Why do social scientists disagree about the right way to do causal inference?

Reading guide: *The last **ten** pages contain the bibliography and a technical appendix. Both of these can be skipped when reading.*

Abstract

There are irreconcilable differences between two main approaches to causal inference in social science: Rubin's Causal Model (RCM) and Pearl's Causal Model (PCM). In this paper, I argue that these methodological differences can be largely explained by their different metaphysics of causation. Specifically, I show that the biggest methodological point of contention between RCM and PCM concerns whether we can estimate individual causal effects in actual causation causes. Building on this insight, I show that both RCM and PCM reduce causation to structural equations. However, on RCM these equations are *different* across individuals whereas on PCM they are the *same*. I then suggest how this seemingly small difference can explain most methodological differences between RCM and PCM.

I Introduction

[1] There is a fierce debate about how to conduct causal inference in social science (Pearl, 2012; Imbens, 2020). On one side are the advocates of Pearl's Causal Model (PCM) who use graphs to represent the causal links between different variables. On the other side are advocates of Rubin's Causal Model (RCM) who examine real-world cases which approximate controlled experiments. So, why do they disagree about causal inference? I answer this question in two steps. First, I show that there are irreconceivable methodological differences between PCM and RCM. Second, I argue that these differences can be attributed to the fact that they assume two slightly different metaphysics of causation.

[1] *Things that need to be done: 1) Put graph terminology in appendix (if we don't use the language too much); 2) Correct response to interventionist theory and RCM: Hu (2022) or Reiss (2009) should have a response; 3) Papers to read: Hitchock (2007) Prevention, Preemption, and the Principle of Sufficient Reason and Markus (2021); , 4) Check if the last part is readable on its own (for LSE), 5) Much more discussion is needed about the randomness example, e.g., does causal sufficiency holds, how does Naftali W's objection to one-to-one comparisons relates to it, a Naftali W objection based on email etc; 6) Merge the two sections about the objections into one: should save a lot of space; 7) Use of word identification: maybe I actually need to define it; 8) Might need to limit notation in the last part, e.g., I removed the j notation in PCM's representation but maybe I can drop the model vs mechanism stuff too*

PCM advocates disagree with RCM advocates about many things and usually have a good response from their own standpoint to any objections, and *vice versa*. When PCM advocates claim that the two frameworks are mathematically equivalent but PCM provides a richer framework for representing causal links, RCM advocates can reject the axioms of the equivalence proof. I will show that most such methodological disagreements reduce to a disagreements about what I call the *heterogeneity problem*. This refers to our ability to estimate individual causal effects in cases of actual causation. PCM advoctes believe that this is possible which RCM deny. Despite the importance of the *heterogeneity problem*, the disagreements do not seem resolvable on purely methodological grounds.

Should we give up on resolving the debate? Luckily, the metaphysics of causation can tell us not only why such methodological differences exist but also why PCM and RCM work in practice. Drawing on Papineau (2021), I suggest reducing causation to a system of structural equations, i.e., the structural equations theory of causation. Papineau argues that his version of the theory is sufficient to explain why PCM works. I show that this is not always true, given the functional forms which he assumes on the structural equations underlying his theory.

For this reason, I modify his theory in two ways. First, I propose a restricted set of alternative functional forms that are sufficient to do the job. Second, I suggest to change the theory, so that it also explain why **R**CM works. I then argue that RCM and PCM rely on two slightly different versions of the theory: on PCM the structural equations are the *same* for all people whereas on RCM they *vary* across individuals. This feature allows me to explain how RCM and PCM understand heterogeneity and, consequently, many other contentious methodological points between PCM and RCM.

To support this argument, I begin by describing PCM (Section II) and RCM (Section III). Next, I discuss their methodological differences by studying the arguments of PCM advocates against RCM (Section IV) and analogically the arguments of RCM-sympathetic economists against PCM (Section V). I then show why the traditional metaphysical theories of causation cannot justify why RCM and PCM work (Section VI). Last, I outline the structural equations theory of causation and suggest how it can explain the methodological differences (Section VII).

II Pearl's Causal Model

II.a Set-up

How does PCM-based causal inference works? Let us consider a simple example before discussing the more fundamental assumptions of PCM. Suppose that we are interested in the effects of neighbourhoods *early* in life (N), i.e., the area in which a person was brought up, on income *later* in life (I).[2] Different authors have stressed different channels for neighbourhood effects (Chyn and Katz, 2021). Based on such research, we can argue that neighbourhood effects on income are fully mediated via education (E) and health (H), so that neighbourhoods have no independent effect on income.[3] Figure 1 summarises these considerations. Given some additional assumptions discussed above, we can interpret the graph as causal.

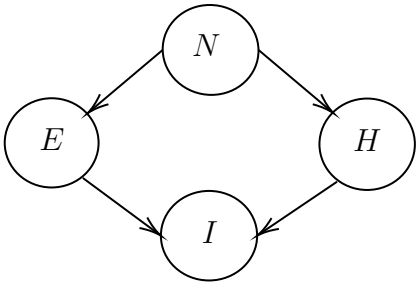

Figure 1: Neighbourhood effects on income

Before proceeding, it is useful to introduce some graph theory terminology. Graphs in this paper represent how a set of variables is connected. Each node represents a different variable, e.g., $\{N, E, H, I\}$ in Figure 1. Nodes are connected by *edges*. An edge is *directed* if it points from one node to another node. For any directed edge $N \to E$, we say that N is a *parent* of E and E is a *child* of N. More generally, all the predecessors of a node E, including its parents, are its *ancestors* whereas all its successors, including its children, are its *descendants*. Any collection of one or more consecutive edges is called a *path* where a *directed* path is a path containing edges directed only in one direction. A graph is *acyclic* if there is no directed path which starts from node N and ends in the same node, e.g., $N \to E \to I \to N$. These definitions

[2]Neighbourhoods are a very popular topic in social science (Sampson et al., 2002; Chyn and Katz, 2021). There is also substantial evidence for their importance (Chetty et al., 2016).

[3]Robert (1999) established $N \to H$, Kauppinen (2008) found $N \to E$, Card (1999) discussed $E \to I$ and Baicker and Finkelstein (2011) suggested that health insurance increases financial stability ($H \to I$). Other potential mediators, e.g., crime, are omitted for brevity.

are important since Pearl is largely interested in Directed Acyclic Graphs (DAGs) which is a necessary assumption in many of his theorems.

Without additional assumptions, DAGs do not necessarily have a causal interpretation: they could simply be summarising a probability distribution in a succinct way as in non-causal Bayesian nets. How can we then give Figure 1 a causal reading? While there are other options,[4] the approach favoured by Pearl assumes the existence of a system of structural equations, in which the variables on the right-hand side directly determine the variable on the left-hand side:

$$y_k = f_k(pa_k, u_k)$$

where pa_k is a vector with the observed parents of y_k, k refers to the k-th equation in our system, u_k is an probabilistically independent error term capturing unobserved factors and $f_k(.)$ is some unspecified functional form. Crucially, while pa_k affect y_k deterministically, probability enters y_k via the errors u_k. In our example about neighbourhoods, the complete system is:

$$Education = f_E(Neighborhood, u_E) \tag{1}$$

$$Health = f_H(Neighborhood, u_H) \tag{2}$$

$$Income = f_I(Education, Health, u_I) \tag{3}$$

where u-s are independent errors. Once we have our structural equations and give them a causal reading, they can be represented in a DAG.

II.b Causal inference

We have seen Pearl's view of the theory underlying the DAGs. However, this does not tell us how to use PCM for causal inference. Here I focus on one aspect of causal inference: causal discovery, i.e., how to uncover the true causal structure from data.[5] To conduct causal discovery, we need to make certain assumptions about the true causal model. Studying such assumptions will allow

[4]The main alternative assumes probabilistic causation and provides conditions, under which DAGs can be read as *causal* Bayesian nets (Spohn, 2009). Pearl argues against this approach in Chapter 1 of his book (2009).

[5]Another use of PCM in causal inference is *causal effects identification*. This tells us if we can estimate a particular causal effect given our graph. I do not discuss this topic because identification methods are valid only if the underlying DAG captures genuine causal relations, i.e., discovery seems more important than identification.

us to make claims of the type: 'Suppose we have a set of correlations. If assumptions 1), 2), etc hold in the true causal model, then we can use our correlations to discover the true model'. In other words, we assume that there exists some unobservable underlying causal structure. Using our evidence, we formulate a structural model which should approximate the true causal structure. We then need to make certain assumptions on the true causal structure. Otherwise, we cannot be sure that our hypothesised structural model is truly approximating the true causal structure. The trouble is that many such assumptions are empirically unverifiable which makes causal inference difficult.

We can illustrate this idea by examining two common assumptions made on the true causal structure: *faithfulness* and *causal sufficiency*. First, *faithfulness* states that the absence of a causal link[6] implies the absence of a correlation (Zhang and Spirtes, 2016, p.1114). If we find no correlation between two variables (potentially after conditioning), they are not causally linked directly. There are various counterexamples to faithfulness, in which two variables have zero correlation but are actually causally linked (Andersen, 2013; Papineau, 2021). Here I provide a verbal version of such a counterexample but Appendix A.a provides the graphical equivalent.

Suppose we are interested in the gender wage gap and find that conditional on occupation there is zero correlation between gender and income. This is plausible given evidence that women tend to choose lower paid occupations (Blau and Kahn, 2017). Does that mean that gender has no causal effect on income outside of occupation? Not necessarily. Gender might still affect earnings through two separate paths which exactly offset each other, leading to a zero correlation. For example, note that women are more likely to attend university (Goldin et al., 2006), and that education increases earnings (Card, 1999). So, gender has a *positive* effect on income via education. At the same time, there might still be gender differences in wage negotiating ability (Card et al., 2016). As a result, gender may have a direct *negative* effect on income which *exactly* cancels out the *positive* effect mediated by education. This is a faithfulness failure: gender and income are conditionally uncorrelated, albeit gender still affects income. We can now see why faithfulness is problematic (aside from being unverifiable[7]). If it fails in the

[6]Two variables V and W are causally linked if V (in)directly causes W or if W (in)directly causes V or if they have a (in)direct common cause U (Papineau, 2021, p.8).

[7]One might respond that faithfulness still has testable implications (Zhang and Spirtes, 2008). However, the fact remains that the assumption cannot be directly verified.

true model, any *hypothesised* causal links which assume it might not be really causal.

The next assumption is *causal sufficiency* which allows us to rule out the effects of unobserved causes (Scheines, 1997; Papineau, 2021). If causal sufficiency holds, then we can never find a common cause of both variables in any of the hypothesised causal links in our model. The assumption does *not* require the inclusion of all variables affecting *the main variables of interest* but only variables affecting *both variables in a hypothesised causal links*. In Figure 1, we do *not* have to include gender if it affects income directly but is not a common cause in any of the hypothesised causal link. We do need to include parental income if it affects both neighbourhoods and income. If causal sufficiency is incorrectly assumed for a causal link, this link will not be truly causal. While there are discovery algorithms for datasets that do not satisfy causal sufficiency (Spirtes et al., 2009, Chapter 6), they are much less helpful, e.g., their output is not necessarily a DAG (Pearl, 2009, §2.6.). However, it might be that faithfulness and causal sufficiency actually hold in the true model.[8] In that case, there is a plethora of algorithms which are guaranteed to discover the true structure, e.g., Pearl (2009, p.50).

III Rubin's Causal Model

The basic primitive in RCM are the *potential outcomes* (POs) (Holland, 1986; Imbens and Rubin, 2015). Drawing on randomised control trials, POs are usually defined relative to a binary *treatment*. More formally, the POs for unit j are:

$$Y_{j,t}(D=1) \quad and \quad Y_{j,t}(D=0)$$

where D indicates if j got the treatment. In the example from Figure 1, the treatment could be living in a good relative to a bad neighbourhood whereas $Y_{j,t}$ is j's income. We also assume that j's POs at time t are not the same as j's POs at time s (Imbens and Rubin, 2015, p.8). Intuitively, this is because the effect of an Aperol Spritz on my happiness in the *morning* is not the same as its effect in the *evening* after a long day of work.

[8] Technically, we also need the *Causal Markov Condition* and *minimality*. See Stern and Eva (2022) for details on these assumptions.

A crucial feature of POs in RCM is that treatment D can only be a variable, which we can imagine manipulating in a hypothetical experiment (Imbens and Rubin, 2015, p.21). There is 'no causation without manipulation' (Holland, 1986). This rules out studying treatments such as race because we cannot imagine designing an experiment in which we change somebody's race. While this is partly a matter of degree, it is much easier to imagine giving a person an aspirin than changing their race. RCM also allows interventions that influence race perception, so long as they are easy to implement (e.g. see Bertrand and Mullainathan, 2004). In any case, only variables which we can imagine manipulating in a hypothetical albeit realistic experiment can be causes (Holland, 1986, §7).

If we decide to use POs directly to estimate individual causal effects, we will face the *fundamental problem of causal inference*. For each j, we observe either $Y_{j,t}(D = 1)$ or $Y_{j,t}(D = 0)$ but not both. So, we cannot calculate the *individual* treatment effects. Luckily, in randomised control trials, we can get an unbiased estimate of the *average* treatment effect, e.g., Theorem 6.1 in Imbens and Rubin (2015). This quantity is still useful as it tells us how people in our sample respond on average to the treatment.

Very often, however, we cannot conduct such experiments (for ethical or legal reasons) and need to rely on observational studies. It is, therefore, important to study the assumptions required for unbiasedness. This will allow us to check if they hold in a particular observational study and if we can use it to learn a causal effect. The stable unit treatment value assumption (SUTVA) is one such assumption (Imbens and Rubin, 2015, pp.9-13). One part of SUTVA requires that whether j is assigned treatment should have no bearing on whether i is assigned treatment, i.e., j's treatment should be independent from i's treatment.[9] While this assumption is not necessary for treatment effect estimation,[10] it is often imposed due to data limitations, similarly to faithfulness in PCM.

Other important assumptions also concern the treatment assignment mechanism. Specifically, the assignment mechanism in randomised control trials in contrast to observational study is controlled. This ensures that treatment is genuinely allocated at random, so that we can

[9] I do not discuss SUTVA's homogeneous-treatment part because of space constraints. See Imbens and Rubin (2015, pp.9-13) for details.

[10] A burgeoning literature is designing RCM method for situations when SUTVA fails (DiTraglia et al., 2020).

get an unbiased estimate of the average treatment effect. In observational studies without randomisation, we can only get the causal effect if we make some additional assumptions on the assignment mechanism. One way to proceed is to assume selection (into treatment) on unobservables (Cerulli, 2015).[11] On this assumption, treatment assignment depends not just on observable characteristics but also on unobservable ones. So, we cannot use standard techniques such as linear regression to estimate the causal effect, as we will run into omitted variable bias.

In response to this problem, methodologists have developed techniques such as instrumental variables, difference-in-difference and synthetic control methods that work under selection on unobservables (Athey and Imbens, 2017). While such techniques require additional assumptions, they show that RCM causal inference can work even if we do not observe all variables that affect both treatment and outcome. This key feature also explains how Chetty and Hendren (2018) can estimate neighbourhood effects when treatment is not randomly assigned.

IV PCM objections to RCM

We have now introduced two seemingly different approaches towards causal inference. There is a lively debate between their advocates (Pearl, 2012; Gelman, 2009; Imbens, 2020). In Sections IV and V, I contribute to a recent literature in philosophy of science (Markus, 2021; Weinberger, 2022) that tries to uncover the main points of contention in this debate. Here I respond to two objections raised by PCM advocates against RCM: (i) while PCM and mathematically equivalent to RCM, PCM is a better analytical framework and (ii) RCM tell us nothing about mechanisms.

IV.a Pearl's equivalence claim

Pearl claims to have proved the mathematical equivalence of RCM and PCM: any theorem in RCM has an equivalent theorem in PCM (Pearl 2012; Pearl, 2009, Chapter 7; Galles and Pearl, 1998; Glymour et al., 2016, p.126; Gelman, 2009). However, RCM prevents us from utilising the 'inferential and representational powers of diagrams' (Pearl, 2012). Consequently, PCM provides us with a richer framework to represent most problems encountered in causal inference and so

[11] The main alternative is selection on observables which requires that treatment assignment is fully determined by observable variables. Unfortunately, this assumption is known to be almost always false (LaLonde, 1986).

we should abandon RCM.

To make the equivalence proofs, Pearl outlines all assumptions embedded in his definition of a structural model, among which is the existence of POs (Pearl, 2009, Definition 7.1.4). Next, he presents a set of three axioms which are necessary and sufficient for deriving the definition of a structural model (Theorems 7.3.3 and 7.3.5). In other words, all properties which Pearl includes in his definition of a structural model (including POs) can be inferred from the axioms. So, the three basic axioms are the building blocks for any more complex statements made within structural models: any claim involving POs can be reduced to a claim expressed in the axioms. This entails that any result which Rubin derives using POs can be translated into PCM.

While I do not doubt Pearl's mathematical derivation, I do doubt whether Rubin will accept his definition of POs. The reason is that it implies that the *same* structural model applies to all units. Consider Pearl's definition of POs:

$$Y(do(D = 1), M(b)) \tag{4}$$

where Y is the outcome of interest, D is a binary treatment indicator, b are background factors and M is a model which connects the variables of interest. The do-operator $do(.)$ provides rules which we can use to calculate the effect of specific interventions within M, e.g., setting $D = 1$.[12] For Pearl, POs are inseparable from the model M linking b.

This feature of POs within PCM is important, since it suggests that causal effects will be the same for two people with the same b. It is also reflected in one of Pearl's axioms: the axiom of composition (Pearl, 2009, p.229).[13] It implies that if two people share the same characteristics b in model M, they will have the same POs once we intervene to $D = 1$ where these predicted POs will be the same as the actual POs. This means that $D = 1$ will have exactly the same effect on their POs. For example, if two people share the same education and health in Figure 1, changing their neighbourhood will have exactly the same effect on their incomes.

Let us now see how POs in RCM compare to POs in PCM. If we stick to do-calculus, RCM

[12] I will not discuss $do(.)$ in detail, since it helps causal identification, not discovery.

[13] More formally, composition states that the only variable changed *directly* by the intervention is the treatment indicator, given M. The descendants of the treatment indicator change but only *indirectly* as a result of the intervention.

POs are given by $Y_j(do(D=1))$ where j denotes the unit of observation (Imbens and Rubin, 2015) and where I drop time indexing for brevity. In this definition, Rubin allows POs to differ across people, independently of the underlying structural model, so it is *not* always true that:

$$\underbrace{Y(do(D=1), M(b_j))}_{POs\ in\ PCM} = \underbrace{Y_j(do(D=1))}_{POs\ in\ RCM}$$

Even if i and j share the same characteristics ($b_i = b_j$), they might not share the same M on RCM. So, the difference between Rubin and Pearl boils down to a disagreement about how POs are generated across observationally equivalent people. For Pearl, the variation between individuals can be fully captured by the variables in M whereas for Rubin this might not be the case. Rubin will reject the equivalence claims not simply because Pearl defines POs within a model but because the model imposes the same POs on two people with the same b and, therefore, the same causal effect. This distinction matters since Markus (2021) suggests that defining POs within a model is the issue which fails to capture the essence of Rubin's response.

This reasoning somewhat simplifies things. Both Pearl and Rubin allow for the effect of random shocks to affect POs. For Pearl, probabilistically independent random disturbances are allowed to affect POs independently of the other causes. However, for Rubin, they can affect both the POs *and* the causal effect of the treatment. Even if the POs for i and j are affected by equal random shocks, the causal effect might still be different for Rubin: there might be an additional random shock in RCM, affecting just the causal effect itself. This point relates closely to the *heterogeneity problem* about whether we can estimate causal effects in actual causation cases which is crucial for understanding the methodological debate.

IV.b Mechanisms

Pearl's equivalence claims fails and so does his argument for the superiority of PCM. However, PCM advocates may raise a familiar objection (e.g. Deaton and Cartwright, 2018) against RCM. Not only does RCM (i) tells us nothing about the mechanism by which a treatment affects a potential outcome, but it also (ii) tells us nothing about the background conditions required for the causal effect of interest. While the latter is a serious problem for randomised control trials,

it is less of an issue for RCM methods which rely on observational data such as instrumental variables. There is also a flourishing literature on generalising the results from one study to new settings (Athey et al., 2020).

What about the objection that RCM ignores mechanism? One might respond that this is actually a key strength of RCM. While sometimes there might be a clear mechanism behind a causal link, very often there might be too much heterogeneity across people. So, imposing a single model M will be inappropriate or even wrong. This does not mean that we can never model the mechanism but only that modelling it correctly might be more difficult than assumed by PCM advocates. If we believe that methodology should be driven by pragmatic considerations (Weinberger, 2022), there is nothing wrong in using different methods to solve different problems. We can first use RCM establish a causal link and then use other techniques to study the underlying mechanism. On its own, RCM does not exclude such an approach.[14]

V Economists against PCM

Having seen how PCM advocates criticise RCM, here I turn the table around and consider objections to PCM from an RCM standpoint. Specifically, I discuss objections raised by economists who dislike PCM due to its perceived connection to structural equations *modelling* (Pearl, 2009, §7). Structural equations modelling is a method that gained popularity among economists in the 1960s, but it came to be regarded as an 'empirical failure' in the 1970s (Heckman, 2000). This raises the question why economists gave up this PCM-like method. I consider two of their objections: (i) causal sufficiency almost never holds and (ii) PCM cannot estimate individual-specific effects.

Before proceeding, note that I 'translated' the original objections (Liu, 1960; Leamer, 1983; Lucas and Sargent, 1997) into the language of PCM. This was necessary because structural equations modelling uses a different semantics from PCM. While I only present the translated arguments, Moev (2022) has recently considered how to relate the two frameworks.

[14]One might wonder if PCM advocates will object to other assumptions of RCM. They cannot object to SUTVA since it is also an issue in PCM (Ogburn and VanderWeele, 2014). A more controversial assumption is consistency which seems to be differently understood in the two frameworks (Weinberger, 2022, §3).

V.a Identification and causal sufficiency

Causal sufficiency requires that we include all variables that are common causes of the two variables involved in the causal link of interest (Section II). Crucially, it is a necessary condition for identifying a particular causal effect. Causal sufficiency becomes problematic when *unobservable* variables affect our causal link because we cannot verify it empirically. Unfortunately, in social sciences 'any effects that can possibly be there typically are' there (Imbens, 2020, p.1140), meaning that it is usually easy to find a common cause of both variables in a causal link. In our stock example (Figure 1), we omitted the level of criminal activity in an area which might affect both neighbourhood quality and income (Kling et al., 2005). So, economists can argue that PCM rarely identifies causal effects, since causal sufficiency usually fails.[15]

At this point, we might worry that causal sufficiency is *always* needed: RCM-based techniques will also fail unless we assume causal sufficiency. Thus, RCM advocates have no right to attack PCM for causal sufficiency. Nevertheless, causal sufficiency in RCM is less demanding than in PCM. The reason is that it can be translated into PCM as an assumption called selection on observables which requires that treatment assignment is fully determined by observables (Dale and Krueger, 2002). However, we saw that many RCM methods do not need this assumption: they are specifically designed to work under selection on *un*observables, i.e., when causal sufficiency fails. Furthermore, RCM usually explores the causal links between *two* variables only whereas PCM often explores the links between *many* variables. Why is the number of variables important? If we only study one causal link as in RCM, we should ensure causal sufficiency only for that link. If we study many causal links as in PCM, we should ensure causal sufficiency for all links. So, in PCM we need to guarantee causal sufficiency for many more links than in RCM. Unfortunately, this is difficult even for one link in social science. In that sense, causal sufficiency is more demanding in PCM applications.

A PCM advocate might acknowledge this point but respond that DAGs assuming causal sufficiency are a useful starting point in our research. We can then consider, for which hypothesised links causal sufficiency is reasonable, and, subsequently, examine more complex models,

[15] Originally, macroeconomists attacked structural equation modelling users for making shaky identification claims because they excluded important variables from their models (Liu, 1960; Sims, 1980). Ironically, this had to be done in order to ensure identification. See for details Moev (2022, §§3-4).

in which it does not hold. So, frequent violations of causal sufficiency does not mean that we cannot use PCM for causal inference.

V.b Individual-invariant coefficients

We noted above that Rubin objects to Pearl's definition of POs within a structural model because it entails that two observationally equivalent people necessarily have the same causal effect, i.e., on PCM their effects are individually invariant. Why do economists also make such a fuss about individual invariant effects?[16] The reason is Pearl's claim that PCM can handle actual causation cases, i.e., how much neighbourhood change the income for a particular individual (Pearl, 2009, §10). This implies that PCM can identify individual causal effects. However, this is in direct contrast to the fundamental problem of causal inference in PCM: we do not observe the POs both with and without treatment. For this reason, RCM methods are designed to work under a lot of heterogeneity in individual effects (Imbens and Wooldridge, 2009, p.7). The question if we can estimate individual causal effects in actual causation cases is at the crux of the methodological debate. I call this issue the *heterogeneity problem* and later show how it helps to explain most disagreements between RCM and PCM.

So, RCM advocates will object to PCM because it mistakenly claims to resolve the *heterogeneity problem*. How can RCM advocates support this claim? One strategy would be to provide examples of individual causal effects in actual causation cases which PCM cannot handle. Specifically, consider a single equation determining income:

$$Income_j = \beta Education_j + u_j \tag{5}$$

where u_j is a probabilistically independent error term and where causal sufficiency holds.[17] In (10), we can clearly see the individual-invariant effects, i.e., β is the same for everybody. So, the incomes of two people with the same education will differ if and only if their error terms differ. This implication looks unreasonable. For example, there is substantial evidence that the returns

[16] Historically, economists attacked PCM for assuming *time*-invariant effects (Lucas, 1983, p.25), but this should be translated into assuming *individual*-invariant effects (Moev, 2022).

[17] That is, there are no common causes of income and education which we have missed.

to education differ across genders (Dougherty, 2005).

Thus, we might want to introduce such variation by modelling the true causal effect as $\beta_j = \beta + \epsilon_j$. In this formulation, ϵ_j does not enter directly into either education or income but only into the causal effect. RCM methods can still tell us something about the average treatment effect under relatively general conditions on the probability distribution of ϵ_j (Imbens and Wooldridge, 2009). Unfortunately, it is not obvious how we can add such unobserved randomness in the arrows of a DAG. We cannot treat u_j as a mediator since it is unobserved. PCM allows us to introduce randomness into variables but not into causal effects. In situations, where randomness can enter into the causal effect, PCM will be unable to capture actual causation. In other words, RCM advocates are right in saying that PCM cannot always resolve the *heterogeneity problem*. Appendix A.b provides another example of such a situation.

VI Traditional metaphysical theories

We considered four clash points between RCM and PCM: (i) Pearl's equivalence claims, (ii) RCM's inability to handle mechanisms, (iii) the importance of causal sufficiency in PCM and (iv) the *heterogeneity problem*. In my view, the deepest disagreement is about the *heterogeneity problem* because it can explain why PCM and RCM disagree about the other three things. It immediately suggests why (i) Pearl's equivalence claims is false. It also helps us see why (ii) RCM does not examine mechanisms. It is hard enough to estimate one specific causal effect, let alone the whole mechanism, in which this effect is embedded. Moreover, given how different causal effects and mechanisms are across individuals, RCM advocates cannot lightly assume causal sufficiency. This explains why they are unwilling to assume causal sufficiency for all links in a DAG as in PCM (iv).

While the *heterogeneity problem* is key for understanding this methodological debate, this insight does not tell us *why* PCM and RCM assume it has different solutions in the first place. For that purpose, we need to venture into the metaphysics of causation. This approach is justified because we cannot explain why different methods work without talking about metaphysics, i.e., 'metaphysics and methodology should go hand in hand' (Cartwright, 2007, Chapter 9). So, I

argue that PCM and RCM depend on two slightly different metaphysics of causation which can explain their responded to the heterogeneity.

Unfortunately, I cannot base my argument on the traditional theories of causation because they cannot explain why causal inference based on RCM or PCM works. Consequently, they also cannot explain their methodological differences. We need an alternative theory. Before introducing such a theory, I show why the mainstream versions of the probabilistic, regularity, counterfactual and interventionist theories will not do the job (Beebee et al., 2009).[18]

VI.a Regularity theory

The regularity theory (Psillos, 2009, p.131) states that event c causes event e iff:

(i) Events c and e are contiguous in spacetime;

(ii) c precedes e temporally;

(iii) All events of type C are regularly followed by all events of type E.

In this definition, c and e refer to specific *events*, e.g., Alexei's neighbourhood and income, whereas C and E refer to *event-types*, e.g., Kensington and being rich. Conditions (i) and (ii) ensure that c and e are two closely linked events with the correct temporal order. Condition (iii) introduces the idea of regularity between event-types where a regularity is a lawlike universality and not just a generalisation over separate instances (Psillos, 2009). So, if we observe that everybody who was brought up in Kensington is rich, then bringing up Alexei in Kensington will make him get rich.

Unfortunately, the regularity theory might incorrectly classify events linked by common causes as cases of causation. Consider the fact that on Earth all event-types *day* are followed by event-types *night*. By the regularity theory, there is nothing to stop us from saying that today will cause tonight to appear. This is clearly false since today and tonight have a common cause: earth rotation. Our theory of causation needs to rule out such regularities from common cause.

We can easily deal with such counterexamples in PCM and RCM. In PCM we need causal sufficiency before concluding that today will cause tonight. We can try adding causal sufficiency

[18] I exclude Salmon's process theory because it seems less applicable to social sciences.

to the regularity theory but it is not obvious how this assumption can be defined in terms of regularities. In RCM the (to)day-causing-(to)night example is ill-defined. Our POs are night and our treatment is day: $Night_i(Day = 1)$ and $Night_i(Day = 0)$. To conduct causal inference, we need to observe at least one unit i, on which we can give the treatment $Day = 0$. This is clearly impossible, given earth rotation. In other words, to implement treatment $Day = 0$, we need to stop earth rotation, but this means that we have acknowledged earth rotation as a common cause. So, the basic regularity theory cannot explain why causal inference works because it cannot deal with common cause counterexamples which are not an issue on PCM and RCM.

VI.b Counterfactual theory

This theory (Paul, 2009) states that C is a cause of E iff:

(i) The counterfactual 'if not C, then not E' is true.

The theory tries to reduce causation to counterfactuals which are taken as a primitive. How do we evaluate if a counterfactual is true? Lewis suggests using a similarity semantics: counterfactual $A \rightarrow B$ is true if it is true in the A-closest possible world. In other words, a world in which A and B are both true is closer to our actual world than a world in which A holds but B does not (Menzies and Beebee, 2020, §1.1). Equipped with this similarity semantics, we can evaluate counterfactuals for evidence of causation.

Setting questions about possible worlds aside, a crucial problem with the theory are cases of preemptive prevention (Paul, 2009). Suppose we have two processes $C1$ and $C2$ that are both sufficient to cause E (Collins, 2004). If E is getting rich, then $C1$ and $C2$ could be two lucrative job offers which I am about to receive. Just before $C1$ causes E, $C2$ causes E. In this case, counterfactual 'if not $C2$, then not E' is not true because $C1$ would have caused E if $C2$ had not occurred. $C2$ cannot be E's cause, although intuitively we would like to say $C2$ caused E. Such cases of *late* preemption are particularly challenging for counterfactual theories (Paul, 2009).

While it is unclear if PCM and RCM can handle preemption cases, the main difference with Lewis' theory is that they do not take counterfactuals as a primitive. Counterfactuals in RCM

are simply a useful reading clarifying POs' definition and play no other role. Counterfactuals in PCM emerge out of causal effect queries but rely on a causal modelling rather than a similarity semantics. Even if they are taken as a primitive in PCM, they follow very different inference rules from Lewis' semantics. While Pearl has claimed the equivalence of Lewis' semantics with his own (Glymour et al., 2016, p.116), Briggs (2012) has argued that certain counterfactuals (including *modus ponens*) are evaluated differently in the two frameworks, meaning that there is a counterexample disproving Pearl's claim. Therefore, the counterfactual theory cannot explain why causal inference techniques work.

VI.c Probabilistic theory

This theory states that C causes E iff:

(i) C precedes E temporally

(ii) $Pr(E|C) > Pr(E)$

(iii) There is no B occurring earlier than C such that $Pr(E|C\&B) = Pr(E|B)$

Here C and E are event-types but they can be single events in alternative versions of the theory. Condition (ii) states that the cause must be raising the probability of the effect whereas condition (iii) ensures that we have not missed any common cause B which would render C and E conditionally uncorrelated. The probabilistic theory is not subject to the day-causing-night counterexample due to condition (iii).

There are many well-known issues with the probabilistic theory such as its inability to remain a fully reductive theory of causation in the face of Simpson's paradox (Hitchcock, 2021). In my view, the most obvious reason why the theory fails, however, are counterexamples such as faithfulness failures discussed in Section II. Using our old example, conditional on occupation we get:

$$P(Wage|Gender\&Occupation) = P(Wage|Occupation)$$

The probabilistic theory implies that there is no causal effect of *Gender* on $Wage$ but this is not necessarily true.

Can the probabilistic theory justify PCM? Pearl (2009, §1.3-1.4) acknowledges that he can either use the probabilistic theory *or* the structural approach to give DAGs a causal reading (Section II). He chooses the latter approach partly because it does not rely on the probabilistic theory but also because it allows evaluating counterfactuals (Pearl, 2009, p.27). So, while it can ground parts of PCM, the theory is clearly not Pearl's favoured approach. Similarly, Holland (1986, §5.3) argues that the probabilistic theory is 'quite different' from RCM because (a) it imposes *no* restrictions on what can count as a cause in contrast to RCM and (b) it discusses probabilities on the aggregate, not the individual level. Even if we define probabilities on the individual level, it is not obvious how to aggregate them across subpopulations (Hitchcock, 2021, §2.6).

VI.d Interventionist theory

On this theory, C causes E iff:

(i) Intervening on C in the right way will result in a corresponding change to E (Woodward, 2016).

According to Woodward (2016), intervening 'in the right way' means that we only manipulate[19] the causal route going through C. Then, if we find a change in E, it will certainly indicate a causal effect. In our stock example (Figure 1), intervening on neighbourhoods means setting it to some fixed value such as Kensington, changing nothing else in the model and seeing how much the intervention affects the probability of getting rich.

It is unclear what Pearl's attitude towards the theory is. While Woodward (2016) credits Pearl as advocating an interventionist theory, Papineau (2021) argues that we can only make sense of PCM via the structural theory discussed below. In my view, however, the interventionist theory is not sufficient on its own to justify why PCM works. The do-calculus is inseparable from the idea of intervention within PCM since it provides the rules for evaluating the effect of interventions within models. However, do-calculus is used for the identification of causal effect and is largely independent from causal discovery (Section II). So, interventions are not necessary for conducting causal inference in PCM: they can only be evaluated *within* the causal structural

[19] I use intervening and manipulating interchangeably.

model. In other words, we can evaluate interventions only if we can justify a causal reading of a DAG. So, the interventionist theory is insufficient to explain why PCM works.

Can the theory explain causal inference in PCM? On the surface, the answer seems yes: Rubin's argues for 'no causation without manipulation' (Holland, 1986). We can only calculate causal effect of variables, on which we can imagine intervening. However, to see why the interventionist theory in the form above cannot do the job, note that we did *not* define POs in RCM relative to a model or a causal mechanism. It is not obvious how we can intervene 'in the right way', i.e., only on the causal route going through C. For example, if the causal mechanisms applying to different observations differ, causal routes are not well defined, and the notion of intervening in the right way is ambiguous. How can we then justify Rubin's motto? Manipulability should not be understood as grounding RCM but as a constraint on the type of variables that can have a treatment effect. For Rubin, certain variables such as race and gender lack manipulability (Imbens and Rubin, 2015, Chapter 1). While we *can* use RCM to study their effect, it will not be a valid application of RCM. So, POs can be mathematically defined without the notion of an intervention, but they can only be used in situations where manipulability is satisfied. With that said, the interventionist theory remains much closer to explaining why RCM works than the other traditional theories.

VII Structural equations theory of causation

We need an alternative metaphysical theory to explain the metaphysical differences between PCM and RCM. Inspired by Papineau (2021, 2022), I argue for the structural equations theory of causation. It reduces causation to 'underlying structural equations with probabilistic exogenous terms' (Papineau, 2021, p.248). On this theory, there is a system of structural equations behind every causal mechanism we might want to study. In practice, we only observe bits of these structural equations. We probably do not observe all variables from the true system, for example. This creates the need for causal inference techniques. In other words, if we would like to learn causal effects and we do not observe the true causal mechanism, we need to decide on the best way to leverage the available information which is done by causal inference techniques. Unfortunately,

I show that Papineau's original version is *insufficient* to explain why **P**CM works because it does not impose enough restrictions on the functional form of the structural equations. I then offer a slightly modified version of the theory which deals with the insufficiency problem and also explains why **R**CM works.

We can gain insight into the structural equations theory by getting a bit more formal. There is a set of *true* K^* equations that govern all mechanisms in nature and society. The k-th structural equation for the outcome variable y_k^*:

$$y_k^* = f_k^*(pa_k^*) + u_k^* \tag{6}$$

where pa_k^* is the full set of parents of y_k^* and u_k^* is a probabilistically independent error term which could always be generated by chancy quantum effects (Papineau, 2021, §21). Superscript * indicates *true* variables in contrast to *observed* ones without superscripts. So, (6) states that the variables on the right-hand side determine the outcome variable via the functional form $f_k^*(.)$. The right-hand side only contains variables that have appeared in one of the previous $k-1$ equations or exogenous error terms. This means that the system of equation does not contain cycles, i.e., it is a DAG. Definition 1 which is closely linked with Simon (1953) summarises the theory. Conditions (ii) and (iii) ensure that there is a system of R equations, containing a directed causal path from C to E.

Definition 1 (Structural Equations Theory)**.** *C causes E iff:*

(i) There is a recursive mechanism consisting of K^* equations of the form (6);

(ii) Variables C and E are connected via $1 \leq R \leq K^*$ equations;

(iii) E is an ancestor of C within this system of J equations.

Does Papineau's theory in Definition 1 differ from Hausman's **CP** principle (1998)? There are two differences. First, Papineau's theory is explicitly embodied within structural equations framework. Second, Papineau aims at a reductive theory in contrast to Hausman (Hitchcock, 2000). We should read the equations as lawlike regularities that actually appear in nature. This establishes a connection with the regularity theory of causation (Papineau, 2021, p.31).

My representation of the theory, however, differs in a crucial respect from Papineau's original. The difference is in the restrictions on the functional form of the structural equations. Compare:

$$\underbrace{y_k^* = f_k^*(pa_k^*, u_k^*)}_{\textit{Papineau's eq. (22)}} \qquad vs. \qquad \underbrace{y_k^* = f_k^*(pa_k^*) + u_k^*}_{\textit{Our eq. (6)}} \tag{7}$$

The u_k^*-s are restricted to enter additively into the outcome variable in our formulation. To see why this matters, it is necessary to unpack Papineau's argument for the structural theory (2021, §13). As in Definition 1, he assumes a system of K^* equations. Importantly, each structural equation has an unobserved error which is probabilistically independent from other causes. Papineau then claims that this condition is sufficient to always differentiate the true causal structure from alternatives. This result seemingly confirms his metaphysics: causation can indeed be reduced to a system of structural equations with probabilistically independent error terms.

Why do functional forms matter for this argument? Consider Papineau's main claim:

Claim 1. *If the errors in each true structural equation are independent from other causes, then we can always differentiate the true causal system from alternatives.*

Unfortunately, this claim is not always correct when we work with Papineau's functional form $y_{k,j} = f_k(pa_{k,j}, u_{k,j})$. In Appendix A.c, I provide a formal example of such a situation. However, the example does not tell us *why* Claim 1 is false. I believe that it fails because Papineau needs to prove two separate claims before establishing Claim 1. The first is:

Claim 2. *If the errors in each true structural equation are independent from other causes, (*) then the errors* ***enter*** *the outcome variable independently from other causes.*

According to Claim 2, the probabilistic independence of our errors implies that they actually enter the outcome variable independently from these causes.[20] Then, he needs to establish:

Claim 3. *(*) If the error terms* ***enter*** *the outcome variable independently from other causes, then we can always differentiate the true causal system from alternatives.*

[20] Consider $y_{k,j} = (pa_{1,k,j} + pa_{2,k,j})u_{k,j}$. Here $u_{k,j}$ is probabilistically independent from both parents but do not enter into $y_{k,j}$ independently from them. Appendix A.c discusses this example further.

If Claims 2 and 3 are both true, then Claim 1 is also true. However, the example in Appendix A disproves Claim 2: Papineau's theory fails because it incorrectly assumes the validity of Claim 2 under his functional form. Luckily, Claim 2 is true with the functional form I have suggested because the error terms are constrained to enter additively into the outcome.[21] So, if Claim 3 is correct, then my version of the structural theory implies the validity of Claim 1, meaning that it successfully reduces causation to structural equations.

Having established this point, we can show how the structural theory explains causal inference techniques. The K^* equations and the true variables define the complete causal mechanism. In practice, however, we are often interested in modelling one particular outcome such as income in Figure 1. We usually observe only a subset of all variables and equations from the true mechanism. As a result, we can at most recover K equations of the form:

$$y_k = f_k(pa_k) + u_k \tag{8}$$

where we are interested in modelling y_k. $Pa_{k,j}$ is not necessarily a subset of $pa^*_{k,j}$: we might have not observe the l-th true parent $pa^*_{k,j,l}$ but we might still include one of $pa^*_{k,j,l}$'s *ancestors*. If we are willing to make some additional assumptions such as causal sufficiency, we can now conduct causal inference. So, we can infer causal effects, even when we do not observe the full true system.

In that sense, Papineau's reduction of causation to structural equations remains very faithful to PCM. It makes the structural causal models that Pearl uses for causal readings of DAGs into the metaphysics of causation. We can summarise these ideas by formalising Definition 1. There is a true mechanism of R equations which connects outcome y^*_k and treatment x^*_k as in $y^*_k = M^*(b^*_k, x^*_k)$ where b^*_k are all the relevant ancestors of y^*_k which could be mediators or background factors. However, we are trying to model that mechanism using observed variables $y_k = M(b_k, x_k)$. While $M^*(.)$ refers to the true *mechanism*, $M(.)$ refers to the *model* we use for causal inference.

We can now see how causal inference works in practice. Let the true pathway for the transmission of pain be $M^*(b^*_k, x_k)$ where x_k is damage applied to my hand. The mechanism contains

[21] Our formulation can probably be relaxed to $y^*_k = f^*_k(pa^*_k) + g^*_k(u^*_k)$ where $g^*_k(.)$ is a measurable function.

all the variables (neurons) along the ascending spinal tract. However, as an observer, I can only see the initial damage to my hand (x_k) and the degree of pain I feel (y_k). Thus, I can conclude that more damage to my hand causes more pain, even though I do not observe much of the true mechanism M^* which I approximate with model M.

At this point, we might raise several issues with the structural equations theory. From RCM standpoint, there are two related problems stemming from the definition of POs. As we saw above, it is not obvious if PCM can estimate individual causal effects (Section V). Given its close connection with PCM, the structural equations theory should also have difficulty with these cases. It also implies that if two people share exactly the same characteristics, they will respond in exactly the same way to a treatment.

The other problem with the structural theory is its inability to capture the evolution of causal mechanisms over time. It assumes that the system of structural equations governing my behaviour is the same as the system governing the behaviour of a person in another era. However, it is unlikely that coding skills mattered for incomes in the Middle Ages but they do matter for incomes nowadays. This is not a trivial point because it shows that systems of structural equations are dynamic: different factors get different weighting at different times. In practice, this assumption is often unproblematic. Probably, the same model determines my income now and three years ago. Nevertheless, it remains unclear why we are justified in assuming time-invariant structural equations in our metaphysics.

These considerations suggest that the standard structural equations theory which we can call *Pearl's* structural equations theory (PSET) would probably be unacceptable to RCM advocates. Thus, we need *Rubin's* structural equations theory (RSET) to justify RCM. Luckily, we only need to tweak PSET slightly to get RSET. We begin by making the structural equations in RSET individual-specific. To that aim, we denote individuals as j, so that the true causal mechanism is:

$$y^*_{k,j} = f^*_{k,j}(pa^*_{k,j}) + u^*_{k,j} \tag{9}$$

We allow the functions $f^*_{k,j}(.)$ to be individual-specific for j and the number of equations to differ across individuals These are the only differences with PSET. Specifically, if we add j-notation to PSET, it postulates $f^*_k(pa^*_{k,j})$ whereas PSET postulates $f^*_{k,j}(pa^*_{k,j})$. If we are interested

in how some $y^*_{k,j}$ is affected by treatment $x^*_{k,j}$, we get the individual-specific true mechanism $y^*_{k,j} = M^*_j(b^*_{k,j}, x^*_{k,j})$. For causal inference, we need to approximate the true mechanism with an individual-*specific* model $y_{k,j} = M_j(b_j, x^*_j)$. In contrast, PSET postulates the individual-*invariant* mechanism $y^*_{k,j} = M^*(b^*_j, x^*_j)$ and model $y_{k,j} = M(b_j, x^*_j)$ which have no individual subscripts.

Why would RCM advocates prefer RSET? Causal effects are by definition individual-specific on RSET, even for people with the same covariates. Moreover, RSET allows us to capture changes in the causal mechanism through time. While the $M^*_j(.)$-s for people in the Middle Ages might have contained no place for an effect of coding skills on income, these people passed away and made away for people nowadays whose $M^*_j(.)$-s do contain a place for coding skills.

Markus (2021) has argued that RCM advocates might not like defining POs relative to a model, meaning that they may reject RSET. However, Markus' argument assumes an individual-invariant model. Relaxing this assumption makes models much more acceptable because they are akin to a data-generating process. Statisticians often use such processes when studying the theoretical properties of different estimators.[22] So, if assuming a true but inaccessible data-generating process is acceptable to RCM advocates, then assuming individual-specific inaccessible structural equations should also be acceptable.

How can RSET and PSET explain the methodological differences between RCM and PCM? We can see the different responses to the *heterogeneity problems*. On PSET, individuals share the same mechanisms, so we can use your mechanism as a guide to mine. Once we estimate the parameters of our model we can also derive the causal effects in actual causation cases. In contrast, given the mechanisms are fundamentally different across people on RSET, this is not an option. This also explains why RCM advocates do not generally examine mechanisms. There may or may not be a shared a mechanism: we simply do not know in advance. For this reason, RCM methodologists develop techniques aimed at making as few assumptions as possible on these mechanisms. Furthermore, because we can easily think of violations of causal individuals for a particular individual j given RSET, RCM advocates are skeptical of it. In contrast, individual violations of causal sufficiency are less problematic on PSET.

[22]For example, we might investigate how well the ordinary least squares estimator performs under a data-generating process of a linear model with an omitted variable using asymptotic theory (Wooldridge, 2010, §4.3)

VIII Conclusion

In this paper, I argued that the methodological differences between RCM and PCM can be explained by their different views of the metaphysics of causation. RCM and PCM advocates disagree about many things such as the importance of mechanisms. However, these disagreements boil down to a more fundamental disagreement about the *heterogeneity problem*. Using this insight, I suggested that RCM and PCM both rely on a reduction of causation to structural equations but these equations vary across individuals on RCM. This allow us to see why they see the *heterogeneity problem* differently and, consequently, why they disagree about other methodological issues.

One question which these considerations raise is whether the metaphysical theory behind RCM or behind PCM is superior. There are three reasons why the RCM version is superior. First, there is no obvious metaphysical reason to impose individual-invariant mechanisms. Second, the RCM version allows us to capture the variation in the structural equations over time. Third, the RCM version is better suited to account for how we perceive the world. In practice, on any causal path, there are many unobservable mediators. For this reason, the data would seem to us *as if* it comes from the RCM metaphysics, even if it does not. This is a very brief answer to a much more complex question that I hope to explore in future work.

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A Technical Appendix

A.a Graphical illustration of a faithfulness failure

Recall that in the example in Section II we found that conditional on occupation there is no correlation between gender and income. Figure 2 illustrates this example graphically. We asked whether the absence of correlation between gender and income means that gender has no causal effect on income conditional on occupation. We saw that this is not necessarily the case due to the fact that gender affects income through two separate paths whose effects exactly offset each other. In Figure 2, we can see how gender has a *positive* effect on income via education. This is because women are more likely to attend university, i.e., $G \xrightarrow{+} E$ (Goldin et al., 2006), and that education increases earnings, i.e., $E \xrightarrow{+} I$ (Card, 1999). On the other hand, women's lower wage negotiating ability directly lowers their earnings , i.e., $G \xrightarrow{-} I$ (Card et al., 2016). It is entirely possible that the direct *negative* effect on income *exactly* cancels out the *positive* effect mediated by education. This is a faithfulness failure: gender and income are conditionally uncorrelated, albeit gender still affects income.

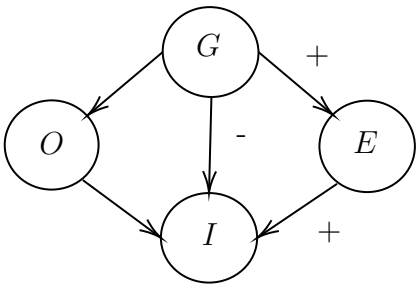

Figure 2: Faithfulness failure

A.b PCM and Actual Causation

This subsection provides another example of an actual causation case which PCM cannot handle. Consider again the single equation determining income:

$$Income_j = \beta Education_j + u_j \tag{10}$$

where u_j is a probabilistically independent error term and where causal sufficiency holds. As noted above, there is substantial evidence that the returns to education differ across people. For example, they might differ across genders, even if gender has no independent effects on either income or education.[23] Crucially, this will not violate causal sufficiency because gender will not be a common cause of income or education. It will only affect the causal effect itself, not the treatment or the outcome variables. While we *can* estimate β in this case, our estimate will not be capturing the true causal effect contrary to Pearl's claim that we can get causal effects in cases of actual causation. It will only provide a summary of the returns to education across many different people. In RCM we generally cannot estimate individual effects as well. However, there is explicit recognition that causal effects are individually varying from the definition of POs which is not the case in PCM.

Here PCM advocates might object that PCM can still allow β_j to vary across individual by adding mediators. If we add gender as mediator, we can model j's causal effect as:

$$\beta_j = \beta + \alpha_1 Gender_j + \alpha_2 Mediator_j \tag{11}$$

where β is the direct effect of education on wages. $Mediator_j$ is a second mediator and α_1 and α_2 give the contribution of the two mediators to β_j. However, this formulation raises more questions than it answers. Given that the effect of education on income differs across individuals, a mediator might have a different effect on different individuals. Education might affect earnings through a path containing two mediators and not just one. Its effect might be mediated through gender and marriage which is known to have contrasting effects on men (Ludwig and Brüderl, 2018) and women (Vagni and Breen, 2021). This reasoning suggests that at some point we must draw the line and say that there are no more mediators. Once this is done, the causal effect which we have estimated will be considered the same across all individuals. Drawing such a line will be necessarily arbitrary. It is not obvious when it will be reasonable to say that there are no more mediators. While such a threshold might exist, it is up to PCM advocates to provide a reasonable criterion for determining it.

[23] While this situation is rather implausible empirically, it is theoretically possible.

A.c Functional form objection to Papineau

This appendix provides a formal example which disproves a crucial assumption in Papineau's structural theory of causation (2021; 2022). More formally, Papineau's claim is:

Claim 4. *If the errors in each true structural equation are independent from other causes, then we can always differentiate the true causal system from alternatives irrespectively of the functional form.*

Unfortunately, the consequent does not always follow from the antecedent. That is, there exist functional forms under which we cannot distinguish the true and the alternative systems, even if the errors are independent. I proceed by providing such a functional form which provides the counterexample. Figure 3 gives the general set-up I consider.

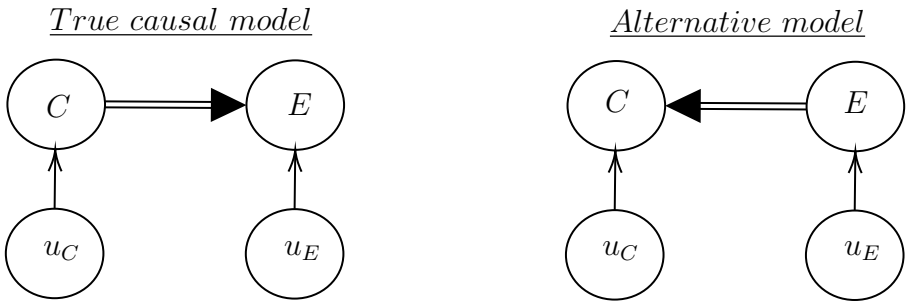

Figure 3: True and alternative DAG

We also assume (i) $E[u_c] = E[u_E] = 0$, (ii) $E[u_c^2] = Var(u_c) \neq 0 \neq Var(u_E) = E[u_E^2]$ and (iii) $E[u_c u_E] = Cov(u_c, u_E) = 0$. I will provide two functional form examples. Both functional forms are allowed by Papineau's formulation but only one of them allows us to distinguish the true causal system from alternatives.

A.c.1 Linearity

If we assume linearity, the DAGs reduces to one of Papineau's example (2021, eq. (18), (20)). The linearity assumption is allowed by both Papineau's functional form and by our functional

form, as postulated in (7). The corresponding structural equations are:

True causal model	*Alternative model*
$C = u_C$	$C = \frac{1}{\beta}E + u_C^*$
$E = \beta C + u_E$	$E = u_E^*$

By substituting the true causal model into the alternative, we get:

$$u_E^* = \beta C + u_E$$
$$u_C^* = u_C - \frac{1}{\beta}E = -\frac{u_E}{\beta}$$

Then, we calculate:

$$Cov(E, u_C^*) = -\frac{E[u_E^2]}{\beta} \neq 0$$

Note two things. First, as expected on the structural theory, in the true model there is extra variation entering into E independent from its cause. Second, this is not the case in the alternative model where $Cov(E, u_C^*) \neq 0$. So, by Papineau's structural theory, we have correctly identified the true causal model.

A.c.2 Nonlinearities

Let us take a functional form allowed by Papineau's theory but not ours (recall eq. 7 above):

True causal model	*Alternative model*
$C = u_C$	$C = (\alpha + E)u_C^*$
$E = (\beta + C)u_E$	$E = u_E^*$

We simply use $f(pa_k, u_k) = (\beta + pa_k)u_k$ instead of the linear $f(pa_k, u_k) = \beta pa_k + u_k$. If $Cov(u_C^*, E) = 0$, we will be unable to distinguish the true from the alternative model. The reason is that in both cases we can separate the outcome variable into causes and independent

errors.

Let us evaluate this idea by following the same procedure as above:

$$u_E^* = (\beta + u_C)u_E$$
$$u_C^* = \frac{C}{\alpha + E} = \frac{u_C}{\alpha + (\beta + C)u_E}$$

Note that $Cov(\alpha + E, u_C^*) = E[(\alpha + E)u_C^*] = E[Eu_C^*]$. So, we have:

$$E[(\alpha + E)u_C^*] = E[u_C] = 0 \implies Cov(E, u_C^*) = 0$$

Similarly, $Cov((\alpha + u_E^*), u_C^*) = E[u_E^* u_C^*] = Cov(u_E^*, u_C^*) = 0$. In both the true and the alternative system, we can reduce the outcome variable into its causes and independent errors. So, the structural equations theory does not tell us which is the correct causal structure.