Statistical Causality from a Decision-Theoretic Perspective

A. PHILIP DAWID

University of Cambridge, UK

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Abstract: We present an overview of the decision-theoretic framework of statistical causality, which is well-suited for formulating and solving problems of determining the effects of applied causes. The approach is described in detail, and is related to and contrasted with other current formulations, such as structural equation models and potential responses. Topics and applications covered include confounding, the effect of treatment on the treated, instrumental variables, and dynamic treatment strategies.

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1 Introduction

After decades of neglect, recent years have seen a flowering of the field of statistical causality, with an impressive array of developments both theoretical and applied. However there is as yet no one fully accepted foundational basis for this enterprise. Rather, there is a variety of formal and informal frameworks for framing and understanding causal questions, and hot discussion about their relationships, merits and demerits: we might mention, among others, structural equation modelling and path analysis (Wright 1921), potential response models (Rubin 1978), functional models (Pearl 2009), and various forms of graphical representation. This plethora of “foundations” leaves statistical causality in much the same state of confusion as probability theory before Kolmogorov.

The aim of this overview paper is to add to the confusion by describing one particular approach, based on decision-theoretic principles, that its author considers superior to the others (in this of course he is fully aligned with others’ attitudes to their own works). I will present the main features of the approach, relate it to and compare it with some other approaches, and show how it works in some simple applications. I consider this approach more straightforward philosophically and mathematically (it requires only a very small extension to standard statistical methods), and easier to comprehend and manipulate, than other approaches that
introduce new ingredients and structures, such as potential responses or deterministic functional relationships. While I do not expect wholesale conversion to the point of view, I hope that readers already knowledgeable in causal inference will, at the very least, find it helpful to look at familiar topics through a fresh pair of spectacles.

For further details and developments of the material in this paper, see Dawid (2000); Dawid (2002); Dawid (2003); Didelez et al. (2006); Dawid (2007a); Dawid and Didelez (2007); Dawid (2010a); Dawid (2010b); Dawid and Didelez (2010); Guo and Dawid (2010); Dawid (2011); Geneletti and Dawid (2011); Dawid and Didelez (2012); Berzuini et al. (2012). The lecture notes Dawid (2007b) contain a fuller exposition of the decision-theoretic approach.

Causality and agency

“Causality” has been a focus of interest for philosophers for millennia, but — as befits any worthwhile philosophical conundrum — all this attention has not resulted in a settled approach to understanding it. In Dawid (2010b) I reviewed a variety of philosophical conceptions and theories, focusing particularly on that which is most germane to my own approach: the agency theory of causality (Price 1991; Hausman 1998; Woodward 2003; Woodward 2013). This interprets causality as being all about how an external manipulation would affect a system: for example, how the quality of the chemical product would respond to adjustments of the lever that controls the pressure in the production process. Much of Statistical Science—in particular, the whole subfield of Experimental Design—aims to address exactly these kinds of questions about the effects of interventions.
on a system, which are indeed a major object of all scientific enquiry.

An important advantage of the agency approach is its clear separation of cause and effect variables, resulting in the elimination of definitional ambiguities associated with the possibility of reverse causation or common causes. But having clean definitions is not enough for practical purposes: we must be able to relate those definitions to properties of the empirical world. Whereas this is relatively unproblematic in cases involving genuine experimentation, it becomes a major headache when we can only observe a system in its natural habitat, and are unable to apply to it the interventions that are essential to understanding its causal properties. The importance of making a clear distinction between intervening and merely observing has been stressed by numerous authors, including [Rubin (1978)](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2270296/), [Meek and Glymour (1994)](https://doi.org/10.1016/S0268-9991(94)80020-4), [Spirtes et al. (2000)](https://doi.org/10.1016/S0268-9991(00)00032-X), [Pearl (2009)](https://doi.org/10.1016/j.jogc.2009.07.005). Most of the recent emphasis of the enterprise of “statistical causality” focuses on observational situations, and attempts to identify conditions under which it is possible to extract causal conclusions from them, and techniques for doing so.

**Effects of causes and causes of effects**

The emphasis in this work will be entirely on the problem of assessing the future effects of an intervention in a system: that is, on identifying the “effects of causes” (EoC). An entirely different problem is that of judging what might have been the cause of an observed outcome: that is to say, identifying the “causes of effects” (CoE). This is very much an issue in legal cases, which may seek to assign responsibility or blame.

Most of the effort to date in statistical causality has focused on CoE problems, an important exception being [Pearl (2009)](https://doi.org/10.1016/j.jogc.2009.07.005) who explores both problems in detail.
However, whereas Pearl, and such others who have dealt with CoE, have used the identical mathematical machinery (in Pearl’s case, based on assumed functional relationships) for both EoC and CoE, I do not consider this appropriate. In [Dawid (2000)] I discussed the relationships and differences between these problems, and their requisite infrastructures, in some detail, and argued that, whereas (unlike for EoC) some form of counterfactual logic appears unavoidable for assessing CoE, the difficulties in appropriately modelling a CoE problem have been underappreciated. Some discussion and analysis of CoE issues in the context of using epidemiological data to address a legal case of toxic tort can be found in [Dawid (2011); Dawid et al. (2013); Dawid et al. (2014)].

Plan of paper

I start in §2 with a simple example that locates statistical causality firmly within the purview of classical statistical decision analysis. Section 3 then explores some variant formulations of this problem, including structural equations and potential responses. In 4 these approaches are explored and compared in the familiar context of statistical experimental design. Section 5 moves the discussion on to the more problematic context of causal inference from observational data, and explores the meaning and representation of the important concept of “no confounding”: from a decision-theoretic approach, this is usefully described in terms of relationships between different “regimes” — e.g., interventional or observational — under which data can, in principle at least, be gathered. It is shown how this and similar requisite properties can be usefully expressed and manipulated in terms of an extension of the probabilistic notion of conditional independence to allow for both stochastic and non-stochastic variables. Section 6 develops
the associated algebraic and graphical theory. In §7 we introduce influence diagrams as useful graphical representations of causal problems, and relate these to the use of directed acyclic graph representations as described by Pearl (2009). The remainder of the paper explores, from the decision-theoretic perspective, a number of important special applications. Section 9 examines the observational identification of causal effects using sufficient covariates, propensity analysis and “do-calculus”, and the possibility of identifying “the effect of treatment on the treated”; §10 considers the use of instrumental variables; and §11 treats problems where a sequence of actions can be applied, over time, in response to intermediate observations and outcomes. The discussion in §12 expresses some scepticisms about currently popular approaches that make unavoidable use of counterfactual reasoning.

2 A Decision Problem

I have a headache and am considering whether or not to take two aspirin tablets. It is generally accepted that aspirin has a beneficial effect on headaches: in some sense — and it will be our task to try to make this more precise — taking aspirin causes headaches to get better faster. The solution to my decision problem is thus intimately bound up with the cause-effect relationship of aspirin on headache.

This observation leads naturally on to a suspicion that some part\footnote{Specifically, the part that aims to understand the effects of applied causes (EoC); problems of identifying the causes of observed effects (CoE) require a different approach.} at least, of the enterprise of “statistical causality” might be fruitfully recast as a special application of standard statistical decision analysis. However, this point of view is not a currently popular one, and indeed there is a variety of other attempts to
interpret causality in a statistical setting. We shall be considering the relationships, similarities and differences between these, and hope to demonstrate that the decision-theoretic approach is more natural, more straightforward, and more useful than its competitors.

To formulate the decision problem, let the binary variable \( X \) denote whether I take aspirin \((X = 1)\) or not \((X = 0)\), and let \( Y \) be the log-time it takes for my headache to go away. I myself can choose \( X \): it is a decision variable, and does not have a probability distribution. Nevertheless, it is still meaningful to consider my conditional distribution, \( P_x \) say, for how the eventual response \( Y \) will turn out, given that I choose \( X = x \). For the moment we assume the distributions \( P_0 \), \( P_1 \) to be known. Where we need to be definite, we shall (purely for simplicity) take them to have the following normal probability density function:

\[
p(y \mid X = x) = (2\pi \sigma^2)^{-\frac{1}{2}} \exp\left(-\frac{(y - \mu_x)^2}{2\sigma^2}\right),
\]

with a mean \( \mu_0 \) or \( \mu_1 \) according as \( x = 0 \) or 1, and variance \( \sigma^2 \) in either case.

The distribution \( P_1 \) [resp., \( P_0 \)] can be interpreted as expressing my hypothetical uncertainty about \( Y \), if I were to decide on action \( X = 1 \) [resp., \( X = 0 \)]. It can incorporate various sources and types of uncertainty, including stochastic effects of external influences arising and acting between the points of treatment application and eventual response. The distributions \( P_1 \) and \( P_0 \) are all that is needed to address my decision problem: I simply need to compare the two different hypothetical distributions for \( Y \), decide which one I prefer, and take the associated decision.

One possible comparison of \( P_1 \) and \( P_0 \) might be in terms their respective means, \( \mu_1 \) and \( \mu_0 \); the “effect” of taking aspirin, rather than nothing, might then be quantified by means of the change in the expected response, \( \delta := \mu_1 - \mu_0 \). Al-
ternatively, we might look at the difference of the means of $Z = e^Y$ under the two possible treatments: $e^{\sigma^2/2}(e^{\mu_1} - e^{\mu_2})$. Or we might compare the variances of $Z$ under the two treatments. Any such comparison of an appropriately chosen feature of the two hypothetical distributions of $Y$ can be regarded as a summary of the causal effect of taking aspirin (as against taking nothing).

More formally, we might apply statistical decision analysis (see e.g. [Raiffa (1968)]) to structure and solve this decision problem. Suppose that I quantify the loss that I will suffer if my headache lasts $y$ minutes by means of a real-valued loss function, $L(y)$. If I were to take the aspirin, my expected loss would be $E_{Y\sim P_1}\{L(Y)\}$; if not, it would be $E_{Y\sim P_0}\{L(Y)\}$. The principles of statistical decision analysis now direct me to choose the treatment leading to the smaller expected loss.

A trivial but fundamentally important point is that, whatever loss function is used, this solution will only involve the two hypothetical distributions, $P_1$ and $P_0$, for $Y$, conditional on taking either action. The “effect of treatment” might be measured by the reduction in expected loss, $E_{P_0}\{L(Y)\} - E_{P_1}\{L(Y)\}$: and the correct decision will be to take aspirin just when this reduction is positive.

Although there is no uniquely appropriate measure of “the effect of treatment”, in the rest of this paper for simplicity we shall focus on the difference of the means of the two hypothetical distributions: $\delta := E_{P_1}(Y) - E_{P_0}(Y)$.

### 3 Alternative Formulations

#### 3.1 Decision-theoretic (DT) model

The essential ingredients of the decision-theoretic analysis above were the two hypothetical distributions for $Y$, conditional on $X = 0$ and on $X = 1$. These
were specialised to be normal:

\[ Y \mid X = x \sim \mathcal{N}(\mu_x, \sigma^2). \]  \hspace{1cm} (2)

We term this a stochastic or decision-theoretic (DT) model. In this formulation, the term average causal effect (ACE) simply denotes the difference of the means of the two hypothetical distributions for \( Y, \mu_1 - \mu_0 \).

### 3.2 Simple structural equation (SSE) model

An alternative way in which the assumptions of (2) are very often expressed is as follows:

\[ Y = \mu_X + E \]  \hspace{1cm} (3)

where

\[ E \sim \mathcal{N}(0, \sigma^2) \]  \hspace{1cm} (4)

and it is implicit that the “error” \( E \) is independent of \( X \). A system of equations such as (3), which may contain hundreds of relationships representing response (“endogenous”) variables as functions of other (both endogenous and “exogenous”) variables as well as of external “error” variables such as \( E \), together with associated explicit or implicit assumptions about the joint distribution of the error terms, constitutes a simple Structural Equation (SSE) model. Such models are popular in econometrics and other fields as representations of causal structures.

The assumptions of the SSE model (3) clearly imply the distributional properties of the DT model (1). Does this mean the SSE model is equivalent to the DT model? To assume this would be to ignore the additional algebraic structure of (3), whereby \( Y \) is represented as a deterministic mathematical function of the two variables \( X \) and \( E \). Unlike the distributional formulation of (1), in (3) all
the uncertainty is compressed into the single variable \(E\), via (1). If we take (3) and its ingredients seriously, we can get more out of it.

It is common, and indeed seems very natural, implicitly to interpret (3) as follows. The values of \(X\) and \(E\) are assigned separately (by the decision maker and by Nature, respectively), and \(Y\) is then determined by the equation. In this case, given that \(E\) takes value \(e\), then if I set \(X\) to \(x\), \(Y\) will take value \(y_x := \mu_x + e\). That is, I will observe the variable \(Y_x := \mu_x + E\). We can regard \(Y_x\) as the potential response to the hypothetical setting \(X = x\). It will become the actual response, \(Y\), when indeed \(X = x\): \(Y = Y_X = \mu_X + E\). Note that, in this interpretation, when in fact I set \(X = 1\), the counterfactual response \(Y_0 = \mu_0 + E\) to \(X = 0\) is still a well-defined function of the ingredients of model (3).

With the above interpretations, if I were to switch my decision from \(X = 1\) to \(X = 0\), then \(Y\) would switch from \(Y_1 = \mu_1 + E\) to \(Y_0 = \mu_0 + E\), with the identical \(E\). The “causal effect” of this switch might then be measured by \(Y_1 - Y_0\), which in this case is the constant \(\mu_1 - \mu_0\). This is a purely algebraic comparison, unrelated to the stochastic properties of the model. It may be termed an individual causal effect (ICE).

Note that all these manipulations rely fundamentally on the implicit assumption that the value of \(E\) remains fixed, irrespective of which decision I take. Such an assumption simply has no counterpart in the DT model (2).

\(^2\)since predicated on a hypothesis that runs counter to known facts
3.3 Extended structural equation (ESE) model

An extension of the SSE model (3) is given by:

\[ Y = \mu_X + E_X \]  \hspace{1cm} (5)

where we now have a pair \( E = (E_0, E_1) \) of error variables, having a bivariate distribution, assumed independent of \( X \). In particular, when \( X = 0 \) we have \( Y = Y_0 := \mu_0 + E_0 \), with \( E_0 \) having its initially assigned distribution; and similarly \( Y = Y_1 := \mu_1 + E_1 \) when \( X = 1 \). And \( Y = Y_X \).

Suppose we model the pair of errors \( E \) as bivariate normal with standard normal margins:

\[ E \sim \mathcal{N}(0, \Sigma), \]  \hspace{1cm} (6)

where \( \Sigma \) (2 \( \times \) 2) has diagonal entries \( \sigma^2 \) and off-diagonal entries \( \rho \sigma^2 \). Then (no matter what the correlation \( \rho \) may be) the DT model (1) for \( Y \) given \( X \) will be obtained. But again, if we take the algebraic structure seriously, and further suppose that the value of \( E \) is unaffected by the choice made for \( X \), we can go further and define potential responses \( Y_x := \mu_x + E_x \), as well as the ICE, \( Y_1 - Y_0 \), which in this case is a random quantity, \( \mu_1 - \mu_0 + E_1 - E_0 \).

It is important to note that the relationship between the ESE model and the induced DT model is many-one: the dependence structure (here embodied in the correlation \( \rho \)) does not enter into the induced DT model.

3.4 Potential response (PR) model

As seen above, starting from a structural equation model (whether simple or extended), we can define the pair \( Y = (Y_0, Y_1) \) of potential responses, and derive its bivariate distribution, in terms of the ingredients of that model. For our ESE
model above, the implied distribution of $Y$ is

$$Y \sim \mathcal{N}(\mu, \Sigma)$$  \hspace{1cm} (7)

with $\mu := (\mu_0, \mu_1)$. Both the value and the distribution of $Y$ are regarded as independent of the applied treatment $X$.

We might alternatively start at this point, simply taking the pair $Y$ as a primitive ingredient of our model, having a bivariate distribution (for example, that of (7)), and again assuming that both the value and the distribution of $Y$ will be unchanged if we change the value of $X$. This is the general potential response (PR) model. The underlying philosophical conception is that both potential responses are real and coexist — even though it is logically impossible to observe both of them together.

Starting from a PR model we can recover a DT model: in particular, if we start with (7) we recover (2). But again, this relationship is many-one.

### 3.5 Functional model

Mathematically, the models introduced in §3.2, §3.3, §3.4 all have the following common functional form:

$$Y = f(X, U),$$  \hspace{1cm} (8)

where $X$ is a decision variable representing the cause of interest; $Y$ is the effect of interest; $U$ is a further extraneous random variable whose value and distribution are taken as independent of $X$; and $f$ is a deterministic function of its arguments.

In the ESE model (5), we can take $U = E$ and $f(x, (e_0, e_1)) = \mu_x + e_x$; the SSE model of (3) is the degenerate case of this having $U = E$ and $f(x, e) = \mu_x + e$.

In the case of a PR model, we can formally take $U$ to be the pair $(Y_0, Y_1)$, and
the function \( f \) to be given by:

\[
f(x, (y_0, y_1)) = y_x.
\]  

(9)

In all the above applications, the variable \( U \) typically represents a somewhat imaginary quantity, that does not correspond to any variable observable in the empirical world. Indeed, were that to be made a requirement, the application of functional models would be limited to the very special situation of complete determinism, with the pair of real variables \((X, U)\) fully determining \( Y \).

A general functional model of the form (8) is mathematically equivalent to a PR model, if we define \( Y_0 = f(0, U) \), \( Y_1 = f(1, U) \). We thus see that (mathematically if not necessarily in terms of their interpretation) PR models, ESE models and general functional models need not be distinguished. Further, any functional model determines a DT model: under model (8) the relevant distribution of \( Y \), given \( X = x \), is simply the marginal distribution of \( f(x, U) \). Conversely, given any DT model, \( Y \mid X = x \sim P_x \), we can construct a functional model corresponding to it in this way: one simple way is as a potential response model (9), in which the marginal distribution of \( Y_x \) is \( P_x \). However, in contrast to the essentially unique cross-correspondence between the other models considered above, the functional representation of a DT model is far from unique—as can again be seen, for example, from the arbitrariness of the dependence parameter \( \rho \) in the PR representation (7) of the stochastic model (2), which can never be identified from data.

4 Causal Inference from Experimental Studies

Having set out a variety of formulations of my basic decision problem, we now address the question: How might I gather and use data to help identify the
required ingredients? From the DT perspective, I need to assess my hypothetical distributions \( P_0 \) and \( P_1 \) for \( Y \), under either treatment choice. These assessments should be informed by (that is, conditioned on) whatever relevant information I may have: for example, the responses observed for other similar headaches (my own, or those of other people), that received one of the two treatments.

We initially restrict attention to the simplest case. Suppose I can observe two groups of people. Each group consists of individuals I can regard as similar to (technically, exchangeable with) me in all features relevant to their development of headaches and reaction to treatment. The first group are then all assigned active treatment (aspirin): \( X = 1 \); while the second group gets the control treatment (no aspirin), \( X = 0 \). Finally I observe the responses of all individuals: let \( Y_{xi} \) denote the response of the \( i \)th individual receiving treatment \( x \).

### 4.1 DT approach

Under the above assumptions, I can model the responses of the treated individuals as being randomly drawn from \( P_1 \), and likewise the responses of the untreated individuals as drawn from \( P_0 \). I can then use completely standard statistical methods to estimate and compare (in any way I choose) the two distributions \( P_1 \) and \( P_0 \). In particular, I have access to all the ingredients required for my

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3The usual operational method is, first to form a single group of individuals “like me”, and, secondly, to randomise the assignment of treatment to its individuals; the resulting treatment/control groups will then each still be exchangeable with me on their pre-treatment characteristics. The second stage by itself ensures internal validity: the treated and untreated groups should be comparable with each other; but without the first stage we will not have external validity, permitting generalisation beyond the data to external cases of interest — in this case, myself.
stochastic decision problem.

For example, under model (2), we model

$$Y_{xi} \sim \mathcal{N}(\mu_x, \sigma^2),$$

(10)

all independently; and can then base inference about the difference $\delta = \mu_1 - \mu_0$ on Student’s $t$-distribution. All this is the bread and butter of the most elementary courses in Statistics. Here we have however emphasised — as may be more rarely done — the assumptions needed to justify the relevance of this inference to the decision problem I face.

4.2 Other approaches

Suppose now we take the ESE model of (5) seriously (this includes the simpler SSE model (3) as the special case $E_0 = E_1$, or, equivalently, $\rho = 1$). Because that model implies all the distributional properties of (2), I can still do all that I did in §4.1 above, and so use the data to help solve my decision problem. But now I might want to do more. For example, I might want to say something about the distribution of my individual causal effect, $\text{ICE} = \mu_1 - \mu_0 + E_1 - E_0$. Can I use the data to help me in this?

Well, the mean of the ICE is just $\delta = \mu_1 - \mu_0$, which I can estimate, as above. But its variance is $2(1 - \rho)\sigma^2$. While I can estimate $\sigma^2$ from my data, the dependence on the correlation $\rho$ is problematic: I could only estimate $\rho$ if I had observations on bivariate pairs, $Y = (Y_0, Y_1)$ — corresponding to observing both potential outcomes for the same individual. Since each individual receives just one of the two treatments, the full pair $Y$ is, logically, always unobservable, and there is simply no way I can estimate $\rho$. In particular, I have no way of distinguishing observationally between the general ESE model (5) and the SSE
model (3). However, these have different implications for \( \text{var(ICE)} \), which is 
\[ 2(1 - \rho)\sigma^2 \] for ESE, and 0 for SSE.

In like fashion, were I to be interested in the mean of the “ratio ICE”, \( \text{viz.} \) \( E(Y_1/Y_0) \); or the estimate of my ICE after having taken aspirin and observed response \( Y = y \), \( \text{viz.} \) \( E(Y_1 - Y_0 \mid X = 1, Y = y) = (1 - \rho)y + (\rho\mu_1 - \mu_0) \); these seemingly innocuous queries could not be addressed by any data I could ever collect, since they all depend on the unknowable value of \( \rho \).

As the ESE model is a special case of a PR model, or a general functional model, all the above caveats apply to those models also. We can thus divide putative causal inferences from such a model into sheep — those that depend only on the marginal distributions, \( P_0 \) and \( P_1 \), of the individual potential responses \( Y_0 \) and \( Y_1 \), and are thus identifiable from data — and goats — those that do not, and so are not identifiable. For example, any putative causal inference that makes essential use of \( \text{var(ICE)} \) is a goat.

It is all too easy to neglect this distinction, and attempt to make a goat-like inference. Given a fully specified ESE/PR/functional model this will be mathematically possible, and it may not be noticed that the answer would be different for a mathematically distinct model that is observationally entirely equivalent to this one because it has the same marginal distributions. Such an apparent causal inference is, to say the least, misleading.

4.3 Treatment-unit additivity

The ESE model (5) would represent the data as

\[ Y_{xi} = \mu_x + E_{xi}, \]  
(11)
For the special case of an SSE model (3), this becomes

\[ Y_{xi} = \mu_x + E_i, \]

(12)
a sum of one term, \( \mu_x \), depending only on the treatment \( x \) applied, and another, \( E_i \), depending only on the unit \( i \) to which it is applied. This property is termed treatment-unit additivity (TUA). It is entirely equivalent to the property that the ICE, \( Y_1 - Y_0 \), has the identical value (viz., \( \mu_1 - \mu_0 \)) across all individuals.

One reason we might like the TUA assumption is as follows. So far I have had to assume that the individuals on whom I have data are “like me” — in particular, we all have the same distribution for our error term \( E \). But this is often unrealistic, since e.g. a clinical trial will typically have stringent recruitment criteria, which I would not satisfy. However, I can relax model (12), so as not to require that my own \( E \) be drawn from the same distribution as the \((E_i)\) in the data (for example, it could have a higher mean or variance). But since my own ICE is still \( \mu_1 - \mu_0 \), which is still estimable from the data, the causal inference about my ICE is unaffected by this relaxation of the SSE model — though it does still rely on TUA.

However an alternative decision-theoretic analysis (which thus does not require TUA) is as follows.\(^4\) Because of different selection criteria, my personal hypothetical distribution \( P_x \) for my response \( Y \), if I take treatment \( X = x \), is allowed be different from \( P_x^* \), the distribution of \( Y \) for the individuals in my data who receive treatment \( X = x \). But suppose (for example) I can model its mean \( \mu_x \) as related to the mean \( \mu_x^* \) of \( P_x^* \) by \( \mu_x = \mu_x^* + \gamma \) for some \( \gamma \) that does not depend on \( x \). Then my own ACE \( \mu_1 - \mu_0 = \mu_1^* - \mu_0^* \) is estimable from the data.

\(^4\)See \[Dawid (2000)\] § 8.1 for a more detailed account.
4.3.1 Neyman and Fisher

Since there is no observational way of distinguishing between the SSE model, for which TUA holds, and the more general ESE model, for which it does not, the arguments in §4.2 would classify any attempt at causal inference that is dependent on the assumption of TUA as a goat. In this light it is interesting to revisit the ill-tempered debate between Neyman and Fisher in Neyman (1935).

The essence of Neyman’s model involves an experiment in which various treatments $t = 1, \ldots, T$ can be applied to various experimental units $u = 1, \ldots, U$, for example plots in a field, which might themselves be described in more detail: for example, in a “randomised blocks” layout, $u = (i, j)$ ($i = 1, \ldots, I; j = 1, \ldots, J$) for the $j$th plot in the $i$th row. The treatments are applied to the units according to a randomisation scheme taking account of the structure of the units. In what is considered to be the first use of a potential response formulation in Statistics, Neyman introduces $y_{tu}$ to represent the response that would be observed on unit $u$, if it were to receive treatment $t$ — with the values $(y_{tu})$ for fixed $u$, as $t$ varies, regarded as having simultaneous existence, even though at most one can be observed. Neyman regards the collection of the $(y_{tu})$, for all $t$ and $u$, as the “unknown parameter”. Statistical inference is based on the distribution of the observed responses brought about by the random assignment of treatments to units.

Neyman introduces the null hypothesis:

$$H_0^*: \text{the value of } y_t \text{ does not depend on } t$$

where $y_t$ denotes the average of the $y_{tu}$ over the $U$ units in the experiment. That is, $y_t$ is the average response that would be obtained if treatment $t$ were applied

\footnote{We use different notation, and ignore certain elaborations irrelevant for current purposes.}
to all the experimental units, and his null hypothesis is that this would be the same for every treatment — allowing, however, that there could be unit-level differences, that just happen to average out. \(^6\) Neyman’s analysis (corrected and extended by Wilk and Kempthorne (1955)) shows that, for certain designs, such as the Latin square, the standard \(F\)-test is a valid test of his \(H_0^*\) only under the assumption of treatment-unit additivity — in which case, it may be noted, \(H_0^*\) becomes equivalent to

\[ H_{0}^{**}: \text{for each unit } u, \text{ the value of } y_{tu} \text{ does not depend on } t. \]

Fisher criticised \(H_0^*\), and the associated test, as based on an entirely inappropriate formulation of the phrase “no differences between the treatments”\(^7\).

From the point of view of the DT approach, it is troubling that, according to Neyman, the standard \(F\)-test is or is not valid according as whether or not we assume TUA — a distinction without any empirically observable consequences. Neyman’s analysis must therefore be classified as a goat.

5 Observational Studies and Confounding

We have so far treated \(X\) as a decision variable, under the control of a human agent rather than Nature — not merely for the actual decision problem I myself face, but likewise for the experimental individuals used to supply inputs for my problem. But often genuine experimentation is impossible, and we have to rely on data already collected, in circumstances over which we had no control — in par-

\(^6\) but need not do so if averaged over some other collection of units

\(^7\) Fisher’s arguments are characteristically intuitive rather than formal — though no less compelling for that — and he is often taken as having favoured \(H_{0}^{**}\), which is still phrased in terms of potential responses, as the appropriate null hypothesis. However my own reading of his remarks does not find any clear commitment to a PR interpretation.
ticular, no control as to who received which treatment. Such observational studies raise serious problems of interpretation and relevance, and great care is needed in drawing conclusions from them (Rosenbaum 2010; Madigan et al. 2014).

So suppose again I have data on a group of individuals whom I can regard as exchangeable with me — but now for whom treatments have already been assigned, I know not how. For each individual I have information (say, for one headache episode) on the treatment applied $X$, and the duration $Y$ (and, typically, also on some further relevant variables). Since I did not have the option to choose which treatment to apply, $X$ is no longer a decision variable: it has become a random variable.

A natural question is whether I can still use (an estimate of) the distribution of $Y$, for those individuals who received treatment $X = 1$, as a proxy for my own hypothetical distribution $P_1$ (and similarly for $X = 0$). Now in order for this to be possible, I must be able to regard the treated patients as similar to (exchangeable with) me, as regards relevant features existing prior to treatment choice. But even though this exchangeability may have been assumed at the level of the whole group, it does not follow that it will hold for the subgroup who got treated, since the treatment decision may itself have been correlated with these features — for example, aspirin may have been given only for really bad headaches. This is the problem of confounding, which obstructs straightforward causal interpretation of observational data. We shall have no confounding only when I can, simultaneously, consider myself as exchangeable, both with those patients who received aspirin, and also with those who received none. This then implies that those two groups of patients must be exchangeable with each other.\footnote{That is, external validity for each group separately implies internal validity.}
which in turn requires that the way in which treatments were applied was oblivious to (independent of) any features of the individuals that could be relevant to their reactions to treatment. The easiest way to ensure this is by randomisation. Although we are here assuming this was not possible, we can sometimes (albeit rarely) attempt to argue that the data can nevertheless be treated as if they had been randomised.

For a functional model \( Y = f(X, U) \), the defining property of “no confounding” is typically taken as requiring independence (in the observational data) between \( X \) and \( U \): in the notation of Dawid (1979) (see §6 below),

\[
X \perp \perp U. \tag{13}
\]

This is trivially equivalent to \( U \perp \perp X \), i.e. the observational distribution of \( U \) given \( X = x \) does not depend on \( x \), thus mimicking a property already assumed for the case that \( X \) is my decision variable. For an ESE model the above requirement translates as \( X \perp \perp E \), and as \( X \perp \perp Y \) for a PR model. (However, since \( U \), \( E \) and \( Y \) typically do not correspond to any empirically observable variables, the mental exercise required to assess whether the above independence properties hold can be perplexing.)

In the next section we consider just why (13) might be considered as expressing “no confounding”, and extend the analysis to the DT interpretation of this concept.

5.1 Regimes

A helpful way to think about (absence of) confounding is in terms of different data-generating regimes and their relationships.

In the above example we can distinguish three such regimes. One of these is
the observational regime, under which the available data have been observed. Then there are two interventional regimes, corresponding to the circumstance in which an external intervention is made to impose one of the two treatments. It is helpful to introduce a non-stochastic variable $F_X$, with values 0, 1 and $\emptyset$ (read as “idle”): $F_X = 0$ labels the interventional regime with $X = 0$; $F_X = 1$ labels the interventional regime with $X = 1$; while $F_X = \emptyset$ labels the observational regime. There will be a joint distribution of all relevant variables for each of these regimes. Thus $F_X$ has the status of a statistical parameter, indexing which distribution we are referring to. Note that (assuming intervention is perfectly successful), $X = x$ with probability 1 in regime $F_X = x$ ($x = 0, 1$), whereas $X$ will be a genuinely stochastic variable in regime $F_X = \emptyset$.

We have previously interpreted a functional model, and its specialisations such as an ESE or PR model, as incorporating an implicit assumption of “stability”: that the relevant variable $U$ should have the same value, and hence the same distribution, no matter which treatment is applied. In fact this assumption is not quite enough: we further need to assume that $U$ has the same distribution, irrespective of the regime that is operating. This is necessary if we are to justify transfer (under suitable conditions) of information from the observational regime to the interventional regimes. Thus suppose (13) holds. We desire to compute, and contrast, the distributions of the response $Y$ under the two interventional regimes. Under intervention with active treatment, $Y = f(1, U)$ with $U$ having its distribution under $F_X = 1$. In the observational regime, we can estimate the conditional distribution of $Y$ given $X = 1$, which is that of $f(1, U)$ given $X = 1$ under $F_X = \emptyset$. On account of (13) (supposed to apply in the observational regime), this is the same as the marginal distribution of $f(1, U)$ under $F_X = \emptyset$. 
But under our extended stability assumptions, \( U \) has the same distribution in all regimes, so this is indeed the same as the desired distribution of \( Y = f(1, U) \) under \( F_X = 1 \). We have thus shown that, taking together the assumptions of stability, we can deduce “no confounding”, expressed as

\[
(Y | F_X = x) \approx (Y | X = x; F_X = \emptyset) \quad (x = 0, 1),
\]

where the symbol \( \approx \) denotes “has the same distribution as”.

At this point we note that the left-hand side of (14) refers to what we have termed the “hypothetical distribution”, \( P_x \), of \( Y \), under intervention with \( X = x \); while the right-hand side refers to a conditional distribution that is, in principle, estimable from empirical data. All the special ingredients of the functional model have evaporated, and we are left with an expression that is fully meaningful within the DT framework. And within that framework we can simply and directly take property (14) (however it may be justified) as the appropriate expression of “no confounding”.

5.2 Conditional independence

An alternative way of expressing (14) is as follows. First note that, since \( X = x \) with probability 1 under regime \( F_X = x \), (14) is equivalent to

\[
(Y | X = x; F_X = x) \approx (Y | X = x; F_X = \emptyset) \quad (x = 0, 1).
\]

This expresses the conditional distribution of \( Y \), given \( X = x \), as a “modular component”, that can be transferred without change between observational and interventional settings. This modular interpretation of “causality” offers a useful pragmatic take on a slippery philosophical concept.

The distributional identity (15) can also be considered as an expression of the
Statistical Causality

conditional independence property (Dawid 1979; Dawid 1980):

\[ Y \perp F_X | X, \] (16)

which says that the distribution of \( Y \), given information both on the value of \( X \) and on the regime \( F_X \) under which that value arose, is in fact the same for all the regimes. In this way we have converted a causal property into a probabilistic one (albeit involving the non-random regime variable \( F_X \)). Since there is a well-established theory of conditional independence (see §6 below), this is a fruitful reinterpretation that will be particularly helpful for both describing and manipulating causal properties. So henceforth we will work with (16), and its DT interpretation, as our formal expression of “no confounding”.

6 Conditional Independence and Graphs

In this section we recapitulate various aspects of the mathematical theory of conditional independence that will be useful for manipulating causal concepts. For further detail see Dawid (1979); Dawid (1980); Dawid (2002); Constantinou (2013).

For random variables \( X, Y, \ldots \), having joint distribution \( P \), we say \( X \) is independent of \( Y \) given \( Z \), and write \( X \perp Y | Z \), to mean that the distribution of \( X \), given \((Y, Z) = (y, z)\) depends only on the value \( z \) of \( Z \). More formally:

**Definition 1** [Conditional independence] We say \( X \) is conditionally independent of \( Y \) given \( Z \), and write \( X \perp Y | Z \), if, for any measurable set \( A \) in the range of \( X \), there exists a function \( w(Z) \) of \( Z \) alone such that \( P(X \in A | Y, Z) = w(Z) \) \( P \)-almost surely.

\[ \square \]

When we need to specify explicitly the underlying joint distribution \( P \) we write e.g. \( X \perp Y | Z \ [P] \). Independence, \( X \perp Y \), is the special case of conditional in-
dependence when the conditioning variable $Z$ is trivial.

### 6.1 Axioms of conditional independence

Among the general properties of probabilistic conditional independence are the following (Dawid 1979). Here we write $W \preceq Y$ to mean that $W$ is a function of $Y$.

- **P1 “Symmetry”**: $X \perp \perp Y \mid Z \Rightarrow Y \perp \perp X \mid Z$
- **P2**: $X \perp \perp Y \mid X$
- **P3 “Decomposition”**: $X \perp \perp Y \mid Z, W \preceq Y \Rightarrow X \perp \perp W \mid Z$
- **P4 “Weak union”**: $X \perp \perp Y \mid Z, W \preceq Y \Rightarrow X \perp \perp Y \mid (W, Z)$
  
  \[
  \begin{aligned}
  X \perp \perp Y \mid Z \\
  \text{and}
  \end{aligned}
  \]

- **P5 “Contraction”**: $X \perp \perp W \mid (Y, Z) \Rightarrow X \perp \perp (Y, W) \mid Z$.

(The descriptive terms are those given by Pearl (1988), Chapter 3).

It is possible to derive many further properties of CI by regarding P1 to P5 as axioms for a logical system, rather than calling on more specific properties of probability distributions.

### 6.2 Extension to non-stochastic variables

In order for Definition 1 to make sense we must be able to talk about distributions for $X$, which thus has to be a random variable; but (subject to appropriate interpretation of the “almost sure” qualification) $Y$ and $Z$ need not be. In particular, this is the case for expression (16), our interpretation of “no confounding”, which involves the non-stochastic regime indicator variable $F_X$. 
We must exercise a little care when applying the notation and theory of §6.1 to non-stochastic variables, to ensure that these always appear, explicitly or implicitly, as conditioning variables. Nevertheless, suitably interpreted, properties P1–P5 do still hold (Dawid 1980; Constantinou 2013). In fact any deduction made using them will be valid, so long as, in both premisses and conclusions, no non-stochastic variables appear in the left-most term in a conditional independence statement (we are allowed to violate this condition in intermediate steps of an argument). So we can apply P1–P5 freely, even in the presence of non-stochastic variables, so long only as we do not attempt to derive any obviously meaningless assertion.

6.3 Graphical representation

There is a remarkable and technically valuable analogy between conditional independence properties holding between random variables, and separation properties of a directed acyclic graph (DAG) (Lauritzen et al. 1990). This enables us to use graphical methods to streamline probabilistic manipulations.

The graphical analogue of probabilistic conditional independence is the following somewhat complex separation property. Let $A$, $B$, $C$ be sets of nodes of the DAG $\mathcal{D}$. We first form the subgraph $\mathcal{D}'$ of $\mathcal{D}$ that contains only the nodes in $A$, $B$ and $C$, together with all their ancestors in $\mathcal{D}$, and all their connecting arrows: this is the relevant ancestral DAG. Next, whenever two nodes in $\mathcal{D}'$ have a common child but are not already joined by an arrow (are “unmarried”), we insert an undirected edge between them, and then convert all remaining edges to be undirected by dropping the arrowheads: this produces the moralised ancestral graph, $\mathcal{G}'$. Finally, in the undirected graph $\mathcal{G}'$, we check whether every connected
path from a node in $A$ to one in $B$ intersects $C$. If so, we say $C$ \textit{d-separates} $A$ \textit{from} $B$\footnote{The name refers to an alternative, but equivalent, way of expressing this separation property, as described by Pearl (1986); Verma and Pearl (1990).} and write $A \perp_D B \mid C$. It can be shown that, at a purely formal level, and with $\leq$ now interpreted as “is a subset of”, $\subseteq$, this separation property satisfies Axioms P1–P5 of $\S$6.1.

Now suppose that each node $v$ of $D$ has an associated random variable $X_v$. Denote $(X_v : v \in A)$ by $X_A$. We say a joint distribution $P$ for all these variables satisfies the \textit{local directed Markov property} with respect to $D$ if, for every node $v$,

$$X_v \perp \perp X_{\text{nd}(v)} \mid X_{\text{pa}(v)},$$

where (using a self-explanatory analogy with a genetic pedigree) $\text{pa}(v)$ denotes the set of “parents” of node $v$, and $\text{nd}(v)$ its “non-descendents”, in $D$. In this case it can be shown that, whenever we find $A \perp_D B \mid C$ (by inspection of the DAG), we can deduce the probabilistic conditional independence property $X_A \perp \perp X_B \mid X_C [P]$. We term this the \textit{moralisation criterion}.

As an example, the directed acyclic graph (DAG) $D$ of Figure 1 describes the relationships between the evidence and other variables figuring in a criminal trial (Dawid and Evett 1997). The graph is constructed so that the each node corresponds to a variable in the problem, and the assumed dependence structure of the variables satisfies the local directed Markov property: each variable is supposed probabilistically conditionally independent of its non-descendents in the graph, conditional on its graph parents. For example, the distribution of $Y_1$ (measured properties of a tuft of fibres found at the scene), given all other variables, is supposed fully determined by the values of $X_3$ (properties of the suspect’s jumper) and of $A$ (an indicator of whether or not the fibres came from
the suspect’s jumper). Likewise, the distribution of $B$ (who left blood on the jumper?), given all variables other than $Y2$ (the type of that blood) and $R$ (whether or not the blood pattern was a spray), in fact only depends on the values of $N$ (the number of offenders) and $C$ (whether or not the suspect was an offender). Such assessments can often be made at a qualitative level, before attempting numerical specification of probabilities. In turn, that specification is simplified because we only need to describe the conditional distribution for each variable given its graph parents.

Suppose now we wish to query whether $(B, R) \perp \perp (G1, Y1) \mid (A, N)$. The relevant ancestral graph $D'$ is shown in Figure 2 and its moralised version $G'$ in Figure 3. We note that in $G'$ it is impossible to trace a path from either of $B$ or $R$ to either $G1$ or $Y1$ without passing through either $A$ or $N$. Thus $(B, R) \perp \perp (G1, Y1) \mid (A, N)$. From this we deduce the probabilistic conditional independence property $(B, R) \perp \perp (G1, Y1) \mid (A, N)$.

**Caution:** Although every DAG thus describes some collection of conditional independence properties, and can be used to manipulate these, by no means every such collection can be represented by a DAG. In full generality, we may need to use algebraic manipulations, successively applying the CI axioms P1–P5 to derive the implicit consequences of an assumed collection of conditional independencies.

6.3.1 **Markov equivalence** Distinct DAGs can have identical separation properties, and so represent identical collections of conditional independencies. They are then termed *Markov equivalent*.

The *skeleton* of a DAG $D$ is the undirected graph obtained by ignoring the directions of the arrows on the edges of $D$. An *immorality* in $D$ is a configuration
of the form $a \rightarrow c \leftarrow b$, where $a$ and $b$ are parents of a common child $c$ but neither $a \rightarrow b$ nor $b \rightarrow a$.

**Theorem 1** (Frydenberg (1990); Verma and Pearl (1991)) Two DAGs $D_0$ and $D_1$ on the same vertex set $V$ are Markov equivalent if and only if they have the same skeleton and the same immoralities.

**Example 1** There are just three possible DAGs on two nodes:

(i). $A \rightarrow B$

(ii). $A \leftarrow B$

(iii). $A \quad B$.

Since DAGs (i) and (ii) have the same skeleton, and neither has any immoralities, they are Markov equivalent: indeed, they embody no conditional independence properties whatsoever. However, DAG (iii), which has a different skeleton, embodies the non-trivial conditional independence restriction $A \perp \!\!\!\!\perp B$.  

**Example 2** Consider the following DAGs on three nodes:

(i). $A \rightarrow B \rightarrow C$

(ii). $A \leftarrow B \leftarrow C$

(iii). $A \leftarrow B \rightarrow C$

(iv). $A \rightarrow B \leftarrow C$.

These all have the same skeleton. However, whereas DAGs (i), (ii) and (iii) have no immoralities, (iv) has one immorality. Consequently, (i), (ii) and (iii) are Markov equivalent to each other, but (iv) is not Markov equivalent to these. Indeed, (i), (ii) and (iii) all express the conditional independence property $A \perp \!\!\!\!\perp C \mid B$, whereas (iv) expresses the marginal independence property $A \perp \!\!\!\!\perp C$.  

\[\square\]
7 Causal Interpretations of DAGs

It is common, and appears very natural, to want to interpret an arrow $a \rightarrow b$ in a DAG as representing some kind of “direct causal dependence” of $b$ on $a$. But this is a potentially dangerous move, since there is nothing in the DAG semantics, as presented above, to justify it. We prefer a different way of introducing causality into a DAG: by explicitly representing regime indicators and applying the moralisation criterion to the resulting influence diagram (ID), a DAG containing both stochastic and non-stochastic variables. As a simple example, the “no confounding” property (16) is represented by the ID of Figure 4.

Consider now the effect of reversing the arrow from $X$ to $Y$, as shown in Figure 5. Without the intervention node $F_X$, the two graphs would have been Markov equivalent (as was the case for Example 1 (i) and (ii)). Now however we can easily see that they no longer represent equivalent assumptions since, although they have the same skeleton, they have different immoralities. Figure 5 expresses the marginal independence property $Y \perp \perp F_X$, and thus makes it explicit that the marginal distribution of $Y$ is the same, no matter whether, or how, $X$ is subjected to intervention. That is, $X$ has no effect on $Y$ (in any regime).

8 Pearlian DAGs

Consider the DAG of Figure 6. Interpreted purely stochastically, it is nothing but a representation of the following conditional independence properties: $A \perp \perp B$; $D \perp \perp (A, B, C)$; and $E \perp \perp (A, B) | (C, D)$; together with all other properties, such as $E \perp \perp B | (A, C)$, deducible from these using P1–P5 (or, equivalently, readable

\footnote{This develops on an idea introduced by Spirtes et al. (2000); see also Pearl (2009) and Lauritzen (2000).}
off the DAG using the moralisation criterion).

In the approach of Pearl (2009), a DAG such as Figure 6 is taken to represent causal properties. A helpful way of understanding Pearl’s interpretation is to consider the DAG as a shorthand for the influence diagram of Figure 7, in which a non-stochastic intervention node has been associated with every stochastic node. Using the moralisation criterion, we can read off from this augmented DAG that, for example, \( C \perp \perp (D, F_A, F_B, F_D, F_E) \mid (A, B, F_C) \). For \( F_C = \emptyset \) (the only non-trivial case), this says that the ‘natural’ conditional distribution of \( C \), given \( A \) and \( B \), is not further affected by additional conditioning on the value of \( D \), nor by whether or not any or all of \( A, B, D \) or \( E \) arose naturally or by intervention. Similar properties hold for the other domain variables. In particular, we can see that the conditional distribution for a node, given its domain parents, when it is allowed to arise naturally, remains unchanged when its parents are set by intervention (and is thus a modular component, invariant across different regimes). The augmented DAG thus automatically encodes (via moralisation semantics) the assumptions made externally by Pearl, without requiring any new ingredients or concepts; and further makes it easy to read off their implications directly. It also makes it clear that, when endowed with Pearl’s causal interpretation, DAGs that are *prima facie* Markov equivalent (such as \( X \rightarrow Y \) and \( X \leftarrow Y \)) are not causally equivalent, since their augmented forms will not be Markov equivalent. For all these reasons, explicit use of augmented DAGs is to be preferred over Pearl’s shorthand form, which in any case courts confusion with the purely stochastic interpretation of a DAG.
Caution: A Pearlian DAG model, or its augmented DAG equivalent, is justified only to the extent that it models the actual the behaviour of the world in the setting to which it is intended to apply. In particular, we must ask whether or not the various interventional situations are indeed related to the non-interventional one in the specific way represented by the DAG. Since such considerations necessarily involve cross-regime comparisons, no assessment of their appropriateness can be made on the basis of purely observational data.

9 Identifying Causal Effects

Suppose we are interested in the “causal effect” of a treatment variable $T$ on a response variable $Y$. In the DT framework, this requires us to identify, and contrast, the two interventional distributions: $P_1$, for $Y$ in regime $F_T = 1$, and $P_0$, for $Y$ in regime $F_T = 0$. For simplicity we again confine attention to the average causal effect

$$ACE := E(Y \mid F_T = 1) - E(Y \mid F_T = 0).$$

With only observational data, gathered in regime $F_T = \emptyset$, we will not be in a position directly to assess these interventional distributions of $Y$. We will thus need to make assumptions to justify and guide computation of the ACE from such data. Since any such assumptions will have to relate distributions across distinct regimes, they will not be empirically testable if we only have observational data. It will however be important to present some sort of convincing argument for the suitability of any assumptions imposed.

At the simplest level, we might assume “no confounding”: $Y \perp \!\!\!\!\!\!\perp F_T \mid T$. In this case we could simply estimate the observational conditional distribution of $Y \mid T = t, F_T = \emptyset$, and take that as the desired interventional distribution of
\( Y \mid F_T = t \ (t = 0, 1). \) Thus under this assumption we will have

\[
\text{ACE} = E(Y \mid T = 1, F_T = \emptyset) - E(Y \mid T = 1, F_T = \emptyset),
\]

which is straightforwardly estimable from observational data.

However, in many realistic contexts the “no confounding” property will be simply unbelievable: that is to say, we will have confounding: \( Y \not\perp\!\!\!\!\perp F_T \mid T. \) Then (19) might fail. Note that this definition of confounding does not require the existence of what are often called confounding variables, or confounders. But to make progress in identifying ACE we will typically have to introduce further variables, with appropriate properties.

### 9.1 Sufficient covariates

A variable \( U \) is a pre-treatment variable if it exists and is (in principle) observable prior to the point at which the treatment decision is taken. In this case its value, and so its distribution, must be the same under both interventional regimes, \( F_T = 0 \) and \( F_T = 1 \). It will frequently (though not invariably) also be the case that its distribution can be considered the same in the relevant observational regime \( F_T = \emptyset \). Then we will have

\[
U \perp\!\!\!\!\perp F_T.
\]

Such a variable is a covariate.

### 9.2 Unconfounder

When we can not assume “no confounding”, we might be able to tell an alternative, more convincing, story, in terms of a (typically multivariate) covariate \( U \): claiming that we will have no residual confounding, after conditioning on \( U \).
Formally,
\[ Y \perp F_T | (U,T). \]  
(21)

For example, if our data arise from an observational study on patients treated by a certain doctor, who might be allocating treatment according to his own observations \( U \) of the general health of the patient, it could be reasonable to suppose that, conditionally on \( U \), we would have no residual confounding. If we can observe \( U \), we can then use the observational distribution of \( Y \) given \( (U,T = t) \) as the distribution of \( Y \) given \( U \) in the interventional regime \( F_T = t \).

A variable satisfying both (20) and (21) is often called a *confounder*, though a more appropriate term might be *unconfounder*. We shall call it a *sufficient covariate*.

The properties (20) and (21) are represented by the ID of Figure 8.

9.2.1 Functional model
Suppose our starting point was a functional model \( Y = f(T,U) \) (which includes (E)SE and PR models). Since the same function is supposed to apply irrespective of the regime operating, (21) holds trivially. We have further assumed that \( U \) has the same value, and hence the same distribution, in all regimes, so (20) holds. That is, formally at least, \( U \) is an unconfounder. However, in such a formulation the variable \( U \) is typically unobservable (this being a logical necessity in the PR approach, where \( U \) is the pair \( Y \) of potential responses), which limits the operational usefulness of this observation.

9.3 Non-confounding
Specialisations of the above structure are obtained when we can assume that either of the arrows marked \( a \) and \( b \) in Figure 8 is absent. It can readily be
checked that in either case we will have $Y \perp \perp F_T \mid T$: no confounding. We might call such a sufficient covariate $U$ a *non-confounder*, and can safely forget that it ever existed: we can simply apply (19).

The ID with arrow $a$ absent represents the additional property $T \perp U \mid F_T$: that in every regime $T$ is independent of $U$. Since this condition holds trivially for the interventional regimes, where $T$ is constant, it merely requires that $T$ be independent of $U$ in the observational regime — that is, that the variables $U$ that putatively might have affected the doctor’s decision did not in fact do so. This property would be perfectly believable if the doctor had tossed a coin to determine his decision, which is why randomised studies can directly address causal queries. But in the case of an observational study, we would need to make some alternative convincing case for this property: then (and only then) we can treat the study as if it had been randomised. This argument is similar to that of §5.1 for functional models, but — since it involves a real rather than a fictitious variable $U$, and stochastic rather than deterministic relationships — supplies a more operational justification for assuming “no confounding”.

The ID with $b$ absent represents the additional property $Y \perp U \mid T$, which says that the conditional distribution of $Y$ given $(T, U)$ (which, by (21), has already been supposed the same in all regimes) does not in fact depend on $U$: that is, $U$ is not predictive of outcome. In that case, even if $U$ is associated with treatment assignment, this will not generate confounding.

\[^{11}\text{i.e., over and above properties (21) and (20).}\]
9.4 Deconfounding

More generally, suppose $U$ is a sufficient covariate that is observed in the observational regime. Define

$$\text{SCE}_U := \text{E}(Y \mid U, F_T = 1) - \text{E}(Y \mid U, F_T = 0),$$

(22)

the specific causal effect of treatment, given $U$. This is a random variable, a function of $U$, whose value $\text{SCE}_U(u)$ when $U = u$ is the average treatment effect in the subgroup of individuals having $U = u$.

Now $T = t$ with probability 1 under $F_t$. Then using (21) we find $\text{E}(Y \mid U, F_T = t) = \text{E}(Y \mid U, T = t, F_T = t) = \text{E}(Y \mid U, T = t, F_T = \emptyset)$\(^{12}\) We deduce

$$\text{SCE}_U = \text{E}(Y \mid U, T = 1, F_T = \emptyset) - \text{E}(Y \mid U, T = 0, F_T = \emptyset),$$

(23)

so that $\text{SCE}_U$ is estimable from observational data. This is a reflection of the fact that we have no confounding conditional on $U$.

Also, by the “extension of the conversation” rule of probability, we have

$$\text{E}(Y \mid F_T = t) = \text{E}\{\text{E}(Y \mid U, F_T = t) \mid F_T = t\}$$

$$= \text{E}\{\text{E}(Y \mid U, F_T = t) \mid F_T = \emptyset\}$$

by (20). It follows that

$$\text{ACE} = \text{E}(\text{SCE}_U \mid F_T = \emptyset).$$

(24)

That is, for any sufficient covariate $U$, the overall average causal effect is the observational expectation of the associated specific causal effect. Since, by (23), $\text{SCE}_U$ is itself an observationally estimable quantity, formula (24) allows us to estimate $\text{ACE}$ whenever we can observe a sufficient covariate.

\(^{12}\)More accurately, these identifications require an additional positivity condition (Guo and Dawid 2010), which will typically be satisfied.
Note that in the PR framework, where we take \( U = Y \), SCE becomes \( Y_1 - Y_0 \), the “individual causal effect”, ICE. Then (24) shows that \( \text{ACE} = \text{E(ICE)} \). However since ICE is necessarily unobservable, this formal identity has no operational content.

9.5 Effect of treatment on the treated

Suppose that I am thinking of taking aspirin, and regard myself as exchangeable with those individuals in the data who did in fact receive aspirin — though not necessarily with those who did not. I can then use the treated group to assess my hypothetical expected response \( \text{E}(Y | F_T = 1) \) for \( Y \), were I to take the aspirin; but it seems I am not in a position to assess the contrasting hypothetical expectation, \( \text{E}(Y | F_T = 0) \), and so cannot assess my personal “effect of treatment”. However, in the presence of a sufficient covariate \( U \) — even if not observed — I may be able to do so.

We define the effect of treated on the treated as

\[
\text{ETT} := \text{E}(\text{SCE}_U | T = 1, F_T = \emptyset). \tag{25}
\]

That is, ETT is the average, in the observational regime, of the specific causal effect (defined relative to \( U \)), over those individuals who did in fact receive the aspirin, \( T = 1 \) — and are thus “like me”.

It might appear that, in the presence of a choice over which sufficient covariate \( U \) to use in (25), that choice might affect the value of ETT. Fortunately it turns out that this is not so, on account of the following result (Geneletti and Dawid 2011):

**Theorem 2** Suppose \( \text{Pr}(T = 1 | F_T = \emptyset) > 0 \). Then, for any sufficient covariate
$U$, ETT defined by (25) satisfies

$$ETT = \frac{E(Y | F_T = \emptyset) - E(Y | F_T = 0)}{\Pr(T = 1 | F_T = \emptyset)}.$$  \hspace{1cm} (26)

We have previously noted that, within the PR framework, we can formally regard the pair $Y$ of potential responses as a sufficient covariate. In that case the SCE becomes the ICE, $Y_1 - Y_0$, and (25) delivers $ETT = E(Y_1 - Y_0 | T = 1, F_T = \emptyset)$, which is the usual PR definition of ETT. However the above argument shows that the PR framework is inessential for defining this quantity.

Formula (26) shows that we can identify ETT whenever we can observe the response $Y$ in the observational regime ($F_T = \emptyset$), and also in a sample of people from whom the treatment was withheld ($F_T = 0$). And although the definition of ETT supposes the existence of some sufficient covariate, it is not necessary to have observations on it.

### 9.6 Reduction of sufficient covariate

Suppose $U$ is a sufficient covariate. A function $V$ of $U$ is a sufficient reduction of $U$ if $V$ is itself a sufficient covariate. Since property (20) for $V$ follows immediately from the same property for $U$, we only need investigate whether property (27) holds for $V$:

$$Y \perp \perp F_T | (V, T).$$  \hspace{1cm} (27)

There are various additional conditions we can impose to ensure this. One is the following:

**Condition 1 (Treatment-sufficient reduction)**

$$T \perp \perp U | (V, F_T = \emptyset).$$  \hspace{1cm} (28)
That is, in the observational regime, the choice of treatment depends on \( U \) only through the value of \( V \).

Note that this condition does not involve the outcome variable \( Y \) — except for the essential requirement that the starting variable \( U \) itself be a sufficient covariate for the effect of \( T \) on \( Y \). Also note that, since \( T \) is constant in any interventional regime, (28) is equivalent to

\[
T \perp \perp U \mid (V, F_T). \tag{29}
\]

Also, since \( V \) is a function of \( U \), we trivially have

\[
V \perp \perp F_T \mid U, \tag{30}
\]

as well as

\[
Y \perp \perp V \mid (U, T, F_T). \tag{31}
\]

The following result now follows on applying the moralisation criterion to the ID of Figure 9, which faithfully represents the conditional independence properties (20), (30), (29), (21) and (31), to deduce (27):

**Theorem 3** Suppose \( U \) is a sufficient covariate, and let be \( V \) be a function of \( U \) such that Condition 1 holds. Then \( V \) is a sufficient covariate.

### 9.6.1 Propensity score

An alternative description of treatment-sufficient reduction is as follows. Using P1, the defining property (28) can be expressed as

\[
U \perp \perp T \mid (V, F_T = \emptyset). \tag{32}
\]

In this form it asserts that, in the observational regime, the conditional distribution of \( U \) given \( V \) is the same, whether further conditioned on \( T = 0 \), or on \( T = 1 \): that is to say, \( V \) is a balancing score for \( U \) (Rosenbaum and Rubin 1983).
Property (32) can also be fruitfully interpreted as follows. Consider the family \( \mathcal{Q} = \{Q_0, Q_1\} \) comprising the pair of observational conditional distributions for \( U \), given, respectively, \( T = 0 \) and \( T = 1 \). Then (32) asserts that \( V \) is a sufficient statistic (in the usual Fisherian sense) for this family. In particular, a minimal treatment-sufficient reduction is obtained as a minimal sufficient statistic for \( \mathcal{Q} \): viz., any \((1,1)\)-function of the likelihood ratio statistic \( \Lambda := q_1(X)/q_0(X) \). We might term such a minimal treatment-sufficient covariate a propensity variable, since one form for it is the treatment-assignment probability

\[
\Pi := \Pr(T = 1 \mid U, F_T = \emptyset) = \pi \Lambda/(1 - \pi + \pi \Lambda)
\]  

(33)

(where \( \pi := \Pr(T = 1 \mid F_T = \emptyset) \)), which is known as the propensity score (Rosenbaum and Rubin 1983). Either \( \Lambda \) or \( \Pi \) supplies a 1-dimensional sufficient reduction of the original, perhaps highly multivariate, sufficient covariate \( U \).13

9.7 do-calculus

We here make use of the notation of Pearl (2009) in which e.g. \( p(y \mid x, \tilde{z}) \) refers to \( \Pr(Y = y \mid X = x, F_Z = z) \), it being implicit that \( z \neq \emptyset \), and all unmentioned intervention variables are idle.

Let \( X, Y, Z, W \) be arbitrary sets of variables in a problem also involving intervention variables. The following rules follow immediately from the definition of conditional independence.14

Rule 1 (Insertion/deletion of observations) If \( Y \perp \!\!\!\!\perp Z \mid (X, F_X \neq \emptyset, W) \) then

\[
p(y \mid \tilde{x}, z, w) = p(y \mid \tilde{x}, w).
\]  

(34)

13However, this property may not be as useful as may first appear (Guo and Dawid 2010).

14We assume throughout any positivity conditions required to ensure that the relevant conditional probabilities are well-defined.
Rule 2 (Action/observation exchange) If \( Y \perp \perp F \mid (X, F_X \neq \emptyset, Z, W) \), then

\[
p(y \mid \bar{x}, \bar{z}, w) = p(y \mid \bar{x}, z, w).
\] (35)

Rule 3 (Insertion/deletion of actions) If \( Y \perp \perp F \mid (X, F_X \neq \emptyset, W) \), then

\[
p(y \mid \bar{x}, \bar{z}, w) = p(y \mid \bar{x}, w).
\] (36)

Successive application of these rules, coupled with the property \( F_X = x \Rightarrow X = x \) and the laws of probability, can sometimes allow one to express a “causal” expression in purely observational terms. This was the essence of the argument in §9.2 above, which (assuming for simplicity that all variables are discrete) can be expressed in general terms as:

**Theorem 4 (Back-door formula)** Suppose that

\[
Z \perp \perp F_X \quad (37)
\]

\[
Y \perp \perp F_X \mid (X, Z). \quad (38)
\]

Then

\[
p(y \mid \bar{x}) = \sum_z p(y \mid Z = z, X = x) p(Z = z). \quad (39)
\]

The most usual application of this *do-calculus* is for a model represented by a Pearlian DAG. However it is easiest to work with the augmented DAG. We first note that conditioning on \( F_X \neq \emptyset \) has the effect of removing all arrows incoming to the set \( X \) other than from \( F_X \). The resulting reduced DAG can then be interrogated, using the usual moralisation criterion, to deduce conditional independence properties that can be used as input to Rules 1–3. In this context it can be shown

\[\text{[15] Pearl’s analysis, like its precursor in} \text{[16] Spirtes et al. (1999)} \text{, works with equivalent, somewhat more complex, formulations in terms of unaugmented DAGs.}\]

\[\text{[16] In fact Rule 1 is now redundant.}\]
that, whenever there exists a reduction of a causal expression to purely observational terms, it can be found by applying the do-calculus.

10 Instrumental Variables

In the presence of an unobserved sufficient covariate $U$, it is typically not possible to estimate the average causal effect, ACE, of a treatment variable $X$ on a response variable $Y$ from observational data. Some progress can be made if we can assume the existence of an observable instrumental variable $Z$, which can be thought of as an imperfect proxy for an intervention. The assumptions required in such a case are typically expressed in informal terms such as (Martens et al. 2006):

(i). $Z$ has a causal effect on $X$

(ii). $Z$ affects the outcome $Y$ only through $X$ ("no direct effect of $Z$ on $Y$"")

(iii). $Z$ does not share common causes with the outcome $Y$ ("no confounding of the effect of $Z$ on $Y$"").

These might be formalised as observational conditional independence properties, such as:

\[ X \perp \!\!\!\! \perp Z \]

\[ U \perp \!\!\!\! \perp Z \] \hspace{1cm} (41)

\[ Y \perp \!\!\!\! \perp Z \mid (X, U). \] \hspace{1cm} (42)

Note the analogy between (41) and (20), and (42) and (21), where (with $T$ relabelled as $X$) $Z$ takes the place of $F_X$. However, unlike the case of an imposed
intervention, $Z$ does not determine the value of $X$, but merely has some association with it, as described by (40). These assumptions are represented by the DAG of Figure 10.  

For all that this might be a fruitful analogy, requirements (40)–(42), and Figure 10 leave something to be desired: since they relate solely to the observational regime, they can not, of themselves, have any causal consequences — at best these are left implicit, which leaves room for confusion. It is far better to make the requisite causal assumptions explicit. We do this by elaborating Figure 10 to explicitly include the nonstochastic regime indicator $F_X$ for $X$, as in Figure 11. For $F_X = \emptyset$ this recovers the assumptions encoded in Figure 10, but in addition it relates the observational structure to what would happen under an intervention to set $X$. In particular, it clarifies that $U$ is assumed to be a sufficient covariate for the effect of $X$ on $Y$, and further encodes:

\[
U \independent Z \mid F_X \quad (43)
\]

\[
Y \independent Z \mid (X, U, F_X). \quad (44)
\]

Properties (44) and (43) extend (41) and (42) to apply under intervention, as well as observationally.

### 10.1 Linear model

Suppose now all the observables are univariate, and we can describe the dependence of $Y$ on $(X, U)$ (which we have assumed the same in all regimes) by a linear model:

\[
E(Y \mid X, U, F_X) = W + \beta X \quad (45)
\]

\footnote{For (40), we need to assume that this is a faithful representation.}

\footnote{Figure 11 also encodes the additional, but inessential, property $Z \independent F_X$.}
for some function $W$ of $U$.

We deduce

$$E(Y \mid F_X = x) = w_0 + \beta x,$$

where $w_0 := E(W \mid F_X = x)$ is a constant independent of $x$, since $U \perp \perp F_X$.

Thus $\beta$ can be interpreted causally, as describing how the mean of $Y$ changes in response to manipulation of $X$. Our aim is to identify $\beta$.

By (44), (45) is also $E(Y \mid X, Z, U, F_X = \emptyset)$. Then

$$E(Y \mid Z, F_X = \emptyset) = E(W \mid Z, F_X = \emptyset) + \beta E(X \mid Z, F_X = \emptyset).$$

But by (43) the first term on the right-hand side is constant. Thus

$$E(Y \mid Z, F_X = \emptyset) = \text{constant} + \beta E(X \mid Z, F_X = \emptyset). \quad (46)$$

Equation (46) relates two functions of $Z$, each of which can be identified from observational data. Consequently (so long as neither side is constant) we can identify the causal parameter $\beta$ from such data. Indeed it readily follows from (46) that (in the observational regime) $\beta = \text{Cov}(Y, Z)/\text{Cov}(X, Z)$, which can be estimated by the ratio of the coefficients of $Z$ in the sample linear regressions of $Y$ on $Z$ and of $X$ on $Z$.

### 10.2 Binary variables

When all the observable variables $Z, X, Y$ are binary, without making further assumptions we can not fully identify the “causal probability” $P(Y = 1 \mid F_X = x)$ from observational data. However, we can develop inequalities it must satisfy.

This approach was instigated by Manski (1990). His inequalities were refined by Balke and Pearl (1997), under the strong additional condition of deterministic
dependence of $X$ on $(Z,U)$ and of $Y$ on $(X,U)$. This condition was shown to be unnecessary by Dawid (2003), where a fully stochastic decision-theoretic approach was developed. In either approach, the analysis involves subtle convex duality arguments.

11 Dynamic Treatment Strategies

In the ID of Figure 12, the $L$’s represent attributes of a patient, the $T$’s treatments that can be applied, and $Y$ a response of interest. These variables are supposed generated in the order shown, each in response to all its predecessors. The non-stochastic regime indicator node $\sigma$ can take value $\emptyset$, indicating the observational regime; otherwise, a value $\sigma = s$ describes a hypothetical treatment strategy, specifying how treatment $T_1$ should be chosen in response to observation of $L_1$, and how $T_2$ should be chosen in response to observation of $(L_1,T_1,L_2)$. Typically such a strategy will prescribe deterministic choices, but there is no difficulty in allowing further randomisation. The task is to infer the consequence, $E(Y | \sigma = s)$, of such a hypothetical strategy from properties of the observational regime $\sigma = \emptyset$.

Figure 12 encodes the following conditional independencies:

\[
L_1 \indep \sigma \quad (47)
\]
\[
L_2 \indep \sigma | (L_1,T_1) \quad (48)
\]
\[
Y \indep \sigma | (L_1,T_1,L_2,T_2). \quad (49)
\]

Condition (49), for example, says that the distribution of $Y$, given the previous variables $(L_1,T_1,L_2,T_2)$, in the observational regime $\sigma = \emptyset$ would also apply.

\[^{19}\text{An alternative interpretation of this condition is in terms of potential outcomes.}\]
under the operation of an imposed strategy $\sigma = s$. This is a “no residual confounding” type of assumption, that might or might not be appropriate. When (47)–(49) apply, we say we have *sequential ignorability*.

We will always have

\[
\begin{align*}
p(l_1, t_1, l_2, t_2, y | \sigma = s) &= p(l_1 | \sigma = s) \\
&\quad \times p(t_1 | l_1, \sigma = s) \\
&\quad \times p(l_2 | t_1, l_1, \sigma = s) \\
&\quad \times p(t_2 | l_1, t_1, l_2, \sigma = s) \\
&\quad \times p(y | l_1, t_1, l_2, t_2, \sigma = s).
\end{align*}
\]

Now (51) and (53) are specified by the strategy $s$. Also, under sequential ignorability, in (50), (52) and (54) we can replace $\sigma = s$ by $\sigma = \emptyset$, so that those terms are estimable from observational data. We will thus have all the ingredients needed to identify the joint distribution of all variables under the strategy $\sigma = s$, and then by marginalisation we can identify the desired consequence, $E(Y | \sigma = s)$. This computation, which can be effectively restructured as a recursion (Dawid and Didelez 2010), reduces to the *g-computation* formula of Robins (1986). That paper (see also Chakraborty and Murphy (2014)) set the problem up in a PR framework, assuming the simultaneous existence of potential responses $(L_{1s}, L_{2s}, Y_s)$ for each possible strategy $\sigma = s$, subject to certain consistency requirements, sequential ignorability then being expressed as a conditional independence property involving these potential responses. Our DT approach is more straightforward to interpret, justify and implement, as well as allowing for randomised strategies.

It will often be unrealistic to impose the “no residual confounding” assump-
tions of sequential ignorability, at least without further justification. Such an assumption might become more reasonable when additional variables are added to the system: variables that could not, however, be usable by the considered strategy $\sigma = s$. In such a case it is possible to add further conditions, generalising those of §9.3, which when acceptable would imply that we will indeed have sequential ignorability. For further details see Dawid and Didelez (2010); Dawid and Constantinou (2014).

12 Discussion

The decision-theoretic language for causality has sometimes been criticised for not being as rich as that of alternative approaches, such as PR models, which can make statements, in their own mathematical terms, that simply have no DT counterpart. I regard this as a strength, not a weakness: formal mathematical expressions (for example, the variance of the ICE—see §3.4) that do not relate directly to features of the real world are at best unnecessary, and at worst dangerously misleading. Within DT we are not plagued with “the fundamental problem of causal inference” (Holland 1986), which is only a self-created problem of the PR approach. The DT approach also fosters healthy scepticism of other methods, such as “principal stratification” (Frangakis and Rubin 2002), that depend crucially on the philosophically perplexing assumption of the real simultaneous existence of potential response pairs (Dawid and Didelez 2012), together with necessarily untestable assumptions about their properties. Within the ambit of problems that are well-posed, the DT framework has all the expressive power necessary, uncluttered by unnecessary and distracting formal mathematical ingredients.
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Figure 1: Directed graph \( D \) for criminal evidence

Figure 2: Ancestral subgraph \( D' \)

Figure 3: Moralised ancestral subgraph \( G' \)

Figure 4: No confounding: \( X \) causes \( Y \)
Figure 5: $X$ does not cause $Y$

Figure 6: A probabilistic DAG

Figure 7: Augmented DAG

Figure 8: Sufficient covariate

Figure 9: Treatment-sufficient reduction
Figure 10: Instrumental variable

Figure 11: Instrumental variable with regimes

Figure 12: Sequential ignorability