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Abstract

Granger and Sims non-causality (GSNC) are compared to non-causality based on concepts popular in the microeconometrics and programme evaluation literature (potential outcome non-causality, PONC). GSNC is defined as a set of restrictions on joint distributions of random variables with observable sample counterparts, whereas PONC combines restrictions on partially unobservable variables (potential outcomes) with different identifying assumptions that relate potential to observable outcomes. Based on a dynamic model of potential outcomes, we find that in general neither of the concepts implies each other without further assumptions. However, identifying assumptions of the sequential selection on observable type provide the link between those concepts, such that GSNC implies PONC, and vice versa.

Keywords

Granger causality, Sims causality, Rubin causality, potential outcome model, dynamic treatments

JEL Classification

C21, C22, C23

1 Introduction*

One of the most important tasks of econometricians is to uncover causal relations between economic variables and distinguish them from associational relationship, also called spurious correlations. Only causal relations are useful for policy advice, because they contain the reaction of the economic variables of interest to policy interventions. In terms of the classical economic theorist like Marshall, or, more recently but in the same spirit, Hicks (1979), it is the effect of the ceteris paribus intervention that is of interest.[1] Econometrics developed two different ways to define what a causal effect is. One concept originated in time series econometrics. The other concept comes from the sphere of microeconometrics and statistics. Although both approaches are frequently applied in their subfields, it seems that they are not yet well understood.

The concept used in time series econometrics is due to Wiener (1956), Granger (1969) and Sims (1972) (e.g. see the review article by Geweke, 1984). Their basic idea is that (non-) causality is very similar, if not the same, than (non-) predictability. Therefore, they consider one variable not to cause another variable, if the current value of the causing variable does not help to predict future values of the variables that might capture the effects of this cause. This statement is conditional on the information set available at each point in time. This concept is in principle (technically) applicable if one cross-sectional unit (e.g. a country) is observed for a sufficiently long period.

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* I am affiliated with ZEW, Mannheim, CEPR and PSI, London, and IZA, Bonn. I am thankful to Jim Heckman for convincing me to write down some of the issues that appear in this paper. Of course, the usual disclaimer applies. I very much appreciate the previous joint work on dynamic potential outcome models with Ruth Miquel, in which we touched on a couple of issues that reappear here. The paper has been written while visiting the Economics Department of the University of Michigan. The hospitality is appreciated.

[1] See the excellent account of these writers and related historical developments in econometrics by Heckman (2000).

The alternative concept currently very popular in microeconometrics, particularly and most explicitly in the programme evaluation literature (e.g. Heckman, LaLonde, and Smith, 1999) is based on the idea that the relevant comparison is between different states of the world, each of which relates to a value of the causing variable. If causation is absent, then the outcomes that would have been realised if those potential states of the world had been true, would be the same. To relate this concept of different states of the world to data, it is necessary to observe different sample units in the different states. Then, so-called identifying assumptions are employed to relate the observed data to the distribution of the potential outcome variables, so that causal effects can be inferred from the 'real world' that is reflected in the data. The statistical formulation of the resulting inference problem is probably due to Neyman (1923) and was extended and popularized by Rubin (1974). Recently, dynamic versions of the potential outcome approach were suggested by Robins (1986) and Lechner and Miquel (2005). In principle, for this approach to be technically applicable there is no need to observe cross-sectional units over time as long as there is enough variation between units.

Apparently, there is nothing specific to those concepts so that they should just be used either in the domain of micro- or time series econometrics. They are based on different general principles that may be applied to all types of data. In particular, when the data have a time as well as a cross-sectional dimension both approaches may be applied. In this case, the dynamic approach to potential outcomes provides a useful framework to compare both concepts on an equal footing, because it addresses heterogeneity issues that are a key concern in the microeconometric literature as well as dynamics that feature most prominent in the time series econometrics

The literature appears to be almost silent on explicit comparisons of those concepts of causality. Heckman (2000) gives a historical account of causality in econometrics but does not attempt a formal comparison underlying both causality concepts. Holland (1986), in his overview of cau-

sality in different fields, briefly analyses Granger causality in a static model of potential outcomes and shows an equivalence of the two concepts under a randomisation condition. The exchange between Granger (1986) and Holland (1986), part of the discussion of that paper, does not really clarify the distinguishing features either.

The contribution of this paper is to fill that gap. We use the nonparametric dynamic model of potential outcomes to analyse the differences between Granger-Sims non-causality and non-causality defined by potential outcomes. We find that in general neither of the concepts implies each other without further assumptions. However, identifying assumptions of the sequential selection on observable type provide the link between those concepts. Once they are added, non-causality based on the Granger-Sims definition implies non-causality based on the dynamic potential outcome version, and vice versa. Thus, if such assumptions are valid, then tests for zero causal effects could be based on both approaches. Moreover, the results of those tests could be interpreted using the different intuitions that are behind the different concepts.

The paper proceeds as follows: Section 2 presents the concepts of (non-) causality based on observable variables. Section 3 presents the causal model based on potential outcomes in its dynamic form and discusses identification assumption. Section 4 relates those concepts to each other and Section 5 concludes.

2 Causality based on observable outcomes: Wiener-Granger-Sims non-causality

Let us define two stochastic processes $D=\{D_t\}$ and $Y=\{Y_t\}$ that may not necessarily be stationary. The data available consist of a random sample $(d_{0i}, d_{1i}, ..., d_{Ti}, \ y_{0i}, y_{1i}, ..., y_{Ti})$ coming from independent and identical draws (i=1, …, N) from the random variables within some time

window of those processes $(D_0, D_1, ..., D_T, Y_0, Y_1, ..., Y_T)$. The question is whether the factors described by *D* are causing changes in the variable *Y*. To set the terminology, we call *Y* the outcome variable (measuring the effect) and *D* the causing variable or treatment variable. The latter term is common in the biometric and econometric evaluation literature.

In its original article Granger (1969, p. 428) explains his concept of causation as "We say that D_t is causing Y_{t+1} if we are better able *to predict* Y_{t+1} using all available information than if the information apart from D_t had been used." (notation adjusted; italics added). He distinguishes between instantaneous causality, when the value of Y_{t+1} can better be predicted with the value of D_t given the history of D_t then without it. In a similar fashion, he considers the case when it takes some periods until the effect manifests itself in the outcome variables. With a similar concept in mind, Sims (1972, p. 545) explains that "… if causality runs from *D* to *Y* only, future values of *D* in the regression [of *Y* on *D* and perhaps other 'exogenous' variables] should have zero coefficients". Furthermore, they also held the view that a cause must precede any effect of it. Initially, the formalization of these concepts used linear predictors.[2] In this context, Hosoya (1977) showed the equivalence of those two concepts (see also Florens and Mouchart, 1985).

Chamberlain (1982), Florens and Mouchart (1982) and Engle, Hendry, and Richard (1983) strengthened them by basing the definitions on properties of conditional distribution functions instead. This has the virtue that the definitions become relevant for all type of economic variables, whether they are related by a linear conditional mean or not. In this paper, we adopt this specification as well. To condense notation the history from period 1 to *t* of *D* and *Y* are denoted by $\underline{D}_t = (D_1, ..., D_t)$ and $\underline{Y}_t = (Y_1, ..., Y_t)$. The initial conditions are collected in

[2] In those times, econometrics was almost entirely concerned with the estimation of linear relations of continuous variables.

$A_0 = (D_0, Y_0)$. Furthermore, letting small letters denote specific values of the random variables Definition 1 gives the technical concept of non-causality:

Definition 1 (GNSC: Granger-Sims non-causality):

$\underline{D}_t$ does not GS-cause Y_{t+1}, if and only if $Y_{t+1} \coprod \underline{D}_t \,|\underline{Y}_t = \underline{y}_t, A_0 = a_0; \quad \forall \underline{y}_t; \forall a_0; \forall t = 1,...,T-1.$ [3]

Note that we slightly deviate from the Chamberlain notation and condition directly on the random variables of the first period observed in the data (initial conditions), as in Engle, Hendry and Richard (1983).[4] The reason is notational simplicity in the comparison of the concepts of causality later on. Similarly, further delays of cause and effect can be introduced but are an unnecessary complication for the purpose of this paper.[5]

Sims (1972) proposed an alternative, but similar definition of non-causality, which in its independence version, proposed by Chamberlain (1982), is given by $(Y_T,...,Y_{t+1}) \coprod D_t \,|\, \underline{Y}_t$. It is direct to see that it is implied by Definition 1 (but not vice versa). Although, it has some intuitive appeal as absence of correlation of a current intervention and future outcomes given past outcomes, ambiguity about the causal meaning comes from not conditioning on the past interventions *D* (which is equivalent of assuming independence of D_t but not of $\underline{D}_t$). Whereas generally in this paper we focus on the (full) effect of *D* on *Y*, the Sims definition only seems to capture part of that, particularly so when the time horizon is finite, as will be assumed here. The lagged effects of the

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[3] $A \coprod (B_1, B_2) \,|\, C = c$ means that *A* and the elements of *B* are jointly independent conditional on *C* taking a value of *c* (i.e. Dawid, 1979). This statement is equivalent to $F(A, B_1, B_2 \,|\, C = c) = F(A \,|\, C)\, F(B_1, B_2 \,|\, C = c)$.

[4] Engle, Hendry and Richard (1983) discuss the related, but not identical concepts of strict exogeneity. Since their discussion focuses on likelihood functions and the role of their parameters in efficient and consistent estimation, it does not lend itself directly to the desired comparison of different concepts of causality.

[5] Dufour and Renault (1998) study long as distinguished from short run causality in a linear model by considering different lag lengths between the outcome variable and the causing and conditioning variables.

intervention may be 'absorbed' in the conditioning set.[6] Thus, for the sake of brevity, we do not consider the Sims (1972) version explicitly. Instead, we chose the name of Granger-Sims non-causality for the relation stated in Definition 1 to give credit to both 'inventors' of this type of causality.

Letting *F(.)* denote a distribution function and using short hand notation for the conditioning values, then Definition 1 is equivalent to $F(\underline{D}_t \mid Y_{t+1}, \underline{Y}_t, A_0) = F(\underline{D}_t \mid \underline{Y}_T, A_0) = F(\underline{D}_t \mid Y_t, A_0)$, i.e. the distribution of $\underline{D}_t$ and its elements does not depend on future outcomes conditional on the history of the process. Therefore, the joint distribution of all random variables has the following expression:

$$
\begin{aligned}
F(\underline{D}_T, \underline{Y}_T \mid A_0) &= F(\underline{D}_T \mid \underline{Y}_T, A_0) F(\underline{Y}_T \mid A_0) = \\
&= \prod_{t=1}^{T} F(D_t \mid \underline{D}_{t-1}, \underline{Y}_T, A_0) \quad \prod_{t=1}^{T} F(Y_t \mid \underline{Y}_{t-1}, A_0) = \\
&= \prod_{t=1}^{T} F(D_t \mid \underline{D}_{t-1}, \underline{Y}_t, A_0) \quad \prod_{t=1}^{T} F(Y_t \mid \underline{Y}_{t-1}, A_0).
\end{aligned}
$$

Furthermore, we have $F(Y_{t+1} \mid \underline{Y}_t, \underline{D}_t, A_0) = F(Y_{t+1} \mid \underline{Y}_t, A_0)$ for all *t*. These conditions have many obvious implications on sample counterparts, which can be used for testing them.

[6] Chamberlain (1982) suggests an alternative and stronger version of the Sims's definition, which leads to the equivalence with the Granger definition in this context. This equivalence holds, as long as all conditioning variables are treated symmetrically, i.e. as long as they can be subsumed in *Y*. Dufour and Tessier (1993) seem to be the first to note this non-equivalence between the Granger and Sims definition when additional 'control' variables are present which are influenced by *D* but are for some reason not included in *Y*. Their analysis is however confined to a linear projection framework. The formulation of this non-equivalence result in terms of independence is contained in Angrist and Kuersteiner (2004).

3 Causal effects defined by potential outcomes: Marshal-Neyman-Rubin causality

3.1 The concept of causality based on potential outcomes

The approach of potential outcomes has its roots in the idea that a causal effect is a reaction of an outcome variable to a manipulation of another variable keeping other factors constant. In economics, this classical ceteris paribus condition is the cornerstone of economics analysis.[7] The factors kept constant in such an intellectual exercise are typically those that are not influenced by the intervention but may influence the outcomes. Typically, this is *really* an intellectual exercise, i.e. a thought experiment, because it requires to imagine how the world would have developed had the specific intervention happened / or not happened. Therefore, additional conditions are required before the data can be used for resolving the causal question. The statistical formulation is probably due to Neyman (1923), Wilks (1932), Cochran and Chambers (1965), and has been highly popularized by the works of Rubin (1974, 1977, etc.; see also the non-technical overviews contained in Heckman, 2000, or Rubin, 2005).

To simplify notation, consider a discrete intervention changing the causing variable D from d to d'. d and d' differ at least once between 1 and T-1. We are interested in the question whether the outcomes would change due to a change in D. As before, we entertain the notion that the cause must precede its effect. To capture the notion of a c.p. change, we define the outcomes as functions of d as well as of other factors u and compare their difference for different values of d and the same value of u. We are interested in the contrast between $Y(d',u)$ and $Y(d,u)$. It has become

[7] See, for example, the classical works by Marshall (1961) and others, as discussed in the historical account of causal analysis by Heckman (2000), or the extensive discussion of ceteris paribus causality provided by Hicks (1979). Furthermore, Heckman (2005) provides an elaborate discussion of the potential outcome model and how it can be imbedded in economic theory.

common to be interested in differences of those *potential outcomes* (*Y(d',u)* and *Y(d,u)*), instead of other functions that may be more difficult to analyze.[8]

Let us define a causal effect of $\underline{D}_t$ on Y_{t+1} given initial conditions as $\theta_{t+1}(\underline{d}_t', \underline{d}_t, u_t) = F[Y_{t+1}(\underline{d}_t', u_t)] - F[Y_{t+1}(\underline{d}_t, u_t)]$. Note that this definition is based on a difference of distribution functions instead of the distribution of the differences of the potential outcomes as it would be common in that literature. The reasons are twofold: Firstly, the second concept is merely an illusion for any other measure than the sample mean for which the mean of the difference equals the difference of the means of the marginal distributions. It has (almost) never been applied in (non- or semiparametric) empirical studies. The reason is that there cannot be any information in the data useful for nonparametric estimation of the joint distribution of the potential outcomes, because no unit can be observed in both states at the same time.[9] Secondly, comparing marginal distributions of potential outcomes is more suited for a comparison with the concept of GSNC and does not distract attention to an issue irrelevant in econometric practice.

Assume that there is no data available on *u*. The case when some components of *u* are observable will be considered below. Therefore, only effects averaged over some population may be estimated from the data, like, $\theta_{t+1}(\underline{d}_t', \underline{d}_t, S_t) = \underset{u_t}{E}[\theta_{t+1}(\underline{d}_t', \underline{d}_t, u_t) \mid u_t \in S_t, A_0] = \underset{u_t}{E}\{F[Y_{t+1}(\underline{d}_t', u_t) \mid u_t \in S_t, A_0]\} - \underset{u_t}{E}\{F[Y_{t+1}(\underline{d}_t, u_t) \mid u_t \in S_t, A_0]\}$, where S_t denotes some population

[8] *Y(d',u)* and *Y(d,u)* are called potential outcomes, because the world cannot be in two different states at any time. Therefore, only *Y(d',u)* or *Y (d,u)* is observed if one of those two states is realised at all. For a fierce attack on such a concept of causality from the statistical side, see for example Dawid (2000). Despite that, this concept appears to be widely used in the sciences and economics, and at least in applied microeconometrics. For a further discussion, see the excellent exposition of the potential outcome approach by Holland (1986).

[9] For attempts to bound effects that are based on the joint distribution, see Heckman, Smith, and Clemens (1997). However, their bounds turn out to be that large as to be only of very limited, if any, relevance in empirical applications.

of interest defined by u_t.[10] There is an issue here whether non-causality should mean that the causal effect is zero for every value of u_t (i.e. $\theta_{t+1}(\underline{d}_t', \underline{d}_t, u_t) = 0$), or just on average for some population. The treatment effect literature places much emphasis on the fact that effects may differ in subpopulations defined by *D*. However, GSNC is formulated as a condition for the population as a whole, conditional on initial conditions. Therefore, we will only consider average effects for the population, denoted by $\theta_{t+1}(\underline{d}_t', \underline{d}_t) = 0$, to allow a comparison that focuses on the key components of different concepts of causality. This means that non-causality in all concepts allow for negative and positive effects at the disaggregated level as long as they wash out for the population. Finally, it should be pointed out that for notational simplicity, this notion suppresses the dependence of the effect on the initial conditions A_0.

Definition 2 (potential outcome non-causality, PONC):

$\underline{D}_t$ does not PO-cause Y_{t+1} if and only if $\theta_{t+1}(\underline{d}_t', \underline{d}_t) = 0, \quad \forall d_t' \neq d_t, \forall t = 1, ..., T-1.$

This notation adapts to Granger's convention with respect to timing of cause and effect. There is a major conceptional difference to the approach presented in the previous section, namely that in the potential outcome approach the definition of the effect and its discovery from the data are two

[10] u_t may contain past values of *u*, but this is suppressed for notational convenience. This notation covers all the usual causal effects that appear in the literature, like average treatment effects, average treatment effects on the treated, local average treatment effects, marginal treatment effects, quantile treatment effects, etc. For an overview of all the different effects discussed in the applied microeconometric literature and an attempt to put them in a unified framework, see Heckman and Vytlacil (2005). The emphasis on effect heterogeneity for different populations that appear in many applied studies based on the potential outcome approach does not appear prominently in GSNC. This is probably due to their different origins and fields of application. The potential outcome approach is used frequently in fields in which cross-sectional effect heterogeneity is considered important and the data have a large cross-sectional dimension. Granger-Sims non-causality originates from the time series literature, which historically is much less concerned with heterogeneity of causal effects and frequently has to rely on only one draw from the population of interest.

distinct steps that are considered separately Therefore, the quantity defined in Definition 2 cannot be empirically tested without further assumptions. The microeconometric literature has discussed numerous ways to identify these causal effects from the data when there are other variables available. To concentrate our analysis on the key conceptional differences between the two definitions of non-causality, we consider the case without further variables other than *D* and *Y*.

3.2 A form of potential outcome causality that can be inferred from the data

The first link of the observed outcome variables to the potential outcomes is the fact that potential outcomes are observed for the value of d_t that is realized in the data (d_{ti}). This is to say that the distribution of the observable outcome conditional on treatment is the same as the distribution of the potential outcome related to that treatment and conditional on it ($F[Y_{t+1} \mid \underline{D}_t = \underline{d}_t, A_0] = F[Y_{t+1}(\underline{d}_t) \mid \underline{D}_t = \underline{d}_t, A_0]$).[11] In the so-called treatment effect literature, this connection is rationalized by the so-called observation rule that can be stated as $Y_{t+1} = \sum_{\underline{d}_t} \underline{1}(\underline{D}_t = \underline{d}_t) Y_{t+1}(\underline{d}_t)$, where $\underline{1}(\cdot)$ denotes the indicator function which is one when the element in the brackets is true.

Even with the observation rule, we still cannot relate this concept of non-causality to data. For example, the observed variables can never uncover an effect like $F[Y_{t+1}(\underline{d}_t') \mid \underline{D}_t = \underline{d}_t, A_0] - F[Y_{t+1}(\underline{d}_t) \mid \underline{D}_t = \underline{d}_t, A_0]$. Although the second term in the difference relates to observables (because it concerns the population that is actually observed in that state, thus $F[Y_{t+1}(\underline{d}_t) \mid \underline{D}_t = \underline{d}_t, A_0] = F[Y_{t+1} \mid \underline{D}_t = \underline{d}_t, A_0]$), the first one does not. Therefore, assumptions are required to relate terms like $F[Y_{t+1}(\underline{d}_t') \mid \underline{D}_t = \underline{d}_t, A_0]$ to random variables for which realizations

[11] For the sake of a compact notation, the dependence of outcomes and treatments on u_t is left implicit for most of this and the following sections. In such cases, u_t is integrated out with respect to some distribution, which is obvious from the specific context.

can be found in the data, namely elements of $(\underline{Y}_T, \underline{D}_T, A_0)$. Robins (1986), and Lechner and Miquel (2005) analyzed such conditions in similar dynamic causal frameworks based on potential outcomes.[12] The former is more geared towards applications in epidemiology and contains assumptions, notation, and causal effects that are not commonly used in econometrics. Therefore, our considerations are based on a simplified version of the econometric dynamic treatment framework suggest by the latter authors.

Within that framework, we formulate conditions that allow to infer some of the $\theta_{t+1}(\underline{d}_t', \underline{d}_t)$ from the data. Without other data than the realizations from $(\underline{Y}_T, \underline{D}_T, A_0)$, the only way to achieve non-parametric point identification of an average causal effect is to assume randomization, i.e. whether unit '*i*' is observed or not, subject to regime *d* or *d'*, is random. We present three different types of such conditional and unconditional randomization assumptions:

Assumption 1 (Full independence assumption, FIA)

$$\underline{Y}_T(\underline{d}_{T-1}) \coprod \underline{D}_{T-1} | A_0 = a_0; \quad \forall \underline{d}_{T-1}; \forall a_0.$$

FIA implies the value of d_t, to which a specific unit is subject to in the next period, is random. Such an assumption would be valid in a classical experimental context, when the units are allocated randomly to different regimes defined by the different values of $\underline{d}_{T-1}$. Using one period only, this is exactly the type of assumption Holland (1986) used for showing equivalence of GSNC and PONC. However, his equivalence result does not necessarily hold in the dynamic context.

[12] These papers are based on so-called selection on observables assumption, which is the route followed below, although in a simplified way. Lechner (2004) proposes matching estimators and shows practical issues in Lechner (2006). Miquel (2002) considered the case of selection on unobservables that requires more data than just the outcomes and treatments. Abbring and Heckman (2005) provide a survey over dynamic causal models.

Though, it may be more plausible that units (economic agents, …) use the information about the past as given by ($\underline{D}_{t-1}, \underline{Y}_t$) to select the state D_t . This randomisation is conditional on the history of treatment and outcome variables. Thus, in period *t* different units of the population may have different probabilities to end up in d_t, depending on their past realisations of the outcome and treatment variables. This assumption is called the weak conditional independence assumption:

Assumption 2 (Weak dynamic conditional independence assumption, W-DCIA)

$$Y_{t+1}(d_t) \coprod D_1 \,|\, Y_1 = y_1, A_0 = a_0; \qquad \forall a_0; \forall \underline{d}_t; \forall y_1; \qquad \forall t = 1,...,T-1;$$
$$Y_{t+1}(\underline{d}_t) \coprod D_\tau \,|\, \underline{Y}_\tau = \underline{y}_\tau, \underline{D}_{\tau-1} = \underline{d}_{\tau-1}, A_0 = a_0; \quad \forall a_0; \forall \underline{d}_t; \forall \underline{y}_t; \forall \tau = 2,...,t; \quad \forall t = 2,...,T-1.$$

Lechner and Miquel (2005) show although population treatment effects are identified based on this assumption, classical treatment on the treated effects, i.e. the effects of the population of those units subject to a specific realisation of $\underline{D}_{T-1}$ are not identified. Thus, this assumption appears as a weak version that however suffices for the purpose of this paper, since any equivalence results that can be obtained under this assumption will also hold under assumption that nest W-DCIA.[13]

Two more assumptions are necessary to use the data together with W-DCIA or FIA to test PONC. Firstly, it is required that realisations of the outcome variables can actually be found for all paths of interest of $\underline{D}_{T-1}$. For W-DCIA, this so-called *common support assumption* must hold conditionally on past outcomes, for FIA it must hold unconditionally. Furthermore, for this notation to cover a ceteris paribus intervention, it is necessary to require that the potential outcomes for a specific state do not depend on the extent of the intervention. In other words, the

[13] To identify all usual treatment effects, Lechner and Miquel (2005) suggest a more restrictive version of the W-DCIA by imposing additional conditions on the way in which past treatments can influence past observed outcomes (strong dynamic conditional independence assumption, S-DCIA).

value of *Y(d,u)* does not depend on the fact that it is compared to *Y(d',u)* or to *Y(d'',u)*. This is called the stable unit treatment value assumption (SUTVA), see Rubin (1980). In what follows, it is understood that SUTVA and the common support assumption hold.[14] Otherwise, the interpretation of both concepts, PONC and GSNC, changes.

If FIA holds, then Property 1 shows how the causal effects can be recovered from the data.

Property 1 (Causal effects with potential outcomes based on FIA)

If FIA holds, the causal effects depend on $(D_0, D_1, ..., D_T, Y_0, Y_1, ..., Y_T)$ as follows:

$$\theta_{t+1}(\underline{d}_t', \underline{d}_t) = F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t', A_0 = a_0) - F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, A_0 = a_0);\ \forall \underline{d}_t, \underline{d}_t'; \forall a_0; \forall t.$$

The proof follows directly by combining FIA ($F[Y_{t+1}(\underline{d}_t) \mid \underline{D}_t = \underline{d}_t', A_0] = F[Y_{t+1}(\underline{d}_t) \mid \underline{D}_t = \underline{d}_t, A_0]$) with the observation rule ($F[Y_{t+1}(\underline{d}_t) \mid \underline{D}_t = \underline{d}_t, A_0] = F[Y_{t+1} \mid \underline{D}_t = \underline{d}_t, A_0]$).

Property 2 (Causal effects with potential outcomes based on W-DCIA)

If W-DCIA holds, the causal effects depend on $(D_0, D_1, ..., D_T, Y_0, Y_1, ..., Y_T)$ as follows:

$$F[Y_{t+1}(\underline{d}_t) \mid A_0 = a_0] = \underset{Y_1 \mid A_0}{E} \underbrace{\underset{Y_2 \mid D_1 = d_1, Y_1, A_0}{E} \cdots \underset{Y_t \mid \underline{D}_{t-1} = \underline{d}_{t-1}, \underline{Y}_{t-1}, A_0}{E}}_{w(\underline{d}_t)} F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, \underline{Y}_t = \underline{y}_t, A_0 = a_0);\ \forall \underline{d}_t; \forall a_0; \forall t.$$

$$\theta_{t+1}(\underline{d}_t', \underline{d}_t) = \underset{Y_1 \mid A_0}{E}[w(\underline{d}_t') F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t', \underline{Y}_t, A_0) - w(\underline{d}_t) F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, \underline{Y}_t, A_0)];\ \forall \underline{d}_t, \underline{d}_t'; \forall a_0; \forall t.$$

The proofs of these properties follow directly from the identification proofs of Lechner and Miquel (2005) and are not repeated here.

[14] An example where SUTVA is violated would be the introduction of a large training programme changing the wages of nonparticipants as well as influencing demand and supply in the labour market. In this case, data of nonparticipants drawn from a world in which these programmes exist cannot proxy data from nonparticipants in a

As can be seen from Property 2, identification is achieved by continuously reweighting the units that receive d_t towards the distributions of characteristics that describes the population of interest. By doing so, the expanding number of conditioning variables and time order of variables is respected.

4 Relation between the different concepts

4.1 General results

Note that Definition 1 summarizes the conditions that GSNC imposes on the data. Definition 2 defines PONC. Properties 1 and 2 define how the PO-causal effects depend on the data if either of the 'identifying' Assumptions 1 or 2 holds. Hence, if GSNC together with those properties imply a zero causal effect ($\theta_{t+1}(\underline{d}_t', \underline{d}_t) = 0; \forall \underline{d}_t, \underline{d}_t'; \forall t$), we conclude that GSNC together with the respective Assumption 1 or 2 implies PONC. Conversely, if the restrictions $\theta_{t+1}(\underline{d}_t', \underline{d}_t) = 0; \forall \underline{d}_t, \underline{d}_t'; \forall t$ imposed on Properties 1 or 2 imply the restrictions of Definition 1, then we conclude that the combination of these assumptions with PONC implies GSNC.

However, before considering the combinations of identifying assumptions with causality definitions, we state the obvious in Theorem 1:

Theorem 1 (GSNC and PONC only)

a) GSNC does not imply PONC.

b) PONC does not imply GSNC.

world where this programme does not exist. Clearly, GSNC and PONC would fail to uncover the 'real' causal effect without further assumptions.

This theorem is true, because PONC, without further assumptions, does not impose any restrictions on the distribution of $(D_0, D_1, ..., D_T, Y_0, Y_1, ..., Y_T)$ that are of relevance to GSNC.

This result may seem trivial. However, it points directly to the important fact that ceteris paribus interventions, which are directly reflected in models based on contrasts of outcomes in two different states of the world have no consequences for the data, if not *enriched* with further (*untestable*) assumptions. In other words, any restrictions put on the data (in the form of testable hypothesis) are silent about underlying causal effects that generated the data if no further untestable assumptions can be added to relate the potential worlds that are required to define effects of c.p. interventions to the data.

Next, consider the experimental assumption (FIA), stating that potential outcomes and causing / treatment variables are independent. Again, we only get negative results in Theorem 2.

Theorem 2 (GSNC and PONC combined with FIA)

Suppose Assumption 1 (FIA) holds and there is common support.

a) GSNC does not imply PONC.

b) PONC does not imply GSNC.

The reason for this non-equivalence is the fact that conditional independence does not imply unconditional independence and unconditional independence does not imply conditional independence. This is the so-called Simpson (1951) paradox. The Simpson paradox implies that all correlations between two random variables may change when conditioned on further variables.

More technically, combining Property 1 with Assumption 1 we see directly that PONC and FIA together imply that $F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t', A_0 = a_0) = F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, A_0 = a_0) = F(Y_{t+1} \mid A_0 = a_0)$, which

is equivalent to $Y_{t+1} \coprod \underline{D}_t \mid A_0$. This condition does not imply $Y_{t+1} \coprod \underline{D}_t \mid \underline{Y}_t, A_0$ (GSNC). Since the converse does not hold either, both parts of Theorem 1 hold.

Theorem 3 shows that the sequential randomisation assumption W-DCIA takes care of the problem arising form Simpson's paradox by conditioning on the lagged outcome variables and thus provides the following equivalence results for the different concepts of causality.

Theorem 3 (GSNC and PONC combined with W-DCIA)

Suppose Assumption 2 (W-DCIA) holds and there is common support.

a) GSNC implies PONC.

b) PONC implies GSNC.

GSNC implies that the distribution of Y_{t+1} given past outcomes does not depend on any of the past D_t, $F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, \underline{Y}_t, A_0) = F(Y_{t+1} \mid \underline{Y}_t, A_0)$. This condition leads to an equality of the inner terms of the causal effects given in Property 3, i.e. $F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t', \underline{Y}_t, A_0) = F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, \underline{Y}_t, A_0)$. Furthermore, because this equality holds for all values of *t*, the weights are identical as well ($w(\underline{d}_t) = w(\underline{d}_t')$). Therefore, GSNC implies PONC if W-DCIA holds.

To show that PONC implies GSNC, it is important to note that W-DCIA comes with an initial condition, i.e. the problem of the first period is essentially static:

$$\theta_2(d_1', d_1) = \underset{Y_1 \mid A_0}{E}[F(Y_2 \mid D_1 = d_1', Y_1, A_0) - F(Y_2 \mid D_1 = d_1, Y_1, A_0)] \overset{!}{=} 0 .$$

Assuming that $F(Y_1 \mid A_0)$ is nonzero in the support of interest (as ensured by the common support assumption), then it must be true that $F(Y_2 \mid D_1 = d_1', Y_1, A_0) = F(Y_2 \mid D_1 = d_1, Y_1, A_0)$. This

however has implication for the causal effect of the next period. Consider the zero causal effect for period 3:

$$\theta_2(\underline{d}_2', \underline{d}_2) = \underset{Y_1|A_0}{E}[\underbrace{\underset{Y_2|D_1=d_1', Y_1, A_0}{E}}_{w(\underline{d}_2')} F(Y_3 \mid \underline{D}_2 = \underline{d}_2', \underline{Y}_2, A_0) - \underbrace{\underset{Y_2|D_1=d_1, Y_1, A_0}{E}}_{w(\underline{d}_2)} F(Y_3 \mid \underline{D}_2 = \underline{d}_2, \underline{Y}_2, A_0)] \overset{!}{=} 0.$$

However, because the zero causal effect from the previous period leads to $F(Y_2 \mid D_1 = d_1', Y_1, A_0) = F(Y_2 \mid D_1 = d_1, Y_1, A_0)$, the weights appearing in the difference are the same ($w(\underline{d}_2') = w(\underline{d}_2)$). However, with nonzero weights guaranteed by common support, this condition on the weights implied by PONC and W-DCIA requires that $F(Y_3 \mid \underline{D}_2 = \underline{d}_2', \underline{Y}_2, A_0) - F(Y_3 \mid \underline{D}_2 = \underline{d}_2, \underline{Y}_2, A_0) = 0$, which in turn implies that the weights for the next period are equal as well ($w(\underline{d}_3') = w(\underline{d}_3)$). Applying this reasoning to every period up to period *T*, it follows that PONC in combination with W-DCIA implies $F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t', \underline{Y}_t, A_0) = F(Y_{t+1} \mid \underline{D}_t = \underline{d}_t, \underline{Y}_t, A_0)$, which is exactly the condition for GSNC. Note that conditioning on some initial conditions as well as definition of zero effects in all periods, plays a key role in this proof.

4.2 Further issues and generalisations

This section takes up some issues that relate to simplifications chosen in this paper with the purpose of clarifying the main differences between the different approaches.

The first such issue relates to additional variables that could be used to condition on. All results hold in any subset defined by variables that are not influenced by treatment variables. Lechner and Miquel (2005) provide the necessary identification results when predetermined variables are added to Assumption 2.

Another interesting type of data that might become available would be instrumental variables, i.e. variables that influence D but do not influence Y other than by changing D. In a world of heterogeneous causal effects that underlies this paper, such variables identify treatment effects for a subpopulation that react to changes in the instruments by changes in D, the so-called compliers (Imbens, Angrist, 1994). Which member of the population belongs to that group can however not be identified. Thus, since GSNC cannot be defined for such an unobservable subpopulation, there is not much sense in comparing GSNC and PONC for that group.

In the comparison between GSNC and PONC, this paper considered PONC for the population instead of subpopulations for the treated. If the latter are explicitly taken into account, then for those effects that are actually identified, the results by Lechner and Miquel (2005) show that the structure of the key elements in the comparison, Properties 1 and 2, remain.

5 Conclusion

The paper highlights the problem to uncover the effects of ceteris paribus interventions with econometric methods. For a long time now, ceteris paribus interventions are typically thought of by economic theorists (like Marshall and Hicks as examples) as comparisons of different states of the world that could have occurred. The paper shows among that Granger-Sims non-causality under some conditions indeed detects the absence of such an effect.

More precisely, we use the dynamic model of potential outcomes for analysing the differences between Granger-Sims non-causality and non-causality defined by potential outcomes for different identifying assumptions. We find that in general neither of these concepts implies each other without further assumptions. However, identifying assumption of the sequential selection on observable type provide the link between those concepts. Once added, non-causality based on the Granger-Sims definition implies non-causality based on the dynamic potential outcome

definition, and vice versa. Thus, if such untestable assumptions are plausible, then tests for zero causal effects could be based on both approaches. Moreover, the results of those tests could be interpreted using the different intuitions that are behind the different concepts.

It is worthwhile noting that our findings are unrelated to the main criticism of the Granger-Sims approach that appeared in Holland (1986) as well as in other papers. The issue is that the availability of new data should lead to additional variables entering the information set. This in turn leads implicitly to a new definition of Granger-Sims non-causality. In other words, knowing more may lead to the result that a variable previously considered a cause becomes a spurious relation. The potential outcome approach in comparison seems somehow immune to that problem, because the identification steps are separated from the estimation steps and the available data. However, the comparison is probably not entirely fair, because in the empirical practice, having new data leads many researchers to change their identifying assumptions by increasing the set of conditioning variables required for the DCIA assumptions to hold, and thus the same phenomena as for Granger-Sims-non-causality may appear.

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