism cannot be difficult to observe as well as easy to observe, context bound as well as universal, contingent as well as law like. It should also be emphasized that this is a relatively short typology of definitional attributes; additional meanings might be unpacked from the burgeoning literature on causal mechanisms, fragmenting the meaning of this key term still further.

Clearly, writers have quite different things in mind when they invoke the mantra of mechanisms. Of course, diversity of usage does not imply that *all* usages of a concept are unhelpful. (After all, we do not cast out concepts such as "justice" or "democracy" simply because of their semantic overload.) My intention in this analytic exercise is not to debunk the concept but rather to point out that there is considerably less consensus on the goal of a mechanism-centered social science than a casual read of the literature might suggest (and less than protagonists of mechanisms seem to suppose). Perhaps this is to be expected, given the methodological breadth of this school of thought. In any case, one cannot help but wonder whether adhesion to this protean term has sometimes served to obscure, rather than to clarify, issues of causality. There are signs that "mechanism" may be following in the footsteps of other methodological concepts—for example, behavioralism, interpretivism, positivism, rational choice, and realism—that have become so overburdened with content and vituperation that they serve more as standard bearers for movements than as tools of understanding.

At this point, I turn from semantic analysis to an analysis of the probable utility of several approaches to the subject. Of course, one cannot possibly cover all interpretations of the concept in this short essay, so it is necessary to pick and choose. Two intertwined methodological arguments are especially widespread among the protagonists of a mechanism-centered social science. The first is a concern that the causal path connecting a causal factor and outcome of interest be carefully specified, and the second is a desire to test that

connection empirically. The implications of these suggested moves will guide our discussion through the rest of the article.

I assume, throughout, that the underlying goal of social science is to generalize about social reality as much as is possible, that is, to understand classes of cases (causal regularities or deterministic "laws") rather than outcomes specific to individual cases. The scope of a causal proposition is of course always a matter of degree; there are no truly universal laws because every causal regularity implies some bounding features. For present purposes, what is important is that the term *mechanism* implies a scope that is greater than a single case. Even if the immediate focus of a study rests on a single case, the *theoretical* interest is on a larger class of cases. This distinction between causal generalizations and a *cause in fact* (for a particular case) will be important for some of the points to follow.

Specifying a Mechanism: How Novel?

One would like to know not only whether X causes Y but also *how* it does so. "A complete explanation," write Kiser and Hechter (1991, p. 5), "must specify a mechanism that describes the process by which one variable influences the other, in other words, how it is that X produces Y." "Explanatory propositions," notes Waldner (2007, p. 146), "are distinguished from non-explanatory propositions by the inclusion of causal mechanisms." And again, "the specification of causal chains is what distinguishes propositions about mechanisms from propositions about correlations" (Mayntz, 2004, p. 241).

The specification of a causal mechanism is closely allied with an appreciation of causal *reasoning* in the assessment of X's relationship to Y. After all, one can hardly state a mechanism without also considering the background conditions of a causal argument, the contrast space against which any causal argument takes place, potential counterarguments, and so forth. To discuss mechanisms is to reason about possible and plausible states of the world as they bear on a particular causal relationship. In reasoning, writers build on their knowledge of a particular context and on general knowledge of the world. They may also play out elaborate reconstructions of the events as they actually occurred or might have occurred to test the relative plausibility of various hypotheses.

These crucial elements of causal explanation are now generally acknowledged, correcting an important omission in the "positivist" account of causation (Braithwaite, Carnap, Hempel, Neurath, Oppenheim, Reichenbach,

Schlick, et al.). The now ubiquitous question—"What is your causal mechanism?"—has prompted a good deal of healthy reflection.

However, one might also wonder how novel these desiderata really are. Is anyone—including "neopositivists" such as Gary King or Donald Rubin—opposed to the specification of causal mechanisms? Where are the *critics* of a mechanism-centered science?[3] The near total absence of opposition to the current movement toward mechanisms suggests that the goals outlined above have been integral to the conduct of science (natural and social) for some time.

Arguably, the specification of a causal mechanism and the employment of causal reasoning are inseparable from the development of a causal theory. Consider some of mechanisms that have garnered attention in recent years, for example, path dependence, transaction costs, networks, cooperation or defection, and collective action constraints. Each of these mechanisms might also be referred to as a theory, theoretical framework, or model, depending on one's predilection. Most were hatched well before the current mechanisms vogue, suggesting again that there is nothing particularly new or different about a mechanism-centered explanation—except that we are now more self-aware of a longstanding practice.

Indeed, the recurring debate over what a "mechanisms approach" might consist of is difficult to distinguish from the discussion about what a good theory or explanation might consist of. For example, Hedstrom and Swedberg (1998b, p. 24) characterize the mechanism approach according to four principles: *action*, *precision*, *abstraction*, and *reduction*. It will be seen that all but the first are also commonly used to describe good theory making (e.g., Gerring, 2005).

Leaving aside questions of definition, let us turn to matters of practice. Not all social science studies are equally attentive to causal pathways. In this vein, Hedstrom (2005, p. 18) takes Blau's (1970) article, "A Formal Theory of Differentiation in Organizations," to task as a flagrant instance of purely covariational logic. Indeed, Blau's argument is reduced to a series of rather baldly stated propositions, for example, "Increasing size generates structural differentiation in organizations along various dimensions at decelerating rates" (Blau, 1970, p. 204). These propositions are intended to consolidate a set of empirical findings (reported elsewhere) and do not include much discussion of what might be going on within these organizations to produce the observed results. There is, in particular, no attempt to make sense of individual-level behavior (a goal that Blau, 1970, p. 216, explicitly eschews). And yet, even here, in this austere "positivist" text, one finds more than an inkling of causal explanation. For example, Blau's (1970, p. 213) second major

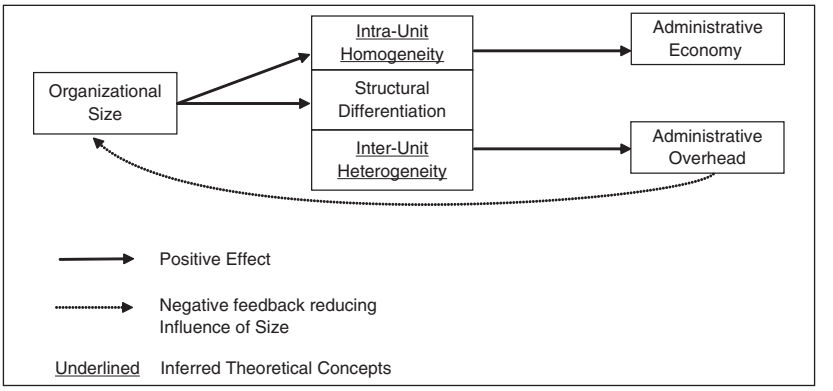

Figure 1. Summary of Blau's argument. Adapted from Blau (1970, p. 218).

proposition reads, "Structural differentiation in organizations enlarges the administrative component, because the intensified problems of coordination and communication in differentiated structures demand administrative attention." Elsewhere (e.g., Blau, 1970, p. 217), he refers repeatedly to the importance of feedback effects in generating the observed patterns of organizational behavior. And the article concludes with a graph of causal pathways (i.e., mechanisms) across the various components of the argument, reproduced here as Figure 1.

It seems unlikely that anyone has ever published an article or book in any field that merely announces a covariational result as causal without *any* discussion of possible causal mechanisms. These are things that social scientists do, more or less self-consciously, when they argue about causes. When Hempel (the arch-positivist in most mechanismic accounts) constructed a heuristic explanation for the French Revolution (an example of the logico-deductive model of explanation), he offered the following theory: "A revolution will tend to occur if there is a growing discontent, on the part of a large part of the population, with certain prevailing conditions" (quoted in Bohman, 1991, p. 19). Surely, this is an appeal to mechanisms.

That said, Hedstrom and others are certainly correct in pointing out that some studies do this more thoroughly and self-consciously than others. Some studies are explicitly about mechanisms; others, like Blau (1970), are more focused on correlations. The relevant question becomes, how much "mechanism talk" is necessary, or desirable? One finds widely varying opinions about how realistic, detailed, and determinate a theory or model must be to

perform its explanatory role. Friedman (1953) articulates a skeptical perspective on the necessity for model realism, a view that many writers have criticized. A mechanism-centered social science would seem to take more seriously the specification of discrete causal pathways than is common, for example, in many economic models. But this by itself does not provide a sharp point of contrast between "mechanismic" and "covariational" social science. Both propound theories, and those theories delve—more or less—into causal mechanisms. It is hard to see how it could be otherwise.

Testing Causal Mechanisms: How Necessary?

Specifying a causal mechanism is sometimes a highly speculative affair. The posited mechanism may be highly specific but at the same time remain entirely unproven, and perhaps highly dubious. This is what prompts some partisans of causal mechanisms to insist that mechanisms not only be specified but also tested empirically wherever their veracity is in doubt. And few would challenge this objective as an ideal. After all, it simply extends the usual injunction to verify or falsify from the principal hypothesis (i.e., the covariational relationship between X and Y) to the causal pathway (Z). However, one might question whether failure to adequately test causal mechanisms should disqualify a study. Is the empirical testing of causal mechanisms a *necessary condition* of causal assessment (as some writers aver)?

There are, to begin with, many instances in the history of science in which the covariational aspect of a causal relationship has been proven prior to the discovery of the causal mechanism at work. Doctors were aware of the curative properties of penicillin and aspirin long before they could figure out why these treatments had their beneficent effects. More generally, one might observe that in a properly conducted experiment (i.e., with a randomized treatment and isolated treatment and control groups) it is often possible to demonstrate that some factor causes a particular outcome even though the pathway remains mysterious (perhaps because it is not amenable to experimental manipulation). We do not hesitate to label these arguments as causal and as definitive (assuming proper experimental protocols have been followed and replications have been conducted in a variety of settings). Thus, common practice in the sciences does not confirm the necessity of testing causal mechanisms—though it certainly confirms their importance.

Moreover, from a policy perspective it is usually more important to know what effect a given treatment has on an outcome than why it has that effect. Granted, the latter are not incidental to the former and can often help to identify

potential externalities. We may know that Policy X causes Policy Outcome Y, but if we do not know why, it may be difficult to anticipate possible unintended effects of Policy X. Even so, the covariational relationship is usually the key component of concern for policy makers. This is true in medicine as it is in the social sciences. We want to know, for example, what effects a voucher program might have on educational achievement and are usually less concerned with ascertaining precisely why it might have these effects.

Testing Causal Mechanisms: How Tractable?

Let us accept, then, that specifying and testing causal mechanisms is a universal goal, but not a requirement, of causal analysis. In some experimental settings it may be possible to prove the existence of a causal relationship between X and Y without having tested the causal pathway(s) lying between them (Z). Indeed, it may even be possible to prove a causal relationship without having a clear supposition about the causal mechanisms at work.

This brings us to the question of how methodologically tractable the routine testing of causal mechanisms might be. We all agree that it is desirable, but to what extent is it possible?[4]

A first methodological difficulty is the problem of infinite causal regress: Within every covariation is a mechanism, and within every mechanism is a mechanism. *At some point*, a study must arrest the empirical process and rely on what we know, or think we know, about the world to reach causal inference. I assume that what proponents of testing causal mechanisms intend is that we should not arrest this process until we are feeling pretty confident about the background assumptions undergirding a causal argument. This means that in the analysis of very proximate covariational relationships—where little causal distance separates X and Y—there may be no point in testing causal mechanisms at all because the pathway is already more or less self-evident. The very notion of a causal mechanism presumes that a distinction can be drawn between the covariational and mechanismic properties of a causal argument; if the covariation is mechanismic (i.e., if the mechanism at work is obvious), then the problem of specifying a mechanism does not arise. It is already accomplished.

Our principal concern, therefore, must be with structural (distal) causes, where X is situated a long way from Y (with multiple intervening factors lying in between). Here, the need for empirical testing of causal mechanisms is readily apparent. However, in precisely the situations where causal mechanisms are most opaque the empirical testing of these mechanisms usually proves most challenging.

As an example, one might consider the relationship between economic development and democracy, a subject that has received a good deal of attention from political scientists over the past decade (Boix & Stokes, 2003; Coppedge, in press; Epstein, Bates, Goldstone, Kristensen, & O'Halloran, 2006; Przeworski, Alvarez, Cheibub, & Limongi, 2000; Rueschemeyer, Stephens, & Stephens, 1992). We agree, more or less, on what the key concepts—economic development, democracy—mean and the various ways they might be operationalized, despite ongoing controversies over conceptualization and measurement (Munck & Verkuilen, 2002). There is a there, there. The same cannot be said for the potential causal mechanisms in this story, which are many and various. Consequently, although there have been many covariational studies (see the above-cited sources), one finds fewer attempts to empirically test the causal mechanisms that might be at work in this relationship. More important, in the latter genre one finds few or no conclusive findings.

This is a fairly typical situation in the social sciences. Recent work on the "resource curse" is virtually united in identifying oil and other mineral resources as a (probabilistic) cause of civil war, bad governance, autocracy, and stagnant long-term growth. Yet, the causal mechanisms at work in this causal story remain opaque. There is a great deal of theoretical speculation and some empirical testing—clearly, authors are taking the question of mechanisms seriously—but few strong conclusions have emerged (Humphreys, 2005; Mehlum, Moene, & Torvik, 2002; Papyrakis & Gerlagh, 2003; Ross, 2001).

Another example is provided by work on the "democratic peace." Here, a strong correlation is found between country dyads that are democratic and peaceful interstate relations. Most researchers regard this as evidence of causality, though there is ongoing debate over whether to regard the democratic peace as probabilistic or deterministic, and a few scholars remain unconvinced that the relationship is causal at all (e.g., Rosato, 2003). In any case, the covariational evidence is strong, and there are many reasons to suspect that war is less likely between two democracies than among dyads in which at least one member is a nondemocracy. Although scholars have gone to extraordinary lengths to test plausible causal mechanisms (Brown, Lynn-Jones, & Miller, 1996; Elman, 1997; Hamberg, 2006; Hasenclever & Weiffen, 2006), the results are again inconclusive (Rosato, 2003). We do not know precisely why democracy discourages violent conflict among nations, even though most scholars are convinced that it does (Debs & Goemans, 2008).

As a final example, one might consider the avalanche of recent work on geography as a long-run factor in political and economic development (e.g.,

Acemoglu, Johnson, & Robinson, 2002; Diamond, 1997; Gallup, Sachs, & Mellinger, 1999; Landes, 1999; Sokoloff & Engerman, 2000). Here, one finds a remarkable degree of scholarly consensus on the causal significance of a variable such as "distance from the equator," despite the evident lack of process-tracing evidence and the bewildering number of ways in which this factor might plausibly influence long-run performance.

Granted, *all* causal hypotheses are accompanied by some degree of uncertainty. Decisive proof or disproof is probably not a realistic goal, even in the natural sciences (and certainly not in the social sciences). The point is that one often finds an imbalance in the literature between covariational arguments that are judged to be reliable and mechanismic arguments that are judged to be highly speculative. Much more greater scholarly consensus can be found with respect to the causal effect of (a) development on democracy, (b) mineral wealth on the quality of governance, (c) democracy on peace, and (d) distance from the equator on economic development, than can be found with respect to the causal mechanisms at work in any of these relationships. We know (or think we know) that X causes Y, but we do not know why, or at least not precisely why.

Testing Causal Mechanisms: What Are the Challenges?

What is it, specifically, that is empirically intractable about factors that come to be defined as causal mechanisms? In considering this question we begin by reconsidering the examples sketched in the previous section—the causal effect of (a) development on democracy, (b) mineral wealth on the quality of governance, (c) democracy on peace, and (d) distance from the equator on economic development. This time, we look explicitly at the causal mechanisms that might be at work in these (presumably causal) relationships.

The effect of economic development on democracy has been credited to (a) urbanization, (b) greater capital mobility, (c) a higher standard of living and lower levels of extreme poverty, (d) greater equality, (e) a stronger working class, (f) a stronger middle class, (g) education (specifically widespread literacy), (h) cultural interconnections with the world, (i) a liberal trade regime, (j) a more extensive and independent media, (k) a more developed civil society (including higher levels of participation and social trust), (l) cross-cutting social cleavages, (m) nonclientelistic elite–mass relationships, and (n) greater identification with national (as opposed to local, tribal) values (see, inter alia, Coppedge, in press, Table 4.3).

The effect of mineral wealth on the enhanced probability of autocratic rule could be explained by three general factors, explains Michael Ross (2001):

> (a) a "rentier effect," which suggests that resource-rich governments use low tax rates and patronage to relieve pressures for greater accountability; (b) a "repression effect," which argues that resource wealth retards democratization by enabling governments to boost their funding for internal security; and (c) a "modernization effect," which holds that growth based on the export of oil and minerals fails to bring about the social and cultural changes that tend to produce democratic government. (pp. 327-328)

The effect of regime type on peace between democracies might be a product of (a) greater transparency (reducing the possibility of surprise attack and the miscalculations of strength that sometimes lead to war), (b) greater normative and cultural affinities (viewed here as a by-product of a nation's experience as a democracy), (c) greater trust and respect (e.g., agreements between nations benefit from credible commitment because they are endorsed by their respective peoples and would be difficult to renege on), (d) liberal trade policies (leading to economic interdependence), (e) democratic norms (implicitly, if not explicitly, opposed to war), (f) the framework of democracy (which makes it more time-consuming to mobilize the country for war, leaving multiple opportunities for diplomats to find a way to step back from the brink), (g) the disinterest of publics everywhere in foreign adventures (which makes it more difficult for leaders in a democratic polity to commit the nation to war), and (h) stronger international institutions (reviews of these arguments can be found in Brown et al., 1996; Elman, 1997; Hamberg, 2006; Hasenclever & Weiffen, 2006; Rosato, 2003).

The effect of distance from the equator on economic development may follow from (a) colonial patterns of settlement, investment, and property rights (intensive and developmental away from the equator, extensive and extractive near the equator), (b) enduring patterns of inequality (high near the equator where geographic conditions were favorable to plantation agriculture, low away from the equator where family farms predominated), (c) the direct effects of an equatorial climate on disease, on human productivity (via heat and disease), and on soil quality, (d) the greater east–west trajectory of Asia (lying predominantly above the equator), relative to other continents and the benefits of technological diffusion that this brings, or (e) the fact that the industrial revolution occurred first in Europe for a variety of reasons that have nothing to do with equatorial climate, soil, and disease conditions and

Europe is a long way from the equator (Acemoglu et al., 2002; Diamond, 1997; Gallup et al., 1999; Landes, 1999; Sokoloff & Engerman, 2000).

Thus, for each of our four examples one finds a litany of theoretically plausible causal mechanisms, most of which have been extensively debated in the literature. Of course, I cannot pretend to have arrived at a comprehensive list of mechanisms for any of these covariational relationships. It should also be noted that many of these mechanisms might be further disaggregated, or differently aggregated. Categories (which are largely dependent on our vocabulary) are often squishy, introducing an element of indeterminacy. If two authors construct different lists of possible causal mechanisms for a given causal effect, their testing procedures and conclusions are likely to vary. The difficulty of testing empirical mechanisms begins with the difficulty of articulating all the possible (theoretically plausible) causal mechanisms for a given X–Y relationship.

The problem of omitted factors deserves special notice. If no list is ever demonstrably complete (there are always novel and heretofore unimagined ways in which X may influence a distal Y), then no testing procedure is definitive. Consider that in testing the effect of X on Y one can arrive at a reasonably definitive answer if the causal factor of interest is amenable to experimental analysis. However, in testing the mechanism(s) that lie between X and Y one has a much more open-ended question to wrestle with. One's task, in effect, is to discover and test all the pathways that lie between an intervention an outcome. Having canvassed the literature, and ransacked one's brain, one constructs a list of five plausible causal mechanisms, each of which (miraculously) is amenable to randomized research designs (X with Z and Z with Y). This may allow one to eliminate one or several hypotheses and may provide plausible confirmation for other hypotheses. However, it will never answer the question of causal mechanisms definitively and completely because of the potential—which can never be entirely overcome—of additional causal mechanisms that one has not contemplated. Indeed, the situation is worse still, for these omitted factors may serve as confounders for the causal factors that one has thought to confirm. In this respect, the contrast between covariational and mechanismic research designs is akin to the contrast between "effects of causes" (the Neyman–Rubin–Holland approach) and "causes of effects" (the search for all causes of a particular outcome; Mahoney & Goertz, 2006). The latter objective is, in some basic philosophic sense, indeterminate.

A third challenge is that mechanisms under review in social science frequently consist of vague and abstract concepts that are rather difficult to operationalize. They either resist measurement or are liable to many plausible

indicators. Sometimes, they consist of continuous causal processes that are, by virtue of this fact, difficult to differentiate from either X or Y. Problems of operationalization plague many of the causal mechanisms listed in the foregoing discussion.

A fourth challenge is that plausible mechanisms connecting X and a distal Y are usually multiple and may be difficult to tease apart from one another. If they cannot be partitioned, they cannot be separately tested, and the question of causal mechanisms must go begging. And even if, by some stroke of luck, each causal pathway can be separated from the rest, and securely operationalized, one is likely to face extreme problems of multicollinearity.

A fifth challenge is the variety of possible interrelationships that may be found among various causal mechanisms that help to explain X's relationship to Y. Z1, Z2, Z3, and Z4 may be independent and additive (each contributing some portion of causal force that together explain variance on Y), they may be substitutes (each providing a sufficient cause for Y), or they may work interaction with one another. Each supposition demands a different research design, and this means of course that the resulting research design cannot be looked on as providing an entirely falsifiable test.

A final challenge is that the same causal mechanism may have opposite effects on an outcome depending on the trigger or the context of an event (Elster, 1998). It has been argued, for example, that resource wealth sometimes diminishes, and sometimes enhances, the probability of a democratic regime outcome (Dunning, 2008). In this sort of situation, it is still possible to calculate a population-level relationship between X and Y (ignoring the heterogeneity of the sample). But it is not possible to say anything definitive about the causal mechanisms at work, unless one has correctly identified the circumstances in which X is positively, and negatively, associated with Y.

Granted, these empirical challenges are not unique to mechanism-based tests; they also impinge on covariational studies. However, they seem to be considerably more common among studies purporting to shed light on causal mechanisms.

Given these obstacles, it is not surprising that scholars' efforts to subject plausible causal mechanisms to empirical testing are rarely definitive. In the prototypical research situation, a number of potential causal paths—Z1, Z2, Z3, and Z4—seem plausibly to lead from X to Y, only one of which (Z1) is easily measured. The dogged scholar tests Z1's relationship to X, and to Y, and—citing strong presumptive-theoretical and empirical-covariational evidence—concludes that Z1 is a, or the, mechanism that explains X's relationship to Y.

Without engaging the particulars of the evidence, several difficulties with this conclusion are clear (and indeed have already been foreshadowed). First, it is still quite possible that other plausible causal pathways (Z2, Z3, and Z4) help to explain the causal relationship of theoretical interest. Second, there may be other pathways (Z5, Z6, and Z7) that neither the scholar nor the literature has managed to identify. Third, conclusions focused on Z1 (leaving aside Z2-7) are subject to a very serious specification problem given that measurement obstacles preclude the inclusion of Z2, Z3, and Z4 (not to mention Z5, Z6, and Z7) in the scholar's causal model. Indeed, Z1 may serve only as a proxy for one or more unmeasured factors if those unmeasured factors turn out to be correlated (singly or cumulatively) with Z1. Under the circumstances, it seems highly possible that Z1 carries no weight whatsoever in the causal model—or, at the very least, that its coefficient (the strength of its relationship to X and Y) is overstated. (Note that these identification problems persist regardless of whether the empirical analysis is focused on a single case, several cases, and/or a large sample of cases and whether the analysis is qualitative or quantitative.)

None of this should be interpreted as discouraging scholars from undertaking empirical testing of causal mechanisms. My point is simply that the barriers to successful analysis are high and ought to be forthrightly acknowledged. Arguably, disciplinary standards for mechanismic testing ought to be somewhat lower than those routinely applied to covariational testing.

Testing Causal Mechanisms in Confirmatory Settings

In the previous discussion I have assumed that relatively little is known about the causal mechanisms that might connect X with Y. Theories are indeterminate (or determinate theories are not credible). This is the inductive style of research that is typical of most social science today (though it often affects to be theory driven). Sometimes, however, research programs are governed closely by the dictates of theory. In this context, it is easier to imagine the exploration of causal mechanisms playing an important empirical role. This is the idea behind "pattern matching," as envisioned by Donald Campbell (1966; also see George & Bennett, 2005; Trochim, 1989). The general question addressed by pattern matching is the following: If Theory A is true (false), what about the world ought to be true (false)?

As a concrete example, let us consider a question that has absorbed economists and public health experts for several decades. We know that there is a strong association between socioeconomic status and health status: The rich

are healthier. But we do not know in which direction the causal arrows should be drawn. Does low status make one sicker (Marmot, 2004), or does sickness cause one to fall to a lower status (Cutler, Deaton, & Lleras-Muney, 2006)? (Usually, status is measured by income or occupation; however, the concept at issue is *relative* status, not absolute income—which everybody agrees has a strong impact on health.) This example provides a typical instance in which extant observational evidence is inconclusive (even though there is a lot of it) and no experimental protocol seems to be forthcoming (for obvious practical and ethical reasons).

Angus Deaton is the foremost exponent of the second view: Health affects poverty. His reasoning is summarized in a recent account by Nancy Cartwright (2007), from whom I quote:

> the income-mortality correlation is due primarily to loss of income from poor health, then it should weaken dramatically in the retired population where health will not affect income. It should also be weaker among women than men, because the former have a weaker attachment to the labour force over the period of employment. In both cases it looks as if these predictions are borne out by the data. Even more importantly, when the data are split between diseases that something can be done about and those that nothing can be done about, then income is correlated with mortality from both—just as it would be if causality ran from health to income. Also, education is weaker or uncorrelated for the diseases that nothing can be done about. It is, [Deaton] argues, hard to see how this would follow if income and education were both markers for a single concept of socio-economic status that was causal for health. (p. 27)

Here we see how patterns drawn from the periphery of the analysis can be mustered to shed light on a causal inference. When, for whatever reason, a direct test of X's effect on Y is impossible, the researcher may cast about for other things that ought to be true *if* there is a causal relationship between X and Y. This style of evidence is often the best that is available. As such, it ought to be granted a more dignified title than is suggested by our use of the terms *ad hoc* and *circumstantial*. One can applaud the role of mechanismic thinking insofar as it legitimates this genre of best-possible evidence.

However, the technique of pattern matching (as applied to causal mechanisms) is constrained by the explicitness and determinacy of the theory under investigation. In the absence of a strong set of theoretical expectations it is impossible to ascertain whether the theory is true or not simply by observing

causal mechanisms. As it happens, many social science theories are rather flaccid, that is, open to multiple interpretations, and highly probabilistic. This limits the utility of this otherwise robust method. It is true that one can easily *dis*confirm a theory that makes specific and determinate predictions about the nature of reality; and its confirmation, though more difficult, is still possible. Indeed, if the predictions are deterministic, they can be disconfirmed by a single case (Dion, 1998). But if the theory is instead rather vague and abstract, and the predictions are probabilistic, meaning that multiple predictions are generated by the theory but none are sufficient to falsify it or to provide strong corroboration (Lakatos, 1978), and if the mechanisms are plural and may be combined in many different ways, then the results of a mechanisms-based investigation are unlikely to be conclusive. This dynamic can be seen in the ongoing controversy over the democratic peace hypothesis, where scholars dispute the implications of evidence about causal mechanisms (Doyle, 2005; Kinsella, 2005; Rosato, 2005; Slantchev, Alexandrova, & Gartzke, 2005).

It is worth noting that the technique of pattern matching, as described here, is commonly employed in nonexperimental branches of the *natural* sciences, where theories tend to issue more specific and determinate predictions (Bechtel & Abrahamsen, 2006; Cartwright, 2007, p. 29; Darden, 2002). As one example, one might consider the effort by a team of geologists to demonstrate the meteorite theory of dinosaur extinction. King, Keohane, and Verba (1994, summarizing Alvarez & Assaro, 1990) explain,

> One hypothesis to account for dinosaur extinction, developed by Luis Alvarez and collaborators at Berkeley in the late, 1970s, posits a cosmic collision: a meteorite crashed into the earth at about 72,000 kilometers an hour, creating a blast greater than that from a full-scale nuclear war. If this hypothesis is correct, it would have the observable implication that iridium (an element common in meteorites but rare on earth) should be found in the particular layer of the earth's crust that corresponds to sediment laid down sixty-five million years ago; indeed, the discovery of iridium at predicted layers in the earth has been taken as partial confirming evidence for the theory. Although this is an unambiguously unique event, there are many other observable implications. For one example, it should be possible to find the meteorite's crater somewhere on Earth. (pp. 11-12)

Although the evidence offered here is by no means conclusive (pattern matching rarely is), it is sufficient to update prior probabilities. But it is not a

social science example, nor is it typical of the sort of theoretical problems that we are generally focused on.

Causal Mechanisms as a Substitute for Covariational Analysis

Typically, the investigation of causal mechanisms serves an adjunct role in the analysis of some covariational relationship. It is there to help confirm or disconfirm—or to further clarify—an observed relationship between X and Y. However, it is also argued that the investigation of causal mechanisms (aka process tracing, colligation, etc.) can sometimes effectively *replace* the (covariational) analysis of X and Y (Bennett, 2003; Mahoney, 2000; Morgan & Winship, 2007, pp. 182-184, 224-230; Pearl, 2000; Roberts, 1996).

The perceived need for an alternative to standard X:Y approaches to causal assessment grows out of an increasing unease with the foibles of observational research. (I assume that despite the encouraging growth of experimental work across the social sciences a majority of studies will continue to employ nonrandomized treatments.) We are, by now, achingly familiar with the assignment (i.e., selection) problem, for which ex post statistical corrections are rarely reliable (Benson & Hartz, 2000; Berk, 2004; Freedman, 1991, 1997; Friedlander & Robins, 1995; Glazerman, Levy, & Myers, 2003; LaLonde, 1986). Observational research that enlists nation-states as units of empirical analysis is usually subject to additional problems of sample bias, small sample size, sample heterogeneity, and limited temporal variation in key parameters. Typical problems in observational research are thereby exacerbated (Kittel, 2006; Rodrik, 2005; Summers, 1991).

In this context, the search for alternative approaches to causal assessment is understandable, and commendable. Consider the following special conditions: (a) all causal mechanisms (Z) connecting X to Y are measurable, (b) Z serves as the exclusive and exhaustive pathway between X and Y, (c) if Z consists of multiple pathways these are isolated from one another (they do not have any reciprocal causal impact) and independently measurable, and (d) Z is caused by X and not by any other factor (except some factor that lies antecedent to X).[5]

To visualize this scenario, let us explore an example drawn from the field of medicine, one that has already elicited a good deal of commentary (Pearl, 2000, pp. 83-84). Suppose we wish to test the hypothesis that smoking causes cancer—until recently, a much debated proposition. Although the correlation between these two factors is high, there are of course many possible confounders. Perhaps people who smoke are also prone to other risky actions that

expose them to a greater risk of cancer, perhaps the same genetic predisposition that leads them into addiction also causes them to contract cancer at a later age, and so forth. As such, the question is unproven, and perhaps unprovable, because we are at present unable to measure these genetic predispositions or the health-averse behavior patterns that might serve to enhance the risk of cancer (independent of smoking). And, for obvious reasons, we are unable to randomize the treatment (smoking). Yet if it is the case that smoking causes a buildup of tar in the lungs and tar is a proximate cause of cancer (insofar as cancer might be caused by tobacco), we may be able to estimate the enhanced risk of cancer that comes with smoking by targeting this causal mechanism. This would be a simple two-stage analysis, allowing us to disregard any confounding effects (from genetics and/or behavior patterns): (a) smoking → tar and (b) tar → cancer. (Of course, we must be willing to discount the possibility that some prior factor, for example, genetics, causes all three phenomena—smoking, tar, *and* cancer.)

The example is compelling. However, we must wonder how common it is to find all four requirements listed above fully satisfied. I have difficulty even constructing a plausible scenario for how this approach might be applied to questions in political science. Under the circumstances, I can only conclude that the occasions that might justify the substitution of mechanismic evidence for covariational evidence are exceedingly rare.

Testing Causal Mechanisms: What Is Distinctive?

The previous sections have demonstrated that we are generally more secure in our knowledge of covariational relationships than in our knowledge of the causal mechanisms at work in these relationships. This, in turn, suggests one of two conclusions. Perhaps, because an explicit methodological focus on mechanisms constitutes a rather new agenda (depending on how one interprets the agenda), greater success in this arena will be forthcoming in the future. We have only to wait for mechanismic knowledge to accrue. Alternatively, there may be something distinctive, methodologically speaking, about mechanismic questions that make them less amenable to empirical proof. Leaving the future aside, let us entertain the latter possibility.

From one perspective, nothing distinguishes the investigation of causal mechanisms from the investigation of covariation except that the former occurs in two stages: X:Z and Z:Y. Each of these investigations is "covariational," to use the lingua franca. This suggests that testing causal mechanisms is at least twice as demanding as testing covariational relationships.

In addition, one must consider the arbitrary nature of "X," "Z," and "Y." Evidently, the role of each factor is assigned by the researcher; they are not inherent features of the phenomenal world. In this light, important differences emerge. Note that scholars choose their principal causal hypotheses carefully, with an eye to their empirical tractability. X and Y are likely to be measurable and testable, at least to some degree. The territory in between X and Y, by contrast, is often a morass. This is a product, again, of the way in which causal hypotheses are identified. Writers must have some confidence that they know what X and Y are, and how they should be operationalized; otherwise, they are unlikely to focus their research on this particular causal question. The question of causal mechanisms is more peripheral, both conceptually and empirically. It is no surprise, therefore, that these secondary relationships are often empirically intractable. Indeed, if they were highly tractable one suspects that they would have become the primary focus of the research—as X or Y, rather than Z.

In this light, one might reconsider the various examples explored above. Suppose that some brilliant (or lucky) scholar discovers a way to differentiate and test the many putative causal pathways that may explain the relationship between economic development and regime type. The list of potential factors includes the bourgeoisie, the middle class, the proletariat, urbanization, basic literacy, higher education, the private sector, and social capital—each of which could be viewed as an outgrowth of economic development, and all of which might affect the likelihood of democratization or the preservation of democracy (once established). It stands to reason that some of these factors will prove more empirically robust than others. Let us imagine that an analysis reveals the number of highly educated intellectuals to be the strongest predictor of regime type in a panel of countries (following Kurzman & Leahey, 2004). Now, in all likelihood, the author of such a study will not publish this finding as a test of causal mechanisms. Rather, she or he will frame it as a finding in its own right. The headline is "intellectuals"—not "development." Note also that a new set of mechanismic questions now arise: Why, precisely, does a larger intellectual class result in a more democratic regime? What are the pathways? As is traditional, this question is addressed by Kurzman and Leahey (2004), but by no means proven.

I conclude that it is a normal feature of academic life for tractable "mechanisms" to be transformed into "covariations," and intractable covariations into causal mechanisms. From this perspective, the problem of testing causal mechanisms has nothing to do with ontological realities and everything to do with the sociology of knowledge. Covariations between X and Y are things that we can demonstrate with a fair degree of assurance. Mechanisms, by

contrast, are things that we speculate might be true but cannot prove in a definitive fashion. This explains why the empirical problem posed by causal mechanisms is unlikely to disappear. It is, at root, a definitional problem.

Conclusions

Some partisans of mechanismic explanations might say that the foregoing discussion illustrates precisely the weaknesses inherent in traditional macro-level social science theorizing. It is this sort of work that we should be *avoiding* rather than trying to repair. If mechanisms are ambiguous and resist empirical definitive testing, then one has no business talking about causality. We should look instead to proximal causal hypotheses where the relationship between X and Y can be understood, that is, where the causal mechanisms are not a jumble of abstract, undifferentiated, and untestable processes but rather are discrete and testable, or can be intuited from what we know about the world. This, broadly stated, is the view embraced by those who would reconfigure social science around the methodology of *microfoundations* (also known as the *micro–macro link* or *reductionism*). This is a plausible conclusion, though not without its own difficulties (reviewed in the extended, online version of this article).

In any case, I want to clarify that this essay has *not* argued against mechanisms as a tool of causal explanation and analysis. It has argued, rather, against what might be called a mechanistic application of mechanisms. As we have seen, the apparently simple aim to achieve "mechanism-centered" explanation has many guises, most of which are worthy of emulation. However, none of these—beyond the simple admonition (implicit in the very act of theorizing) to stipulate a causal mechanism—may be considered a necessary condition of causal explanation. To insist on the rigorous testing of mechanisms as a sine qua non of causal inference would seem to eliminate whole fields of social-scientific inquiry—or, equally bad, would drive social scientists to construct pseudo-tests that scarcely advance our knowledge of the true reality underlying X's relationship to Y. In this light, those who dismiss causal arguments because they are vague and inconclusive with respect to causal mechanisms are practicing a species of methodological dogmatism every bit as dogmatic as the "positivist" methodology they seek to replace.

What we need is intelligent discussion of plausible causal mechanisms, which should be subjected to empirical testing *to the extent that it is feasible*. What we should appreciate is that this objective is rarely fully achievable when one is dealing with distal causal relationships, and it is often unnecessary (in the sense of being trivial) when dealing with proximate causal

relationships. Thus, the "problem" of causal mechanisms is likely to remain problematic, despite our best efforts.

By way of conclusion, it may be helpful to distinguish among four causal issues that, I suspect, often are confused: (a) the definition of *causality* (what is a causal relationship?), (b) the *causal effect* (what is the effect of a given change in X on Y?), (c) *causal explanation* (what does it mean to explain a causal relationship?), and (d) *causal assessment* (how do we know when a causal relationship is present?). These four aspects of causation are all important, but they are not the same things.

The importance of causal mechanisms is probably most apparent at the level of (c). Indeed, I have argued that causal mechanisms are more or less intrinsic to explanation. So it is no surprise that when partisans of causal mechanisms talk about causation they often focus on the task of explanation. Mechanisms may also come into play at the level of definition (a) and assessment (d), though neither of these features could be understood solely through mechanisms. Rarely, it seems, do causal mechanisms help us to measure causal effects (b), for reasons already discussed. In short, causal mechanisms are an integral part of the project of causality. But they are not the entirety, and they never exist in isolation from other considerations. There is no such thing as a mechanism without a causal effect, for instance.

Other approaches to causality emphasize somewhat different aspects of the overall project (Brady, 2002). In this respect, it is possible to identify "schools." However, the schools are not easily distinguished from one another, as this article has shown. In principle, and in practice, there are grounds for a unified vision of causality within the social sciences (Gerring, 2005).

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Notes

1. A longer version of this article, with a more comprehensive set of citations and an additional section focused on the project of microfoundations, is posted on the author's homepage (http://people.bu.edu/jgerring/Methodology.html).
2. Representative works include Bennett (2003), Bunge (1997), Dessler (1991), Elster (1983, 1989, 1998), George and Bennett (2005), Goodin and Tilly (2006), Hechter (1983), Hedstrom (2005), Hedstrom and Swedberg (1998a, 1998b), Little (1998), Mahoney (2001, 2003, 2004), Mayntz (2004), Norkus (2004), Petersen (1999), Pickel (2006), Roberts (1996), Schelling (1978), Steel (2003), Stinchcombe (1991, 2005), Tilly (2001), van den Bergh and Gowdy (2003), and Waldner (2007). Discussions of causal mechanisms in the context of philosophy of science and natural science—though often influential in social science contexts—are of peripheral interest here.
3. A thorough canvas of the literature reveals only one article that is modestly critical of the turn to mechanisms (Norkus, 2004), and this piece does not take issue with the general goal of specifying causal mechanisms. Even King, Keohane, and Verba (1994, p. 85; also see pp. 226-228), the high priests of neopositivist orthodoxy within contemporary political science, admit that "any coherent account of causality needs to specify how the effects are exerted."
4. Discussion of econometric issues involved in testing causal mechanisms—sometimes referred to as mediation analysis—can be found in Imai, Keele, and Yamamoto (2009) and MacKinnon (2008). Here, I am concerned with empirical issues that are not simply econometric in nature (though they are that as well, by extension).
5. This account is based largely on Morgan and Winship (2007, pp. 182-184, 224-230), which, in turn, builds on Pearl (2000).

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