Causal Arrows in Econometric Models

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ABSTRACT

Econometrics applies statistical methods to study economic phenomena. Roughly, by means of equations, econometricians typically account for the response variable in terms of a number of explanatory variables. The question arises under what conditions econometric models can be given a causal interpretation. By drawing the distinction between associational models and causal models, the paper argues that a proper use of background knowledge, three distinct types of assumptions (statistical, extra-statistical, and causal), and the hypothetico-deductive methodology provide sufficient conditions for a causal interpretation of econometric models.

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1. INTRODUCTION

A *vexata quaestio* in philosophy of economics is the extent to which econometric models tell causal stories. The question is certainly not new and occupies much of philosophically-minded discussions of the methodology of economics and, more generally, of the social sciences.

This paper adds to the literature offering its own position. The position I defend stems from the dissatisfaction with a certain class of arguments—which I shall call 'metaphysical arguments'—and from the recognition that another class of arguments—which I shall call 'methodological arguments'—are on the right track but only go half way through.

The paper is organized as follows. In section 2, I explain the particular epistemological viewpoint I adopt in looking at econometric models. The motivation for an epistemological approach to causality comes from the dissatisfaction with 'metaphysical arguments' that assume causal structures generating the observed data instead of explaining how we come to establish whether a given correlation is causal. In section 3, I review traditional 'methodological arguments' given by philosophers of economics and philosophically-minded econometricians. In those arguments two trends can be identified: those who stress the importance of causal mechanisms and those who put the whole burden of the causal interpretation in the assumptions of the model. I then offer in section 4 my own answer. I shall do that in two steps. First, I present an account of the conditions under which econometric models can be given a causal interpretation by defending the following interrelated ideas: (i) there is an important distinction between associational models and causal models; (ii) the difference lies in their respective features, notably whilst associational models just have statistical assumptions, causal models also have extra-statistical and causal assumptions, employ a hypothetico-deductive methodology, and use background knowledge in an essential way at each stage of the model building and model testing process. Second, I discuss the methodology, results, and critiques of a case study on the relations between health and wealth in elderly Americans.

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2. AN EPISTEMOLOGICAL INVESTIGATION INTO ECONOMETRIC MODELS

Let me make clear from the outset what perspective I shall take in looking at econometric models. In the philosophy of causality three broad areas of investigation may be distinguished. The metaphysics of causality is interested in what causality in fact is, in what kind of entities causes are, or in what a causal claim means. The epistemology of causality, instead, investigates how we come to know about causal relations. There is a fleeting borderline between epistemology and methodology, but a line between the two can be drawn nonetheless. Whilst methodology is concerned with problems of scientific methods and aims at developing successful methods for the discovery and confirmation of causal relationships, epistemology is rather interested in the conceptual issues behind those methods. Metaphysical, epistemological, and methodological issues ought not to be conflated and a joint investigation from these three perspectives will hopefully allow us to get a better grip on causation.

This paper entirely locates within the domain of epistemology and methodology. Namely, the focus is on how we come to know about causal relations, regardless of the position one may take about the metaphysics of causation, i.e., about what causation in fact is. The motivation for an epistemological perspective comes from difficulties in 'metaphysical arguments', according to which, simply put, genuine probabilistic dependencies are causal dependencies, i.e. genuine correlations are per se causal. An example is Hausman (1998) who reiterates the idea that true correlations are causal and that if, eventually, the correlation turns out to be spurious this means that we picked out the 'wrong' correlation (see, e.g., Hausman 1998, p.33, 56). Differently put, correlation does prove causation—accidental correlations, in Hausman's view, simply aren't true correlations. To be sure, what is meant by these kinds of argument is that modulo certain conditions—most typically the so-called Markov condition—correlations are causal. However, it is highly a controversial matter whether the Markov condition ensure causality (see for instance Hausman and Woodward 1999 and 2004 and Cartwright 2002 on this point).

There is also another problem with 'metaphysical arguments'. A stock example is the positive correlation between the increasing number of storks and the increasing of birth rates in Alsace. According to arguments à la Hausman, this correlation is accidental, not genuine, hence not causal. Agreed, it might well be the case that genuine correlations be causal, but why we do not believe that the increasing number of storks is causally related to the increasing number of births? The answer, it seems to me, is that our background knowledge does not contain any theory or piece of information that makes this correlation plausible, let alone plausibly causal, in any possible way.

In a similar vein, Cartwright (1989) believes that capacities are responsible for, or give raise to, stable regularities and therefore are the very ontological basis of any observed statistical correlation. Capacities have the peculiar feature of being stable across different background conditions, but the question is, in the first place, how do we know that something is a capacity. At times, Cartwright (1989, pp.148 ff) seems to introduce a circularity in the account because the main point of econometric model is to test stability of relationships between variables; but those relations will be stable in case they are the effects of capacities, therefore stability cannot be a test to know whether something is a capacity in the first place. Steel (2008, pp.82-85) rises a similar worry concerning extrapolation, because if a causal relation has to have the feature of being stable across different contexts, then capacities cannot be what grounds extrapolation inferences exactly because what we are trying to establish is whether (stable) capacities are at work.

Thus, even if we assume that there are true causes that give rise to the observed correlations, the question still remains as to how we come to establish that some correlations, and not others, are causal. Echoing Granger (2003), the question is whether 'causality' applies to the model or the data generating process. I'd opt for the former option, in particular, my interest then lies in the process of model building and model testing, and, within in this process, I aim to highlight the conditions under which econometric models can be given a causal interpretation. This task I shall undertake in section 4.

3. STANDARD VIEWS

The literature in econometrics and social science is indeed vast, but two broad families of arguments can be identified nonetheless. A first family of arguments is centred around the notion of mechanism and the second around the assumptions of econometric models. To be sure, those two families are not mutually exclusive and there in fact a version of the 'mechanism argument' that eventually collapse into the 'assumption argument. Let me explain further.

Mechanisms are usually central for those who advocate a structural approach in econometrics. Simply put, and leaving aside the technicalities, structural models explain economic phenomena by means of (sets of) equations that describe causal mechanisms.

In the philosophy of economics and social science, a noteworthy partisan of the mechanistic approach is Little (1990), who claims that causal analysis in the social sciences is legitimate insofar as models identify social mechanisms. Little believes that such social mechanisms work through the actions of individuals—a position also known as methodological individualism. Hoover (2001), instead, stresses the causal import of the structural approach in econometrics arguing for a reality of macroeconomic structures that does not boil down to the reality of microeconomic relations. Hoover also tags along with mechanistic approaches because a causal structure is, in his view, a "network of counterfactual relations that maps out the underlying mechanisms through which one thing is used to control or manipulate another" (2001, p.24). The idea behind approaches à la Little (but not necessarily à la Hoover) is that there is a causal relation between A and B if and only if there is a mechanism that links A to B—a position hold, in slightly different ways, also by other 'mechanical philosophers'. Nevertheless, the emphasis on mechanisms is famously criticized by Kinkaid and more recently by Reiss. Kinkaid (1996), for instance, thinks it is false that in order to know whether X causes Y at least a mechanism linking X to Y has to be identified. Reiss (2007b and 2008, ch.6), along the same lines, argues that mechanisms might not be the most useful strategy to achieve other goals, for instance measuring concepts such as 'inflation rate' or 'unemployment rate'.

A different emphasis on mechanisms is given by economic theorists, who describe economic mechanisms that work 'ceteris paribus'. Such position is typical of classical economists such as Adam Smith, David Ricardo, Thomas Malthus, and John Stuart Mill, or the Chicago School of Economics. One way or another, it is the economic theory that 'dictates' the mechanism, instead of conceiving the whole modelling procedure as the attempt to identify what the mechanism is and how it works. It is in this vein that, according to Heckman (2008), structural econometric analysis has the following peculiar feature: it aims to model the generation of the outcome (i.e., the dependent variable) taking into account the agent's decisions to undertake a treatment. Thus, the outcome is explicitly modelled "in terms of its determinant as specified by [economic] theory" (p.18, my emphasis).

Within literature of econometricians and statisticians, instead, another trend can be recognized. The goal here is to model the stochastic mechanism that generates the data—in their jargon, the data generating process. The specification of the data generating process, in

turn, depends on the statistical model, and, consequently, the assumptions of model play a crucial role. Freedman (2004), for instance, distinguishes between statistical and causal assumptions and requires interventions to grant causal inferences. The crucial assumptions, in his account, are the causal ones, which eventually consist in assuming that structural equations unveil the causal mechanism that generate the observed data. This way, however, there isn't much difference between this methodological argument and the metaphysical arguments mentioned in the previous section. In fact, under this account the causal interpretation of structural equation consists in assuming that there is mechanism behind but stay silent on how we come to establish whether there is such mechanism. Holland (1986) goes a step further and draws a distinction between associational models and causal models, where the former simply make descriptive claims about conditional distributions and the latter also aim to quantify the causal effect of a treatment or intervention. Stone (1993), finally, focuses specifically on the causal assumptions, ranking them from the strongest—i.e., covariate sufficiency—to the weakest—i.e., ignorable treatment assignment.

All these arguments certainly get right part of what is at stake but not, I contend, the whole story. Mechanisms certainly play a role here. But why? Is it because we assume the existence of a given causal structure that we believe gives rise to the observed distributions? Or is it because we aim to model a causal mechanism? Assumptions are certainly central too. But what is exactly their import in justifying the causal interpretation? The story I want to tell somehow embraces both those views. Notably, I will defend the idea that we have to model mechanisms paying particular attention to the different types of assumptions made in the model.

Somehow, the view defended here is mid-way between the 'deductivist' and 'inductivist' approaches in econometrics (Moneta 2007). In the former, causes are 'given' by the economic theory; although there is some degree of freedom as to what economic theory to choose, once a choice is made, this imposes the restrictions on the model. Econometrics is thus reduced to measuring (statistical) relations between variables, rather then (dis)confirming causal hypotheses. In the latter, causes are inferred from statistical properties of data alone, by imposing to the model the simplest causal structure that allows identification—a methodology that strongly resembles present-day graphical models. The view defended here locates in between those two positions (i) because economic theory has to play a role in the model building and model testing process as part of background knowledge, but does not have to be the sole element to determine the choice of variables and the interpretation of results; (ii) because statistical analyses, where we let "the data speak as much as possible"—to echo Moneta (2007, p.119)— independently of any a priory economic theory can also play a role in the model building process but do not exhaust the causal analysis itself.

4. 'CAUSAL' ECONOMETRIC MODELS

The arguments offered next hinge upon the distinction and comparison of two classes of models: associational models and causal models. It is commonly agreed that associational models just make descriptive claims about conditional distributions, whereas causal models, in addition, aim at evaluating statistical relevance relations to 'quantify' the causal effect of the explanatory variables on the response variable. However, this cannot be the whole story, since it still stays unclear how, in causal models, correlations suddenly turn into causal relations and probabilistic dependencies into causal dependencies. In the following, I argue that differences between associational models and causal models can be identified at three levels: (i)

background knowledge, (ii) assumptions, and (iii) methodology. The difference between associational and causal models is schematically represented in table 1.

Associational Models	Causal Models
Statistical Assumptions	Background knowledge/Causal context Statistical Assumptions Extra-Statistical Assumptions Causal Assumptions Hypothetico-deductive Methodology

Table 1: Associational Models vs. Causal Models

ASSOCIATIONAL MODELS

The goal of associational models is to describe how a given variable (the dependent variable) varies depending on other variables (the independent ones). Associational models are typically used to make exploratory analyses of data in order to see what correlations between variables hold. Background knowledge does not play any particular role in associational models and variables do not play specific causal roles. Associational models rest on a number of standard 'statistical assumptions'. Leaving technicalities aside, we suppose that the model have some characteristics (usually, linearity and normality) such that it is easy to manipulate, easy to estimate statistically, and the resulting estimates have nice properties. We also assume that variables are measured without error, that the errors are not correlated with the independent variables. When these assumptions are satisfied, the conditional distribution correctly describes how variables co-vary. But at this stage there is no necessary causal information conveyed by the parameters, nor it is generally valid to give the regression coefficients the causal interpretation. Also, dependencies described in associational models are symmetric, which doesn't tell much about causation, in fact.

To interpret the coefficients causally means that the coefficients appearing in the equations measure the effect on the dependent variable caused by a change in the independent variable(s). To go beyond the descriptive claims we need (i) accurate knowledge of the causal context, (ii) further assumptions, and (iii) a methodology to confirm/disconfirm causal hypotheses.

CAUSAL MODELS

Causal models are equipped with a much richer apparatus than associational models simply do not have. This involves: (i) background knowledge, (ii) further assumptions, and (iii) hypothetico-deductive methodology.

BACKGROUND KNOWLEDGE

Background knowledge certainly includes the economic theory but also includes general knowledge of the socio-political context, knowledge of demographic characteristics of the population under investigation, or 'institutional' knowledge (i.e., knowledge of the functioning and procedures of an institution such as the Central Bank). This is what many social scientists usually call 'field knowledge'. In some cases, notably when dealing with disciplines that need to include biological variables, background knowledge may also include knowledge of the physical-biological-physiological mechanism. This is the case, for instance, in epidemiology, where one of the objectives is to understand how health variables affect socio-economic variables, or the other way round. Well established scientific theories also belong to filed knowledge. No doubt evidence is important for background knowledge. In particular, evidence of the same putative causal relations operating in different populations may justify further research, or evidence about different causal relations operating in other populations may justify a different modelling strategy. Thus, the use of different/similar data and/or models also belongs to background knowledge.

ASSUMPTIONS

Let us now turn to the assumptions needed in causal models. Beside standard 'statistical assumptions' also made in associational models, causal models have two extra sets of assumptions that I shall call the 'extra-statistical' assumptions and the 'causal' assumptions.

By 'extra-statistical' assumptions I mean all those assumptions that either are not related to the statistical properties of the distributions or have causal meaning but are not subject to statistical test. Among these assumptions we find, for instance, the direction of time, the direction of the causal relations, the causal mechanism.

By 'causal' assumptions I mean all those assumptions that are subject to statistical test and that contribute to interpret causally the relations between variables in the model. Two causal assumptions are particularly important: 'covariate sufficiency' and 'no-confounding'. Covariate sufficiency states that the independent variables are all the variables needed in order to explain the variance of the dependent variable. The assumption of 'no confounding' then plays a complementary role and means, simply put, that we ruled out other factors liable to 'screen off' the variables we took into account. These assumptions are causal because the causal interpretation crucially depends on them in the following sense.

Causal modelling rests on the so-called 'closure of the system'. This assumption says that the system under analysis is not subject to external influences and thus can be separated, so to speak, from the larger web of interrelations in which it is located. Thanks to this assumption we can, at least in principle, detect the causes acting in the system under investigation. The assumption of the closure is related to covariate sufficiency and no-confounding thus: if we put those three pieces of the puzzle together—(weak) closure, covariate sufficiency, and no-confounding—we end up with a causal model that describes a (quasi) hermetic mechanism where all and only the chosen explanatory variables play a role.

HYPOTHETICO-DEDUCTIVE METHODOLOGY

In order to tell causal stories, econometric models have to employ a hypothetico-deductive methodology. Simply put, hypothetico-deductivism is the view according to which scientists

first formulate hypotheses and then test them by seeing whether or not the consequences derived from the hypotheses obtain. Popper (1959), who developed the H-D methodology, was motivated by the need of creating a scientific method in a non-inductive way. However, in causal analysis, hypothetico-deductivism takes a slightly different facet specifically concerning deduction and, mostly, is not strictly falsificationist in character. Yet, it does borrow from the Popperian account the primary role of the hypothesis-formulation stage. I shall get back to this point later. According to the H-D methodology, model building and model testing essentially involve three stages:

1. formulate the causal hypothesis;
2. build the statistical model;
3. draw consequences to conclude to the empirical validity or invalidity of the causal hypothesis.

The causal hypothesis to put forward for empirical testing does not come from a tabula rasa, but emerges from a causal context, namely from background knowledge. The hypothesis formulation stage may also be informed by results of associational models as they suggest interesting correlations to submit to further scrutiny. The rest of the model building and model testing process is meant to (dis)confirm the causal hypothesis. In practice, this requires building the statistical model, and then drawing consequences from the hypothesis in order to test the hypothesis against empirical data.

It is through the estimation of the statistical model and through hypothesis testing that we will (dis)confirm the causal hypothesis. If the model is correctly estimated and fits the data, and if certain conditions are satisfied (notably, exogeneity and invariance) the hypothesized causal link is accepted, rejected otherwise. The hypothetico-deductive structure of causal modelling is thus apparent: a causal relation is first hypothesized and then put forward for empirical testing. That it to say, the causal hypothesis is not directly inferred from the data, as is the case with inductive strategies, but accepted or rejected depending on the results of tests and on background knowledge.

As anticipated above, hypothetico-deductivism in causal modelling does not involve deduction *stricto sensu*, but involves a weaker inferential step of 'drawing consequences' from the hypothesis. Once the causal hypothesis is formulated out of the observation of meaningful co-variations between the putative cause(s) and the putative effect and out of background knowledge, we do not require data to be implied by the hypothesis but just that data conform to it. Here, 'conform' means that the selected indicators and relations among them adequately represent the conceptual variables appearing in the causal hypothesis and the relations among them. Thus, this way of validating the causal hypothesis is not, strictly speaking, a matter of deduction, but surely is, broadly speaking, a deductive procedure. More precisely, it is a hypothetico-deductive procedure insofar as it goes the opposite direction of inductive methodologies: not from rough data to theory, but from theories to data. For a discussion of the H-D methodology, see also Little (1998, ch.9), Cartwright (2007, ch. 2) and Russo (2008, ch. 3 and 2009).

The two main tests of model parameters concern invariance and exogeneity. Invariance tests check whether the relation between two variables is stable across different environments, for instance under intervention or across different panels of the data set. The invariance condition acts as a constraint on the causal relation. This means that invariance does not constitute causation, but is a feature we require in order to interpret the parameters as causal parameters. The idea behind exogeneity is the following. Exogeneity is a condition of separability of inference. Given a data set containing a number of variables of interest, the goal

of modelling will be to decompose an initial joint probability distribution of all the variables into a sequence of marginal and conditional probability distributions. This (statistically) corresponds to identify and specify the variables playing a specific causal role and the way those causal variables interrelate. Background knowledge is of primary importance in guiding the choice of the marginal-conditional decomposition.

A CASE STUDY: HEALTH AND WEALTH IN ELDERLY AMERICANS

A study published in the Journal of Econometrics (Adams et al 2003) examined the possible causal links between health and socioeconomic status (SES) in elderly retired Americas. The links between health and SES have been the object of numerous studies. A survey of the literature shows that a significant association holds for a variety of health variables and alternative measures of SES. Also, much discussion has been devoted to the mechanisms behind this association, and yet there have been relatively few studies that allow causal paths to be definitively identified. This study adopts a particular methodology and applies statistical methods to test for the absence of direct causal links from SES to health, and from health conditions to wealth.

Adams et al. examine a sample from the population of elderly Americans aged 70 and older, in particular they examine whether innovations in health and wealth in a panel are influenced by their past values and by the past values of other relevant variables. Data come from three surveys performed between 1993 and 1998, providing information about health, wealth, and demographic information of individuals. A first analysis of data shows a significant association between health and SES. On the basis of background knowledge, including previous studies and knowledge of the socio-demographic conditions of elderly Americans, Adams et al. hypothesize that the links between health and wealth conditions can be represented as (roughly) sketched in fig. 1.

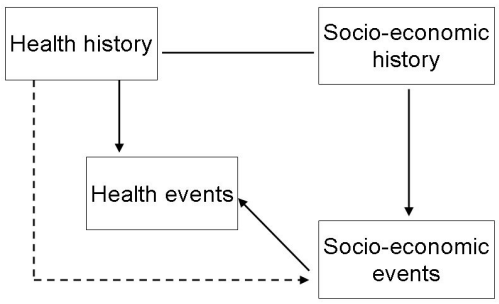

Fig.1 - Possible causal links between health and wealth

In the graph, arrows represent causal relations; when the line is dotted, the causal relation is absent. Thus, Adams et al., based on background knowledge and preliminaries analyses of data, expect that no significant causal link be found from health history of retired people to their present wealth status. On the contrary, they expect that socio-economic history, through their current wealth status, does affect their current health.

Following the approach of Granger (1969), two specific causal hypotheses are put forward for empirical testing: (i) there is no causal link from health to SES, and (ii) there is no causal link

from SES to health. Those two hypotheses are evaluated by means of non-causality test and invariance tests. Very informally, the structure of non-causality tests, in the approach of Granger (1969), is as follows: the present state of a variable (say, current health of individuals in the sample), causally depends on all its history (that is all the health history of individuals in the sample) and on the history of other relevant variables (say, SES of individuals in the sample). Thus, if SES and health turn out to be independent, then we say that SES does not Granger-cause health. Invariance tests, instead, aim to check whether model parameters stay stable across different panels.

In testing the significance of SES on a number of health outcomes, the authors draw the following conclusions: SES is, usually, strongly associated with health; however, the link is weaker when they control for previous health problems; also, the association between SES and mental and chronic illness is stronger than the association between SES and acute and sudden outset health conditions. Adams et al. suggest that those findings reflect the differential coverage of Medicare; in fact, insurances typically do not cover mental illness.

This simple presentation of the case study already illustrates the hypothetico-deductive methodology used. Look at where causality is; causality is not at merely the output, pulled out of the statistical model. Instead, the two causal hypotheses are put forward for empirical testing within the model, and then accepted or rejected depending on the results of test and on whether the results are congruent with background knowledge. To illustrate the features of causal models discussed above, let us discuss each feature separately.

Background knowledge and causal context. In Adams et al. work, it includes previous studies on the relations between health and wealth. In particular, the association between health and wealth holds for a variety of health variables and alternative measures of SES. Therefore, a causal interpretation of those correlations is plausible and worth testing.

Causal hypothesis. In the health and wealth case study the causal hypothesis is split into two: (i) is no causal link from health to SES, and (ii) there is no causal link from SES to health. In order to evaluate the causal impact of SES on health and vice-versa, Adams et al. build a model following the approach of Granger and perform non-causality and invariance tests on the causal hypotheses specified.

Statistical assumptions. The statistical analysis fits the approach of Granger, which is essentially based on regression methods and where standard statistical assumptions hold.

Extra-statistical assumptions. In this case study two extra-statistical assumptions are at stake: causal priority and causal ordering. In fact, in Granger-approach it is explicit that the history of variable causally determines the current value of the variable. Accordingly, statistical tests are about whether heath histories determine current wealth and whether wealth histories determine current health.

Causal assumptions. For the most part, Adams et al. discuss the assumption of covariate sufficiency, that is all the relevant explanatory variables have been included. Another important assumption concerns and structure of the causal relation: by adopting Granger's approach, instantaneous causality is ruled out, at least for sufficiently brief time intervals.

Tests. Within a 'Granger-causality' framework, Adams et al. test model parameters for invariance across different panels. More explicitly, they test (i) whether the conditional distribution 'current health given wealth history' and (ii) whether the conditional distribution 'current wealth given health history' hold stable across different panels analyzed. The invariance conditions (largely) fails in the first case, i.e. variations in health generally do not correspond to variations in wealth, hence, the hypothesis of no causality is accepted. On the other hand, the conditional distribution 'current wealth given health history' results (relatively) stable and therefore the hypothesis of no causality is rejected.

Hypothetico-deductive methodology. Adams et al. are clearly using a hypothetico-deductive methodology in their analysis. They first formulate the causal hypotheses out of background knowledge and then, iteratively, build the model and perform statistical tests to confirm or disconfirm the causal hypotheses. Background knowledge and causal context play a primary role at each stage: from the choice of variable until the evaluation of results. A clear identification of the steps in the process of model building and model testing allows to identify possible leaks or points of disagreement between scientists. Whilst there is a shared consensus on the background of this study (see the commentaries in the same volume of Adams et al.'s paper), a number of commentators suggested that a structural approach would be better suited to study the links between health and wealth (Hausman (2003), Heckman (2003), Hoover (2003)). The particular form of the invariance tests is also debated. For instance, Hoover (2003) agrees that invariance is an important indicator for the presence of a causal relationships, but doubts on the cogency of the evidence used by Adams et al. Analogously, remarks on the chosen definition and tests of non-causality also come from Florens (2003). Adda et al (2003) object to their suggestion that the observed links between SES and health are the effect of the access people have to health infrastructures. The reason is that Adda et al. apply the very same methodology to two different data sets, one from UK and one from Sweden, and get very similar results. However, since medical systems are very different in those countries, coverage of Medicare cannot be what produces these results. As a consequence, policies intervening in that direction would be, in Adda et al.'s view, misplaced.

EPISTEMOLOGICAL MORALS

Let me know state more precisely the epistemological morals to draw from the arguments given above. I undertook this path of investigation to overcome the dissatisfaction with available arguments that either assume a causal structure instead of saying how we come to establish it, or with methodological arguments that stop too early in praising the role of assumptions in causal models. In order to make causal claims we need different types of assumptions. The distinction I drew between statistical, extra-statistical, and causal assumptions is meant to make clear the import of each of them towards establishing causal relations. For instance, extra-statistical assumptions such as causal ordering, usually come from knowledge of the causal contexts, are typically not tested in the model, and yet participate in causal attribution because they serve, for instance, to fix which variables are the causes and which variables are the effects. Nevertheless, the main point of this distinction is to show that it is not the case that the stricter the statistical tests the greater our confidence in the causal interpretation. Causal relations are not pulled out of statistical hats. The hat, so to speak, is itself causal. The conceptual hypothesis is in fact a causal claim the evaluation of which demands for empirical testing. In other words, causality is a matter of confirmation, or borrowing the statistical vocabulary, it is a matter of accepting or rejecting a given causal hypothesis.

Let me linger more on this 'causal hat'. Model building and model testing take place in a causal context. Background knowledge plays a major role in the preliminary analyses of data, in the selection of variables, and in the formulation of causal hypotheses. It is also on the basis of background knowledge that the choice of testing for invariance some conditional distributions, but not others, relies. It is, finally, on the basis of background knowledge that results are evaluated. The importance of background knowledge, however, goes beyond the choice of variables and for the formulation of the causal hypothesis. It is also important in order to rule out highly implausible causal relations before starting the whole machinery of

model building and model testing. In manipulationist accounts à la Woodward (2003), the relation between the increasing number of storks and the increasing number of births is not causal because it would turn out not to be invariant under intervention; namely, no manipulation on the variable 'number of storks' would lead to changes in the variable 'number of births'. Nevertheless, should we go as far as testing for invariance in cases where we can exclude that the correlation is causal on the basis of background knowledge?

In the literature, the crucial role of the causal context and of the conceptual framework has been stressed by many eminent scientists and philosophers—jut to name a few: Fisher (1925), Haavelmo (1944), Kendall and Stuart (1961), Suppes (1970), Humphreys (1989). And yet, this emphasis hasn't been given the proper importance. It has oft been taken as a platitude about causal modelling—causal methods rely on background knowledge in an obvious sense—or as a 'hidden challenge'—what in fact background knowledge taught us that we did not already know? In other words, if model building and model testing are largely based on background knowledge, how do we go beyond it and gain causal knowledge?

What allows us to go beyond background knowledge is the hypothetico-deductive methodology of causal models. This is a dynamic methodology that allows a *va et vient* between established theories and establishing theories. Established scientific theories are (and ought to be) used to formulate the causal hypothesis and to evaluate the plausibility of results on theoretical grounds. But causal models also participate in establishing new theories by generalizing results of single studies. This reflects the idea that science is far from being monolithic, discovering immutable and eternal truths. If the model fits the data, model parameters are (sufficiently) invariant and the relations are congruent with background knowledge, then we can say, to the best our knowledge, that we hit upon a causal relation. But what if one of these conditions fails? A negative result may trigger further research by improving the modelling strategies, or by collecting new data, thus leading to new discoveries that, perhaps, discard background knowledge.

A last corollary of this epistemological regard on econometric models is that, by and large, causal models model mechanisms. As mentioned above, hypothetico-deductive causal models aim to (dis)confirm a causal hypothesis; the causal hypothesis, however, states something more than simply a causal relation—it's about a causal mechanism. Let me develop this idea further.

What do causal models do? Causal models model the properties of a social system. In particular, they model the relations between the properties or characteristics of the system, which are represented by variables. To model the properties of a social system means to give the scheme, or the skeleton, of how these properties relate to each other. However, this causal mechanism is not modelled in terms of spatio-temporal processes and interactions à la Salmon (1984 and 1990) but is statistically modelled. Concepts typical of statistical causality—e.g., statistical relevance, comparison of conditional probabilities, and screening-off—are used in order to identify the types of relationships that hold among the variables of interest. This is how the causal model models the causal mechanism governing the social system. In particular, causal models seek to uncover stable variational relations between the characteristics of the system.

This view of mechanisms is not perfectly in line with more 'physical' views such as those of Glennan (1996), Machamer et al. (2000), Bechtel and Abrahms (2005), or Craver (2007). However, it is not in opposition either. Craver, for instance, echoing the definition given by Machamer et al (2000), conceives of mechanisms as "a set of entities and activities organized such that they exhibit the phenomenon to be explained" (Craver 2007, p.5), and calls it a "skeletal description". Such characterization of mechanisms, I believe, is broad enough as to account for mechanisms in various domains. Should we take the entities to be neurons and the

activities neurotransmitters release, the above skeletal description will well fit neuro-mechanisms. Should we take entities to be socio-demo-economic variables, and activities to be their influence on other socio-demo-economic variables, the above skeletal description will fit equally well social mechanisms. The degree of 'physical' reality one wishes to give to entities and activities may lead to different accounts — notably, to a different ontological commitment to the existence — of mechanisms. In the social contexts we do not necessarily need to endorse the view that elements and relations should always have 'physical' counterparts. This would lead us into debates concerning social ontology and methodological individualism, which, of course, are far beyond the scope of the present discussion.

5. CONCLUDING REMARKS

Econometrics applies statistical methods to study economic relations. By means of structural models, econometricians typically account for the response variable in terms of a number of explanatory variables. Under what conditions can econometric models be given a causal interpretation? This paper defended the view that (i) a proper use of background knowledge, (ii) statistical, extra-statistical, and causal assumptions, and (iii) hypothetico-deductive methodology provides sufficient conditions for the causal interpretation. On the contrary, associational models do not allow any causal interpretation as they are just equipped with statistical assumptions. This goes against the views that causal structures are simply assumed and are that which is responsible for the observed correlations, and against the views that 'bootstrap' causal relations from stringent statistical tests alone. The causal interpretation is instead a matter of confirmation. In hypothetico-deductive terms, the goal is to confirm (or disconfirm) a hypothesis that is explicitly causal. Nancy Cartwright famously expressed this idea with the motto 'No causes in, no causes out'. In spite of appearance, the account is not circular, and if there is a circle at all, it is virtuous and not vicious. To put causality in the model means that the evaluation of causal relations is relative to some conceptual and formal framework; in this framework we find background knowledge, causal assumptions, and hypothetico-deductive methodology. The account offered gives sufficient conditions that allow to interpret econometric models causally. It is worth emphasize that such account deeply depends on the epistemological perspective I adopted. By focusing on how we come to establish causal relations rather than on what causation in fact is, I implicitly endorsed an epistemic view of causality very much in line with the view defended by Williamson (2005). According to this view, results of econometric models are not immutable and eternal causal truths, but justified beliefs, which are fallible. This fact is indeed mirrored in the hypothetico-deductive methodology of causal models which, being dynamic and flexible, allows us to revise our causal beliefs by building and testing new 'causal' econometric models.

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