Appropriate Causal Models and the Stability of Causation

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Abstract

Causal models defined in terms of structural equations have proved to be quite a powerful way of representing knowledge regarding causality. However, a number of authors have given examples that seem to show that the Halpern-Pearl (HP) definition of causality [Halpern and Pearl 2005] gives intuitively unreasonable answers. Here it is shown that, for each of these examples, we can give two stories consistent with the description in the example, such that intuitions regarding causality are quite different for each story. By adding additional variables, we can disambiguate the stories. Moreover, in the resulting causal models, the HP definition of causality gives the intuitively correct answer. It is also shown that, by adding extra variables, a modification to the original HP definition made to deal with an example of Hopkins and Pearl [2003] may not be necessary. Given how much can be done by adding extra variables, there might be a concern that the notion of causality is somewhat unstable. Can adding extra variables in a "conservative" way (i.e., maintaining all the relations between the variables in the original model) cause the answer to the question "Is $X = x$ a cause of $Y = y$?" to alternate between "yes" and "no"? It is shown that we can have such alternation infinitely often, but if we take normality into consideration, we cannot. Indeed, under appropriate normality assumptions. Adding an extra variable can change the answer from "yes' to "no", but after that, it cannot change back to "yes".

1 Introduction

Causal models defined in terms of structural equations have proved to be quite a powerful way of representing knowledge regarding causality. For example, they have been used to find causes of errors in software [Beer et al. 2012] and have been shown to be useful in predicting human attributions of responsibility [Gerstenberg and Lagnado 2010; Lagnado, Gerstenberg, and Zultan 2013]. However, a number of authors [Glymour et al. 2010; Hall 2007; Livengood 2013; Spohn 2008; Weslake 2015] have given examples that seem to show that the Halpern-Pearl (HP) definition of causality [Halpern and Pearl 2005] gives intuitively unreasonable answers. One contribution of this paper is to show that these "problematic" examples can be dealt with in a relatively uniform way, by being a little more careful about the choice of causal model.

The need to choose the causal model carefully has been pointed out frequently [Blanchard and Schaffer 2013; Hall 2007; Halpern and Pearl 2005; Halpern and Hitchcock 2010; Hitchcock 2001; Hitchcock 2007]. A causal model is characterized by the choice of variables, the equations relating them, and which variables we choose to make exogenous and endogenous (roughly speaking, which are the variables we choose to take as given and

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which we consider to be modifiable). Different choices of causal model for a given situation can lead to different conclusions regarding causality. The choices are, to some extent, subjective. While some suggestions have been made for good rules of thumb for choosing random variables (e.g., in [Halpern and Hitchcock 2010]), they are certainly not definitive. Moreover, the choice of variables may also depend in part on the variables that the modeler is aware of.

In this paper, I consider the choice of representation in more detail in five examples. I show that in all these examples, the model originally considered (which I call the "naive" model) does not correctly model all the relevant features of the situation. I argue that we can see this because, in all these cases, there is another story that can be told, also consistent with the naive model, for which we have quite different intuitions regarding causality. This suggests that a more detailed model is needed to disambiguate the stories. In the first four cases, what turns out to arguably be the best way to do the disambiguation is to add (quite well motivated) extra variables, which, roughly speaking, capture the mechanism of causality. In the final example, what turns out to be most relevant is the decision as to which variables to make exogenous. Once we model things more carefully, the HP approach gives the expected answer in all cases.

As already observed by Halpern and Hitchcock [2015], adding extra variables also lets us deal with two other concerns that resulted in changes to the original HP definition. In Section 4, I consider an example due to Hopkins and Pearl [2003] that motivated one of the changes. After showing how this example can be dealt with by adding an extra variable in a natural way (without modifying the original HP definition), I show that this approach generalizes: we can always add extra variables so as to get a model where the original HP definition can be used. In Section 5, I discuss an example due to Hiddleston [2005] that motivated the addition of normality considerations to the basic HP framework (see Section 2). Again, adding an extra variable deals with this example.

All these examples show that adding extra variables can result in a cause becoming a non-cause. Can adding variables also result in a non-cause becoming a cause? Of course, without constraints, this can easily happen. Adding extra variables can fundamentally change the model. Indeed, even if we insist that variables are added in a conservative way (so as to maintain all the relations between the variables in the original model), $X = x$ can alternate infinitely often between being a cause of $Y = y$ and not being a cause. But, in a precise sense, this requires the new variables we add to take on abnormal values. Once we talk normality into consideration, this cannot happen. If $X = x$ is not a cause of $Y = y$, then adding extra variables to the model cannot make $X = x$ a cause of $Y = y$.

The rest of this paper is organized as follows. In the next section, I review the HP definition (and the original definition) and its extension to deal with normality, as discussed in [Halpern and Hitchcock 2015]. I discuss the five examples in Section 3. In Section 4, I discuss how adding extra variables can deal with the Hopkins-Pearl example and, more generally, can obviate the need to modify the original HP definition. In Section 5, I discuss the extent to which adding extra variables can avoid the need to taking normality into account. In Section 6, I discuss issues of stability. I conclude in Section 7 with some discussion of the implications of these results.

2 Review

In this section, I briefly review the definitions of causal structures, the HP definition(s) of causality, and the extension that takes into account normality given by Halpern and Hitchcock. The exposition is largely taken from [Halpern 2008]. The reader is encouraged to consult [Halpern and Pearl 2005], and [Halpern and Hitchcock 2015] for more details and intuition.

2.1 Causal models

The HP approach assumes that the world is described in terms of random variables and their values. Some random variables may have a causal influence on others. This influence is modeled by a set of *structural equations*. It is conceptually useful to split the random variables into two sets: the *exogenous* variables, whose values are determined by factors outside the model, and the *endogenous* variables, whose values are ultimately determined

by the exogenous variables. For example, in a voting scenario, we could have endogenous variables that describe what the voters actually do (i.e., which candidate they vote for), exogenous variables that describe the factors that determine how the voters vote, and a variable describing the outcome (who wins). The structural equations describe how the outcome is determined (majority rules; a candidate wins if A and at least two of B, C, D, and E vote for him; etc.).

Formally, a *causal model* M is a pair $(\mathcal{S}, \mathcal{F})$, where $\mathcal{S}$ is a *signature*, which explicitly lists the endogenous and exogenous variables and characterizes their possible values, and $\mathcal{F}$ defines a set of *modifiable structural equations*, relating the values of the variables. A signature $\mathcal{S}$ is a tuple $(\mathcal{U}, \mathcal{V}, \mathcal{R})$, where $\mathcal{U}$ is a set of exogenous variables, $\mathcal{V}$ is a set of endogenous variables, and $\mathcal{R}$ associates with every variable $Y \in \mathcal{U} \cup \mathcal{V}$ a nonempty set $\mathcal{R}(Y)$ of possible values for Y (that is, the set of values over which Y *ranges*). For simplicity, I assume here that $\mathcal{V}$ is finite, as is $\mathcal{R}(Y)$ for every endogenous variable $Y \in \mathcal{V}$. $\mathcal{F}$ associates with each endogenous variable $X \in \mathcal{V}$ a function denoted F_X such that $F_X : (\times_{U \in \mathcal{U}} \mathcal{R}(U)) \times (\times_{Y \in \mathcal{V} - \{X\}} \mathcal{R}(Y)) \rightarrow \mathcal{R}(X)$. This mathematical notation just makes precise the fact that F_X determines the value of X, given the values of all the other variables in $\mathcal{U} \cup \mathcal{V}$. If there is one exogenous variable U and three endogenous variables, X, Y, and Z, then F_X defines the values of X in terms of the values of Y, Z, and U. For example, we might have $F_X(u, y, z) = u + y$, which is usually written as $X = U + Y$.[1] Thus, if $Y = 3$ and $U = 2$, then $X = 5$, regardless of how Z is set.

The structural equations define what happens in the presence of external interventions. Setting the value of some variable X to x in a causal model $M = (\mathcal{S}, \mathcal{F})$ results in a new causal model, denoted $M_{X=x}$, which is identical to M, except that the equation for X in $\mathcal{F}$ is replaced by $X = x$.

Following [Halpern and Pearl 2005], I restrict attention here to what are called *recursive* (or *acyclic*) models. This is the special case where there is some total ordering $\preceq$ of the endogenous variables (the ones in $\mathcal{V}$) such that, unless $X \preceq Y$, Y is independent of X, that is, $F_Y(\ldots, x, \ldots) = F_Y(\ldots, x', \ldots)$ for all $x, x' \in \mathcal{R}(X)$. I write $X \prec Y$ if $X \preceq Y$ and $X \neq Y$. If $X \prec Y$, then the value of X may affect the value of Y, but the value of Y cannot affect the value of X. It should be clear that if M is an acyclic causal model, then given a *context*, that is, a setting $\vec{u}$ for the exogenous variables in $\mathcal{U}$, there is a unique solution for all the equations. We simply solve for the variables in the order given by $\prec$. The variables that come first in the order, that is, the variables X such that there is no variable Y such that $Y \prec X$, depend only on the exogenous variables, so their value is immediately determined by the values of the exogenous variables. The values of variables later in the order can be determined once we have determined the values of all the variables earlier in the order.

2.2 A language for reasoning about causality

To define causality carefully, it is useful to have a language to reason about causality. Given a signature $\mathcal{S} = (\mathcal{U}, \mathcal{V}, \mathcal{R})$, a *primitive event* is a formula of the form $X = x$, for $X \in \mathcal{V}$ and $x \in \mathcal{R}(X)$. A *causal formula (over $\mathcal{S}$)* is one of the form $[Y_1 \leftarrow y_1, \ldots, Y_k \leftarrow y_k]\varphi$, where

- φ is a Boolean combination of primitive events,
- $Y_1, \ldots, Y_k$ are distinct variables in $\mathcal{V}$, and
- $y_i \in \mathcal{R}(Y_i)$.

Such a formula is abbreviated as $[\vec{Y} \leftarrow \vec{y}]\varphi$. The special case where $k = 0$ is abbreviated as φ. Intuitively, $[Y_1 \leftarrow y_1, \ldots, Y_k \leftarrow y_k]\varphi$ says that φ would hold if Y_i were set to y_i, for $i = 1, \ldots, k$.

A causal formula ψ is true or false in a causal model, given a context. As usual, I write $(M, \vec{u}) \models \psi$ if the causal formula ψ is true in causal model M given context $\vec{u}$. The $\models$ relation is defined inductively. $(M, \vec{u}) \models X = x$ if the variable X has value x in the unique (since we are dealing with acyclic models) solution to the equations in M in context $\vec{u}$ (that is, the unique vector of values for the exogenous variables that simultaneously

[1] The fact that X is assigned $U + Y$ (i.e., the value of X is the sum of the values of U and Y) does not imply that Y is assigned $X - U$; that is, $F_Y(U, X, Z) = X - U$ does not necessarily hold.

satisfies all equations in M with the variables in $\mathcal{U}$ set to $\vec{u}$). The truth of conjunctions and negations is defined in the standard way. Finally, $(M, \vec{u}) \models [\vec{Y} \leftarrow \vec{y}]\varphi$ if $(M_{\vec{Y}=\vec{y}}, \vec{u}) \models \varphi$. I write $M \models \varphi$ if $(M, \vec{u}) \models \varphi$ for all contexts $\vec{u}$.

2.3 The definition(s) of causality

The HP definition of causality, like many others, is based on counterfactuals. The idea is that A is a cause of B if, if A hadn't occurred (although it did), then B would not have occurred. But there are many examples showing that this naive definition will not quite work. To take just one example, consider the following story, due to Ned Hall and already discussed in [Halpern and Pearl 2005], from where the following version is taken.

> Suzy and Billy both pick up rocks and throw them at a bottle. Suzy's rock gets there first, shattering the bottle. Since both throws are perfectly accurate, Billy's would have shattered the bottle had it not been preempted by Suzy's throw.

We would like to say that Suzy's throw is a cause of the bottle shattering, and Billy's is not. But if Suzy hadn't thrown, Billy's rock would have hit the bottle and shattered it.

The HP definition of causality is intended to deal with this example, and many others.

Definition 2.1: $\vec{X} = \vec{x}$ is an *actual cause of* φ *in* $(M, \vec{u})$ if the following three conditions hold:

AC1. $(M, \vec{u}) \models (\vec{X} = \vec{x})$ and $(M, \vec{u}) \models \varphi$.

AC2. There is a partition of $\mathcal{V}$ (the set of endogenous variables) into two subsets $\vec{Z}$ and $\vec{W}$[2] with $\vec{X} \subseteq \vec{Z}$ and a setting $\vec{x}'$ and $\vec{w}$ of the variables in $\vec{X}$ and $\vec{W}$, respectively, such that if $(M, \vec{u}) \models Z = z$ for all $Z \in \vec{Z}$ (i.e., z is the value of the random variable Z in the real world), then both of the following conditions hold:

(a) $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W} \leftarrow \vec{w}]\neg\varphi$.

(b) $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}, \vec{W}' \leftarrow \vec{w}, \vec{Z}' \leftarrow \vec{z}]\varphi$ for all subsets $\vec{W}'$ of $\vec{W}$ and all subsets $\vec{Z}'$ of $\vec{Z}$, where I abuse notation and write $\vec{W}' \leftarrow \vec{w}$ to denote the assignment where the variables in $\vec{W}'$ get the same values as they would in the assignment $\vec{W} \leftarrow \vec{w}$, and similarly for $\vec{Z}' \leftarrow \vec{z}$.

AC3. $\vec{X}$ is minimal; no subset of $\vec{X}$ satisfies conditions AC1 and AC2.

The tuple $(\vec{W}, \vec{w}, \vec{x}')$ is said to be a *witness* to the fact that $\vec{X} = \vec{x}$ is a cause of φ.

AC1 just says that $\vec{X} = \vec{x}$ cannot be considered a cause of φ unless both $\vec{X} = \vec{x}$ and φ actually happen. AC3 is a minimality condition, which ensures that only those elements of the conjunction $\vec{X} = \vec{x}$ that are essential for changing φ in AC2(a) are considered part of a cause; inessential elements are pruned. Without AC3, if dropping a lit cigarette is a cause of a fire then so is dropping the cigarette and sneezing. AC3 serves here to strip "sneezing" and other irrelevant, over-specific details from the cause.

AC2 is the core of the definition. We can think of the variables in $\vec{Z}$ as making up the "causal path" from $\vec{X}$ to φ. Intuitively, changing the value of some variable in X results in changing the value(s) of some variable(s) in $\vec{Z}$, which results in the values of some other variable(s) in $\vec{Z}$ being changed, which finally results in the value of φ changing. The remaining endogenous variables, the ones in $\vec{W}$, are off to the side, so to speak, but may still have an indirect effect on what happens. AC2(a) is essentially the standard counterfactual definition of causality, but with a twist. If we want to show that $\vec{X} = \vec{x}$ is a cause of φ, we must show (in part) that if $\vec{X}$ had a different value, then so too would φ. However, the effect on φ of changing the value of the variables in $\vec{X}$ may not obtain unless we also change the values of some of the "off path" variables in $\vec{W}$. Intuitively, setting $\vec{W}$ to $\vec{w}$ eliminates

[2] I occasionally use the vector notation ($\vec{Z}$, $\vec{W}$, etc.) to denote a set of variables if the order of the variables matters, which it does when we consider an assignment such as $\vec{W} \leftarrow \vec{w}$.

some side effects that may mask the effect of changing the value of $\vec{X}$. For example, if Billy and Suzy both throw rocks at a bottle and hit it simultaneously, shattering it, but one rock would have sufficed to shatter the bottle, then to show that Billy's throw is a cause of the bottle shattering, we consider a setting where Suzy does not throw. Then if Billy doesn't throw, the bottle doesn't shatter, while if he throws it does shatter. We do require that, although the values of variables on the causal path (i.e., the variables $\vec{Z}$) may be perturbed by the change to $\vec{W}$, this perturbation has no impact on the value of φ. As I said when defining AC2, if $\vec{u}$ is the actual context and $(M, \vec{u}) \models \vec{Z} = \vec{z}$, then z is the value of the variable Z in the actual situation. We capture the fact that the perturbation has no impact on the value of φ by saying that if some variables Z on the causal path were set to their values in the context $\vec{u}$, φ would still be true, as long as $\vec{X} = \vec{x}$. Roughly speaking, AC2(b) says that if the variables in $\vec{X}$ are reset to their original value, then φ holds, even if only a subset $\vec{W}'$ of the variables in $\vec{W}$ are set to their values in $\vec{W}$ and and even if some variables in $\vec{Z}$ are set to their original values (i.e., the values in $\vec{z}$). The fact that AC2(b) must hold even if only a subset $\vec{W}'$ of the variables in $\vec{W}$ are set to their values in $\vec{w}$ (so that the variables in $\vec{W} - \vec{W}'$ essentially act as they do in the real world; that is, they are allow to vary freely, according to the structural equations, rather than being set to their values in $\vec{w}$) and only a subset of the variables in $\vec{Z}$ are set to their values in the actual world says that we must have φ even if some things happen as they do in the actual world. See Sections 3.1 and 4 for further discussion of and intuition for AC2(b).

The original HP paper [Halpern and Pearl 2001] used a weaker version of AC2(b). Rather than requiring that $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}, \vec{W}' \leftarrow \vec{w}, \vec{Z}' \leftarrow \vec{z}]\varphi$ for all subsets $\vec{W}'$ of $\vec{W}$, it was required to hold only for $\vec{W}$. That is, the following condition was used instead of AC2(b).

AC2(b′) $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}, \vec{W} \leftarrow \vec{w}, \vec{Z}' \leftarrow \vec{z}]\varphi$ for all subsets $\vec{Z}'$ of $\vec{Z}$.

The change from AC2(b′) to AC2(b) may seem rather technical, but it has some nontrivial consequences. One of the contributions of this paper is to examine whether it is necessary; see Section 4 for details.

To deal with other problems in the HP definition, various authors have added the idea of *normality* to the definition. This can be done in a number of ways. I now briefly sketch one way that this can be done, following the approach in [Halpern and Hitchcock 2015]. (See Section 5 for some discussion of the need for normality.)

Take a *world* (in a model M) to be a complete assignment of values to the endogenous variables in M.[3] (See the discussion after Corollary 6.4 for why it is conimportant that a world is an assignment only to the endogenous variables, and not all the variables, including the exogenous variables.) We assume a partial preorder $\succeq$ on worlds, that is, a reflexive transitive relation.[4] Intuitively, if $s \succeq s'$, then s is at least as normal, or typical, as s'. We can use normality in the definition of causality in two ways. Say that a world s is a *witness world* for $\vec{X} = \vec{x}$ being a cause of φ in $(M, \vec{u})$ if there is a witness $(\vec{W}, \vec{w}, \vec{x}')$ to $\vec{X} = \vec{x}$ being a cause of φ and $s = s_{\vec{X}=\vec{x}', \vec{W}=\vec{w}, \vec{u}}$, where $s_{\vec{X}=\vec{x}', \vec{W}=\vec{w}, \vec{u}}$ is the world that results by setting $\vec{X}$ to $\vec{x}'$ and $\vec{W}$ to $\vec{w}$ in context $\vec{u}$. We can then modify AC2(a) so as to require that we consider $\vec{X} = \vec{x}$ to be a cause of φ in $(M, \vec{u})$ only if the witness world s for $\vec{X} = \vec{x}$ being a cause is such that $s \succeq s_{\vec{u}}$, where $s_{\vec{u}}$ is the world determined by context $\vec{u}$; call this modified version AC2(a$^+$). AC2(a$^+$) says that, in determining causality, we consider only possibilities that result from altering atypical features of a world to make them more typical, rather than vice versa. This captures an observation made by Kahneman and Miller [1986] regarding human ascriptions of causality. An *extended causal model* is a causal model together with a preorder $\succeq$ on worlds. Say that $\vec{X} = \vec{x}$ *is a cause of* φ *according to the extended HP definition in* $(M, \vec{u})$, where M is an extended causal model, if $\vec{X} = \vec{x}$ is a cause of φ using AC2(a$^+$) rather than AC2(a).

A somewhat more refined use of normality is to use it to "grade" causes. Say that s is a *best witness* for $\vec{X} = \vec{x}$ being a cause of φ if s is a witness world for $\vec{X} = \vec{x}$ being a cause of φ and there is no other witness world s' for $\vec{X} = \vec{x}$ being a cause of φ such that $s' \succ s$. (Note that there may be more than one best witness.) We can then grade candidate causes according to the normality of their best witnesses (without requiring that there must be a witness s such that $s \succeq s_{\vec{u}}$). Experimental evidence suggests that people are focusing on the cause with the best

[3] In [Halpern and Hitchcock 2015], a world is defined as a complete assignment of values to the *exogenous* variables, but this is a typo.

[4] $\succeq$ is not necessarily a partial order; in particular, it does not necessarily satisfy *antisymmetry* (i.e., $s \succeq s'$ and $s' \succeq s$ does not necessarily imply $s = s'$).

witness (according to their subjective ordering on worlds); see, e.g., [Cushman, Knobe, and Sinnott-Armstrong 2008; Hitchcock and Knobe 2009; Knobe and Fraser 2008].

3 The Examples

In this section, I consider examples due to Spohn [2008], Weslake [2015], Hall [2007], Glymour et al. [2010], and Livengood [2013]. I go through these examples in turn. I set the scene by considering the rock-throwing example mentioned above.

3.1 Throwing rocks at bottles

A naive model of the rock-throwing story just has three *binary* random variables *ST*, *BT*, and *BS* (for "Suzy throws", "Billy throws", and "bottle shatters"). The fact that the variables are binary means that they take values in $\{0, 1\}$. The values of *ST* and *BT* are determined by the context; the value of *BS* given by the equation $BS = ST \vee BT$: the bottle shatters if Suzy or Billy throws.[5] Call this model M_{RT}. For simplicity, suppose that there is just one exogenous variable. Let u be the context that results in $ST = BT = 1$: Suzy and Billy both throw. M_{RT} is described in Figure 1. (Although I have included the exogenous variable here, in later figures exogenous variables are omitted for ease of presentation.)

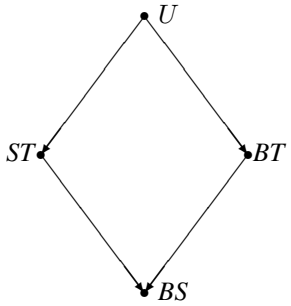

Figure 1: M_{RT}: the naive rock-throwing model.

As already pointed out by Halpern and Pearl [2005], in M_{RT} Suzy and Billy play completely symmetric roles. Not surprisingly, both $ST = 1$ and $BT = 1$ are causes of $BS = 1$ according to the HP definition. Clearly, M_{RT} cannot be used to distinguish a situation where Suzy is a cause from one where Billy is a cause.

In the story as given, people seem to agree that Suzy's throw is a cause and Billy's throw is not, since Suzy's rock hit the bottle and Billy's did not. M_{RT} does not capture this fact. Following Halpern and Pearl [2005], we extend M_{RT} so that it can express the fact that Suzy's rock hit first by adding two more variables:

- *BH* for "Billy's rock hits the (intact) bottle", with values 0 (it doesn't) and 1 (it does); and
- *SH* for "Suzy's rock hits the bottle", again with values 0 and 1.

The equations are such that $SH = ST$ (Suzy's rock hits the bottle if Suzy throws), $BH = BT \wedge \neg SH$ (Billy's rock hits an intact bottle if Billy throws and Suzy's rock does not hit), and $BS = SH \vee BH$ (the bottle shatters if either Suzy's rock or Billy's rock hit it). Now if Suzy and Bill both throw ($ST = 1$ and $BT = 1$), Suzy's rock hits the bottle ($SH = 1$), so that Billy's rock does not hit an intact bottle ($BH = 0$). Call the resulting model M'_{RT}. M'_{RT} is described in Figure 2 (with the exogenous variable omitted).

[5]Here and elsewhere, I follow the fairly standard mathematical convention of eliding the "and only if" in definitions. What is intended here is that the bottle shatters *if and only if* Suzy or Billy throws.

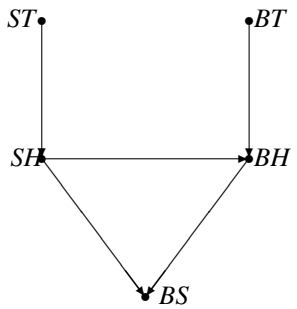

Figure 2: M'_{RT}: the better rock-throwing model.

In this model, $BT = 1$ is *not* a cause. For example, if we take $\vec{Z} = \{BT, BH, BS\}$ in AC2 and set $ST = 0$, then while it is the case that $BS = 0$ if $BT = 0$ and $BS = 1$ if $BT = 1$, it is not the case that $BT = 1$ if we set BH to its original value of 0. Similar arguments work for all other partitions into $\vec{Z}$ and $\vec{W}$. The key is to consider whether BH is in $\vec{W}$ or $\vec{Z}$. If BH is in $\vec{W}$, then how we set BT has no effect on the value BS, so $BT = 1$ cannot be cause. And if BH is in $\vec{W}$, then we get the same problem with with AC2(b^o) or AC2(b^u) as above, since it is easy to see that at least one of SH or ST must in $\vec{W}$, and $\vec{w}$ must be such that whichever is in $\vec{W}$ is set to 0. I leave the details to the reader.

This example shows that it necessary in AC2(b) to allow some variables, but not necessarily all, variables in $\vec{Z}$ to be set to their original values. For example, if we take $\vec{W} = \{ST\}$, $\vec{Z} = \{BT, BH, BS, SH\}$, and $\vec{w} = 0$, to show that $BT = 1$ is not a cause of $BS = 1$, we must set $BH = 0$, its original value, but we do *not* want to set $SH = 1$. Setting BH to 0 captures the intuition that Billy's throw is not a cause because, in the actual world, his rock did not hit the bottle ($BH = 0$). By AC2(b), to establish $BT = 1$ as a cause of $BS = 1$, setting BT to 1 would have to force $BS = 1$ even if $BH = 0$, which is not the case.

3.2 Spohn's example

The next example is due to Spohn [2008].

Example 3.1: There are four endogenous binary variables, A, B, C, and S, taking values 1 (on) and 0 (off). Intuitively, A and B are supposed to be alternative causes of C, and S acts as a switch. If $S = 0$, the causal route from A to C is active and that from B to C is dead; and if $S = 1$, the causal route from A to C is dead and the one from B to C is active. There are no causal relations between A, B, and S; their values are determined by the context. The equation for C is $C = (\neg S \wedge A) \vee (S \wedge B)$.

Suppose that the context is such that $A = B = S = 1$, so $C = 1$. The HP definition yields $B = 1$ and $S = 1$ as causes of $C = 1$, as we would hope. But, unfortunately, it also yields $A = 1$ as a cause of $C = 1$. The argument is that in the contingency where S is set to 0, if $A = 0$, then $C = 0$, while if $A = 1$, then $C = 1$. This does not seem so reasonable. Intuitively, if $S = 1$, then the value of A seems irrelevant to the outcome. Considerations of normality do not help here; all worlds seem to be equally normal.

But now consider a slightly different story. This time, we view B as the switch, rather than S. If $B = 1$, then $C = 1$ if either $A = 1$ or $S = 1$; if $B = 0$, then $C = 1$ only if $A = 1$ and $S = 0$. That is, $C = (B \wedge (A \vee S)) \vee (\neg B \wedge A \wedge \neg S)$. Although this is perhaps not as natural a story as the original, such a switch is surely implementable. In any case, a little playing with propositional logic shows that, in this story, C satisfies exactly the same equation as before: $(\neg S \wedge A) \vee (S \wedge B)$ is equivalent to $(B \wedge (A \vee S)) \vee (\neg B \wedge A \wedge \neg S)$. The key point is that, unlike the first story, in the second story, it seems to me quite reasonable to say that $A = 1$ is a cause of $C = 1$ (as are $S = 1$ and $B = 1$). Having $A = 1$ is necessary for the first "mechanism" to work.

Given that we have different causal intuitions for the stories, we should model them differently. One way to distinguish them is to add two more endogenous random variables, say D and E, that describe the ways that C could be 1. In Spohn's original story, we would have the equation $D = \neg S \wedge A$, $E = S \wedge B$, and $C = D \vee E$. In this model, since $D = 0$ in the actual context, it is not hard to see that $A = 1$ is *not* a cause of $C = 1$, while $B = 1$ and $S = 1$ are, as they should be. Thus, in this model, we correctly capture our intuitions for the story.

To capture the second story, we can add variables D' and E' such that $D' = B \wedge (A \vee S)$, $E' = \neg B \wedge A \wedge S$, and $C = D' \vee E'$. In this model, it is not hard to see that all of $A = 1$, $B = 1$, and $S = 1$ are causes of $C = 1$.

This approach of adding extra variables leads to an obvious question: What is the role of these variables? I view D and E (resp., D' and E') as "structuring" variables, that help an agent "structure" a causal story. Consider Spohn's original story. We can certainly design a circuit where there is a source of power at A and B, a physical switch at S, and a bulb at C that turns on ($C = 1$) if either there is a battery at A ($A = 1$) and the switch is turned left ($S = 0$) or there is battery at B ($B = 1$) and the switch is turned right ($S = 1$). In this physical setup, there is no analogue of D and E. Nevertheless, to the extent that we view the models as a modeler's description of what is going, a modeler could usefully introduce D and E to describe the conditions under which $C = 1$, and to disambiguate this model from one where, conceptually, we might want to think of other ways that C could be 1 (as in the story with D' and E').

Note that we do not want to think of D as being *defined* to take the value 1 if $A = 1$ and $S = 0$. For then we could not intervene to set $D = 0$ if $A = 1$ and $S = 0$. Adding a variable to the model commits us to being able to intervene on it.[6] In the real world, setting D to 0 despite having $A = 1$ and $S = 0$ might correspond to the connection being faulty when the switch is turned left. Indeed, since the equation for C is the same in both stories, it is only at the level of interventions that the difference between the two stories becomes meaningful. ∎

3.3 Weslake's example

The next example is due to Weslake [2015, Example 10].

Example 3.2: A lamp L is controlled by three switches, A, B, and C, each of which has three possible positions, -1, 0, and 1. The lamp switches on iff two or more of the switches are in same position. Thus, $L = 1$ iff $(A = B) \vee (B = C) \vee (A = C)$. Suppose that, in the actual context, $A = 1$, $B = -1$, and $C = -1$. Intuition suggests that while $B = -1$ and $C = -1$ should be causes of $L = 1$, $A = 1$ should not be; since the setting of A does not match that of either B or C, it has no causal impact on the outcome. The HP definition indeed declares $B = -1$ and $C = -1$ to be causes; unfortunately, it also declares $A = 1$ to be a cause. For in the contingency where $B = 1$ and $C = -1$, if $A = 1$ then $L = 1$, while if $A = 0$ then $L = 0$. Adding defaults to the picture does not solve the problem.

Just as in the Spohn example, we can tell another story where the observed variables have the same values, and are connected by the same structural equations. Now suppose that $L = 1$ iff either (a) none of A, B, or C is in position -1, (b) none of A, B, or C is in position 0, or (c) none of A, B, or C is in position 1. It is easy to see that the equations for L are literally the same as in the original example. But now it seems more reasonable to say that $A = 1$ *is* a cause of $L = 1$. Certainly $A = 1$ causes $L = 1$ as a result of no values being 0; had A been 0, then the lamp would still have been on, but now it would be as a result of no values being -1. Considering the contingency where $B = 1$ and $C = -1$ "uncovers" the causal impact of A.

Again, we can capture the distinction between the two stories by adding more variables. For the second story, we can add the variables $NOT(-1)$, $NOT(0)$, and $NOT(1)$, where $NOT(i)$ is 1 iff none of A, B, or C are i. Then $L = NOT(-1) \vee NOT(0) \vee NOT(1)$. Now the HP definition makes $A = 1$ a cause of $L = 1$ (as well as $B = -1$ and $C = -1$). For Weslake's original story we can add the variables $TWO(-1)$, $TWO(0)$, and $TWO(1)$, where $TWO(i) = 1$ iff at least two of A, B, and C are i, and take $L = TWO(-1) \vee TWO(0) \vee TWO(1)$. Now the HP definition does not make $A = 1$ a cause of $L = 1$ (although, of course $B = 1$ and $C = 1$ continue to be causes).

[6] I thank Chris Hitchcock for stressing this point.

Once again, I think of the variables $NOT(-1)$, $NOT(0)$, and $NOT(1)$ (resp., $TWO(-1)$, $TWO(0)$, and $TWO(1)$) as "structuring" variables, that help the modeler distinguish the two scenarios. They are conceptually meaningful even if they don't have a physical analogue. ∎

3.4 Hall's example

Hall's [2007] gives an example that's meant to illustrate how a bad choice of variables leads to unreasonable answers. I repeat it here because, although I agree with his main point (that, indeed, is one of the main points of this paper!), I disagree with one of his conclusions. What I present is actually a slightly simplified version of his example that retains all the necessary features.

Consider a model M with four endogenous variables, A, B, D, and E. The values of A and D are determined by the context. The values of B and E are given by the equations $B = A$ and $E = D$.[7] Suppose that the context u is such that $A = D = 1$. Then clearly, in context (M, u), $A = 1$ is a cause of $B = 1$ and not a cause of $E = 1$, while $D = 1$ is a cause of $E = 1$ and not of $B = 1$. The problem comes if we replace A in the model by X, where intuitively, $X = 1$ iff the context would have been such that A and D agree (i.e., $X = 1$ in the context where $A = D = 1$ or $A = D = 0$). Now we can recover the value of A from that of D and X; it is easy to see that $A = 1$ iff $X = D = 1$ or $X = D = 0$. Thus, we can rewrite the equation for B by taking $B = 1$ iff $X = D = 1$ or $X = D = 0$. Formally, consider a model M' with endogenous variables X, B, D, and E; the context determines the value of X and D; the equation for B is that given above; and we still have the equation $E = D$. Now let u be the context where $X = D = 1$. In (M', u), it is still the case that $D = 1$ is a cause of $E = 1$, but now $D = 1$ is also a cause of $B = 1$.

Hall [2007] says "This result is plainly silly, and doesn't look any less silly if you insist that causal claims must always be relativized to a model." I disagree. To be more precise, I would argue that Hall has in mind a particular picture of the world, that captured by model M. Of course, if that is the "right" picture of the world, the conclusion that $D = 1$ is a cause of $B = 1$ is indeed plainly silly. But consider the following two stories. We are trying to determine the preferences of two people, Betty and Edward, in an election. $B = 1$ if Betty is recorded as preferring the Democrats and $B = 0$ if Betty is recorded as preferring the Republicans, and similarly for E. In the first story, we send Alice to talk to Betty and David to talk to find out their preferences (both are assumed to be truthful and good at finding things out). When Alice reports that Betty prefers the Democrats ($A = 1$) then Betty is reported as preferring the Democrats ($B = 1$); similarly for David and Edward. Clearly, in this story (which is modeled by M) $D = 1$ causes $E = 1$, but not $B = 1$.

But now suppose instead of sending Alice to talk to Betty, Xavier is sent to talk to Carol, who knows only whether Betty and Edward have the same preferences. Carol tells Xavier that they indeed have the same preferences ($X = 1$). Upon hearing that $X = D = 1$, the vote tabulator correctly concludes that $B = 1$. This story is modeled by M'. But in this case it strikes me as perfectly reasonable that $D = 1$ should be a cause of $B = 1$. This is true despite that fact that if we had included the variable A in M', it would have been the case that $A = B = 1$.

3.5 Glymour et al.'s example

The next example is due to Glymour et al. [2010].

Example 3.3: A ranch has five individuals: $a_1, \ldots, a_5$. They have to vote on two possible outcomes: staying at the campfire ($O = 0$) or going on a round-up ($O = 1$). Let A_i be the random variable denoting a_i's vote, so $A_i = j$ if a_i votes for outcome j. There is a complicated rule for deciding on the outcome. If a_1 and a_2 agree (i.e., if $A_1 = A_2$), then that is the outcome. If $a_2, \ldots, a_5$ agree, and a_1 votes differently, then the outcome is given by a_1's vote (i.e., $O = A_1$). Otherwise, majority rules. In the actual situation, $A_1 = A_2 = 1$ and $A_3 = A_4 = A_5 = 0$, so by the first mechanism, $O = 1$. The question is what were the causes of $O = 1$.

[7] Hall [2007] also has variables C and F such that $C = B$ and $F = E$; adding them does not affect any of the discussion here (or in Hall's paper).

Using the naive causal model with just the variables $A_1, \ldots, A_5, O$, and the obvious equations describing O in terms of $A_1, \ldots, A_5$, it is almost immediate that $A_1 = 1$ is a cause of $O = 1$. Changing A_1 to 0 results in $O = 0$. Somewhat surprisingly, in this naive model, $A_2 = 1$, $A_3 = 0$, $A_4 = 0$, and $A_5 = 0$ are also causes.[8] To see that $A_2 = 1$ is a cause, consider the contingency where $A_3 = 1$. Now if $A_2 = 0$, then $O = 0$ (majority rules); if $A_2 = 1$, then $O = 1$, since $A_1 = A_2 = 1$, and $O = 1$ even if A_3 is set back to its original value of 0. To see that $A_3 = 0$ is a cause, consider the contingency where $A_2 = 0$, so that all voters but a_1 vote for 0 (staying at the campsite). If $A_3 = 1$, then $O = 0$ (majority rules). If $A_3 = 0$, then $O = 1$, by the second mechanism (a_1 is the only vote for 0), while if A_2 is set to its original value of 1, then we still have $O = 1$, now by the first mechanism.

But all this talk of mechanisms (which is also implicit in Glymour et al. [2010]; in footnote 11, they say that setting A_2 back to its original value of 1 "brings out the original result, but in a different way") suggests that the mechanism should be part of the model. There are several ways of doing this. One is to add three new variables, call them M_1, M_2, and M_3. These variables have values in $\{0, 1, 2\}$, where $M_j = 0$ if mechanism j is active and suggests an outcome 0, $M_j = 1$ if mechanism j is active and suggests an outcome of 1, and $M_j = 2$ if mechanism j is not active. (We actually don't need the value $M_3 = 2$; mechanism 3 is always active, because there is always a majority with 5 voters, all of whom must vote.) Note that at most one of the first two mechanisms can be active. We have obvious equations linking the value of M_1, M_2, and M_3 to the values of $A_1, \ldots, A_5$.

Now the value of O just depends on the values of M_1, M_2, and M_3: if $M_1 \neq 2$, then $O = M_1$; if $M_2 \neq 2$, then $O = M_2$, and if $M_1 = M_2 = 2$, then $O = M_3$. It is easy to see that in this model, if $A_1 = A_2 = 1$ and $A_3 = A_4 = A_5 = 0$, then none of $A_3 = 0$, $A_4 = 0$, and $A_5 = 0$ is a cause. $A_1 = 1$ is a cause, as we would expect, as are $A_2 = 1$ and $M_2 = 1$. This seems reasonable: the second mechanism was the one that led to the outcome, and it required $A_1 = A_2 = 1$.

Now suppose that we change the description of the voting rule. We take $O = 1$ if one of the following two mechanisms applies:

- $A_1 = 1$ and it is not the case that both $A_2 = 0$ and exactly one of A_3, A_4, and A_5 is 1.
- $A_1 = 0$, $A_2 = 1$, and exactly two of A_3, A_4, and A_5 are 1.

It is not hard to check that, although the description is different, O satisfies the same equation in both stories. But now it does not seem so unreasonable that $A_2 = 1$, $A_3 = 0$, $A_4 = 0$, and $A_5 = 0$ are causes of $O = 1$. And indeed, if we construct a model in terms of these two mechanisms (i.e., add variables M_1' and M_2' that correspond to these two mechanisms), then it is not hard to see that $A_1 = 1$, $A_2 = 1$, $A_3 = 0$, $A_4 = 0$, and $A_5 = 0$ are all causes.

Here the role of the structuring variables M_1, M_2, and M_3 (resp. M_1' and M_2') as descriptors of the mechanism being invoked seems particularly clear. For example, setting $M_1 = 2$ says that the first mechanism will not be applied, even if $A_1 = A_2$; setting $M_1 = 1$ says that we act as if both a_1 and a_2 voted in favor, even if that is not the case. ∎

3.6 Livengood's voting examples

As Livengood [2013] points out, voting can lead to some apparently unreasonable causal outcomes (at least, if we model things naively). He first considers Jack and Jill, who live in an overwhelmingly Republican district. As expected, the Republican candidate wins with an overwhelming majority. Jill would normally have voted Democrat, but did not vote because she was disgusted by the process. Jack would normally have voted Republican, but did not vote because he (correctly) assumed that his vote would not affect the outcome. In the naive model, both Jack and Jill are causes of the Republican victory. For if enough of the people who voted Republican had switched to voting Democrat, then if Jack (or Jill) had voted Democrat, the Democrat would have won, while he would not have won had they abstained. Notice that, in this argument, Jack and Jill are treated the same way; their preferences make no difference.

[8] Glymour et al. point out that $A_1 = 1$, $A_3 = 0$, $A_4 = 0$, and $A_5 = 0$ are causes; they do not mention that $A_2 = 1$ is also a cause.

We can easily construct a model that takes these preferences into account. One way to do so is to assume that their preferences are so strong that we may as well take them for granted. Thus, the preferences become exogenous; the only endogenous variables are whether or not they vote. In this case, Jack's not voting is not a cause of the outcome, but Jill's not voting is.

More generally, with this approach, a voter whose preference is made exogenous and is a strong supporter of the victor does not count as a cause of victory. This does not seem so unreasonable. After all, in an analysis of a close political victory in Congress, when an analyst talks about the cause(s) of victory, she points to the swing voters who voted one way or the other, not the voters that were taken to be staunch supporters of one particular side.

That said, making a variable exogenous seems like a somewhat draconian solution to the problem. It also does not allow us to take into account smaller gradations in depth of feeling. At what point should a preference switch from being endogenous to exogenous? We can achieve the same effect in an arguably more natural way by using normality considerations. In the case of Jack and Jill, we can take voting for a Democrat to be highly abnormal for Jack, and voting for a Republican to be highly abnormal for Jill. To show that either Jack (resp., Jill) is a cause of the victory, we need to consider a contingency where Jack (resp., Jill) votes for the Democratic candidate. This would be a change to a highly abnormal world in the case of Jack, but to a more normal world in the case of Jill. Thus, if we use normality as a criterion for determining causality, Jill would count as a cause, but Jack would not. If we use normality as a way of grading causes, Jack and Jill would still both count as causes for the victory, but Jill would be a much better cause. More generally, the more normal it would be for someone to vote Democrat, the better a cause that voter would be. The use of normality here allows for a more nuanced gradation of cause than the rather blunt approach of either making a variable exogenous or endogenous.

Now, following Livengood [2013], consider a vote where everyone can either vote for one of three candidates. Suppose that the actual vote is 17–2–0 (i.e., 17 vote for candidate A, 2 for candidate B, and none for candidate C). Then not only is every vote for candidate A a cause of A winning, every vote for B is also a cause of A winning. To see this, consider a contingency where 8 of the voters for A switch to C. Then if one of the voters for B votes for C, the result is a tie; if that voter switches back to B, then A wins (even if some subset of the voters who switch from A to C switch back to A).

Is this reasonable? What makes it seem particularly unreasonable is that if it had just been a contest between A and B, with the vote 17–2, then the voters for B would not have been causes of A winning. Why should adding a third option make a difference?

In some cases it does seem reasonable that adding a third option makes a difference. For example, we speak of Nader costing Gore a victory over Bush in the 2000 election. But, as Livengood [2013] points out, we don't speak of Gore costing Nader a victory, although in a naive HP model of the situation, all the voters for Gore are causes of Nader not winning as much as the voters for Nader are causes of Gore not winning. The discussion above points a way out of this dilemma. If a sufficiently large proportion of Bush and Gore voters are taken to be such strong supporters that they will never change their minds, and we make their votes exogenous, then it is still the case that Nader caused Gore to lose, but not the case that Gore caused Nader to lose. Similar considerations apply in the case of the 17–2 vote. (Again, we can use normality considerations to give arguably more natural models of these examples.)[9]

4 Do we need AC2(b)?

In this section, I consider the extent to which we can use AC2(b$'$) rather than AC2(b), and whether this is a good thing.

[9] As a separate matter, most people would agree that Nader entering the race was a cause of Gore not winning, while Gore entering the race was not a cause of Nader not winning. Here the analysis is different. If Nader hadn't entered, it seems reasonable to assume that there would have been no other strong third-party candidate, so just about all of Nader's votes would have gone to Bush or Gore, with the majority going to Gore. On the other hand, if Gore hadn't entered, there would have been another Democrat in the race replacing him, and most of Gore's votes would have gone to the new Democrat in the race, rather than Nader.

4.1 The Hopkins-Pearl example

I start by examining the Hopkins-Pearl example that was intended to show that AC2(b′) was inappropriate. The following description is taken from [Halpern and Pearl 2005].

Example 4.1: Suppose that a prisoner dies either if A loads B's gun and B shoots, or if C loads and shoots his gun. Taking D to represent the prisoner's death and making the obvious assumptions about the meaning of the variables, we have that $D = (A \wedge B) \vee C$. Suppose that in the actual context u, A loads B's gun, B does not shoot, but C does load and shoot his gun, so that the prisoner dies. That is, $A = 1$, $B = 0$, and $C = 1$. Clearly $C = 1$ is a cause of $D = 1$. We would not want to say that $A = 1$ is a cause of $D = 1$, given that B did not shoot (i.e., given that $B = 0$). However, suppose that we take the obvious model with the random variables A, B, C, D. With AC2(b′), $A = 1$ is a cause of $D = 1$. For we can take $\vec{W} = \{B, C\}$ and consider the contingency where $B = 1$ and $C = 0$. It is easy to check that AC2(a) and AC2(b′) hold for this contingency, so under the original HP definition, $A = 1$ is a cause of $D = 1$. However, AC2(b) fails in this case, since $(M, u) \models [A \leftarrow 1, C \leftarrow 0](D = 0)$. The key point is that AC2(b) says that for $A = 1$ to be a cause of $D = 1$, it must be the case that $D = 0$ if only some of the values in $\vec{W}$ are set to $\vec{w}$. That means that the other variables get the same value as they do in the actual context; in this case, by setting only A to 1 and leaving B unset, B takes on its original value of 0, in which case $D = 0$. AC2(b′) does not consider this case.

Nevertheless, as pointed out by Halpern and Hitchcock [2015], we can use AC2(b′) if we have the "right" model. Suppose that we add a new variable E such that $E = A \wedge B$, so that $E = 1$ iff $A = B = 1$, and set $D = E \vee C$. Thus, we have captured the intuition that there are two ways that the prisoner dies. Either C shoots, or A loads and B fires (which is captured by E). It is easy to see that (using either AC2(b) or AC2(b′)) $B = 0$ is *not* a cause of $D = 1$. ∎

As I now show, the ideas of this example generalize. But before doing that, I define the notion of a conservative extension.

4.2 Conservative extensions

In the rock-throwing example, adding the extra variables converted $BT = 1$ from being a cause to not being a cause of $BS = 1$. Similarly, adding extra variables affected causality in all the other examples above. Of course, without any constraints, it is easy to add variables to get any desired result. For example, consider the rock-throwing model M'_{RT}. Suppose that we add a variable BH_1 with equations that set $BH_1 = BT$ and $BS = SH \vee BT \vee BH_1$. This results in a new "causal path" from BT to BS going through BH_1, independent of all other paths. Not surprisingly, in this model, $BT = 1$ is indeed a cause of $BS = 1$.

But this seems like cheating. Adding this new causal path fundamentally changes the scenario; Billy's throw has a new way of affecting whether or not the bottle shatters. While it seems reasonable to refine a model by adding new information, we want to do so in a way that does not affect what we know about the old variables. Intuitively, suppose that we had a better magnifying glass and could look more carefully at the model. We might discover new variables that were previously hidden. But we want it to be the case that any setting of the old variables results in the same observations. That is, while adding the new variable refines the model, it does not fundamentally change it. This is made precise in the following definition.

Definition 4.2: A causal model $M' = ((\mathcal{U}', \mathcal{V}', \mathcal{R}'), \mathcal{F}')$ is a *conservative extension* of $M = ((\mathcal{U}, \mathcal{V}, \mathcal{R}), \mathcal{F})$ if $\mathcal{U} = \mathcal{U}'$, $\mathcal{V} \subseteq \mathcal{V}'$, and, for all contexts $\vec{u}$, all variables $X \in \mathcal{V}$, and all settings $\vec{w}$ of the variables in $\vec{W} = \mathcal{V} - \{X\}$, we have $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X = x)$ iff $(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X = x)$. That is, no matter how we set the variables other than X, X has the same value in context $\vec{u}$ in both M and M'. ∎

According to the definition, M' is a conservative extension of M iff, for certain formulas ψ involving only variables in $\mathcal{V}$, namely, those of the form $[\vec{W} \leftarrow \vec{w}](X = x)$, $(M, \vec{u}) \models \psi$ iff $(M', \vec{u}) \models \psi$. As the following lemma shows, this is actually true for all formulas involving only variables in $\mathcal{V}$, not just ones of a special form.

Lemma 4.3: *Suppose that M' is a conservative extension of $M = ((\mathcal{U}, \mathcal{V}, \mathcal{R}), \mathcal{F})$. Then for all causal formulas φ that mention only variables in $\mathcal{V}$ and all contexts $\vec{u}$, we have $(M, \vec{u}) \models \varphi$ iff $(M', \vec{u}) \models \varphi$.*

Proof: Since M is a recursive model, there is some total order $\preceq$ on the endogenous variables such that, unless $X \preceq Y$, Y is independent of X in M; that is, unless $X \preceq Y$, changing the value of X has no impact on the value of Y according to the structural equations in M, no matter what the setting of the other variables. It is almost immediate from the definition of conservative extension that, for all $X, Y \in \mathcal{V}$, Y is independent of X in M iff Y is independent of X in M'. Also note that if $X \prec Y$, then it is not the case that $Y \prec X$, so if $X \prec Y$, then X is independent of Y (in both $(M, \vec{u})$ and $(M', \vec{u})$). Say that X is independent of a set $\vec{W}$ of endogenous variables in $(M, \vec{u})$ if X is independent of Y in $(M, \vec{u})$ for all $Y \in \vec{W}$.

Suppose that $\mathcal{V} = \{X_1, \ldots, X_n\}$. Since M is a recursive model, we can assume without loss of generality that these variables are ordered so that $X_1 \prec \cdots \prec X_n$ I now prove by induction on j that, for all $\vec{W} \subseteq \mathcal{V}$, all settings $\vec{w}$ of the variables in $\vec{W}$, all contexts $\vec{u}$, and all $x_j \in \mathcal{R}(X_j)$, we have $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_j = x_j)$ iff $(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_j = x_j)$.

For the base case of the induction, given $\vec{W}$, let $\vec{W}' = \mathcal{V} - (\vec{W} \cup \{X_1\})$, and let $\vec{w}'$ be an arbitrary setting of the variables in $\vec{W}'$. Then we have

	$(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_1 = x_1)$	
iff	$(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}' \leftarrow \vec{w}'](X_1 = x_1)$	[since X_1 is independent of $\vec{W}$ in $(M, \vec{u})$]
iff	$(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}' \leftarrow \vec{w}'](X_1 = x_1)$	[since M' is a conservative extension of M]
iff	$(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_1 = x_1)$	[since X_1 is independent of $\vec{W}$ in $(M', \vec{u})$].

This completes the proof of the base case. Suppose that $1 < j < n$ and the result holds for $1, \ldots, j-1$; I prove it for j. Given $\vec{W}$, now let $\vec{W}' = \mathcal{V} - (\vec{W} \cup \{X_j\})$, let $\vec{W}'_1 = \vec{W}' \cap \{X_1, \ldots, X_{j-1}\}$, and let $\vec{W}'_2 = \vec{W}' - \vec{W}'_1$. Since $\vec{W}'_2$ is contained in $\{X_{j+1}, \ldots, X_n\}$, X_j is independent of $\vec{W}'_2$ in $(M, \vec{u})$ and in $(M', \vec{u})$. Choose $\vec{w}'_1$ such that $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](\vec{W}'_1 = \vec{w}'_1)$. Since $\vec{W}'_1 \subseteq \{X_1, \ldots, X_{j-1}\}$, by the induction hypothesis, $(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}](\vec{W}'_1 = \vec{w}'_1)$. It easily follows that we have $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_j = x_j)$ iff $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}'_1 \leftarrow \vec{w}'_1](X_j = x_j)$, and similarly for M'. Thus,

	$(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_j = x_j)$	
iff	$(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}'_1 \leftarrow \vec{w}'_1](X_j = x_j)$	[as observed above]
iff	$(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}'_1 \leftarrow \vec{w}'_1, \vec{W}'_2 \leftarrow \vec{w}'_2](X_j = x_j)$	[since X_j in independent of $\vec{W}'_2$ in $(M, \vec{u})$]
iff	$(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}'_1 \leftarrow \vec{w}'_1, \vec{W}'_2 \leftarrow \vec{w}'_2](X_j = x_j)$	[since M' is a conservative extension of M]
iff	$(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}, \vec{W}'_1 \leftarrow \vec{w}'_1](X_j = x_j)$	[since X_j is independent of $\vec{W}'_2$ in $(M', \vec{u})$]
iff	$(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}](X_j = x_j)$	[as observed above].

This completes the proof of the inductive step.

Since, in general $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](\psi_1 \wedge \psi_2)$ iff $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}]\psi_1 \wedge [\vec{W} \leftarrow \vec{w}]\psi_2$ and $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}]\neg\psi_1$ iff $(M, \vec{u}) \models \neg[\vec{W} \leftarrow \vec{w}]\psi_1$, and similarly for M', an easy induction shows that $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}]\psi$ iff $(M', \vec{u}) \models [\vec{W} \leftarrow \vec{w}]\psi$ for an arbitrary Boolean combination ψ of primitive events that mentions only variables in $\mathcal{V}$. Another easy induction shows that $(M, \vec{u}) \models \psi$ iff ∎

4.3 Avoiding AC2(b)

I now show that we can always use AC2(b′) instead of AC2(b), if we add extra variables.

Theorem 4.4: *If $X = x$ is not a cause of $Y = y$ in $(M, \vec{u})$ using AC2(b), but is a cause using AC2(b′), then there is a model M' that is a conservative extension of M such that $X = x$ is not a cause of $Y = y$ using AC2(b′).*

Proof: Suppose that $(\vec{W}, \vec{w}, x')$ is a witness to $X = x$ being a cause of $Y = y$ in $(M, \vec{u})$ using AC2(b′). Let $(M, \vec{u}) \models \vec{W} = \vec{w}^*$. We must have $\vec{w} \neq \vec{w}^*$, for otherwise it is easy to see that $X = x$ would be a cause of $Y = y$ in $(M, \vec{u})$ using AC(2b) with witness $(\vec{W}, \vec{w}, x')$.

If M' is a conservative extension of M with additional variables $\mathcal{V}'$, say that $(\vec{W}', \vec{w}', x')$ *extends* $(\vec{W}, \vec{w}, x')$ if $\vec{W} \subseteq \vec{W}' \subseteq \vec{W} \cup \mathcal{V}'$ and $\vec{w}'$ agrees with $\vec{w}$ on the variables in $\vec{W}$.

I now construct a conservative extension M' of M in which $X = x$ is not a cause of $Y = y$ using AC2(b′) with a witness extending $(\vec{W}', \vec{w}, x')$. Of course, this just kills one witness. I then show that we can construct further extensions to kill all other witnesses to $X = x$ being a cause of $Y = y$ using AC2(b′).

Let M' be obtained from M by adding one new variable N. All the variables have the same equations in M and M' except for Y and (of course) N. The equations for N are easy to explain: if $X = x$ and $\vec{W} = \vec{w}$, then $N = 1$; otherwise, $N = 0$. The equations for Y are the same in M and M' (and do not depend on the value of N) except for two special cases. To define these cases, for each variable $Z \in \mathcal{V} - \vec{W}$, if $x'' \in \{x, x'\}$, define $z_{x'', \vec{w}}$ as the value such that $(M, \vec{u}) \models [X \leftarrow x'', \vec{W} \leftarrow \vec{w}](Z = z_{x'', \vec{w}})$. That is, $z_{x'', \vec{w}'}$ is the value taken by Z if X is set to x'' and $\vec{W}$ is set to $\vec{w}$. Let $\vec{V}'$ consist of all variables in $\mathcal{V}$ other than Y, let $\vec{v}'$ be a setting of the variables in $\vec{V}'$, and let $\vec{Z}'$ consist of all variables in $\vec{V}' - \vec{W}$ other than X. Then we want the equations for Y in M' to be such that for all $j \in \{0, 1\}$, we have

$$(M, \vec{u}) \models [\vec{V}' \leftarrow \vec{v}'](Y = y'') \text{ iff}$$
$$(M', \vec{u}) \models [\vec{V}' \leftarrow \vec{v}', N \leftarrow j](Y = y'')$$

unless the assignment $\vec{V}' \leftarrow \vec{v}'$ results in either (a) $X = x$, $\vec{W} = \vec{w}$, $Z = z_{x, \vec{w}}$ for all $Z \in \vec{Z}'$, and $N = 0$ or (b) $X = x'$, $\vec{W} = \vec{w}$, $Z = z_{x', \vec{w}}$ for all $Z \in \vec{Z}'$, and $N = 1$. (Note that in both of these cases, the value of N is "abnormal". If $X = x$, $\vec{W} = \vec{w}$ and $Z = z_{x, \vec{w}}$ for all $z \in \vec{Z}'$, then N should be 1; if we set X to x' and change the values of the variables in $\vec{Z}'$ accordingly, then N should be 0.) If (a) holds, $Y = y'$ in M'; if (b) holds, $Y = y$.

I now show that M' has the desired properties and, in addition, does not make $X = x$ a cause in new ways.

Lemma 4.5:

(a) It is not the case that $X = x$ is a cause of $Y = y$ using AC2(b′) in $(M', \vec{u})$ with a witness that extends $(\vec{W}, \vec{w}, x')$.

(b) M' is a conservative extension of M.

(c) If $X = x$ is a cause of $Y = y$ in $(M', \vec{u})$ using AC2(b) (resp. AC2(b′)) with a witness extending $(\vec{W}', \vec{w}', x'')$ then $X = x$ is a cause of $Y = y$ in $(M, \vec{u})$ using AC2(b) (resp. AC2(b′)) with witness $(\vec{W}', \vec{w}', x'')$.

Proof: For part (a), suppose, by way of contradiction, that $X = x$ is a cause of $Y = y$ using AC2(b′) in $(M', \vec{u})$ with a witness $(\vec{W}', \vec{w}', x')$ that extends $(\vec{W}, \vec{w}, x')$. If $N \notin \vec{W}'$, then $\vec{W}' = \vec{W}$. But then, since $(M', \vec{u}) \models N = 0$ and $(M', \vec{u}) \models [X \leftarrow x, \vec{W} \leftarrow \vec{w}, N = 0](Y = y')$, it follows that $(M', \vec{u}) \models [X \leftarrow x, \vec{W} \leftarrow \vec{w}](Y = y')$, so AC2(b′) fails, contradicting the assumption that $X = x$ is a cause of $Y = y$. Now suppose that $N \in \vec{W}'$. There are two cases, depending on how the value of N is set in $\vec{w}'$. If $N = 0$, then again, since $(M', \vec{u}) \models [X \leftarrow x, \vec{W} \leftarrow w, N \leftarrow 0](Y = y')$, AC2(b′) fails; and if $N = 1$, then since $(M', \vec{u}) \models [X \leftarrow x', \vec{W} \leftarrow \vec{w}, N \leftarrow 1](Y = y)$, AC2(a) fails. So, in all cases, we get a contradiction to the assumption that $X = x$ is a cause of $Y = y$ using AC2(b′) in $(M', \vec{u})$ with a witness $(\vec{W}', \vec{w}', x')$ that extends $(\vec{W}, \vec{w}, x')$.

For part (b), note that the only variable in $\mathcal{V}$ for which the equations in M and M' are different is Y. Consider any setting of the variables in $\mathcal{V}$ other than Y. Except for the two special cases noted above, the value of Y is clearly the same in M and M'. But for these two special cases, as was noted above, the value of N is "abnormal", that is, it is not the same as its value according to the equations given the setting of the other variables. It follows that for all settings $\vec{v}$ of the variables $\vec{V}'$ in $\mathcal{V}$ other than Y and all values y'' of Y, we have $(M, \vec{u}) \models (\vec{V}' \leftarrow \vec{v}](Y = y'')$ iff $(M', \vec{u}) \models (\vec{V}' \leftarrow \vec{v}](Y = y'')$. Thus, M' is a conservative extension of M.

For part (c), suppose that $X = x$ is a cause of $Y = y$ in $(M', \vec{u})$ using AC2(b) (resp. AC2(b′)) with witness $(\vec{W}'', \vec{w}'', x'')$. Let $\vec{W}'$ and $\vec{w}'$ be the restrictions of $\vec{W}''$ and $\vec{w}''$, respectively, to the variables in $\mathcal{V}$. If $N \notin \vec{W}''$ (so that $\vec{W}'' = \vec{W}'$) then, since M' is a conservative extension of M, it easily follows that $(\vec{W}', \vec{w}', x'')$ is a witness to $X = x$ being a cause of $Y = y$ in $(M, \vec{u})$ using AC2(b) (resp. AC2(b′)). If $N \in \vec{W}''$, it suffices to show that $(\vec{W}', \vec{w}', x'')$ is also a witness to $X = x$ being a cause of $Y = y$ in $(M', \vec{u})$; that is, N does not play an essential role in the witness. I now do this.

If $N = 0$ is a conjunct of $\vec{W}'' = \vec{w}''$, since the equations for Y are the same in M and M' except for two cases, the only way that $N = 0$ can play an essential role in the witness is if setting $\vec{W}' = \vec{w}'$ and $X = x$ results in $\vec{W} = \vec{w}$ and $Z = z_{x,\vec{w}}$ for all $Z \in \vec{Z}'$ (i.e., we are in the first of the two cases where the value of Y does not agree in $(M, \vec{u})$ and $(M', \vec{u})$). But then $Y = y'$, so if this were the case, AC2(b) (and hence AC2(b′)) would not hold. Similarly, if $N = 1$ is a conjunct of $\vec{W}'' = \vec{w}''$, N plays a role only if $x'' = x'$ and setting $\vec{W}' = \vec{w}'$ and $X = x'$ results in results in $\vec{W} = \vec{w}$ and $Z = z_{x',\vec{w}}$ for all $Z \in \vec{Z}'$ (i.e., we are in the second of the two cases where the value of Y does not agree in $(M, \vec{u})$ and $(M', \vec{u})$). But then $Y = y$, so if this were the case, AC2(a) would not hold, and again we would have a contradiction to $X = x$ being a cause of $Y = y$ in $(M', \vec{u})$ with witness $(\vec{W}'', \vec{w}'', x'')$. Thus, $(\vec{W}', \vec{w}', x'')$ must be a witness to $X = x$ being cause of $Y = y$ in $(M', \vec{u})$, and hence also in $(M, \vec{u})$. This completes the proof of part (c). ∎

Lemma 4.5 is not quite enough to complete the proof of Theorem 4.4. There may be several witnesses to $X = x$ being a cause of $Y = y$ in $(M, \vec{u})$ using AC2(b′). Although we have removed one of the witnesses, some others may remain, so that $X = x$ may still be a cause of $Y = y$ in $(M', \vec{u})$. But by Lemma 4.5(c), if there is a witness to $X = x$ being a cause of $Y = y$ in $(M', \vec{u})$, it must extend a witness to $X = x$ being a cause of $Y = y$ in $(M, \vec{u})$. We can repeat the construction of Lemma 4.5 to kill this witness as well. Since there are only finitely many witnesses to $X = x$ being a cause of $Y = y$ in $(M, \vec{u})$, after finitely many extensions, we can kill them all. After this is done, we have a causal model M^* extending M such that $X = x$ is not a cause of $Y = y$ in $(M^*, \vec{u})$ using AC2(b′). ∎

It is interesting to apply the construction of Theorem 4.4 to Example 4.1. The variable N added by the construction is almost identical to E. Indeed, the only difference is that $N = 0$ if $A = B = C = 1$, while $E = 1$ in this case. But since $D = 1$ if $A = B = C = 1$ and $N = 0$, the equations for D are the same in both causal models if $A = B = C = 1$. While it seems strange, given our understanding of the meaning of the variables, to have $N = 0$ if $A = B = C = 1$, it is easy to see that this definition works equally well in showing that $A = 1$ is not a cause of $D = 1$ using AC2(b′) in the context where $A = 1$, $B = 0$, and $C = 1$.

4.4 Discussion

Theorem 4.4 suggests that, by adding extra variables appropriately, we can go back to the definition of causality using AC2(b′) rather than AC2(b). This has some technical advantages. For example, with AC2(b′), causes are always single conjuncts [Eiter and Lukasiewicz 2002; Hopkins 2001]. As shown in [Halpern 2008], this is not in general the case with AC2(b); it may be that $X_1 = x_1 \wedge X_2 = x_2$ is a cause of $Y = y$ with neither $X_1 = x_1$ nor $X_2 = x_2$ being causes (see also Example 6.6). It also seems that testing for causality is harder using AC2(b). Eiter and Lukasiewicz [2002] show that, using AC2(b′), testing for causality is NP-complete for binary models (where all random variables are binary) and Σ_2-complete in general; with AC2(b), it seems to be Σ_2-complete in the binary case and Π_3-complete in the general case [Aleksandrowicz, Chockler, Halpern, and Ivrii 2014].

On the other hand, adding extra variables may not always be a natural thing to do. For example, in Beer et al.'s [2012] analysis of software errors using causality, the variables chosen for the analysis are determined by the program specification. Moreover, Beer et al. give examples where AC2(b) is needed to get the intuitively correct answer. Unless we are given a principled way of adding extra variables so as to be able to always use AC2(b′), it is not clear how to automate an analysis. In addition, as we saw above, adding the extra variable N as in Theorem 4.4 rather than E result in an "unnatural" model. There does not always seem to be a "natural" way of adding extra variables so that AC2(b′) suffices (even assuming that we can agree on what "natural" means!).

Adding extra variables also has an impact on complexity. Note that, in the worst case, we may have to add an extra variable for each pair (W, w) such that there is a witness (W, w, x') for $X = x$ being a cause of $Y = y$. In all the standard examples, there are very few witnesses (typically 1–2), but I have been unable to prove a nontrivial bound on the number of witnesses.

More experience is needed to determine which of AC2(b) and AC2(b′) is most appropriate. Fortunately, in many cases, the causality judgment is independent of which we use.

5 Normality

As was already observed in [Halpern and Hitchcock 2015], the example that motivated the use of normality considerations can also be dealt with by adding variables to the model in an arguably reasonable way. Consider the following example, given by Hitchcock [2007], based on an example due to Hiddleston [2005].

Example 5.1: Assassin is in possession of a lethal poison, but has a last-minute change of heart and refrains from putting it in Victim's coffee. Bodyguard puts antidote in the coffee, which would have neutralized the poison had there been any. Victim drinks the coffee and survives. Is Bodyguard's putting in the antidote a cause of Victim surviving? Most people would say no, but according to the HP definition (with either AC2(b) or AC2(b′)), it is. For in the contingency where Assassin puts in the poison, Victim survives iff Bodyguard puts in the antidote. ∎

What makes this particularly troubling is that the obvious naive model is isomorphic to the naive model in the rock-throwing example (illustrated in Figure 1). Specifically, if we take A (for "assassin does not put in poison"), B (for "bodyguard puts in antidote"), and VS (for "victim survives"), then $VS = A \vee B$, just as $BS = ST \vee BT$. However, while most people agree that $ST = 1$ a cause of $BS = 1$ in this, they do not view $A = 1$ as a cause of $VS = 1$. Using normality considerations, we can say that $A = 1$ is not a cause because the witness world, where $A = 0$, is less normal than the actual world. It is not normal to put poison in coffee. But would we feel differently in a universe where poisoning occurred frequently, or was normal in the sense that it was accepted practice?

Arguably a better solution to this problem, already suggested in [Halpern and Hitchcock 2015], is to add an additional variable. Suppose we add a variable PN to the model, representing whether a chemical reaction takes place in which poison is neutralized, where $PN = \neg A \wedge B$ (A puts in the poison and B puts in the antidote) and $VS = A \vee PN$, it is easy to check that now $B = 1$ is no longer a cause of $VS = 1$. Intuitively, the antidote is a cause of the victim living only if it actually neutralized the poison.

Blanchard and Schaffer [2013] have used this example and others to argue that we do not need to use normality at all in determining causality. I do not agree. As we have seen, thinking in terms of normality helps in the Livengood voting example; there are many other examples given in [Halpern and Hitchcock 2015] where the use of normality, and in particular the ability to use normality to allow for gradation of causality, seems to be helpful. Moreover, as I mentioned earlier, people seem to take normality considerations into account. Finally, in the case of normality, we do not yet have an analogue to Theorem 4.4 that says that we can always add extra variables to remove the need for normality. There may well be examples where normality solves the problem, while no number of extra variables will deal with it.

6 The Stability of (Non-)Causality

The examples in Section 3 raise a potential concern. Consider the rock-throwing example again. Adding extra variables changed $BT = 1$ from being a cause of $BS = 1$ to not being a cause. Could adding even more variables convert $BT = 1$ back to being a cause? Could it then alternate further?

These questions of stability have been raised before. Strevens [2008] provides an example where what Strevens calls a cause can become a non-cause if extra variables are added according to Woodward's [2003]

definition of causality;[10] Eberhardt [2014] shows that this can also happen for *type causality* ("smoking causes cancer" rather than "Mr T.'s smoking for 20 years caused him to get cancer") using Woodward's definition. Here I consider the situation in more detail for the HP definition and show that it can get much worse. In general, we can convert an event from being a cause to a non-cause and then back again infinitely often.

Consider an arbitrary model M with variables A and B and a context u such that $(M, u) \models A = 1 \wedge B = 1$, but $A = 1$ is not a cause of $B = 1$ in (M, u). I now show how to extend M in a conservative way so as to make $A = 1$ a cause of $B = 1$. Add a new binary variable to M, say X_1, to get a model M'. Normally $X_1 = 1$. The equations for all variables are the same in M and M' unless $A = X_1 = 0$. If $A = X_1 = 0$, then $B = 0$. But if $A = 1$ then $B = 1$, no matter what the value of X_1. It easily follows that $A = 1$ is a cause of $B = 1$, with witness $(\{X_1\}, 0, 0)$. It is then not hard to then add a variable Y_1 to "neutralize" the effect of X_1, so that $A = 1$ is not a cause of $B = 1$. Repeating this construction infinitely often, we get a sequence of models where the the answer to the question of whether $A = 1$ is a cause of $B = 1$ alternates infinitely often.

I now formalize this. Specifically, I construct a sequence $M_0, M_1, M_2, \ldots$ of causal models and a context u such that M_{n+1} is a conservative extension M_n, $A = 1$ is not a cause of $B = 1$ in the causal settings (M_n, u) where n is even and $A = 1$ is a cause of $B = 1$ in the the causal settings (M_n, u) where n is odd. That is, the answer to the question "Is $A = 1$ a cause of $B = 1$?" alternates as we go along the sequence of models.

M_0 is just the model with two binary endogenous variables A and B with one binary exogenous variable U. The variables A and B are independent of each other; their value is completely determined by the context. In the context u_1 where $U = 1$, $A = B = 1$. In the context u_0 where $U = 0$, $A = B = 0$. Clearly, $A = 1$ is not a cause of $B = 1$ in (M_0, u_1).

The models $M_1, M_2, M_3, \ldots$ are defined inductively. For $n \geq 0$, we get M_{2n+1} from M_{2n} by adding a new variable X_{n+1}; we get M_{2n+2} from M_{2n+1} by adding a new variable Y_{n+1}. Thus, for $n \geq 0$, the model M_{2n+1} has the endogenous variables $A, B, X_1, \ldots, X_{n+1}, Y_1, \ldots, Y_n$ and the model M_{2n+2} has the endogenous variables $A, B, X_1, \ldots, X_{n+1}, Y_1, \ldots, Y_{n+1}$. All these models have just one binary exogenous variable U. For $n \geq 0$, the exogenous variable determines the value of $A, X_1, \ldots, X_{n+1}$ in models M_{2n+1} and M_{2n+2}; in the context u_j, these variables all have value j. In addition, in u_0, $B = 0$, no matter how the other variables are set. If $n \geq 1$, then in M_{2n} and M_{2n+1}, the equation for Y_j is just $Y_j = X_j$, $j = 1, \ldots, n$; X_j determines Y_j. In (M_{2n-1}, u_1), $B = 1$ unless either (a) $A = 0$ and either $X_n = 0$ or for some $j < n$, $X_j = Y_j = 0$, or (b) $A = 1$ and $X_j \neq Y_j$ for some $j < n$. In M_{2n}, if $U = 1$, then $B = 1$ unless either (a) $A = 0$ and for some $j \leq n$, $X_j = Y_j = 0$ or (b) $A = 1$ and $X_j \neq Y_j$ for some $j \leq n$. Intuitively, $B = 1$ unless $A = 0$ and X_j and Y_j both take on the exceptional value 0 (or just X_n does, if there is no corresponding Y_n), or $A = 1$ and X_j is different from Y_j (which is also an exceptional circumstance).

Theorem 6.1: *For all $n \geq 0$, M_{n+1} is a conservative extension of M_n. Moreover, $A = 1$ is not a cause of $B = 1$ in (M_{2n}, u_1) and $A = 1$ is a cause of $B = 1$ in (M_{2n+1}, u_1).*

Proof: Fix $n \geq 0$. To see that M_{2n+1} is a conservative extension of M_{2n}, note that for the variables $A, B, X_1, \ldots, X_n, Y_1, \ldots, Y_n$ that appear in both M_{2n} and M_{2n+1}, the equations for all variables but B are the same in M_{2n} and M_{2n+1}. It thus clearly suffices to show that, no matter what the value of U, for every setting of the variables $A, X_1, \ldots, X_n, Y_1, \ldots, Y_n$, the value of B is the same in both M_{2n} and M_{2n+1}.[11] If $U = 0$, $B = 0$ in both M_{2n} and M_{2n+1}. If $U = 1$, in M_{2n+1}, no matter how $A, X_1, \ldots, X_n, Y_1, \ldots, Y_n$ are set, $X_{n+1} = 1$. And if $X_{n+1} = 1$, then the value of B depends on the values of $A, X_1, \ldots, X_n, Y_1, \ldots, Y_n$ in M_{2n+1} in the same way that it does in M_{2n}.

The argument that M_{2n+2} is a conservative extension of M_{2n+1} is almost identical. Now we have to show that, no matter what the value of U, for every setting of the variables $A, X_1, \ldots, X_{n+1}, Y_1, \ldots, Y_n$, the value of B is the same in both M_{2n+1} and M_{2n+2}. Again, this is immediate if $U = 0$. If $U = 1$, since $Y_{n+1} = X_{n+1}$ in M_{2n+2}, the result again follows easily.

[10] Actually, Strevens considered what Woodward called a *contributing cause*.

[11] Of course, if $n = 0$, there are no variables $X_1, \ldots, X_n, Y_1, \ldots, Y_n$, so it suffices to show that for all settings of A, the value of B is the same in M_0 and M_1. A similar comment applies elsewhere when $n = 0$.

To see that $A = 1$ is a cause of $B = 1$ in (M_{2n+1}, u_1), take $\vec{W} = \{X_{n+1}\}$. It is immediate that $(M_{2n+1}, u_1) \models [A \leftarrow 0, X_{n+1} \leftarrow 0](B = 0)$, so AC2(a) holds. Moreover, $(M_{2n+1}, u_1) \models [A \leftarrow 1, X_{n+1} \leftarrow 0](B = 1)$ and $(M_{2n+1}, u_1) \models [A \leftarrow 1, X_{n+1} \leftarrow 1](B = 1)$, so AC2(b) holds. (Note that all of $X_1, \ldots, X_n, Y_1, \ldots, Y_n$ get the same value if the context $U = 1$ whether or not X_{n+1} is set to 1.)

Finally, to see that $A = 1$ is not a cause of $B = 1$ in (M_{2n}, u_1), suppose, by way of contradiction, it is a cause, with witness $(\vec{W}, \vec{w}, 0)$. For AC2(a) to hold, we must have $(M_{2n}, u_1) \models [A \leftarrow 0, \vec{W} \leftarrow \vec{w}](B = 0)$. Thus, there must some $j < n$ such that $\{X_j, Y_j\} \subseteq \vec{W}$ and $\vec{w}$ is such that X_j and Y_j are set to 0. But then let $\vec{W}' = \vec{W} - \{X_j\}$. Then we must have $(M_{2n}, u_1) \models [A \leftarrow 1, \vec{W}' \leftarrow \vec{w}](B = 0)$, because if $U = 1$, then X_j is set to 1, and this is not overridden by $\vec{W}'$, and if $A = 1$, $X_j = 1$, and $Y_j = 0$, then $B = 0$. Thus, AC2(b) does not hold. This completes the argument. ∎

Theorem 6.1 is somewhat disconcerting. It seems that looking more and more carefully at a situation should not result in our view of $X = x$ being a cause of $Y = y$ alternating between "yes" and "no", at least, not if we do not discover anything inconsistent with our understanding of the relations between previously known variables. Yet, Theorem 6.1 shows that this can happen. Moreover, the construction used in Theorem 6.1 can be applied to *any* model M such that $(M, \vec{u}) \models A = 1 \wedge B = 1$, but A and B are independent of each other (so that, in particular, $A = 1$ is not a cause of $B = 1$), to get a sequence of models $M_0, M_1, \ldots$, with $M = M_0$ and M_{n+1} a conservative extension of M_n such that the truth of the statement "$A = 1$ is a cause of $B = 1$ in $(M_n, \vec{u})$" alternates as we go along the sequence.

While disconcerting, I do not believe that, in fact, this is a problem. A child may start with a primitive understanding of how the world works, and believe that just throwing a rock causes a bottle to shatter. Later he may become aware of the importance of the rock actually hitting the bottle. Still later, he may become of other features critical to bottles shattering. This increased awareness can and should result in causality ascriptions changing. However, in practice, there are very few new features that should matter. We can make this precise by observing that, most new features that we become aware of are almost surely irrelevant to the bottle shattering except perhaps in highly abnormal circumstances. If the new variables were relevant, we probably would have become aware of them sooner. (Recall the gloss that I gave above when introducing the variable X_n: the value $X_n = 0$, which was needed to establish $A = 1$ being a cause of $B = 1$, was an abnormal value.)

As I now show, once we take normality into account, under reasonable assumptions, non-causality is stable. To make this precise, I must first extend the notion of conservative extension to extended causal models so as to take the normality ordering into account.

Definition 6.2: An extended causal model $M' = (\mathcal{S}', \mathcal{F}', \succeq')$ is a *conservative extension* of an extended causal model $M = (\mathcal{S}, \mathcal{F}, \succeq)$ if the causal model $(\mathcal{S}', \mathcal{F}')$ underlying M' is a conservative extension of the causal model $(\mathcal{S}, \mathcal{F})$ underlying M according to Definition 4.2 and, in addition, the following condition holds, where $\mathcal{V}$ is the set of endogenous variables in M:

CE. For all contexts $\vec{u}$, if $\vec{W} \subseteq \mathcal{V}$, then $s_{\vec{W}=\vec{w},\vec{u}} \succeq s_{\vec{u}}$ iff $s_{\vec{W}=\vec{w},\vec{u}} \succeq' s_{\vec{u}}$.

∎

Roughly speaking, CE say that the normality ordering when restricted to worlds characterized by settings of the variables in $\mathcal{V}$ is the same in M and M'. (Actually, CE says less than this. I could have taken a stronger version of CE that would be closer to this English gloss: if $\vec{W} \cup \vec{W}' \subseteq \mathcal{V}$, then $s_{\vec{W}=\vec{w},\vec{u}} \succeq s_{\vec{W}'=\vec{w}',\vec{u}}$ iff $s_{\vec{W}=\vec{w},\vec{u}} \succeq' s_{\vec{W}'=\vec{w}',\vec{u}}$. The version of CE that I consider suffices to prove the results below, but this stronger version seems reasonable as well.)

For the remainder of this section, I work with extended causal models M and M', and so use the extended HP definition of causality that takes normality into account, although, for ease of exposition, I do not mention this explicitly. As above, I take $\succeq$ and $\succeq'$ to be the preorders in M and M', respectively.

I now provide a condition that almost ensures that non-causality is stable. Roughly speaking, I want it to be abnormal for a variable to take on a value other than that specified by the equations. Formally, say that *in world s,*

V takes on a value other than that specified by the equations in $(M, \vec{u})$ if, taking $\vec{W}^*$ to consist of all endogenous variables in M other than V, if $\vec{w}^*$ gives the values of the variables in $\vec{W}^*$ in s, and v is the value of V is s, then $(M, \vec{u}) \models [\vec{W}^* \leftarrow \vec{w}^*](V \neq v)$. For future reference, note that it is easy to check that if $\vec{W} \subseteq \vec{W}^*$ and $(M, \vec{u}) \models [\vec{W} \leftarrow \vec{w}](V \neq v)$, then V takes on a value other than that specified by the equations in $s_{\vec{W} \leftarrow \vec{w}, \vec{u}}$. Finally, say that $(M, \vec{u})$ *respects the equations for* V if, for all worlds s such that V takes on a value in s other than that specified by the equations in $(M, \vec{u})$, we have $s \not\succeq s_{\vec{u}}$ (where $\succeq$ is the preorder on worlds in M).

Recall from the proof of Theorem 6.1 that to show that $A = 1$ is a cause of $B = 1$ in (M_{2n+1}, u_1), we considered a witness world where $X_{n+1} = 0$ and $A = 0$. Once we take normality into account, if we require that the normality ordering in M_{2n+1} be such that (M_{2n+1}, u_1) respects that equations for X_{n+1}, a world where $X_{n+1} = 0$ is less normal than $s_{\vec{u}}$, so cannot be used to satisfy AC2(a). As the following theorem shows, this observation generalizes.

Theorem 6.3: *If M and M' are extended causal models such that (a) M' is a conservative extension of M, (b) $\vec{X} = \vec{x}$ is not a cause of φ in $(M, \vec{u})$, and (c) $(M', \vec{u})$ respects the equations for all the endogenous variables that are in M' but not in M, then either $\vec{X} = \vec{x}$ is not a cause of φ in $(M', \vec{u})$ or there is a strict subset $\vec{X}_1$ of $\vec{X}$ such that $\vec{X}_1 = \vec{x}_1$ is a cause of φ in $(M, \vec{u})$, where $\vec{x}_1$ is the restriction of $\vec{x}$ to the variables in $\vec{X}_1$.*

Proof: Suppose that the assumptions of the theorem hold and that $\vec{X} = \vec{x}$ is a cause of φ in $(M', \vec{u})$ with witness $(\vec{W}, \vec{w}, \vec{x}')$. I show that there is a strict subset $\vec{X}_1$ of $\vec{X}$ such that $\vec{X}_1 = \vec{x}_1$ is a cause of φ in $(M, \vec{u})$, where $\vec{x}_1$ is the restriction of $\vec{x}$ to the variables in $\vec{X}_1$.

Let $\mathcal{V}$ be the set of endogenous variables in M, let $\vec{W}_1 = \vec{W} \cap \mathcal{V}$, let $\vec{Z}_1 = \mathcal{V} - \vec{W}$, and let $\vec{w}_1$ be the restriction of $\vec{w}$ to the variables in $\vec{W}_1$. Since $\vec{X} = \vec{x}$ is not a cause of φ in $(M, \vec{u})$, it is certainly not a cause with witness $(\vec{W}_1, \vec{w}_1, \vec{x}')$. Thus, either (i) $(M, \vec{u}) \models \vec{X} \neq \vec{x} \vee \neg\varphi$ (i.e., AC1 is violated); (ii) $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W}_1 \leftarrow \vec{w}_1]\varphi$ (i.e., AC2(a) is violated), (iii) there exist subsets $\vec{W}_1'$ of $\vec{W}_1$ and $\vec{Z}_1'$ of $\vec{Z}_1$ such that if $(M, \vec{u}) \models \vec{Z}_1' = \vec{z}_1$ (i.e., $\vec{z}_1$ gives the actual values of the variables in $\vec{Z}_1'$), then $(M, \vec{u}) \not\models [\vec{X} \leftarrow \vec{x}, \vec{W}_1' \leftarrow \vec{w}_1, \vec{Z}_1' \leftarrow \vec{z}_1]\neg\varphi$ (i.e., AC2(b) is violated), (iv) $s_{\vec{X}=\vec{x}', \vec{W}_1=\vec{w}_1, \vec{u}} \not\succeq s_{\vec{u}}$ (i.e., the normality condition in AC2(a$^+$) is violated), or (v) there is a strict subset $\vec{X}_1$ of $\vec{X}$ such that $\vec{X}_1 = \vec{x}_1$ is a cause of φ in $(M, \vec{u})$, where $\vec{x}_1$ is the restriction of $\vec{x}$ to the variables in $\vec{X}_1$ (i.e., AC3 is violated). I now show that none of (i)–(iv) can hold, which suffices to prove the result.

Since M' is a conservative extension of M, by Lemma 4.3, if (i) or (iii) holds, then the same statement holds with M replaced by M', showing that $\vec{X} = \vec{x}$ is not a cause of φ in $(M', \vec{u})$ with witness $(\vec{W}, \vec{w}, \vec{x}')$, contradicting our assumption. If (ii) holds, it is still consistent that AC2(a) holds in M' with witness $(\vec{W}, \vec{w}, \vec{x}')$. However if, for each variable $V \in \vec{W} - \vec{W}_1$, if v is the value of V in $\vec{w}$ and we have $(M', \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W}_1 \leftarrow \vec{w}](V = v)$, then $(M', \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W} \leftarrow \vec{w}]\varphi$, and AC2(a) also fails in $(M', \vec{u})$. On the other hand, if $(M', \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W}_1 \leftarrow \vec{w}](V \neq v)$ for some $V \in \vec{W} - \vec{W}_1$, then, in the world $s_{\vec{X}=\vec{x}', \vec{W} \leftarrow \vec{w}, \vec{u}}$, the variable V takes on a value other than that specified by the equations in $(M', \vec{u})$. Since, by assumption, $(M', \vec{u})$ respects the equations for V, we have $s_{\vec{X}=\vec{x}', \vec{W} \leftarrow \vec{w}, \vec{u}} \not\succeq' s_{\vec{u}}$, contradicting the assumption that $\vec{X} = \vec{x}$ is a cause of φ in $(M', \vec{u})$ with witness $(\vec{W}, \vec{w}, \vec{x}')$. Either way, if (ii) holds, we get a contradiction. Finally, if (iv) holds, by CE, we must have $s_{\vec{X}=\vec{x}', \vec{W}_1=\vec{w}_1, \vec{u}} \not\succeq' s_{\vec{u}}$. Moreover, as we observed in the argument for (ii), we must have $(M', \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W}_1 \leftarrow \vec{w}](V = v)$ for each variable $V \in \vec{W} - \vec{W}_1$, where v is the value of V in $\vec{w}$, or else we get a contradiction to $\vec{X} = \vec{x}$ being a cause of φ in $(M', \vec{u})$ with witness $(\vec{W}, \vec{w}, \vec{x}')$. But this means that $s_{\vec{X}=\vec{x}', \vec{W}_1=\vec{w}_1, \vec{u}} = s_{\vec{X}=\vec{x}', \vec{W}=\vec{w}, \vec{u}}$, so $s_{\vec{X}=\vec{x}', \vec{W}=\vec{w}, \vec{u}} \not\succeq' s_{\vec{u}}$, and again we get a contradiction to $\vec{X} = \vec{x}$ being a cause of φ in $(M', \vec{u})$ with witness $(\vec{W}, \vec{w}, \vec{x}')$. ∎

We immediately get that single-variable non-causality is stable.

Corollary 6.4: *If (a) $X = x$ is not a cause of φ in $(M, \vec{u})$, (b) M' is a conservative extension of M, and (c) $(M', \vec{u})$ respects the equations for all the endogenous variables that are in M' but not in M, then $X = x$ is not a cause of φ in $(M', \vec{u})$.*

While these results shows that we get stability of causality, it comes at a price: the assumption that the normality ordering respects the equations for a variable relative to a context $\vec{u}$ is clearly quite a strong one. Although it may seem reasonable to require that it be abnormal for the new variables not to respect the equations in $\vec{u}$, recall that the normality ordering is placed on worlds, which are complete assignments to the endogenous variables, not on complete assignments to both endogenous and exogenous variables. Put another way, in general, the normality ordering does not take the context into account. To see why this is important, note that in almost all of our examples of causality in a context $\vec{u}$ in a model M, the witness does not respect the equations of $\vec{u}$. For example, to show that Suzy's throw is a cause of the bottle shattering in the context u where both Suzy and Billy throw rocks, we consider a witness world where neither Suzy nor Billy throw. This world clearly does not respect the equations of u, where Suzy and Billy do throw rocks. Nevertheless, if we ignore the context, it does not seem so abnormal that neither Suzy nor Billy throw rocks.

Thus, saying that the normality ordering respects the equations for a variable V relative to $\vec{u}$ is really saying that, as far as V is concerned, what happens in $\vec{u}$ is really the normal situation. In the assassin example used to prove Theorem 6.1, it might be better to think of the variable A_n as being three-valued: $A_n = 0$ if assassin $\#n$ is present and puts in poison, $A_n = 1$ if assassin $\#n$ is present and does not put in poison, and $A_n = 2$ if assassin $\#n$ is not present. Clearly the normal value is $A_n = 2$. Take u to be the context where, in model M_{2n+1}, $A_n = 2$. While the potential presence a number of assassins makes bodyguard putting in antidote (part of) a cause in (M_{2n+1}, u), it is no longer part of a cause once we take normality into account. Moreover, here it does seem reasonable to say that violating the equations for A_n relative to u is abnormal.

These observations suggest why, in general, although the assumption that respects the equations for the variables in $\mathcal{V}' - \mathcal{V}$ relative to the context $\vec{u}$ is a strong one, it may not be unreasonable in practice. Typically, the variables that we do not mention take on their expected values, and thus are not even noticed.

The requirement that we are talking about single-variable causality in Corollary 6.4 has some bite, but not much. Stability of non-causality does not hold in general, even with the abnormality assumption, as Example 6.6 below shows. However, I can show that there can be at most one change from non-causality to causality. It follows that we cannot get an infinite sequence of causal models, each one a conservative extension of the one before, where the answer to the question "Is $\vec{X} = \vec{x}$ a cause of φ?" alternates from "Yes" to "No" and back again under reasonable (ab)normality assumptions. Indeed, as is shown in the following corollary, we cannot even get such a sequence of length 3.

Corollary 6.5: *If (a) M_2 is a conservative extension of M_1, (b) M_3 is a conservative extension of M_2, (c) $\vec{X} = \vec{x}$ is a cause of φ in $(M_1, \vec{u})$ and $(M_3, \vec{u})$, (d) $(M_2, \vec{u})$ respects the equations for all endogenous variables in M_2 not in M_1, and (e) and (e) $(M_3, \vec{u})$ respects the equations for all endogenous variables in M_3 not in M_2, then $\vec{X} = \vec{x}$ is also a cause of φ in $(M_2, \vec{u})$.*

Proof: Suppose, by way of contradiction, that there is a sequence M_1, M_2, and M_3 of models and a context $\vec{u}$ satisfying the conditions of the theorem, but $\vec{X} = \vec{x}$ is not a cause of φ in $(M_2, \vec{u})$. By Theorem 6.3, there must be a strict subset $\vec{X}_1$ of $\vec{X}$ such that $\vec{X}_1 = \vec{x}_1$ is a cause of φ in $(M_2, \vec{u})$, where $\vec{x}_1$ is the restriction of $\vec{x}$ to the variables in $\vec{X}_1$. But $\vec{X}_1 = \vec{x}_1$ cannot be a cause of φ in $(M_1, \vec{u})$, for then, by AC3, $\vec{X} = \vec{x}$ would not be a cause of φ in $(M_1, \vec{u})$. By Theorem 6.3 again, there must be a strict subset $\vec{X}_2$ of $\vec{X}_1$ such that $\vec{X}_2 = \vec{x}_2$ s a cause of φ in $(M_1, \vec{u})$, where $\vec{x}_2$ is the restriction of $\vec{x}$ to $\vec{X}_2$. But then, by AC3, $\vec{X} = \vec{x}$ cannot be a cause of φ in $(M_1, \vec{u})$, giving us the desired contradiction. ∎

The following example, which is a variant of the example in [Halpern 2008] showing that a cause may involve more than one conjunct, shows that Corollary 6.5 is the best that we can hope for. It is possible for a non-cause to become a cause if it has more than one conjunct.

Example 6.6: A votes for a candidate. A's vote is recorded in two optical scanners B and C. D collects the output of the scanners. The candidate wins (i.e., *WIN* $= 1$) if any of A, B, or D is 1. The value of A is determined by the exogenous variable. The following structural equations characterize the the remaining variables: $B = A$,

$C = A$, $D = B \wedge C$, $WIN = A \vee B \vee D$. Call the resulting causal model M. In the actual context u, $A = 1$, so $B = C = D = WIN = 1$. Assume that all worlds in M are equally normal.

I claim that $B = 1$ is a cause of $WIN = 1$ in (M, u). To see this, take $\vec{W} = \{A\}$. Consider the contingency where $A = 0$. Clearly if $B = 0$, then $WIN = 0$, while if $B = 1$, $WIN = 1$. It is easy to check that AC2 holds. Moreover, since $B = 1$ is a cause of $WIN = 1$ in (M, u), by AC3, $B = 1 \wedge C = 1$ cannot be a cause of $WIN = 1$ in (M, u).

Now consider the model M' that is just like M, except that there is one more exogenous variable D', where $D' = B \wedge \neg A$. The equation for WIN now becomes $WIN = A \vee D' \vee D$. All the other equations in M' are the same as those in M. Roughly speaking, D' acts like BH in the rock-throwing example. Define the normality ordering in M' so that it respects the equations for D' in (M', u): all worlds where $D' = B \wedge \neg A$ are equally normal, all worlds where $D' \neq B \wedge \neg A$ are also equally normal, but less normal than worlds where $D' = B \wedge \neg A$.

It is easy to see that M' is a conservative extension of M. Since D' does not affect any variable but WIN and all the equations except that for WIN are unchanged, it suffices to show that for all settings of the variables other than D' and WIN, WIN has the same value in context u in both M and M'. Clearly if $A = 1$ or $D = 1$, then $WIN = 1$ in both M and M'. So suppose that we set $A = D = 0$. Now if $B = 1$, then $D' = 1$ (since $A = 0$), so again $WIN = 1$ in both M and M'. On the other hand, if $B = 0$, then $D' = 0$, so $WIN = 0$ in both M and M'. Condition CE clearly holds as well.

Finally, as I now show, $B = 1 \wedge C = 1$ is a cause of $WIN = 1$ in (M', u). To see this, first observe that AC1 clearly holds. For AC2, let $\vec{W} = \{A\}$ (so $\vec{Z} = \{B, C, D, D', WIN\}$) and take $w = 0$ (so we are considering the contingency where $A = 0$). Clearly, $(M, u) \models [A \leftarrow 0, B \leftarrow 0, C \leftarrow 0](WIN = 0)$, so AC2(a) holds, and $(M, u) \models [A \leftarrow 0, B \leftarrow 1, C\ gets 1](WIN = 1)$. Moreover, $(M, \vec{u}) \models [B \leftarrow 1, C \leftarrow 1](WIN = 1)$, and $WIN = 1$ continues to hold even if D is set to 1 and/or D' is set to 0 (their values in (M, u)). Thus, AC2(b) holds.

It remains to show that AC3 holds and, in particular, that neither $B = 1$ nor $C = 1$ is a cause of $WIN = 1$ in (M', u). The argument is the same for both $B = 1$ and $C = 1$, so I just show it for $B = 1$. Roughly speaking, $B = 1$ is not a cause of $WIN = 1$ for essentially the same reason that $BT = 1$ is not a cause of $BS = 1$. For suppose that $B = 1$ were a cause. Then we would have to have $A \in \vec{W}$, and we would need to consider the contingency where $A = 0$ (for otherwise $WIN = 1$ no matter how we set B). Now we need to consider two cases: $D' \in \vec{W}$ and $D' \in \vec{Z}$. If $D' \in \vec{W}$, then if we consider the contingency where $D' = 0$, we have $(M', u) \models [A \leftarrow 0, B \leftarrow 1, D' \leftarrow 0](WIN = 0)$, so AC2(b) fails (no matter whether C and D are in $\vec{W}$ or $\vec{Z}$). And if we consider the contingency where $D' = 1$, then AC2(a) fails, since $(M', u) \models [A \leftarrow 0, B \leftarrow 0, D' \leftarrow 1](WIN = 1)$. Now if $D' \in \vec{Z}$, note that $(M, u) \models D' = 0$. Moreover, as we have observed, $(M, u) \models [A \leftarrow 0, B \leftarrow 1, D' \leftarrow 0](WIN = 0)$, so again AC2(b) fails (no matter whether C or D are in $\vec{W}$ or $\vec{Z}$). Thus, $B = 1$ is not a cause of $WIN = 1$ in (M', u). Thus, $B = 1 \wedge C = 1$ goes from not being a cause of $WIN = 1$ in (M, u) to being a cause of $WIN = 1$ in (M', u).

Now consider the model M'' which is just like M' except that it has one additional variable D'', where $D'' = D \wedge \neg A$ and the equation for WIN becomes $WIN = A \vee D' \vee D''$. All the other equations in M'' are the same as those in M'. Define the normality ordering in M'' so that it respects the equations for both D' and D'' in (M'', u).

It is easy to check that M'' is a conservative extension of M'. Since D'' does not affect any variable but WIN and all the equations except that for WIN are unchanged, it suffices to show that for all settings of the variables other than D'' and WIN, WIN has the same value in context u in both M and M'. Clearly if $A = 1$ or $D' = 1$, then $WIN = 1$ in both M' and M''. And if $A = D = 0$, then $D' = 1$ iff $D = 1$, so again the value of WIN is the same in M' and M''. Condition CE clearly holds as well.

Finally, I claim that $B = 1 \wedge C = 1$ is no longer a cause of $WIN = 1$ in (M'', u). Suppose, by way of contradiction, that it is, with witness $(\vec{W}, \vec{w}, \vec{x}')$. $A = 0$ must be a conjunct of $\vec{W} = \vec{w}$. It is easy to see that either $D' = 0$ is a conjunct of $\vec{W} = \vec{w}$ or $D' \notin \vec{W}$, and similarly for D''. Since $D' = D'' = 0$ in the context u, and $(M'', u) \models [A \leftarrow 0, D' \leftarrow 0, D'' \leftarrow 0](WIN = 0)$, it easily follows that AC2(b) does not hold, no matter whether D' and D'' are in $\vec{W}$.

Thus, $B = 1 \wedge C = 1$ goes from not being a cause of *WIN* $= 1$ in (M, u) to being a cause of *WIN* $= 1$ in (M', u) to not being a cause of of *WIN* $= 1$ in (M'', u). ∎

7 Conclusions

This paper has demonstrated the HP definition of causality is remarkably resilient, but it emphasizes how sensitive the ascription of causality can be to the choice of model. The focus has been on showing that the choice of variables is a powerful modeling tool. But it is one that can be abused. One lesson that comes out clearly is the need to have variables that describe the mechanism of causality, particularly if there is more than one mechanism. However, this is hardly a general recipe. Rather, it is a heuristic for constructing a "good" model. As Halpern and Hitchcock [2010] point out, constructing a good model is still more of an art than a science.

The importance of the choice of variables to the ascription of causality leads to an obvious question: to what extent is the choice of variables determined by the story. Certainly some variables are explicit in a causal story. If we talk about Suzy and Billy throwing rocks at a bottle, which shatters, it seems pretty clear that a formal model needs to have variables that talk about Suzy and Billy throwing rocks, and the bottle shattering. Furthermore, if the story says that Suzy's rock hits first, it also seems clear that we need variables in the formal model to capture the fact that Suzy's rock hit first. Unfortunately, there is more than one way to capture this fact using variables. Here I used the variables *SH* and *BH*, as was done in [Halpern and Pearl 2005]. But in [Halpern and Pearl 2005], another model was also presented, where the there are time-indexed variables (e.g., a family of variables BS_k for "bottle shatters at time k"). In the model with time-indexed variables it is still the case that Suzy's throw is a cause of the bottle shattering and Billy's throw is not. The point here is that the story does not make explicit which variables should be used. While a modeler must ultimately justify whatever variables are used in terms of how well they capture the intent of the story, there is clearly a lot left to the modeler's judgment here. (A similar point is made in [Halpern and Hitchcock 2010].)

A second lesson of this paper is that there is an interplay between the choice of variables and normality considerations. Moreover, normality considerations can play quite an important role in dealing with issues regarding the stability of causality and non-causality. There are doubtless other lessons that will be learned as we get more experience with causal modeling. Structural models are a powerful tool for modeling causality, but they have to be handled with care!

References

Aleksandrowicz, G., H. Chockler, J. Y. Halpern, and A. Ivrii (2014). The computational complexity of structure-based causality. In *Proc. Twenty-Eighth National Conference on Artificial Intelligence (AAAI '14)*, pp. 974–980.

Beer, I., S. Ben-David, H. Chockler, A. Orni, and R. J. Trefler (2012). Explaining counterexamples using causality. *Formal Methods in System Design 40*(1), 20–40.

Blanchard, T. and J. Schaffer (2013). Cause without default. unpublished manuscript.

Cushman, F., J. Knobe, and W. Sinnott-Armstrong (2008). Moral appraisals affect doing/allowing judgments. *Cognition 108*(1), 281–289.

Eberhardt, F. (2014). Direct causes and the trouble with soft intervention. *Erkenntnis 79*(4), 755–777.

Eiter, T. and T. Lukasiewicz (2002). Complexity results for structure-based causality. *Artificial Intelligence 142*(1), 53–89.

Gerstenberg, T. and D. Lagnado (2010). Spreading the blame: the allocation of responsibility amongst multiple agents. *Cognition 115*, 166–171.

Glymour, C., D. Danks, B. Glymour, F. Eberhardt, J. Ramsey, R. Scheines, P. Spirtes, C. M. Teng, and J. Zhang (2010). Actual causation: a stone soup essay. *Synthese 175*, 169–192.

Hall, N. (2007). Structural equations and causation. *Philosophical Studies 132*, 109–136.

Halpern, J. Y. (2008). Defaults and normality in causal structures. In *Principles of Knowledge Representation and Reasoning: Proc. Eleventh International Conference (KR '08)*, pp. 198–208.

Halpern, J. Y. and C. Hitchcock (2010). Actual causation and the art of modeling. In R. Dechter, H. Geffner, and J. Halpern (Eds.), *Causality, Probability, and Heuristics: A Tribute to Judea Pearl*, pp. 383–406. London: College Publications.

Halpern, J. Y. and C. Hitchcock (2015). Graded causation and defaults. *British Journal for the Philosophy of Science 66*(2), 413–457.

Halpern, J. Y. and J. Pearl (2001). Causes and explanations: A structural-model approach. Part I: Causes. In *Proc. Seventeenth Conference on Uncertainty in Artificial Intelligence (UAI 2001)*, pp. 194–202.

Halpern, J. Y. and J. Pearl (2005). Causes and explanations: A structural-model approach. Part I: Causes. *British Journal for Philosophy of Science 56*(4), 843–887.

Hiddleston, E. (2005). Causal powers. *British Journal for Philosophy of Science 56*, 27–59.

Hitchcock, C. (2001). The intransitivity of causation revealed in equations and graphs. *Journal of Philosophy XCVIII*(6), 273–299.

Hitchcock, C. (2007). Prevention, preemption, and the principle of sufficient reason. *Philosophical Review 116*, 495–532.

Hitchcock, C. and J. Knobe (2009). Cause and norm. *Journal of Philosophy 106*, 587–612.

Hopkins, M. (2001). A proof of the conjunctive cause conjecture. Unpublished manuscript.

Hopkins, M. and J. Pearl (2003). Clarifying the usage of structural models for commonsense causal reasoning. In *Proc. AAAI Spring Symposium on Logical Formalizations of Commonsense Reasoning*.

Kahneman, D. and D. T. Miller (1986). Norm theory: comparing reality to its alternatives. *Psychological Review 94*(2), 136–153.

Knobe, J. and B. Fraser (2008). Causal judgment and moral judgment: two experiments. In W. Sinnott-Armstrong (Ed.), *Moral Psychology, Volume 2: The Cognitive Science of Morality*, pp. 441–447. Cambridge, MA: MIT Press.

Lagnado, D. A., T. Gerstenberg, and R. Zultan (2013). Causal responsibility and counterfactuals. *Cognitive Science 37*, 1036–1073.

Livengood, J. (2013). Actual causation in simple voting scenarios. *Nous 47*(2), 316–345.

Spohn, W. (2008). Personal email.

Strevens, M. (2008). Comments on woodward, *Making Things Happen*. *Philosophy and Phenomenology 77*(1), 171–192.

Weslake, B. (2015). A partial theory of actual causation. *British Journal for the Philosophy of Science*. To appear.

Woodward, J. (2003). *Making Things Happen: A Theory of Causal Explanation*. Oxford, U.K.: Oxford University Press.