Chapter 4
Structural Modelling, Exogeneity, and Causality

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4.1 Causal Analysis in the Social Sciences

4.1.1 Goals of Causal Analysis

Whilst it might seem uncontroversial that the health sciences search for causes – that is, for causes of disease and for effective treatments – the causal perspective is less obvious in social science research, perhaps because it is apparently harder to glean general laws in the social sciences than in other sciences, due the probabilistic character of human behaviour. Thus the search for causes in the social sciences is often perceived to be a vain enterprise and it is often thought that social studies merely *describe* the phenomena.

On the one hand, an explicit causal perspective can already be found in pioneering works of Adolphe Quetelet (1869) and Emile Durkheim (1897) in demography and sociology respectively, and the social sciences have taken a significant step in *quantitative causal analysis* by following Sewall Wright's path analysis (1934), first applied in population genetics. Subsequent developments of path analysis – such as structural models, covariance structure models or multilevel analysis – have the merit of making the concept of cause operational by introducing causal relations into the framework of statistical modelling. However, these developments in causal modelling leave a number of issues at stake, for instance a deeper understanding of exogeneity and its causal importance.

On the other hand, an explicit causalist perspective still needs justification. Different social sciences study society and humans from different angles and perspectives. Sociology studies the structure and development of human society, demography attends to the vital statistics of populations, economics studies the management of goods and services, epidemiology studies the distribution of disease in human populations and the factors determining that distribution, etc. In spite of

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these differences, social sciences share a common objective: to understand, predict and intervene on individuals and society. In these three moments of the scientific demarche, knowledge of causes becomes essential. The importance of causal knowledge is twofold. Firstly, we pursue a *cognitive goal* in detecting causes and thus in gaining general knowledge of the causal mechanisms that govern the development of society. Secondly, such general causal knowledge is meant to guide and inform social policies, that is we also pursue an *action-oriented goal*. If the social sciences merely *described* phenomena, it would not be possible to design efficient policies or prescribe treatments that rely on the results of research.

As stated above, the social sciences do not establish laws as physics does. Whether this is an intrinsic issue of these sciences, or merely a contingent issue due to the specifity of social problems, is still matter of debate and falls far beyond the scope of the present paper. In the following, we will rather reverse the perspective and try to tackle the issue: under what conditions can structural models give us causal knowledge?

4.1.2 Variation and Regularity in Causal Analysis

The first thing worth mentioning is that we need to abandon the paradigm of regularity as regular succession of events in time, a heritage of Hume, in favour of a more flexible framework. Hume believed that causality lies in the constant conjunction of causes and effects. In his *Treatise* Hume (1748) says that, in spite of the impossibility of providing rational foundations for the existence of objects, space, or causal relations, believing in the existence of causal relations is a "built in" habit of human nature. In particular, belief in causal relations is granted by experience. For Hume, simple impressions always precede simple ideas in our mind, and by introspective experience we also know that simple impressions are always associated with simple ideas. Simple ideas are then combined in order to form complex ideas. This is possible thanks to imagination, which is a normative principle that allows us to order complex ideas according to (i) resemblance, (ii) contiguity in space and time, and (iii) causality. Of the three, causation is the only principle that takes us beyond the evidence of our memory and senses. It establishes a link or connection between past and present experiences with events that we predict or explain, so that all reasoning concerning matters of fact seems to be founded on the relation of cause and effect. The causal connection is thus part of a principle of association that operates in our mind. Regular successions of impressions are followed by regular successions of simple ideas, and then imagination orders and conceptualizes successions of simple ideas into complex ideas, thus giving birth to causal relations. The famed problem is that regular successions so established by experience clearly lack the logical necessity we would require for causal successions. Hume's solution is that if causal relations cannot be established a priori, then they must be grounded in our experience, in particular, in our *psychological habit* of witnessing effects that regularly follow causes in time and space.

If we want causality to be an empirical and testable matter rather than a psychological one, we need to replace the Humean paradigm of regularity with a paradigm of *variation*. In this framework structural models do not only aim at finding regular successions of events. Rather, causal models *model* causal relations by analysing suitable *variations* among variables of interest (see Russo 2006, 2008). Differently put, causal models are governed by a *rationale of variation*, not of regularity. A rationale is a principle of some opinion, action, hypothesis, phenomenon, model, reasoning, or the like. The quest for a rationale of causality is then the search for the principle that guides causal reasoning and thanks to which we can draw causal conclusions. This principle lies in the notion of *variation*.

The rationale of variation manifestly emerges, for instance, in the basic idea of probabilistic theories of causality and in the interpretation of structural equations. Probabilistic theories of causality, see Suppes (1970), focus on the difference between the conditional probability $P(E\,|C\,)$ and the marginal probability $P(E)$. To compare conditional and marginal probability means to analyse a statistical relevance relation, i.e. probabilistic independence. The underlying idea is that if C is a cause of E, then C must be statistically relevant for E. Hence, the *variation* hereby produced by C in the effect E will be detected because the conditional and the marginal probability differ. Analogously, quantitative probabilistic theories focus on the difference between the conditional distribution $P(Y \leq y\,|X \leq x\,)$ and the marginal distribution $P(Y \leq y)$. Again, to compare conditional distribution with marginal distribution means to measure the variation produced by the putative cause X on the putative effect Y.

In structural equation models, the basic idea is that, given a system of equations, we can test whether variables are interrelated through a set of linear relationships, by examining the *variances* and *covariances* of variables. Sewall Wright, as early as 1934, has taught us to write the covariance of any pair of observed variables in terms of path coefficients. The path coefficient quantifies the (direct) causal effect of X on Y; given the numerical value of the path coefficient β, the equation $Y = \beta X + \varepsilon$ claims that a unit increase in X would result in a β unit increase of Y. In other words, β quantifies the *variation* of Y associated to a *variation* of X, provided that X doesn't have null variance. Another way to put it is that structural equations attempt to quantify the change in X that accompanies a unit change in Y. It is worth noting that the equality sign in structural equations does not state an algebraic equivalence. Jointly with the associated graph, the structural equation is meant to uncover a causal structure. That is, given a structural equation of the simple form $Y = \beta X + \varepsilon_1$, the reverse equation $X = \gamma Y + \varepsilon_2$ is not causally equivalent. Pearl (2000, pp. 159–160) makes a similar point.

4.1.3 Background Knowledge in Causal Analysis

Variation, however, is not itself a causal notion and consequently cannot guarantee, alone, the causal interpretations of probabilistic inequalities. Good epistemology ought to tell us under what conditions, i.e. what the constraints are, for variations

to be causal. A complete account of the guarantee of the causal interpretation should focus on the difference between purely associational models and causal models, pointing to the features proper to the richer apparatus of causal models (see Russo 2008; Russo 2006). For a model to be causal, we shall particularly focus on two types of constraints: background knowledge and structural stability. In a nutshell, concomitant variations will be deemed causal if they are structurally stable and if they are congruent with background knowledge; see also e.g. Engle et al. (1983), Florens and Mouchart (1985), Hendry and Richard (1983) or Thomas (1996). In this way regularity, which would be better understood here in terms of invariance of the model's structure (variables and relations), becomes a constraint that participates in the causal interpretation of variations.

On the one hand, background knowledge, both theoretical and empirical, serves three roles: (i) it provides a relevant causal context for the formulation of hypotheses, (ii) it guides the choice of variables and of the relations to be tested for structural stability, and (iii) it constitutes the sounding board for results as they have to be congruent with background knowledge. On the other hand, structural stability is a constraint we impose on a relation for being causal, in order to rule out accidental relations. Differently put, the crucial step in Hume's argument is significantly different from the rationale hereby proposed. We claim that we firstly look for variations. Once concomitant variations are detected, a condition of invariance or structural stability (among others) is imposed on them. What does structural stability give us? Not logical necessity, nor mere constant conjunction as Hume advocated. Invariance, which is an empirical feature, recalls Humean regularity but the scope of the former is wider than that of the latter. Structural stability is a condition required in order to ensure that the model correctly specifies the data generating process and that the model does not confuse accidental and/or spurious relations with causal ones. It is worth noting that, in the search for structurality, background knowledge and invariance play a complementary role. In particular, unexplained stable relations may lead to questioning background knowledge and eventually to modifying it.

It might be objected that if structural stability does not give us logical necessity either, it does not any better than regularity. Undoubtedly necessity is an essential feature for those who would like the social sciences to discover universal laws, or for those who question their scientific legitimacy on this ground. However, independently of whether it is a built-in impossibility of the social sciences to glean *laws*, this would be a too rigid framework, for society and individuals are too mutable objects of study to be fettered in immutable and even regular deterministic or probabilistic *laws*.

The philosophical gain of adopting this paradigm is twofold. Firstly, we go beyond the Humean tradition that somehow denies causation by reducing it to regularity. Secondly, we do not fall into untestable metaphysical positions either, because structural models stay at the level of *knowledge*. Let us clarify this last point. Structural modelling intends to represent an underlying causal structure, mathematically, by means of equations, and pictorially, by means of directed acyclic graphs. However, structural models don't pretend to attain the ontic level, i.e. to open the black box, so to speak. They stay at the level of field knowledge and theory: if concomitant

variations between, say, X and Y are structurally stable and are congruent with available field knowledge, then we have no reasons not to *believe* that X causes Y. In this sense structural models mediate epistemic access to causal relations without claiming that the *true* causes have been discovered. Differently put, structural modelling allows us to take a sensible causalist stance that guides actions and policies without overflowing into untestable metaphysical claims.

The practical gain of adopting this paradigm is having a clearer understanding of the causal import of background knowledge and of testing stability. Those aspects, in fact, turn out to be of fundamental importance for the interpretation of results.

4.1.4 Probabilistic Modelling in Causal Analysis

Structural models belong to the category of *probabilistic* models. This leads us to consider also the following issue. Is a *probabilistic* characterization of causation a symptom of indeterministic causality or rather of our incomplete and uncertain knowledge? In physics, quantum mechanics raised quite substantial issues about the possibility of indeterminism. However, *whether or not* the world is actually indeterministic, needs not to be decided once and for all. In fact, from an epistemological viewpoint, a probabilistic characterization of causal relations in structural models only commits us to state that our knowledge is incomplete and uncertain. Our endeavour to gain causal knowledge requires reducing, as far as possible, bias and confounding by building good structural models, that is models that pick up structurally stable relations consistent with background knowledge.

So far we have seen that the concept of variation plays a crucial role in the interpretation of structural equations. A simple form of a structural equation such as $Y = \beta X + \varepsilon$, can be interpreted as follows: variations in X lead to or are responsible for variations in Y. In other words, X is statistically relevant for Y, i.e. $P(Y|X) \neq P(Y)$. However, statistical relevance, and consequently also variation, are symmetrical notions. So how do we know that X causes Y and not the other way around? There are three different but nonetheless related elements that participate in determining the direction of the causal relations: background knowledge, invariance, and time. Let us focus on time. In the social sciences we need temporal direction. This is for several reasons.

Firstly, causal mechanisms – be they physiological, social or socio-physiological – are embedded in time. Smoking at time t causes cancer at t' ($t < t'$), but not the other way around. To give another example, use of contraceptives is followed by changes in the intensity and tempo of fertility. Secondly, although the two causal relations *marriage dissolution influences migration* and *migration influences marriage dissolution* both make sense, we need to know whether marriage dissolution or migration is the temporally prior cause for cognitive and/or policy reasons. One out of the two claims might be eventually disproved due to problems of observability or lack of theory. For instance, the causal chain *migration influences*

marriage dissolution might be incorrect: although marriage dissolution is observed *after* migration, there might exist a temporally prior process – marital problems and the subsequent decision to divorce – causing migration.

This oversimplified example clearly shows that causal modelling requires a constant interplay between observation, theory and testing. Indeed, this is the core of a hypothetico-deductive methodology of structural modelling (see Russo 2008; Russo 2006). Causal hypotheses need to be confirmed or disconfirmed (i.e. accepted or rejected in the statistical jargon) based on empirical testing: the model has to fit observations, but the causal hypothesis itself has to be formulated, along with the model building stage, in accordance with available well established theories and background knowledge. However, we also need structural models to be flexible enough to revise our theories in the light of new data disconfirming prior theories.

Following the H-D methodology, causal hypotheses are confirmed or disconfirmed depending on the results of empirical testing. Suppose, for the sake of the argument, that the causal hypothesis is rejected. Such a negative result can be nonetheless useful as it can suggest that improvement is needed in the theory backing the causal model, or that data may contain some source of bias. In other words, the rejection of a causal hypothesis can trigger further research. Suppose now, again for the sake of the argument, that the causal hypothesis is accepted. Such a positive result is not an immutable one, written on the stone, so to speak. Although the causal hypothesis is not rejected, this may be subject to revision (and even to rejection) in the future, due to new discoveries. It is worth stressing that the acceptance of the causal model is highly dependent on its structural stability. Unlike the traditional falsificationist account (see Popper 1959), hypothetico-deductivism in structural modelling allows and indeed encourages us to use at any stage of research all available information. Williamson (2005) also makes a similar point in putting forward a hybrid of inductive and hypothetico-deductive methodologies in which the hypothesising stage is always informed by previous results, whether positive or negative. This is indeed the advantage of handling structural models that are assumed to represent underlying causal structures without pretending to uncover immutable metaphysical causes. The following sections make more explicit and formal these ideas about causality and structural modelling.

4.2 Structural Modelling

4.2.1 The Meaning of Structurality

Inspired by the seminal works of Wright, Haavelmo, Blalock, Pearl and others, we will develop in this section a structural modelling approach to causation. In essence, a model is deemed structural if it uncovers a structure underlying the data generating process. As discussed in Section 4.1.3, this approach systematically blends two ingredients. First, the model must be congruent with background knowledge: modelling the data generating process must be operated in the light of the current

information on the relevant field. Second, the model must show stability in a wide sense: both the structure of the model and the parameters have to be stable or invariant with respect to a large class of interventions or of modifications of the environment. Often, but not always, structural models make use of latent variables. By integrating out the latent variables, the statistical model is thus obtained as the marginal distribution of the manifest or observable variables. It is crucial to note that this concept of structural modelling is wider than the framework of structural equations models, also known as covariance structure models or LISREL type models, widely used in psychology or in sociology, and of simultaneous equations models, widely used in econometrics.

A first consequence of this approach is that the notion of causality becomes relative to the model itself, rather than to the data, as is the case, for instance, in the Granger-type concept of causality. Also, this means that we do not aim at making metaphysical claims about causal relations, but rather at saying when we have enough reasons – specifically, reasons about background knowledge and about structural stability – to believe that we hit upon a causal relation. A second consequence of this model-based concept of causality, involving both background knowledge and stability, is that the model does not simply derive from theory as is often the case in the econometric tradition.

Therefore structural modelling is much more than a sophisticated statistical tool. Good structural modelling ought to be accompanied by a broad and sensible account of what a statistical model is and represents, of what statistical inference is, and of what rationale guides model building and testing. The last point has been dealt with in the previous section. The first and the second will be the object of the following sections. We first recall the formal nature of a statistical model and of the basic concepts of conditional modelling and of exogeneity, we then define the concept of causality in such a framework.

4.2.2 The Statistical Model

Formally, a statistical model $\mathbf{M}$ is a set of probability distributions, explicitly:

$$\mathbf{M} = \{S, P^{\omega}: \ \omega \in \Omega\} \tag{4.1}$$

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 $\omega \in \Omega$, P^{ω} is a probability distribution on the sample space, also called the sampling distribution; thus, ω is a characteristic, also called parameter, of the corresponding distribution and Ω 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: $\omega = (\mu, \sigma^2)$.

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. 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).

If 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 an assumption is true. A (frequentist) statistician may however want to test in due course whether a hypothesis is confirmed or not. 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.

4.2.3 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 corresponds to a stochastic representation of the world and is a vehicle for the learning-by-observing.

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 models, also called associational or descriptive models, and exploratory data analysis, also called 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 a causal relation and leaves open a wide scope for interpreting the meaning of the results.

The second family consists in the so-called structural models. "Structural" conveys the idea of a representation of the world that is stable under a large class of interventions or of modifications of the environment. 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. 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 aims at capturing an underlying structure; modelling this underlying structure requires taking into account the contextual knowledge of the field of application. The characteristics, or parameters, of a structural model are of interest because they correspond to relevant 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 associations without linking those associations to stable properties of the world.

The invariance condition of a structural model is actually a complex issue. Two aspects have to be considered. A first one is a condition of stability of the causal relation. The idea is that each variable depends upon a set of other variables through a relationship that remains invariant when those other variables are subject to external influence. This condition allows us to predict the effects of changes in the environment or of interventions. A second condition is the stability of the distributions to ensure that the parameters will not be affected by changes in the environment or interventions.

4.3 Conditional Models, Exogeneity and Causality

4.3.1 Conditional Models

Originally, the concept of exogeneity appears with regression models. A first, and naive, approach was to consider an exogenous variable as a non-random variable, the endogenous variable being the only random one. That this approach was unsatisfactory became clear when considering complex models where the same variable could be exogenous in one equation and endogenous in another one. A first progress came through a proper recognition of the nature of a conditional model. Here, we

present a heuristic account of the basic concepts; for a more formal presentation, see Mouchart and Oulhaj (2000) and Oulhaj and Mouchart (2003).

Let us start with *an (unconditional) parameterized* statistical model $\mathbf{M}_{\mathbf{X}}^{\omega}$ given in the following form:

$$\mathbf{M}_{\mathbf{X}}^{\omega} = \{p_X(x\,|\omega) : \omega \in \Omega\} \tag{4.2}$$

where for each $\omega \in \Omega$, $p_X(x\,|\omega)$ is a (sampling) probability density on an underlying sample space corresponding to a (well-defined) random variable X and Ω is the parameter space, aimed at describing the set of sampling distributions considered to be of interest. A conditional model is constructed through embedding this concept into the usual concept of an unconditional statistical model (4.2). For expository purposes, this paper only considers the case where a random vector X of observations is decomposed into $X' = (Y', Z')$ (where $'$ denotes transposition) and the model is conditional on Z.

The basic idea of a conditional model is the following: starting from a global model $\mathbf{M}_{\mathbf{X}}^{\omega}$ as given in (4.2), each sampling density $p_X(x\,|\omega)$ is first decomposed through a marginal-conditional product:

$$p_X(x\,|\omega) = p_Z(z\,|\phi)\, p_{Y|Z}(y\,|z,\theta) \qquad \omega = (\phi, \theta) \tag{4.3}$$

where $p_Z(z\,|\phi)$ is the marginal density of Z, parametrized by ϕ, and $p_{Y|Z}(y\,|z,\theta)$ is the conditional density of $(Y\,|Z)$, parametrized by θ. Next, one makes specific assumptions on the conditional component leaving virtually unspecified the marginal component. Thus a conditional model may be represented as follows:

$$\mathbf{M}_{\mathbf{Y}}^{\mathbf{Z},\theta;\Phi} = \left\{p_X(x\,|\omega) = p_Z(z\,|\phi)\, p_{Y|Z}(y\,|z,\theta) \qquad \omega{=}(\theta,\phi) \in \Omega = \Theta \times \Phi\right\} \tag{4.4}$$

where Φ parametrizes a typically large family of sampling probabilities on Z only and for each $\theta \in \Theta$, $p_{Y|Z}(y\,|z,\theta)$ represents a conditional density of $(Y\,|Z)$. The essential features of a conditional model are therefore:

1. θ indexes a well specified family of conditional distributions. This family constitutes the kernel of the concept of a conditional model. The concept of conditional model relates, however, to a family of joint distributions $p_X(x\,|\omega)$ obtained by crossing the family of conditional densities $p_{Y|Z}(y\,|z,\theta)$ with a family of marginal distributions $p_Z(z\,|\phi)$.
2. ϕ is a nuisance parameter which is identified by definition (because Φ is a set of distributions of Z). Furthermore θ and ϕ are variation free. The notation $\mathbf{M}_{\mathbf{Y}}^{\mathbf{Z},\theta;\Phi}$ conveys the idea that θ is the only parameter of actual interest, leaving to ϕ no explicit role.
3. The modelling restrictions are concentrated on the conditional component, i.e. the set $P_Y^{Z,\theta} : \theta \in \Theta$ embodies the main hypotheses of the model, whereas in most cases, the set Φ embodies a minimal amount of restrictions, typically only the hypotheses necessary to guarantee essential properties for the inference on

θ, such as identifiability or convergence of estimators. For instance, in a linear regression model, suitable asymptotic properties of the Ordinary Least Squares estimators require conditions such as stationarity or ergodicity of the process generating the explanatory variables. Consequently, in most situations, but not in all, Φ represents a "thick" subset of the set of all probability distributions of Z. The role of Φ is to stress the random character of Z at the same time as the vague specification of its data generating process; Φ may nevertheless play an important role because its specification may determine desirable properties of the estimators of θ, the parameter of interest. Oulhaj and Mouchart (2003) provides more information on conditional models.

Let us give an example. Consider four variables: tabacism (T), cancer of the respiratory system (C), asbestos exposure (A) and socio-economic status (SES). A global (unconditional) model would consider a family of distributions on the four variables (T, C, A, SES) parametrized by, say, ω, as in (4.2). A conditional modelling approach would run as follows. Suppose we are interested in the impact of T, A and SES on C. Attention would therefore focus on a particular component of the global model, namely the conditional distribution of C given T, A and SES, leaving the marginal distribution of T, A and SES with a minimum amount of specification. In other words, for each distribution indexed by ω in the global model (4.2), we have in mind a marginal-conditional decomposition as in (4.3):

$$p_{C,T,A,SES}(c, t, a, ses\,|\omega) = p_{C|T,A,SES}(c\,|t, a, ses, \theta)\, p_{T,A,SES}(t, a, ses\,|\phi)$$
$$\omega = (\phi, \theta) \tag{4.5}$$

The basic idea of the conditional model, as in (4.4), is to endow the global model (4.2) with two properties. Firstly, the parameters characterizing the marginal (ϕ) and the conditional (θ) components are independent. Here, "independence" means "variation-free" in a sampling theory framework, i.e. $\omega = (\theta, \phi) \in \Omega = \Theta \times \Phi$, or independent in the (prior) probability in a Bayesian framework, i.e. $\phi \perp\!\!\!\perp \theta$ in Bayesian terms. Secondly the marginal component is left almost unspecified, i.e. the set Φ represents a "very large" set of possible distributions for (T, A, SES).

4.3.2 Conditional Model and Exogeneity

Suppose we analyze data set $X = (Y, Z)$. A challenging issue is to decide whether it is admissible, in the sense of losing no relevant information, to only specify a conditional model $\mathbf{M}_{\mathbf{Y}}^{\mathbf{Z},\theta;\Phi}$ rather than specifying the model $\mathbf{M}_{\mathbf{X}}^{\omega}$. This is the issue of exogeneity.

The motivation for specifying a conditional model rather than a model on the complete data set X is parsimony: some specifications on the marginal process may not be avoided for ensuring suitable properties of the inference on the parameters of the conditional process but by specifying less stringently the marginal process,

generating Z, one looks for protection against specification error. The cost could however be substantial if the marginal process generating Z contains relevant information, an example of which is given in Section 4.5.1.

Formally, the condition of exogeneity is therefore: the parameter of interest should only depend on the parameters identified by the conditional model and the parameters identified by the marginal process should be "independent" of the parameters identified by the conditional process. It should be stressed that the independence among parameters has no bearing on a (sampling) independence among the corresponding variables.

In order to make the argument more transparent, we slightly modify the notation. In Section 4.3.1 we constructed a model on the X-space, where $X = (Y', Z')'$, by crossing a family of distributions on Z, indexed by ϕ, and a family of conditional distributions on $(Y \,|Z)$, indexed by θ, and eventually obtained a joint model, parametrized by $\omega = (\phi, \theta)$. We now start from a joint model on X, parametrized by ω, and deduce from the decomposition (4.3) the parameters characterizing the family of marginal distributions of Z, denoted by θ_Z, and the parameters characterizing the family of conditional distributions of $(Y \,|Z)$, denoted by $\theta_{Y|Z}$. Equation (4.3) is accordingly rewritten as follows:

$$p_X(x \,|\omega) = p_Z(z \,|\theta_Z)\, p_{Y|Z}\left(y \,\middle|\, z, \theta_{Y|Z}\right) \tag{4.6}$$

where θ_Z, respectively $\theta_{Y|Z}$, represents the parameter identified by the marginal, respectively conditional, process. The condition of independence, namely:

$$\left(\theta_Z, \theta_{Y|Z}\right) \in \Theta_Z \times \Theta_{Y|Z} \quad \text{or} \quad \theta_Z \perp\!\!\!\perp \theta_{Y|Z} \tag{4.7}$$

is a *condition of (Bayesian) cut* (see Barndorff-Nielsen (1978) in a sampling theory framework, and Florens et al. (1990) in a Bayesian framework), and is deemed to allow for a separation between the inference on the parameters of the marginal process and the inference on the parameters of the conditional process. More explicitly, condition (4.7) implies that any inference on θ_Z, respectively, $\theta_{Y|Z}$, be based only on the marginal, respectively conditional, model characterized by the marginal distributions $p_Z(z \,|\theta_Z)$, respectively conditional distributions $p_{Y|Z}(y \,|z, \theta_{Y|Z})$.

This condition, along with the condition that the parameter of interest, say λ, depends only on the parameters identified by the conditional process, i.e. $\lambda = f(\theta_{Y|Z})$, formalizes the concept of "losing no relevant information" when basing the inference on the conditional model rather than on the complete model, characterized by the distributions $p_X(x \,|\omega)$. In this setting, the concept of exogeneity appears as a binary relation between a function of the data, namely Z, and a function of the parameters, namely λ. Thus, Florens et al. (1990) suggests the expression "Z and λ are mutually exogenous" (or Z is exogenous for λ), to stress the idea that a variable is not exogenous by itself but is exogenous in a particular inference problem. Treating Z as exogenous means therefore that the (marginal) process generating Z is minimally specified (and may be heuristically qualified as "left unspecified") *and* that

the inference on the parameter of interest, although based on the joint distribution of all the variables in X, is nevertheless invariant with respect to any specific choice of the marginal distribution of Z. Summarizing: exogeneity is the condition that makes admissible the use of the conditional model as a reduction of the complete model.

The consequences of a failure of exogeneity may be twofold. There may be a loss of efficiency in the inference if the failure comes from a restriction (equality or inequality), or a lack of independence in a Bayesian framework, between the parameters of the marginal model and those of the conditional model. There may also be an impossibility of finding a suitable, e.g. unbiased or consistent, estimator if the parameter of interest is not a function of $\theta_{Y|Z}$ only. A typical example, well known in the field of simultaneous equations in econometrics, is that the parameter of interest in a structural equation may not be a function of the parameters identified by the conditional model corresponding to a specific equation.

4.3.3 Exogeneity and Causality

In general, the specification of a parameter of interest is a contextual rather than a statistical issue. A most usual rationale for specifying the parameter of interest is based on the notion of a *structural model*. In this framework, Russo (2006) approach causality as *exogeneity in a structural conditional model*. In the very simple case of two variables Y and Z, this concept may be paraphrased as follows: if the conditional distribution of Y given Z is structurally stable and reflects a good scientific knowledge of the field, there is no reason not to believe that Z causes Y. This approach might be considered empirical because the observations providing the ground for a causal interpretation are not only the data under immediate scrutiny but also the whole body of observations underlying the "field knowledge" and leading accordingly to the present state of scientific knowledge. In this sense, causal attribution "Z causes Y" is an issue of structural modelling, namely this is the question whether the conditional model characterized by $p_{Y|Z}(y \,|z, \theta_{Y|Z})$ is actually *structural*.

4.4 Confounding, Complex Systems and Completely Recursive Systems

4.4.1 Confounders and Confounding

In many circumstances, the same effect can be produced by several causes or the same cause can produce several effects. We may however focus our interest on a particular cause, say X and a particular effect, say Y. In this case, the causal relation $X \rightarrow Y$ can be subject to confounding. In epidemiology and in demography, for example, when one examines the impact of a treatment/exposure on a response/outcome, a confounding variable – or confounder – is often defined as a

variable associated both with the putative cause and with its effect, see e.g. Jenicek and Cléroux (1982), Elwood (1988). Sometimes the definition is more precise, such as in Anderson et al. (1980) or in Leridon and Toulemon (1997). According to these authors, a variable is a confounder whenever two conditions simultaneously hold:

1. The risk groups differ on this variable;
2. The variable itself influences the outcome.

Some authors gloss condition 1 adding that the variable, as a background factor, should not be a consequence of the putative cause, see e.g. Schlesselman (1982).

For instance, if we examine the impact of cigarette smoking on the incidence of cancer of the respiratory system, a variable such as exposure to asbestos dust confounds the relation between smoking and this type of cancer. Indeed, exposure to asbestos dust and smoking are associated, i.e. proportionally there are more persons exposed to asbestos in the smoking group than in the non-smoking group. Condition 1 is therefore satisfied. In addition, inhalation of asbestos dust is a strong cause of cancer of the pleura; condition 2 is thus also satisfied. Cancer is the outcome variable in this example, smoking a potential cause, and exposure to asbestos a confounder. Vice-versa if one were to examine the impact of asbestos exposure on the incidence of cancer of the respiratory system, smoking this time would be the confounding factor, as it is associated with asbestos exposure and is a cause of lung cancer. This simplified example is discussed in Russo (2006) but a real study would also consider other causal factors and paths, and the synergy between smoking and asbestos exposure.

Condition 1 needs to be clarified however; on this subject, see also McNamee (2003). Why are smoking and asbestos exposure associated? In demography and in epidemiology, one knows 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 higher *SES*. The causal graph can therefore be drawn as in Fig. 4.1, where A represents exposure to asbestos, T tabacism, and C cancer incidence. It is worth noting that Fig. 4.1 incorporates two assumptions, namely: $A \perp\!\!\!\perp T \,|\, SES$ and $C \perp\!\!\!\perp SES \,|\, A, T$.

This graph shows that tabacism 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. Note that *SES* is also a common cause of T and C as it has an impact on cancer through the intervening or intermediate variable A. However an association between two variables such as smoking and asbestos exposure could also be due

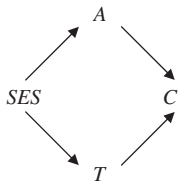


Fig. 4.1 Socio-economic status, smoking, asbestos exposure and cancer of the respiratory system

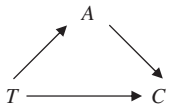


Fig. 4.2 The relation between T and A, A being an intervening variable between T and C

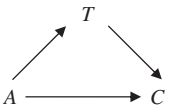


Fig. 4.3 The relation between T and A, A being a common cause of T and C

to a causal relation between them. T could be a cause of A or vice-versa. The two corresponding causal graphs are given in Fig. 4.2 and 4.3 respectively.

This distinction leads to a more precise definition of a confounder: a confounding variable, or confounder, is a variable which is a common cause of both the putative cause and its outcome (Bollen 1989; Pearl 2000; Wunsch 2007). In graphical representations, a common cause is a common ancestor to both putative cause and effect. For example, A is a confounder in Fig. 4.3 because in this model it is a common cause of both T and C. For the same reason, SES is a confounder in Fig. 4.1, as it is a common cause of both T and C (the latter via A). In Fig. 4.2, A is not a common cause of T and C; therefore A is not a confounder. Notice that confounding is always relative to a particular cause and a particular effect. The confounder can be either latent (i.e. unobserved) or observed; the issue of latent confounding is considered in Section 4.5. This definition avoids taking an intervening (intermediate) variable between the putative cause and the outcome such as in Fig. 4.2 as a confounder, even though it is associated with the putative cause (as the latter has a causal influence on the former) and it has an impact on the outcome.

Judea Pearl (2000) proposes two criteria for controlling confounding bias: the back-door and the front-door. The *back-door criterion* tackles the problem of which variables to control for in cases of possible confounding of a cause (C) and effect (E) relation. A variable or a set of variables Z should be controlled for, according to the back-door criterion, if (i) Z is not a descendant of the cause C and (ii) Z blocks every path between C and E that contains an arrow into C. For example, in Fig. 4.4 taken from Pearl (2000), the sets (X_4, X_3) and (X_3, X_5) meet the back-door criterion by blocking every path between C and E containing an arrow into C, while (X_3) alone does not. The variable X_3 is a collider depending upon the inverted fork X_1 and X_2. If we condition on X_3, the variables X_1 and X_2 become dependent

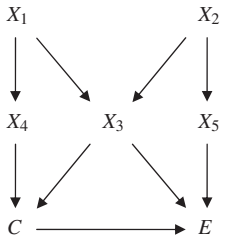


Fig. 4.4 An example of Pearl's back-door criterion

(Pearl 2000; Wunsch 2007) and thus controlling for the sole variable X_3 does not block the path $(C,X_4,X_1,X_3,X_2,X_5,E)$.

The *front-door criterion* uses the presence of an intervening variable between cause and effect to estimate the causal relation. As an example of Pearl's front-door criterion, consider the relation between smoking and lung cancer. If the impact of smoking on lung cancer is mediated by the amount of tar in the lungs, one can estimate on the one hand, the impact of smoking on the amount of tar and on the other hand, the impact of the amount of tar on lung cancer. If these relations are not confounded by other variables, one can then combine the two effects in order to obtain an estimate of the impact of smoking on lung cancer. If the relations between smoking and tar and between tar and lung cancer are confounded, it is sometimes possible to assess the two relations in the absence of confounding if one can control for another variable causing tar accumulation (such as environmental pollution) which blocks the back-door paths from smoking to tar and from tar to lung cancer An example is given in Pearl (2000, pp. 67 and 83). An application of the front-door criterion to the more complex problem of the causal effect of Catholic schooling on learning is given in Morgan and Winship (2007, p. 183).

4.4.2 Complex Systems and Completely Recursive Systems

In the previous sections, only small systems of a few variables have been discussed. Let us now consider a decomposition of X into p components: $X = (X_1, X_2, \ldots X_p)$. Once p increases, the analysis sketched above requires more structure because unrestricted systems become quickly unmanageable. In this section, we show how to use field knowledge with the purpose of obtaining a recursive decomposition of complex systems, giving space to further contextually meaningful restrictions.

Suppose that the components of X have been ordered in such a way that in the complete marginal-conditional decomposition:

$$p_X(x\,|\omega) = p_{X_p|X_1,X_2,\ldots X_{p-1}}\left(x_p\,\middle|x_1, x_2, \ldots x_{p-1}, \theta_{p|1,\ldots p-1}\right) \cdot p_{X_{p-1}|X_1,X_2,\ldots X_{p-2}}\left(x_{p-1}\,\middle|x_1, x_2, \ldots x_{p-2}, \theta_{p-1|1,\ldots p-2}\right)\ldots p_{X_1}(x_1\,|\theta_1) \tag{4.8}$$

each component of the right hand side may be considered as a structural model with mutually independent parameters, i.e. in a sampling theory framework:

$$\omega = \left(\theta_{p|1,\ldots p-1}, \theta_{p-1|1,\ldots p-2} \ldots, \theta_1\right) \in \Theta_{p|1,\ldots p-1} \times \Theta_{p-1|1,\ldots p-2} \ldots \times \Theta_1 \tag{4.9}$$

Equations (4.8) and (4.9) characterize a *completely recursive system*. For $p = 3$, Equation (4.8) may be represented by Fig. 4.5, for $p = 4$ by Fig. 4.6. Once the

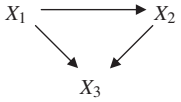


Fig. 4.5 First 3 components of a completely recursive system

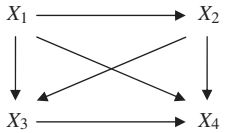


Fig. 4.6 First 4 components of a completely recursive system

value of p increases, graphical representations become quickly unmanageable *unless* some assumptions, in the form of conditional independences, operate simplifications on the system. This is indeed a main issue in structural modelling: field knowledge aims not only at ordering the components of X to obtain (4.8), but also at bringing in more structure than in the complete system (4.8).

More specifically, statistical modelling of complex systems raises several issues:

1. Given a p-dimensional vector of variables to be modelled, is field knowledge sufficient for ordering the variables in such a way that one may obtain a completely recursive system as in (4.8), i.e. in such a way that each component X_j is univariate? It often happens, in particular in econometrics, that it is not possible to disentangle recursively the process generating a vector of variables, in other words that some components X_j are subvectors of X rather than univariate random variables. For instance, Mouchart and Vandresse (2005) handles a case where the data are made of vectors, the components of which are price and attribute of a set a contracts concluded through a bargaining process. The data and the contextual information do not allow to know whether the prices have been bargained after or before the attributes have been agreed upon. This is a case of *simultaneity* where the model describes a process generating a vector of (so-called "endogenous") variables conditionally on a vector of exogenous variables, in such a way that the equations of the model do not correspond to a marginal-conditional decomposition. The econometric literature, particularly between the Sixties and the Eighties, is rich in developing this class of models, called "simultaneous equation models".
2. Endowing each distribution of (4.8) with a structural interpretation amounts to saying that each of these distributions represents a contextually relevant data generating process. Parsimony recommends focusing the attention on the processes of actual interest and is made operational by selecting a subvector $(X_{r+s}, X_{r+s-1}, \ldots, X_r)$ of X such that the joint distribution of $(X_{r+s}, X_{r+s-1}, \ldots, X_r \,|X_1, \ldots X_{r-1})$ gathers all data generating processes of actual interest. In such a case the subvector $(X_1, \ldots X_{r-1})$ becomes globally exogenous for the system of interest.

4.5 Partial Observability and Latent Variables

4.5.1 A Three-Component System

In this paper, the concept of causality is not rooted in latent variables, as in the literature on counterfactuals (see for instance Morgan and Winship 2007). However, this section shows that when latent variables are present in a structural model, causal attribution becomes substantially more complex.

Historically, latent variables have been object of interest since at least the Forties and early Fifties, see e.g. Reiersøl (1950), Neyman and Scott (1948, 1951). Latent variables appear in measurement error models and in factor analytic and LISREL type models, among others. Also those models and simultaneous equation models have been shown to be mathematically equivalent as they are all based on the idea that mathematical expectations are required to lie in a linear space (Florens et al. 1976, 1979). The last years have seen a voluminous amount of publications on the large role of latent variables in statistical modelling. Thus Chapter 1 of Skrondal and Rabe-Hesketh (2004) speaks of "the omni-presence of latent variables", and the book presents an interesting account of methodological issues and of applications. Rabe-Hesketh et al. (2004) suggest how to use a latent variable framework as a unifying device for a large class of models including multilevel and structural equation models.

We begin by considering a three-variate case and next extend the analysis to a p-dimensional vector. Consider a three-variate completely recursive system, represented in Fig. 4.7, for data in the form $X = (Y, Z, U)$:

$$p_X(x\,|\theta) = p_{Y|Z,U}\left(y\,|z, u, \theta_{Y|Z,U}\right) p_{Z|U}\left(z\,|u, \theta_{Z|U}\right) p_U(u\,|\theta_U) \tag{4.10}$$

where each of the three components of the right hand side may be considered as structural models with mutually independent parameters, i.e. in a sampling theory framework:

$$\theta = \left(\theta_{Y|Z,U}, \theta_{Z|U}, \theta_U\right) \in \Theta_{Y|Z,U} \times \Theta_{Z|U} \times \Theta_U \tag{4.11}$$

This diagram suggests that U causes Z and (U, Z) cause Y. Thus, according to the definition offered above, U is a confounding variable for the effect of Z on Y. Also, Equations (4.10) and (4.11) say that U is exogenous for $\theta_{Z|U}$ and that (U, Z) are jointly exogenous for $\theta_{Y|Z,U}$.

Now suppose that U is not observable. It might be tempting to collapse the diagram in Fig. 4.7 into that of Fig. 4.8. Formally, Fig. 4.8 may be obtained by integrating the latent variable U out of (4.10):

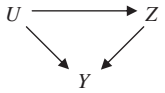


Fig. 4.7 3-component completely recursive system

Fig. 4.8 2-component system

$$p_{Y|Z}\left(y\left|z,\theta_{Y|Z}\right.\right)=\frac{\int p_{Y|Z,U}\left(y\left|z,u,\theta_{Y|Z,U}\right.\right)p_{Z|U}\left(z\left|u,\theta_{Z,U}\right.\right)p_U\left(u\left|\theta_U\right.\right)du}{\int\int p_{Y|Z,U}\left(y\left|z,u,\theta_{Y|Z,U}\right.\right)p_{Z|U}\left(z\left|u,\theta_{Z,U}\right.\right)p_U\left(u\left|\theta_U\right.\right)du\,dy} \tag{4.12}$$

$$p_Z\left(z\left|\theta_Z\right.\right)=\int p_{Z|U}\left(z\left|u,\theta_{Z|U}\right.\right)p_U\left(u\left|\theta_U\right.\right)du \tag{4.13}$$

Therefore:

$$\theta_{Y|Z}=f_1\left(\theta_{Y|Z,U},\theta_{Z,U},\theta_U\right)\qquad\theta_Z=f_2\left(\theta_{Z|U},\theta_U\right) \tag{4.14}$$

Two remarks are in order:

1. In general, Z is not exogenous anymore because (4.14) shows that the parameter $\theta_{Y|Z}$ and θ_Z are, in general, not independent; indeed some components of $\theta_{Z|U}$ and of θ_U may be common to $\theta_{Y|Z}$ and θ_Z. Therefore, Fig. 4.8 is an inadequate simplification of Fig. 4.7 (see however next remark);
2. the non-observability of U typically implies a loss of identification: the functions f_1 and f_2 are *not* one-to-one; thus Z might still be exogenous because potentially common parameters in $\theta_{Y|Z}$ and θ_Z might not be identified;

One might also look for further conditions deemed to recover the exogeneity of Z. A simplifying assumption frequently used is the sampling independence between Z and U:

$$Z\perp\!\!\!\perp U\,|\theta \tag{4.15}$$

This assumption implies that $\theta_{Z|U}$ is now written as θ_Z and Fig. 4.7 becomes Fig. 4.9 suggesting that U and Z both cause Y (without U causing Z).

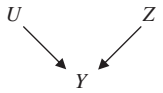
Under condition (4.15), when U is not observable Fig. 4.8 is again obtained under the following integration of U:

$$p_{Y|Z}\left(y\left|z,\theta_{Y|Z}\right.\right)=\int p_{Y|Z,U}\left(y\left|z,u,\theta_{Y|Z,U}\right.\right)p_U\left(u\left|\theta_U\right.\right)du \tag{4.16}$$

Therefore:

$$\theta_{Y|Z}=f_3\left(\theta_{Y|Z,U},\theta_U\right) \tag{4.17}$$

Fig. 4.9 3-component completely recursive system with marginal independence



is independent of θ_Z and the exogeneity between Z and $\theta_{Y|Z}$ may be recovered. In particular, under condition (4.15), U is not a common cause of Z and Y anymore, but, from (4.17), the meaning of $\theta_{Y|Z}$ comes from a combination of the causal action of U along with that of Z, represented by $\theta_{Y|Z,U}$, and of the distribution of U, represented by θ_U.

An example may be useful to better grasp some difficulties. Suppose, for simplifying the argument, that the joint distribution of X in (4.10) is multivariate normal; thus the regression functions are linear and the conditional variances are homoscedastic, i.e. they do not depend on the value of the conditioning variables. Let us compare the following two regression functions:

$$E\left[Y \,\middle|\, Z, U, \theta_{Y|Z,U}\right] = \alpha_0 + Z\alpha_1 + U\alpha_2 \tag{4.18}$$

$$\begin{aligned}\alpha_1 &= [cov\,(Y, Z\,|U\,)]\,[V\,(Z\,|U\,)]^{-1} \\ &= \left[cov\,(Y, Z) - cov\,(Y, U)\,[V\,(U)]^{-1}\,cov\,(U, Z)\right] \\ &\quad \times \left[V\,(Z) - cov\,(Z, U)\,[V\,(U)]^{-1}\,cov\,(U, Z)\right]^{-1}\end{aligned} \tag{4.19}$$

$$E\left[Y \,\middle|\, Z, \theta_{Y|Z}\right] = \beta_0 + Z\beta_1 \quad \beta_1 = [cov\,(Y, Z)]\,[V\,(Z)]^{-1} \tag{4.20}$$

Therefore, if the effect on Y of the cause Z is measured by the regression coefficient, the correct measure would be α_1 rather than β_1, once the conditional model generating $(Y\,|Z, U\,)$ is structural. Note that, in this particular case, $\alpha_1 = \beta_1$ when $Z \perp\!\!\!\perp U$, but this is a particular feature of the normal distribution for which $Z \perp\!\!\!\perp U$ implies that $cov(Y, Z\,|U\,) = cov(Y, Z)$, and $cov(Y, U\,|Z\,) = cov(Y, U)$, which is in general not true. Moreover, $\alpha_1 = \beta_1$ is also true when $\alpha_2 = 0$, i.e. when $Y \perp\!\!\!\perp U\,|Z$, which is contextually different from $Z \perp\!\!\!\perp U$.

This example makes two issues explicit:

(i) measuring the effect of a cause should be operated relatively to a completely specified structural model; failing to properly recognize this issue may lead to fallacious conclusions because in general: $\alpha_1 \neq \beta_1$

(ii) prima facie ancillary specifications, such as a normality assumption, may be more restrictive than first thought; indeed, under a normality assumption, the hypotheses $Z \perp\!\!\!\perp U$ and $Y \perp\!\!\!\perp U\,|Z$ each imply that $\alpha_1 = \beta_1$, although they are contextually different once the normality assumption is not retained. This happens because, in the normal case, independence is equivalent to uncorrelatedness, and because the regression functions are linear.

4.5.2 The General Case

A difficult issue in structural modelling is bound to the fact that many theories in the social sciences involve latent or nonobservable variables. These are introduced in order to help structuring a theoretical framework; think, for instance, of the concept of "anomy" in sociology or of "permanent income" in economy. In such a case, the

initial model includes both latent and manifest or observable variables, from which a statistical model is obtained by integrating out all the latent variables. A typical benefit of such an approach is to obtain a statistical model with more structure, i.e. more restrictions, than a "saturated" statistical model constructed independently of a structural approach. A well-known case is provided by the LISREL type model, or covariance structure model. However this structural approach has also a cost, sometimes difficult to handle. Indeed, the analysis performed around the simplest case of one unobservable variable along with two observable variables, given through Equations (4.12) and (4.13), suggests that the analysis of exogeneity *at the level of the statistical model bearing on the manifest variables only* soon becomes intractable, jeopardizing most exogeneity properties and making the interpretation of the identifiable parameters difficult.

4.6 Discussion and Conclusion

Philosophers have wandered for long time in search of the ultimate *concept* of causality, i.e. in search of what causality in fact *is*. Hume (1748), unable to find what gives logical necessity to causal relations, came to the conclusion that causality is nothing more than a regular succession of events deemed to be causal only thanks to our psychological habit to experience such regular sequences. In his *System of Logic*, John Stuart Mill, as early as 1843, put forward an experimentalist notion of cause. Causes are *physical*, i.e. one physical fact is said to be the cause of another. In the *System of Logic* the experimental approach is seen as the privileged way for ascertaining what phenomena are related to each other as causes and effects. We have, says Mill, to follow the Baconian rule of varying the circumstances, and for this purpose we may have recourse to observation and experiment. Mill believed that his four methods – Method of Agreement, Method of Difference, Method of Residues, and Method of Concomitant Variation – were particularly well suited to natural science contexts but not at all to social sciences. The inapplicability of the experimental method to the social sciences ruled them out straight away from the realm of the sciences and still nowadays leads to a skeptical despair about the very possibility of establishing causal relations in social contexts.

Causal analysis has indeed proved to be a challenging enterprise in the social sciences. There are at least two difficulties in establishing causal relations. A first difficulty is, as just mentioned, that a pure randomized experimentation is rarely possible. A second one, already discussed in the Introduction, is that society and individuals are too mutable to generate "laws of social physics" *à la* Quetelet. However, is this reason enough to give up causal analysis? Should we then content ourselves with Humean regular successions?

Interestingly enough, Durkheim (1895, Chapter VI) strongly argued against the Millian attempt to dismiss social sciences as *sciences* and therefore against any attempt to dismiss causal analysis. In particular, he maintained that the method of concomitant variation is fruitfully used in sociology and indeed this is what makes

sociology scientific. Although an explicit causalist perspective has been adopted by the forefathers of quantitative causal analysis, in more recent times practising scientists have, mistakenly, hardly ever taken a clear stance in this respect. As we have suggested in the opening of this paper, a cognitive goal and an action-oriented goal justify our effort in making causality an empirical and testable, i.e. scientific, matter.

We have argued that structural modelling tries to make causality meaningful and operational and we have seen that this objective can be achieved if two fundamental ingredients are incorporated. The first one is an epistemological element – viz. the rationale of variation, and the second is a methodological element – viz. the concept of *structural* model. Structural modelling aims at uncovering a structure underlying the actual data generating process. Clearly there is an infinity of conceivable structural models leading to a same statistical model "explaining" the data under scrutiny. A main issue for the model builder is selecting one of those structures, taking into account the knowledge of the field and desirable properties of invariance/stability. Thus the practical implication of this paper is twofold. Firstly, causation may be attributed only within a structural model reflecting the state of knowledge of the domain considered. Secondly, the structural stability of the relationships *and* of the parameters of the distributions should be thoroughly checked. This approach is therefore at variance with purely statistical ones where causation is supposedly tested from correlations without making explicit a suitable structural model. Furthermore, causation should not be attributed from a model only based on purely theoretical considerations. Finally, the search for agreement with background knowledge and for structural stability leaves a lesser role to the goodness of fit.

However, although the development of a more adequate rationale of causality and of an accurate concept of structural model give a meaningful framework for causal analysis, we claimed that specific issues still needed to be addressed, e.g. exogeneity and confounding. In this causal framework, the concepts of exogeneity and of confounding have been explicitly defined. On the one hand, exogeneity is a condition of separability of inference that allows us to concentrate on the conditional distribution leaving aside the marginal one. On the other hand, we have adopted a definition of confounders as common ancestors of both cause and effect. However, we have shown that the impact of confounders complicates substantially the analysis and the operational interpretation of exogeneity, because a variable may lose its exogenous status under the impact of a latent confounder. Furthermore, if a latent variable U is a determinant of an outcome Y but is independent of another cause Z of this outcome, Z remains exogenous but, at the level of the manifest variables, the measure of the effect of Z on Y depends upon the original causal effect of Z and upon the distribution of the latent variable U.

Let us now give some general conclusions. In the framework of structural modelling what is the meaning of the claim X *causes* Y? Not metaphysical: by means of structural modelling we do not pretend to attain the ontic level and to discover the true and ultimate causes. If causal claims cease to have metaphysical meaning, then they must have an epistemic one: we have reasons to *believe* that X causes Y. Causality thus becomes a matter of *knowledge* generated by the sensible use

of structural modelling. A major task of epistemology and methodology is then to make explicit the conditions under which our causal beliefs are justified and to inform us correctly about causal relations in the world. The net advantage of spousing an epistemic view is to avoid committing to the discovery of the "true" causes or of the "true" model. Instead, causal beliefs are part of our knowledge of the world, and thus are naturally subject to change and improvement.

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