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STATISTICAL MODELLING AND CAUSALITY IN THE SOCIAL SCIENCES

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Introduction

Philosophers and statisticians have been debating on causality for a long time. However, these discussions have been led quite independently from each other. An objective of this paper is to pursue a fruitful dialogue between philosophy and statistics. As is well known, at the beginning of the 20th century, some philosophers and statisticians dismissed the concept of causality altogether. It will suffice to mention Bertrand Russell (1913) and Karl Pearson (1911). Almost a hundred years later, causality still represents a central topic both in philosophy and statistics.

In the social sciences, including research on public health, most studies are concerned with the possible causes, determinants, factors, *etc.* of a set of observations. In particular, for planning or policy reasons, it is important to know what causes which effects. In order to attain causal knowledge, many social scientists appeal to statistical modelling to confirm or disconfirm their hypotheses about possible causal relations among the variables they consider, taking care of controlling for relevant covariates and especially for possible confounding factors.

To what extent can a statistical model say something about *causal* relations among variables? In this paper, we will attempt an answer by examining a special class of statistical models, *i.e. structural models*. The discussion, however, will not be confined to a mere examination of statistical methods, since a considerable effort will be made to consider causality from an *epistemological* perspective. To put it otherwise, this paper does not address the *nature* of causation itself, nor the analysis of various causal structures, nor the elaboration of complex causal structures; rather, we will be concerned with the question of how we come to uncover causal relations by means of statistical modelling.

The practice of statistical modelling raises substantial issues of ontological nature. The latter is not the purpose of this paper. Nevertheless, as statistical models are a common way of gaining scientific knowledge, we begin in section I by stating a moderate realist position concerning the relationship between reality and observation. This point of view is not necessarily shared by all philosophers and scientists.

I. Models and Scientific knowledge

In order to gain cognitive access to reality, scientists typically construct models; social scientists are no exception to this rule (Blalock 1964, Franck 2002). Broadly speaking, a model may contain statements, schemes, figures and mathematical expressions. The notion of model is central to present-day philosophy of science; so far, however, no proposed account of what a model is has managed to attract universal consensus. The following remarks are thus aimed at singling out the characteristics of what we take models to be in the context of statistical modelling in social sciences.

A model is not a personal mental image of some reality. Models are not private psychological entities but intersubjective constructions which can typically be found in

scientific textbooks and articles, and taught to students. For a social scientist in general, a model is not a set-theoretical structure that satisfies or makes true a given set of statements; this formal, mathematical, notion of model has influenced many a philosopher of science (Suppes 2002, van Fraassen 1980, Giere 1999). Social scientists take the core of a model to be a set of assumptions, *i.e.* statements, which aim at providing a simplified account of a complex reality. Of course, this view does not prevent the possible satisfaction of these assumptions by some set-theoretical structures, *i.e.* models in the mathematical sense.

Models however, as the term is used by social scientists, are not purely mathematical. Although the vast majority of scientific models contain mathematical assumptions, they usually contain non mathematical components such as explanatory statements, pictures, schemata *etc.* Giere (1999) compares models with geographical maps. But maps function essentially as pictures of some territory, whereas the models used by social scientists contain both pictorial elements, such as drawings, figures *etc.*, and components which are not figurative, such as statements. Provisionally, we will take a model to be an abstract object which permits partial cognitive access to some real systems. Systems are portions of reality which can be purely observable or also contain non observable parts.

Realists claim that at least some parts of a model correspond to some elements or aspects of a real system. This claim is notoriously controversial. Yet, the empirical success of a model, *i.e.* its ability to lead to sensible explanations and to correct, precise and even novel observational predictions, provides good grounds for the realist's belief in such partial correspondence. For many realists, a model is a – at least possible – representation of a real system. "Representation" is a hard-to-define, elusive concept. For social scientists a representation is not necessarily an image or a picture. Imaging is a particular case of representing in which there is some isomorphism or identity of form between the image and what is pictured. In a picture of a system, the organization of the elements of the picture mirrors the arrangement of the corresponding elements in the real system. Since models contain statements and since statements are not pictures of possible facts, *pace* Wittgenstein (1961), some parts of the model are not images. For our present purpose, it will be sufficient to say that parts of a model may correspond to some aspects of a represented system. No model pretends to capture all aspects of a real system. Some characteristics are consciously or unconsciously disregarded. Modelling consists in abstracting and results in constructing a simplified representation of a complex reality; thus, it always involves some degree of idealization. More specifically, a moderate realist approach takes models as approximations of a complex reality, in the sense that some of the statements implied by the model are only approximately true. In section III, statistical models are viewed as partial representations of a data generating process, possibly interpreted as the underlying reality. The random component of the statistical model constitutes the unexplained part of the model. Some idealizations and approximations are explicit and explain why a model does not fit the data perfectly; however, some discrepancies with the observations or data are left unexplained.

Fitting the data certainly is a widely agreed condition of adequacy. But what does fitting the data mean? Making true predictions or statistically accurate predictions within certain limits surely is part of the story. A model itself determines what counts as a relevant observational result since those results must have corresponding counterparts in the model; typically, the observational results are denoted by the values of variables. However, a model by itself does not contain the criteria for deciding if a given observational result counts as a confirmation or a refutation. Such a decision is external to the model and hinges on the amount of error deemed acceptable for practical purposes. This is the case in the natural sciences too: in some circumstances a model of geometrical optics may be considered adequate, while in other contexts we will have to use models of wave or quantum mechanical

optics. Thus, good data fitting or empirical adequacy is also judged in function of the ends and purposes pursued (Giere 1999).

Contrary to realists, empiricists such as van Fraassen (1980), restrict the correspondence of a model with reality to the data level. Unobservational components in the model may have counterparts in reality, but we have no means to assure ourselves that they exist because we do not have empirical access to them. As an empiricist, van Fraassen holds that the only cognitive access to reality is observation. Realists, usually called scientific realists, claim that we have reasons to believe in the existence of some unobservable entities or processes as well. Some theoretical statements of our models can be asserted to be true provided they play a role in achieving empirical adequacy, especially in accounting for new observations. Some parts of schemata or figures may have a counterpart in reality and this can be attested by empirical evidence and measurements. For example, the angle between the atoms of a molecule can be measured. Usually, the correspondence of an element of a drawing *etc.* with reality can be expressed by a proposition. Here, we will espouse a moderate form of realism according to which models permit to have cognitive access to some unobservable aspects of real systems. Granted, models are falsifiable and science is not the locus of definitive and infallible truth.

This moderate realist position has a bearing on the status of causality. A social scientist does not rest content with good data fitting but also attempts to construct models which provide a causal explanation of those data. A causal structure or relationship among variables is first articulated within the model. In some instances, it can be held as a working hypothesis. If a model achieves a good statistical fit with the data, especially when it succeeds in encompassing hitherto unknown data, then it is reasonable to believe that the model hits upon a real causal relationship. And we could capitalize on this in order to construct new models for different, but related, situations.

II. Data

Science tries to make sense out of observations, but the latter first have to be…observed. The selection of what one observes depends upon our underlying research questions and theoretical constructs. According to what we are looking for/at, we can use our eyes, a microscope, an electrocardiograph, *etc.* In demography for example, most data are collected by some sort of form: a census form, a birth certificate, a survey questionnaire, an inscription in a population register, and so on. The facts thus collected are however often far from perfect for the scientific enterprise. First, the data may contain voluntary or non-voluntary errors: erroneous income declarations, age-heaping in some less developed countries, sampling biases, *etc.*, including how the sample was drawn; for example, interviewing only hospital patients gives a biased image of the health of the whole population.

Then, there is the issue of time. The time-ordering of events is a prerequisite for causal analysis: causes should precede their effects in time, though this criterion is disputed by some (Horwich 1987). A cross-sectional observational scheme, as one knows, does not enable us to disentangle age, period, and cohort (APC), or their durational equivalent, effects. For example, it may show that hearing decreases with age and so presently does smoking, but the former is an age effect and the second is a cohort effect. Retrospective studies give a time-dimension to the data and enable us to distinguish APC effects or to time-order events relative to their possible causes, but they are influenced by recall lapses and only those persons alive and present can obviously be interviewed. If one can afford them, prospective longitudinal studies avoid these pitfalls, but they can be affected by loss to follow-up and they thus possibly lead progressively to a selected population.

Recall bias and selection bias are particularly difficult to model and correct (Freedman, 1999). In retrospective or prospective studies, one also has to choose an adequate time-frame: if the observation extends far into the future or the past, loss to follow-up and recall lapses respectively increase, as does the cost of the prospective survey! If the time-frame is too short, we might miss an important lagged effect, such as the deleterious impact of a drug occurring only several years after use. Case-control studies can be of help here, though they are rarely used in the social sciences.

Another serious observational problem in demography and in the social sciences in general is the fact that many of our variables are abstract notions such as social status or intelligence, which cannot be observed directly. In structural modelling, such as in the LISREL approach, one would call them *latent variables.* For measurement and comparisons, we must therefore agree on a common definition of these abstract concepts and on a procedure for deriving empirical measures satisfying the reliability (repeatable measures) and construct validity (accurate reflection) criteria (Babbie 2000). The problem is especially acute when contexts differ: may we use the same definition and indicators of education in Europe and in Africa, for example? Probably not. Taking into account the purpose of the study, the definition of a concept should determine a partition such that an object either is or is not subsumed under the definition, notwithstanding the possible existence of some cases of fuzzy membership. The possible various facets or dimensions of the concept should be clearly pointed out. Such a procedure is furthermore helpful in selecting the multiple indicators of the concept needed for empirical measurement. Even biological variables such as sterility are not always obvious or clearly defined; one needs in this case to distinguish between primary and secondary sterility and to avoid taking sub-fertility for infertility (Habbema *et al.* 2004). The indicators of these forms of (in)fertility will not be the same. According to our moderate realist stance, we can attain (provisional) causal knowledge of the system under analysis, in spite of these observational problems.

III. Causality and Statistical modelling

The statistical model

Formally, a statistical model **M** is a set of probability distributions; more precisely:

$$M = \left\{ S, P^{\theta} \theta \in \Theta \right\}$$

where S, called the *sample space* or observation space, is the set of all possible values of a given observable variable (or vector of variables) and for each $\theta \in \Theta$, P^{θ} is a probability distribution on the sample space, also called the *sampling distribution*; thus, θ is a characteristic, also called *parameter*, of the corresponding distribution and Θ describes the set of all possible sampling distributions belonging to the model. The basic idea is that the data can be analyzed *as if* they were a realization of one of those distributions. For example, in a univariate normal model, the sample space S is the real line and the normal distributions are characterized by a bivariate parameter, for instance the expectation (μ) and the variance (σ^2); in this case: $\theta = \left(\mu, \sigma^2 \right)$.

A *statistical model* is based on a *stochastic representation of the world.* Its randomness delineates the frontier or the internal limitation of the statistical explanation, since the random component represents what is *not* explained by the model. For instance, in the simplest case of repeated measurements (x) of the weight of a given object, the statistical model, derived from the equation $x = \mu + \varepsilon$, explains the expected measurement as the "true" weight (μ) of

the object to which is added an unexplained error of measurement (ε) modelled as a random variable with zero mean.

A *statistical model* is made of *a set of assumptions* under which the data are to be analyzed. Typical assumptions of statistical models are: the observed random variables follow or not identical distributions; the observations are, or are not, independent; the basic sampling distributions are, or are not, continuous and may pertain, or not, to a family characterized by a finite number of parameters (*e.g.* the normal distributions). When we deal with multivariate models, adequate assumptions might involve linearity (in the parameters and/or in the variables), non measurement error, or non correlation of error terms.

In particular, we often assume the model to be linear or approximately so. This is a matter of convenience, since a linear model is easy to manipulate, its parameters are easily estimated and the resulting estimators have nice properties. Often assumed, linearity may also be tested. The same holds for normal distributions. We may also assume that variables are measured without error and that the errors are not correlated with the independent variables.

If the statistical assumptions are satisfied, the statistical model correctly describes co-variations between variables, but no causal interpretation is allowed yet. In other words, it is not necessary that causal information be conveyed by the parameters, nor is it generally legitimate to give the regression coefficients a causal interpretation. It is worth noting that in specifying the assumptions typical of a statistical model, the problem is not to evaluate whether or not an assumption is true. A (frequentist) statistician may however want to test in due course whether a hypothesis is acceptable or not. In a sense, if a model-builder could prove that an assumption were (exactly) true, this would not be an assumption anymore, but a description of the real world. Rather, the main issue is to evaluate whether an assumption is useful, in the sense of making possible a process of *learning-by-observing* on some aspects of interest of the real world.

Statistical inference and structural models.

Statistical inference is concerned with the problem of *learning-by-observing* and is *inductive* since it implies drawing conclusions about what has not been observed from what has been observed. Therefore, statistical inference is always uncertain and the calculus of probability is the natural, and in a sense logically necessary tool (see *e.g.* de Finetti 1937, Savage 1954), for expressing the conclusions of statistical inference. Therefore, the stochastic aspect of statistical models involves a stochastic representation of the world *and* a vehicle for the learning-by-observing process.

Here, two aspects ought to be distinguished. On the one hand, learning-by-observing conveys the idea of learning about some features of interest, namely the characteristics of a distribution or the values of a future realization. On the other hand, learning-by-observing is also concerned with the problem of accumulating information as observations accumulate. These two aspects actually refer to the usefulness of the model. Structural models are precisely designed for making the process of statistical inference meaningful and operational.

To better understand the idea behind this last claim, it is worth distinguishing two families of models. In the first family we find *purely statistical* or *empirical* models, also called associational or descriptive models, exploratory data analysis or data mining. In these approaches, the assumptions are either not made explicit or restricted to a minimum allowing us to interpret descriptive summaries of data. Interest may accordingly focus on the distributional characteristics of one variable at a time, such as mean or variance, or on the associational characteristics among several variables, such as correlation or regression coefficients. It is worth noting that the absence or the reduced number of assumptions constituting the underlying model make these associational studies insufficient to infer any causal relations.

The second family consists in the so-called *structural* or *causal* models. "Structural" conveys the idea of a representation of the real world that is stable under a large class of interventions or of modifications of the environment. As a matter of fact, structural models incorporate not only observable, or manifest, variables but also, in many instances, unobservable, or latent, variables. The possible introduction of latent variables is motivated by the help they provide in making the observations understandable; for instance, the notion of "intelligence quotient" or of "associative imagination" might help to shape a model which explains how an agent succeeds in answering the questions of a test in mathematics. Thus a structural model may capture an underlying structure of the world. Modelling this underlying structure requires taking into account the contextual knowledge of the field of application in order to uncover the structural stability.

Structural models are also called "causal models". Here, the concept of causality is *internal* to a model which is itself stable, in the sense of *structurally stable*. The characteristics, or parameters, of a structural model are of interest because they correspond to intrinsic properties of the observed reality and can be safely used for accumulating statistical information, precisely because of their structural stability. In this context, a structural model is opposed to a "purely statistical model", understood as a model that accounts for observable regularities without linking those regularities to stable properties of the real world.

The *invariance* condition of a structural model is actually a complex issue. This is a condition of stability not of the causal variables, but of the causal relation itself. The idea is that each variable is determined by a set of other variables through a relationship that remains *invariant* when those other variables are subject to external influence. It is in this sense that we call the model "structurally stable". This condition allows us to predict the effects of changes and interventions. *Stability* of distributions is also assumed to ensure that the (conditional) independencies between variables will not be jeopardized by variations in the parameters (Pearl 2000 calls this condition "stability", but is also known as "DAG-isomorphism" (Pearl 1988) or "faithfulness" (Spirtes *et al.* 1993)).

Besides the assumptions of stability, or of invariance, the construction of structural models typically involves other assumptions such as: covariate sufficiency, no-confounding, independence of error terms, recursivity *etc.* It is worth pointing out that the correctness and usefulness of structural or causal models also rest on a set of untested, and often untestable, assumptions, which nevertheless play a fundamental role. In particular, at the *building* stage the direction of time is usually assumed to point from the past to the future. However, reasoning may be reversed at the *inference* stage; for instance, in a medical application, inference may concern a diagnosis conditional on observed symptoms, even though the pathology has been active before the symptoms appear.

Conditional models and exogeneity

By means of causal modelling, the social scientist often has to acknowledge that all the variables of interest cannot be taken into account by a unique structural model, because of too severe an environmental instability. The purpose of modelling becomes, under such circumstances, to uncover some structural, or stable, aspects of an unstable reality. In order to do that, a usual strategy consists in separating the data into two parts: $X = (Y, Z)$, along with a *marginal-conditional decomposition*, namely, in terms of density functions:

$$p(x \mid \theta) = p(z \mid \theta) \cdot p(y \mid z, \theta)$$

where the *marginal model*, constituted by $\{p(z \mid \theta), \theta \in \Theta\}$, describes how the data Z (alone) have been generated. The *conditional model*, constituted by $\{p(y \mid z, \theta), \theta \in \Theta\}$, describes the

conditional distribution of $(Y \mid Z)$, that is to be interpreted as describing the data generating process of the random variable *Y* relatively to a particular value of *Z* and, therefore, does not take into account the randomness of Z. In social science contexts, the standard practice is to gather in the marginal model the more unstable aspects of the real world, as conjectured on the basis of background and contextual knowledge. This makes plausible the assumption that only the conditional component of the model is structural. The burden of the assumptions then bears on the conditional distribution of the variables Y, leaving virtually free the marginal distributions of the conditioning variables Z.

In such a case, only the parameters describing the conditional distribution are considered of interest and, reciprocally, a variable Z is said to be *exogenous*, for a given parameter of interest, if this parameter of interest is a function of only the parameters identified by the model conditional on the exogenous variables, and if the parameters identified by the marginal model and by the conditional model respectively are independent in a Bayesian sense (or variation-free in a classical sense):

$$p(y,z|\theta) = p(y|z,\theta_1) \cdot p(z|\theta_2) \text{ with } \theta = (\theta_1,\theta_2) \in \Theta_1 \times \Theta_2$$

The condition of exogeneity justifies analyzing separately the marginal and the conditional sub-models. Note that the existence of a latent common cause for both Y and Z typically violates the condition of exogeneity.

Taking into account the previous definition of exogeneity, this leads us to the following concept of causality: *a causal variable is an exogenous variable in a given structural model*. Similarly to Suppes (1970), this concept of causality relies on conditional modelling, but this paper insists on the necessary structural stability of causal models. A simple example, given in Appendix I, illustrates the notions just introduced.

What makes causality a complex issue?

Exogeneity does not exhaust the problem of causality; indeed, structural stability is crucial for interpreting exogeneity as causality. Furthermore, structural stability makes sense only if consistent with the background knowledge. Up to now, we have considered a concept of exogeneity as a prerequisite to the causal interpretation of structural models. The latter are however more complex than a single marginal-conditional decomposition would suggest (Holland 1986).

We will now examine two *epistemological* aspects of causality. On the one hand, a *single* "marginal-conditional decomposition" may not properly account for the complexity of the relationships within a large number of variables of interest. On the other hand, a proper analysis of the concept of causality requires, at some stage, to acknowledge the role of time. The first facet will be labelled "atemporal", in order to pinpoint aspects for which the role of time is not essential. A simplified example which illustrates the various issues in this section is developed in Appendix II.

We begin with those atemporal features. The issue of determinism is definitely controversial. Structural equations may be considered functions in which probabilities come in through error terms representing some lack of knowledge (Cartwright 1989, Hausman 1998, Woodward 2003). We may otherwise adopt the statistician's viewpoint: the world of the statistician is stochastic, the error terms representing what is *not* explained. In truth, the epistemological perspective here endorsed allows us to set aside the *metaphysical* problem of determinism, *i.e.* whether or not the world is actually deterministic.

The complexity of the relationships among a large number of variables of interest requires to take further features into consideration. In some cases it is possible to order a vector of variables of interest in such a way that a systematic marginal-conditional

decomposition, *i.e.* $p(x_1, x_2, x_3) = p(x_1) \cdot p(x_2 \mid x_1) \cdot p(x_3 \mid x_1, x_2), \ldots,$ is structurally stable. In such a case the model is said to be *recursive*. When some components cannot be disentangled, for instance if $p(x_2, x_3 \mid x_1)$ cannot be structurally decomposed into $p(x_2 \mid x_1) \cdot p(x_3 \mid x_1, x_2)$ some authors speak of causal loops (for more details see Wold 1949 and 1954).

Though a causal factor may have a limited impact on an effect, and in this case may possibly be neglected, it is often assumed that a strong association between a possible cause and its effect is more likely to reflect a causal relationship (Elwood, 1988). A strong relationship might however be due to confounding. By ruling out other factors liable to screen-off the impact of the covariates taken into account, the assumption of *no confounding* has therefore a complementary role to covariate sufficiency. However it should be noticed that, as pointed out by Stone (1993, 459) "good explanations (of the notion of confounding) are surprisingly rare" in spite of "appearing in most epidemiology texts and (being) ubiquitous in the quantitative social sciences". Take a three-variable case *Y*, *Z*, *W*, for example, where we wish to measure the impact of a variable *Y* on another variable *Z*. If *Y* is associated with *W and* if the latter influences *Z* too, then part of the covariation between *Y* and *Z* will be due to the hidden presence of *W* if this latter variable is not controlled for, *i.e.*, is not taken into account in the model. In this case of model misspecification, *W* is called a confounding factor when measuring the relation between *Y* and *Z*.

Confounding is also related to the so-called Simpson's paradox, the resolution of which is based on the fact that the two conditions $Y \perp Z \mid W$ and $Y \perp Z$ are not linked by any logical implication, neither their negations. For example, ranking countries according to their crude death rate (the ratio of yearly deaths to the mid-year population size) can lead to very different and even inverse conclusions than if these same countries were ranked according to their expectation of life at birth. The second mortality indicator controls for population age structure while the first does not. If countries differ significantly according to their age structure, the latter confounds the actual mortality differentials existing between these countries as mortality is strongly related to age. Hidden confounders may therefore lead to inadequate measures of cause–effect relationships and possibly to structural instability of the model. If our background knowledge suggests the presence of possible confounders, the latter should always be included - data permitting - among the covariates.

By *causal asymmetry*, we mean that the two propositions "*Z* causes *Y*" and "*Y* causes *Z*" cannot hold at the same time: either the direction of causation is known and is unique or the direction of causation is unknown and this leads to a situation of simultaneity in the model.

The temporal aspect of a causal mechanism is generally accepted as an important component of the modelling effort. A first issue is evidently that of the *direction of time*. As mentioned above, we assume that the causal mechanism follows the direction of time from the past to the future and therefore that effects occur after their causes. Secondly, the problem of feedback loops is solved taking into account *causal priority*, *i.e.* the fact that causes precede their effects in time: $Z_t \rightarrow Y_{t'} \rightarrow Z_{t''}$, where $t < t' < t''$. That is, Y is an intervening variable between two temporally distinct values of *Z* and these two together guide the choice of *causal ordering*, *i.e.* the temporal order in which variables are observed. As we have pointed out in section II, however, causes and effects may appear to be simultaneous because the time-frame underlying our data is not always adequate for temporally ordering causes and effects. This is the reason why longitudinal dynamic models should always be preferred to cross-sectional ones if the purpose is to distinguish causes from effects.

Hypothetico-deductive methodology

Causal modelling is not concerned with the question whether the true causes of an effect are disclosed but rather with the issue of a good representation (of the world) which embodies a *causal* mechanism. For a moderate scientific realist, the process of model building involves a continuous interaction between a prior knowledge of the field and a sequence of statistical procedures for elaborating and testing the successive hypotheses, with the understanding that the true causes are attainable at least in principle. In practice, causal attribution incorporates accordingly educated guesses. Because, as explained above, causality is a property *within* a structural model, rather than a *prima facie* empirical problem, it is impossible to *deduce* causes from correlations in a purely statistical model. But this does not lead to a Humean sceptical despair: causal modelling is indeed a promising tool for causal attribution. In other words what vindicates causal models is the hypothetico-deductive strategy employed in much of contemporary science.

By hypothetico-deductive methodology we mean a procedure that will account for data obtained through observations and/or experimentation and that will confirm or disconfirm a given causal model by confrontation with empirical evidence (see below). Hypothetico-deductive methodology was developed already in the 17th century within experimental methods. In recent times, the hypothetico-deductive methodology has been also theorized within the covering law model of explanation (Hempel and Oppenheim 1948).

Three remarks are in order. Firstly, a hypothetico-deductive methodology is employed in case we have at our disposal enough well confirmed theories and background knowledge to formulate a prior causal hypothesis. If this is not the case, exploratory statistical methods provide a useful tool to detect in the data a tentative structure to be further analyzed by means of the structural modelling methodology. Williamson (2005) makes a similar point about the combined used of inductive and hypothetico-deductive methods for causal analysis by means of Bayesian networks.

Secondly, from a logical point of view, a model is false if at least one of its assumptions is false. However, as mentioned above, models are deemed to be useful rather than true. Consequently, also false models can be *useful* depending on the problem at hand. In other words, although the model is not the "true" model, it can more or less faithfully represent reality and thus be useful in order to understand (at least) some aspects of the world.

Thirdly, deduction ought not to be confused with the hypothetico-deductive methodology and induction ought not to be confused with the inductive methodology. In fact, there is a sharp difference between hypothetico-deductive or inductive strategies on the one hand, and deductive or inductive inferences, on the other hand. The former are procedures for testing or for formulating hypotheses, whereas the latter are types of inference. On the one hand, H-D strategies *confirm* (or disconfirm) hypotheses, while inductive strategies are employed to *discover* hypotheses. On the other hand, deduction is a *non-ampliative inference* from what is known to what is known, whereas induction is an *ampliative* inference from what is known to what is *not* known yet.

Structural models discussed above are hypothetico-deductive (H-D) models, for which empirical testing is performed through two stages:

(i) prior theorizing of out-of-sample information, including in particular the selection of variables deemed to be of interest, the formulation of a causal hypothesis (also called the conceptual hypothesis), *etc.*;

(ii) iteratively:

a. building the statistical model;

b. testing the adequacy between the model and the data to accept the empirical validity or non validity of the causal hypothesis.

Causal modelling requires accurate knowledge of the causal context: previous studies, well confirmed scientific theories or background knowledge are essential. The conceptual hypothesis states a hypothesized causal structure, *i.e*. a causal claim about the association between variables to be put forward for empirical testing. However, the evaluation of the conceptual hypothesis cannot be done only *a priori* but also requires empirical testing. Indeed, this is what the whole statistical set-up is built for. Differently put, causality is a matter of confirmation, or borrowing the statistical vocabulary, a matter of accepting or rejecting a given hypothetical model. Therefore, if (i) statistical assumptions are satisfied, (ii) the model fits the data, and (iii) the model is structurally stable, then the proposed causal link is provisionally accepted.

This strategy is hypothetico-deductive because the causal claim is not inferred from the data, as in inductive methods, but confirmed or disconfirmed in the given causal context and relative to the structural model. This is, in particular, at variance from algorithms in TETRAD (Spirtes *et al.* 1993), which are supposed to allow the *inductive inference* of causal relationships from a data set regardless of any prior conceptual framework. Note that H-D strategy so described is general enough to provide a scheme of scientific practice, without commitment to a strict covering law model or to the use of any particular measure of confirmation; on this point see Williamson (2005).

IV. Some remarks on causation at the population and at the individual level

The problem of levels of causation arises because causal conclusions drawn from statistical models concern populations as well as individuals, although probability distributions and their parameters are typically defined relative to the population. Nonetheless, populations *are* made up of individuals. Regardless of how individuals influence group behaviour and *vice versa*, we wonder how to make sense of the following issue. Epidemiological studies establish a causal relationship between smoking and lung cancer. This conclusion is based on data concerning individuals of a particular population. Yet, one might be interested in Harry's chance of getting lung cancer given that he smokes, or in the probability that his smoking caused him to contract lung cancer.

This remark leads us to the fruitful distinction between population-level causation and individual-level causation, although we are left with at least two problems. On the one hand, we face the *methodological* problem of handling the heterogeneity of individual characteristics and, on the other, the practical problem of causal attribution and/or diagnosis for a given individual. It is worth pointing out that to advocate two levels of causation is *not* tantamount to saying that causation operates in a different manner at the two levels.

The methodological problem can be rephrased as follows. The concept of causality that emerges from structural modelling involves two complementary aspects. Firstly, the validity of a statement such as "smoking caused Harry's cancer" depends on the validity of a model deemed to be structural. Secondly, the concept of a structural model is a statistical concept that refers to some population of reference, and a statement such as "smoking causes lung cancer" virtually refers to each individual of a population of reference. Also, the H-D methodology just discussed points to the issue that, as a matter of fact, the structural model on which causality is based is not justified *a priori*, but has to be uncovered by blending field knowledge and statistical methods.

Heterogeneity of individual characteristics, *i.e.* the problem raised by variables that are causal *and* non observable, has the consequence that our models, although structural, are nonetheless imperfect. The complexity of the problem of heterogeneity has led to an extremely vast body of literature. Solving the problem goes far beyond the scope of this work, and we shall be content to mention that it has been tackled and criticized from several

perspectives. For instance, multilevel analysis (see Courgeau 2004) tries to get at an understanding of individual and population behaviours under the assumption that the grouping of individuals according to various levels introduces an influence of the group on its members and *vice versa*. In health sciences, *e.g.* in biostatistics, frailty models are used to model heterogeneity of populations (Vaupel and Yashin, 2001). The counterfactual approach tries to cope with the problem of heterogeneity by setting different hypothetical initial conditions. However, in spite of the intuitive appealing of counterfactual reasoning, the non observability of such different settings has been the object of several criticisms (see Dawid 2001)

Let us now come back to the practical problem. Can a physician decide whether to prescribe a treatment or not on the basis of a causal model? The answer is not straightforward. On the one hand, the answer ought to be positive, were the physician sure of covariate sufficiency. However, this is not a realistic situation. A good physician does not blindly follow what a structural model prescribes. He would also take into account the specific characteristics of his patients.

Nonetheless, this practical problem hides an epistemological one. In fact, the physician's decision depends on the relationships between causality at the population level and at the individual level. The physician's incertitude about the population level causal evidence is due to the methodological difficulties mentioned above: we wonder (i) what the causal variables are and whether it is possible at all to provide a sufficient list, and (ii) what mechanisms operate among the variables deemed to be causal.

In spite of this, what we discover about the average relation between smoking and lung cancer at the population level, can guide causal attribution in the case of Harry through a simple tool of probabilistic reasoning, namely Bayes' theorem. In fact, Bayes' theorem allows us to calculate the posterior probability of the cause for a given individual, provided that the population risk is interpreted as a prior probability for this individual, taking into account his specific characteristics.

Concluding remarks

Statistical models are stochastic representations of the real world. Typically, structural models are conditional statistical models characterized by parameters that are stable over a large class of interventions or of environmental changes. It is *within* these structural models that we formulate causal statements. From a statistical viewpoint, causality can be defined in terms of exogeneity in a structural model. Nonetheless, exogeneity is not enough if we consider causality from an *epistemological* perspective. A more complex and rich concept of causality may be worked out once we consider the fundamental role of assumptions made in structural models, of background and contextual knowledge and of the hypothetico-deductive methodology. These considerations, however, do not enable us to attain a unique and consensual *definition* of causality. But at least this allows us to attain a concept of causality that is *internal* or relative to the structural model itself. This is not to deny the existence of causation. Rather, this is to emphasize that our knowledge of causal relations – at least in the social sciences – depends on structural models that mediate epistemic access to causal relations. In these concluding remarks we shall focus on some epistemological and practical consequences of our approach.

From an epistemological (and also methodological) point of view, it is worth distinguishing (i) the seeking for a basic concept of causality from (ii) the analysis and examination of complex causal structures. Causality is not directly observable but intrinsically latent, as the fact that we cannot infer causation from correlation shows. This means that causality is, above all, a matter of perspective. According to a realist perspective, causality

exists whether or not we can observe it, and the problem turns out to be how to detect *true* causal relations. An empiricist approach is more interested in what we can say about causal relations starting from observations. Our emphasis on exogeneity and structural stability accommodates both approaches. On the one hand, exogeneity is a well-defined concept; on the other hand, structural stability raises interpretational problems because what we decide to test for structural stability depends on background knowledge.

In the social sciences, structural models analyze causal structures that are much more complex than simple causal relations such as *X* causes *Y*. In section III we mentioned some practical difficulties such as covariate sufficiency, no confounding, etc. However, we did not develop a systematic analysis of those issues – this will be the object of another paper.

Some practical implications can be drawn from this paper. Firstly, data should ideally enable the time-ordering of events, as causes precede their effects in time. If this is not possible, e.g. if we have no control over the data collection process, background knowledge can sometimes supply the necessary information on causal priority. For instance, that tobacco consumption causes lung cancer and not the other way around, belongs to our background knowledge and should be used in the time-ordering of events. Secondly, a causal explanation cannot be derived purely from the data themselves. In addition, causal explanations require background knowledge, theoretical assumptions, and a sound methodological approach. Causality is thus bond to structural modelling, and the final test of a causal model remains structural stability accompanied by a sensible interpretation of results. Thirdly, each of the various stages of the modelling endeavour (collecting prior evidence, obtaining data and checking their quality, defining concepts and constructing indicators, developing causal hypotheses, choosing the methods of analysis) should be undertaken very thoroughly: today's causal modelling is indeed the building-block of tomorrow's background knowledge.

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Appendix I: A simple example of exogeneity and of causality

In this appendix we illustrate through a very simple example some basic notions regarding structural modelling and exogeneity. We also take this opportunity to show the progressive nature of model specification.

Let us consider the following economic situation. We start with annual data on the price $p(t)$ and the quantity sold $q(t)$ of a well-defined holiday destination; imagine, for instance, a full-board stay in a three star hotel during the first two weeks of August on a well-known beach. Without further specification, a simple model would consist in assuming that the pair of variables $\left(p(t), q(t)\right)$ is generated by a bivariate normal distribution with expectations $(\mu_{p(t)}, \mu_{q(t)})$, variances $\left(\sigma^2_{p(t)}, \sigma^2_{q(t)}\right)$ and covariance $\sigma_{p(t),q(t)}$. The argument "t" in the parameters indicates that the characteristics of the process change over time: in the statistical jargon the parameters are purely "incidental". Although acceptable from an economic point of view, this parametric instability leads to a non operational model: each new observation $\left(p(t), q(t)\right)$ introduces 5 new parameters and no observation might possibly put the model into question, let alone refute it. We should add more structure, based on contextual rather than empirical knowledge.

Suppose now that the market operates as follows. In January, each year, the tour operator prints a catalogue in which the price $p(t)$ is announced. From January to July the buyers enter their orders, compounding the aggregated quantity $q(t)$ at the announced price $p(t)$. This suggests to decompose the joint distribution, generating $\left(p(t), q(t)\right)$, into a *marginal distribution* generating $p(t)$, with parameters $\left(\mu_{p(t)}, \sigma^2_{p(t)}\right)$ and a *conditional distribution* generating $\left(q(t) \mid p(t)\right)$: $P\left[p(t), q(t)\right] = P\left[p(t)\right] \cdot P\left[q(t) \mid p(t)\right]$.

The motivation for this decomposition is that the economic context suggests that the *marginal process* captures the behaviour of the tour operator (the supply side) where the expectations $\mu_{p(t)}$ expresses, in particular, the (changing) expectations on the evolution of the costs (cost of kerosene for air flight, local cost of accommodation *etc.*) and the variances $\sigma^2_{p(t)}$ reflects the indeterminateness of the behaviour, in particular due to changing levels of uncertainty. Similarly, the economic context suggests that the *conditional process* generating $\left(q(t) \mid p(t)\right)$ captures the consumer behaviour (demand side), taking in particular into account that in our societies once a catalogue is printed the buyer does not try to bargain on the price: (*s*) he either accepts the price, and buys, or rejects the price, and does not buy (and possibly looks for a cheaper offer…) and therefore behaves *"as if"* the price were fixed, *i.e.* not random.

Suppose now that we are only interested in the supply side behaviour. As shown above the parameters $\left(\mu_{p(t)}, \sigma^2_{p(t)}\right)$ cannot be estimated because of their incidental nature. Thus we should add more structure, typically in making these expectations and variances known functions of past observations and of a finite number of parameters (for instance α and β in: $\mu_{p(t)} = \alpha + \beta\, p(t-1)$). By doing so, we aim at capturing a parameter constancy: this is one of the major objectives of statistical modelling.

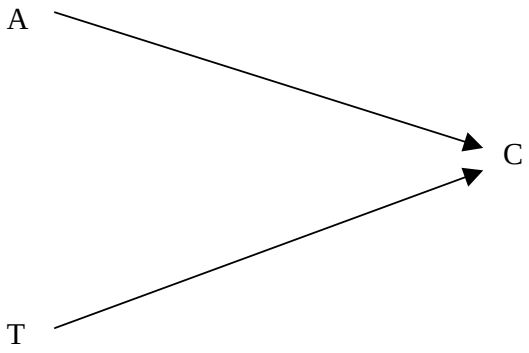
Suppose now that we are only interested in the demand side. Again for the same reason as before we should endow the conditional expectation $E\left[q(t)\,|\,p(t)\right]$ and the conditional variance $V\left[q(t)\,|\,p(t)\right]$ with some structure. For the sake of simplicity, let us assume that $E\left[q(t)\,|\,p(t)\right]=\gamma+\delta p(t)$ and $V\left[q(t)\,|\,p(t)\right]=\sigma^2$, which is independent of t (an homoscedasticity assumption). Apart from the so-introduced structural stability, we should now face the issue of exogeneity; this is the question whether it is "admissible" (*i.e.* without loss of information) to consider only the conditional model, *i.e.* to treat the price $p(t)$ *"as if"* the price was not random. The exogeneity of the price involves two different aspects. Firstly, do we grant that the conditional distribution actually captures the behaviour we are interested in? Thus, do we grant that the demand really behaves under "given" price, that only the (current) price is relevant for the buying behaviour, that the conditional variance really is constant etc?

Quite a different question is the following: do we accept that the randomness of the price gives no information on the parameters of the conditional model? In other words, do we accept that the unknown value of the parameters of the conditional model is independent of the values of the parameters of the marginal model? If so, the statistical jargon would say that the parameters of the marginal models and the parameters of the conditional model are "variation-free", in a sampling theory approach, or are "a priori independent" in a Bayesian approach. Thus the exogeneity problem involves two aspects. The first one regards the parameters of interest: are we really - *i.e.* contextually - interested in the parameters of the conditional model? The second aspect regards the statistical efficiency: may we ignore the random character of the conditioning variable?

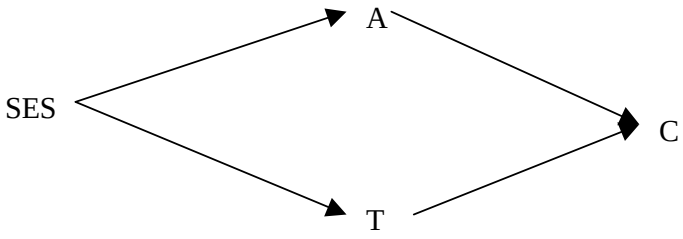
Does the exogeneity of the price in the conditional model generating $\left(q(t)\,|\,p(t)\right)$ imply that "the price causes the quantity"? The answer is "yes" in quite a specific sense, *i.e.* under several provisos. Firstly, it is a concept of causality internal to a specific model: the price $p(t)$ "causes" the quantity in a particular model which is assumed to represent a demand behaviour but the price does not cause the quantity "in general". Secondly, this concept of causality is relative to a particular family of models, namely models that are both conditional and structural: the price causes the quantity because the conditional model, generating $\left(q(t)\,|\,p(t)\right)$ is assumedly structural. In other words, to the best of the scientist's knowledge, analyzing the data on the price alone, by means of a marginal model generating $p(t)$ only, would give no information about the characteristics of the conditional distribution *and* this conditional distribution is by assumption stable under a large class of interventions.

Appendix II: The epidemiology of lung cancer

The following example is taken from the field of epidemiology. It is well known that two major determinants of lung cancer are smoking and exposure to asbestos dust (American Cancer Society, http://www.cancer.org, accessed on November 23, 2005). A case-control study, for example, can indeed show that those having lung cancer have smoked more and/or have been more exposed to asbestos dust on average than those who do not have lung cancer. There are furthermore two types of lung cancer which should be distinguished in an epidemiological perspective, *small cell lung cancer* and *non-small cell lung cancer*, but this distinction has not been taken into account here. A first simple model would therefore consider tabagism (T) and asbestos exposure (A) as two independent causes of lung cancer (C), leading to the following causal graph



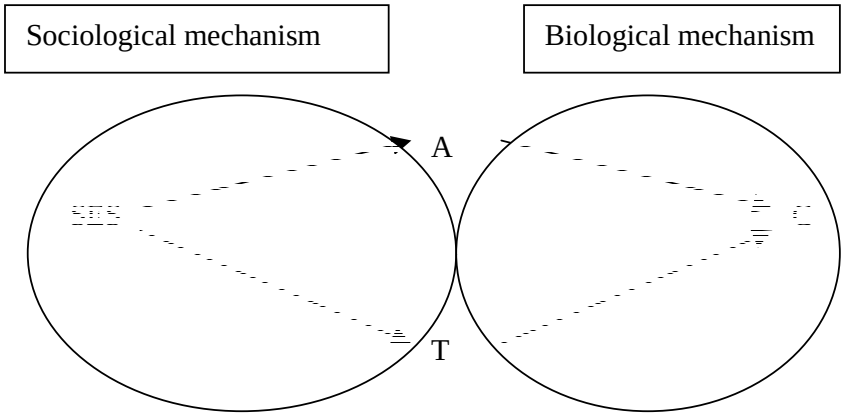
One also knows however that both smoking and asbestos exposure are dependent upon one's socio-economic status (SES): those with a lower SES tend more to smoke and work in unhealthy environments than those with a high SES. The causal graph can therefore be redrawn as follows:



This graph shows that tabagism and asbestos exposure are in fact not independent from one another as they are both related to one's SES, *i.e.* they have a common cause. It is also known that other risk factors - such as radon, occupational chemicals, and environmental tobacco smoke – also play a role on the incidence of lung cancer. In the present simplified

model, we have neglected these other possible causes, though a more complex model could take them into account too. For example, there could be another path from SES to C through exposure to occupational chemicals. We also do not consider in this simplified model the possible interaction effect of smoking and asbestos on lung cancer (this could possibly be represented by adding a node "$T*A$" linked with an arrow to the node "C"). Finally, we neglect the time lag between exposure to smoking and asbestos, and the development of lung cancer. In more realistic epidemiological models, exposure duration would moreover be taken into account if the data are available.

From a structural modelling point of view, two fundamentally different mechanisms should however be distinguished: one is a *biological* mechanism leading from exposure (to tabagism, to asbestos,…) to lung cancer, and another one is a *sociological* mechanism by which different social categories differ in smoking behaviour and occupation. As the two mechanisms are quite different from one another, the global model should therefore be decomposed into two sub-models, a structural model relating firstly SES to smoking and asbestos exposure, and a second structural model relating these latter variables to lung cancer incidence, as is depicted in the next figure. In the first structural model, SES would be the exogenous variable and tabagism and asbestos exposure the endogenous variables, and in the second structural model both tabagism and asbestos exposure would be the exogenous variables while lung cancer incidence would be the endogenous variable. A demographer, as a social scientist, would focus mainly on the first sub-model while an epidemiologist, with a public health background, would deal more with the second. These considerations point out the fact that the concept of exogeneity is always relative to one's structural model; as is the case here, the same variables can be endogenous in one model and exogenous in another model.



The model can now be formalised as follows. For the sake of simplicity, suppose that our data base is made of 4 variables only, namely: SES, T, A and C. The model just sketched corresponds to decomposing the joint distribution of these variables as follows:

$$p(SES, T, A, C) = p_1(SES) \cdot p_2(T, A \mid SES) \cdot p_3(C \mid T, A)$$

One should notice the following issues:

(i) the three components $p_1(SES)$, $p_2(T, A \mid SES)$ and $p_3(C / T, A)$ are deemed to represent three different structural mechanisms (therefore assumedly structurally stable);

(ii) from what has been said, the first mechanism $p_1(SES)$ is not investigated and left unspecified;

(iii) the third mechanism $p_3(C \mid T, A)$ incorporates the assumption that T and A are sufficient, *i.e.* that the distribution of C given T, A and SES does not depend on SES : $C \perp SES \mid T, A$.

(iv) the second mechanism $p_2(T, A / SES)$ reveals an issue of simultaneity: the mechanism generating T and A given SES is not decomposed into two univariate mechanisms, such as for instance $p_{2.1}(T \mid SES)$ and $p_{2.2}(A \mid SES, T)$; the reason why that decomposition has not been operated is outside the scope of this paper but one possible reason might be that interest focuses on the third mechanism and that similarly to the first one the specification of the second one is only a partial one;

(v) the structure of this model suggests that the association between SES and C, as possibly illustrated as :

SES ⟶ C

may be statistically significant but contextually non interpretable, unless as a composition of associations rooted in different mechanisms; that is to say, from a philosophical point of view, the above figure does not make any ontological sense, for, strictly speaking, SES does not *cause* cancer. Therefore, in an intervention analysis, an action simply on SES is not likely to decrease the prevalence of lung cancer: intervention should be targeted either on the sociological, or on the biological, mechanism.

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