

# Causal attribution in block-recursive social systems: A structural modeling perspective

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## Abstract

One method for causal analysis in the social sciences is structural modeling. Structural models, as used in this article, model the (causal) mechanism for a social phenomenon by recursively decomposing the multivariate distribution of the variables of interest. Often, however, one does not achieve a complete decomposition in terms of single variables but in terms of “blocks” of variables only. Papers giving an overview of this issue are nevertheless rare. The purpose of this article is to categorize distinct types of block-recursive and to examine, in a multidisciplinary perspective, the implications of block-recursive for causal attribution. A probabilistic approach to causality is first developed in the framework of a structural model. The article then examines block-recursive due to the presence of contingent conditions, of interaction, and of conjunctive causes. It also discusses causal attribution when information on the ordering of the variables is incomplete. The article concludes by emphasizing, in particular, the importance of properly specifying the population of reference.

## Keywords

Causality, block-recursive, contingent conditions, interaction, conjunctive causes

## Recursivity and structural modeling

Causal attribution is the problem of establishing what causes what. In the past decades, a number of quantitative approaches have been developed, notably the so-called *structural modeling* approach. The latter, at least in the account given by Mouchart and Russo (2011) and Wunsch et al. (2014), models the *mechanism* of the phenomenon of interest. This is particularly useful in social science, in which experimentation is often limited for practical or ethical reasons that are beyond the scope of this discussion. The way in which mechanistic modeling and structural modeling intersect is of particular interest here.

Glennan and Illari (2018: Chapter 1) explain that in the social sciences, as well as in the life sciences, the search for mechanisms has been a reaction to the tradition of logical empiricism, which depicted the scientific enterprise as the search for laws (of nature) and explanation as an exercise in subsuming particular events under the said laws. Glennan and Illari propose a formulation that is supposed to capture the main features of mechanisms in the social and life sciences. They call it *minimal mechanism*: “A mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the

phenomenon.” This formulation blends earlier attempts to generally characterize what a mechanism is, notably those given by Illari and Williamson (2012) and by Glennan (2017).

In different disciplines, the search, discovery, and validation of mechanisms take different forms. See, for instance, Demeulenaere (2011) for a thorough discussion of mechanisms in sociology. As mentioned above, in quantitative social science, mechanisms can be modeled using “structural models.” These models, which will be presented in detail in the following section, have the characteristic of modeling a given phenomenon by elucidating its *probabilistic structure*.

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Mouchart and Russo (2011) and Wunsch et al. (2014) argue that such probabilistic structure can be interpreted as a mechanism along the lines of Glennan and Illari's (2018) "minimal mechanism" characterization. Interpreting probabilistic structures as mechanisms allows doing simultaneously two things—on the one hand, to cash out the notion of causality as a particular property of the model, notably *exogeneity*, and, on the other hand, to provide an account of how quantitative social studies can be explanatory, notably by providing an appropriate description of the functioning of the mechanism (see also Russo, 2011).

Simply put, the kernel of a structural model is the recursive decomposition of the joint distribution of the variables of interest. The explanatory endeavor is to *give structure* to this joint distribution, by "breaking it down" into conditional distributions representing cause–effect relations. The recursive decomposition of the initial joint distribution, in other words, serves to identify the several sub-mechanisms that are "responsible for the phenomenon." Under a condition of exogeneity, causes are then identified with the conditioning variables in a sub-mechanism generating an outcome of interest.

A structural model, in the sense of this article, is taken as a particular case of a causal model and is identified by three main features. Each of these three conditions is relative to a population of reference, the specification of which is an essential part of the modeling enterprise, from the formulation of the research question and data collection to the interpretation of results. The specification of the population of reference is even more important in the case of a block-recursive decomposition, which is the object of the present discussion. These three conditions are summarized below, and the reader is directed to Mouchart et al. (2009) for a full discussion:

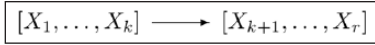
1. A recursive decomposition of the joint distribution of variables, to be interpreted as a sequence of sub-mechanisms, reflecting the causal ordering of the variables and their function in each sub-mechanism. This sequence, decomposing the putative mechanism into a series of relevant sub-mechanisms, makes causal assessment feasible. The recursive decomposition, or mechanism, can be visually represented by a directed acyclic graph (DAG).
2. Congruence with background knowledge, including preliminary analysis of the data. The causal ordering of the variables is usually based on prior knowledge, including information on the temporal ordering of the variables and on the context or environment in which they take place. These elements also play a role in the specification of the population of reference.
3. Invariance or stability of the recursive decomposition for the population of reference, in a specific context and period of time. This is in opposition to the idea of a "universal" theory or model, as can be the case in

economics, for example. This is also in opposition to a model that would be different for each observation. Invariance is a condition for separating the incidental aspects from the structural aspects of the behavior of the statistical units and allows, therefore, establishing causal claims that are valid for the population under study.

This structural modeling perspective on causality can be taken as a strategy for modeling causal assertions derived from background knowledge. This perspective is rooted in a precise statistical concept of causality, namely, that of exogeneity in a recursive conditional model with an explicit condition of exogeneity, where the latter is taken as a condition of separation of inference. This condition allows the inference on the parameters of a conditional model to be operated independently of the inference on the parameters of the model generating the conditioning variables, in line with the concept of a cut in a statistical model (see Barndorff-Nielsen, 1978; Florens and Mouchart, 1985).

Recursive systems require a unidirectional relationship among the variables, with no simultaneous feedback. It often happens, however, that a complete ordering of variables cannot be accomplished, either because some causes occur jointly, as contingent conditions (such as Mackie's (1965) INUS (Insufficient but Necessary part of a condition which is itself Unnecessary but Sufficient for the result) causes) or as conjunctive causes (i.e. acting jointly on the outcome), or because one does not have the information required for determining the order or causal priority among the variables. In these cases, the recursive decomposition is not complete anymore, in the sense that some of the conditional distributions are not univariate but concern vectors—or "blocks"—of variables. One then speaks of a *block-recursive system* (BRS). The purpose of this article is to categorize distinct types of block-recursivity and to examine the implications of block-recursivity for causal attribution. A probabilistic approach to causality will be developed on the basis of a structural or causal model, in the framework of a statistical model embodying a partial and non-deterministic explanation of a social phenomenon.

The term "block recursivity" has been used in the statistical literature with different meanings. In particular, an interesting paper (Wermuth, 1992) uses this term for analyzing structures in correlation matrices, without paying a systematic attention to causality. As the analysis is confined to multivariate normal distributions, particular features of the latter underlie the analysis, in particular linear and homoscedastic regressions, identity between uncorrelatedness and stochastic independence, along with various analytical specificities (see, for instance, Mouchart et al., 2009: Section 4.5.1). At variance from Wermuth's approach, this article is essentially distribution-free, in the sense that no distributional assumptions are assumed, and uncorrelatedness is not identified with stochastic independence. Moreover, the concept of



**Figure 1.** A primitive block-recursive system.

recursivity is based on a (complete or partial) ordering of the variables.

This article has been conceived as a methodological contribution written in a multidisciplinary perspective by a social scientist, a statistician, and a philosopher of science. The statistical models underlying the analysis are distribution-free; moreover, no estimation methods are discussed. Of course, the latter should be considered when implementing the methodological approach developed here.

The order of exposition is as follows. In section “Block-recursivity: endogenous and exogenous blocks,” the concept of block-recursivity is presented as a partial development of a complete structural model. The article then examines different types of block-recursivity due to the presence of contingent conditions (section “Causal attribution in the presence of contingent conditions”), of interaction (section “Causal attribution in the presence of interaction”), and of conjunctive causes (section “Causal attribution in the presence of conjunctive causes”). Section “Causal attribution when information on the ordering of the variables is insufficient” discusses causal attribution when information on the ordering of variables is insufficient. The final section “Discussion and conclusion” discusses, among others, the importance, for causal attribution, of correctly specifying the population of reference.

## Block-recursivity: endogenous and exogenous blocks

In this section, a specific method for causal attribution, namely, structural modeling, is examined. The starting step is the elaboration of a statistical model representing the data generating process (DGP) by means of a family of multivariate distributions of the variables of interest. A structural/causal model, in the sense of this article, is provided by a completely recursive decomposition of the DGP. More specifically, consider a random vector of  $r$  components  $X = (X_1, X_2, \dots, X_i, \dots, X_r)$ , where the component  $X_i$  may be either a univariate random variable or a random vector. The recursive decomposition is obtained through a systematic marginal-conditional decomposition of the joint distribution of  $X$  that can be expressed as

$$P_X = P_{X_1} P_{X_2|X_1} \cdots P_{X_i|X_1, X_2, \dots, X_{i-1}} \cdots P_{X_r|X_1, X_2, \dots, X_{r-1}} \quad (1)$$

where  $P_{X_i|X_1, X_2, \dots, X_{i-1}}$  represents the conditional distribution of  $X_i$  given  $X_1, X_2, \dots, X_{i-1}$ . Among the possible recursive decompositions (namely,  $r!$ ), the structural, or causal,

approach selects the one that may be interpreted as a decomposition of the global mechanism into an ordered sequence of acting sub-mechanisms (more details are given in Mouchart et al., 2009).

The recursive decomposition is the cornerstone of the explanatory power of a structural model because it endows the joint distribution of  $X$  with the interpretation that each component of the decomposition stands for one of the sub-mechanisms that compose the DGP of  $X$ . The recursive decomposition is built in such a way that the identified sub-mechanisms are interpretable from background knowledge. Background knowledge is an elusive concept and is crucial for most modeling practices across the sciences. In the social sciences, background knowledge includes all available information about the population of reference, from basic demographic characteristics to the broad sociopolitical context, and on the phenomenon under consideration. All these elements play an important role in, for example, variable selection and in the marginal-conditional decomposition of the joint distribution.

The order of the decomposition of  $X$  is crucial for the interpretability of the components as sub-mechanisms. In addition, characterizing a sub-mechanism often leads to eliminating, in this sub-mechanism, some of the variables appearing previously in the ordering of variables. In a completely recursive decomposition, each sub-mechanism is accordingly composed of an endogenous outcome variable and of conditioning variables that may be interpreted as jointly causing this endogenous variable. In general, a recursive decomposition may be represented by a DAG (Pearl, 2000).

If in equation (1) each component  $X_i$  is a univariate random variable, this decomposition is *completely recursive*; otherwise it is *partially recursive* or *block-recursive*. This last case is the object of this article.

A primitive BRS may be represented by the DAG given in Figure 1, where  $[X_1, \dots, X_k]$  stands for a block of “exogenous” variables and  $[X_{k+1}, \dots, X_r]$  stands for a block of “endogenous” variables.

This DAG corresponds to the following recursive decomposition of the joint distribution of  $(X_1, \dots, X_k, X_{k+1}, \dots, X_r)$

$$P_{X_1, \dots, X_r} = P_{X_1, \dots, X_k} P_{X_{k+1}, \dots, X_r | X_1, \dots, X_k} \quad (2)$$

Neither of the two factors  $P_{X_1, \dots, X_k}$  and  $P_{X_{k+1}, \dots, X_r | X_1, \dots, X_k}$  is recursively decomposed. Thus, no sub-mechanisms are identified. There is only a causal relation between blocks of variables, namely, the vector  $(X_1, \dots, X_k)$  is assumed to cause the vector  $(X_{k+1}, \dots, X_r)$ , and there is no simultaneous feedback of the latter on the former.

A primitive BRS is a natural first step in the development of a structural, or causal, model and aims at settling the following issues:

1. What variables  $(X_1, \dots, X_r)$  should enter the analysis?

2. What is the mechanism of interest, and which variables should be viewed as exogenous and endogenous?

The answer to questions (1) and (2) does not come from statistical considerations alone. Instead, background knowledge is essential here. As a first step in the analysis, it is implicitly assumed that in equation (2) the interest is focused on the last factor  $P_{X_{k+1}, \dots, X_r | X_1, \dots, X_k}$ . Decomposition (2) is crucially dependent on one's background knowledge of the field.

The next step should endeavor at obtaining a complete recursive decomposition of the factor of interest, namely

$$P_{X_{k+1}, \dots, X_r | X_1, \dots, X_k} = P_{X_{k+1} | X_1, \dots, X_k} P_{X_{k+2} | X_1, \dots, X_{k+1}} \cdots P_{X_r | X_1, \dots, X_{r-1}} \quad (3)$$

In many cases, it is not possible to complete this step and one has to deal with a BRS or with a system in which blocks of variables remain that cannot be causally ordered. It is then impossible to conduct a complete causal analysis, in the sense that the causal ordering of some variables possibly remains latent or is not causally relevant.

The focus here is on the model specifying  $P_{X_{k+1}, \dots, X_r | X_1, \dots, X_k}$ , accepting, in this article, that  $X_{k+1}$  may be univariate or multivariate. The qualification of this model as a *block-recursive model* means that  $P_{X_1, \dots, X_k}$  is not completely recursively decomposed. If it were, Mouchart et al. (2016) show how to analyze cause–effect relations by means of controlling suitably chosen variables. In this article, particular attention is paid to specifying different types of causal relations, according to various characteristics of the incomplete decomposition of  $P_{X_1, \dots, X_k}$ . This incompleteness problem may be due, in particular, to the presence of contingent conditions, interaction among causes, conjunctive causes, or the lack of information on the ordering of some variables, examples being given for each case. The following sections examine situations where this incompleteness problem has different sources.

## Causal attribution in the presence of contingent conditions

In the study of the effects of a cause on an outcome, one usually takes for granted a set, or block, of contingent conditions that are not recursively ordered but serve as background to the causal relations and are nevertheless necessary for the occurrence of the outcome. To give an example, the presence of gravity and oxygen is assumed in studies focusing on normal physiological human functions on earth. A cause is therefore usually embedded in a combination of contingent conditions that are necessary for the cause to operate in a specific context or *causal field* (a term coined by John Anderson, 1938). The French physicist Francis Halbwachs (1971) speaks of a *complexe causal*.

The contingent approach to causality is best exemplified by the well-known *INUS condition* proposed by the philosopher J.L. Mackie (1965, 1974); Mackie starts with the example of a fire (the outcome) breaking out in a house. It could be due to a spark caused by a short-circuit, or due to the overturning of a lighted oil stove, or due to a strike of lightning, and so on; all are possible causes of the outcome. But the fire could not have occurred without necessary or contingent (context-dependent) conditions such as oxygen, inflammable material, the lack of an automatic sprinkler, and so on. In this block of standing conditions, the latter are not causally ordered. Following Mackie (1974), there are usually triggering causes (such as a spark) and predisposing causes (such as inflammable material), the latter being part of the causal field but most often actually not called a cause in the circumstances, although they are part of the “full cause” (in Mackie’s terms) of the outcome. For Mackie, the actual cause, such as a short-circuit, is thus an INUS. Distinguishing between causes and conditions depends on an adequate specification of background knowledge and, in social science contexts, of the population of reference.

A probabilistic version of the INUS condition, called INUP causation, has been proposed by Ellett and Ericson (1983):  $Y$  is an INUP cause of  $Z$  if  $Y$  is neither a sufficient nor a probabilistic cause but is a non-redundant part of a set  $S$  of conditions which is an unnecessary but a probabilistic cause of  $Z$ . In other words, although  $Y$  does not by itself influence  $Z$ , the set or block  $S$  (which has  $Y$  as part) does influence  $Z$  in a probabilistic manner. Mackie’s INUS condition has been largely discussed in the literature; for instance, see Pearl (2000: Chapter 10) for the determination of the actual cause of an outcome, in a causal modeling approach which is close to the structural modeling one presented in the previous section.

Contingent or standing conditions reflect a state of stability, or absence of change, of variables that are necessary for the working of the causal (sub-)mechanism. The concept of stability is relative to a specific context. Within this context, a stable variable should, however, not be considered as a cause because causality relates to a concept of variation of an outcome due to a variation of a causing variable via a suitable (sub-)mechanism (see Russo, 2009). For this reason, a contingent stable condition is not viewed as a cause, as by definition it does not vary.

Debates on causality in philosophy and epidemiology made much use of Mackie’s INUS definition to emphasize that causes are not always, or necessarily, “one” object, event, or variable, but rather one *part* within a set, or block, of (INUS) causes and conditions. So, for instance, Mackie’s approach explains quite well disease causation, and it is no chance that a very similar model has been developed in epidemiology by Rothman (the so-called “Rothman’s pies”; see Rothman, 1976; Rothman et al., 2008; for a discussion, see Illari and Russo, 2014: Chapter 4). Comparatively little attention has been given, however, to the *conditions*. In simple examples like the fire, mentioned above, it is pretty easy



to identify what conditions should be in place so that INUS causes can operate. It is in fact part of our “common knowledge” that fire can develop only in the presence of oxygen. In many other cases, however, understanding the conditions is as much important (and difficult) as understanding the causes. Specifying the population of reference helps precisely in this respect.

In a structural or causal model, as the one presented earlier in sections “Recursivity and structural modeling” and “Block-recursivity: endogenous and exogenous blocks,” the conditions that make INUS causes possible to operate are typically not explicitly included in the statistical model. They are rather part of the general description of the context, including the population of reference, in which the mechanisms take place. This context should nevertheless be described as a part of the explanation of the phenomenon studied. For example, if one compares contraceptive behaviors in Sweden and Chad, the sociocultural contexts of both countries should be described, as they are particularly relevant for the topic studied. Causal models in the social sciences are actually valid only for specific contexts and time periods, in contrast to the laws of physics, for example, and the context and period should always be defined. The point is that these descriptions should be studied in more detail; in other words, the *conditions* should be studied as much as the *causes*. To understand why studying the conditions may make a difference in the causal analysis, consider the following illustrative example, a modification of Mackie’s original one.

Consider now two houses, where a spark occurs in both houses due to a short-circuit. The houses have similar fixtures and fittings, except that house A is full of inflammable material and house B is not. Due to this difference, house A catches fire but house B does not. Once again, if the focus is on house A, the spark would be considered as the cause of the fire. Compare now what happened to house A and to house B. This time, the presence of inflammable material in house A (vs its absence in house B) would most probably be considered here as a main cause of the fire, rather than the spark, as a spark has occurred in both houses. In this case, the spark could become a part of the context. The variation of a causing variable should thus be taken as a *difference-making* factor that produces (or leads to) the outcome, in this case the fire.

In social science research, the identification of the conditions is not as easy as in the simple example above, but is nonetheless essential. Consider the following example from demographic research. In the study of the fertility transition, Ansley Coale (1973) has specified three prerequisites that have to be fulfilled together before women possibly embrace voluntary limitation of fertility within marriage. Following Coale, women must be ready, willing, and able in order to practice birth control. More explicitly, according to Coale,

1. Fertility must be within the calculus of conscious choice;

2. Reduced fertility must be perceived as advantageous;
3. Effective techniques of fertility reduction must be available.

From this point of view, the conjunction *ready*  $\wedge$  *willing*  $\wedge$  *able* is a prerequisite for the decision to practice birth control, or not, that is, a contingent condition, and, as Coale shows, there is no necessary causal ordering among these three conditions. One can say that the three variables form a block or set of necessary conditions that have to be satisfied prior to the decision of practicing, or not, contraception. Studying the block of *conditions* is, in cases like this, of utmost importance: one cannot get a good grasp of choices about birth control unless one understands what makes them possible. Understanding how these conditions are acting goes beyond a summary narrative of the sociodemographic or political context. Background knowledge certainly plays an essential role here, but what is required, at times, is a proper causal analysis, using the tools presented earlier in sections “Recursivity and structural modeling” and “Block-recursivity: endogenous and exogenous blocks.”

More specifically, one may start with a population of reference composed of women of fertile age in a given sociocultural context. If  $R, W, A, F$  stand for a binary coding of *ready*, *willing*, *able*, *fertility regulation*, a simple structural model could be written as  $P(F | R.W.A = 1) = p_1$  and  $P(F | R.W.A = 0) = p_0$ . Among the women who are *ready*  $\wedge$  *willing*  $\wedge$  *able* (i.e.  $R.W.A = 1$ ), a main issue is to decide what other variables enter the conditional process generating fertility control  $F$ , as some of these women have recourse to contraception and others do not. For the reference population composed of women who are ready, willing, and able, one should then attempt at developing a structural model generating the outcome, that is, fertility control, taking the available background knowledge into account. The women who are not *ready*  $\wedge$  *willing*  $\wedge$  *able* (i.e.  $R.W.A = 0$ ) constitute another population of reference that would require another structural model generating the possible outcome (fertility control).

The contingent condition concept, however, needs to be clarified. A contingent condition is a necessary condition for an outcome to occur and is taken for granted, or not, according to the context. To give an example, in the natural sciences, the presence of oxygen is taken for granted on our earth but not on the moon. In the social science context, an analogous reasoning can be made. For instance, in a modern society, Coale’s three prerequisites are usually taken for granted for a large proportion of the population, making  $p_0$  of negligible interest. This would not be the case in a traditional society.

Thus, the presence of necessary or contingent conditions raises the issue of explicitly defining the population of reference. These conditions form a block that might possibly be explained by a structural model that would enrich the causal analysis, compared to the case of an unexplained block of conditions.

## Causal attribution in the presence of interaction

In the presence of disjunctive causes acting separately, there might also be *interaction effects* between the causes that jointly affect the outcome as a block; these effects are often called *moderator effects* in the psychological literature (Baron and Kenny, 1986). The impact of a cause on an outcome can be *moderated* (weakened) by another cause, but it can also be *amplified* (strengthened). The term *moderator* is, however, used in both the cases. For example, the interaction between two drugs can alter the effect of the drugs. Contraceptive pill users also taking anticonvulsants have much higher contraceptive failure rates. Moreover, the hormonal contraceptive may result in therapeutic failure of the antiepileptic drug and aggravate seizure (Reimers et al., 2015).

To give another example, both smoking and asbestos exposure alone are a cause of lung cancer, but it has been shown that the joint impact of asbestos exposure and smoking, among asbestos workers, is much greater than additive (Frost et al., 2011). In other words, in this last case, there is a significant interaction between these two causes of lung cancer. One also says that the effect of one of the causes has “moderated” the impact of the other cause. The total impact of the two causes on the outcome derives from their separate action plus their joint interaction, that is, their disjunctive effects plus their conjunctive effects. Interaction effects are not always predictable and are often not assumed beforehand; they are usually discovered when analyzing the data. One should, therefore, always test for possible interaction effects at the analysis stage of the model.

Interaction effects are commonly taken into account, in linear statistical models, by adding multiplicative terms to the equation; see, for example, Stock and Watson (2003: Section 6.3) and especially Aiken and West (1991) for an older but thorough analysis of interaction in multiple regression. More specifically, once a recursive structural/causal model has been developed, for each causal variable having a direct effect on an outcome, one should condition on the other variables having a direct effect on this outcome (see Mouchart et al., 2016). This takes care of possible interaction effects between the direct causes of an outcome and also controls for potential confounders. For example, in the smoking-asbestos case, one would, respectively, study the effect of smoking on lung cancer among those not exposed to asbestos, the effect of asbestos exposure among the non-smokers, and finally the impact on lung cancer among the smokers who are also exposed to asbestos.

More precisely, suppose variables  $X$  and  $Z$  have a direct effect on an outcome variable  $Y$ . As the direct effect of  $X$  or  $Z$  on  $Y$  is characterized by the conditional distribution  $p_{Y|X,Z}$ , it is important to condition the outcome  $Y$  on both  $X$  and  $Z$  in order to detect possible cases with interaction, where the direct effect of  $X$  (or  $Z$ ) depends on the value of  $Z$  (or  $X$ ), as opposed

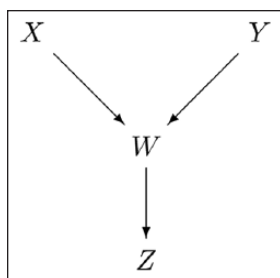
to cases without interaction where the direct effect of  $X$  (or  $Z$ ) on  $Y$  is not affected by the value of  $Z$  (or  $X$ ). More generally, taking into account interaction effects calls for disentangling the effects of the block of direct causes on an outcome, such as  $X$  and  $Z$  on  $Y$ . Note that interaction effects cannot be adequately represented in a DAG. As pointed out by Rehder (2015), causal networks and DAGs show the dependency structure among variables but cannot represent the functional form of the cause–effect relations.

A more complex form of interaction has been examined by Preacher et al. (2007), that is, moderated mediation. This can happen when an indirect effect on an outcome variable is moderated by another variable. Five illustrative models are discussed by these authors, the moderator impacting either the outcome variable or the mediator, or both. Preacher et al. (2007) focus their analysis on the particular case of linear models, such as path analysis or structural equation models (SEMs), and derive estimates of the conditional indirect effects and of their statistical significance using bootstrapping. More generally, whatever the distributional assumptions and the causal graph, for each (causal) variable having a direct effect on a result variable, one should condition on the other variable(s) having a direct effect on this result variable, following the generic rule recalled above. This takes into consideration both interaction effects and confounding.

As discussed in Mouchart et al. (2016), an interaction or moderator effect may be due to an intrinsic non-additivity of direct effects or to neglecting, in a model, the action of other variables, as might occur when these variables are unknown to the model builder or when the variables are known but not observable. For example, if the variables are discrete, with an ordered confounder such as age there might be a regular trend in the measures from one cell to the other. Moreover, detecting an interaction may be a subtle issue because it can depend on some analytical features of the model. For instance, if the conditional expectation of  $Y$  has the form  $\exp\{\alpha_0 + \alpha_1 X + \alpha_2 Z\}$ , differentiating the conditional expectation reveals an interaction effect, whereas the log of the conditional expectation is simply additive without interaction. Elwood (1992: 153) points out that the term “interaction” is meaningful only if the underlying model which is regarded as the non-interaction situation is described.

## Causal attribution in the presence of conjunctive causes

The example taken from Ansley Coale, in section “Causal attribution in the presence of contingent conditions,” considered the joint or conjunctive variable *ready*  $\wedge$  *willing*  $\wedge$  *able* as a necessary contingent condition for possibly opting for contraception. Conjunctive variables that are not recursively ordered, but observed as a block, can also be causes in some DGPs, with different levels of complexities. The following situations will exemplify two different structures of complexities, namely, the case of a two-component glue, an



**Figure 2.** The two-component adhesive.

illustrative example drawn from the natural sciences, and the case of complex profiles in quantitative criminology.

### The two-component adhesive

Consider the example of a two-component (2C) adhesive, a resin and a hardener, where the use of only one of the components produces no gluing effect, whereas mixing the two components in a proper ratio ensures the adhesive effect by chemical reaction. In this case, the two components act as a block of conjunctive causes: both need to be present to produce the outcome. There is furthermore no causal ordering among the two causes in this example, the conjunction of glues acting together as a block on the outcome. Note that in this example, the 2C glues are not contingent or standing conditions, as in section “Causal attribution in the presence of contingent conditions,” but are actual causes because a change in the state of the variables has occurred. The two components have to be mixed and then applied. Note also that this case differs from interaction: the two components have no separate effects on the outcome but only a joint effect. More generally, conjunctive causes are those that need to occur jointly for an effect to come about.

Following Rehder (2011, 2015), one can decompose the distribution of all the variables of interest in order to take into account the presence of conjunctive causes (see Rehder’s, 2011 and 2015; Figure 1) and to represent the ensuing DGP in a structural modeling perspective. More formally, let  $X$  stand for the resin and  $Y$  for the hardener,  $Z$  being the adhesive effect. The latter can only be produced by the conjunction of  $X$  and  $Y$ . Let  $W$  stand for this conjunction, possibly along with other conditions determining the adhesive effect. Thus, the corresponding recursive decomposition can be written as

$$P_{X,Y,W,Z} = P_X P_Y P_{W|X,Y} P_{Z|W} \quad (4)$$

The corresponding DAG is represented in Figure 2, identifying two sub-mechanisms, namely,  $P_{W|X,Y}$  generating  $W$  (explicating Rehder’s Figure 1 in Rehder, 2011, 2015) and  $P_{Z|W}$  generating the adhesive effect  $Z$ .

The adhesive effect is represented by the sub-mechanism  $P_{Z|W}$ , without explicating the two components  $X$  and  $Y$ . In the simplest case,  $W$  would be a binary variable coding the

conjunction, or not, of the two components, that is,  $W = 1$  if there is conjunction of  $X$  and  $Y$ , and  $W = 0$  otherwise. In more elaborate cases, the adhesive effect might depend not only on the conjunction but also on some characteristics of the components, for instance, their quality, their dosage, and so on. Let  $Q_X$  and  $Q_Y$  stand for these relevant characteristics,  $D$  for the dosage,  $E$  for some environmental characteristics possibly influencing the chemical reaction of the two components, and  $C$  for a binary variable coding the decision to put the two components  $X$  and  $Y$  together. In such a case,  $W$  is a binary function of  $(Q_X, Q_Y, D, E, C)$ . It should be noticed that the sub-mechanism  $P_{Z|W}$  is essentially a chemical mechanism, whereas  $P_{W|X,Y}$  is a complex sub-mechanism combining material variables  $(Q_X, Q_Y, D, E)$  and a decisional variable  $C$ .

### Conjunctive analysis of case configurations

Another interesting approach to conjunctive causality can be found in Miethe et al. (2008). They develop, in the case of categorical variables, a so-called method of “conjunctive analysis of case configurations” (CACC) that is useful in detecting patterns (or case configurations) of conjunctive causes impacting an outcome, that is, a particular combination of elements inducing the outcome. One first selects the independent and dependent variables—that is, the putative causes and the outcome—relying on background knowledge and identifies the complete list of possible case configurations on the basis of all independent variables. CACC then proceeds by putting each observation into its respective case configuration or profile of attributes and analyzing the relative distribution of the outcome across these configurations. The authors apply the approach to the risk of imprisonment of drug offenders according to the conjunction of various legal and extralegal factors (such as type of offense, race, prior sentencing record, etc.) that are expected to influence current sentencing decisions, all variables having been dichotomized. In general, the categorical variables allow for an enumeration of profiles defined by their conjunctions. Empirical inspection of the data permits the detection of the more risky profiles. In the example considered by Miethe et al. (2008), one observes that, as a result of the choice of the variables, the possible profiles are quite different in their frequency and in their impact on the variable of interest, that is, prison sentencing.

To give another example, Mieczkowski and Beauregard (2010) have applied CACC to examine the profile of factors associated with lethal outcomes in sexual assaults. In the case configurations, they have taken three domains conjunctively into account: victim characteristics, situational characteristics, and crime characteristics.

As no explanatory mechanism is explicitly postulated between the independent and dependent variables, CACC has initially been conceived mainly as a tool for exploring data. The analysis becomes unwieldy when there are many

variables and several categories per variable, as the size of the display matrix expands quickly. One could, however, create an ordinal indicator variable accounting for the number of conditions that are satisfied and possibly include it into a recursive decomposition. Actually, the situation can be causally quite complex. For example, individual, neighborhood, and social network characteristics can jointly impact drug use, but the latter can also determine where people live and with whom they interact (Linton et al., 2017).

Although the approach is essentially descriptive, the analysis is nevertheless oriented toward the search of the causes of an effect. In particular, the population of reference and the individual variables are chosen on the basis of background knowledge. This approach is, however, not structural, in the sense of sections “Recursivity and structural modeling” and “Block-recursivity: endogenous and exogenous blocks,” as an ordered sequence of explanatory sub-mechanisms is not identified. Thus, the variables defining the profiles are not ensured to be exogenous. This descriptive approach exhibits relevant features of the phenomenon of interest, but the interpretation of the results requires caution from a causal perspective.

### **Causal attribution when information on the ordering of the variables is insufficient**

There is another possible source of blocks of non-ordered variables in a causal model, namely, the lack of information on the ordering of the variables due to partial background knowledge or to inadequate observation of the temporal sequence of events. For the latter, this is typically the case when events are not recorded, or made available, on a continuous-time basis but by discrete periods of time. Depending on the length of the period, it is not always possible to see whether the putative cause occurred before or after the outcome, that is, to determine causal priority. For example, during a yearly period of time, one may observe both a person’s change of residence and his or her change in occupation. Is the former a cause of the latter or vice versa? The temporal priority of the cause over the effect is impossible to determine in this case, as one only knows what happened during the whole year. Both events are observed together, as a block, and cannot be time-ordered, except if one interviews the agents on the order of the event: Did they change residence before the change in occupation, or vice versa? Was one the cause of the other?

To give a more detailed example, Mouchart and Vandresse (2010) faced the following issue when analyzing contracts in the freight sector. The data were made up of slightly more than 100 contracts providing the characteristics of each contract, such as distance covered, weight of the freight, urgency requirements, and price. These data were the results of negotiations between the provider and the user of the transport service, each contract being negotiated individually. The

absence of data on the sequence of the steps of the negotiation led these authors to conclude that it was not possible to operate a recursive decomposition and to disentangle the behaviors of supply and demand. Instead, they could only analyze the joint distribution of the data and, by means of a double frontier analysis, evaluate the imperfection of the market and the bargaining power of each agent for each contract.

As Cox (1992) has pointed out, and as discussed in Wunsch et al. (2010), time can be embedded in background knowledge, and in this case, the causal ordering of variables can approximately be determined. To give a trivial example, if one observes for a woman both a conception and a birth during the year, one knows that conception has preceded birth, and not the other way around, excluding the case where a birth is followed by another conception during the same time interval. The causal ordering can be determined here from other aspects of background knowledge, such as knowledge of relevant biological mechanisms, or other. Similarly, and more to the point, from cross-sectional survey data one can study fertility differentials by educational status, as one’s schooling is usually prior to one’s childbearing. Though observed as a block at a point in time, these variables can nevertheless be causally ordered on the basis of one’s knowledge of the field. To give another example, among smokers at a given moment, one should observe more persons with respiratory problems than among non-smokers, knowing that smoking usually precedes the disease and is a high risk factor for the latter. Once again, subject-matter knowledge may be used here to establish the presumed causal ordering of variables.

In cases like the study of contracts in the freight sector mentioned above, a way out is of course to improve data collection. For example, in the field of stock markets, high-frequency intraday data are now being collected by the BEDOFIH database in France (see the Eurofidai website). This presently enables research on trades and orders on a continuous basis, with microsecond accuracy (i.e. 0.000001 of a second). The time-ordered sequence of events can thus be established. However, as this is not always possible, appealing to all available background knowledge becomes essential.

In philosophy of science, recursivity is typically taken as part of the very definition of a causal model, so causes are assumed to occur prior to their effects. For instance, Clarke et al. (2014) examine recursive and non-recursive models of causal mechanisms and show that time-ordering the variables is necessary for taking causal cycles into account, such as feedback loops and reciprocal causation. On the other hand, a static equilibrium distribution or network, where the variables are not indexed by time, can be of interest, but then the relations among the variables have no causal interpretation.

Causal attribution is therefore impossible when information on the ordering of the variables is insufficient. This raises problems of identification. First, identification among models, meaning that two different models may be



observationally equivalent, should be clearly distinguished from parameter identification, that is, for a given parameterization of a given model two parameter values may be observationally equivalent (where observationally equivalent is taken in the sense of a same distribution). In both econometrics and social sciences, SEMs have been constructed and endowed with parametric restrictions, ensuring parametric identification without implying a recursive structure (see, for instance, Nagase and Kano, 2017). In these cases, putative causal relations are not uniquely identified; this would require another model, possibly observationally equivalent. Typically, such a new alternative model would necessitate further information on the DGP, either through additional empirical information or through supplementary contextual knowledge. In a nutshell, as Kahn and Whited (2017) have emphasized, identification is not causality, and vice versa.

## Discussion and conclusion

In a multidisciplinary perspective, this article considers a structural approach to causality which connects statistical modeling in the social sciences to the vast philosophical literature on causality, and more specifically on mechanisms. The version of structural modeling presented here is based on a concept of causality as a property defined within a statistical model. The latter is constructed according to explicit requirements of structurality, involving the decomposition of a complex DGP in terms of an ordered sequence of sub-mechanisms through a completely recursive decomposition of the joint distribution of the variables. In other words, the initial set of possible explanatory variables is analyzed through a sequence of sub-mechanisms leading to the event investigated. This decomposition is not always possible, either because it would be irrelevant (as in the 2C glue example) or because of an insufficiency of the available data, in which case a systematic analysis of causality, such as investigating mediation or moderator effects, becomes unfeasible.

This article explores how to proceed in cases where only a block-recursive, or partially recursive, decomposition of the joint process is available. In particular, it is shown that in many cases a preliminary, though imperfect, causal analysis can be performed on the basis of a detailed study of each context, including contingent conditions, and/or of possible additional evidence. Making causal analysis context-dependent implies developing an explicit specification of the population of reference, data permitting.

The population of reference is taken as the population exposed to the occurrence of the event being studied, in a specific context of time and space. For example, in the study of present-day fertility in Europe, the population of reference can be taken as the set of women at fertile ages, for example, 14–50 years. However, not all these women will give birth during the period considered and a structural model, or

mechanism, should be developed in order to understand why some do and others do not. The population of reference is thus progressively decomposed into smaller subsets exposed to risk, dependent on the sub-mechanisms that are proposed in the recursive decomposition. The individuals composing a population of reference are characterized by a common value of different characteristics and standing conditions, some being observed and some not, but all initially assumed to take part in the causal structure of the mechanism being investigated.

As an example of the specification of a population of reference, using a structural modeling approach, Gourbin et al. (2017) examine women's contraceptive use at fertile ages in urban Africa, the latter being restricted to the urban setting in the capital regions of Burkina Faso, Ghana, Morocco, and Senegal, as defined in the censuses, and the time period being that of *Demographic and Health Surveys* (DHS) carried out in these countries in the years 2003–2005. These specifications, and the thorough discussion in the article of the socio-cultural contexts, are essential for a proper understanding of the empirical results of this analysis. This work illustrates the fact that the recursive decomposition is crucially dependent on the interpretation given to each sub-mechanism, according to the context, and that the sub-mechanisms are specific to the population of reference.

In a block-recursive model, the above requirements must also be satisfied, as far as possible. In particular, the population of reference should be defined, the context studied, the congruence with background knowledge, and the stability of the results checked. The interpretation of the results should then be adjusted to take into account the incompleteness of the recursive decomposition. This issue is particularly important when information on the ordering of the variables is insufficient (section “Causal attribution when information on the ordering of the variables is insufficient”).

The topic of block-recursiveity also raises the problem of the closure of a model, that is, the question of how far should a model develop the issue of “the causes of the causes.” For example, when both smoking and exposure to asbestos are considered as two causes of lung cancer, a first issue is to consider the block “smoking and exposure to asbestos,” appraising how thoroughly one should try to understand the reasons of the interaction effect between the two causes. A second consideration is that studying the causes of these two causes can be an important factor in grasping the functioning of these causes.

The analysis of BRSs also illustrates the importance and usefulness of another classic notion of causal analysis: background conditions and INUS causes. While the literature on causality has thoroughly examined the status of cause (for instance, in probabilistic frameworks), much less has been said about the *conditions*. Our illustrative examples, as well the examples taken from the practice of demography, show that understanding the role of the conditions and specifying

the population of reference are of utmost importance. What is more, often these conditions are “packed” into blocks, which is why an analysis using appropriate structural models is required.

To conclude, structural modeling has already proven to be a successful method for causal attribution in several scientific domains and, in this article, has allowed pointing out the delicate issue of block-recursivity. However, much remains to be said about specific notions involved, and especially how these connect to concepts developed in the philosophy of causality.

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### References

- Aiken LS and West SG (1991) *Multiple Regression: Testing and Interpreting Interactions*. Thousand Oaks, CA: SAGE.
- Anderson J (1938) The problem of causality. *Australasian Journal of Psychology and Philosophy* 16(2): 127–142.
- Barndorff-Nielsen O (1978) *Information and Exponential Families in Statistical Theory*. New York: John Wiley.
- Baron RM and Kenny DA (1986) The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology* 51(6): 1173–1182.
- Clarke B, Leuridan B and Williamson J (2014) Modelling mechanisms with causal cycles. *Synthese* 191(8): 1651–1681.
- Coale AJ (1973) The demographic transition reconsidered. In: *IUSSP proceedings of the international population conference* (Vol.1), Ordina edn, Liège, pp. 53–72.
- Cox DR (1992) Causality: Some statistical aspects. *Journal of the Royal Statistical Society Series A* 155(2): 291–301.
- Demeulenaere P (ed.) (2011) *Analytical Sociology and Social Mechanisms*. Cambridge: Cambridge University Press.
- Ellett FS and Ericson DP (1983) The logic of causal methods in social sciences. *Synthese* 57(1): 67–82.
- Elwood JM (1992) *Causal Relationships in Medicine*. Oxford: Oxford University Press.
- Florens JP and Mouchart M (1985) Conditioning in dynamic models. *Journal of Time Series Analysis* 53(1): 15–35.
- Frost G, Darnton A and Harding AH (2011) The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971–2005). *Annals of Occupational Hygiene* 55(3): 239–247.
- Glennan S (2017) *The New Mechanical Philosophy*. Oxford: Oxford University Press.
- Glennan S and Illari P (2018) *The Routledge Handbook of Mechanisms and Mechanical Philosophy*. Oxford: Routledge.
- Gourbin C, Wunsch G, Moreau L, et al. (2017) Direct and indirect paths leading to contraceptive use in urban Africa: An application to Burkina Faso, Ghana, Morocco and Senegal. *Revue Quetelet/Quetelet Journal* 5(1): 33–70.
- Halbwachs F (1971) Causalité linéaire et causalité circulaire en physique. In: Piaget J (ed.) *Les théories de la causalité*. Paris: P.U.F, pp. 39–111.
- Illari P and Russo F (2014) *Causality: Philosophical Theory Meets Scientific Practice*. Oxford: Oxford University Press.
- Illari P and Williamson J (2012) What is a mechanism? Thinking about mechanisms across the sciences. *European Journal for Philosophy of Science* 2(1): 119–135.
- Kahn RJ and Whited TM (2017) Identification is not causality, and vice versa. Available at: <https://ssrn.com/abstract=3014878>
- Linton SL, Haley DF, Hunter-Jones J, et al. (2017) Social causation and neighborhood selection underlie associations of neighborhood factors with illicit drug-using social networks and illicit drug use among adults relocating from public housing. *Social Science & Medicine* 185: 81–90.
- Mackie JL (1965) Causes and conditions. *American Philosophical Quarterly* 2(4): 245–264.
- Mackie JL (1974) *The Cement of the Universe: A Study of Causation*. Oxford: Clarendon Press.
- Mieczkowski T and Beauregard E (2010) Lethal outcome in sexual assault events: A conjunctive analysis. *Justice Quarterly* 27(3): 332–361.
- Miethe TD, Hart TC and Regoeczi WC (2008) The conjunctive analysis of case configurations: An exploratory method for discrete multivariate analyses of crime data. *Journal of Quantitative Criminology* 4(2): 227–241.
- Mouchart M and Russo F (2011) Causal explanation: Recursive decompositions and mechanisms. In: Illari PM, Russo F and Williamson J (eds) *Causality in the Sciences*. Oxford: Oxford University Press, pp. 317–337.
- Mouchart M and Vandresse M (2010) A double-frontier approach for measuring market imperfection. *Special Issue of Annals of Operations Research on Efficiency and Productivity* 173(1): 137–144.
- Mouchart M, Russo F and Wunsch G (2009) Structural modelling, exogeneity, and causality. In: Engelhardt H, Kohler HP and Fürnkranz-Prskawetz A (eds) *Causal Analysis in Population Studies: Concepts, Methods, Applications*. Dordrecht: Springer, pp. 59–82.
- Mouchart M, Wunsch G and Russo F (2016) Controlling variables in social systems: A structural modelling approach. *Bulletin of Sociological Methodology* 132(1): 5–25.
- Nagase M and Kano Y (2017) Identifiability of nonrecursive structural equation models. *Statistics and Probability Letters* 122: 109–117.
- Pearl J (2000) *Causality: Models, Reasoning, and Inference*. Cambridge: Cambridge University Press.
- Preacher KJ, Rucker DD and Hayes AF (2007) Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research* 42(1): 185–227.

- Rehder B (2011) Reasoning with conjunctive causes. In: Carlson L, Hoelscher C and Shipley TF (eds) *Proceedings of the 33rd Annual Conference of the Cognitive Science Society*. Austin, TX: Cognitive Science Society, pp. 1406–1411.
- Rehder B (2015) The role of functional form in causal-based categorization. *Journal of Experimental Psychology: Learning, Memory, and Cognition* 41(3): 670–692.
- Reimers A, Brodtkorb E and Sabers A (2015) Interactions between hormonal contraception and antiepileptic drugs: Clinical and mechanistic considerations. *Seizure* 28: 66–70.
- Rothman KJ (1976) Causes. *American Journal of Epidemiology* 104(6): 587–592.
- Rothman KJ, Greenland S and Lash TL (2008) *Modern Epidemiology*. Riverwoods, IL: Wolters Kluwer; Lippincott Williams & Wilkins.
- Russo F (2009) *Causality and Causal Modelling in the Social Sciences: Measuring Variations* (Methodos Series, Vol. 5). Dordrecht: Springer.
- Russo F (2011) Correlational data, causal hypotheses, and validity. *Journal for General Philosophy of Science* 42(1): 85–107.
- Stock JH and Watson MW (2003) *Introduction to Econometrics*. Boston, MA: Addison-Wesley.
- Wermuth N (1992) On block-recursive linear regression equations (with discussion). *Revista Brasileira De Probabilidade E Estatística* 6: 1–56.
- Wunsch G, Mouchart M and Russo F (2014) Functions and mechanisms in structural-modelling explanations. *Journal for General Philosophy of Science* 45(1): 187–208.
- Wunsch G, Russo F and Mouchart M (2010) Do we necessarily need longitudinal data to infer causal relations? *Bulletin of Sociological Methodology* 106(1): 5–18.

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