

# Integrated Inferences

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# Preface

This book has four main parts:

- Part I introduces causal models and a Bayesian approach to learning about them and drawing inferences from them.
- Part II applies these tools to strategies that use process tracing, mixed methods, and “model aggregation.”
- Part III turns to design decisions, exploring strategies for assessing what kind of data is most useful for addressing different kinds of research questions given knowledge to date about a population or a case.
- In Part IV we put models into question and outline a range of strategies one can use to justify and evaluate causal models.

We have developed an R package—`CausalQueries`—to accompany this book, hosted on Cran. In addition, a supplementary Guide to Causal Models serves as a guide to the package and provides the code behind many of the models used in this book.



# Chapter 1

## Introduction

Here is the key idea of this book.

Quantitative social scientists spend a lot of time trying to understand causal relations between variables by looking across large numbers of cases to see how outcomes differ when potential causes differ. This strategy relies on variation in causal conditions across units of analysis, and the quality of the resulting inferences depends in large part on what forces give rise to that variation.

Qualitative social scientists, like historians, spend a lot of time looking at a smaller set of cases and seek to learn about causal relations by examining evidence of causal processes in operation within these cases. Qualitative scholars rely on theories of how things work, theories that specify what should be observable within a case if indeed an outcome were generated by a particular cause.

These two approaches seem to differ in what they seek to explain—individual-level or population-level outcomes; in the forms of evidence they require—cross-case variation or within-case detail; and in what they need to assume—knowledge of assignment processes or knowledge of causal processes.

The central theme of this book is that this distinction, though culturally real (Goertz and Mahoney (2012)), is neither epistemologically deep nor analytically helpful. Social scientists can work with causal models that simultaneously exploit cross-case variation and within-case detail, that address both case-level and population-level questions, and that both depend on,

and contribute to developing theories of how things work.<sup>1</sup>

We describe an approach to doing this in which researchers *form* causal models, *update* those models using data, and then *query* the models to get answers to particular causal questions. This framework is very different from standard statistical approaches in which researchers focus on selecting the best estimator to estimate a particular estimand of interest. In a causal models framework, the model itself gets updated, not the estimate: we begin by learning about processes, and only then draw inferences about particular causal relations of interest, either at the case level or at the population level.

We do not claim that a causal-model-based approach is the best or only strategy suited to addressing causal questions. There are plenty of settings in which other approaches would likely work better. But we do think that the approach holds considerable promise — allowing researchers to combine disparate data in a principled way to ask a vast range of causal questions, helping integrate theory and empirics in a compelling way, and providing coherent guidance on research design — and that it should have a place in the applied researcher’s toolkit.

Our goals in this book are to motivate this approach; provide an introduction to the theory of structural causal models; provide practical guidance for setting up, updating, and querying causal models; and show how the approach can inform key research-design choices, especially case-selection and data-collection strategies.

## 1.1 The Case for Causal Models

There are three closely related motivations for embracing a causal models approach. One is a concern over the limits of design-based inference. A second is an interest in integrating qualitative knowledge with quantitative approaches. A third is an interest in better connecting empirical strategies to theory.

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<sup>1</sup>Indeed, though we will sometimes follow convention and refer to “within case” and “cross-case” observations, all data are data on cases and enter into analysis in the same fundamental way: we are always asking how consistent a given data pattern is with alternative sets of beliefs.

### 1.1.1 The limits to design-based inference

To caricature positions a bit, consider the difference between an engineer and a skeptic. The engineer tackles problems of causal inference using models: theories of how the world works, generated from past experiences and applied to the situation at hand. They come with prior beliefs about a set of mechanisms operating in the world and, in a given situation, will ask whether the conditions are in place for a known mechanism to operate effectively. The skeptic, on the other hand, maintains a critical position, resisting the importation of beliefs that are not supported by evidence in the case at hand.

The engineer’s approach echoes what was until recently a dominant orientation among social scientists. At the turn of the current century, much analysis—both empirical and theoretical—took the form of modelling processes (“data generating processes”) and then interrogating those models.

Over the last two decades, however, skeptics have raised a set of compelling concerns about the assumption-laden nature of standard regression analysis, while also clarifying how valid inferences can be made with limited resort to models in certain research situations. The result has been a growth in the use of design-based inference techniques that, in principle, allow for model-free estimation of causal effects (see Dunning (2012), Gerber et al. (2004), Druckman et al. (2011), Palfrey (2009) among others). These include lab, survey, and field experiments and natural-experimental methods exploiting either true or “as-if” randomization by nature. With the turn to experimental and natural-experimental methods has come a broader conceptual shift, with a growing reliance on the “potential outcomes” framework which provide a clear language for thinking about causation (see Rubin (1974), Splawa-Neyman et al. (1990) among others) without having to invoke fully specified models of data-generating processes.

The ability to estimate average effects and to characterize uncertainty—for instance calculating  $p$ -values and standard errors—without resort to models is an extraordinary development. In Fisher (2017)’s terms, with these tools, randomization processes provide a “reasoned basis for inference,” placing empirical claims on a powerful footing.

Excitement about the strengths of these approaches has been mixed with various concerns regarding how the approach shapes inquiry. We highlight two.

The first concern—raised by many in recent years (e.g., Thelen and Mahoney (2015))—is about design-based inference’s scope of application. While experimentation and natural experiments represent powerful tools, the range of research situations in which model-free inference is possible is inevitably limited. For a wide range of causal conditions of interest both to social scientists and to society, controlled experimentation is impossible, and true or “as-if” randomization is absent. Moreover, limiting our focus to those questions for which, or situations in which, exogeneity can be established “by design” would represent a dramatic narrowing of social science’s ken. To be clear, this is not an argument against experimentation or design-based inference when these can be used; rather it is an argument for why social science needs a broader set of tools.

The second concern is more subtle. The great advantage of design-based inference is that it liberates researchers from the need to rely on models to make claims about causal effects. The risk is that, in operating model-free, researchers end up learning about effect sizes but not about models. Yet often the model is the thing we want to learn about. Our goal as social scientists is to come to grips with how the world works, not simply to collect propositions about the effects that different causes have had on different outcomes in different times and places. It is through models that we derive an understanding of how things might work in contexts and for processes and variables that we have not yet studied. Thus, our interest in models is intrinsic, not instrumental. By taking models out of the equation, as it were, we limit the potential for learning about the world.

### 1.1.2 Qualitative and mixed-method inference

Recent years have seen the elucidation of the inferential logic behind “process tracing” procedures used in qualitative political science and other disciplines. On our read of this literature, the logic of process tracing in these accounts depends on a particular form of model-based inference.<sup>2</sup> While process trac-

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<sup>2</sup>As we describe in Humphreys and Jacobs (2015), the term “qualitative research” means many different things to different scholars, and there are multiple approaches to mixing qualitative and quantitative methods. There we distinguish between approaches that suggest that qualitative and quantitative approaches address distinct, if complementary, questions; those that suggest that they involve distinct measurement strategies; and those that suggest that they employ distinct inferential logics. The approach that we em-

ing as a method has been around for more than three decades (e.g., George and McKeown (1985)), its logic has been most fully laid out by qualitative methodologists in political science and sociology over the last 15 years (e.g., Bennett and Checkel (2015), George and Bennett (2005), Brady and Collier (2010), Hall (2003), Mahoney (2010)). Whereas King et al. (1994) sought to derive qualitative principles of causal inference within a correlational framework, qualitative methodologists writing in the wake of “KKV” have emphasized and clarified process-tracing’s “within-case” inferential logic: in process tracing, explanatory hypotheses are tested based on observations of what happened within a case, rather than on observation of covariation of causes and effects across cases.

The process-tracing literature has also advanced increasingly elaborate conceptualizations of the different kinds of probative value that within-case evidence can yield. For instance, qualitative methodologists have explicated the logic of different test types (“hoop tests”, “smoking gun tests”, etc.) involving varying degrees of specificity and sensitivity (Collier (2011), Mahoney (2012)).<sup>3</sup> Other scholars have expressed the leverage provided by process-tracing evidence in Bayesian terms, moving from a set of discrete test types to a more continuous notion of probative value (Fairfield and Charman (2017), Bennett (2015), Humphreys and Jacobs (2015)).<sup>4</sup>

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ploy in Humphreys and Jacobs (2015) connects most with the third family of approaches. Most closely related, in political science, is work in Glynn and Quinn (2011), in which researchers use knowledge about the empirical joint distribution of the treatment variable, the outcome variable, and a post-treatment variable, alongside assumptions about how causal processes operate, to tighten estimated bounds on causal effects. In the present book, however, we move toward a position in which fundamental differences between qualitative and quantitative inference tend to dissolve, with all inference drawing on what might be considered a “qualitative” logic in which the researcher’s task is to confront a pattern of evidence with a theoretical logic.

<sup>3</sup>A smoking-gun test is a test that seeks information that is only plausibly present if a hypothesis is true (thus, generating strong evidence for the hypothesis if passed); a hoop test seeks data that should certainly be present if a proposition is true (thus generating strong evidence against the hypothesis if failed); and a doubly decisive test is both smoking-gun and hoop (for an expanded typology, see also Rohlfing (2013)).

<sup>4</sup>In Humphreys and Jacobs (2015), we use a fully Bayesian structure to generalize Van Evera’s four test types in two ways: first, by allowing the probative values of clues to be continuous; and, second, by allowing for researcher uncertainty (and, in turn, updating) over these values. In the Bayesian formulation, use of process-tracing information is not formally used to conduct tests that are either “passed” or “failed”, but rather to update beliefs about different propositions.

Yet, conceptualizing the different ways in which probative value might operate leaves a fundamental question unanswered: what gives within-case evidence its probative value with respect to causal relations? We do not see a clear answer to this question in the current process-tracing literature. Implicitly—but worth rendering explicit—*the probative value of process-tracing evidence depends on researcher beliefs that come from outside of the analysis in question.* We enter a research situation with a model of how the world works, and we use this model to make inferences given observed patterns in the data — while at the same time updating those models based on the data.

A key aim of this book is to demonstrate the role that models can — and, in our view, must — play in drawing case-level causal inferences and to clarify conditions under which these models can be defended. To do so we draw on an approach to specifying causal models developed originally in computer science and that predates this work in qualitative methodology. The broad approach, described in Cowell et al. (1999) and Pearl (2009), is consistent with the potential outcomes framework, and provides rules for updating on population and case level causal queries from different types of data.

In addition to clarifying the logic of qualitative inference, we will argue that such causal models can also enable the systematic integration of qualitative and quantitative forms of evidence. Social scientists are increasingly developing mixed-method research designs, research strategies that combine quantitative with qualitative forms of evidence (Small, 2011). A typical mixed-methods study includes the estimation of causal effects using data from many cases as well as a detailed examination of the processes taking place in a few cases. Now-classic examples of this approach include Lieberman’s study of racial and regional dynamics in tax policy (Lieberman (2003)); Swank’s analysis of globalization and the welfare state (Swank (2002)); and Stokes’ study of neoliberal reform in Latin America (Stokes (2001)). Major recent methodological texts provide intellectual justification of this trend toward mixing, characterizing small-*n* and large-*n* analysis as drawing on a single logic of inference and/or as serving complementary functions (King et al. (1994); Collier et al. (2004)). The American Political Science Association now has an organized section devoted in part to the promotion of multi-method investigations, and the emphasis on multiple strategies of inference research is now embedded in guidelines from many research funding agencies (Creswell and Garrett, 2008).

However, while scholars frequently point to the benefits of mixing correlational and process-based inquiry (e.g., Collier et al. (2010), p.~181), and have sometimes mapped out broad strategies of multi-method research design (Lieberman (2005), Seawright and Gerring (2008)), they have rarely provided specific guidance on how the integration of inferential leverage should unfold. In particular, the literature has not supplied specific principles for aggregating findings—whether mutually reinforcing or contradictory—across different modes of analysis.<sup>5</sup> As we aim to demonstrate in this book, however, grounding inference in causal models provides a very natural way of combining information of the  $X, Y$  variety with information about the causal processes connecting  $X$  and  $Y$ . The approach that we develop here can be readily addressed both to the case-oriented questions that tend to be of interest to qualitative scholars and to the population-oriented questions that tend to motivate quantitative inquiry.

As will become clear, when we structure our inquiry in terms of causal models, the conceptual distinction between qualitative and quantitative inference becomes hard to sustain. Notably, this is not because all causal inference depends fundamentally on covariation but because in a causal-model-based inference, what matters for the informativeness of a piece of evidence is how that evidence alters beliefs about a model, and in turn, a query. While the apparatus that we present is formal, the approach—in asking how pieces of evidence drawn from different parts of a process map on to a base of theoretical knowledge—is arguably most closely connected to process tracing in its core logic.

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<sup>5</sup>A small number of exceptions stand out. In the approach suggested by Gordon and Smith (2004), for instance, available expert (possibly imperfect) knowledge regarding the operative causal mechanisms for a small number of cases can be used to anchor the statistical estimation procedure in a large-N study. Western and Jackman (1994) propose a Bayesian approach in which qualitative information shapes subjective priors which in turn affect inferences from quantitative data. Relatedly, in Glynn and Quinn (2011), researchers use knowledge about the empirical joint distribution of the treatment variable, the outcome variable, and a post-treatment variable, alongside assumptions about how causal processes operate, to tighten estimated bounds on causal effects. Seawright (2016) presents an informal framework in which case studies are used to test the assumptions underlying statistical inferences, such as the assumption of no-confounding or the stable-unit treatment value assumption (SUTVA).

### 1.1.3 Connecting theory and empirics

The relationship between theory and empirics has been a surprisingly uncomfortable one in political science. In a recent intervention, for instance, Clarke and Primo (2012) draw attention to and critique political scientists' widespread reliance on the "hypothetico-deductive" (H-D) framework, in which a theory or model is elaborated, empirical predictions derived, and data sought to test these predictions and the model from which they derive. Clarke and Primo draw on decades of scholarship in the philosophy of science pointing to deep problems with the H-D framework, including with the idea that the truth of a model logically derived from first principles can be *tested* against evidence.

In fact the relationship between theory and evidence in social inquiry is often surprisingly unclear both in qualitative and quantitative work. We can perhaps illustrate it best, however, by reference to qualitative work, where the centrality of theory to inference has been most emphasized. In process tracing, theory is what justifies inferences. In their classic text on case study approaches, George and Bennett (2005) describe process tracing as the search for evidence of "the causal process that a theory hypothesizes or implies" (6). Similarly, Hall (2003) conceptualizes the approach as testing for the causal-process-related observable implications of a theory; Mahoney (2010) indicates that the events for which process tracers go looking are those posited by theory (128); and Gerring (2006) describes theory as a source of predictions that the case-study analyst tests (116). Theory, in these accounts, is supposed to help us figure out where to look for discriminating evidence.

What is not clear, however, is how researchers can derive within-case empirical predictions from theory and how exactly doing so provides leverage on a causal question. From what elements of a theory can scholars derive informative within-case observations? Of the many possible observations suggested by a theory, how can we determine which would add probative value to the evidence already at hand? How do the evidentiary requisites for drawing a causal inference, given a theory, depend on the particular causal question of interest—on whether, for instance, we are interested in identifying the cause of an outcome in a case, estimating an average causal effect, or identifying the pathway through which an effect is generated? Perhaps most confusingly, if the theory tells us what to look for to draw an inference, can the inferences be about the theory itself or are we constrained to make theory

dependent inferences? In short, how exactly can we ground causal inferences from within-case evidence in background knowledge about how the world works?

Much quantitative work in political science features a similarly weak integration between theory and research design. The modal inferential approach in quantitative work, both observational and experimental, involves looking for correlations between causes and outcomes, with less regard for intervening or surrounding causal relationships.<sup>6</sup> If a theory suggests a *set* of relations, it is common to examine these separately—does *A* cause *B* does *B* cause *C*? are relations stronger or weaker here or there?—without standard procedures for bringing the disparate pieces of evidence together to form theoretical conclusions. More attention has been paid to empirical implications of theoretical models than to theoretical implications of empirical models.

In this book, we seek to show how scholars can simultaneously make fuller and more explicit use of theoretical knowledge in designing their research projects and analyzing data and make use of data to update on theoretical models. Like Clarke and Primo, we treat models not as veridical accounts of the world but as maps: maps, based on prior theoretical knowledge, about causal relations in a domain of interest. Also, as in Clarke and Primo’s approach, we do not write down a model in order to test its veracity (though, in later chapters, we do discuss ways of justifying and evaluating models). Rather, our focus is on how we can systematically *use* causal models — in the sense of *mobilizing background knowledge of the world* — to guide our empirical strategies and inform our inferences. Grounding our empirical strategy in a model allows us, in turn, to learn about features of the model itself as we encounter the data.

## 1.2 Key contributions

This book draws on methods developed in the study of Bayesian networks, a field pioneered by scholars in computer science, statistics, and philosophy (see especially Pearl (2009)). Bayesian networks, a form of causal model,

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<sup>6</sup>There are of course many exceptions, including work that uses structural equation modeling, and research that focuses specifically on understanding heterogeneity and mediation processes.

have had limited traction to date in political science. Yet the literature on Bayesian networks and their graphical counterparts, directed acyclic graphs (DAGs), is a body of work that addresses very directly the kinds of problems with which qualitative and quantitative scholars routinely grapple.<sup>7</sup>

Drawing on this work, we show in the chapters that follow how a theory can be formalized as a causal model represented by a causal graph and a set of structural equations. Engaging in this modest degree of formalization yields enormous benefits. It allows us, for a wide range of causal questions, to specify causal questions clearly and assess what inferences to make about queries from new data.

For students engaging in process tracing, the benefits of this approach are multiple. In particular, the framework that we develop in this book provides:

- A grounding for assessing the “probative value” of evidence drawn from different parts of any causal network. The approach yields a principled and transparent approach to answering the question: how should the observation of a given piece of data affect my causal beliefs about a case?
- A transparent, replicable method of aggregating inferences from observations drawn from different locations in a causal network. Having collected multiple pieces of evidence from different parts of a causal process or case context, what should I end up believing about the causal question of interest?

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<sup>7</sup>For application to quantitative analysis strategies in political science, Glynn and Quinn (2007) give a clear introduction to how these methods can be used to motivate strategies for conditioning and adjusting for causal inference. García and Wantchekon (2015) demonstrate how these methods can be used to assess claims of external validity. With a focus on qualitative methods, Waldner (2015) uses causal diagrams to lay out a “completeness standard” for good process tracing. Weller and Barnes (2014) employ graphs to conceptualize the different possible pathways between causal and outcome variables among which qualitative researchers may want to distinguish. Generally, in discussions of qualitative methodology, graphs are used to capture core features of theoretical accounts, but are not developed specifically to ensure a representation of the kind of independence relations implied by structural causal models (notably what is called in the literature the “Markov condition”). Moreover, efforts to tie these causal graphs to probative observations, as in Waldner (2015), are generally limited to identifying steps in a causal chain that the researcher should seek to observe.

- A common approach for assessing a wide variety of queries (estimands). We can use the same apparatus to learn *simultaneously* about different case-level causal questions, such as “What caused the outcome in this case?” and “Through what pathway did this cause exert its effect?”
- Guidance for research design. Given finite resources, researchers must make choices about where to look for evidence. A causal model framework can help researchers assess, *a priori*, the relative expected informativeness of different evidentiary and case-selection strategies, conditional on how they think the world works and the question they want to answer.

The approach also offers a range of distinctive benefits to researchers seeking to engage in mixed-method inference and to learn about general causal relations, as well as about individual cases. The framework’s central payoff for multi-method research is the systematic integration of qualitative and quantitative information to answer any given causal query. We note that the form of integration that we pursue here differs from that offered in other accounts of multi-method research. In Seawright (2016)’s approach, for instance, one form of data — quantitative *or* qualitative — is always used to draw causal inferences, while the other form of data is used to test assumptions or improve measures employed in that primary inferential strategy. In the approach that we develop in this book, in contrast, we are always using *all* information available to update on causal quantities of interest. In fact, within the causal models framework, there is no fundamental difference between quantitative and qualitative data, as both enter as values of nodes in a causal graph. This formalization — this reductive move — may well discomfit some readers. And we acknowledge that our approach undeniably involves a loss of some of what makes qualitative research distinct and valuable. Yet, this translation of qualitative and quantitative observations into a common, causal model framework offers major advantages. Beyond the integration of different forms of information, these advantages include:

- Transparency. The framework makes manifest precisely how each form of evidence enters into the analysis and shapes conclusions.
- Learning across levels of analysis. In a causal model approach, we use case-level information to learn about populations and general theory.

At the same time, we use what we have learned about populations to sharpen our inferences about causal relations within individual cases.

- Cumulation of knowledge. A causal model framework provides a straightforward, principled mechanism for building on what we have already learned. As we see data, we update our model; and then our updated model can inform the inferences we draw from the next set of observations. Models can, likewise, provide an explicit framework for positing and learning about the generalizability and portability of findings across research contexts.
- Guidance for research design. With a causal model in hand, we can formally assess key multi-method design choices, including the balance we should strike between breadth (the number of cases) and depth (intensiveness of analysis in individual cases) and the choice of cases for intensive analysis.

Using causal models also has substantial implications for common methodological intuitions, advice, and practice. To touch on just a few of these implications:

- Our elaboration and application of model-based process tracing shows that, given plausible causal models, process tracing’s common focus on intervening causal chains may be much less productive than other empirical strategies, such as examining moderating conditions.
- Our examination of model-based case-selection indicates that for many common purposes there is nothing particularly especially about “on the regression line” cases or those in which the outcome occurred, and there is nothing necessarily damning about selecting on the dependent variables. Rather optimal case selection depends on factors that have to date received little attention, such as the population distribution of cases and the probative value of the available evidence.
- Our analysis of clue-selection as a decision problem shows that the probative value of a piece evidence cannot be assessed in isolation, but hinges critically on what we have already observed.

The basic analytical apparatus that we employ here is not new. Rather, we see the book’s goals as being of three kinds. First, we aim to import insights: to introduce political scientists to an approach that has received little attention in the discipline but that can be useful for addressing the sorts of causal questions with which political scientists are commonly preoccupied. As a model-based approach, it is a framework especially well suited to a field of inquiry in which exogeneity frequently cannot be assumed by design—that is, in which we often have no choice but to be engineers.

Second, we draw connections between the Bayesian networks approach and key concerns and challenges with which students in social sciences routinely grapple. Working with causal models and DAGs most naturally connects to concerns about confounding and identification that have been central to much quantitative methodological development. Yet we also show how causal models can address issues central to process tracing, such as how to select cases for examination, how to think about the probative value of causal process observations, and how to structure our search for evidence, given finite resources.

Third, we provide a set of usable tools for implementing the approach. We provide intuition and software, the **CausalQueries** package, that researchers can use to make research design choices and draw inferences from the data.

There are also important limits to this book’s contributions and aims. First, while we make use of Bayesian inference throughout, we do not engage here with fundamental debates over or critiques of Bayesianism itself. (For excellent treatments of some of the deeper issues and debates, see, for instance, Earman (1992) and Fairfield and Charman (2017).)

Second, this book does not address matters of data-collection (e.g., conducting interviews, searching for archival documents) or the construction of measures. For the most part, we assume that data are either at hand or can be gathered, and we bracket the measurement process itself. That said, a core concern of the book is using causal models to identify the *kinds* of evidence that qualitative researchers will want to collect. In Chapter 7, we show how causal models can tell us whether observing an element of a causal process is potentially informative about a causal question; and in Chapter ?? we demonstrate how we can use models to assess the likely learning that will arise from different clue-selection strategies. We also address the problem of measurement error in Chapter 9, showing an approach to using causal

models to learn about error from the data.

Finally, while we will often refer to the use of causal models for “qualitative” analysis, we do not seek to assimilate all forms of qualitative inquiry into a causal models framework. Our focus is on work that is squarely addressed to matters of causation; in particular, the logic that we elaborate is most closely connected to the method of process tracing. More generally, the formalization we introduce here—the graphical representation of beliefs and the application of mathematical operations to numerically coded observations—will surely strike some readers as reductive and not particularly “qualitative.” It is almost certainly the case that, as we formalize, we leave behind some of what makes qualitative research distinctive and valuable. Our aim in this book is not to discount the importance of those aspects of qualitative inquiry that resist formalization, but to show some of things we *can* do if we are willing to formalize.

### 1.3 The Road Ahead

The book is divided into four main parts.

In the first part of the book, we set out the basics. In Chapter 2, following a review of the common potential-outcomes approach to causality, we introduce the concept and key components of a causal model. Chapter 3 illustrates how we can represent of causal beliefs in the form causal models by translating the arguments of a several prominent works of political science into causal models. In Chapter 4, we set out a range of causal questions that researchers might want to address — including questions about case-level causal effects, population-level effects, and mechanisms — and define these queries within a causal model framework. Chapter 5 offers a primer on the key ideas in Bayesian inference that we will mobilize in later sections of the book. In Chapter 6, we map between causal models and theories, showing how we can think of any causal model as situated within a hierarchy of complexity: within this hierarchy, any causal model can be justified by references to a “lower level”, more detailed model that offers a theory of why things work the way do that the higher level. This conceptualization is crucial insofar as we use more detailed (lower-level) models to generate empirical leverage on relationships represented in simpler, higher-level models.

The second part of the book shows how we can use causal models to undertake process-tracing and mixed method inference. Chapter 7 lays out the logic of case-level inference from causal models: the central idea here is that what we learn from evidence is always conditional on the prior beliefs embedded in our model. In Chapter 8, we illustrate model-based process-tracing with an application to the substantive issue of economic inequality’s effects on democratization. Chapter 9 moves to mixed data problems: situations in which a researcher wants to use “quantitative” (broadly,  $X, Y$ ) data on a large set of cases and more detailed (“qualitative”) data on some subset of these cases. We show how we can use any arbitrary mix of observations across a sample of any size (greater than 1) to update on all causal parameters in a model, and then use the updated model to address the full range of general and case-level queries of interest. In Chapter 10, we illustrate this integrative approach by revisiting the problem of inequality and democracy introduced in Chapter 8. Finally, in Chapter 11, we take the project of integration a step further by showing how we can use models to integrate findings across *studies* and across *settings*. We show, for instance, how we can learn jointly from the data generated by an observational study and an experimental study of the same causal domain and how models can help us reason in principled ways about the transportability of findings across contexts.

The third part of the book unpacks what causal models can contribute to research design. Across Chapters ??, ??, and ?? we demonstrate how researchers can mobilize their models, as well as prior observations, to determine what kind of new evidence is likely to be most informative about the query of interest, how to strike the balance between extensiveness and intensiveness of analysis, and which cases to select for in-depth process tracing.

The fourth and final part of the book steps back to put the model-based approach into question. Until this point, we have been advocating an embrace of models to aid inference. But the dangers of doing this are demonstrably large. The key problem is that with model-based inference, the inferences are only as good as the model. In the end, while we advocate a focus on models, we know that skeptics are right to distrust them. This final part approaches this problem from two perspectives. In Chapter ??, we demonstrate the *possibility* of justifying models from external evidence, though we do not pretend that the conditions for doing so will arise commonly. In Chapter ??, drawing on common practice in Bayesian statistics, we present a set of strategies that researchers can use to evaluate and compare the validity

of models, and to investigate the degree to which findings hinge on model assumptions.

In the concluding chapter we summarize what we see as the main advantages of a causal-model-based approach to inference, draw out a set of key concerns and limitations of the framework, and identify what we see as the key avenues for progress in model-based inference.

Here we go.

# **Part I**

## **Foundations**



# Chapter 2

## Causal Models

Causal claims are everywhere. Causal knowledge is often the end goal of empirical social science. It is also a key *input* into causal inference.<sup>1</sup> Causal assumptions are also hidden in seemingly descriptive statements: claims that someone is guilty, or exploited, or powerful, or weak, involve beliefs about how things would be were conditions different. Even when scholars carefully try to avoid causal claim-making, causal verbs—depends, drives, produces, influences—tend to surface.

But while causal claims are commonplace, it is not always clear (1) what exactly is meant by a causal relation and (2) how causal knowledge about one thing can be marshaled to justify causal claims about another. For our purposes, the counterfactual view of causality addresses the first question. Causal models address the second.

### 2.1 The counterfactual model

We begin with what we might think of as a meta-model, the counterfactual model of causation. At its core, a counterfactual understanding of causation captures a simple notion of causation as “difference-making.”<sup>2</sup> In the coun-

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<sup>1</sup>As nicely put by Cartwright et al. (1994), no causes in, no causes out, a point we return to more formally later.

<sup>2</sup>The approach is sometimes attributed to David Hume, whose writing contains ideas both about causality as regularity and causality as counterfactual. On the latter, Hume’s

terfactual view, to say that  $X$  caused  $Y$  is to say: *had  $X$  been different,  $Y$  would have been* different.

The causal effect, in this view, is the difference between two things that might have happened. This means that *by definition, causal effects are not measurable quantities*. They are not differences between possible observations in the world, but, at best, differences between outcomes in the world and counterfactual outcomes. They need to be inferred not measured.

Moreover, in this view, the antecedent, “had  $X$  been different,” imagines a *controlled* change in  $X$ —an intervention that altered  $X$ ’s value—rather than a naturally arising difference in  $X$ . The counterfactual claim, then, is not that  $Y$  is different in those cases in which  $X$  is different; it is, rather, that if one could somehow have *made*  $X$  different,  $Y$  would have been different. In the terminology of Pearl (2000), we represent this quantity using a “do” operator:  $Y(\text{do}(X = x))$  is the value of  $Y$  when  $x$  is *set* to  $x$ .

Consider a simple example. Students with teacher A perform well without studying. Students with teacher B perform well if they study, and do not perform well if they do not study. Moreover, only students with teacher B in fact study. And all perform well.

When we say that one of teacher B’s students did well *because* they studied, we are comparing the outcome that they experienced to the outcome they would have experienced if they had had teacher B (as they did) but (counterfactually) had not studied. Notably, we are *not* comparing their realized outcome to the outcome they would have experienced if they had been among the people that in fact didn’t study (i.e., if they had had teacher A).

A second example. Consider the claim that Switzerland democratized ( $D = 1$ ) because it had a relatively low level of economic inequality ( $I = 0$ ) (drawing on the logic of Boix (2003)). In the counterfactual view, this is equivalent to saying that, had Switzerland *not* had a high level of equality, the country would not have democratized. High economic equality made a difference. The comparison for the causal statement is with the outcome Switzerland would have experienced under an intervention that boosted its historical level of economic inequality — *not* with how Switzerland would have performed if it had been one of the countries that *in fact* had higher levels of

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key formulation is, “if the first object had not been, the second never had existed” (Hume and Beauchamp, 2000, Section VIII). More recently, the counterfactual view has been set forth by Splawa-Neyman et al. (1990) and Lewis (1973). See also Lewis (1986).

inequality, cases that likely differ from Switzerland in other causally relevant ways.

Along with this notion of causation as difference-making, we also want to allow for *variability* in how  $X$  acts on the world.  $X$  might sometimes make a difference, for some units of interest, and sometimes not. High levels of equality might generate democratization in some countries or historical settings but not in others. Moreover, while equality might make democratization happen in some times in places, it might *prevent* that same outcome in others. We need a language to describe these different types of relations.

### 2.1.1 Potential outcomes

The “potential outcomes” framework is useful for describing the different kinds of counterfactual causal relations that might prevail between variables Rubin (1974). In this framework we characterize how a given unit responds to a causal variable by positing the outcomes that it *would* take on at different values of the causal variable.

A setting in which it is quite natural to think about potential outcomes is medical treatment. Imagine some individuals in a diseased population are observed to have received a drug ( $X = 1$ ) while others have not ( $X = 0$ ). Assume that, subsequently, a researcher observes which individuals become healthy ( $Y = 1$ ) and which do not ( $Y = 0$ ). Given the assignments of all other individuals,<sup>3</sup> we can treat each individual as belonging to one of four unobserved response “types,” defined by the outcomes that the individual *would have* if they received or did not receive treatment:<sup>4</sup>

- **adverse:** Those individuals who would get better if and only if they do not receive the treatment

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<sup>3</sup>We noted that we are conditioning on the assignments of others. If we wanted to describe outcomes as a function of the *profile* of treatments received by others we would have a more complex types space. For instance in an  $X \rightarrow Y$  model with 2 individuals we would report how  $(Y_1, Y_2)$  respond to  $(X_1, X_0)$ ; each vector can take on four values producing a type space with  $4^4$  types rather than  $2^2$ . The complex type space could be reduced back down to four types again, however, if we invoked the assumption that the treatment or non-treatment of one patient has no effect on the outcomes of other patients—an assumption known as the stable unit treatment value assumption (SUTVA).

<sup>4</sup>See Copas (1973)} for an early classification of this form. The literature on probabilistic models also refers to such strata as “canonical partitions” or “equivalence classes.”

- **beneficial:** Those who would get better if and only if they do receive the treatment
- **chronic:** Those who will remain sick whether or not they receive treatment
- **destined:** Those who will get better whether or not they receive treatment

Table 2.1 maps the four types ( $a, b, c, d$ ) onto their respective potential outcomes. In each column, we have simply written down the outcome that a patient of a given type would experience if they are not treated, and the outcome they would experience if they are treated. In each cases we are imagining *controlled* changes in treatment: the responses if treatments are changed without changes to other background conditions about the case.

Table 2.1: . Potential outcomes: What would happen to each of four possible types of case if they were or were not treated.

	Type a	Type b	Type c	Type d
	adverse effects	beneficial Effects	chronic cases	destined cases
Outcome if not treated	Healthy	Sick	Sick	Healthy
Outcome if treated	Sick	Healthy	Sick	Healthy

We highlight that, in this framework, case-level causal relations are treated as deterministic. A given case has a set of potential outcomes. Any uncertainty about outcomes enters as incomplete knowledge of a case’s “type,” not from underlying randomness in causal relations. This understanding of causality—as ontologically deterministic, but empirically imperfectly understood—is compatible with views of causation commonly employed by qualitative researchers (see, e.g., Mahoney (2008)), and with understandings of causal determinism going back at least to Laplace (1901).

As we will also see, we can readily express this kind of incompleteness of knowledge within a causal model framework: indeed, the way in which causal models manage uncertainty is central to how they allow us to pose questions of interest and to learn from evidence. There are certainly situations we could imagine in which one might want to conceptualize potential outcomes

themselves as random (for instance, if individuals in different conditions play different lotteries). But for the vast majority of the settings we consider, not much of importance is lost if we treat potential outcomes as deterministic but possibly unknown: at the end of the day something will occur or it will not occur, we just do not know which it is.

### 2.1.2 Generalization

Throughout the book, we generalize from this simple setup. Whenever we have one causal variable and one outcome, and both variables are binary (i.e., each can take on two possible values, 0 or 1), there are only four sets of possible potential outcomes, or “types.” More generally, for variable,  $Y$ , we will use  $\theta^Y$  to capture the unit’s “type”: the way that  $Y$  responds to its potential causes.<sup>5</sup> We, further, add subscripts to denote particular types. Where there are four possible types, for instance, we use the notation  $\theta_{ij}^Y$ , where  $i$  represents the case’s potential outcome when  $X = 0$  and  $j$  is the case’s potential outcome when  $X = 1$ .

Adopting this notation, for a causal structure with one binary causal variable and a binary outcome, the four types can be represented as  $\{\theta_{10}^Y, \theta_{01}^Y, \theta_{00}^Y, \theta_{11}^Y\}$ , as shown in Table 2.2:

Table 2.2: . Generalizing from Table 2.1, the table gives for each causal type the values that  $Y$  would take on if  $X$  is set at 0 and if  $X$  is set at 1.

	Type a	Type b	Type c	Type d
	$\theta^Y = \theta_{10}^Y$	$\theta^Y = \theta_{01}^Y$	$\theta^Y = \theta_{00}^Y$	$\theta^Y = \theta_{11}^Y$
Set $X = 0$	$Y(0) = 1$	$Y(0) = 0$	$Y(0) = 0$	$Y(0) = 1$
Set $X = 1$	$Y(1) = 0$	$Y(1) = 1$	$Y(1) = 0$	$Y(1) = 1$

Returning to the matter of inequality and democratization to illustrate, let  $I = 1$  represent a high level of economic equality and  $I = 0$  its absence; let

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<sup>5</sup>Later, we will refer to these as “nodal types.”

$D = 1$  represent democratization and  $D = 0$  its absence. A  $\theta_{10}^D$  (or  $a$ ) type is a case in which a high level of equality, if it occurs, *prevents* democratization in a country that would otherwise have democratized. The causal effect of high equality in a case,  $i$ , of  $\theta_{10}^D$  type is  $\tau_i = -1$ . A  $\theta_{01}^D$  type (or  $b$  type) is a case in which high equality, if it occurs, generates democratization in a country that would otherwise have remained non-democratic (effect  $\tau_i = 1$ ). A  $\theta_{00}^D$  type ( $c$  type) is a case that will not democratize regardless of the level of equality (effect  $\tau_i = 0$ ); and a  $\theta_{11}^D$  type ( $d$  type) is one that will democratize regardless of the level of equality (again, effect  $\tau_i = 0$ ).

In this setting, a causal *explanation* of a given case outcome amounts to a statement about its type. The claim that Switzerland’s high level of equality was a cause of its democratization is equivalent to saying that Switzerland democratized and is a  $\theta_{01}^D$  type. To claim that Sierra Leone democratized because of low inequality is equivalent to saying that Sierra Leone democratized and is a  $\theta_{10}^D$  type. To claim, on the other hand, that Malawi democratized for reasons having nothing to do with its level of economic equality is to characterize Malawi as a  $\theta_{11}^D$  type (which already specifies its outcome).

Now, let us consider more complex causal relations. Suppose now that there are two binary causal variables  $X_1$  and  $X_2$ . We can specify any given case’s potential outcomes for each of the different possible combinations of causal conditions—there now being four such conditions, as each causal variable may take on 0 or 1 when the other is at 0 or 1.

As for notation, we now need to expand  $\theta$ ’s subscript since we need to represent the value that  $Y$  takes on under each of the four possible combinations of  $X_1$  and  $X_2$  values. We construct the four-digit subscript with the ordering (and, in general, mapping any two parents alphabetically into  $X_1$  and  $X_2$ ). The next equation connects this notation to the “do” notation used to convey the idea that conditions are controlled.

$$Y_{hijk} \left\{ \begin{array}{l} h = Y|do(X_1 = 0, X_2 = 0) \\ i = Y|do(X_1 = 1, X_2 = 0) \\ j = Y|do(X_1 = 0, X_2 = 1) \\ k = Y|do(X_1 = 1, X_2 = 1) \end{array} \right. \quad (2.1)$$

Thus, for instance,  $\theta_{0100}^Y$  means that  $Y$  is 1 if  $X_1 = 1$  and  $X_2 = 0$  and is 0 otherwise.

We now have 16 causal types: 16 different patterns that  $Y$  might display in response to changes in  $X_1$  and  $X_2$ . The full set is represented in Table 2.3, which also makes clear how we read types off of four-digit subscripts. For instance, the table shows us that for nodal type  $\theta_{0101}^Y$ ,  $X_1$  has a positive causal effect on  $Y$  but  $X_2$  has no effect. On the other hand, for type  $\theta_{0011}^Y$ ,  $X_2$  has a positive effect while  $X_1$  has none.

We also capture interactions here. For instance, in a  $\theta_{0001}^Y$  type,  $X_2$  has a positive causal effect if and only if  $X_1$  is 1. In that type,  $X_1$  and  $X_2$  serve as “complements.” For  $\theta_{0111}^Y$ ,  $X_2$  has a positive causal effect if and only if  $X_1$  is 0. In that setup,  $X_1$  and  $X_2$  are “substitutes.”

Table 2.3: With two binary causal variables, there are 16 causal types: 16 ways in which  $Y$  might respond to changes in the other two variables.

$\theta^Y$	if $X_1 = 0, X_2 = 0$	if $X_1 = 1, X_2 = 0$	if $X_1 = 0, X_2 = 1$	if $X_1 = 1, X_2 = 1$
$\theta_{0000}^Y$	0	0	0	0
$\theta_{1000}^Y$	1	0	0	0
$\theta_{0100}^Y$	0	1	0	0
$\theta_{1100}^Y$	1	1	0	0
$\theta_{0010}^Y$	0	0	1	0
$\theta_{1010}^Y$	1	0	1	0
$\theta_{0110}^Y$	0	1	1	0
$\theta_{1110}^Y$	1	1	1	0
$\theta_{0001}^Y$	0	0	0	1
$\theta_{1001}^Y$	1	0	0	1
$\theta_{0101}^Y$	0	1	0	1
$\theta_{1101}^Y$	1	1	0	1
$\theta_{0011}^Y$	0	0	1	1
$\theta_{1011}^Y$	1	0	1	1
$\theta_{0111}^Y$	0	1	1	1
$\theta_{1111}^Y$	1	1	1	1

This is a rich framework in that it allows for all possible ways in which a set of multiple causes can interact with each other. Often, when seeking to explain the outcome in a case, researchers proceed as though causes are

necessarily *rival*, where  $X_1$  being a cause of  $Y$  implies that  $X_2$  was not. Did Malawi democratize because it was a relatively economically equal society *or* because of international pressure to do so? In the counterfactual model, however, causal relations can be non-rival. If two out of three people vote for an outcome under majority rule, for example, then both of the two supporters caused the outcome: the outcome would not have occurred if *either* supporter's vote were different. This typological, potential-outcomes conceptualization provides a straightforward way of representing this kind of complex causation.

Because of this complexity, when we say that  $X$  caused  $Y$  in a given case, we will generally mean that  $X$  was *a* cause, not *the* (only) cause. Malawi might not have democratized if *either* a relatively high level of economic equality *or* international pressure had been absent. For most social phenomena that we study, there will be multiple, and sometimes a great many, difference-makers for any given case outcome.

We will mostly use  $\theta_{ij}^Y$ -style notation in this book to refer to types. We will, however, occasionally revert to the simpler  $a, b, c, d$  designations when that helps ease exposition. As types play a central role in the causal-model framework, we recommend getting comfortable with both forms of notation before going further.

Using the same framework, we can generalize to structures in which a unit has any number of causes and also to cases in which causes and outcomes are non-binary. As one might imagine, the number of types increases rapidly (very rapidly) as the number of considered causal variables increases, as it also does as we allow  $X$  or  $Y$  to take on more than 2 possible values. For example, if there are  $n$  binary causes of an outcome, then there can be  $2^{(2^n)}$  types of this form: that is,  $k = 2^n$  combinations of values of causes to consider, and  $2^k$  distinct ways to react to each combination. If causes and outcomes are ternary instead of binary, we have  $3^{(3^n)}$  causal types of this form. Yet, the basic principle of representing possible causal relations as patterns of potential outcomes remains unchanged, at least as long as variables are discrete.

### 2.1.3 Summaries of potential outcomes

So far, we have focused on causal relations at the level of an individual case. Causal relations at the level of a population are, however, simply a summary of causal relations for cases, and the same basic ideas can be used. We could, for instance, summarize our beliefs about the relationship between economic equality and democratization by saying that we think that the world is comprised of a mixture of  $a$ ,  $b$ ,  $c$ , and  $d$  types, as defined above. We could get more specific and express a belief about what proportions of cases in the world are of each of the four types. For instance, we might believe that  $a$  types and  $d$  types are quite rare while  $b$  and  $c$  types are quite common. Moreover, our belief about the proportions of  $b$  (positive effect) and  $a$  (negative effect) cases imply a belief about equality's *average* effect on democratization as, in a binary setup, this quantity is simply the proportion of  $b$  types minus the proportion of  $a$  types. Such summaries allow us to move from discussion of the cause of a single outcome to discussions of average effects, a distinction that we take up again in Chapter 4.

## 2.2 Causal Models and Directed Acyclic Graphs

So far we have discussed how a single outcome is affected by one or more possible causes. However, these same ideas can be used to describe more complex relations between collections of variables — for example, with one variable affecting another directly as well as indirectly via its impact on some mediating variable.

Potential outcomes tables can be used to describe such complex relations. However, as causal structures become more complex—especially, as the number of variables in a domain increases—a causal model can be a powerful organizing tool. In this section, we show how causal models and their visual counterparts, directed acyclic graphs (DAGs), can represent substantive beliefs about counterfactual causal relationships in the world. The key ideas in this section can be found in many texts (see, e.g., Halpern and Pearl (2005) and Galles and Pearl (1998)), and we introduce here a set of basic principles that readers will need to keep in mind in order to follow the argumentation in this book.

As we shift to talking about networks of causal relations between variables we will also shift our language. When talking about causal networks, or causal graphs, we will generally refer to variables as “nodes.” And we will sometimes use familial terms to describe relations between nodes. For instance, two nodes directly connected by an arrow are known as “parent” and “child,” while two nodes with a child in common (both directly affecting the same variable) are “spouses.” We can also say that  $I$  is an “ancestor” of  $D$  (a node upstream from  $D$ ’s parent) and conversely that  $D$  is a descendant of  $I$  (a node downstream from  $I$ ’s child).

Return to our running democratization example, but suppose now that we have more fully specified beliefs about how the level of economic inequality can have an effect on whether a country democratizes. We might believe that inequality affects the likelihood of democratization by generating demands for redistribution, which in turn can cause the mobilization of lower-income citizens, which in turn can cause democratization. We might also believe that mobilization itself is not just a function of redistributive preferences but also of the degree of ethnic homogeneity, which shapes the capacities of lower-income citizens for collective action. In this model  $R$  is a parent of  $M$ ,  $I$  is an ancestor of  $M$  but not a parent.  $R$  and  $E$  are spouses and  $M$  is their child. We can visualize this model as a Directed Acyclic Diagram (DAG) in Figure 2.1.

Fundamentally, we treat causal models in this book as formal representations of *beliefs* about how the world works—or, more specifically, about causal relations within a given domain. We might use a causal model to capture our own beliefs, a working simplification of our beliefs, or a set of potential beliefs that one might hold. The formalization of *prior* beliefs in the form of a causal model is the starting point for research design and inference in this book’s analytic framework. Using the democratization example, we will now walk through the three components of a causal model in which our beliefs get embedded: nodes, functions, and distributions.

### 2.2.1 Components of a Causal Model

The three components of a causal model are (i) the nodes—that is, the set of variables we are focused on and how are they defined (ii) the functional relations—which nodes are “explained” by which other nodes and how, and

A Model of Inequality's Effect on Democratization

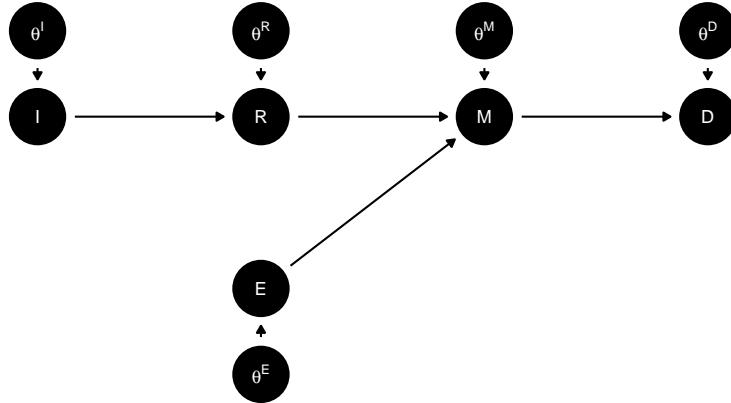


Figure 2.1: A simple causal model in which high inequality ( $I$ ) affects the democratization ( $D$ ) via redistributive demands and mass mobilization ( $M$ ), which is also a function of ethnic homogeneity ( $E$ ). The arrows show relations of causal dependence between variables. The graph does not capture the ranges of the variables and the functional relations between them.

(iii) probability distributions over unexplained elements of a model.

### 2.2.1.1 The nodes

The first component of a causal model is the set of variables (nodes) across which the model characterizes causal relations. On the graph in Figure 2.1, the five included variables are represented by the five lettered nodes. (In addition we mark  $\theta^D$  on the graph though, as will be made clear, we will not think of this as a variable.)

In identifying the nodes, we also need to specify the *ranges* over which they can vary. We might specify, for instance, that all nodes in the model are binary, taking on the values 0 or 1. We could, alternatively, define a set of categories across which a node ranges or allow a node to take on any real number value or any value between a set of bounds.<sup>6</sup>

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<sup>6</sup>If we let  $\mathcal{R}$  denote a set of ranges for all nodes in the model, we can indicate  $X$ 's range, for instance, by writing  $\mathcal{R}(X) = \{0, 1\}$ . The nodes in a causal model together with their ranges—the triple  $(\mathcal{U}, \mathcal{V}, \mathcal{R})$ —are sometimes called a *signature*,  $\mathcal{S}$ .

Notice that some of these nodes have arrows pointing *into* them:  $R$ ,  $M$ , and  $D$  are endogenous nodes, meaning that their values are determined entirely by other nodes in the model.

Other nodes have arrows pointing *out* of them but no arrows pointing into them:  $I$  and  $E$ .  $I$  and  $E$  are “exogenous” nodes; they influence other nodes in the model but themselves have no causes specified in the model.

The  $\theta$  terms require a little more explanation since they do not describe substantive nodes. In our discussion above, we introduced  $\theta$  notation for representing types. Generally we can think of  $\theta$  terms on a causal graph as unobservable and unspecified features of a causal domain that affect outcomes. These might include random processes (noise) or contextual features that we are unable to identify or do not understand. We imagine them pointing into every node, whether indicated or not. In all cases the work they do is to characterize what the value of a node will be, given the value of its parents.

We note that our notation for representing these unobservable, unspecified influences differs from that commonly found in the literature on causal models. In many treatments, these components are themselves referred to as “exogenous” variables, and often labelled as sets  $\mathcal{U}$ , to be distinguished from the endogenous—named—variables often labelled as  $\mathcal{V}$ . We will generally use  $\theta$  to denote these unobserved, unspecified influences to emphasize their particular role, as direct objects of interest in causal inquiry. As we will show, we can think of  $\theta$  nodes as capturing the functional relations between endogenous variables and as being quantities of direct interest for causal inquiry. We more fully develop this point — returning to the notion of  $\theta$  terms as receptacles for causal effects — below.

### 2.2.1.2 The functions

Next, we need to specify our beliefs about the causal relations among the nodes in our model. How is the value of one node affected by, and how does it affect, the values of others? For each endogenous node—each node influenced by others in the model—we need to express beliefs about how its value is affected by its parents, its immediate causes.

The DAG already represents a critical part of these beliefs: the arrows, or directed edges, tell us *which nodes we believe to be direct causal inputs*

into other nodes. So, for instance, we believe that democratization ( $D$ ) is determined jointly by mobilization ( $M$ ) and some exogenous, unspecified factor (or set of factors),  $\theta^D$ . As we have said, we can think of  $\theta^D$  as all of the other influences on democratization, besides mobilization, that we either do not know of or have decided not to explicitly include in the model. We believe, likewise, that  $M$  is determined by  $I$  and an unspecified exogenous factor (or set of factors),  $\theta^M$ . And we are conceptualizing inequality ( $I$ ) and ethnic heterogeneity ( $E$ ) as shaped solely by factors exogenous to the model, captured by  $\theta^I$  and  $\theta^E$ , respectively.

Beyond the beliefs captured by the arrows in a DAG, we can express more specific beliefs about causal relations in the form of a causal function.<sup>7</sup> Specifying a function means writing down whatever general or theoretical knowledge we have about the direct causal relations between nodes. A function specifies how the value that one node takes on is determined by the values that other nodes—its parents—take on.

We can specify this relationship in a vast variety of ways. It is useful however to distinguish broadly between parametric and non-parametric approaches. We take a non-parametric approach in this book, but it is helpful to juxtapose that approach to a parametric one.

**Parametric approaches.** A parametric approach specifies a functional form that relates parents to children. For instance, we might model one node as a linear function of another and write  $D = \alpha + \beta M$ , where  $\beta$  is a parameter that we do not know the value of at the outset of a study but about which we wish to learn. If we believe  $D$  to be linearly affected by  $M$  but also subject to forces that we do not yet understand and have not yet specified in our theory, then we might write:  $D = \alpha + \beta M + \theta^D$ . In this function,  $\alpha, \beta$  might be the parameters of interest, with  $\theta^D$  treated merely as a random disturbance. We can be still more agnostic by, for example, including parameters that govern how other parameters operate. Consider, for instance the function,  $D = \beta M^{\theta^D}$ . Here,  $D$  and  $M$  are linearly related if  $\theta^D = 1$ , but not otherwise.

Note that functions can be written to be quite specific or extremely general, depending on the state of prior knowledge about the phenomenon under investigation. The use of a structural model *does not require precise knowledge*

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<sup>7</sup>The collection of all causal functions in the model can be denoted as  $\mathcal{F}$ .

of specific causal relations, even of the functional forms through which two nodes are related.

**The non-parametric approach.** With discrete data, causal functions can take fully *non-parametric* form, allowing for *any possible relation* between parents and children. We use this framework for most of this book and thus spend some time developing the approach here.

We begin by returning to the concept of types. Drawing on our original four types from earlier in this chapter, we know that we can fully specify causal relations between a binary  $M$  and a binary  $D$  by allowing the node  $\theta_D$  to range across four possible values  $\{\theta_{10}^D, \theta_{01}^D, \theta_{00}^D, \theta_{11}^D\}$ . For instance,  $\theta_{10}^D$  represents a negative causal effect of  $M$  on  $D$  while  $\theta_{00}^D$  represents  $D$  remaining at 0 regardless of  $M$ . Put differently,  $\theta^D$  represents the non-parametric function that relates  $M$  to  $D$ . We can formally specify  $D$ 's behavior as a function of  $M$  and  $\theta^D$  in the following way:

$$D(M, \theta_{ij}^D) = \begin{cases} i & \text{if } M = 0 \\ j & \text{if } M = 1 \end{cases}$$

Note that  $\theta^D$  ranges over *all possible* functional forms between these two binary variables.

How should we think about what kind of *thing*  $\theta^D$  is, in a more substantive sense? It is probably most helpful to think of  $\theta^D$  as an unknown and possibly random factor that conditions the effect of mobilization on democratization, determining whether  $M$  has a negative effect, a positive effect, no effect with democratization never occurring, or no effect with democratization bound to occur regardless of mobilization. Importantly, however, while we might think of  $\theta^D$  as an unknown or random quantity, in this formulation  $\theta^D$  should not be thought of as a nuisance — as “noise” that we would like to get rid of — but as *the quantity that we want to learn about*: we want to know whether  $M$  likely had a positive, negative, or no effect on  $D$ . We elaborate on this point at much greater length in Chapter 4.

We can similarly use  $\theta$  terms to capture causal relations involving any number of parent nodes. Every substantively defined node,  $J$ , in a graph can be thought of as having a  $\theta^J$  term pointing into it, and the (unobservable) value of  $\theta^J$  represents the mapping from  $J$ 's parents (if it has any) to the value of  $J$ .

Applied to the binary nodes in Figure 2.1,  $\theta^J$  ranges as follows:

- **Nodes with no parents:** For an exogenous node, like  $I$  or  $E$ ,  $\theta^J$  represents an external “assignment” process can take on one of two values,  $\theta_0^J$ , meaning that  $J$  is “assigned” to 0 or  $\theta_1^J$ , meaning that  $J$  is assigned to 1. For instance,  $\theta_0^I$  typifies a case in which exogenous forces have generated low inequality.
- **Binary nodes with 1 binary parent:** For endogenous node  $R$ , with only one parent ( $I$ ),  $\theta^R$  takes on one of four values of the form  $\theta_{ij}^R$  (our four original types,  $\theta_{10}^R$ ,  $\theta_{01}^R$ , etc.).
- **Binary nodes with 2 binary parents:**  $M$  has two parent nodes. Thus,  $\theta^M$  will take on a possible 16 values of the form  $\theta_{hijk}^M$  ( $\theta_{0000}^M$ ,  $\theta_{0001}^M$ , etc.), using the syntax detailed earlier in this chapter.

**Nodal types and causal types.** For analytic applications later in the book, we will want to be able to think both about the type operating at a particular *node* and about *collections* of types operating across a model. We thus refer to  $\theta^J$  as a unit’s *nodal causal type*, or simply *nodal type*, for  $J$ . We refer to the collection of nodal types across all nodes for a given unit (i.e., a case) as the case’s *unit causal type*, or simply *causal type*, denoted by the vector  $\theta$ .

If we hypothetically knew a unit’s causal type—all nodal types for all nodes—then we would know everything there is to know about that unit. Since the nodal types of exogenous nodes include *values* of all exogenous nodes, and the nodal types of all endogenous nodes specify how those nodes respond to all of their parents, a unit’s causal type fully specifies all nodal values as well as all *counterfactual* nodal values for a unit.

We will sometimes refer to the values of  $\theta$  as a unit’s *context*. This is because  $\theta$  captures all exogenous forces acting on a unit. This includes the assignment process driving the model’s exogenous nodes (in our example,  $\theta^I$  and  $\theta^E$ ) as well as all contextual factors that shape causal relations between nodes ( $\theta^R$ ,  $\theta^M$ , and  $\theta^D$ ). Put differently,  $\theta$  captures both how a unit reacts to situations and which situations it is reacting to. One implication is that there is no *formal* distinction between a unit’s type and a unit’s situation—between, say, a hungry person, and a person who has had no food.

Nodal types, causal types

term	symbol	meaning
nodal type	$\theta^J$	The way that node $J$ responds to the values of its parents. Example: $\theta_{10}^Y$ : $Y$ takes the value 1 if $X = 0$ and 0 if $X = 1$ .
causal type	$\theta$	A causal type is a concatenation of nodal types, one for each node. Example: $(\theta_0^X, \theta_{00}^Y)$ , is a causal type that has $X = 0$ and that has $Y = 0$ no matter what the value of $X$ .

A few important aspects of causal functions are worth highlighting.

1. These functions express *causal* beliefs. When we write  $D = \beta M$  as a function, we do not just mean that we believe the values of  $M$  and  $D$  in the world to be linearly related. We mean that we believe that the value of  $M$  *determines* the value of  $D$  through this linear function. Functions are, in this sense, *directional* statements, with causes on the righthand side and an outcome on the left.
2. The collection of simple functions that map from the values of parents of a given node to the values of that node are sufficient to represent potentially complex webs of causal relations. For each node, we do not need to think through entire sequences of causation that might precede it. We need only specify how we believe it to be affected by its parents—that is to say, those nodes pointing directly into it. Our outcome of interest,  $D$ , may be shaped by multiple, long chains of causality. To theorize how  $D$  is generated, however, we write down how we believe  $D$  is shaped by its parent—its direct cause,  $M$ . We then, separately, express a belief about how  $M$  is shaped by *its* parents,  $R$  and  $E$ . A node’s function must include as inputs all, and only, those nodes that point directly into that node.<sup>8</sup>
3. As in the general potential-outcomes framework, all relations in a causal model are conceptualized as deterministic at the case level. Yet,

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<sup>8</sup>The set of a node’s parents is required to be minimal in the sense that a node is not included among the parents if, given the other parents, the child does not depend on it in any state that arises with positive probability.

there is not as much at stake here as one might think at first; by this we simply mean that a node's value is *determined* by the values of its parents *along with* any stochastic or unknown components. We express uncertainty about causal relations, however, as unknown parameters, such as the causal types  $\theta$ .

### 2.2.1.3 The distributions

Putting causal structure and causal functions together gives us a *structural causal model*. In a structural causal model, all endogenous nodes are, either directly or by implication, functions of a case's context (the values of the set of exogenous nodes).<sup>9</sup> What we have not yet inscribed into the model, however, is beliefs about how *likely* or *common* different kinds of contexts might be.

Thus, for instance, a structural causal model consistent with Figure 2.1 stipulates which nodes may have effects on which other nodes. But it says nothing in itself about the distribution of values of either the exogenous nodes or of the causal relations between nodes.<sup>10</sup> We have not said anything, for instance, about how common high inequality is across the relevant domain of cases or how common ethnic homogeneity is. Put differently, we have said nothing about the *distribution* of  $\theta^I$  or of  $\theta^E$ . Similarly, we have said nothing yet about how commonly mobilization has positive, negative, or null effects of democratization—that is, the distribution of  $\theta^D$ —or about how commonly  $I$  and  $E$  have different possible joint causal effects on  $M$  (the distribution of  $\theta^M$ ).

In many research situations, we will have or want to posit a set of prior beliefs about how the world works under different conditions and about what kinds of conditions are more likely than others. We can express these beliefs about

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<sup>9</sup>More formally, a **structural causal model** over signature  $\mathcal{S} = <\mathcal{U}, \mathcal{V}, \mathcal{R}>$  is a pair  $<\mathcal{S}, \mathcal{F}>$ , where  $\mathcal{F}$  is a set of ordered structural equations containing a function  $f_i$  for each element  $Y \in \mathcal{V}$ . We say that  $\mathcal{F}$  is a set of ordered structural equations if no node is its own descendant and if no element in  $\mathcal{U}$  is parent to more than one element of  $\mathcal{V}$ . This last condition can be achieved by shifting any parent of multiple children in  $\mathcal{U}$  to  $\mathcal{V}$ . This definition thus includes an assumption of acyclicity, which is not found in all definitions in the literature.

<sup>10</sup>Thus  $P(d|i, e, u_D)$  would be defined by this structural model (as a degenerate distribution), but  $P(i)$ ,  $P(e)$ ,  $P(u_D)$ , and  $P(i, e, u_D)$  would not be.

context as probability distributions over the model’s  $\theta^J$  terms. For instance, our structural causal model might tell us that  $E$  and  $R$  can jointly affect  $M$ . We might, then, add to this a belief about  $\theta^M$  such that, in the population of interest, redistribution rarely has a positive effect on mobilization when ethnic homogeneity is low. We would thus be putting a low probability on the nodal types for  $M$  in which  $R$  has a positive effect on  $M$  when  $E = 0$ , relative to  $M$ ’s other nodal types.<sup>11</sup>

We might add to this the belief that  $E = 1$  in only 10% of cases in the population of interest, thus setting a 0.1 probability on  $\theta_1^E$ . Note that these two beliefs jointly imply that  $R$  will rarely have a positive effect on  $M$ .

As with functions, we can also (and typically would) build uncertainty into our beliefs about the shares of different nodal types in the population. We do this by specifying a *distribution* over possible “share” allocations.<sup>12</sup> For instance, we can specify a distribution over the shares of cases with ethnic homogeneity ( $\theta_1^E$ ), and a distribution over the shares of  $\theta^M$  types, with our degrees of uncertainty captured by each distribution’s variance. (More on these distributions in Chapter 5.)

In the default setup, we assume that each  $\theta$  term ( $\theta^I, \theta^E, \theta^R$ , etc.) is generated independently of the others.

While this is not without loss of generality, it is not as constraining as it might at first appear: any graph in which two  $\theta$  terms are *not* independent can be replaced by a graph in which these two terms are themselves generated by a common, third  $\theta$  term.<sup>13</sup> This independence feature is critical for being able to read off relations of conditional independence from a graph (see Box below). If it cannot be defended then the graph needs to be modified to communicate that  $\theta$ s are not independent, typically using two headed arrows. More on this in section 2.3.1.

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<sup>11</sup>Ordering the parent nodes alphabetically, the types we would be setting to a low probability would be  $\theta_{0010}^M, \theta_{0110}^M, \theta_{0111}^M, \theta_{0011}^M$ .

<sup>12</sup>More strictly our uncertainty is over probabilities. However it is sometimes more intuitive to describe uncertainty over shares. The distinction is not important for the applications later in which we typically assume units are independently drawn from a large population.

<sup>13</sup>Operationally, in the **CausalQueries** package, we can specify nodal types as having joint distributions.

Once we introduce beliefs about the distribution of values of the exogenous terms (in our setup, the  $\theta$  terms) in a model, we have specified a *probabilistic causal model*. We need not say much more, for the moment, about the probabilistic components of causal models. But to foreshadow the argument to come, our prior beliefs about the likelihoods of different contexts play a central role in the framework that we develop here. We will see how the encoding of contextual knowledge—beliefs that some kinds of conditions and causal effects are more common than others—forms a key foundation for causal inference. At the same time, our expressions of *uncertainty* about context represent scope for learning: it is the very things that we are, at a study’s outset, uncertain about that we can update our beliefs about as we encounter evidence.

## 2.3 Graphing models and using graphs

While we have been speaking to causal graphs throughout this chapter, we want to take some time to unpack their core features and uses. A key benefit of causal models is that they lend themselves to graphical representations; in turn, graphs constructed according to particular rules can aid causal analysis. In the next subsection we discuss a set of rules for representing a model in graphical form. The following subsection then demonstrates how access to a graph facilitates causal inference.

### 2.3.1 Rules for graphing causal models

The diagram in Figure 2.1 is a causal DAG (Hernán and Robins, 2006). We endow it with the interpretation that an arrow from a parent to a child means that a change in the parent can, under some circumstances, induce a change in the child. Though we have already been making use of this causal graph to help us visualize elements of a causal model, we now explicitly point out a number of general features of causal graphs as we will be using them throughout this book. Causal graphs have their own distinctive “grammar,” a set of rules that give them important analytic features.

**Directed, acyclic.** A causal graph represents elements of a causal model as a set of nodes (or vertices), representing nodes, connected by a collection

of single-headed arrows (or directed edges). We draw an arrow from node  $A$  to node  $B$  if and only if we believe that  $A$  can have a direct effect on  $B$ . The resulting diagram is a *directed acyclic* graph (DAG) if there are no paths along directed edges that lead from any node back to itself—i.e., if the graph contains no causal cycles. The absence of cycles (or “feedback loops”) is less constraining than it might appear at first. In particular if one thinks that  $A$  today causes  $B$  tomorrow which in turn causes  $A$  today, we can represent this as  $A_1 \rightarrow B \rightarrow A_2$  rather than  $A \leftrightarrow B$ . That is, we timestamp the nodes, turning what might informally appear as feedback into a non-cyclical chain.

**Meaning of missing arrows.** The *absence* of an arrow between  $A$  and  $B$  means that  $A$  is not a direct cause of  $B$ .<sup>14</sup> Here lies an important asymmetry: drawing an  $A \rightarrow B$  arrow does not mean that we know that  $A$  *does* directly cause  $B$ ; but omitting such an arrow implies that we know that  $A$  *does not* directly cause  $B$ . We say more, in other words, with the arrows we omit than with the arrows that we include.

Returning to Figure 2.1, we have here expressed the belief that redistributive preferences exert no direct effect on democratization; we have done so by *not* drawing an arrow directly from  $R$  to  $D$ . In the context of this model, saying that redistributive preferences have no direct effect on democratization is to say that any effect of redistributive preferences on democratization *must* run through mobilization; there is no other pathway through which such an effect can operate. This might be a way of encoding the knowledge that mass preferences for redistribution cannot induce autocratic elites to liberalize the regime absent collective action in pursuit of those preferences.

The same goes for the effects of  $I$  on  $M$ ,  $I$  on  $D$ , and  $E$  on  $D$ : the graph in Figure 2.1 implies that we believe that these effects also do not operate directly, but only along the indicated, mediated paths.

**Sometimes-causes.** The existence of an arrow from  $A$  to  $B$  does not imply that  $A$  always has a direct effect on  $B$ . Consider, for instance, the arrow running from  $R$  to  $M$ . The existence of this arrow requires that  $R$  appears somewhere in  $M$ ’s functional equation, as a node’s functional equation must include all nodes pointing directly into it. Imagine, though, that  $M$ ’s causal function were specified as:  $M = RE$ . This function would allow for the

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<sup>14</sup>By “direct” we mean that the  $A$  is a parent of  $B$ : i.e., the effect of  $A$  on  $B$  is not fully mediated by one or more other nodes in the model.

*possibility* that  $R$  affects  $M$ , as it will whenever  $E = 1$ . However, it would also allow that  $R$  will have no effect, as it will when  $E = 0$ .

**No excluded common causes.** Any cause common to multiple nodes on a graph must itself be represented on the graph. If  $A$  and  $B$  on a graph are both affected by some third node,  $C$ , then we must represent this common cause. Thus, for instance, the graph in Figure 2.1 implies that  $I$  and  $E$  have no common cause. If in fact we believed that a country's level of inequality and its ethnic composition were both shaped by, say, its colonial heritage, then this DAG would *not* be an accurate representation of our beliefs about the world. To make it accurate, we would need to add to the graph a node capturing that colonial heritage and include arrows running from colonial heritage to both  $I$  and  $E$ .

This rule ensures that the graph captures all potential correlations among nodes that are implied by our beliefs. If  $I$  and  $E$  are in fact driven by some common cause, then this means not just that these two nodes will be correlated but also that each will be correlated with any consequences of the other. For instance, a common cause of  $I$  and  $E$  would also imply a correlation between  $R$  and  $E$ .  $R$  and  $E$  are implied to be independent in the current graph but would be implied to be correlated if a common node pointed into both  $I$  and  $E$ .

Of particular interest in Figure 2.1 is the implied independence of  $\theta^J$ 's from one another. Imagine, for instance, that the distribution of  $\theta^D$  were different if  $I = 0$  or  $I = 0$ . This would represent a classic form of confounding: the assignment of cases to values on the explanatory node would be correlated with the case's potential outcomes on  $D$ . The omission of any such pathway is precisely equivalent to expressing the belief that  $I$  is exogenous, i.e., (as if) randomly assigned.

**Representing unobserved confounding.** It may be however that there are common causes for nodes that we simply do not understand. We might believe that some unknown factor (partially) determines both  $I$  and  $D$ , which is the same as saying that  $\theta^I$  and  $\theta^D$  are not independently distributed. If we were to represent the  $\theta$  terms on the graph we might then want to represent a single term  $(\theta^I, \theta^D)$  that points into both  $I$  and  $D$ . Usually however the  $\theta$  terms are omitted from graphs and in this case we would represent the unobserved confounding by adding a dotted line, or a two headed arrow, connecting nodes whose unknown components are not independent. Figure

2.2 illustrates. We address this kind of unobserved confounding later in the book and show how we can seek to learn about the joint distribution of nodal types in such situations.

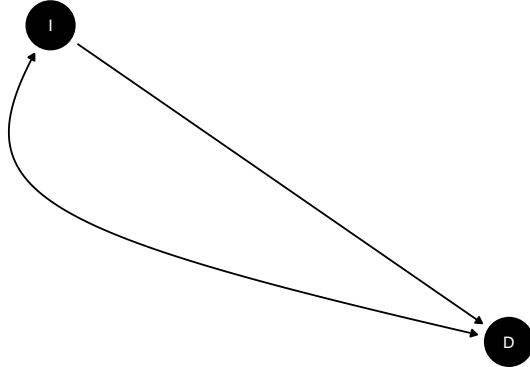


Figure 2.2: A DAG with unobserved confounding

**Licence to exclude nodes.** The flip side of the “no excluded common causes” rule is that a causal graph, to do the work it must do, does not need to include everything we know about a substantive domain of interest. We may know quite a lot about the causes of economic inequality, for example. But we can safely omit any other factor from the graph as long as *it does not affect multiple nodes in the model*. Indeed,  $\theta^I$  in Figure 2.1 already implicitly captures all factors that affect  $I$ , just as  $\theta^D$  captures all factors *other than* mobilization that affect democratization. We may be aware of a vast range of forces shaping whether countries democratize, but choose to bracket them for the purposes of an examination of the role of economic inequality. This bracketing is permissible as long as none of these unspecified factors also act on other nodes included in the model.

**We can't read functional equations from a graph.** As should be clear, a DAG does not represent all features of a causal model. What it does record is which nodes enter into the structural equation for every other node: what can directly cause what. But the DAG contains no other information about the form of those causal relations. Thus, for instance, the DAG in Figure 2.1 tells us that  $M$  is function of both  $R$  and  $E$ , but it does not tell us whether that joint effect is additive ( $R$  and  $E$  separately increase mobilization) or interactive (the effect of each depends on the value of the other), or whether

either effect is linear, concave, or something else. This lack of information about functional forms often puzzles those encountering causal graphs for the first time: surely it would be convenient to visually differentiate, say, additive from interactive effects. As one thinks about the variety of possible causal functions, however, it quickly becomes clear that there would be no simple visual way of capturing all possible functional relations. Moreover, causal graphs do not require functional statements to perform their main analytic purpose—a purpose to which we now turn.

### 2.3.2 Conditional independence from DAGs

If we encode our prior knowledge using the grammar of a causal graph, we can put that knowledge to work for us in powerful ways. In particular, the rules of DAG-construction allow for an easy reading of the *conditional independencies* implied by our beliefs. (For another, somewhat more extended treatment of the ideas in this section, see Rohrer (2018).)

To begin thinking about conditional independence, it can be helpful to conceptualize dependencies between nodes as generating *flows of information*. Let us first consider a simple relationship of dependence. Returning to Figure 2.1, the arrow running from  $I$  to  $R$ , implying a direct causal dependency, means that we expect  $I$  and  $R$  to be correlated. Put differently, observing the value of one of these nodes also gives us information about the value of the other. If we measured redistributive preferences, the graph implies that we would also be in a better position to infer the level of inequality, and vice versa. Likewise,  $I$  and  $M$  are also linked in a relationship of dependence: since inequality can affect mobilization (through  $R$ ), knowing the the level of inequality would allow us to improve our estimate of the level of mobilization and vice versa.

In contrast, consider  $I$  and  $E$ , which are in this graph indicated as being *independent* of one another. Learning the level of inequality, according to this graph, would give us no information whatsoever about the degree of ethnic homogeneity, and vice-versa.

Moreover, sometimes what we learn depends on *what we already know*. Suppose that we already knew the level of redistributive preferences. Would we then be in a position to learn about the level of inequality by observing the level of mobilization? According to this graph we would not: since the causal

link—and, hence, flow of information between  $I$  and  $M$ —runs through  $R$ , and we already know  $R$ , there is nothing left to be learned about  $I$  by also observing  $M$ . Anything we could have learned about inequality by observing mobilization is already captured by the level of redistributive preferences, which we have already seen. In other words, if we were not to include  $R$  in the causal model, then  $I$  and  $M$  would be dependent and informative about each other. When we do include  $R$  in the causal graph,  $I$  and  $M$  are independent of one another and, hence, uninformative about each other. We can express this idea by saying that  $I$  and  $M$  are *conditionally independent given  $R$* .

We say that two nodes,  $A$  and  $C$ , are “conditionally independent” given a set of nodes  $\mathcal{B}$  if, once we have knowledge of the values in  $\mathcal{B}$ , knowledge of  $A$  provides no information about  $C$  and vice-versa. Taking  $\mathcal{B}$  into account thus “breaks” any relationship that might exist unconditionally between  $A$  and  $C$ .

To take up another example, suppose that war is a cause of both military casualties and price inflation, as depicted in Figure 2.3. Casualties and inflation will then be (unconditionally) correlated with one another because of their shared cause. If we learn that there have been military casualties, this information will lead us to think it more likely that there is also war and, in turn, price inflation (and vice versa). However, assuming that war is their only common cause, we would say that military casualties and price inflation are *conditionally independent given war*. If we already know that there is war, then we can learn nothing further about the level of casualties (price inflation) by learning about price inflation (casualties). We can think of war, when observed, as blocking the flow of information between its two consequences; everything we would learn about inflation from casualties is already contained in the observation that there is war. Put differently, if we were just to look at cases where war is present (i.e., if we hold war constant), we should find no correlation between military casualties and price inflation; likewise, for cases in which war is absent.

Relations of conditional independence are central to the strategy of statistical control, or covariate adjustment, in correlation-based forms of causal inference, such as regression. In a regression framework, identifying the causal effect of an explanatory node,  $X$ , on a dependent node,  $Y$ , requires the assumption that  $X$ ’s value is conditionally independent of  $Y$ ’s potential out-

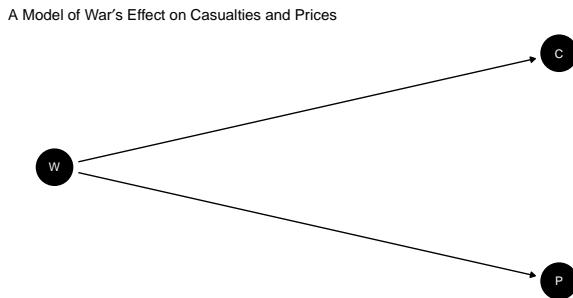


Figure 2.3: This graph represents a simple causal model in which war ( $W$ ) affects both military casualties ( $C$ ) and price inflation ( $P$ ).

comes (over values of  $X$ ) given the model’s covariates. To draw a causal inference from a regression coefficient, in other words, we have to believe that including the covariates in the model “breaks” any biasing correlation between the value of the causal node and its unit-level effect.

As we will explore, however, relations of conditional independence are also of more general interest in that they tell us, given a model, *when information about one feature of the world may be informative about another feature of the world, given what we already know*. By identifying the possibilities for learning, relations of conditional independence can thus guide research design. We discuss these research-design implications in Chapter 7, but focus here on showing how relations of conditional independence operate on a DAG.

To see more systematically how a DAG can reveal conditional independencies, it is useful spell out three elemental structures according to which which information can flow across a causal graph:

- (1a) Information can flow unconditionally along a path of arrows pointing in the same direction. In Panel 1 of Figure 2.4, information flows across all three nodes. Learning about any one will tell us something about the other two.
- (1b) Learning the value of a node along a path of arrows pointing in the same direction *blocks* flows of information across that node. Knowing the value of  $B$  in Panel 1 renders  $A$  no longer informative about  $C$ , and vice versa: anything that  $A$  might tell us about  $C$  is already captured by the information about  $B$ .

(a) A path of arrows pointing in the same direction



(b) A forked path



(c) An inverted fork (collision)



Figure 2.4: Three elemental relations of conditional independence.

(2a) Information can flow unconditionally across the branches of any forked path. In Panel 2 learning only  $A$  can provide information about  $C$  and vice-versa.

(2b) Learning the value of the node at the forking point blocks *flows* of information across the branches of a forked path. In Panel 2, learning  $A$  provides no information about  $C$  if we already know the value of  $B$ .<sup>15</sup>

(3a) When two or more arrowheads collide, generating an inverted fork, there is no unconditional flow of information between the incoming sequences of arrows. In Panel 3, learning only  $A$  provides no information about  $C$ , and vice-versa.

(3b) Collisions can be sites of *conditional* flows of information. In the jargon of causal graphs,  $B$  in Panel 3 is a “collider” for  $A$  and  $C$ .<sup>16</sup> Although information does not flow unconditionally across colliding sequences, it does flow across them *conditional* on knowing the value of the collider node or any of its downstream consequences. In Panel 3, learning  $A$  *does* provide new information about  $C$ , and vice-versa, *if* we also know the value of  $B$  (or, in principle, the value of anything that  $B$  causes).

<sup>15</sup>Readers may recognize this statement as the logic of adjusting for a confound that is a cause of both an explanatory node and a dependent node in order to achieve conditional independence.

<sup>16</sup>In the familial language of causal models, a collider is a child of two or more parents.

The last point is somewhat counter-intuitive and warrants further discussion. It is easy enough to see that, for two nodes that are correlated unconditionally, that correlation can be “broken” by controlling for a third node. In the case of collision, two nodes that are *not* correlated when taken by themselves *become* correlated when we condition on (i.e., learn the value of) a third node, the collider. The reason is in fact quite straightforward once one sees it: if an outcome is a joint function of two inputs, then if we know the outcome, information about one of the inputs can provide information about the other input. For example, if I know that you have brown eyes, then learning that your mother has blue eyes makes me more confident that your father has brown eyes.

Looking back at our democratization DAG in Figure 2.1,  $M$  is a collider for  $R$  and  $E$ , its two inputs. Suppose that we again have the functional equation  $M = RE$ . Knowing about redistributive preferences alone provides no information whatsoever about ethnic homogeneity since the two are determined independently of one another. On the other hand, imagine that we already know that there was no mobilization. Now, if we observe that there *were* redistributive preferences, we can figure out the level of ethnic homogeneity: it must be 0. (And likewise in going from homogeneity to preferences.)

Using these basic principles, conditional independencies can be read off of any DAG. We do so by checking every path connecting two nodes of interest and ask whether, along those paths, the flow of information is open or blocked, given any other nodes whose values are already observed. Conditional independence is established when *all* paths are blocked given what we already know; otherwise, conditional independence is absent.

Following Pearl (2000), we will sometimes refer to relations of conditional independence using the concept of *d-separation*. We say that variable set  $\mathcal{C}$  *d*–separates variable set  $\mathcal{A}$  from variable set  $\mathcal{B}$  if  $\mathcal{A}$  and  $\mathcal{B}$  are conditionally independent given  $\mathcal{C}$ . We say that  $\mathcal{A}$  and  $\mathcal{B}$  are *d*–connected given  $\mathcal{C}$  if  $\mathcal{A}$  and  $\mathcal{B}$  are *not* conditionally independent given  $\mathcal{C}$ .

### 2.3.3 Simplifying models

It is very easy to write down a model that is too complex to use effectively. In such cases we often seek simpler models that are consistent with models we have in mind but contain fewer nodes or more limited variation. In general

this is possible but caution has to be taken to ensure that simplified models are indeed consistent with the original model.

Fortunately the mapping between graphs and relations of conditional independence give guidance for determining when and how it is possible to simplify models. We focus discussion on simplifications that involve node elimination and conditioning on nodes.

### 2.3.3.1 Eliminating nodes

If we want to eliminate a node the key rule is that the new model (and graph) must take into account:

- (a) all *dependencies* among remaining nodes and
- (b) all *variation* generated by the eliminated node.

We can work out what this means, separately, for eliminating *endogenous* nodes and for eliminating *exogenous* nodes.

#### *Eliminating endogenous nodes*

Eliminating an endogenous node means removing a node with parents (direct causes) represented on the graph. If the node also has one or more children, then the node captures a dependency: it links its parents to its children. When we eliminate this node, preserving these dependencies requires that all of the eliminated node's parents adopt—become parents of—all of the eliminated node's children. Thus, for instance if we had a model in which  $A \rightarrow M \leftarrow B$  and  $M \rightarrow Y$ , if we were to eliminate  $M$ ,  $M$ 's parents ( $A$  and  $B$ ) would need to adopt  $M$ 's child,  $Y$ .

More intuitively, when we simplify away a mediator, we need to make sure that we preserve the causal relationships being mediated—both those among substantive variables and any random shocks at the mediating causal steps.<sup>17</sup>

#### *Eliminating exogenous nodes*

---

<sup>17</sup>Eliminating endogenous nodes may also operate via “encapsulated conditional probability distributions” (Koller and Friedman, 2009) wherein a system of nodes,  $\{Z_i\}$  is represented by a single node,  $Z$ , that takes the parents of  $\{Z_i\}$  not in  $\{Z_i\}$  as parents to  $Z$  and issues the children of  $(Z_i)$  that are not in  $(Z_i)$  as children. However, this is not a fundamental alteration of the graph.

What about eliminating exogenous nodes—nodes with no parents? For the most part, exogenous nodes cannot be eliminated, but must either be replaced by or incorporated into  $U$  (or  $\theta$ ) terms. The reason is that we need preserve any dependencies or variation generated by the exogenous node. Figure 2.5 walks through four different situations in which we might want to simplify away the exogenous node,  $X$ . (Here we use the more generic  $U$  notation, though the same principles apply if these are type-receptacles  $\theta$ .)

```
## Warning in grid.Call.graphics(C_text,
## as.graphicsAnnot(x$label), x$x, x$y, : conversion
## failure on '(c2) Simplification: X absorbed into \hat{I},
## term' in 'mbcsToSbcs': dot substituted for <ce>

## Warning in grid.Call.graphics(C_text,
## as.graphicsAnnot(x$label), x$x, x$y, : conversion
## failure on '(c2) Simplification: X absorbed into \hat{I},
## term' in 'mbcsToSbcs': dot substituted for <b8>
```

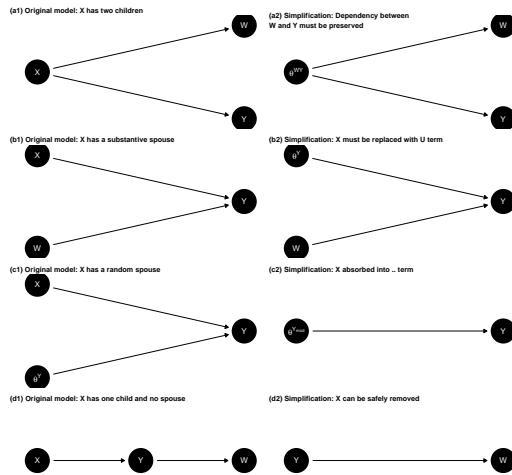


Figure 2.5: Here we represent the basic principles for eliminating exogenous nodes.

- *Multiple children.* In (a1), we start with a model in which  $X$  has two children, thus generating a dependency between  $W$  and  $Y$ . If we

eliminate  $X$ , we must preserve this dependency. We can do so, as pictured in (a2), by replacing  $X$  with a  $\theta$  term that points into both  $W$  and  $Y$ . By convention, we could, alternatively, convey the same information with a dashed, undirected line between  $W$  and  $Y$ . Though we are no longer specifying what it is that connects  $W$  and  $Y$ , the correlation itself is retained.

- *Substantive spouse.* In (b1),  $X$  has a spouse that is substantively specified,  $W$ . If we eliminate  $X$ , we have to preserve the fact that  $Y$  is not fully determined by  $W$ ; *something* else also generates variation in  $Y$ . We thus need to replace  $X$  with a  $\theta$  term,  $\theta^Y$ , to capture the variation in  $Y$  that is not accounted for by  $W$ .
- *$\theta$ -term spouse.* In (c1),  $X$  has a spouse that is *not* substantively specified,  $U^Y$ . Eliminating  $X$  requires, again, capturing the variance that it generates as a random input. As we already have a  $\theta$  term pointing only into  $Y$ , we can substitute in  $\theta_{\text{mod}}^Y$ , which represents both  $U^Y$  and the variance generated by  $X$ .<sup>18</sup>
- *One child, no spouse.* In (d1),  $X$  has only one child and no spouse. Here we can safely eliminate  $X$  with no loss of information. It is always understood that every exogenous node has some cause, and there is no loss of information in simply eliminating a node's causes if those causes are exogenous and do not affect other endogenous nodes in the model. In (d2) we are simply not specifying  $Y$ 's cause, but we have not lost any dependencies or sources of variance that had been expressed in (d1).

One interesting effect of eliminating a substantive exogenous node can be to render seemingly deterministic relations effectively probabilistic. In moving from (b1) to (b2), we have taken a component of  $Y$  that was determined by  $X$  and converting it into a random disturbance. Just as we can explain a more probabilistic claim with a less probabilistic theory, we can derive claims from simplified models with greater probabilism from theories with greater determinism.

We can apply these principles to a model of any complexity. We illustrate a wider range of simplifications by starting with Figure 2.6. In Figure 2.7, we

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<sup>18</sup>This aggregation cannot occur if  $\theta^Y$  also has another child,  $W$ , that is not a child of  $X$  since then we would be representing  $Y$ 's and  $W$ 's random components as identical, which they are not in the original graph.

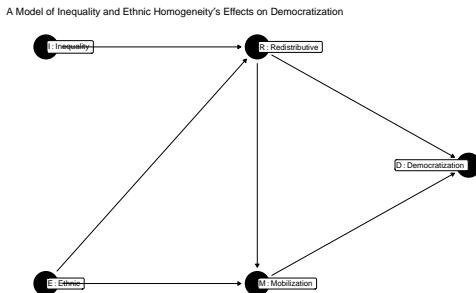


Figure 2.6: A model from which multiple simpler models can be derived.

show all permissible reductions of the more elaborate model. We can think of these reductions as the full set of simpler claims (involving at least two nodes) that can be derived from the original model. In each subgraph,

- we mark eliminated nodes in grey;
- those nodes that are circled must be replaced with  $\theta$  terms; and
- arrows represent the causal dependencies that must be preserved.

Note, for instance, that neither  $S$  (because it has a spouse) nor  $X$  (because it has multiple children) can be simply eliminated; each must be replaced with a  $\theta$  term. Also, the simplified graph with nodes missing can contain arrows that do not appear at all in the graph: eliminating  $C$ , for instance, forces an arrow running from  $X$  to  $R$  (though that is there already) and another running from  $X$  to  $Y$ , as  $X$  must adopt  $M$ 's children. The simplest elimination is of  $Y$  itself since it does not encode any dependencies between other variables.

### 2.3.3.2 Conditioning on nodes

Another way to simplify a model is to condition on the value of a node. When we condition on a node, we are restricting the model in scope to situations in which that node's value is held constant. Doing so allows us to eliminate the node as well as all arrows pointing into it or out of it. Consider three different situations in which we might condition on a node:

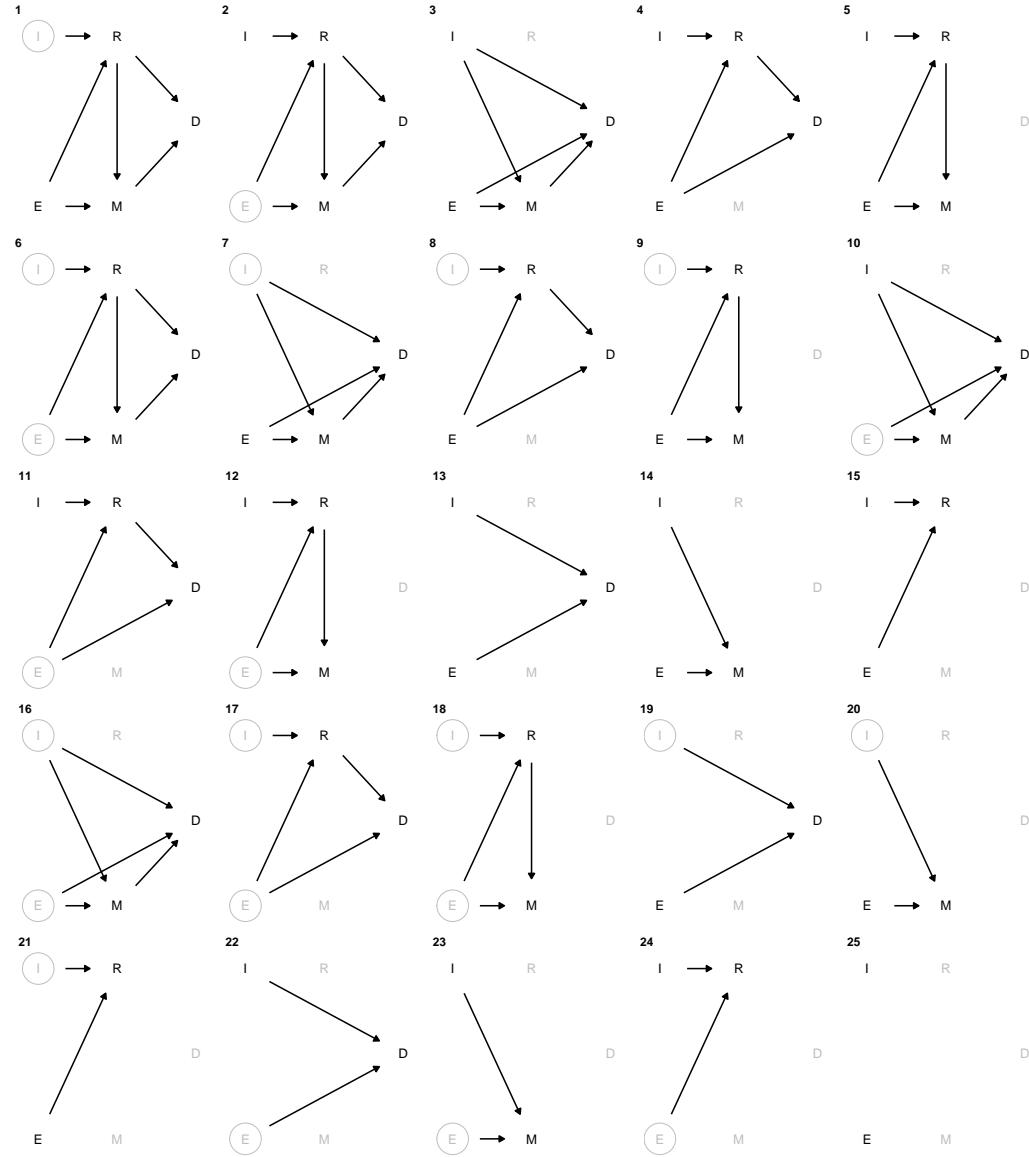


Figure 2.7: Simplifications of the model of Figure X. Nodes that are eliminated are marked in grey; circles denote exogenous nodes that are replaced in subgraphs by unidentified variables. (A circled node pointing into two other nodes could equivalently be indicated as an undirected edge connecting the two.)

- *Exogenous, with multiple children.* In simplifying (a1) in Figure 2.5, we need to be sure we retain any dependence that  $X$  generates between  $W$  and  $Y$ . However, recalling the rules of conditional independence on a graph (see Chapter 2), we know that  $W$  and  $Y$  are *independent* conditional on  $X$ . Put differently, if we restrict the analysis to contexts in which  $X$  takes on a constant value, the model implies that  $Y$  and  $W$  will be uncorrelated across cases. As fixing  $X$ 's value breaks the dependence between  $Y$  and  $W$ , we can drop  $X$  (and the arrows pointing out of it) without having to represent that dependence.
- *Exogenous, with spouse.* In simplifying (b1) or (c1) in Figure 2.5, we need to account for the variation generated by  $X$ . If we fix  $X$ 's value, however, then we eliminate this variation by assumption and do not need to continue to represent it (or the arrow pointing out of it) on the graph.
- *Endogenous.* When we condition on an endogenous node, we can eliminate the node as well the arrows pointing into and out of it. We, again, leverage relations of conditional independence here. If we start with model  $X \rightarrow M \rightarrow Y$  and we condition on the mediator,  $M$ , we sever the link between  $Y$  and  $X$ , rendering them conditionally independent of one another. We can thus remove  $M$ , the arrow from  $X$  to  $M$ , and the arrow from  $M$  to  $Y$ . In the new model, with  $M$  fixed,  $Y$  will be entirely determined by the random disturbance  $\theta^Y$ .

### 2.3.4 Retaining probabilistic relations

We have highlighted the graphical implications of node elimination or node conditioning but importantly the distribution over  $\theta$  also needs to be preserved faithfully in a move to a simpler model.

In sum, we can work with models that are simpler than our causal beliefs: we may believe a model to be true, but we can derive from it a sparser set of claims. There may be intervening causal steps or features of context that we believe matter, but that are not of interest for a particular line of inquiry. While these can be removed, we nonetheless have to make sure that their *implications* for the relations remaining in the model are not lost. Understanding the rules of reduction allow us to undertake an important task: checking which simpler claims are and are not consistent with our full belief set.

## 2.4 Conclusion

In this chapter, we have shown how we can inscribe causal beliefs, rooted in the potential outcomes framework, into a causal model. In doing so, we have now set out the foundations of the book’s analytic framework. Causal models are both the starting point for analysis in this framework and the object about which we seek to learn. Before moving on to build on this foundation, we aim in the next chapter to offer further guidance by example on the construction of causal models, by illustrating how a set of substantive social scientific arguments from the literature can be represented in causal model form.

## 2.5 Chapter Appendix

### 2.5.1 Steps for constructing causal models

### 2.5.2 Model construction in code

Our `gbiqq` package provides a set of functions to implement all of these steps concisely for *binary* models – models in which all nodes are dichotomous.

```
# Steps 1 and 2
# We define a model with three binary nodes and specified edges between them:
model <- make_model("X -> M -> Y")

# Unrestricted functional forms are allowed by default, though these can
# also be reduced. Here we impose monotonicity at each step
# by removing one type for M and one for Y
model <- set_restrictions(model, labels = list(M = "10", Y="10"))

# Step 4
# We set priors over the distribution of (remaining) causal types.
# Here we set "jeffreys priors"
model <- set_priors(model, distribution = "jeffreys")
```

```
# We now have a model defined as an R object.  
# You might plot it like this:  
hj_ggdag(model=model)  
  
# Later we will ask questions of this model and update it using data.
```

These steps are enough to fully describe a binary causal model. Later in this book we will see how we can ask questions of a model like this but also how to use data to train it.

### 2.5.3 Rules for moving between levels

### 2.5.4 Reading conditional independence from a graph

We illustrate how to identify the relations of conditional independence between  $A$  and  $D$  in Figure 2.8.

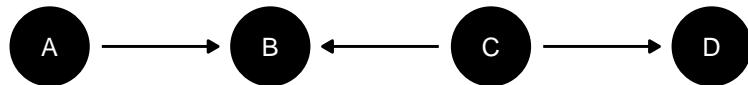


Figure 2.8: An exercise:  $A$  and  $D$  are conditionally independent, given which other node(s)?

Are  $A$  and  $D$  independent:

- unconditionally?

Yes.  $B$  is a collider, and information does not flow across a collider if the value of the collider node or its consequences is not known. Since no information

can flow between  $A$  and  $C$ , no information can flow between  $A$  and  $D$  simply because any such flow would have to run through  $C$ .

- if you condition on  $B$ ?

No. Conditioning on a collider opens the flow of information across the incoming paths. Now, information flows between  $A$  and  $C$ . And since information flows between  $C$  and  $D$ ,  $A$  and  $D$  are now also connected by an unbroken path. While  $A$  and  $D$  were independent when we conditioned on nothing, they cease to be independent when we condition on  $B$ .

- if you condition on  $C$ ?

Yes. Conditioning on  $C$ , in fact, has no effect on the situation. Doing so cuts off  $B$  from  $D$ , but this is irrelevant to the  $A$ - $D$  relationship since the flow between  $A$  and  $D$  was already blocked at  $B$ , an unobserved collider.

- if you condition on  $B$  and  $C$ ?

Yes. Now we are doing two, countervailing things at once. While conditioning on  $B$  opens the path connecting  $A$  and  $D$ , conditioning on  $C$  closes it again, leaving  $A$  and  $D$  conditionally independent.

Analyzing a causal graph for relations of independence represents one payoff to formally encoding our beliefs about the world in a causal model. We are, in essence, drawing out implications of those beliefs: given what we believe about a set of direct causal relations (the arrows on the graph), what must this logically imply about other dependencies and independencies on the graph, conditional on having observed some particular set of nodes? We show in a later chapter how these implications can be deployed to guide research design, by indicating which parts of a causal system are potentially informative about other parts that may be of interest.

# Chapter 3

## Illustrating Causal Models

In this short chapter, we provide more of a sense of how we can encode prior knowledge in a causal model by asking how we might construct models in light of extant scholarly works. We undertake this exercise by drawing on three well-known publications in comparative politics and international relations: Paul Pierson’s seminal book on welfare-state retrenchment (Pierson, 1994); Elizabeth Saunders’ research on leaders’ choice of military intervention strategies (Saunders, 2011); and Przeworski and Limongi’s work on democratic survival (Przeworski and Limongi, 1997), an instructive counterpoint to Boix’s (Boix, 2003) argument about a related dependent variable. For each, we represent the causal knowledge that we might plausibly think we take away from the work in question in the form of a causal model.

Readers might represent these knowledge bases differently; our aim here is only to illustrate how causal models are constructed, rather than to defend a particular representation (much less the works in question) as accurate.

For each exercise below, we focus on a specific argument in the literature in order to fix in place a relatively clear set of background causal beliefs and simplify the exposition. We emphasize, however, that *in general* a causal model should be thought of as a representation of our state of knowledge or beliefs about causal relations within a domain, rather than as a representation of a specific argument. Suppose, for instance, that we are interested in testing a specific argument in which  $X$  affects  $Y$  through the mediator  $M$ . In constructing a causal model to guide our empirical analysis, we cannot simply draw that argument in DAG form ( $X \rightarrow M \rightarrow Y$ ) and leave it at that. In line

with the principles relating to conditional independence outlined in Chapter 2, we must consult our beliefs about this causal domain in a broader sense. For instance, given what we know about the domain from prior observations or studies, is it plausible that  $X$  could affect  $Y$  through a pathway that does not go through  $M$ ? If we believe it is possible, then we must also draw a direct  $X \rightarrow Y$  arrow, or our causal model will steer us wrong — even if our primary aim is to examine the pathway through  $M$ . Otherwise, our DAG will contain a relation of conditional independence ( $X$  being conditionally independent of  $Y$  given  $M$ ) that we do not believe holds. Thus, while we draw on specific works in the illustrations in this chapter, we urge readers to remember that in practice one would want to characterize a broader prior knowledge base in relation to a causal domain in generating a causal model.

We aim to illuminate a number of features of causal models and their construction with these exercises. The examples that we work through variously illustrate how graphs capture beliefs about relations of conditional independence; the potential causal complexity embedded in the causal structures implied by common social-scientific arguments; and the elements of a causal model that cannot be read from a graph. For each work, we discuss both a parametric rendering of the causal functions and a non-parametric formulation built on nodal types.

### 3.1 Welfare state reform

The argument in Pierson's 1994 book *Dismantling the Welfare State?* challenged prior notions of post-1980 welfare-state retrenchment in OECD countries as a process driven primarily by socioeconomic pressures (slowed growth, rising unemployment, rising deficits, aging populations) and the rise of market-conservative ideologies (embodied for instance by the ascendance of Thatcher and Reagan). Pierson argues that socioeconomic and ideological forces put retrenchment on the policy agenda, but do not ensure its enactment because retrenchment is a politically perilous process of imposing losses on large segments of the electorate. Governments will only impose such losses if they can do so in ways that allow them avoid blame for doing so—by, for instance, making the losses hard to perceive or the responsibility for them difficult to trace. These blame-avoidance opportunities are themselves conditioned by the particular social-program

structures that governments inherit.

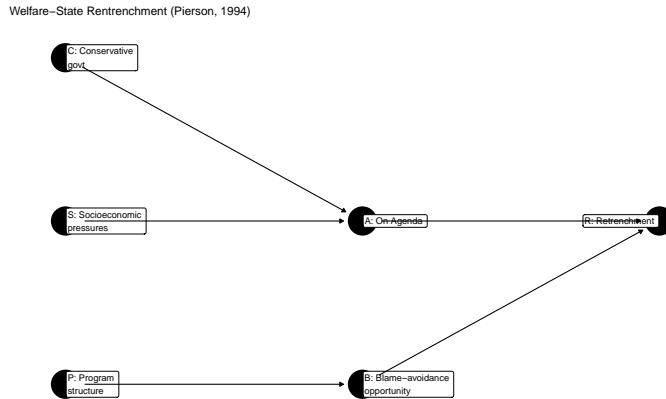


Figure 3.1: A graphical representation of Pierson (1994).

While the argument has many more specific features (e.g., different program-structural factors that matter, various potential strategies of blame-avoidance), its essential components can be captured with a relatively simple causal model. We propose such a model in graphical form in Figure 3.1. Here, the outcome of retrenchment ( $R$ ) hinges on whether retrenchment makes it onto the agenda ( $A$ ) and on whether blame-avoidance strategies are available to governments ( $B$ ). Retrenchment emerges on the policy agenda as a consequence of both socioeconomic developments ( $S$ ) and the ascendance of ideologically conservative political actors ( $C$ ). Inherited program structures ( $P$ ), meanwhile, determine the availability of blame-avoidance strategies. To avoid cluttering the graph, we do not represent the  $\theta$  terms, but it is implied that every node on this graph has a  $\theta$  node pointing into it.

A few features of this graph warrant attention. As we have discussed, it is the omitted arrows in any causal graph that imply the strongest statements. The graph implies that  $C$ ,  $S$ , and  $P$ —which are neither connected along a directed path nor downstream from a common cause—are independent of one another. This implies, for instance, that whether conservatives govern is independent of whether program structures will allow for blame-free retrenchment. Thus, as Pierson argues, a Reagan or Thatcher can come to power but nonetheless run up against an opportunity structure that would make retrenchment politically perilous. Given the absence of bidirectional

arrows indicating confounding, the graph similarly implies that the nodal types for all nodes are independent of one another.

Further, this graph represents the belief that any effect of program structures on retrenchment *must* run through their effects on blame-avoidance opportunities. One could imagine relaxing this restriction by, for instance, drawing an arrow from  $P$  to  $A$ : program structures might additionally affect retrenchment in other ways, such as by conditioning the fiscal costliness of the welfare state and thus helping to determine whether reform makes it onto the agenda. If the current state of knowledge suggested that program structures could affect retrenchment via a pathway other than blame-avoidance opportunities, then we would indeed want to include a direct  $P \rightarrow A$  arrow.

Where two variables *are* connected by an arrow, moreover, this does not imply that a causal effect will always operate. Consider, for instance, the arrow pointing from  $A$  to  $R$ . The fact that  $A$  sometimes affects  $R$  and sometimes does not is, in fact, central to Pierson's argument: conservatives and socioeconomic pressures forcing retrenchment on the agenda will *not* generate retrenchment if blame-avoidance opportunities are absent.

The graph also reflects a choice about where to begin. We could, of course, construct a causal account of how conservatives come to power, how socioeconomic pressures arise, or why programs were originally designed as they were. Yet it is perfectly permissible for us to bracket these antecedents and start the model with  $C$ ,  $S$ , and  $T$ , as long as we do not believe that these variables have any antecedents in common. If they do have common causes, then this correlation should be captured in the DAG.<sup>1</sup>

The DAG itself tells us about the possible direct causal dependencies but is silent on the ranges of and functional relations among the variables. How might we express these? With three endogenous variables, we need three functions indicating how their values are determined. Moreover, every variable pointing directly into another variable must be part of that second variable's function.

Let us assume that all variables (including the implied  $\theta$  terms) are binary, with each condition either absent or present.

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<sup>1</sup>In DAG syntax, this correlation can be captured by placing the common cause(s) explicitly on the graph or by drawing a dashed line between the correlated nodes, leaving the source of the correlation unspecified.

One option would be to take a parameteric approach and imagine specific functions connecting parents to children, with  $\theta$  terms representing exogenous “noise.” For instance, we can capture quite a lot of Pierson’s theoretical logic with the following quite simple functional equations:

- $A = CS\theta^C$ , capturing the idea that retrenchment makes it on the agenda only if conservatives are in power *and* socioeconomic pressures are high.
- $B = P\theta^P$ , implying that blame-avoidance opportunities arise only when program structures take a particular form.
- $R = AB\theta^R$ , implying that retrenchment will occur only if it is on the agenda and blame-avoidance opportunities are present.

In each equation, the  $\theta$  term allows for exogenous forces that might block the outcome from occurring. In the last functional equation, for instance, retrenchment will only occur if retrenchment is on the agenda and blame-avoidance opportunities are present, but even if both are present, the effect on retrenchment also hinges on the value of  $\theta^R$ . When  $\theta^R = 1$ , the  $AB$  combination has a positive causal effect on retrenchment. When  $\theta^R = 0$ ,  $AB$  has no causal effect: retrenchment will not occur regardless of the presence of  $AB$ . We can think of  $\theta^R$  as capturing a collection of features of a case’s context that might render the case susceptible or not susceptible to an  $AB$  causal effect. For instance, Pierson’s analysis suggests that a polity’s institutional structure might widely diffuse veto power such that stakeholders can block reform even when retrenchment is on the agenda and could be pursued without electoral losses. We could think of such a case as having a  $\theta^R$  value of 0, implying that  $AB$  has no causal effect. A  $\theta^R = 1$  case, with a positive effect, would be one in which the government has the institutional capacity to enact reforms that it has the political will to pursue.

Alternatively, we could take a non-parameteric approach, as we generally do in the remainder of this book. In a non-parametric setup, each node’s  $\theta$  term captures that node’s nodal type. Each value of a  $\theta$  term’s range represents a possible way in which the node might respond to its parents. We would define  $\theta^A$  as taking on one of 16 values (16 types, given 2 parent nodes);  $\theta^B$  as taking on one of four values; and  $\theta^R$  as taking on one of 16 values; with  $\theta^C$  and  $\theta^S$  each taking on one of two values.

Then choices on the probability distributions fully reflect the ways these variables relate to each other. Note that the parametric argument given above can be thought of as a special case of the non-parametric representation with all probability mass placed on a small set of possible nodal types. Thus the central thrust of Pierson's argument could then be represented in nodal-type form as:

- $\theta^A = \theta_{0001}^A$
- $\theta^B = \theta_{01}^B$
- $\theta^R = \theta_{0001}^R.$

In practice, however we would allow for a richer probability *distribution* over each  $\theta$ , representing beliefs over the assignment process or causal relations operating at each node. Beliefs about the distribution of exogenous conditions would be captured in distributions over the values of  $\theta^C$ ,  $\theta^S$ , and  $\theta^P$ . How we handle distributions over  $\theta^A$ ,  $\theta^B$ , and  $\theta^R$  depends on the degree of confidence that we want to express in Pierson's argument. To represent the belief that Pierson's argument is correct with certainty and operates in uniform, deterministic fashion across units, we would simply have degenerate distributions for  $\theta^A$ ,  $\theta^B$ , and  $\theta^R$ , with a probability of 1.0 placed on the respective nodal types shown above. To capture uncertainty about the functional relations on any graph or if we believe that there is some heterogeneity of effects across units we would disperse probability density across types for each  $\theta$ . For instance, for  $\theta^R$  we might want to put some weight on  $\theta_{0011}^R$  (blame-avoidance opportunities alone are enough to generate retrenchment),  $\theta_{0101}^R$  (conservative leaders alone are enough),  $\theta_{0111}^R$  (either is enough), and  $\theta_{0000}^R$  (retrenchment will not happen even when both conditions are present), while perhaps putting greatest weight on  $\theta_{0001}^R$ .<sup>2</sup>

## 3.2 Military Interventions

Saunders (2011) asks why, when intervening militarily abroad, do leaders sometimes seek to transform the *domestic* political institutions of the states they target but sometimes seek only to shape the states' *external* behaviors.

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<sup>2</sup>In notation that we use later, these beliefs would be represented with a  $\lambda^R$  vector.

Saunders' central explanatory variable is the nature of leaders' causal beliefs about security threats. When leaders are "internally focused," they believe that threats in the international arena derive from the internal characteristics of other states. Leaders who are "externally focused," by contrast, understand threats as emerging strictly from other states' foreign and security policies.

These basic worldviews, in turn, affect the cost-benefit calculations leaders make about intervention strategies—in particular, about whether to try to transform the internal institutions of a target state—via two mechanisms. First, an internal focus (as opposed to an external focus) affects leaders' perceptions of the likely security gains from a transformative intervention strategy. Second, internal vs. external focus affects the kinds of strategic capabilities in which leaders invest over time (do they invest in the kinds of capabilities suited to internal transformation?); and those investments in turn affect the costliness and likelihood of success of alternative intervention strategies. Calculations about the relative costs and benefits of different strategies then shape the choice between a transformative and non-transformative approach to intervention.

At the same time, leaders can only choose a transformative strategy if they decide to intervene at all. The decision about whether to intervene depends, in turn, on at least two kinds of considerations. The first is about fit: a leader is more likely to intervene against a target when the nature of the dispute makes the leader's preferred strategy appear feasible in a given situation. Second, Saunders allows that forces outside the logic of her main argument might also affect the likelihood of intervention: in particular, leaders may be pushed to intervene by international or domestic audiences.

Figure 3.2 depicts the causal dependencies in Saunders' argument in DAG form (again, with all  $\theta$  terms implied). Working from left to right, we see that whether or not leaders are "internally focused" ( $F$ ) affects the expected net relative benefits of transformation ( $B$ ), both via a direct pathway and via an indirect pathway running through investments in transformative capacities ( $T$ ). Characteristics of a given dispute or target state ( $D$ ) likewise influence the benefits of transformation ( $B$ ). The decision about whether to intervene ( $I$ ) is then a function of three factors: internal focus ( $F$ ), the expected relative net benefits of transformation ( $B$ ), and audience pressures ( $A$ ). Finally, the choice of whether to pursue a transformative strategy ( $S$ ) is

a function of whether or not intervention occurs at all ( $I$ ), and of cost-benefit comparisons between the two strategies ( $B$ ).

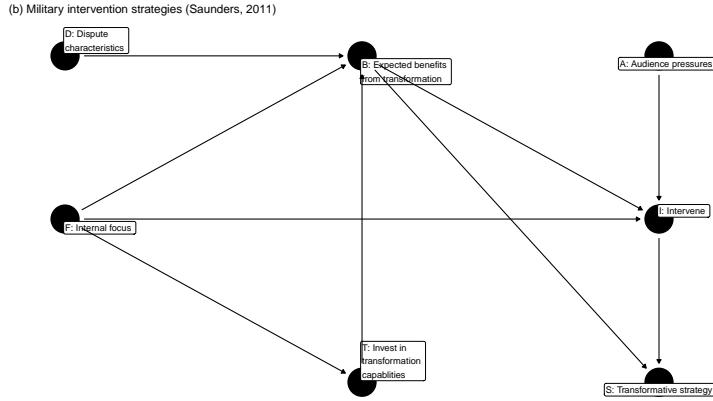


Figure 3.2: A graphical representation of Saunders' (2011) argument.

This DAG illustrates how readily causal graphs can depict the multiple pathways through which a given variable might affect another variable, as with the multiple pathways linking  $F$  to  $I$  and  $B$  (and, thus, all of its causes) to  $S$ . In fact, this graphical representation of the dependencies in some ways throws the multiplicity of pathways into even sharper relief than does a narrative exposition of the argument. For instance, Saunders draws explicit attention to how causal beliefs operate on expected net benefits via both a direct and indirect pathway, both of which are parts of an indirect pathway from  $F$  to the outcomes of interest,  $I$  and  $S$ . What is a bit easier to miss without formalization is that  $F$  also acts *directly* on the choice to intervene as part of the feasibility logic: when leaders assess whether their generally preferred strategy would be feasible if deployed against a particular target, the generally preferred strategy is itself a product of their causal beliefs. The DAG also makes helpfully explicit that the two main outcomes of interest—the choice about whether to intervene and the choice about how—are not just shaped by some of the same causes but are themselves causally linked, with the latter depending on the former.

Omitted links are also notable. For instance, the lack of an arrow between  $D$  and  $A$  suggests that features of the dispute that affect feasibility have no effect on audience pressures. If we instead believed there could be other

connections—for instance, that audiences take feasibility into account in demanding intervention—then we would want to include a  $D \rightarrow A$  arrow.

Turning to variable ranges and functional equations, it is not hard to see how one might readily capture Saunders' logic in a fairly straightforward set-theoretic manner. All variables except  $S$  could be treated as binary with, for instance,  $F = 1$  representing internally focused causal beliefs,  $T = 1$  representing investments in transformative capabilities,  $B = 1$  representing expectations that transformation will be more net beneficial than non-transformation,  $D = 1$  meaning that a dispute has characteristics that make transformation a feasible strategy, and so on. Although there are two strategies, we in fact need three values for  $S$  because it must be defined for all values of the other variables—i.e., it must take on a distinct categorical value if there is no intervention at all. We could then define functions, such as:

- $B = FTD$ , implying that transformation will only be perceived to be net beneficial in a case if and only if the leader has internally focused causal beliefs, the government is prepared for a transformative strategy, and the dispute has characteristics that make transformation feasible.
- $I = (1 - |B - F|) + (1 - (1 - |B - F|))A$ , implying that intervention can occur under (and only under) either of two alternative sets of conditions: if the generally preferred strategy and the more net-beneficial strategy in a given case are the same (i.e., such that  $B - F = 0$ ) or, when this alignment is absent (i.e., such that  $|B - F| = 0$ ), where audiences pressure a leader to intervene.

As illustrated in the Pierson example, in a non-parametric framework, each parametric functional equation represents one nodal type for the relevant  $\theta$ . For instance, though we spare the reader the complexities of the corresponding subscript notation, there is a single value of  $\theta^B$  under which the conditions  $F = 1, P = 1$ , and  $T = 1$  generate  $B = 1$ , and we get  $B = 0$  otherwise. Likewise, there exists a single value of  $\theta^I$  under which  $B = 1, F = 1$  and  $B = 0, F = 0$  produce  $I = 1$ , for either value of  $A$ ; and  $A$  has a positive effect on  $I$  whenever  $B \neq F$ . To work with this model, we would specify a probability distribution over all possible nodal types for each node on the graph.

This example also nicely illustrates how much potential causal complexity a moderately intricate argument and causal graph implies. The number of

*possible* nodal types at each node depends on how many parents that node has. Looking at the endogenous nodes here, we have one node with one parent ( $T$ ), implying 4 nodal types; one node with two parents ( $S$ ), implying 16 nodal types; and two nodes with 3 parents ( $B$  and  $I$ ), implying 256 nodal types each. If we now conceptualize the set of possible “causal types” as containing all distinct combinations of nodal types—all ways in which a case might behave across all of its nodes (see Chapter 2)—then this graph implies about 4 million different ways in which the values of exogenous nodes ( $D$ ,  $F$ , and  $A$ ) might jointly produce patterns of outcomes. Saunders’ argument effectively represents one of these 4 million possible sets of relations.

The framework that we outline in this book allows for updating on arguments like Saunders’: we can ask how likely the specific causal type implied by this argument is relative to other causal types. Yet, as we will see, the approach lends itself to a much broader view of causal inquiry. In general, we will use data to update beliefs over *all* causal types allowed for in a model, and then use these updated beliefs to answer any number of causal questions about relations in the model. For instance, we can use the same data and updated model to ask about the average effect of internal focus on intervention; the relative importance in this effect of the expected-benefits pathway over the direct pathway; about individual steps in the causal chain, such as the effect of expected benefits on choice of strategy; and so on.

### 3.3 Development and Democratization

Przeworski and Limongi (1997) argue that democratization occurs for reasons that are, with respect to socioeconomic or macro-structural conditions, largely idiosyncratic; but once a country has democratized, a higher level of economic development makes democracy more likely to survive. Economic development thus affects whether or not a country is a democracy, but only after a democratic transition has occurred, not before. Thus, in their description—and contrary to Boix (2003)—democratization is “exogenous”: it is not determined by other variables in the model. The dynamic component of Przeworski and Limongi’s argument—the fact that both the presence of democracy and the causal effect of development on democracy depend on whether a democratic transition occurred at a previous point in time—forces us to think about how to capture over-time processes in a causal

model.

We represent Przeworski and Limongi's argument in the DAG in Figure 3.3. The first thing to note is that we can capture dynamics by considering democracy at different points in time as separate nodes. According to the graph, whether a country is a democracy in a given period ( $D_t$ ) is a function, jointly, of whether it was a democracy in the previous period ( $D_{t-1}$ ) and of the level of per capita GDP in the current period (as well as of other unspecified forces  $\theta^{D_t}$ , not pictured).

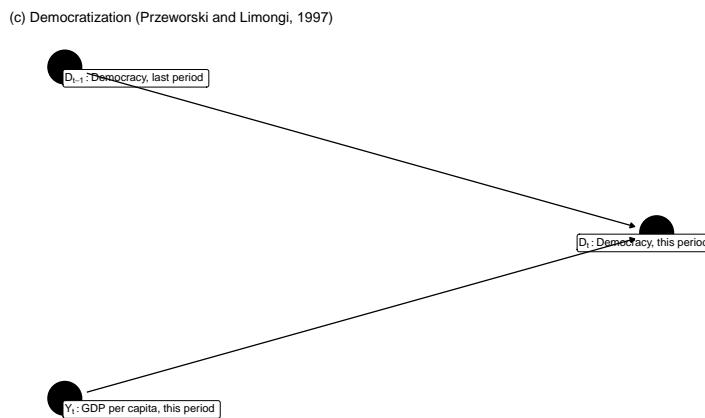


Figure 3.3: A graphical representation of Przeworski and Limongi's argument, where  $D_{t-1}$ =democracy in the previous period;  $GDP_t$ =per capita GDP in the current period;  $D_t$ =democracy in the current period.

Second, the arrow running from  $GDP_{t-1}$  to  $D_t$  means that  $GDP$  *may* affect democracy, not that it always does. Indeed, Przeworski and Limongi's argument is that development's effect depends on a regime's prior state: GDP matters for whether democracies continue to be democracies, but not for whether autocracies go on to become democracies. The absence of an arrow between  $D_{t-1}$  and  $GDP_{t-1}$ , however, implies a (possibly incorrect) belief that democracy and  $GDP$  in the last period are independent of one another.

Inspection of this figure highlights, we think, a curious feature of this argument. The key claim—that the switch *to* democracy does not depend on income—is not readable from the graph. The reason is simply that, given this argument, being a non democracy in one period given you were a democracy in a previous period does depend on income. Your state in the second period

does depend on income. Specifically there is a *counterfactual* dependence—income causes a state to be democratic even if it does not cause it to *transition to* democracy. The effect of income may well be asymmetric depending on whether you start as a democracy or start as an autocracy but this asymmetry has to be captured by the specification of the functional relations; it is not captured by the graph.

For a parametric representation of this asymmetric relationship we can specify a function in which *GDP* can reduce the likelihood of a transition *away* from democracy but does affect the probability of a transition *to* democracy, which should be exogenously determined. One possible translation of the argument into functional terms is:

$$D_t = \mathbb{1}(\theta^{D_t} + (1 - D_{t-1})p + D_{t-1}Y_{t-1}q) > 0$$

where

- $D_t$  and  $D_{t-1}$  are binary, representing current and last-period democracy, respectively
- $p$  is a parameter representing the probability that an autocracy democratizes
- $q$  is a parameter representing the probability that a democracy with a GDP of 1 remains democratic
- $Y_{t-1}$  represents national per capita GDP, scaled to 0-1.
- $\theta^{D_t}$  represents a random, additional input into democracy
- the indicator function, 1, evaluates the inequality and generates a value of 1 if and only if it is true

Unpacking the equation, the likelihood that a country is a democracy in a given period rises and falls with the expression to the left of the inequality operator. This expression itself has two parts, reflecting the difference between the determinants of *transitions to* democracy (captured by the first part) and the determinants of democratic *survival* (captured by the second). The first part comes into play—i.e., is non-zero—only for non-democracies. For non-democracies, the expression evaluates simply to  $p$ , the exogenous probability of democratization. The second part is non-zero only for democracies, where it evaluates to  $q$  times  $Y_{t-1}$ : thus, remaining democratic is more likely as national income rises. The inequality is then evaluated by asking

whether the expression on the left passes a threshold. Thus, higher values for the expression increase the likelihood of democracy while the randomness of the  $\theta^{D_t}$  threshold captures the role of other, idiosyncratic inputs. The mean and variance of  $\theta^{D_t}$  capture the overall likelihood of being a democracy as well as the importance of unspecified factors.<sup>3</sup> In a model like this it would be natural to seek to estimate parameters  $p$  and  $q$  as well as trying to understand the distribution of  $\theta^{D_t}$ .

We can also represent the asymmetry in the binary set up with causal types that we developed in the last chapter.

Type  $\theta_{0001}^{D_t}$  is a type for which the regime type *will stay as they are* if they are wealthy, but will become authoritarian if they are not wealthy. To be clear, wealth still affects whether or not a state is a democracy, rather than an autocracy, in this period–counterfactually—but wealth does not make a non democracy become a democracy. In other words it causes a case to *be* a democracy but not to *become* a democracy. This type can be distinguished from a  $\theta_{0011}^{D_t}$  type in which a non democracy becomes a democracy when income is high.

Although we do not engage with dynamic models in this book, it is instructive to think through the implications of a distribution of causal types for a dynamic process. Say we were to imagine that income were constant but that in each period one half of units were of type  $\theta_{0001}^{D_t}$ , and one half of type  $\theta_{1111}^{D_t}$  (with types drawn afresh each period). Say that in an initial period, half the units were democracies and half had high income and there was no relation between these two features. Then in the next period we would have that half of cases would be democracies (regardless of income), half of which would be surviving democracies and half new democracies; of the other half, one quarter would be surviving democracies (surviving *because of* their income), and the other three quarters would be autocracies, one third of which would be “backsliders” because of their poverty. Similar transitions occur in future periods until eventually the wealthy states are all stable democracies and the

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<sup>3</sup>Note how, while the functional equation nails down certain features of the process, it leaves others up for grabs. In particular, the parameters  $p$  and  $q$  are assumed to be constant for all autocracies and for all democracies, respectively, but their values are left unspecified. And one could readily write down a function that left even more openness—by, for instance, including an unknown parameter that translates  $y$  into a change in the probability of reversion or allowing for non-linearities, with unknown parameters, in this effect.

poorer states transition between democracy and autocracy back and forth randomly each period.

In this approach there are no parameters  $p$  or  $q$  to be estimated. Rather the focus is entirely on the distribution of nodal types.

# Chapter 4

## Causal Queries

Although scholars share a broad common interest in causality there is tremendous heterogeneity in the kinds of causal questions that scholars ask. Consider the relationship between inequality and democratization. We might seek, for instance, to know inequality's average impact on democratization across some set of cases. Alternatively, we might be interested in a particular case—say, Mongolia in 1995—and want to know whether inequality would have an effect *here*. That is a question about causal effects at the case level. Alternatively we might wonder whether the level of democracy in Mongolia in 1995 is *due to* the level of inequality in that case—this is quite a distinct question (in the same way that establishing that poison would make you sick does not imply that you are sick because of poison). Or we may be interested in *how* causal effects unfold, inquiring about the pathway or mechanism through which inequality affects democratization—a question we can also ask at two levels. We can ask whether inequality affected democratization in Mongolia through mobilization of the masses; or we can ask how commonly inequality affects democratization through mobilization across a broad set of cases. Pushing further we might ask a counterfactual question of the form: would inequality have produced democratization had mobilization been prevented from occurring.

Distinct methodological literatures have been devoted to the study of average causal effects, the analysis of case-level causal effects and explanations, and the identification of causal pathways. Fortunately each of these questions can be readily captured as specific queries asked of (and answerable from)

a causal model. As described by Pearl (2010), the goal is to deploy an “*algorithm* that receives a model  $M$  as an input and delivers the desired quantity  $Q(M)$  as the output.” More specifically, we demonstrate how, given the structure we described in Chapter 2, causal queries can be represented as question about one or more *nodes* on a causal graph. When we assimilate our causal questions into a causal model, we are placing what we want to know in formal relation to both what we *already* know and what we can potentially *observe*. As we will see in later chapters, this move allows us then to deploy the model to generate strategies of inference: to determine which observations, if we made them, would be likely to yield the greatest leverage on our query, given our prior knowledge about the way the world works. And by the same logic, once we see the evidence, this integration allows us to “update” on our query—figure out in systematic fashion what we *have* learned—in a manner that takes background knowledge into account.

In the remainder of this chapter, we walk through the conceptualization and causal-model interpretation of five key causal queries:

- Case-level causal effects
- Case-level causal attribution
- Case-level explanation
- Average causal effects
- Causal pathways

These five are in no way exhaustive of the causal questions that can be captured in causal graphs, but they are among the more common social scientific investigations.

## 4.1 Case-level causal effects

The simplest causal question is whether some causal effect operates in an individual case. Does  $X$  have an effect on  $Y$  in this case? For instance, is Yemen in 1995 a case in which a change in economic inequality would produce a change in whether or not the country democratizes? We could put

the question more specifically as a query about a causal effect in a particular direction, for instance: Does inequality have a positive effect on democratization in the case of Yemen in 1995?

In counterfactual terms, a query about case-level causation is a question about what would happen if we could manipulate a variable in the case: if we could hypothetically intervene to change  $X$ 's value in the case, (how) would  $Y$ 's value change? To ask whether a positive (or negative) effect operates for a case is to ask whether a particular counterfactual relation holds in that case. If we assume a setup with binary variables for simplicity, to ask whether inequality has a positive effect on democratization is to ask: if we set  $I$  to 0 would  $D$  take on a value of 0, *and* if we set  $I$  to 1, would  $D$  take on a value of 1? (*Both* of these conditions must hold for  $I$  to have a positive effect on  $D$ .)

We can easily represent this kind of query in the context of a causal model. We show the DAG for such a model in Figure 4.1. As introduced in Chapter 2,  $\theta^Y$  here represents the nodal type characterizing  $Y$ 's response to  $X$  and, if  $X$  and  $Y$  are binary, it can take on one of four values:  $\theta_{10}^Y$ ,  $\theta_{01}^Y$ ,  $\theta_{00}^Y$ , and  $\theta_{11}^Y$  (which map onto our  $a, b, c$  and  $d$  types, respectively). Importantly, given that the value of nodes (or variables) is allowed to vary across cases, this setup allows for  $\theta^Y$ —the causal effect of  $X$  on  $Y$ —to vary across cases. Thus,  $X$  may have a positive effect on  $Y$  in one case (with  $\theta^Y = \theta_{01}^Y$ ), and a negative ( $\theta^Y = \theta_{10}^Y$ ) or no effect ( $\theta^Y = \theta_{00}^Y$  or  $\theta_{11}^Y$ ) on  $Y$  in other cases.

In this model, then, the query, “What is  $X$ 's causal effect in this case?” simply becomes *a question about the value of the nodal type  $\theta^Y$* .

Two natural variants of this question are, “What is the expected effect of  $X$  on  $Y$ ?” and ”What is the probability that  $X$  matters for  $Y$ ? Answering the question requires estimating the probability that  $X$  has a positive effect minus the probability that it has a negative effect:  $\Pr(\theta^Y = \theta_{01}^Y) - \Pr(\theta^Y = \theta_{10}^Y)$ . Answering the second involves assessing  $\Pr(\theta^Y = \theta_{01}^Y \text{ OR } \theta^Y = \theta_{10}^Y)$ .

We can conceptualize this same question even if the model involves more complex relations between  $X$  and  $Y$ . The question itself does not depend on the model having a particular form. For instance, consider a mediation model of the form  $X \rightarrow M \rightarrow Y$ . In this model, a positive effect of  $X$  on  $Y$  can emerge either from a chain of positive effects of  $X$  on  $M$  and of  $M$  on  $Y$  or from a chain of negative effects, while a negative effect of  $X$  on  $Y$  can emerge from a chain of opposite-signed effects. Thus, answering the question

A DAG Capturing a Case-Level Causal Effect



Figure 4.1: This DAG is a graphical representation of the simple causal setup in which the effect of  $X$  on  $Y$  in a given case depends on the case's nodal type, represented by  $\theta^Y$ . With a single binary causal variable of interest, we let  $\theta^Y$  take on values  $\theta_{ij}^Y$ , with  $i$  representing the value  $Y$  takes on if  $X = 0$  and  $j$  representing the value  $Y$  takes on if  $X = 1$ . With a binary framework outcome,  $\theta^Y$  ranges over the four values:  $\theta_{00}^Y$ ,  $\theta_{10}^Y$ ,  $\theta_{01}^Y$  and  $\theta_{11}^Y$ .

means estimating:

$$\Pr((\theta^M = \theta_{01}^M \& \theta^Y = \theta_{01}^Y) \text{ OR } (\theta^M = \theta_{10}^M \& \theta^Y = \theta_{10}^Y)) - \Pr((\theta^M = \theta_{01}^M \& \theta^Y = \theta_{10}^Y) \text{ OR } (\theta^M = \theta_{10}^M \& \theta^Y = \theta_{01}^Y))$$

*Answering* the question now requires guesses about nodal types for  $M$  and for  $Y$ , not just nodal types for  $Y$ . Thus the question remains the same, and answerable, under different models, but the answer to the question might involve summaries of the values of different nodes.

## 4.2 Case-level causal attribution

A query about causal attribution is related to, but different from, a query about a case-level causal effect. When asking about  $X$ 's case-level effect, we are asking, “*Would* a change in  $X$  cause a change in  $Y$  in this case?” The question of causal attribution asks: “*Did*  $X$  cause  $Y$  to take on the value it did in this case?” More precisely, we are asking, “Given the values that  $X$  and  $Y$  *in fact* took on in this case, would  $Y$ 's value have been different if  $X$ 's value had been different?”

For instance, given that we know that inequality in Taiwan was relatively low and that Taiwan democratized in 1996, was low inequality a *cause* of

Taiwan's democratization in 1996? Or: given low economic inequality and democratization in Taiwan in 1996, would the outcome in this case have been different if inequality had been high?

This goes beyond simply asking whether Taiwan is a case in which inequality has a causal effect on democratization. Whereas a case-level causal effect is defined in terms of the  $\theta$  nodes on endogenous variables, we define a causal-attribution query in terms of a larger set of nodes. To attribute  $Y$ 's value in a case to  $X$ , we need to know not only whether this is the kind of case in which  $X$  could have an effect on  $Y$  but also whether the context is such that  $X$ 's value *in fact* made a difference.

Consider, for instance, the general setup in Figure 4.2. Here,  $Y$  is a function of two variables,  $X_1$  and  $X_2$ . This means that  $\theta^Y$  is somewhat more complicated than in a setup with one causal variable:  $\theta^Y$  must here define  $Y$ 's response to all possible combinations of  $X_1$  and  $X_2$ , including interactions between them.

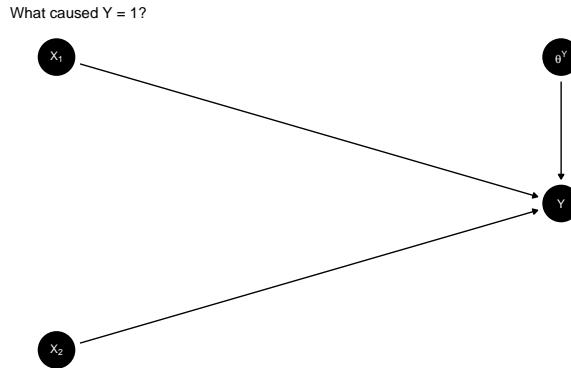


Figure 4.2: This DAG is a graphical representation of the simple causal setup in which  $Y$  depends on two variables  $X_1$  and  $X_2$ . How  $Y$  responds to  $X_1$  and  $X_2$  depends on  $\theta^Y$ , the DAG itself does not provide information on whether or how  $X_1$  and  $X_2$  interact with each other.

We examined the set of nodal types for a set up like this in Chapter 2 (see Table 2.3). In the table, there are four column headings representing the four possible combinations of  $X_1$  and  $X_2$  values. Each row represents one possible pattern of  $Y$  values as  $X_1$  and  $X_2$  move through their four combinations.

One way to conceptualize the size of the nodal-type “space” is to note that

$X_1$  can have any of our four causal effects (the four binary types) on  $Y$  when  $X_2 = 0$ ; and  $X_1$  can have any of four causal effects when  $X_2 = 1$ .<sup>1</sup> This yields 16 possible response patterns to combinations of  $X_1$  and  $X_2$  values.

A query about causal attribution—whether  $X_1 = 1$  caused  $Y = 1$ —for the model in Figure 4.2, needs to be defined in terms of both  $X_2$  and  $\theta^Y$ . Parallel to our Taiwan example, suppose that we have a case in which  $Y = 1$  and in which  $X_1$  was also 1, and we want to know whether  $X_1$  caused  $Y$  to take on the value it did. Answering this question requires knowing whether the case’s type is such that  $X_1$  would have had a positive causal effect on  $Y$ , *given the value of  $X_2$*  (which we can think of as part of the context). Thus, given that we start with knowledge of  $X_1$ ’s and  $Y$ ’s values, our query about causal attribution amounts to a query about two nodal types: (a)  $\theta^{X_2}$  (which gives  $X_2$ ’s value) and (b)  $\theta^Y$ , specifically whether its value is such that  $X_1$  has a positive causal effect given  $X_2$ ’s value.

Suppose, for instance, that we were to observe  $X_2 = 1$ . We then need to ask whether the nodal type,  $\theta^Y$ , is such that  $X_1$  has a positive effect when  $X_2 = 1$ . Consider  $\theta_{0111}^Y$  (type 8 in Table 2.3).<sup>2</sup> This is a nodal type in which  $X_1$  has a positive effect when  $X_2 = 0$  but no effect when  $X_2 = 1$ . Put differently,  $X_2 = 1$  is a sufficient condition for  $Y = 1$ , meaning that  $X_1$  makes no difference to the outcome when  $X_2 = 1$ .

In all we have four qualifying  $Y$ -types:  $\theta_{0001}^Y$ ,  $\theta_{1001}^Y$ ,  $\theta_{0101}^Y$ ,  $\theta_{1101}^Y$ . In other words, we can attribute a  $Y = 1$  outcome to  $X_1 = 1$  when  $X_2 = 1$  and  $Y$ ’s nodal type is one of these four.

Thus, a question about causal attribution is a question about the *joint* value of a set of nodal types: about whether the *combination* of context and the nodal type(s) governing effects is such that changing the causal factor of interest would have changed the outcome.

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<sup>1</sup>This is precisely equivalent to noting that  $X_2$ ’s effect on  $Y$  can be of any of the four types when  $X_1 = 0$  and of any of the four types when  $X_1 = 1$ .

<sup>2</sup>A reminder that, with two-parent nodes, the nodal-type subscript ordering is  $Y|(X_1 = 0, X_2 = 0); Y|(X_1 = 1, X_2 = 0); Y|(X_1 = 0, X_2 = 1); Y|(X_1 = 1, X_2 = 1)$ .

## 4.3 Actual causes

So far we have been dealing with causes in the standard counterfactual sense: antecedent conditions a change in which would have produced a different outcome. Sometimes, however, we are interested in identifying antecedent conditions that were not counterfactual difference-makers but that nonetheless *generated* or *produced* the outcome. Consider, for instance, a situation in which an outcome was overdetermined: multiple conditions were present, each of which on their own, *could* have generated the outcome. Then none of these conditions caused the outcome in the counterfactual sense; yet one or more of them may have been distinctively important in *producing* the outcome. The concept of an *actual cause* can be useful in putting a finer point on this kind of causal question.

A motivating example used in much of the literature on actual causes (e.g. Hall, 2004) imagines two characters, Sally and Billy, simultaneously throwing stones at a bottle. Both are excellent shots and hit whatever they aim at. Sally's stone hits first, and so the bottle breaks. However, Billy's stone *would* have hit had Sally's not hit, and would have broken the bottle. Did Sally's throw cause the bottle to break? Did Billy's?

By the usual definition of causal effects, neither Sally's nor Billy's action had a causal effect: without either throw, the bottle would still have broken. We commonly encounter similar situations in the social world. We observe, for instance, the onset of an economic crisis and the breakout of war—either of which would be sufficient to cause the government's downfall—but with (say) the economic crisis occurring first and toppling the government before the war could do so. In this situation, neither economic crisis nor war in fact made a difference to the outcome: take away either one and the outcome remains the same.

To return to the bottle example, while neither Sally's nor Billy's throw is a counterfactual cause, there is an important sense in which Sally's action obviously broke the bottle, and Billy's did not. We can formalize this intuition by defining Sally's throw as the *actual cause* of the outcome. Using the definition provided by (Halpern, 2015), building on (Halpern and Pearl, 2005) and others, we say that a condition ( $X$  taking on some value  $x$ ) was an actual cause of an outcome (of  $Y$  taking on some value  $y$ ), where  $x$  and  $y$  may be collections of events, if:

1.  $X = x$  and  $Y = y$  both happened
2. there is some set of variables,  $\mathcal{W}$ , such that if they were fixed at the levels that they actually took in the case, and if  $X$  were to be changed, then  $Y$  would change (where  $\mathcal{W}$  can also be an empty set)
3. no strict subset of  $X$  satisfies 1 and 2 (there is no redundant part of the condition,  $X = x$ )

The definition thus describes a condition that *would* have been a counterfactual cause of the outcome if we were to imagine holding constant some set of events that in fact occurred (and that, in reality, might not have been constant if the actual cause had not in fact occurred).

Let us now apply these 3 conditions to the Sally and Billy example. Conditions 1 and 3 are easily satisfied, since Sally *did* throw and the bottle *did* break (Condition 1), and “Sally threw” has no strict subsets (Condition 3).

Condition 2 is met if Sally’s throw made a difference, counterfactually speaking — with the important caveat that, in determining this, we are permitted to condition on (to fix in the counterfactual comparison) any event or set of events that actually happened (or on none at all). To see why Condition 2 is satisfied, we have to think of there being three steps in the process: (1) Sally and Billy throw, (2) Sally’s or Billy’s rock hits the bottle, and (3) the bottle breaks. In actuality, Billy’s stone did not hit the bottle, so we are allowed to condition on that fact in determining whether Sally’s throw was a counterfactual cause. Conditioning on Billy’s stone not hitting, the bottle would *not* have broken had Sally not thrown.

From the perspective of counterfactual causation, it may seem odd to condition on Billy’s stone not hitting the bottle when thinking about Sally not throwing the stone—since Sally’s throwing the stone was the very thing that prevented Billy from hitting the bottle. Yet Halpern argues that this is an acceptable thought experiment for establishing the importance of Sally’s throw since conditioning is constrained to the actual facts of the case. Moreover, the same logic shows why Billy is not an actual cause. The reason is that Billy’s throw is only a cause in those conditions in which Sally did not hit the bottle. But because Sally *did* actually hit the bottle, we are not permitted to condition on Sally not hitting the bottle in determining actual causation. We thus cannot—even through conditioning on actually occurring events—construct any counterfactual comparison in which Billy’s throw is a counterfactual cause of the bottle’s breaking.

The striking result here is that there can be grounds to claim that a condition was the actual cause of an outcome even though, under the counterfactual definition, the effect of that condition on the outcome is 0. (At the same time, all counterfactual causes are automatically actual causes; they meet Condition 2 by conditioning on nothing at all, an empty set  $\mathcal{W}$ .) One immediate methodological implication follows: since actual causes need not be causes, there are risks in research designs that seek to understand causal effects by tracing back actual causes—i.e., the way things actually happened. If we traced back from the breaking of the bottle, we might be tempted to identify Sally’s throw as the cause of the outcome. We would be right only in an actual-causal sense, but wrong in the standard, counterfactual causal sense. Chains of events that appear to “generate” an outcome are not always causes in that sense.<sup>3</sup>

As with other causal queries, the question “Was  $X = x$  the actual cause of  $Y = y$ ?” can be redefined as a question about which combinations of nodal types produce conditions under which  $X$  could have made a difference. To see how, let us run through the Billy and Sally example again, but formally in terms of a model. Consider Figure 4.3, where we represent Sally’s throw ( $S$ ), Billy’s throw ( $B$ ), Sally’s rock hitting the bottle ( $H^S$ ), Billy’s rock hitting the bottle ( $H^B$ ), and the bottle cracking ( $C$ ). Each endogenous variable has a  $\theta$  term associated with it, capturing its nodal type. We capture the possible “preemption” effect with the arrow pointing from  $H^S$  to  $H^B$ , allowing whether Sally’s rock hits to affect whether Billy’s rock hits.<sup>4</sup>

For Sally’s throw to be an actual cause of the bottle’s cracking, we need first to establish that Sally threw ( $\theta^S = \theta_1^S$ ) and that the bottle cracked ( $\theta^C = \theta_1^C$ ) (Condition 1). Condition 3 is automatically satisfied in that  $\theta^S = \theta_1^S$  has no strict subsets. Turning now to Condition 2, we need Sally’s throw to be a counterfactual cause of the bottle cracking if we condition on the value of some set of nodes remaining fixed at the values they in fact took on. As discussed above, we know that we can meet this criterion if we condition on

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<sup>3</sup>Perhaps more surprising, it is possible that the expected causal effect is negative but that  $X$  is an actual cause in expectation. For instance, suppose that 10% of the time Sally’s shot intercepts Billy’s shot but without hitting the bottle. In that case the average causal effect of Sally’s throw on bottle breaking is  $-0.1$  yet 90% of the time Sally’s throw is an actual cause of bottle breaking (and 10% of the time it is an actual cause of non-breaking). For related discussions, see Menzies (1989).

<sup>4</sup>We do not need an arrow in the other direction because Sally throws first.

Billy's throw not hitting. To make this work, we need to ensure, first, that Sally's throw hits if and only if she throws: so  $\theta^{H^S} = \theta_{01}^{H^S}$ . Next, we need to ensure that Billy's throw does not hit whenever Sally's does: this corresponds to any of the four nodal types for  $H^B$  that take the form  $\theta_{xx00}^{H^B}$ , meaning that  $H^B = 0$  whenever  $H^S = 1$ . Note that the effect of Billy throwing on Billy hitting when Sally has *not* thrown—the first two terms in the nodal-type's subscript—does not matter since we have already selected a value for  $\theta^S$  such that Sally does indeed throw.

Finally, we need  $\theta^C$  to take on a value such that  $H^S$  has a positive effect on  $C$  when  $H^B = 0$  (Billy doesn't hit) since this is the actual circumstance on which we will be conditioning. This is satisfied by any of the four nodal types of the form  $\theta_{0x1x}^C$ . This includes, for instance, a  $\theta^C$  value in which Billy's hitting has no effect on the bottle (perhaps Billy doesn't throw hard enough!): e.g.,  $\theta_{0011}^C$ . Here, Sally's throw is a counterfactual cause of the bottle's cracking. And, as we have said, all counterfactual causes are actual causes. They are, simply, counterfactual causes when we hold *nothing* fixed ( $\mathcal{W}$  in Condition 2 is just the empty set).

Notably, we do not need to specify the nodal type for  $B$ : given the other nodal types identified, Sally's throw will be the actual cause regardless of whether or not Billy throws. If Billy does not throw, then Sally's throw is a simple counterfactual cause (given the other nodal types).

The larger point is that actual cause queries can, like all other causal queries, be defined as questions about the values of nodes in a causal model. When we pose the query, was Sally's throw an actual cause of the bottle cracking, we are in effect asking whether the case's combination of nodal types (or its causal type) matches  $\theta_1^S, \theta_x^B, \theta_{xx00}^{H^B}, \theta_{01}^{H^S}, \theta_{0x1x}^C$ .

Likewise, if want to ask *how often* Sally's throw is an actual cause, in a population of throwing rounds, we can address this query as a question about the joint *distribution* of nodal types. We are then asking how common the qualifying combinations of nodal types are in the population given the distribution of types at each node.

Actual causes are conceptually useful whenever there are two sufficient causes for an outcome, but one preempts the operation of the other. For instance, we might posit that both the United States' development of the atomic bomb was a sufficient condition for U.S. victory over Japan in World War II, and that U.S. conventional military superiority was also a sufficient condition

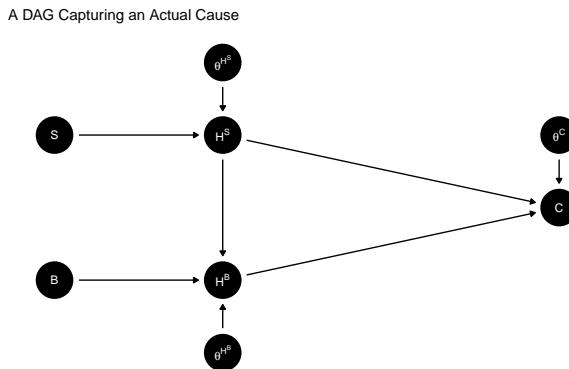


Figure 4.3: This DAG is a graphical representation of the simple causal setup in which the effect of  $X$  on  $Y$  in a given case depends on the case's nodal type for  $Y$ , represented by  $\theta^Y$ .

and would have operated via a land invasion of Japan. Neither condition was a counterfactual cause of the outcome because both were present. However, holding constant the *absence* of a land invasion, the atomic bomb was a difference-maker, rendering it an actual cause. The concept of actual cause thus helps capture the sense in which the atomic bomb distinctively contributed to the outcome, even if it was not a counterfactual cause.

An extended notion (Halpern, 2016, p 81) of actual causes restricts the imagined counterfactual deviations to states that are more likely to arise (more “normal”) than the factual state. We will call this notion a “notable cause.” We can say that one cause,  $A$ , is “more notable” than another cause,  $B$ , if a deviation in  $A$  from its realized state is (believed to be) more likely than a deviation in  $B$  from its realized state.

For intuition, we might wonder why a Republican was elected to the presidency in a given election. In looking at some minimal winning coalition of states that voted Republican, we might distinguish between a set of states that *always* vote Republican and a set of states that usually go Democratic but voted Republican this time. If the coalition is minimal winning, then every state that voted Republican is a cause of the outcome in the standard (difference-making) sense. However, only the states that usually vote Democratic are notable causes since it is only for them that the counterfactual scenario (voting Democratic) was more likely to arise than the factual

scenario. In a sense, we take the “red” states’ votes for the Republican as given—placing them, as it were, in the causal background—and identify as “notable” those conditions that mattered and easily could have gone differently. By the same token, we can say that, among those states that voted Republican this time, those that more commonly vote Democratic are *more* notable causes than those that less commonly vote Democratic.

How notable a counterfactual cause is can be expressed as a claim about the distribution of a set of nodal types. For instance, if we observe  $R^j = 1$  for state  $j$  (it voted Republican), then the notability of this vote directly increases in our belief about the probability that  $\theta^{R^j} = \theta_0^{R^j}$ : the probability that the state’s vote could have gone the other way.

## 4.4 Average causal effects

A more general query asks about an average causal effect in some population. In counterfactual terms, a question about average causal effects is: if we manipulated the value of  $X$  for all cases in the population—first setting  $X$  to one value for all cases, then changing it to another value for all cases—by how much would the average value of  $Y$  in the population change? Like other causal queries, a query about an average causal effect can be conceptualized as learning about a node in a causal model.

We can do this by conceiving of any given case as being a member of a population composed of different nodal types. When we seek to estimate an average causal effect, we seek information about the *shares* of these nodal types in the population.

More formally and adapted from Humphreys and Jacobs (2015), we can use  $\lambda_{ij}^Y$  to refer to the *share* of cases in a population that has nodal type  $\theta_{ij}^Y$ . Thus, given our four nodal types in a two-variable binary setup,  $\lambda_{10}^Y$  is the proportion of cases in the population with negative effects;  $\lambda_{01}^Y$  is the proportion of cases with positive effects; and so on. One nice feature of this setup, with both  $X$  and  $Y$  as binary, is that the average causal effect can be simply calculated as the share of positive-effect cases less the share of negative-effect cases:  $\lambda_{01}^Y - \lambda_{10}^Y$ .

Graphically, we can represent this setup by including  $\lambda^Y$  in a more complex causal graph as in Figure 4.4. As in our setup for case-level causal effects,

$X$ 's effect on  $Y$  in a case depends on (and only on) the case's nodal type,  $\theta^Y$ . The key difference is that we now model the case's type not as exogenously given, but as a function of two additional variables: the distribution of nodal types in a population and a random process through which the case's type is "drawn" from that distribution. We represent the type distribution as  $\lambda^Y$  (a vector of values for the proportions  $\lambda_{10}^Y, \lambda_{01}^Y, \lambda_{00}^Y, \lambda_{11}^Y$ ) and the random process drawing a  $\theta^Y$  value from that distribution as  $U^\theta$ .

In this model, our causal query—about  $X$ 's average causal effect—is thus defined by the vector  $\lambda^Y$ , and specifically by the shares of negative- and positive-causal-effect cases, respectively, in the population. What is  $X$ 's average effect on  $Y$  amounts to asking: what are the values of  $\lambda_{10}^Y$  and  $\lambda_{01}^Y$ ? As with  $\theta^Y$ ,  $\lambda^Y$  is not directly observable. And so the empirical challenge is to figure out what we *can* observe that would allow us to learn about  $\lambda^Y$ 's component values?<sup>5</sup>

We can, of course, likewise pose queries about other population-level causal quantities. For instance, we could ask for what proportion of cases in the population  $X$  has a positive effect: this would be equivalent to asking the value of  $\lambda_{01}^Y$ , one element of the  $\lambda^Y$  vector. Or we could ask about the proportion of cases in which  $X$  has no effect, which would be asking about  $\lambda_{00}^Y + \lambda_{11}^Y$ .

## 4.5 Causal Paths

To develop richer causal understandings, researchers often seek to describe the causal path or paths through which effects propagate. Consider the DAG in Figure 4.5, in which  $X$  can affect  $Y$  through two possible pathways: directly and via  $M$ . Assume again that all variables are binary, taking on values of 0 or 1. Here we have nodal types defining  $M$ 's response to  $X$  ( $\theta^M$ ) and defining  $Y$ 's response to both  $X$  (directly) and  $M$  ( $\theta^Y$ ).

Suppose that we observe  $X = 1$  and  $Y = 1$  in a case. Suppose, further, that we have reasonable confidence that  $X$  has had a positive effect on  $Y$  in this case. We may nonetheless be interested in knowing whether that

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<sup>5</sup>Note also that  $\lambda^Y$  can be thought of as itself drawn from a distribution, such as a Dirichlet. The hyperparameters of this underlying distribution of  $\lambda$  would then represent our uncertainty over  $\lambda$  and hence over average causal effects in the population.

A DAG with Nodal Type Drawn from a Population-level Distribution of Nodal Types

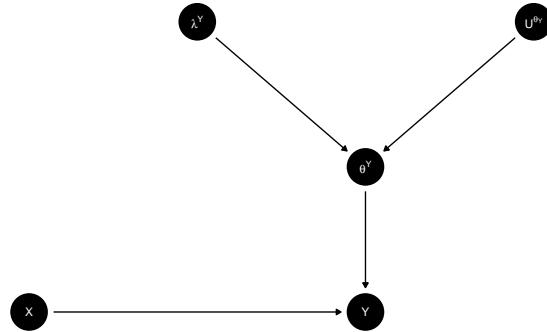


Figure 4.4: This DAG is a graphical representation of a causal setup in which cases are drawn from a population composed of different nodal types. As before,  $X$ 's effect on  $Y$  is a function of a causal-type variable,  $\theta^Y$ . Yet here we explicitly model the process through which the case's type is drawn from a distribution of types in a population. The variable  $\lambda$  is a vector representing the multinomial distribution of nodal types in the population while  $U^\theta$  is a random variable representing the draw of each case from the distribution defined by  $\lambda$ . A case's nodal type,  $\theta^Y$ , is thus a joint function of  $\lambda^Y$  and  $U^{\theta^Y}$ .

causal effect ran *through M*. We will refer to this as a query about a causal path. Importantly, a causal path query is not satisfied simply by asking whether some mediating event along the path occurred. We cannot, for instance, establish that the top path in Figure 4.5 was operative simply by determining the value of  $M$  in this case—though that will likely be useful information.

Rather, the question of whether the mediated (via  $M$ ) causal path is operative is a composite question of two parts: First, does  $X$  have an effect on  $M$  in this case? Second, does that effect—the difference in  $M$ 's value caused by a change in  $X$ —in turn *cause* a change in  $Y$ 's value? In other words, what we want to know is whether the effect of  $X$  on  $Y$  depends on—that is, *will not operate without*—the effect of  $X$  on  $M$ .<sup>6</sup> Framing the query in this way makes clear that asking whether a causal effect operated via a given path is in fact asking about a specific set of causal effects lying along that path.

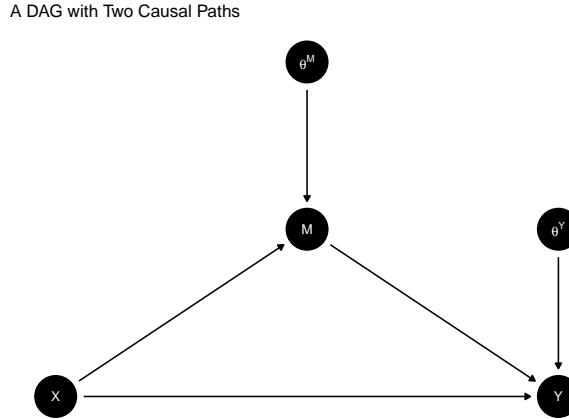


Figure 4.5: Here  $X$  has effects on  $Y$  both indirectly through  $M$  and directly.

As we can show, we can define this causal-path query as a question about

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<sup>6</sup>A very similar question is taken up in work on mediation where the focus goes to understanding quantities such as the “indirect effect” of  $X$  on  $Y$  via  $M$ . Formally, the indirect effect would be

$$Y(X = 1, M = M(X = 1, \theta^M), \theta^Y) - Y(X = 1, M = M(X = 0, \theta^M), \theta^Y))$$

, which captures the difference to  $Y$  if  $M$  were to change in the way that it would change due to a change in  $X$ , but without an actual change in  $X$  (Pearl, 2009, p 132, Imai et al. (2010)).

specific nodes on a causal graph. In particular, a causal path can be defined in terms of the values of  $\theta$  nodes: specifically, in the present example, in terms of  $\theta^M$  and  $\theta^Y$ . To see why, let us first note that there are two combinations of effects that would allow  $X$ 's positive effect on  $Y$  to operate via  $M$ : (1)  $X$  has a positive effect on  $M$ , which in turn has a positive effect on  $Y$ ; or (2)  $X$  has a negative effect on  $M$ , which has a negative effect on  $Y$ .

Thus, in establishing whether  $X$  affects  $Y$  through  $M$ , the first question is whether  $X$  affects  $M$  in this case. Whether or not it does is a question about the value of  $\theta^M$ . We know that  $\theta^M$  can take on four possible values corresponding to the four possible responses to  $X$ :  $\theta_{10}^M, \theta_{01}^M, \theta_{00}^M, \theta_{11}^M$ . For sequence (1) to operate,  $\theta^M$  must take on the value  $\theta_{01}^M$ , representing a positive effect of  $X$  on  $M$ . For sequence (2) to operate,  $\theta^M$  must take on the value  $\theta_{10}^M$ , representing a negative effect of  $X$  on  $M$ .

$\theta^Y$  defines  $Y$ 's response to different combinations of two other variables—here,  $X$  and  $M$ —since *both* of these variables point directly into  $Y$ . Where  $X$  can have both a mediated effect through  $M$  and a direct effect,  $X$  and  $M$  also potentially *interact* in affecting  $Y$ . Another way to think about this setup is that  $M$  is not just a possible mediator of  $X$ 's indirect effect;  $M$  is also a potential *moderator* of  $X$ 's direct effect. This results in sixteen possible values for  $\theta^Y$ —again as shown above in Table 2.3.

What values of  $\theta^Y$  then are compatible with the operation of the  $M$  causal path? Let us first consider this question with respect to sequence (1), in which  $X$  has a positive effect on  $M$ , and that positive effect is necessary for  $X$ 's positive effect on  $Y$  to occur. For this sequence to operate,  $\theta^M$  must take on the value of  $\theta_{01}^M$ . When it comes to  $\theta^Y$ , then, what we need to look for types in which  $X$ 's effect on  $Y$  *depends on M's taking on the value it does as a result of X's positive effect on M*.

We are thus looking for nodal types that capture two kinds of counterfactual causal relations operating on nodes. First,  $X$  must have a positive effect on  $Y$  when  $M$  changes as it does as a result of  $X$ 's positive effect on  $M$ . Second, that change in  $M$ , generated by a change in  $X$ , must be *necessary* for  $X$ 's positive effect on  $Y$  to operate. The thought experiment here thus imagines a situation in which  $X$  changes from 0 to 1,<sup>7</sup> but  $M$  does *not* change to the

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<sup>7</sup>This is the natural thought experiment when explaining a case with realized value of  $X = 1$ , in which the outcome can be thought of as having been generated by a change from  $X = 0$ . The identification of types does hinge, however, on the direction in which

value that it should as a result of this change in  $X$ . We then inspect our types to see if  $Y$  would change from 0 to 1 in this situation. It is only if  $Y$  would *not* change to 1 in this situation that we have identified a nodal type for which the  $M$ -mediated path matters. It is this thought experiment that isolates the causal significance of the path that runs through  $M$ .

Assuming a positive effect of  $X$  on  $M$  ( $\theta^M = \theta_{01}^M$ ), we thus need to apply the following set of queries to  $\theta^Y$ :<sup>8</sup>

1. Is  $X = 1$  a counterfactual cause of  $Y = 1$ , given  $X$ 's positive effect on  $M$ ? Establishing this positive effect of  $X$  involves two queries:
  - a) Where  $X = 0$ , does  $Y = 0$ ? As we are assuming  $X$  has a positive effect on  $M$ , if  $X = 0$  then  $M = 0$  as well. We thus look down the  $X = 0, M = 0$  column and eliminate those types in which we do not observe  $Y = 0$ . This eliminates types 9 through 16.
  - b) Where  $X = 1$ , does  $Y = 1$ ? Again, given  $X$ 's assumed positive effect on  $M$ ,  $M = 1$  under this condition. Looking down the  $X = 1, M = 1$  column, we eliminate those types where we do not see  $Y = 1$ . We retain only types 2, 4, 6, and 8.
2. Is  $X$ 's effect on  $M$  necessary for  $X$ 's positive effect on  $Y$ ? That is, do we see  $Y = 1$  *only* if  $M$  takes on the value that  $X = 1$  generates ( $M = 1$ )? To determine this, we inspect the *counterfactual* condition

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we imagine types changing. In other situations, we might observe  $X = Y = 0$  and thus conceive of the outcome as having been generated by a change from  $X = 1$  to  $X = 0$  (again, assuming a positive effect of  $X$  on  $Y$ ). When we do this, query 2 below changes: we are now looking for types in which  $Y = 1$  when  $X = 0$  but  $M = 1$ . (Does  $Y$  stay at 1 when  $X$  moves to 0 but  $M$  doesn't?) The queries are then satisfied by types 6 and 8, rather than 2 and 6.

<sup>8</sup>Using standard potential outcomes notation, we can express the overall query, conditioning on a positive effect of  $X$  on  $M$ , via the inequality  $Y(1, M(1)) - Y(0, M(0)) > Y(1, M(0)) - Y(0, M(0))$ . The three specific queries formulated below simply correspond to the three unique elements of this expression. We can also readily map the path query that we are defining here—does the positive effect of  $X$  on  $Y$  depend on  $X$ 's effect on  $M$ —onto a query posed in terms of indirect effects. For instance, in our binary setup, conditioning our path query on a positive causal effect of  $X$  on  $Y$ , a positive effect of  $X$  on  $M$ , and an imagined change from  $X = 0$  to  $X = 1$  generates precisely the same result (identifies the same  $\theta^Y$  types) as asking which  $\theta^Y$  types are consistent with a positive indirect effect of  $X$  on  $Y$ , conditioning on a positive total effect and  $X = 1$ .

in which  $X = 1$  and  $M = 0$ , and we ask: does  $Y = 0$ ? Of the four remaining types, only 2 and 6 pass this test.

Under these and only these two values of  $\theta^Y$ — $\theta_{0001}^Y$  and  $\theta_{0101}^Y$ —we will see a positive effect of  $X$  on  $Y$  for which the  $M$ -mediated path is causally necessary, given a positive effect of  $X$  on  $M$ . These two  $\theta^Y$  values are also different from one another in an interesting way. For type  $\theta_{0101}^Y$ ,  $X$ 's effect on  $Y$  runs strictly through  $M$ : if  $M$  were to change from 0 to 1 *without*  $X$  changing,  $Y$  would still change from 0 to 1.  $X$  is causally important for  $Y$  *only* insofar as it affects  $M$ . In a case of type  $\theta_{0001}^Y$ , then, anything else that similarly affects  $M$  would generate the same effect on  $Y$  as  $X$  does. In type  $\theta_{0001}^Y$ , however, both  $X$ 's change to 1 *and* the resulting change in  $M$  are necessary to generate  $Y$ 's change to 1;  $X$ 's causal effect thus requires both the mediated and the unmediated pathway. And here  $X$  itself matters in the counterfactual sense; for a case of type  $\theta_{0001}^Y$ , some other cause of  $M$  would *not* generate the same effect on  $Y$ .

We can undertake the same exercise for sequence (2), in which  $X$  first has a negative effect on  $M$ , or  $\theta^M = \theta_{10}^M$ . Here we adjust the three queries for  $\theta^Y$  to take account of this negative effect. Thus, we adjust query 1a so that we are looking for  $Y = 0$  when  $X = 0$  and  $M = 1$ . In query 1b, we look for  $Y = 1$  when  $X = 1$  and  $M = 0$ . And for query 2, we want types in which  $Y$  fails to shift to 1 when  $X$  shifts to 1 but  $M$  stays at 1. Types  $\theta_{0010}$  and  $\theta_{1010}$  pass these three tests.

In sum, we can define a query about causal paths as a query about the value of  $\theta$  terms on the causal graph. For the graph in Figure 4.5, asking whether  $X$ 's effect runs via the  $M$ -mediated path is asking whether one of four combinations of  $\theta^M$  and  $\theta^Y$  hold in case:

- $\theta^M = \theta_{01}^M$  and ( $\theta^Y = \theta_{0001}^Y$  or  $\theta_{0101}^Y$ )
- $\theta^M = \theta_{10}^M$  and ( $\theta^Y = \theta_{0010}^Y$  or  $\theta_{1010}^Y$ )

It is worth noting how different this formulation of the task of identifying causal pathways is from widespread understandings of process tracing. Scholars commonly characterize process tracing as a method in which we determine whether a mechanism was operating by establishing whether the events lying along that path occurred. As a causal-model framework makes

clear, finding out that  $M = 1$  (or  $M = 0$ , for that matter) does not establish what was going on causally. Observing this intervening step does not by itself tell us what value  $M$  *would* have taken on if  $X$  had taken on a different value, or whether this would have changed  $Y$ 's value. We need instead to conceive of the problem of identifying pathways as one of figuring out the *counterfactual* response patterns of the variables along the causal chain. As we will demonstrate later in the book, explicitly characterizing those response patterns as nodes in a causal model helps us think systematically about empirical strategies for drawing the relevant inferences.

## 4.6 General procedure

We have been able to associate a collection of causal types to each of the causal queries we have described in this chapter. But we have not described a general method for doing so. We do that now.<sup>9</sup>

The algorithm calculates the full set of outcomes on all nodes, given each possible causal type and a collection of controlled conditions (“**do** operations”). Then each causal type is marked as satisfying the query or not. This in turn then tells us the *set* of types that satisfy a query. Quantitative queries, such as the probability of a query being satisfied, or the average treatment effect, can then be calculated by taking the measure of the set of causal types that satisfies the query.

First some notation.

Let  $n$  denote the number of nodes. Label the nodes  $V_1, \dots, V_n$  subject to the requirement that each node's parents precede it in the ordering. Let  $pa_j$  denote the set of values of the parents of node  $j$  and let  $V_j(pa_j, \theta_t)$  denote the value of node  $j$  given the values of its parents and the causal type  $\theta_t$ .

The primitives of a query are questions about the values of outcomes,  $V$ , given some set of controlled operations  $x$ .

- let  $x = (x_1, \dots, x_n)$  denote a set of **do** operations where each  $x_i$  takes on

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<sup>9</sup>In particular we describe the algorithm used by the **CausalQueries** package. This approach is not the efficient but it is intuitive and can be used for arbitrarily complex queries.

a value in  $\{-1, 0, 1\}$ . here -1 indicates “not controlled”, 0 means set to 0 and 1 means set to 1 (this set can be expanded if  $V$  is not binary)

- let  $V(x, \theta_t)$  denote the values  $V$  (the full set of nodes) takes given  $\theta_t$
- a “simple query” is a function  $q(V(x, \theta_t))$  which returns TRUE if  $V(x, \theta_t)$  satisfies some condition and FALSE otherwise.

Queries are summaries of simple queries. For instance, for nodes  $X$  and  $Y$ :

- Query  $Q_1 : 1(Y(X = 1) = 1)$  asks whether  $Y = 1$  when  $X$  is set to 1. This requires evaluating one simple query.
- Query  $Q_2 : 1(Y(X = 1) = 1) \& 1(Y(X = 0) = 0)$  is composed of two simple queries: the first returns true if  $Y$  is 1 when  $X$  is set to 1, the second returns true if  $Y$  is 0 when  $X$  is set to 0; both conditions holding corresponds to a positive effect on a unit.
- Query  $Q_3 : E((1(Y(X = 1) = 1) \& (Y(X = 0) = 0)) - (1(Y(X = 1) = 0) \& 1(Y(X = 0) = 1)))$  asks for the average treatment effect, represented here using four simple queries: the expected difference between positive and negative effects. This query involves weighting by the probability of the causal types.

Then to calculate  $V(x, \theta_t)$ :

1. Calculate  $v_1$ , the realized value of the first node,  $V_1$ , given  $\theta_t$ . This is given by  $v_1 = x_1$  if  $x_1 \neq -1$  and by  $\theta_t^{V_1}$  otherwise.
2. For each  $j \in 2 \dots n$  calculate  $v_j$  using either  $v_j = x_j$  if  $x_j \neq -1$  and  $V_j(pa_j, \theta_t)$  otherwise, where the values in  $pa_j$  are determined in the previous steps.

We now have the outcomes,  $V$ , for all nodes given the operations  $x$  and so can determine  $q(V(x))$ . From there we can calculate summaries of simple queries across causal types.

A last note on conditional queries. Say we are interested in an attribution query of the form: what is the probability that  $X$  causes  $Y$  in a case in which  $X = 1$  and  $Y = 1$ . In this case define simple query  $q_1$  which assesses whether  $X$  causes  $Y$  for a given  $\theta_t$  and simple query  $q_2$  which assesses whether  $X = 1$  and  $Y = 1$  under  $\theta_t$ . We then calculate the conditional query by conditioning on the set of  $\theta$ s for which  $q_2$  is true and evaluating the share of these for which  $q_2$  is true (weighting by the probability of the causal types).

## 4.7 Appendix

We demonstrate how queries are calculated using the `CausalQueries` package for a chain model of the form  $X \rightarrow M \rightarrow Y$ . We imagine a model of this form in which we assume no negative effects of  $M$  on  $X$  or  $M$  on  $Y$ . We will also suppose that in fact  $X = 1$ , always. Doing this keeps the parameter space a little smaller for this demonstration but also serves to demonstrate that a causal model can make use of the counterfactual possibility that a node takes on a particular value even if it never does in fact.

We then ask two questions:

Q1. What is the probability that  $X$  causes  $Y$ ? (“POS”) Q2. What is the probability that  $X$  causes  $Y$  in cases in which  $X = 1$  and  $Y = 1$ ? (“POC”)

To answer these two queries we define simple query  $q_1$  which assesses whether  $X$  causes  $Y$  for each  $\theta$  and a second simple query  $q_2$  which assesses whether  $X = 1$  and  $Y = 1$  for each  $\theta$ . In this example the first simple query involves some `do` operations, the second does not.

The answers to these two simple queries are shown in the table below, where each row corresponds to a causal type.

```
model <- make_model("X -> M -> Y") %>%
  set_restrictions("X[] == 0") %>%
  set_restrictions("M[X=1] < M[X=0]") %>%
  set_restrictions("Y[M=1] < Y[M=0]")

q1 <- "Y[X = 1] > Y[X = 0]"
q2 <- "X == 1 & Y == 1"

df <- data.frame(
  a1 = CausalQueries:::map_query_to_causal_type(model, q1)$types,
  a2 = CausalQueries:::map_query_to_causal_type(model, q2)$types,
  p = get_type_prob(model))
```

The answer to the overall queries are then (1) the expected value of (the answers to)  $q_1$  and weights  $p$  and (2) the expected value of (the answers to)  $q_1$  given  $q_0$  and weights  $p$ :

Table 4.1: Set of causal types in the model that satisfy q1 and q2 along with the probability of the type.

	a1	a2	p
X1.M00.Y00	FALSE	FALSE	0.1111
X1.M01.Y00	FALSE	FALSE	0.1111
X1.M11.Y00	FALSE	FALSE	0.1111
X1.M00.Y01	FALSE	FALSE	0.1111
X1.M01.Y01	TRUE	TRUE	0.1111
X1.M11.Y01	FALSE	TRUE	0.1111
X1.M00.Y11	FALSE	TRUE	0.1111
X1.M01.Y11	FALSE	TRUE	0.1111
X1.M11.Y11	FALSE	TRUE	0.1111

Table 4.2: Calculated answers to two queries.

POS	POC
0.1111	0.2

```
df %>% summarize(POS = weighted.mean(a1, p),
                    POC = weighted.mean(a1[a2], p[a2]))
```

Given the equal weighting on causal types, these answers reflect the fact that for 5 of 9 causal types we expect to see  $X = 1$  and  $Y = 1$  but that; the causal effect is present for only 1 of 9 causal types and for 1 of the 5 causal types that exhibit  $X = 1$  and  $Y = 1$ .

# Chapter 5

## Bayesian Answers

Bayesian methods are just sets of procedures to figure out how to update beliefs in light of new information.

We begin with a prior belief about the probability that a hypothesis is true. New data then allow us to form a posterior belief about the probability of the hypothesis. Bayesian inference takes into account the consistency of the evidence with a hypothesis, the uniqueness of the evidence to that hypothesis, and background knowledge about the problem.

In the next section we review the basic idea of Bayesian updating. The following section applies it to the problem of updating on causal queries given a causal model and data.

### 5.1 Bayes Basics

For simple problems, Bayesian inference accords well with our intuitions. Once problems get slightly more complex however, our intuitions often fail us.

#### 5.1.1 Simple instances

Say I draw a card from a deck. The chances it is a Jack of Spades is just 1 in 52. If I tell you that the card is indeed a spade and asked you now what

are the chances it is a Jack of Spades, you should guess 1 in 13. If I told you it was a heart you should guess there is no chance it is a Jack of Spades. If I said it was a face card and a spade you should say 1 in 3.

All those answers are applications of Bayes' rule. In each case the answer is derived by assessing what is possible, given the new information, and then assessing how likely the outcome of interest among the states that are possible. In all the cases you calculate:

$$\text{Probability Jack of Spades} \mid \text{Information} = \frac{\text{Is Jack of Spades Consistent with Information}}{\text{How many cards are consistent with Information}}$$

The same logic goes through when things are not quite so black and white.

Now consider two slightly trickier examples.

**Interpreting Your Test Results.** Say that you take a test to see whether you suffer from a disease that affects 1 in 100 people. The test is good in the sense that if you have the disease it will say you have it with a 99% probability. If you do not have it, then with a 99% probability, it will say that you do not have it. The test result says that you have the disease. What are the chances you have it? You might think the answer is 99%, but that would be to mix up the probability of the result given the disease with the probability of the disease given the result. In fact the right answer is 50%, which you can think of as the share of people that have the disease among all those that test positive. The logic is most easily seen if you think through it using frequencies (see Hoffrage and Gigerenzer (1998) for this problem and ways to address it). If there were 10,000 people, then 100 would have the disease and 99 of these would test positive. But 9,900 would not have the disease and 99 of these would test positive. So the people with the disease that test positive are half of the total number testing positive.

As an equation this might be written:

$$\begin{aligned} \text{Probability You have the Disease} \mid \text{Test} &= \frac{\text{How many have the disease and test positive?}}{\text{How many test positive?}} \\ &= \frac{99}{99 + 99} \end{aligned} \tag{5.3}$$

**Two-Child Problem** Consider last an old puzzle described in Gardner (1961). *Mr Smith has two children, A and B. At least one of them is a boy. What are the chances they are both boys?* To be explicit about the puzzle, we will assume that the information that one child is a boy is given as a truthful answer to the question “is at least one of the children a boy?” Assuming that there is a 50% probability that a given child is a boy, people often assume the answer is 50%. But surprisingly the answer is 1 in 3. The information provided rules out the possibility that both children are girls and so the right answer is found by readjusting the probability that two children are boys based on this information. As an equation:

$$\text{Probability both are boys} \mid \text{Not both girls} = \frac{\text{Probability both boys}}{\text{Probability they are not both girls}} = \frac{1 \text{ in } 4}{3 \text{ in } 4}$$

### 5.1.2 Bayes’ Rule for Discrete Hypotheses

Formally, all of these equations are applications of Bayes’ rule which is a simple and powerful formula for deriving updated beliefs from new data.

The formula is given as:

$$\Pr(H|\mathcal{D}) = \frac{\Pr(\mathcal{D}|H) \Pr(H)}{\Pr(\mathcal{D})} \quad (5.4)$$

$$= \frac{\Pr(\mathcal{D}|H) \Pr(H)}{\sum_{H'} \Pr(\mathcal{D}|H') \Pr(H')} \quad (5.5)$$

where  $H$  represents a hypothesis,  $\mathcal{D}$  represents a particular realization of new data (e.g., a particular piece of evidence that we might observe), and the summation runs over an exhaustive and exclusive set of hypotheses.

Looking at the formula we see that the posterior belief derives from three considerations.

First the strength of our prior level of confidence in the hypothesis,  $\Pr(H)$ . The greater the prior likelihood that our hypothesis is true, the greater the chance that new data consistent with the hypothesis has *in fact* been generated by a state of the world implied by the hypothesis.

Second, the likelihood: how likely are we to have observed these data if the hypothesis were true,  $\Pr(\mathcal{D}|H)$ ?

Third, the probability of the data—the denominator. How likely are we to have observed these data regardless of whether the hypothesis is true or false,  $\Pr(\mathcal{D})$ ?

The last two quantities capture how consistent the data are with our hypothesis and how specific the data are to our hypothesis. As shown in the equation above, the third quantity can usefully be written in terms of the different ways (alternative Hypotheses,  $H'$ ) that the data could come about.

Note that answering the third question requires putting probabilities on an exclusive and exhaustive set of hypotheses. However, it does not require a listing of all possible hypotheses, just some exhaustive collection of hypotheses. For example we might have the notion of the probability that the accused’s fingerprints would be on the door if she were or were not guilty without having to decompose the “not guilty” into a set of hypotheses regarding who else might be guilty. Also, while the hypotheses that enter the formula do have to be mutually exclusive, that does not prevent you from drawing inferences about hypotheses that are not mutually exclusive. For instance you might form posteriors over which of 100 people is guilty, but then use the posterior to ask what is the probability that a man is guilty, or that an old person is guilty. The fact that “man” and “old” are not mutually exclusive in no way means you cannot try to learn about both of these hypotheses.

### 5.1.3 Bayes’ Rule for Continuous Parameters and The Dirichlet family

This basic formula extends in a simple way to collections of continuous variables. For example, say we are interested in the value of some parameter vector  $\theta$  (as a vector,  $\theta$  can contain many quantities we are uncertain about), we can calculate this, given a prior probability distribution over possible values of  $\theta$ ,  $p$ , and given data  $D$  as:

$$p(\theta|\mathcal{D}) = \frac{p(\mathcal{D}|\theta)p(\theta)}{\int_{\theta'} p(\mathcal{D}|\theta')p(\theta')d\theta'}$$

Bayes rule requires the ability to express a prior distribution but it does not require that the prior have any particular properties other than being probability distributions.

In practice however when we are dealing with continuous parameters, it can be useful to make use of “off the shelf” distributions.

For the framework developed in this book, we will often be interested in forming beliefs about the *share* of units that are of a particular type. For this type of question we will make quite heavy use of “Dirichlet” distributions: a family of distributions that capture beliefs about shares.

Consider, for example, the share of people in a population that voted; this is a quantity between 0 and 1. Two people might both believe that the turnout was around 50% but may differ in how certain they are about this claim. One might claim to have no information and to believe that any turnout rate between 0 and 100% is equally likely, giving an expected turnout of 50%; another might be completely confident that the number is 50% and entertain no other possibilities.

We can capture such beliefs quite well by using the Beta distribution—a special case of the Dirichlet. The Beta is a distribution over the  $[0, 1]$  that is governed by two parameters,  $\alpha$  and  $\beta$ . In the case in which both  $\alpha$  and  $\beta$  are 1, the distribution is uniform – all values are seen as equally likely. As  $\alpha$  rises large outcomes are seen as more likely and as  $\beta$  rises, lower outcomes are seen as more likely. If both rise proportionately the expected outcome does not change but the distribution becomes tighter.

An attractive feature of the Beta distribution is that if one has a prior  $\text{Beta}(\alpha, \beta)$  over the probability of some event (e.g. that a coin comes up heads), and then one observes a positive case, the Bayesian posterior distribution is also a Beta with parameters  $\alpha + 1, \beta$ . Thus in a sense if people start with uniform priors and build up knowledge on seeing outcomes, their posterior beliefs should be Beta distributions.

Figure 5.1 shows a set of such distributions, starting with one that has greater variance than uniform (with alpha and beta both set to 0.5 this corresponds to the non informative “Jeffrey’s prior”), and including the uniform case (both alpha and beta set to 1), then cases in which multiple negative and positive outcomes have been seen.

Dirichlet distributions generalize the Beta to the situation in which there are

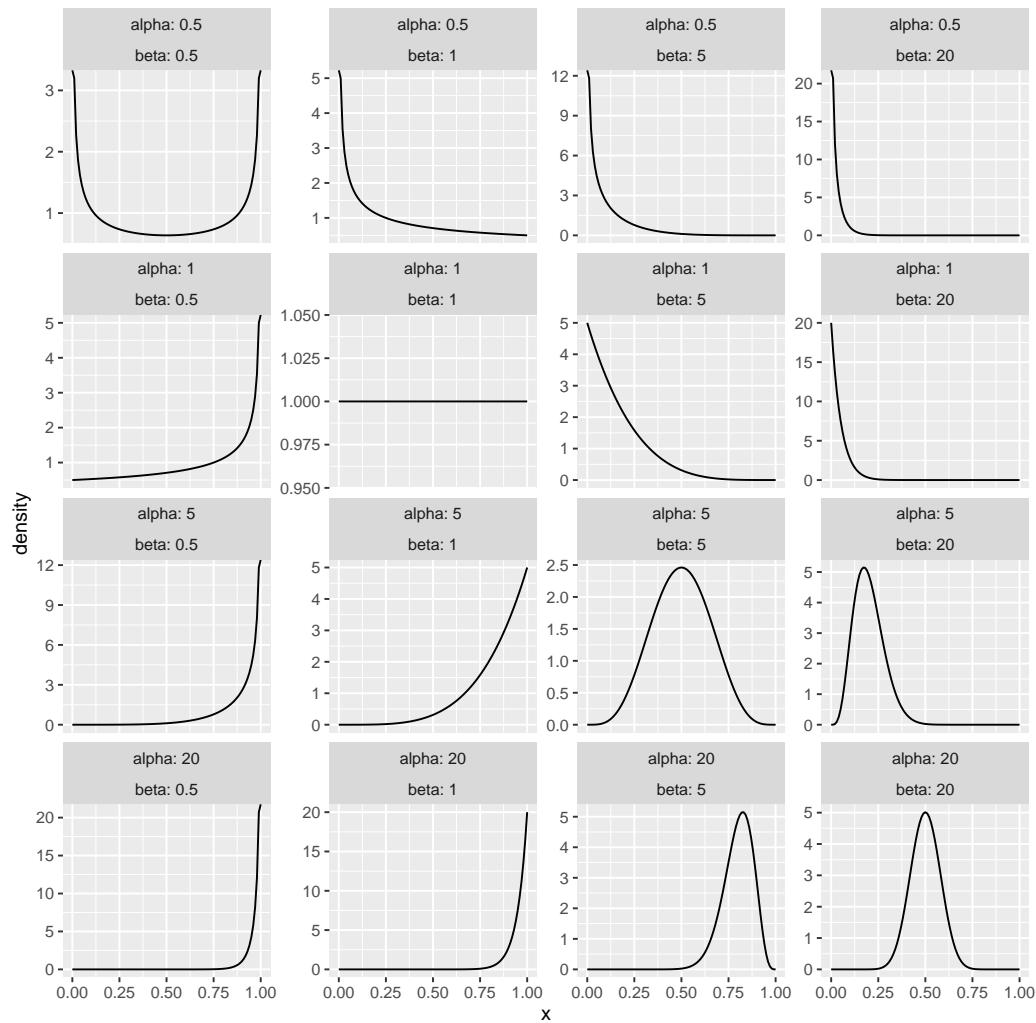


Figure 5.1: Beta distributions

beliefs not just over a proportion, or a probability, but over collections of probabilities. For example, if four outcomes are possible and each is likely to occur with probability  $\theta_k$ ,  $k = 1, 2, 3, 4$ , then beliefs about these probabilities are distributions over a three-dimensional unit simplex—that is, all 4 element vectors of probabilities that add up to 1. The distribution has as many parameters as there are outcomes and these are traditionally recorded in a vector,  $\alpha$ . Similar to the Beta distribution, an uninformative prior (Jeffrey's prior) has  $\alpha$  parameters of (.5, .5, .5, ...) and a uniform (“flat”) distribution has  $\alpha = (1, 1, 1, \dots)$ .

As with the Beta distribution, the Dirichlet updates in a simple way. If you have a Dirichlet prior with parameter  $\alpha = (\alpha_1, \alpha_2, \dots)$  and you observe outcome 1, for example, then the posterior distribution is also Dirichlet with parameter vector  $\alpha' = (\alpha_1 + 1, \alpha_2, \dots)$ .

#### 5.1.4 Moments

In what follows we often refer to the “posterior mean” or the “posterior variance.” These are simply summary statistics of the posterior distribution, or moments, and can be calculated easily once the posterior is known. For example the posterior mean of a parameter  $\theta_1$ —just one in a collection of parameters stored in  $\theta$ —is simply  $\bar{\theta}_1|\mathcal{D} = \int \theta_1 p(\theta|\mathcal{D})d\theta$ . Note importantly that this is calculated using the posterior over the entire vector  $\theta$ . Similarly the posterior variance is  $\int (\theta_1 - (\bar{\theta}_1|\mathcal{D}))^2 p(\theta|\mathcal{D})d\theta$ .

#### 5.1.5 Bayes estimation in practice

Although the principle of Bayesian inference is quite simple, in practice calculating posteriors for continuous parameters is computationally complex.

In principle, with continuous parameters there is an infinity of possible parameter values. Analytic solutions are not, in general, easy to come by and so researchers use some form of sampling.

Imagine for instance you were interested in forming a posterior on the share of U.S. voters intending to vote Democrat, given polling data. (This is not truly continuous, but with large elections it might as well be.)

One approach is to coarsen the parameter space: we calculate the probability of observing the polling data given possible values  $\theta = 0, \theta = .1, \theta = .2, \dots, \theta = 1$ , and, apply Bayes rule to form a posterior for each of these possibilities. The downside of this approach is that, for a decent level of precision, it becomes computationally expensive with large parameter spaces, and parameter spaces get large quickly. For instance, if we are interested in vote shares, we might find .4, .5, and .6 too coarse and want posteriors for 0.51 or even 0.505; the latter would require calculations for 200 parameter values. If we had *two* parameters that we wanted to slice up each into 200 possible values, we would then have 40,000 parameter pairs to worry about. What's more, *most* of those calculations would not be very informative if the real uncertainty all lies in some small (though possibly unknown) range – such as between 40% and 60%.

An alternative approach is to use variants of Markov Chain Monte Carlo sampling. Under these approaches, parameter vectors are sampled and their likelihood is evaluated. If a sampled parameter vector is found to have high likelihood, then new parameter vectors near it are drawn with a high probability. Based on the likelihood associated with these new draws, additional draws are made. The result is a chain of draws that build up to approximate the posterior distribution. The output from these procedures is not a set of probabilities for each possible parameter vector but rather a set of draws of parameter vectors from the posterior distribution.

Many algorithms have been developed to achieve these tasks efficiently; in all of our applications we rely on the `stan` procedures which use MCMC methods, specifically the Hamiltonian Monte Carlo (HMC) algorithm and the no-U-turn sampler (NUTS). Details on these approaches are given in the Stan Reference Manual (Stan et al., 2020).

## 5.2 Bayes applied

### 5.2.1 Simple Bayesian Process Tracing

Process tracing in its most basic form seeks to use within-case evidence to draw inferences about a case. For example, with a focus on whether  $X$  caused  $Y$ , data on a within-case “clue,”  $K$ , is used to make inference about whether

or not the outcome in that case was generated by the case’s treatment status on  $X$ . We refer to the within-case evidence gathered during process tracing as *clues* in order to underline their probabilistic relationship to the causal relationship of interest. Readers familiar with the framework in Collier et al. (2004) can usefully think of our “clues” as akin to causal process observations, although we highlight that there is no requirement that the clues be generated by the causal process.

To make inferences, the analyst looks for clues that will be observed with some probability if the case is of a given type and that will *not* be observed with some probability if the case is *not* of that type.

It is relatively straightforward to express the logic of process tracing in Bayesian terms, a step that will aid the integration of qualitative with quantitative causal inferences. As noted by others (e.g. Bennett (2008), Beach and Pedersen (2013), Rohlfing (2012)), there is an evident connection between the use of evidence in process tracing and Bayesian inference. See Fairfield and Charman (2017) for a detailed treatment of Bayesian approach in qualitative research.

To illustrate, suppose we are interested in regime collapse. We already have  $X, Y$  data on one authoritarian regime: we know that it suffered economic crisis ( $X = 1$ ) and collapsed ( $Y = 1$ ). We want to know what caused the collapse. To make progress we will try to draw inferences given a “clue.” Beliefs about the probabilities of observing clues for cases with different causal effects derive from theories of, or evidence about, the causal process connecting  $X$  and  $Y$ . Suppose we theorize that the mechanism through which economic crisis generates collapse runs via diminished regime capacity to reward its supporters during an economic downturn. A possible clue to the operation of a causal effect, then, might be the observation of diminishing rents flowing to regime supporters shortly after the crisis. If we believe the theory, then this is a clue that we might believe to be highly probable for cases of type  $b$  that have experienced economic crisis (where the crisis in fact caused the collapse) but of low probability for cases of type  $d$  that have experienced crisis (where the collapse occurred for other reasons).

To make use of Bayes rule we need to:

1. define our parameters, which are the key quantities of interest
2. provide prior beliefs about the parameters of interest

3. define a likelihood function
4. provide the probability of the data
5. plug these into Bayes' rule to calculate a posterior on the parameters of interest

We discuss each of these in turn, using our  $a, b, c, d$  type notation for simplicity.

**Parameters.** The inferential challenge is to determine whether the regime collapsed *because* of the crisis (it is  $b$  type) or whether it would have collapsed even without it ( $d$  type). We do so using further information from the case—one or more clues. We use the variable  $K$  to register the outcome of the search for a clue, with  $K=1$  indicating that a specific clue is searched for and found, and  $K=0$  indicating that the clue is searched for and not found.

Let  $j \in \{a, b, c, d\}$  refer to the type of an individual case. Our hypothesis, in this initial setup, consists simply of a belief about  $j$  for the case under examination: specifically whether the case is a  $b$  type ( $j = b$ ). The parameter of interest is the causal type.

**Prior.** We then assign a prior degree of confidence to the hypothesis ( $p = \Pr(H)$ ). This is, here, our prior belief that an authoritarian regime that has experienced economic crisis is a  $b$ .

**Likelihood.** The likelihood,  $\Pr(K = 1|H)$  is the probability of observing the clue, when we look for it in our case, if the hypothesis is true—i.e., here, if the case is a  $b$  type. The key feature of a clue is that the probability of observing the clue is believed to depend on the case's causal type. In order to calculate the probability of the data we will in fact need two such probabilities: we let  $\phi_b$  denote the probability of observing the clue for a case of  $b$  type ( $\Pr(K = 1|j = b)$ ), and  $\phi_d$  the probability of observing the clue for a case of  $d$  type ( $\Pr(K = 1|j = d)$ ). The key idea in many accounts of process tracing is that the *differences* between these probabilities provides clues with “probative value,” that is, the ability to generate learning about causal types. The likelihood,  $\Pr(K = 1|H)$ , is simply  $\phi_b$ .

**Probability of the data.** This is the probability of observing the clue when we look for it in a case, *regardless* of its type, ( $\Pr(K = 1)$ ). More specifically, it is the probability of the clue in a treated case with a positive outcome. As such a case can only be a  $b$  or a  $d$  type, this probability can be calculated simply from  $\phi_b$  and  $\phi_d$ , together with our beliefs about how

likely an  $X = 1, Y = 1$  case is to be a  $b$  or a  $d$  type. This probability aligns (inversely) with Van Evera's concept of "uniqueness."

**Inference.** We can now apply Bayes' rule to describe the learning that results from process tracing. If we observe the clue when we look for it in the case, then our *posterior* belief in the hypothesis that the case is of type  $b$  is:

$$\Pr(j = b|K = 1, X = Y = 1) = \frac{\phi_b p}{\phi_b p + \phi_d(1 - p)}$$

In this exposition we did not make use of a causal model in a meaningful way—we simply need the priors and the clue probabilities.

In fact, however, these numbers can be derived from a causal model. To illustrate, imagine a simple causal model in which the  $X, Y$  relationship is completely mediated by  $K$ . In particular, suppose, from background knowledge of the conditional distribution of outcomes given their causes, we have that:

- $\Pr(K = 1|X = 0) = 0, \Pr(K = 1|X = 1) = .5$
- $\Pr(Y = 1|K = 0) = .5, \Pr(Y = 1|K = 1) = 1$

This data is consistent with a world in which half  $b$  and  $c$  types in the first step and half  $b$  and  $d$  types in the second step. Assume that the case at hand is sampled from this world.

Then we can calculate that the prior probability,  $p$ , that  $X$  caused  $Y$  given  $X = Y = 1$  is  $p = \frac{1}{3}$ .<sup>1</sup> We can also calculate the probability that  $K = 1$  for a treated  $b$  and  $d$  case respectively as  $\phi_b = 1$  and  $\phi_d = 0.5$  (convince yourself of these numbers!). We then get:

$$\Pr(j = b|K = 1, X = Y = 1) = \frac{1 \times \frac{1}{3}}{1 \times \frac{1}{3} + 0.5 \times \frac{2}{3}} = 0.5$$

---

<sup>1</sup>Given  $X = 1, Y = 1$  is consistent with  $b$  types at both stages, which arises with probability .25, or with a  $d$  type in the second stage, which arises with probability .5. The conditional probability is therefore  $.25/.75 = 1/3$ .

We thus shift our beliefs from a prior of  $\frac{1}{3}$  to a posterior of  $\frac{1}{2}$ . In contrast had we *not* observed the clue our posterior would have been 0.

As should be clear from the above, the inferential leverage in process tracing comes from differences in the probability of observing  $K = 1$  for different causal types. Thus, the logic described here generalizes Van Evera's familiar typology of tests by conceiving of the certainty and uniqueness of clues as lying along a continuum.

Van Evera's four tests ("smoking gun," "hoop," "straw in the wind," and "doubly decisive") represent, in this sense, special cases—particular regions that lie on the boundaries of a "probative-value space." To illustrate the idea, we represent the range of combinations of possible probabilities for  $\phi_b$  and  $\phi_d$  as a square in Figure ??CluesInferences1) and mark the spaces inhabited by Van Evera's tests. As can be seen, the type of test involved depends on both the relative *and* absolute magnitudes of  $\phi_b$  and  $\phi_d$ . The probative value of a test depends on the difference between them. Thus, a clue acts as a smoking gun for proposition " $b$ " (the proposition that the case is a  $b$  type) if it is highly unlikely to be observed if proposition  $b$  is false, and more likely to be observed if the proposition is true (bottom left, above diagonal). A clue acts as a "hoop" test if it is highly likely to be found if  $b$  is true, even if it is still quite likely to be found if it is false. Doubly decisive tests arise when a clue is very likely if  $b$  and very unlikely if not. It is, however, also easy to imagine clues with probative qualities lying in the large space amidst these extremes.<sup>2</sup>

In this illustration, we note that we draw both the priors and the probative value from a causal model. If we altered the model—for example, if we had a stronger first stage and so a larger value for  $\Pr(K = 1|X = 0)$ —this would alter both our prior,  $p$ , and our calculations of  $\phi_d$ . An implication of this is that, although one might be tempted to think of the priors and the probative values as independent quantities, and contemplate how inferences change as priors change (as we did for example in the treatment in Humphreys and Jacobs (2015)), keeping probative value fixed, that kind of thought experiment may assume values that are not justified by an underlying model.

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<sup>2</sup>We thank Tasha Fairfield for discussions around this graph which differs from that in Humphreys and Jacobs (2015) by placing tests more consistently on common rays originating from (0,0) and (1,1).

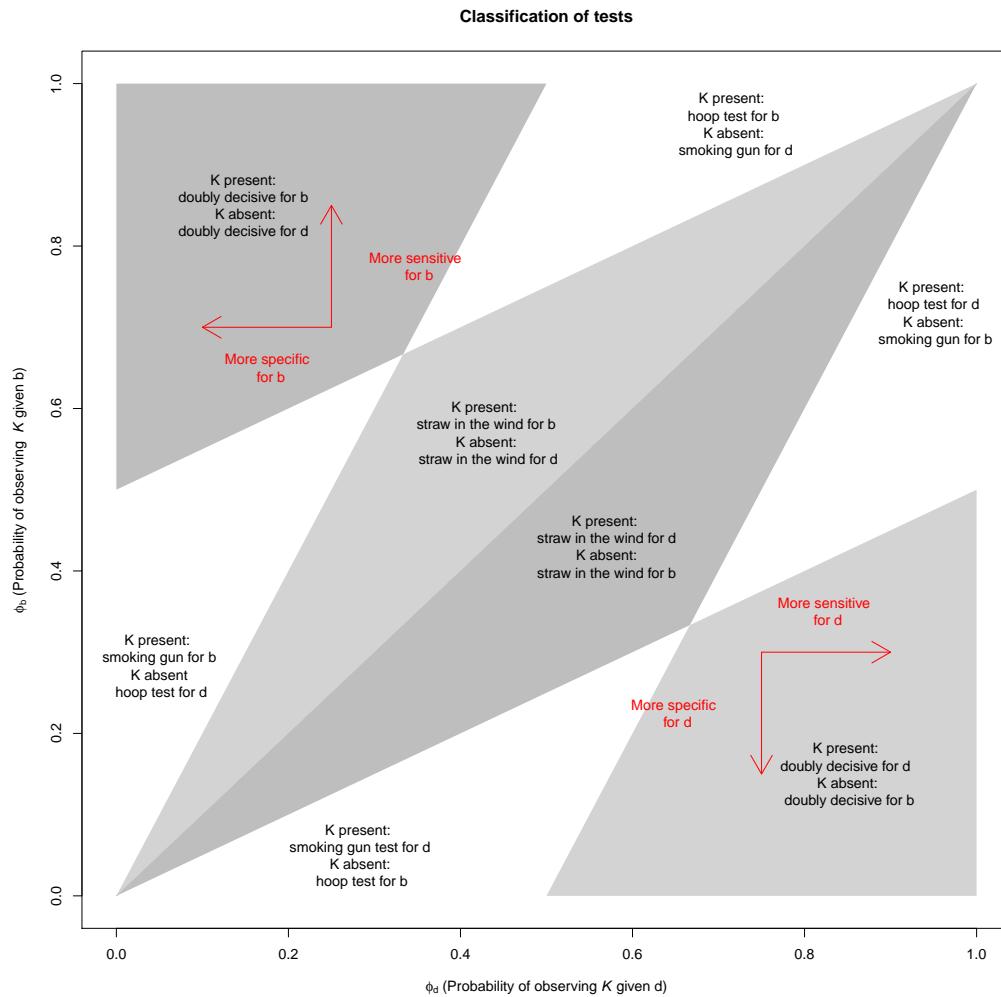


Figure 5.2: A mapping from the probability of observing a clue if the proposition that a case is a  $b$  type is true ( $\phi_b$ ) or false ( $\phi_d$ ) to a generalization of the tests described in Van-Evera (1997).

### 5.2.2 A Generalization: Bayesian Inference on Queries

In Chapter 4, we described queries of interest as queries over nodal types in causal models.

Once queries are defined in terms of nodal types, the formation of beliefs, given data  $W$ , about queries follows immediately from application of Bayes rule.

Let  $Q(\theta)$  define the value of the query in context  $\theta$ . The updated beliefs about the query are given by the distribution:

$$P(q|W) = \int_{\theta:Q(\theta)=q} P(\theta|W)d\theta = \int_{\theta:Q(\theta)=q} \frac{P(W|\theta)P(\theta)}{\int_{\theta'} P(W|\theta')P(\theta')d\theta'} d\theta$$

This expression gathers together all the causal types (combinations of nodal types) that satisfy a query and assesses how likely these are, collectively, given the data.<sup>3</sup> For an abstract representation of the relations between assumptions, queries, data, and conclusions, see Figure 1 in Pearl (2012).

Return now to Mr Smith’s puzzle. We can think of the two “nodal types” here as the sexes of the two children, child  $A$  and child  $B$ . The query here is  $Q$ : “Are both boys?” The statement “ $Q = 1$ ” is equivalent to the statement,  $A$  is a boy &  $B$  is a boy. Thus it takes the value  $q = 1$  under just one causal type, when both nodes have been assigned to the value “boy.” Statement  $q = 0$  is the statement (“ $A$  is a boy &  $B$  is a girl” or “ $A$  is a girl &  $B$  is a boy” or “ $A$  is a girl &  $B$  is a girl”). Thus  $q = 0$  in three contexts. If we assume that each of the two children is equally likely to be a boy or a girl with independent probabilities, then each of the four contexts is equally likely. The result can then be figured out as  $P(Q = 1) = \frac{1 \times \frac{1}{4}}{1 \times \frac{1}{4} + 1 \times \frac{1}{4} + 1 \times \frac{1}{4} + 0 \times \frac{1}{4}} = \frac{1}{3}$ . This answer requires summing over only one causal type.  $P(Q = 0)$  is of course the complement of this, but using the Bayes formula one can see that it can be found by summing over the posterior probability of three causal types in which the statement  $Q = 0$  is true.

We will often want to think about our causal queries being collections of states of the world — i.e., of causal types. Returning to our discussion of

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<sup>3</sup>Learning about roots from observed data is sometimes termed *abduction*; see Pearl (2009), p 206.

queries in Chapter 4, suppose we start with the model  $X \rightarrow M \rightarrow Y$ , and our query is whether  $X$  has a positive effect on  $Y$ . This is a query that is satisfied by four sets of causal types: those in which  $X$  has a positive effect on  $M$  and  $M$  has a positive effect on  $Y$ , with  $X$  being either 0 or 1; and those in which  $X$  has a negative effect on  $M$  and  $M$  has a negative effect on  $Y$ , with  $X$  being either 0 or 1. Our inferences on the query will thus involve gathering these different causal types, and their associated posterior probabilities, together.

One way to think intuitively about Bayesian updating is to say that we update more strongly in favor of the hypothesis for which the evidence is *least* damaging to the *most-likely ways* in which the hypothesis could be true. Suppose our prior belief was that it was much more unlikely that  $M$  had a negative effect on  $Y$ , than that  $M$  had a positive effect on  $Y$ . This makes one of the ways in which  $X$  could have a positive effect on  $Y$  (the chain of negative effects) much less likely than the other way in which  $X$  could have a positive effect on  $Y$  (the chain of positive effects). This means that evidence against a chain of negative effects and evidence against a chain of positive effects will not be equally consequential for our query: in particular, we will update more strongly against the query if we find evidence against a chain of positive effects than if we find evidence against a chain of negative effects. Evidence against a chain of positive effects speaks against the *most* likely way in which the query could be true, whereas evidence against a chain of negative effects speaks against a way the query could be true that we did not think was very likely to begin with.

## 5.3 Features of Bayesian updating

Bayesian updating has implications that may not be obvious at first glance. These will matter for all forms of inference we examine in this book, but they can all be illustrated in simple settings.

### 5.3.1 Priors matter

The amount of learning that results from a given piece of new data depends strongly on prior beliefs. We have already seen this with the example of

interpreting our test results above. Figure 5.3 illustrates the point for process tracing inferences.

In each subgraph of Figure 5.3 , we show how much learning occurs under different scenarios. The horizontal axis indicates the level of prior confidence in the hypothesis and the curve indicates the posterior belief that arises if we do (or do not) observe the clue. As can be seen, the amount of learning that occurs—the shift in beliefs from prior to posterior—depends a good deal on what prior we start out with. For a smoking gun test, the amount of learning is highest for values roughly in the 0.2 to 0.4 range—and then declines as we have more and more prior confidence in our hypothesis. For a hoop test, the amount of learning when the clue is *not* observed is greatest for hypotheses in which we have middling-high confidence (around 0.6 to 0.8), and minimal for hypotheses in which we have a very high or a very low level of confidence.

The implication here is that our inferences with respect to a hypothesis must be based not just on the search for a clue predicted by the hypothesis but also on the *plausibility* of the hypothesis, based on other things we know. Suppose, for instance, that we fail to observe evidence that we are 90 percent sure we *should* observe if a hypothesized causal effect has occurred: a strong hoop test is failed. But suppose that the existing literature has given us a very high level of confidence that the hypothesis *is* right. This high prior confidence, sometimes referred to as a “base rate,” is equivalent to believing that the causal effect exists in a very high proportion of cases. Thus, while any given case with a causal effect has only a 0.1 chance of not generating the clue, the high base rate means that the vast majority of cases that we observe without the clue will nonetheless be cases with causal effects. Thus, the failure of even a strong hoop test, involving a highly certain prediction, should only marginally reduce our confidence in a hypothesis that we strongly expect to be true.

A similar line of reasoning applies to smoking gun tests involving hypotheses that prior evidence suggests are very unlikely to be true. Innocent people may be very unlikely to be seen holding smoking guns after a murder. But if a very high proportion of people observed are known to be innocent, then a very high proportion of those holding smoking guns will in fact be innocent—and a smoking-gun clue will be far from decisive.

We emphasize two respects in which these implications depart from common intuitions. First, we cannot make *general* statements about how decisive

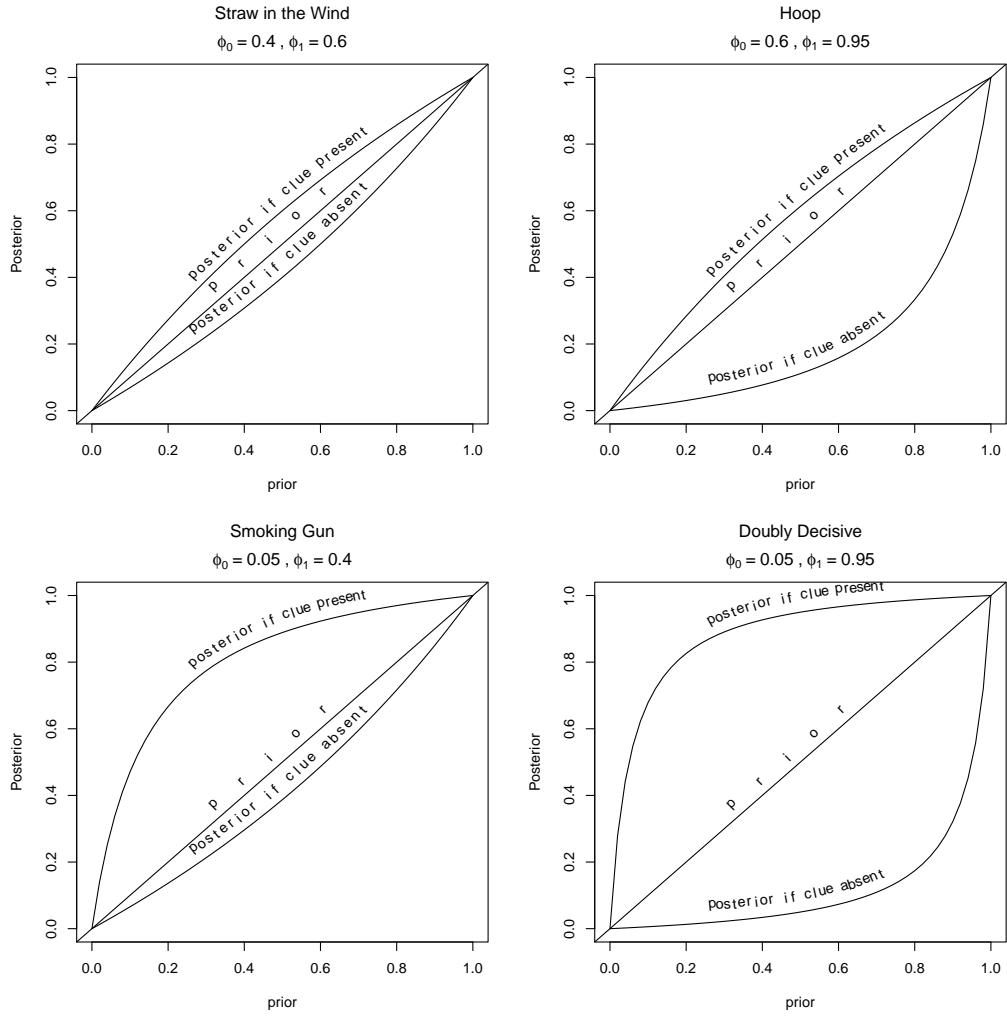


Figure 5.3: Figure shows how the learning from different types of tests depends on priors regarding the proposition. A smoking gun test has the greatest impact on beliefs when priors are middling low and the clue is observed; a “hoop test” has the greatest effect when priors are middling high and the clue is not observed.

different categories of test, in Van Evera's framework, will be. It is commonly stated that hoop tests are devastating to a theory when they are failed, while smoking gun tests provide powerful evidence in favor of a hypothesis. But, in fact the amount learned depends not just on features of the clues but also on prior beliefs.

Second, although scholars frequently treat evidence that goes against the grain of the existing literature as especially enlightening, in the Bayesian framework the contribution of such evidence may sometimes be modest, precisely because received wisdom carries weight. Thus, although the discovery of *disconfirming* evidence—an observation thought to be strongly inconsistent with the hypothesis—for a hypothesis commonly believed to be true is more informative (has a larger impact on beliefs) than *confirming* evidence, this does not mean that we learn more than we would have if the prior were weaker. But it is not true as a general proposition that we learn more the bigger the “surprise” a piece of evidence is. The effect of disconfirming evidence on a hypothesis about which we are highly confident will be *smaller* than it would be for a hypothesis about which we are only somewhat confident. When it comes to very strong hypotheses, the “discovery” of disconfirming evidence is very likely to be a false negative; likewise, the discovery of supporting evidence for a very implausible hypothesis is very likely to be a false positive. The Bayesian approach takes account of these features naturally.<sup>4</sup>

### 5.3.2 Simultaneous, joint updating

When we update we often update over multiple quantities. When we see a smoking gun, for instance, we might update our beliefs that the butler did it, but we might also update our beliefs about how likely we are to see smoking guns – maybe they are not as rare as we thought!

Intuitively we might think of this updating as happening sequentially – first of all, we update over the general proposition, then we update over the particular claim. But in fact we update over both quantities at once.

Here we elaborate on the intuition using an example of Bayesian process tracing, in which updating occurs over both the nodal type ( $j$ ) and beliefs

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<sup>4</sup>We note, however, that one common intuition—that little is learned from disconfirming evidence on a low-plausibility hypothesis or from confirming evidence on a high-plausibility one—is correct.

about the probabilities with which clues are observed for each type ( $\phi$  values).

Suppose that we observe a case with values  $X = 1, Y = 1$ . We begin by defining a prior probability distribution over each parameter. Suppose that we establish a prior categorical distribution reflecting uncertainty over whether the case is a  $b$  type (e.g., setting a probability of 0.5 that it is a  $b$  and 0.5 that is a  $d$  type). We also start with priors on  $\phi_b$  and  $\phi_d$ . For concreteness, suppose that we are certain that the clue is unlikely for a  $d$  type ( $\phi_d = .1$ ), but we are very uncertain about  $\phi_b$ ; in particular, we have a uniform prior distribution over  $[0, 1]$  for  $\phi_b$ . Note that, even though we are very uncertain about  $\phi_b$ , the clue still has probative value, arising from the fact that the expected value of  $\phi_b$  is higher than that of  $\phi_d$ .

Suppose that we then look for the clue in the case and observe it. This observation shifts posterior weight away from a belief that the case is a  $b$ . See Figure 5.4 for an illustration. Yet it *simultaneously* shifts weight toward a higher value for  $\phi_b$  and a lower value for  $\phi_d$ . The reason is that the observed clue has a relatively high likelihood *both* for combinations of parameter values in which the case is a  $d$  and  $\phi_b$  is low *and* for combinations in which the case is a  $b$  and  $\phi_b$  is *high* (or, equivalently, in this example, where  $\phi_d$  is low). The marginal posterior distribution of  $\phi_b$  will thus be shifted upward relative to its prior marginal distribution. The joint posterior distribution will also reflect a dependency between the probability that the case is a  $b$  vs. a  $d$ , on the one hand, and  $\phi_b$  and  $\phi_d$  on the other.

Figure 5.5 provides a second example, this time showing the joint distribution for the belief that  $X$  causes  $Y$  in cases in which  $X = 1, Y = 1$  and the relative likelihood that  $K = 1$  when  $X$  causes  $Y$  (and  $X = 1, Y = 1$ ) relative to the likelihood that  $K = 1$  when  $X$  does not cause  $Y$  (and  $X = 1, Y = 1$ ). In this case the joint posterior is derived from a model in which probative value of  $K$  is inferred from data, rather than assumed by researchers. We develop more models of this form in later chapters but for now highlight only that in simple setups the distribution of beliefs over queries and over the informativeness of clues are likely not independent of each other.

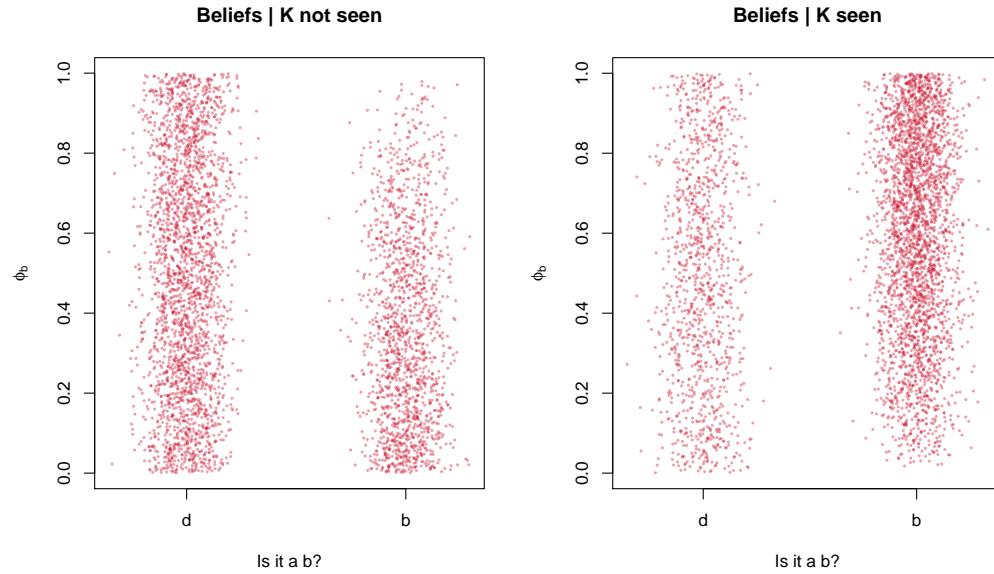


Figure 5.4: Joint posteriors distribution on whether a case is a *b* or *d* and on the probability of seeing a clue for a *b* type ( $\phi_b$ ).

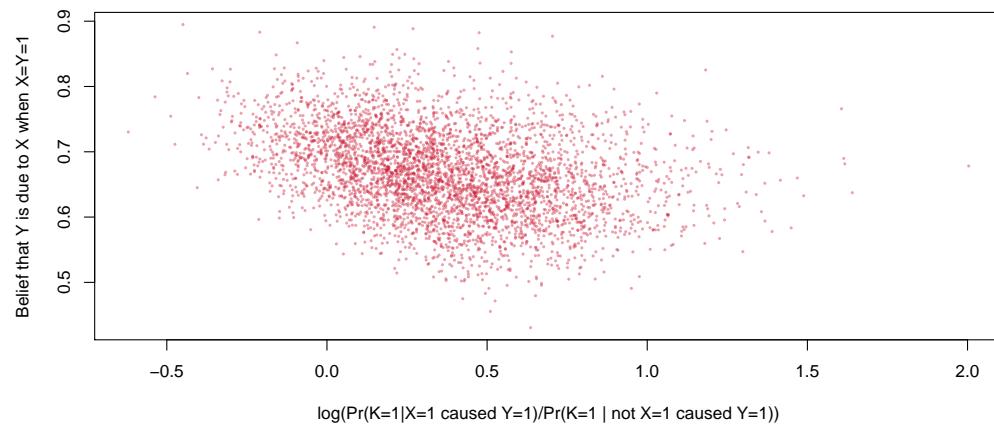


Figure 5.5: Correlated beliefs on queries and probative value

### 5.3.3 Posteriors are independent of the ordering of data

We often think of learning as a process in which we start off with some set of beliefs—our priors—we gather data,  $D_1$ , and update our beliefs, forming a posterior; we then observe new data and we update again, forming a new posterior, having treated the previous posterior as a new prior. In such cases it might seem natural that it would matter which data we saw first and which later.

In fact, however, Bayesian updating is blind to ordering. If we learn first that a card is a face card and second that it is black, our posteriors that the card is a Jack of Spades go from 1 in 52 to 1 in 12 to 1 in 6. If we learn first that the card is black and second that it is a face card, our posteriors that it is a Jack of Spades go from 1 in 52 to 1 in 26 to 1 in 6. We end up in the same place in both cases. And we would have had the same conclusion if we learned in one go that the card is a black face card.

The math here is easy enough. Our posterior given two sets of data  $D_1, D_2$  can be written:

$$p(\theta|D_1, D_2) = \frac{p(\theta, D_1, D_2)}{p(D_1, D_2)} = \frac{p(\theta, D_1|D_2)p(D_2)}{p(D_1|D_2)p(D_2)} = \frac{p(\theta, D_1|D_2)}{p(D_1|D_2)}$$

or, equivalently:

$$p(\theta|D_1, D_2) = \frac{p(\theta, D_1, D_2)}{p(D_1, D_2)} = \frac{p(\theta, D_2|D_1)p(D_1)}{p(D_2|D_1)p(D_1)} = \frac{p(\theta, D_2|D_1)}{p(D_2|D_1)}$$

In other words our posteriors given both  $D_1$  and  $D_2$  can be thought of as the result of updating on  $D_2$  given we already know  $D_1$  or the result of updating on  $D_1$  given we already know  $D_2$ .

This fact will be useful in applications. In practice we might assume that we have beliefs based on background data  $D_1$ , for example regarding general relations between  $X$  and  $Y$  and a flat prior, and we then update again with new data on  $K$ . Rather than updating twice, the fact that updating is invariant to order means that we can assume a flat prior and update once given data on  $X$ ,  $Y$ , and  $K$ .



# Chapter 6

## Theories as causal models

In Chapter 3, we described a set of theories and represented them as causal models. But so far we haven't been very explicit in what we mean by a theory or how theory maps onto a causal-model framework.

In this book, we will think of theory as a type of *explanation*: a theory provides an account of how or under what conditions a set of causal relationships operate. We generally express both a theory and the claims being theorized as causal models. The theory is then a model that *implies* another model—possibly with the help of some data.

To fix ideas: a simple claim might be that “ $A$  caused  $B$  in case  $j$ ”. This claim is itself a model, albeit a very simple one. The theory that supports this model might be of the form “ $A$  always causes  $B$ ”, “ $A$  always causes  $B$  whenever  $C$  (and  $C$  holds in case  $j$ )”, or “ $A$  invariably causes  $B$  and invariably  $B$  causes  $C$ ”. These all have in common that they are arguments that could be provided to support the simple claim; in each case, if you believe the theory you believe the implication.

The rest of this short chapter builds out this idea and uses it to provide a way of characterizing when a theory is useful or not. In the first section, we consider multiple senses in which one model might imply, and thus serve as a *theory of*, another model. For one thing, we consider how one causal structure can imply (theorize) another causal structure, by including additional new nodes and nodal types that explain how or when causal effects in the original model will unfold. Next, we consider how the causal-type *ranges* of models can relate to one another: one model can imply another model when

the former’s causal types constitute a subset of the latter’s. In this situation, the theory represents a more specific, stronger claim about the kinds of causal effects that are operating. We then turn to logical relations between probabilistic models. We show how the distributions over nodal types in a simpler model structure can be underwritten by distributions over nodal types in a more detailed model structure. Here, a claim about the prevalence (or probability) of causal effects in a causal network is justified by claims about the prevalence or probability of causal effects in a more granular rendering of that network. Finally, we show how a probabilistic model plus *data* can provide a theoretical underpinning for a new, stronger model.

Second, we consider how theories-as-models can be useful. In embedding theorization within the world of causal models, we ultimately have an empirical objective in mind. Theorizing a causal relationship of interest, in our framework, means elaborating our causal beliefs about the world in greater detail. As we show in later chapters, theorizing in the form of a causal model allows us to generate research designs: to identify sources of inferential leverage and to explicitly and systematically link observations of components of a causal system to the causal questions we seek to answer. In the second section of this chapter, however, we provide a high-level conceptualization of the empirical gains from theory.

In the chapter’s third and final section, we show how our formalization of theory maps onto *formal* theory as usually understood, showing how we can generate a causal model from a game-theoretic model.

## 6.1 Models as *theories of*

Let us say that a causal model,  $M'$ , is a *theory of*  $M$  if  $M$  is implied by  $M'$ . It is a theory *because* it has implications. Otherwise it is a conclusion, an inference, or claim. A theory,  $M'$ , might itself sit atop—be supported by—another theory,  $M''$ , that implies  $M'$ . To help fix the idea of theory as “supporting” or “underlying” the model(s) it theorizes, we refer to the theory,  $M'$ , as a *lower-level* model relative to  $M$  and refer to  $M$  as a *higher-level* model relative to its theorization,  $M'$ .<sup>1</sup>

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<sup>1</sup>We note that our definition of theory differs somewhat from that given in Pearl (2009) (p207): there a theory is a (functional) causal model and a restriction over  $\times_j \mathcal{R}(U_j)$ , that

Both structural models and probabilistic models—possibly in combination with data—imply other models. We discuss each in turn.

### 6.1.1 Implications of structural causal models

Structural models can imply multiple other simpler structural models. Similarly structural models can be implied by multiple more involved models.

We imagine two forms of lower level model, those that involve “type splintering” and those that involve “type reduction.”

**Type splintering theorization.** Theorization often involves a refinement of causal types, implemented through the addition of nodes. Take the very simple model,  $M$ , represented in Figure 6.1(a). The model simply states that  $X$  has (or *can* have) a causal effect on  $Y$ .

What theories might justify  $M$ ? This question can be rephrased as “what models imply model  $M$ ?”. The figure points to two possibilities. Both models  $M'$  and  $M''$  imply model  $M$ . They can be thought of as *theories*, or lower-level model, of  $M$ .

Model  $M'$  differs by the addition of a node,  $K$ , in the causal chain between  $X$  and  $Y$ . We can say that  $M'$  is a *theory* of  $M$  for two reasons. First it provides a *justification*—if you believe  $M'$  you should believe  $M$ : if  $X$  affects  $Y$  through  $K$ , then  $X$  affects  $Y$ . But as well as a justification it also provides an *explanation* of  $M$ . Suppose we already *know* that  $X$  affects  $Y$  but want to know *why*. If we ask, “why does  $X$  affect  $Y$ ?”,  $M'$  provides an answer:  $X$  affects  $Y$  because  $X$  affects  $K$ , and  $K$  affects  $Y$ .

Model  $M''$  differs by the addition of a node,  $C$ , that moderates the effect of  $X$  on  $Y$ .  $M''$  justifies  $M$  in the sense that if you believe  $M''$  you should believe  $M$ . It provides an explanation of a kind also: if you believe model  $M''$  you likely believe that the relation between  $X$  and  $Y$  is what it is because of  $C$ . Had  $C$  been different the causal relation between  $X$  and  $Y$  might have been also.

A key idea is that both  $M'$  and  $M''$  involve a redefinition of  $\theta^Y$ . That is we see a change in the endogenous nodes but these in turn imply a change in

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is, over the collection of contexts envisionable. Our definition also considers probabilistic models as theories, allowing statements such as “the average effect of  $X$  on  $Y$  is 0.5.”

the interpretation of the exogenous nodes pointing into existing endogenous nodes (such as  $Y$  in this example). We can think of part of  $\theta^Y$  being splintered off and captured by  $\theta^K$  or  $C$ .

Return to models  $M$  and  $M'$  in Figure 6.1(a). Importantly, in moving from the higher- to the lower-level model, we have effectively *split* the nodal-type term  $\theta^Y$  into two parts:  $\theta^{Y_{\text{lower}}}$  and  $\theta^K$ . Intuitively, in the higher-level model, (a),  $Y$  is a function of  $X$  and  $\theta^Y$ , the latter representing all things other than  $X$  than can affect  $Y$ . Or, in the language of our nodal-type setup,  $\theta^Y$  represents all of the (unspecified) sources of variation in  $X$ 's effect on  $Y$ . When we insert  $K$  into the model, however,  $X$  now does not directly affect  $Y$  but only does so via  $K$ . Further, we model  $X$  as acting on  $K$  in a manner conditioned by  $\theta^K$ , which represents all of the (unspecified) factors determining  $X$ 's effect on  $K$ . The key thing to notice here is that  $\theta^K$  now represents *a portion of the variance that  $\theta^Y$  represented in the higher-level graph*: some of the variation in  $X$ 's effect on  $Y$  now arises from variation in  $X$ 's effect on  $K$ , which is captured by  $\theta^K$ . So, for instance,  $X$  might have no effect on  $Y$  because  $\theta^K$  takes on the value  $\theta_{00}^K$ , so that  $X$  has no effect on  $K$ . Put differently, any effect of  $X$  on  $Y$  must arise from an effect of  $X$  on  $K$ ; so  $\theta^K$ 's value must be either  $\theta_{01}^K$  or  $\theta_{10}^K$  for  $X$  to affect  $Y$ .<sup>2</sup> What  $\theta^K$  represents, then, is that part of the original  $\theta^Y$  that arose from some force other than  $X$  operating at the *first* step of the causal chain from  $X$  to  $Y$ . So now,  $\theta^Y$  in the lower-level graph is not quite the same entity as it was in the higher-level graph. In the original graph,  $\theta^Y$  represented *all* sources of variation in  $X$ 's effect on  $Y$ . In the lower-level model, with  $K$  as mediator,  $\theta^Y$  represents only the variation in  $K$ 's effect on  $Y$ . Put differently,  $\theta^Y$  has been expunged of any factors shaping the first stage of the causal process, which now reside in  $\theta^K$ . We highlight this change in  $\theta^Y$ 's meaning by referring in the second model to  $\theta^{Y_{\text{lower}}}$ .

Consider next model  $M''$  panel (c) in Figure 6.1, which also supports (implies) the higher-level model in panel (a). The logical relationship between models (a) and (c), however, is somewhat different. Here the lower-level model *specifies* one of the conditions that comprised  $\theta^Y$  in the higher-level model. In specifying a moderator,  $C$ , we have extracted  $C$  from  $\theta^Y$ , leaving

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<sup>2</sup>As we emphasize further below, it is in fact only the random, unknown component of the  $X \rightarrow K$  link that makes the addition of  $K$  potentially informative as a matter of research design: if  $K$  were a deterministic function of  $X$  only, then knowledge of  $X$  would provide full knowledge of  $K$ , and nothing could be learned from observing  $K$ .

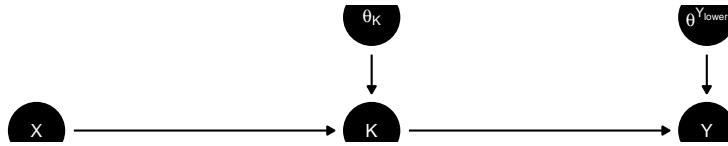
$\theta^{Y_{\text{lower}}}$  to represent all factors *other than C* that condition  $Y$ 's response to its parents. More precisely,  $\theta^{Y_{\text{lower}}}$  now represents the set of nodal types defining how  $Y$  responds jointly to  $X$  and  $C$ . Again, the relabeling as  $\theta^{Y_{\text{lower}}}$  reflects this change in the term's meaning. Whereas in Model  $M'$  we have extracted  $\theta^K$  from  $\theta^Y$ , in Model  $M''$ , it is  $C$  itself that we have extracted from  $\theta^Y$ , substantively specifying what had been just a random disturbance.

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(a) A Higher-Level Model, M



(b) Lower-Level Model, M': Disaggregating via Mediation



(c) Lower-Level Model M'': Disaggregating via Moderation

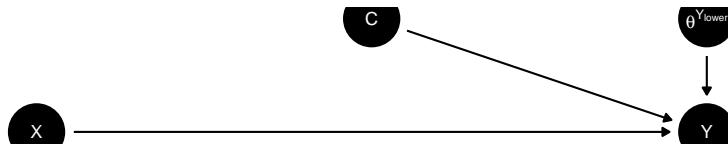


Figure 6.1: Here we represent the simple claim that one variable causes another, and two theories — lower-level models — that could explain this claim. Both model (b) and model (c) involve theorization via disaggregation of nodes.

**Type-reducing theorization.** There is a second way in which we might imagine a model being implied by another model that does not involve a change in nodes. Let  $\Theta(\mathcal{M}_1)$  denote the set of causal types in model  $\mathcal{M}_1$ . Then we can say that  $\mathcal{M}_0$  implies  $\mathcal{M}_1$  if  $\Theta(\mathcal{M}_0) \subseteq \Theta(\mathcal{M}_1)$ . Informally this means that any relation admitted by theory  $\mathcal{M}_0$  is representable in model

$\mathcal{M}_1$ , though the converse may not be true. We can think of a theory of  $\mathcal{M}_1$  as a *restriction* of ranges of  $\Theta(\mathcal{M}_1)$ .

We illustrate the idea in Figure 6.2. In panel (a) of the figure, we have a model,  $\mathcal{M}_1$  in which  $Z$  can have both a direct and an indirect effect (via  $X$ ) on  $Y$ . Suppose that we believed that  $\mathcal{M}_1$  was technically true but overly permissive, in the sense that it allowed for causal relations that we do not in fact believe are operating. We might believe, for instance, that  $Z$  has no direct effect on  $Y$  and that  $Z$  has no negative effects on  $X$  — the beliefs we would need to hold to treat  $Z$  as an instrument for  $X$ . We could thus write down a lower-level model,  $\mathcal{M}_0$ , in which we have *reduced* the type space accordingly. Specifically, in  $\mathcal{M}_0$ , we would restrict the nodal types at  $Y$  to only the  $\theta_{0000}^Y, \theta_{1100}^Y, \theta_{0011}^Y$ , and  $\theta_{1111}^Y$ ; and we would reduce the nodal types at  $X$  to  $\theta_{00}^X, \theta_{01}^X$ , and  $\theta_{11}^X$ . In panel (b), we (somewhat loosely) represent  $\mathcal{M}_0$ . We have now eliminated the arrow from  $Z$  to  $Y$  to represent the dropping of all nodal types involving a direct effect of  $Z$  on  $Y$ ; not pictured is the monotonicity assumption at  $X$ . However, we have relabeled the nodal-type nodes for both  $X$  and  $Y$  to represent the fact that these are different objects from the nodal type nodes in the higher-level model.<sup>3</sup>

Thus, while we can theorize by adding substantive nodes to a model and thus splitting types, we can also theorize by maintaining existing nodes but constraining relations among them. In both forms of theorization, we start with a model that allows for a broad, and possibly unknown, range of possibilities: for instance, a broad range of paths through which or conditions under which  $X$  might affect  $Y$  or a broad range of causal effects operating at each node. Theorization of both forms then involves making a *stronger* claim: for instance, a claim about *how* or *when*  $X$  affects  $Y$  (via type-splintering) or a claim about the *particular* causal effects operating at a given node (via type-reduction). In both forms of theory, believing the stronger claim in the lower-level model implies believing the weaker claim in the higher-level model. Further, both modes of theorization also map nicely onto common ways in which we think about theory-development in the social sciences: we theorize mechanisms, sources of heterogeneity, and directions of effects (starting with a belief that  $X$  affects  $Y$ , for instance, and moving to a more

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<sup>3</sup>We drop the arrow in Figure 6.2, however, in order to help visually convey the difference between the two models. In fact, we would construct  $\mathcal{M}_0$  by placing restrictions at nodes in  $\mathcal{M}_1$ , rather than by changing the model's structure, so that the allowed types in  $\mathcal{M}_0$  form a subset of those in  $\mathcal{M}_1$ .

constrained belief about whether that effect is positive or negative).

Finally, as we speak to below, theorization of both forms can generate gains for causal inference, by allowing us to use data in ways that we are unable to use it in the higher-level model.

### Preserving (conditional) independencies

Not all potential mappings from higher- to lower-levels are permitted. In particular, when theorizing, we may *add* but may not *remove* independencies implied by the original model. If two variables are independent—or conditionally independent given a third variable—in one model, then this same relation of independence (or conditional independence) must be captured in any theory of that model. For instance, if we start with a model of the form  $X \rightarrow Y \leftarrow W$ , where  $W$  and  $X$  are independent, we could not theorize this model by adding an arrow from  $X$  to  $W$ . A theory can have *additional* conditional independencies not present in the higher-level model, as in the example in Figure 6.2. But we may not theorize *away* (conditional) independencies insisted on by our higher-level claim.

This is a key part of what it means for the lower-level model to *justify* the higher-level model. A model makes claims about what is (conditionally) independent of what. The claims about conditional independence implied by the higher-level model must therefore be warranted by (conditional) independencies operating in the lower-level model. If we introduce new dependencies via theorization, then our higher-level model (which excludes these dependencies) would no longer be justified by the lower-level model.

#### 6.1.2 Probabilistic causal models

At the structural level, then, there are two types of theory, or two types of relations between levels of model: those defined by type-splintering and those defined by type-reduction. In general, we will want to be working with probabilistic causal models—i.e., those that include distributions over nodal types. We can describe straightforwardly how distributions in a higher-level model relate to—and must change with—distributions at the lower level. Indeed, it is these relations that unlock the opportunity for reaping empirical gains from theory.

**Theoretical implications of probabilistic models.** Suppose we start

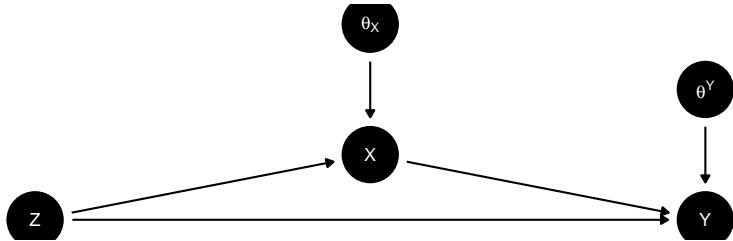
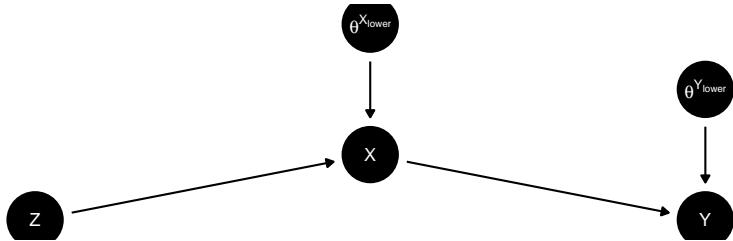
(a) A higher-Level Model,  $M_1$ (b) A lower-Level Model,  $M_0$ , with types reduced

Figure 6.2: Here we represent theorization via type-reduction. Though we show the removal of an arrow to help convey the idea, we would in fact reduce types by imposing restrictions on the nodal types at Y within the same DAG.

with the mediation model in panel (b) of Figure 6.1. We then add to it a distribution over  $\theta^K$  and  $\theta^Y_{lower}$ , giving us a probabilistic causal model that we will denote  $\mathcal{M}_{lower}^p$ .  $\mathcal{M}_{lower}^p$ , in turn, implies a higher-level probabilistic model,  $\mathcal{M}_{higher}^p$ , formed from the structure of Model (a) in Figure 6.1, and a *particular* distribution over  $\theta^Y$ : specifically,  $\theta^Y$  will have the distribution that preserves the causal relations implied by the beliefs in  $\mathcal{M}_{lower}^p$ . Thus, for instance, the probability that  $X$  has a positive effect on  $Y$  in  $\mathcal{M}_{higher}^p$  is  $\theta_{01}^Y$ ; the probability that  $X$  has a positive effect on  $Y$  in  $\mathcal{M}_{lower}^p$  is  $\theta_{01}^{K_{lower}}\theta_{01}^{Y_{lower}} + \theta_{10}^{K_{lower}}\theta_{10}^{Y_{lower}}$ . Consistency then requires that  $\theta_{01}^{M_{lower}}\theta_{01}^{Y_{lower}} + \theta_{10}^{K_{lower}}\theta_{10}^{Y_{lower}} = \theta_{01}^{Y_{higher}}$ . So the value of  $\theta_{01}^{Y_{higher}}$  is *implied* by  $\theta_{01}^{K_{lower}}, \theta_{01}^{Y_{lower}}, \theta_{10}^{K_{lower}}, \theta_{10}^{Y_{lower}}$ , but not vice-versa.

**Deducing models from theory and data.** Now we can see what happens if we bring data to the lower-level model. A probabilistic causal model coupled with data implies another probabilistic causal model via Bayes rule. For this reason, we can fruitfully think of an initial model as being a *theory of* an updated model, coupled with data. Thought of in this way we have clarity over what is meant when we turn to theory to support a claim, but also what is meant when we seek to justify a theory. We might imagine a scholar arguing: “ $\mathcal{M}_1$ :  $X$  caused  $Y$  in country  $j$ .” When pushed for a justification for the claim they provide the lower level model: “ $\mathcal{M}_0$  : the average effect of  $X$  on  $Y$  in countries with feature  $C = 1$  is 0.95, making it likely that  $X$  caused  $Y$  in this case.” Here  $\mathcal{M}_1$  is implied by  $\mathcal{M}_0$  coupled with data  $C = 1$ . If pushed further as to why that theory is itself credible they might point to a lower level model consisting of structural model  $X \rightarrow Y \leftarrow C$  plus flat priors coupled with data on  $X, Y$  and  $C$ . At each stage, as more justification is provided, the researcher formally provides lower-level models.

Moving up, as more data is provided, more “specific” higher level models emerge, justified by lower models plus data. These models are more specific in the sense that they are implied by the higher level models, plus data, but not vice versa. But they are also (generally) more specific in a second sense: that they make stronger statements about how causal processes operate.<sup>4</sup> They place greater weight on a smaller set of causal claims.<sup>5</sup>

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<sup>4</sup>This is not universally true, a point we return to below.

<sup>5</sup>In frequentist frameworks we often think of analysis as implementing up-or-down empirical tests against data to parse between theories that should be maintained and theories that should be rejected. In a Bayesian framework we think more continuously of

As the simplest illustration, we might imagine beginning with an  $X \rightarrow Y$  model,  $\mathcal{M}_1$ , in which,  $X$  and  $Y$  are binary, and we believe that  $Y$  possibly responds to  $X$ . If we have “flat” priors over causal types, in the sense described in Chapter 5, then our prior uncertainty over the proposition that  $X$  causes  $Y$ , under this model, is large; as is our uncertainty that  $Y = 1$  is due to  $X = 1$  in a given case. In other words, given our theory, we are uncertain about the proposition  $\mathcal{M}_3$ :  $X$  caused  $Y$ . However, if we then receive a lot of data,  $\mathcal{D}$ , showing strong relations between  $X$  and  $Y$ , then our updated model  $\mathcal{M}_2$ , formed from combining  $\mathcal{D}$  and  $\mathcal{M}_1$  allows us to infer that  $X$  caused  $Y$  in this case with greater certainty.

Thus our new theory  $\mathcal{M}_2$  is (a) formally similar to  $\mathcal{M}_1$ , (b) formed as a product of past theory plus evidence, here justified by  $\mathcal{M}_1$  given data  $\mathcal{D}$ , and (c) capable of providing sharper implications than past theory.<sup>6</sup>

In this way, Bayesian updating provides a simple and coherent way of thinking about the integration of theory and data.

## 6.2 Gains from theory

We now turn to consider how to think about whether a theory is *useful*. We are comfortable with the idea that theories, or models more generally, are wrong. Models are not full and faithful reflections of reality; they are maps designed for a particular purpose. We make use of them because we think that they *help* in some way.

But how do they actually help, and can we quantify the gains we get from using them?

We think we can. Using the notion of hierarchies of models, imagine we begin with model  $\mathcal{M}_1$ , which together with data  $\mathcal{D}$ , implies claim  $\mathcal{M}_2$ . We then posit theory  $\mathcal{M}_0$  of  $\mathcal{M}_1$ , so  $\mathcal{M}_0$  implies  $\mathcal{M}_1$ . But when we bring  $\mathcal{D}$  to  $\mathcal{M}_0$  we

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shifting our beliefs across causal possibilities within a multi-dimensional theoretical space.

<sup>6</sup>As a general matter an updated theory may not provide sharper claims for all queries. That is, in practice, posterior variance over queries can increase with more data. As a simple illustration: say, we start out thinking that the probability that an outcome is due to conditions A, B, or C is .9, .05, and .05, respectively. If I find evidence that convinces me that A is not the cause, then I shift (a) to greater certainty about whether A was the cause but (b) greater uncertainty about whether B was the cause.

get a new model,  $\mathcal{M}'_2$ , that is different—and, hopefully, better—than  $\mathcal{M}_2$ . Our gain from theory  $\mathcal{M}_0$  should be some summary of how much better  $\mathcal{M}'_2$  is than  $\mathcal{M}_2$ .

Here is an illustration using a theory that allows use of the “front-door criterion.”

Imagine that we have data on three variables,  $X$ ,  $Y$ , and  $K$ . We begin, however, with a model  $\mathcal{M}_1$  with confounding:  $C \rightarrow X \rightarrow Y \leftarrow C$ .  $\mathcal{M}_1$  includes nodes for two of the three variables we have data on,  $X$  and  $Y$ , but not  $K$ . Assume, further, that we do not have data on node  $C$ , the confound.

Suppose that we observe a strong correlation between  $X$  and  $Y$  and infer  $\mathcal{M}_2$ : that  $X$  is a likely cause  $Y$ . Our inference under  $\mathcal{M}_2$  is, however, quite uncertain because we are aware that the correlation may be due to the confound  $C$ .

Suppose now that we posit the lower-level model  $\mathcal{M}_0$ :  $C \rightarrow X \rightarrow K \rightarrow Y \leftarrow C$ .  $\mathcal{M}_0$  now lets us make better use of data  $K$ . If we observe, for instance, that  $X$  and  $K$  are uncorrelated, then we infer with confidence that in fact  $X$  did not cause  $Y$ , despite the correlation.

Thus, in return for specifying a theory of  $\mathcal{M}_1$ , we have been able to make better use of data and form a more confident conclusion. In this case, stating the theory,  $\mathcal{M}_0$ , does not alter our *priors* over our query. Our prior over the effect of  $X$  on  $Y$  may be identical under  $\mathcal{M}_1$  and  $\mathcal{M}_0$ —but our conclusions, given data, differ because the theory lets us make use of the data on  $K$ , which we could not do under  $\mathcal{M}_1$  (which did not include  $K$ ).

In other situations, we might imagine invoking a theory that does not necessarily involve new data but that allows us to make different, perhaps tighter inferences using the same data. An example might be the invocation of a type-reducing theory that involves a monotonicity restriction or exclusion restriction that allows for identification of a quantity that would not be identifiable without the theory.

Thus, one reason to theorize our models — develop lower-level models that make stronger claims — is to be able to reap greater inferential leverage from the more elaborate theory.

But are we, in fact, better off?

We might imagine answering the question in different ways: from an internal

or external position, and from an *ex ante* or *ex post* perspective.

In all cases we ask how much better do we do as a result of making use of a theory.

If we are willing to posit an external ground truth, then we can define “better” in objective terms. An *ex post*, objective way of operationalizing “better” is to assess the size of the error we make relative to the ground truth, from an inference that uses a theory, compared to an inference that does not make use of the theory. An objective *ex ante* approach might be to ask what the expected error is from conclusions one draws given a theory. For instance: how wrong are we likely to be if we base our best guess on our posterior mean? “How wrong” might be operationalized in terms of mean squared error—the square of the distance between the truth and the posterior mean.<sup>7</sup>

A more subjective approach would be to ask about the reductions in posterior variance. *Ex post* we can define “better” as the reduction in posterior variance from conclusions that make use of a theory compared to conclusions that do not. We might also think about the *expected posterior variance*: how certain do you expect you will be after you make use of this new information?

More formally, imagine a situation in which there is an unknown parameter  $q$  and we have a data strategy that produces a distribution over data  $k$ , given  $q$ . Let  $p(q, k)$  denote the joint prior distribution over  $q$  and  $k$  with marginal distributions  $p(k)$  and  $p(q)$ . For any  $k$  there is posterior estimate  $q_k$ .

The squared error, given  $k$  is just  $(q - q_k)^2$ .

The *expected* squared error is:

$$ESE := \int_q \int_k (q_k - q)^2 p(k, q) dk dq$$

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<sup>7</sup>Other loss functions could be used, including functions that take account of the costs of collecting additional data or to the risks associated with false diagnoses. For instance, in Heckerman et al. (1991), an objective function is generated using expected utility gains from diagnoses generated based on new information over diagnoses based on what is believed already. In their treatment (Heckerman et al., 1991, Equation 6), the expected value of new information  $K$ , given existing information  $W$  is:  $\sum K P(K|W)(EU(d(Q, W, K)|W, K) - EU(d(Q, W)|W, K))$  where  $EU$  is expected utility and  $d$  is the optimal inference (diagnosis) given available data. Note that the diagnosis can take account of  $K$  when it is observed, but the expected utility depends on  $K$  whether or not it is observed, as  $K$  carries information about the state of interest.

This takes the error one might get with respect to any true value of the parameter ( $q$ ), given the data one might see given  $q$  and the inferences one might draw.

For any  $k$  we might write the posterior variance as  $v_k$ .

The *expected* posterior variance can be written:

$$EV := \int_k v_k p(k) dk$$

This takes the posterior variance, given some data, over all the possible data one might see given marginal distribution  $p(k)$ .

Interestingly, if we assess expectations using the same priors as you use for forming posteriors the expected posterior variance and expected squared error are equivalent (Scharf, 1991). To see this, we take advantage of the fact that  $p(q, k) = p(k)p(q|k) = p(q)p(k|q)$  and that  $p(q|k)$  gives the posterior distribution of  $q$  given  $k$ . We then have:

$$ESE = \int_q \int_k (q_k - q)^2 p(q, k) dk dq \quad (6.1)$$

$$= \int_k \int_q (q_k - q)^2 p(k)p(q|k) dq dk \quad (6.2)$$

$$= \int_k \left[ \int_q (q_k - q)^2 p(q|k) dq \right] p(k) dk \quad (6.3)$$

$$= \int_k v_k p(k) dk = EV \quad (6.4)$$

Note that the key move is in recognizing that  $p(q|k)$  is in fact the posterior distribution on  $q$  given  $k$ . In using this we assume that the same distribution is used for assessing error and for conducting analysis—that is we take the researcher's prior to be the relevant one for assessing error.

Moreover, it is easy to see that whenever inferences are sensitive to  $K$ , the *expected* variance of the posterior will be lower than the variance of the prior. This can be seen from the law of total variance, written here to highlight the gains from observation of  $K$ , given what is already known from

observation of  $W$ .<sup>8</sup>

$$\text{Var}(Q|W) = E_{K|W}(\text{Var}(Q|K, W)) + \text{Var}_{K|W}(E(Q|K, W))$$

However, although *expected* posterior variance goes down, it is still always possible that posterior variance rises. The increase in uncertainty does not, however, mean you haven't been learning. Rather, you have learned that things aren't as simple as you thought.

One way to capture this idea that, although we are more uncertain, we think we are better off now than we were, is to ask: how much better are our guesses having observed  $K$  compared to what we would have guessed before, *given* what we know having observed  $K$ ? We will call this "Wisdom" to reflect the idea that it values appreciation of justifiable uncertainty:

$$\text{Wisdom} = \int (q_0 - q)^2 - (q_k - q)^2 p(q|k) dq$$

This metric captures how much better off we are with the guess we have made given current data ( $q_k$ ) compared to the guess we would have made if we had a theory that did not let us make use of it ( $q_0$ ), knowing what we know having observe  $K$  ( $p(q|k)$ ).

An advantage of this conceptualization is that we can still record gains in learning even if the learning operates such that the posterior variance is larger than the prior variance. Even so, the implications for strategy are the same since wisdom is maximized by a strategy that reduces expected squared error.

Thus expected wisdom, is:

$$\text{Expected Wisdom} = \int_q (q_0 - q)^2 dq - \int_k \int_q (q_k - q)^2 p(q, k) dq dk \quad (6.5)$$

$$= \text{Prior variance} - \text{Expected Posterior Variance} \quad (6.6)$$

We close with a reminder. Although expected reduction in variance and expected wisdom are both positive, both are are fundamentally subjective

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<sup>8</sup>See Raiffa and Schlaifer (1961). A similar expression can be given for the expected posterior variance from learning  $K$  in addition to  $W$  when  $W$  is not yet known. See, for example, Proposition 3 in Geweke and Amisano (2014).

ideas, that presuppose the theory is correct. In contrast the expected error measure can be assessed under rival theoretical propositions and so allow for the real possibility that the gains of invoking a theory are negative.

## 6.3 Formal theories and causal models

It is relatively easy to see how the ideas above play out for what might be called empirical models. But in social sciences “theory” is a term sometimes reserved for what might be called analytic models. In this last section we work through how to use this framework when seeking to bring analytic models to data.

Let’s start with analytic models. As an example we might consider the existence of “Nash equilibria.” Nash considered a class of settings (“normal form games”) in which each player  $i$  can choose an action  $\sigma_i$  from set  $\Sigma_i$  and receives a payoff  $u_i$  that depends on the actions of all players. A particular game,  $\Gamma$  is the collection of players, action sets, and payoffs.

Nash’s theorem relates to the existence of a collection of strategies with the property that each strategy would produce the greatest utility for each player given the strategies of the other players. Such a collection of strategies is called a Nash equilibrium.

The claim that such a collection of strategies exists in these settings is an analytic claim. Unless there are errors in the derivation of the result, the claim is true in the sense that the conclusions follow from the assumptions. There is no evidence that we could go looking for in the world to assess the claim. The same can be said of the theoretical claims of many formal models in social sciences; they are theoretical conclusions of the if-then variety (Clarke and Primo, 2012).

We will refer to theories of this form as “analytic theories.”

When researchers refer to a theory of populism or a theory of democratization however they generally do not have such pure theories in mind. Rather they have in mind what might be called “applied theories” (or perhaps more simply “scientific theories” or “empirical theories”): general claims about the relations between objects in the world. The distinction here corresponds to

the distinction in Peressini (1999) between “pure mathematical theories” and “mathematized scientific theories.”<sup>9</sup>

Applied theory, in this sense, is a collection of claims with *empirical* content: an applied theory refers to a set of propositions of causal relations in the world that might or might not hold, and is susceptible to assessment using data. These theories might look formally a lot *like* analytic theories but it is better to think of them as translations at most. The relations between nodes of an applied theory are a matter of conjecture not a matter of necessity.<sup>10</sup>

Though it is not standard practice, formal models produced by game theorists can often be translated and then represented using the notation of structural causal models in this way. Moreover, doing so may be fruitful. Using the approach described above we can then assess the utility of the applied theory, if not the pure theory itself.

For two players, for instance, we might imagine a representation of a game as shown in Figure 6.3.

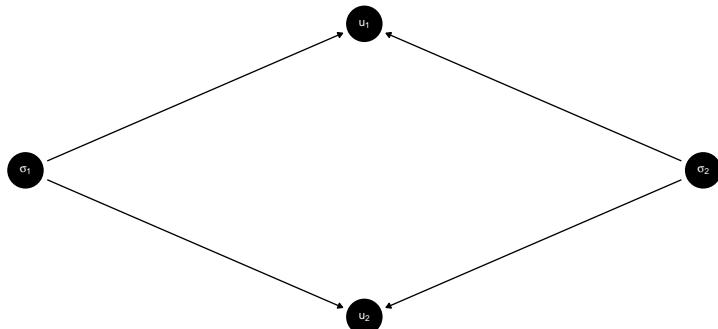


Figure 6.3: Formal structure of a normal form game.

Here the only functional equations are the utility functions. The utilities, given actions, are the implications of the theory, and so this is just a theory of how outcomes depend on social actions. It is not—yet—a behavioral theory.

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<sup>9</sup>Or see the distinction, for instance in in Keynes, between pure theory and applied theory.

<sup>10</sup>Peressini (1999) distinguishes between “applied mathematical theories” and “mathematized scientific theories” on the grounds that not all mathematized theories are an application of a pure theory.

In contrast to Nash's theorem regarding the existence of equilibria, a behavioral theory might claim that in problems that can be represented as normal form games, players indeed play Nash equilibrium. This is a theory about how people act in the world. We might call it Nash's theory.

How might this theory be represented as a causal model? Figure 6.4 provides one representation.

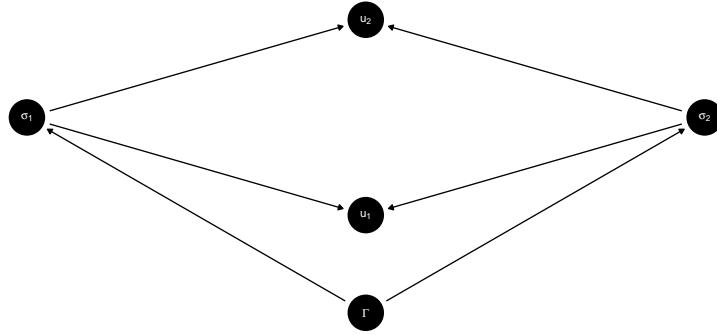


Figure 6.4: Formal structure of a normal form game.

Here beliefs about the game form ( $\Gamma$ ) results in strategy choices by actors. If players play according to Nash's theory, *the functional equations for the strategy choices are given by the Nash equilibrium solution itself*, with a refinement in case of multiplicity.

This model represents what we expect to happen in a game under Nash's theory and we can indeed see if the relations between nodes in the world look like what we expect under the theory. But it does not provide much of an *explanation* for behavior.

A lower level causal model might help. In Figure 6.5 the game form  $\Gamma$  determines the beliefs about what actions the other player would make (thus  $\sigma_2^e$  is 1's belief about 2's actions). The functional equations for  $\sigma_2^e$  and  $\sigma_1^e$  might, for instance, be the Nash equilibrium solution itself: that is, players expect other players to play according to the Nash equilibrium (or in the case of multiple, a particular equilibrium selected using some refinement). The beliefs in turn, together with the game form (which contains  $u_1, u_2$ ), are what cause the players to select a particular action. The functional equation for  $\sigma_1$  might thus be  $\sigma_1 = \arg \max_{\sigma} u_1(\sigma, \sigma_2^e)$ .

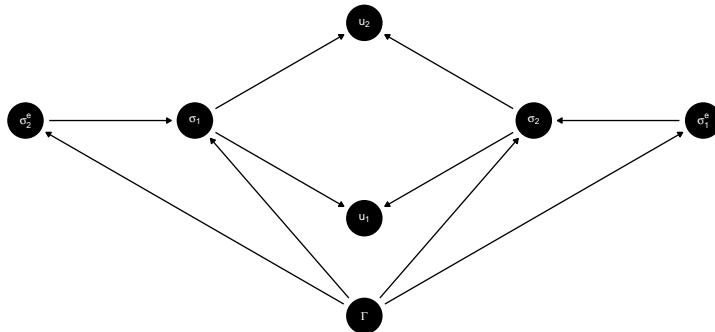


Figure 6.5: Formal structure of a normal form game.

This representation implies a set of relations that can be compared against empirical patterns. Do players indeed hold these beliefs when playing a given game? are actions indeed consistent with beliefs in ways specified by the theory. It provides a theory of beliefs and a theory of individual behavior as well as an explanation for social outcomes.

The model in Figure 6.5 provides a foundation of sorts for Nash's theory. It suggests that players play Nash equilibria *because* they expect others to and they are utility maximizers. But this is not the only explanation that can be provided; alternatively behavior might line up with the theory without passing through beliefs at all as suggested in some accounts from evolutionary game theory that show how processes might select for behavior that corresponds to Nash even if agents are unaware of the game they are playing.

One might step still further back and ask *why* would actors form these beliefs, or take these actions, and answer in terms of assumptions about actor rationality. Figure 6.6 for instance is a model in which actor rationality might vary and might influence beliefs about the actions of others as well as reactions to those beliefs. Fully specified functional equations might specify not only how actors act when rational but also how they react when they are not. In this sense the model in Figure 6.6 both nests Nash's theory and provides an explanation for why actors conform to the predictions of the theory.

In a final elaboration we can represent a kind of underspecification of Nash's theory that make it difficult to take the theory to data. In the above we assumed that players chose actions based on expectations that the other

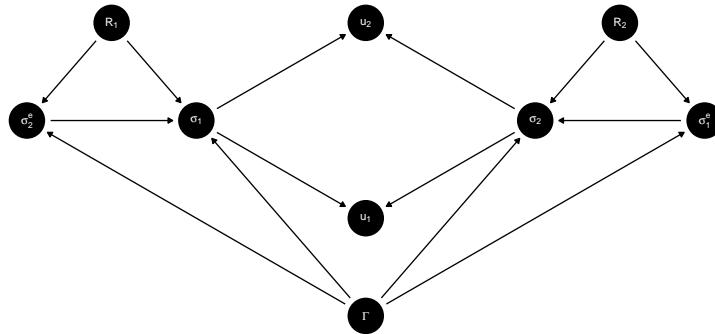


Figure 6.6: Formal structure of a normal form game.

player would play the Nash equilibrium—or that the theory would specify which equilibrium in the case of multiplicity. But it is well known that Nash’s theory often does not provide a unique solution. This indeterminacy can be captured in the Causal model as shown in Figure 6.7 where a common shock—labelled  $\nu$ , and interpreted as norms—interacts with the game form to determine the expectations of other players.

The functional equation for expectations can then allow for the possibility that (i) there is a unique equilibrium invariably chosen and played by both (ii) or a guarantee that players are playing one or other equilibrium together but uncertainty over which one is played, or (iii) the possibility that players are in fact out of sync, with each playing optimal strategies given beliefs but nevertheless not playing the same equilibria.

Nash’s theory likely corresponds to position (ii). It can be captured by functional equations on beliefs given  $\nu$  but the theory does not specify  $\nu$ , in the same way that it does not specify  $\Gamma$ .

We highlight three points from this discussion.

First the discussion highlights that thinking of theory as causal models does not force a sharp move away from abstract analytic theories; close analogues of these can often be incorporated in the same framework. This is true even for equilibrium analysis that seems to involve a kind of simultaneity on first blush.

Second, the discussion highlights how the causal modelling framework can make demands for specificity from formal theories. For instance specifying a

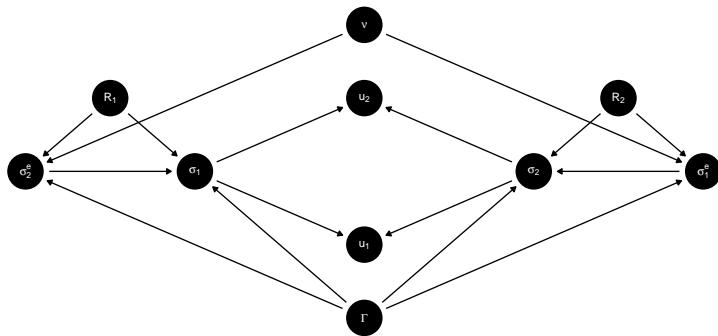


Figure 6.7: A normal form game with a representation of equilibrium selection norms.

functional relations from game form to actions requires a specification of a selection criterion in the event of multiple equilibria. Including agent rationality as a justification for the theory invites a specification for what would happen absent rationality.

Third the example shows a way of building a bridge from pure theory to empirical claims. One can think of Nash's theory as an entirely data free set of claims. When translate into an applied theory—a set of proposition about the ways actual players *might* behave—and represented as a causal model, we are on a path to being able to use data to refine the theory. Thus we might begin with a formal specification like that in Figure 6.7 but with initial uncertainty about player rationality, optimizing behavior, and equilibrium selection. This theory nests Nash but does not presume the theory to be a valid description of processes in the world. Combined with data, however, we shift to a more refined theory that selects Nash from the lower level model.

Finally, we can then apply the ideas of section 6.2 to applied formal theories and ask: is the theory useful? For instance, does data on player rationality help us better understand the relationship between game structure and welfare?

## **Part II**

# **Model-Based Causal Inference**



# Chapter 7

## Process Tracing with Causal Models

### 7.1 Process tracing and causal models

This chapter demonstrates how we can use causal models to conduct confirmatory process tracing: that is, to draw causal inferences about a single case from a causal model with data provided at the case-level.

#### 7.1.1 The intuition

We first walk through the basic intuition and then provide a more formal account.

When we undertake process tracing, we seek to answer a causal question about a given case. The key insight driving our approach is that the inference about a causal question for a case is a claim about **which causal types (collections of nodal types) are both likely ex ante (given prior knowledge) and consistent with the data**.<sup>1</sup>

The question of interest can be about any number of case-level causal features, including questions about a case-level causal effect, the pathway

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<sup>1</sup>This differs from the task for mixed methods that we will address in Chapter 9 as these concern claims about the distribution of causal types in populations.

through which an effect operates, an actual cause, or causal attribution. We use observations from the case itself to address this query. We do so via a procedure in which we first encode prior knowledge in the form of a causal model, then use data to learn about features of the model, and finally take what we have learned about the model and map it into our query.

Given a causal model, we form posteriors over queries as follows:

1. **Specify all possible causal types for a model.** A causal type, recall, is a particular combination of nodal types for all nodes in a unit. That is, a single causal type specifies both a specific set of values of all exogenous variables in a model and the values that all endogenous variables *would* potentially take on for all possible values of the exogenous variables. For a simple, binary  $X \rightarrow Y$  model, the number of possible causal types will be 2 (the number of possible values  $X$ , the exogenous node, can take on) times 4 (the number of possible nodal types for  $Y$ , the endogenous node). Three of these causal types would be:
  - Type 1: ( $X = 1$ ) and ( $Y = 1$  if  $X = 1$ ,  $Y = 0$  if  $X = 0$ ).
  - Type 2: ( $X = 0$ ) and ( $Y = 1$  if  $X = 1$ ,  $Y = 0$  if  $X = 0$ ).
  - Type 3: ( $X = 1$ ) and ( $Y = 1$  if  $X = 1$ ,  $Y = 1$  if  $X = 0$ ).

Whatever the model, we generate a complete set of all possible causal types.

2. **Specify priors over causal types.** We report how likely we think it is that a given unit is of a particular causal type. In the simplest situation, we might place 0 weight on some causal types (which might be ruled out by background theory, for example) and equal weight on the others.
3. **Specify the query in terms of causal types.** For instance, for the simple  $X \rightarrow Y$  model, the query “ $Y$  responds positively to  $X$ ” can be thought of as a collection of causal types:  $Q=\{\text{Type 1, Type 2}\}$ .
4. **Once we observe the data, specify the set of causal types that are consistent with those data.** For instance, if we observe  $X = 1, Y = 1$  we might specify the data-consistent set as  $\{\text{Type 1, Type 3}\}$ , excluding Type 2 with which these data are inconsistent.

5. **Update.** Updating is then done by adding up the prior probabilities on all causal types that are consistent with both the data and the query, and dividing this sum by the sum of prior probabilities on all causal types that are consistent with the data (whether or not they are consistent with the query).

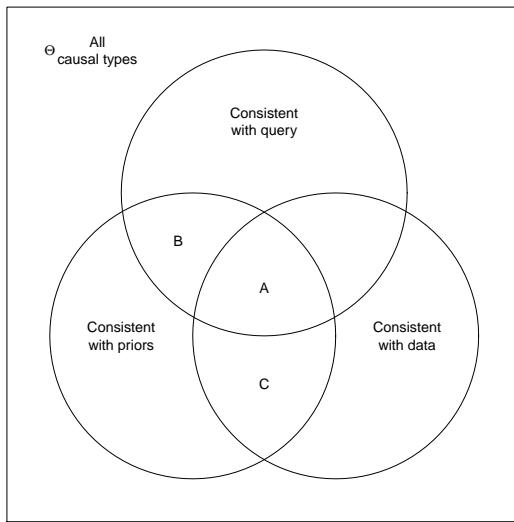


Figure 7.1: Logic of simple updating on arbitrary queries.

This process is represented graphically in Figure 7.1, where we can think of probabilities as proportionate to areas. Our causal model defines the causal-type space. We then proceed by a process of elimination. Only some of the causal types in the model are consistent with prior knowledge. Only some are consistent with the data that we observe. Finally, any query itself maps onto a subset of the possible causal types. The causal types that remain in contention once we have observed the evidence are those at the intersection of consistency with priors and consistency with the data.  $A$  represents those types that are *also* consistent with a given answer to the query (say,  $X$  has a positive effect on  $Y$ ).

Thus, our belief about the query before we have seen the data is the probability of all causal types consistent with our priors and with the query ( $A + B$ ) as a proportion of all types consistent with our priors. Once we have seen the data, we have reduced the permissible types to  $A + C$ . Our posterior

belief on the query is, then, the probabilities of those remaining types that are consistent with the query as a share of the probabilities of *all* remaining types, or  $A/(A + C)$ .

We now turn to a formalization of these ideas.

### 7.1.2 A formalization of the general approach

The general approach to inference draws on the components we outlined in chapters 2 to 4: graphical causal models (DAGs), queries, and priors. Coupled with data these elements provide grounds for causal inferences. We continue to focus on a situation with binary variables, though describe later how this can be extended. We walk through the procedure for simple models, though note that the approach outlined here can be applied to *any* causal model with discrete variables and to any queries defined over the model.

The process tracing procedure operates as follows.

#### 7.1.2.1 The model

First we need a model.

##### A DAG

We begin with a DAG, or graphical causal model. As discussed in Chapter 2, a DAG identifies a set of variables and describes the parent-child relations between them, indicating for each variable which other variables are its direct (possible) causes. These relationships, in turn, tell us which (non-descendant) variables a given variable is *not* independent of given the other variables in the model.

##### Nodal types

Once we have specified a DAG, we can determine the full set of possible nodal types: the types defining the value that a variable will take on given the values of its parents, which we have denoted with  $\theta^j$  values for node  $j$ , as in  $\theta_0^X$  or  $\theta_{10}^Y$ . At each node, the range and number of possible nodal types is defined by the number of parents that that node has and the number of values the variables can take on. For instance, assuming all variables to be

binary, if  $Y$  has parents  $X$  and  $W$ , then there are  $2^{(2^2)} = 16$ ) possible causal types for the  $Y$  node.

### Causal types

From the set of all possible nodal types for a DAG, we get the set of all possible causal types by simply elaborating all possible permutations of nodal types.

#### 7.1.3 Priors

Our background beliefs about a causal domain will usually consist of more than just beliefs about which variables have causal connections; they will also typically contain beliefs about what *kinds* of effects operate between variables. That is, they will contain beliefs about which types are possible or, more generally, are more or less common in the world. We express these beliefs over causal effects as probability distributions over the nodal types. Beliefs about causal types are implied by beliefs about nodal types. In cases with unobserved confounding beliefs are defined over the joint distributions of nodal types.

For process tracing, our beliefs over nodal type  $\theta^j$ , say, simply capture the subjective probability we have that the type takes on different values. We do not *need* to defend this belief to use the machinery. We use  $\lambda_x^j$  to denote the probability that  $\theta^j = \theta_x^j$ . Often however it helps with intuition to think of a given case of interest – the one we are studying and seek to learn about – as being drawn at random from a population and to think about our beliefs for the *single* case as stemming from our beliefs about the population from which it is drawn. In that case  $\lambda_x^j$  can be thought of as a *share*. So, for instance, our prior belief about the probability that inequality has a positive effect on democratization in Mexico in 1999 is our belief about how commonly inequality has a positive effect on democratization in the population of cases that are “like” Mexico in 1999.<sup>2</sup>

A  $\lambda^j$  is simply a vector of numbers (or proportions), one for each possible nodal type, with all numbers non negative and summing to 1. So, for in-

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<sup>2</sup>The reference population for a case is defined based on whatever we already know about the case. Thus, for instance, if we already know that the case has  $Y = 1$  before we begin process tracing, then the relevant population for the formation of prior beliefs is all cases in which  $Y = 1$ .

stance,  $\lambda^Y$  for our current example would be a vector with four values, each of which expresses a probability on one of the four nodal types at  $Y$ . So we might have  $\lambda_{01}^Y = 0.1$ ,  $\lambda_{11}^Y = 0.05$ , and so on – with the  $\lambda^Y$  values summing to 1 because these values are defined over the full set of possible nodal types for  $Y$ . For the purposes of this chapter we take  $\lambda$  as given—you start with beliefs; in later chapters however, when we move beyond single cases,  $\lambda$  becomes the quantity of interest.

Consider now beliefs over causal types. Let's start with with a situation in which we assume that the nodal types are independent of one another. We can think of this as a situation in which there is no confounding that is not captured in the graph – no variable missing from the model that is a common ancestor of multiple nodes in the model. In this situation, our beliefs over causal types are simply the product of our beliefs over the component nodal types (since the joint probability of independent events is simply the product of their individual probabilities). For instance, one causal type might be “a unit in which  $X = 1$  and in which  $Y = 1$  no matter what value  $X$  takes.” In this case the probability that a case is of this causal type is  $\Pr(\theta^X = \theta_1^X) \Pr(\theta^Y = \theta_{11}^Y) = \lambda_1^X \lambda_{11}^Y$ .

The simplest way in which we can express beliefs about the differential probabilities of different causal possibilities is by *eliminating* nodal types that we do not believe to be possible—setting their parameter values to 0. Suppose, for instance, that we are examining the effect of ethnic diversity on civil war in a case. We might not know whether ethnic diversity causes civil war in this case, but we might have sufficient background knowledge to believe that ethnic diversity never has a *negative* effect on civil war: it never prevents a civil war from happening that would have happened in the absence of ethnic diversity. We would thus want to set the parameter value for a negative causal effect to 0. If we then know nothing about the relative frequencies of the three remaining nodal types for  $Y$ , we may (following the principle of indifference), frequency of positive effects, null effects with civil war destined to happen, and null effects with civil war never going to happen, assigning a weight of  $\frac{1}{3}$  to each of them.

In a situation of unobserved confounding, our beliefs over causal types are still well defined, though they are no longer the simple product of beliefs over nodal types. In this situation we need to describe a joint distribution over nodal types. In practice we can do this by specifying a probability for one

nodal type and a conditional probability for another. Let us imagine for instance, in a simple  $X \rightarrow Y$  model, that we believe that some unobserved factor both affects both the likelihood of  $X = 1$  and also  $X$ 's effect on  $Y$ : maybe, for instance,  $X$  is more likely to be assigned to 1 where  $X$  has a positive effect. This is the same as saying that the probability that  $\theta^X$  and  $\theta^Y$  are correlated. Now, the probability of any combination of  $\theta^X$  and  $\theta^Y$  must be calculated using the joint probability formula,  $\Pr(A, B) = \Pr(A)\Pr(B|A)$ .<sup>3</sup> Thus, for instance,  $\Pr(\theta^Y = \theta_{01}^Y, \theta^X = \theta_1^X) = \Pr(\theta^Y = \theta_{01}^Y)\Pr(\theta^X = \theta_1^X|\theta^Y = \theta_{01}^Y)$ . To form priors over causal types in this situation, we need to posit beliefs about a set of more complex, conditional probabilities for  $X$ 's type. Specifically, we need to posit, *for those cases* with a positive effect of  $X$  on  $Y$ , what are the chances a case is “assigned” to  $X = 1$ ; *for those cases* with a negative effect, and similarly for other nodal types.

Thus for instance we represent  $\Pr(\theta_1^X, \theta_{01}^Y) = \Pr(\theta_1^X)\Pr(\theta_{01}^Y|\theta_1^X)$  which we write as  $\lambda_1^X, \lambda_{01}^{Y|\theta_1^X}$ . The notation is awkward but the key thing is that we have a well defined set of beliefs that we need to take into account to assess the probability of different causal types.

In the **CausalQueries** package we represent the relationship parameters and causal types using a “parameter matrix.” This matrix has a row for each parameter, a column for each causal type, and cell entries that indicate whether the parameter forms part of the type probability. In models without confounding we have a parameter for each nodal type. In the  $X \rightarrow Y$  model, for instance, the causal causal type  $(\theta_1^X, \theta_{10}^Y)$ , has a 1 in the  $\lambda_1^X$  row and a 1 in the  $\lambda_{10}^Y$  row only, indicating that the product of these probabilities gives the probability of the causal type. In models with confounding the logic is the same except that we have rows for as many conditional probabilities as we need to fully characterize joint distributions.

### 7.1.3.1 Possible data types.

A *data type* is a particular pattern of data that we could potentially observe for a given case. More specifically, a data type is a set of values, one for each node in a model. For instance, in our  $X, W, Y$  setup,  $X = 1, W = 0, Y = 0$  would be one data type.

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<sup>3</sup>In words, the probability of  $A$  and  $B$  occurring is equal to the probability of  $A$  occurring times the probability of  $B$  occurring *given* that  $A$  occurs.

Importantly, absent intervention, each possible causal type *maps deterministically into a single data type*. One intuitive way to think about why this is the case is that a causal type tells us (a) the values to which all exogenous variables in a model are assigned and (b) how all endogenous variables respond to their parents. Given these two components, only one set of node values is possible. For example, causal type  $\theta = (\theta^X = \theta_1^X, \theta^W = \theta_0^W, \theta^Y = \theta_{0100}^Y)$  implies data  $X = 1, W = 0, Y = 1$ . Absent intervention, there is no other set of data that can be generated by this causal type.

Equally importantly, however, *the mapping from causal types to data types is not one-to-one*. More than one causal type can generate the same case-level data pattern. For instance, the causal type  $\theta = (\theta^X = \theta_1^X, \theta^W = \theta_0^W, \theta^Y = \theta_{1101}^Y)$  will also generate the data type,  $X = 1, W = 0, Y = 1$ . Thus, observing this data type leaves us with ambiguity about the causal type by which it was generated.

In the `CausalQueries` package we use an *ambiguity matrix* to summarize the mapping between causal types and data types. There is a row for each causal type and a column for each data type and an entry of 1 indicates that the causal type generates the data type. Each row has a single 1 but each column can have many 1s—an indicator of the ambiguity we have regarding causal types when we observe data types.

Table 7.1: . An ambiguity matrix, mapping from data types to causal types for a simple  $X \rightarrow Y$  model.

Data types →	X0Y0	X1Y0	X0Y1	X1Y1	Priors on causal types
Causal types ↓					
$\theta_0^X, \theta_{00}^Y$	1	0	0	0	0.1
$\theta_1^X, \theta_{00}^Y$	0	1	0	0	0.1
$\theta_0^X, \theta_{10}^Y$	0	0	1	0	0.1
$\theta_1^X, \theta_{10}^Y$	0	1	0	0	0.1
$\theta_0^X, \theta_{01}^Y$	1	0	0	0	0.2
$\theta_1^X, \theta_{01}^Y$	0	0	0	1	0.2
$\theta_0^X, \theta_{11}^Y$	0	0	1	0	0.1
$\theta_1^X, \theta_{11}^Y$	0	0	0	1	0.1

As models get more complex, the numbers of causal and data types multiply,

though generally the number of causal types increases faster than the number of data types. For a simple mediation model ( $X \rightarrow M \rightarrow Y$ ) there are  $2^3 = 8$  data types—possible combinations of values for  $X, M, Y$  but  $2 \times 4 \times 4$  causal types.

The ambiguities matrix tells us what types are data consistent and, in doing so, shapes our inferences. Table 7.2 shows a portion of the ambiguities matrix for the  $X \rightarrow M \rightarrow Y$  model, with priors on causal types appended in the final column. In this model if we observe  $X = 1, M = 0, Y = 0$  we have ambiguities over causal types. These data tell us that  $\theta_1^X = \theta_0^X$ . But they do not tell us whether  $M$ 's type is such that  $X$  has a negative effect on  $M$  ( $\theta_{10}^M$ ) or  $X$  has no effect with  $M$  fixed at 0 ( $\theta_{00}^M$ ). Similarly, we do not know whether  $M$  has a positive effect on  $Y$  ( $\theta_{01}^Y$ ) or no effect with  $Y$  fixed at 0 ( $\theta_{00}^Y$ ). This leaves four combinations of nodal types—four causal types—that are consistent with the data. These types are picked out by the ambiguities matrix.

### 7.1.4 Updating on types given the data.

Once we observe actual data in a case, we can then update on the probabilities assigned to each causal type. The logic is simple. When we observe a set of data from a case, we place 0 probability on all causal types that could not have produced these data; we then scale up the probabilities on all causal types that could have.

As a simple example imagine we have an  $X \rightarrow Y$  model with equal prior weights (1/8) on each of the eight possible causal types. Now, suppose that we observe the data  $X = 1, Y = 1$ , i.e., data type  $X1Y1$ . This data is consistent with some causal types but not others. Only two causal types are consistent with the data:  $\theta_1^X, \theta_0^Y$  and  $\theta_0^X, \theta_1^Y$ . We therefore put 0 weight on all other causal types and scale up the remaining probabilities so that they sum to 1 (preserving the ratio between them). The result gives *posterior* probabilities on the causal types. We display an “updated” ambiguity matrix, with excluded data types and causal types removed, in Table 7.3.

Before we see any data on the case at hand, then, we believe (based on our beliefs about the population to which the case belongs) that there is a 1/8 probability that the case is one in which  $X$  is assigned to 1 and has a positive effect on  $Y$ ; and 1/8 probability that it's a case in which  $X$  gets assigned

Table 7.2: Excerpt from the Ambiguity matrix for a chain model. Rows are causal types, columns are data types. Last column shows possible priors over rows.

	X0M0Y0	X1M0Y0	X0M1Y0	X1M1Y0	X0M0Y1	X1M0Y1	prior
X0M00Y00	1	0	0	0	0	0	0.02
X1M00Y00	0	1	0	0	0	0	0.02
X0M10Y00	0	0	1	0	0	0	0.02
X1M10Y00	0	1	0	0	0	0	0.02
X0M01Y00	1	0	0	0	0	0	0.04
X1M01Y00	0	0	0	1	0	0	0.04
X0M11Y00	0	0	1	0	0	0	0.02
X1M11Y00	0	0	0	1	0	0	0.02
X0M00Y10	0	0	0	0	1	0	0.02
X1M00Y10	0	0	0	0	0	1	0.02
X0M10Y10	0	0	1	0	0	0	0.02
X1M10Y10	0	0	0	0	0	1	0.02
X0M01Y10	0	0	0	0	1	0	0.04
X1M01Y10	0	0	0	1	0	0	0.04
X0M11Y10	0	0	1	0	0	0	0.02
X1M11Y10	0	0	0	1	0	0	0.02

to 1 and has no effect on  $Y$  (and so  $Y$  is 1 regardless of  $X$ ). Seeing the  $X = 1, Y = 1$  data, we now believe that there is a 1/2 probability that the case is of the former type, and a 1/2 probability that it is of the latter type. Had our prior beliefs on types been different from each other, the posterior beliefs would have scaled up accordingly.

Table 7.3: . Ambiguities in an  $X \rightarrow Y$  model after observing  $X = 1, Y = 1$  in a case.

Data types →	X1Y1	Priors on causal types	Posteriors on causal types
<b>Causal types ↓</b>			
$\theta_1^X, \theta_{01}^Y$	1	1/8	1/2
$\theta_1^X, \theta_{11}^Y$	1	1/8	1/2

We now walk through how this work for the more complex  $X \rightarrow M \rightarrow Y$  model, and the ambiguity matrix in Table 7.2. If we observe the data  $X = 1, M = 0, Y = 0$ , for instance, this exercise would yield the updated ambiguity matrix in Table ??(tab:ambigmedupdate). Here we have eliminated all rows (causal types) with a 0 in the relevant data-type column ( $X1M0Y0$ ) and formed the posteriors by scaling up the priors in the retained rows.

Table 7.4: . An updated version of the ambiguity matrix in Table 7.2, after observing  $X = 1, M = 0, Y = 0$  in a case.

Data types →	X1M0Y0	Priors on causal types	Posteriors on causal types
<b>Causal types ↓</b>			
$\theta_1^X, \theta_{00}^M, \theta_{00}^Y$	1	0.02	0.1667
$\theta_1^X, \theta_{10}^M, \theta_{00}^Y$	1	0.02	0.1667
$\theta_1^X, \theta_{00}^M, \theta_{01}^Y$	1	0.04	0.3333
$\theta_1^X, \theta_{10}^M, \theta_{01}^Y$	1	0.04	0.3333

A notable feature of the logic of single-case process tracing is that the relative probabilities on the retained causal types never change. If we start out

believing that causal type  $A$  is twice as likely as causal type  $B$ , and both  $A$  and  $B$  are retained once we see the data, then  $A$  will be twice as likely as  $B$  in our posteriors. All updating occurs by *eliminating* causal types from consideration and zeroing in on those that remain.

	type	X1M0Y0	prior	posterior
X1M00Y00	X1M00Y00	1	0.02	0.1667
X1M10Y00	X1M10Y00	1	0.02	0.1667
X1M00Y01	X1M00Y01	1	0.04	0.3333
X1M10Y01	X1M10Y01	1	0.04	0.3333

#### 7.1.4.1 Partial data

A similar logic applies if partial data are observed: that is, if we do not collect data for all nodes in the model. The one difference is that, now, rather than reducing to one column we entertain the possibility of any data *type* consistent with the *observed data*. In general, more than one data type will be consistent with partial data. For instance, suppose that we observe  $X = 1, Y = 0$  but do not observe  $M$ 's value. These are data that are consistent with both the data type  $X1M0Y0$  and the data type  $X1M1Y0$  (since the unobserved  $M$  could be either 0 or 1). We thus retain both of these data-type columns as well as all causal types consistent with *either* of these data types. This gives the updated ambiguity matrix in Table 7.5. We note that, with these partial data, we are not able to update as strongly. For instance, for the causal type  $\theta_1^X, \theta_{00}^M, \theta_{00}^Y$ , instead of updating to a posterior probability of 0.1667, we update to a posterior of only 0.0833 – because there is a larger set of causal types with which these partial data are consistent.

Table 7.5: . An updated version of the ambiguity matrix in Table 7.2, after observing partial data in case:  $X = 1, Y = 0$ , with  $M$  unobserved.

Data types →	X1M0Y01M1Y0			Priors on causal types	Posteriors on causal types
Causal types ↓					
$\theta_1^X, \theta_{00}^M, \theta_{00}^Y$	1	0	0.02		0.0833
$\theta_1^X, \theta_{10}^M, \theta_{00}^Y$	1	0	0.02		0.0833

Data types →	X1M0Y0	M1Y0	Priors on causal types	Posteriors on causal types
$\theta_1^X, \theta_{01}^M, \theta_{00}^Y$	0	1	0.04	0.1667
$\theta_1^X, \theta_{11}^M, \theta_{00}^Y$	0	1	0.02	0.0833
$\theta_1^X, \theta_{01}^M, \theta_{10}^Y$	0	1	0.04	0.1667
$\theta_1^X, \theta_{11}^M, \theta_{10}^Y$	0	1	0.02	0.0833
$\theta_1^X, \theta_{00}^M, \theta_{01}^Y$	1	0	0.04	0.1667
$\theta_1^X, \theta_{10}^M, \theta_{01}^Y$	1	0	0.04	0.1667

**Updating on queries.** We now have a posterior probability for each causal type for the case at hand. The causal question we are interested in answering, our query, may not be about causal types *per se*. It is about a query that can be expressed as a *combination* of causal types, as described in Chapter 4.

For instance, suppose we are working with the model  $X \rightarrow M \rightarrow Y$ ; and that our question is, “Did  $X = 1$  cause  $Y = 1$ ?” This question is asking both:

1. Does  $X = 1$  in this case?
2. Does  $X$  have a positive effect on  $Y$  in this case?

The causal types that qualify are those, and only those, in which the answer to both is “yes.”

Meeting condition (1) requires that  $\theta_1^X = \theta_1^X$ .

Meeting condition (2) requires that  $\theta^M$  and  $\theta^Y$  are such that  $X$  has an effect on  $M$  that yields a positive effect of  $X$  on  $Y$ . This could occur via a positive  $X \rightarrow M$  effect linked to a positive  $M \rightarrow Y$  effect or via a negative  $X \rightarrow M$  effect linked to a negative  $M \rightarrow Y$  effect.

Thus, the qualifying causal types in this model are:

- $\theta_1^X, \theta_{01}^M, \theta_{00}^Y$
- $\theta_1^X, \theta_{10}^M, \theta_{10}^Y$

Our *prior* on the query—what we believe before we collect data on the case at hand—is given simply by summing up the prior probabilities on each of the causal types that correspond to the query. Note that we must calculate the prior from the full ambiguity matrix, before excluding types for inconsistency with the data. Returning to the full ambiguity matrix in Table 7.2, we see that the priors on these two types (given the population parameters assumed there) are 0.08 and 0.02, respectively, giving a prior for the query of 0.1.

The posterior on any query is, likewise, given by summing up the posterior probabilities on each of the causal types that correspond to the query, drawing of course from the updated ambiguity matrix. For instance, if we observe the data  $X = 1, M = 1, Y = 1$ , we update to the ambiguity matrix in Table 7.6. Our posterior on the query, “Did  $X = 1$  cause  $Y = 1?$ ” is the sum of the posteriors on the above two causal types. Since  $\theta_1^X, \theta_{10}^M, \theta_{10}^Y$  is excluded by the data, this just leaves the posterior on  $\theta_1^X, \theta_{01}^M, \theta_{01}^Y$ , 0.4444, which is the posterior belief on our query.

If we observe only the partial data,  $X = 1, Y = 1$ , then we update to the ambiguity matrix in Table 7.7. Now both corresponding causal types are included, and we sum their posteriors to get the posterior on the query:  $0.08 + 0.31 = 0.39$ .

Table 7.6: . An updated version of the ambiguity matrix in Table 7.2, after observing  $X = 1, M = 1, Y = 1$  in a case.

Data types →	X1M1YPriors on causal types	Posteriors on causal types
<b>Causal types ↓</b>		
$\theta_1^X, \theta_{01}^M, \theta_{01}^Y$	1	0.08
$\theta_1^X, \theta_{11}^M, \theta_{01}^Y$	1	0.04
$\theta_1^X, \theta_{01}^M, \theta_{11}^Y$	1	0.04
$\theta_1^X, \theta_{11}^M, \theta_{11}^Y$	1	0.02

Table 7.7: . An updated version of the ambiguity matrix in Table 7.2, after observing partial data in case:  $X = 1, Y = 0$ , with  $M$  unobserved.

Data types →	X1M0Y01M1Y0	Priors on causal types	Posteriors on causal types
Causal types ↓			
$\theta_1^X, \theta_{00}^M, \theta_{10}^Y$	1	0	0.02
$\theta_1^X, \theta_{10}^M, \theta_{10}^Y$	1	0	0.02
$\theta_1^X, \theta_{01}^M, \theta_{01}^Y$	0	1	0.08
$\theta_1^X, \theta_{11}^M, \theta_{01}^Y$	0	1	0.04
$\theta_1^X, \theta_{00}^M, \theta_{11}^Y$	0	1	0.02
$\theta_1^X, \theta_{10}^M, \theta_{11}^Y$	0	1	0.02
$\theta_1^X, \theta_{01}^M, \theta_{11}^Y$	1	0	0.04
$\theta_1^X, \theta_{11}^M, \theta_{11}^Y$	1	0	0.02

For more complex models and queries, it can be more difficult to eyeball the corresponding causal types. In practice, therefore, we use the `get_query_types` function in the `CausalQueries` package to do this for us.

## 7.2 Mapping from models to classic qualitative tests

The approach we have elaborated here appears different from that described in the literature on process-tracing tests – such as Collier (2011), Bennett (2008), or Humphreys and Jacobs (2015) – in which one seeks specific evidence that is directly informative about causal propositions: “clues” that arise with different probabilities if one proposition or another is true. In fact, however, the approaches are deeply connected. Specifically, we can think of causal models as providing a *justification* for the probative value that researchers assign to clues in the classic approach.

To see this, let’s write down the probability of observing a given clue conditional on a unit’s causal type using the  $\phi$  notation from Humphreys and Jacobs (2015). Here  $\phi_{jx}$  refers to the probability of observing a clue,  $K$ , in

a case of type  $j$  when  $X = x$ . Assuming an  $X \rightarrow M \rightarrow Y$  model and a prior distribution over the lower-level causal types (the  $\lambda$ 's), we can derive, for an  $X = 1$  case, the probability of seeing the clue if the case is of type  $b$  (positive effect) or of type  $d$  (no effect, and  $Y$  always 1):

$$\begin{aligned}\phi_{b1} &= \frac{\lambda_{01}^K \lambda_{01}^Y}{\lambda_{01}^K \lambda_{01}^Y + \lambda_{10}^K \lambda_{10}^Y} \\ \phi_{d1} &= \frac{\lambda_{11}^Y (\lambda_{01}^K + \lambda_{11}^K) + \lambda_{11}^K \lambda_{01}^Y}{\lambda_{11}^Y + \lambda_{00}^K \lambda_{10}^Y + \lambda_{11}^K \lambda_{01}^Y}\end{aligned}\tag{7.1}$$

These quantities allow for easy mapping between our prior beliefs about our causal query—as expressed in the lower-level model—and the classic process-tracing tests in Van Evera (1997). Figure 7.2 illustrates. In each panel, we manipulate a prior for one or more of the lower-level causal effects, keeping all other priors flat, and we see how probative value changes. As the curves for  $\phi_b$  and  $\phi_d$  diverge, probative value is increasing since there is an increasing difference between the probability of seeing the clue if  $X$  has a positive effect on  $Y$  and the probability of seeing the clue if  $X$  has no effect.

In the left panel, we see that as we place a lower prior probability on  $K$ 's being negatively affected by  $X$ ,<sup>4</sup> seeking  $K = 1$  increasingly takes on the quality of a hoop test for  $X$ 's having a positive effect on  $Y$ . The clue, that is, increasingly becomes something we must see if  $X$  positively affects  $Y$ , with the clue remaining moderately probable if there is no effect. Why? The less likely we believe it is that  $K = 0$  was caused by  $X = 1$ , the less consistent the observation of  $K = 0$  is with  $X$  having a positive causal effect on  $Y$  via  $K$  (since, to have such an effect, if  $X = 1$  and  $K = 0$ , would precisely have to mean that  $X = 1$  caused  $K = 0$ ).

In the second graph, we simultaneously change the prior probabilities of zero effects at both stages in the sequence: of  $K$  and  $Y$  being 1 regardless of the values of  $X$  and  $K$ , respectively.<sup>5</sup> We see here that, as the probabilities of zero effects jointly diminish, seeking  $K = 1$  increasingly becomes a smoking-gun test for a positive effect of  $X$  on  $Y$ : the probability of seeing the clue

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<sup>4</sup>For a given value of  $\lambda_{01}^K$ , we hold the other  $\lambda^K$  values equal by assigning a value of  $(1 - \lambda_{01}^K)/3$  to each.

<sup>5</sup>For a given value of  $\lambda_{11}^Y$ , we hold the other  $\lambda^Y$ 's equal by assigning a value of  $(1 - \lambda_{11}^Y)/3$  to each; likewise for  $\lambda_{11}^K$  and the other  $\lambda^K$  values.

if the case is a  $d$  type diminishes. The reason is that, as zero effects at the lower level become less likely, it becomes increasingly unlikely that  $K = 1$  could have occurred without a positive effect of  $X$  on  $K$ , and that  $Y = 1$  could have occurred (given that we have seen  $K = 1$ ) without a positive effect of  $K$  on  $Y$ .

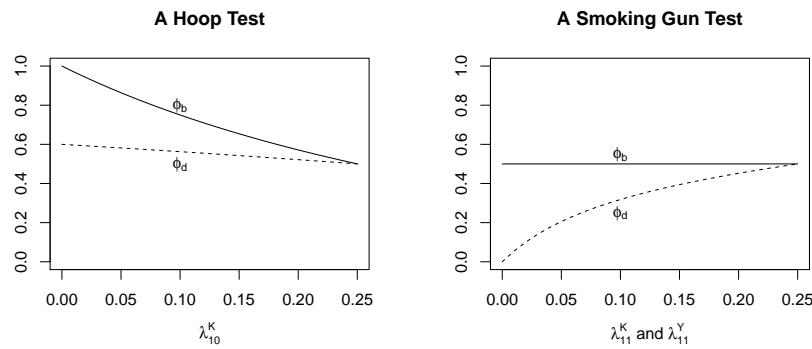


Figure 7.2: The probability of observing  $K$  given causal type for different beliefs on lower-level causal effects. In the left figure, priors on all lower-level causal effects are flat except for the probability that  $X$  has a negative effect on  $K$ . If we believe that it is unlikely that  $X$  has a negative effect on  $K$ ,  $K$  becomes a ‘hoop’ test for the proposition that a case is of type  $b$ . The righthand figure considers simultaneous changes in  $\lambda_{11}^K$  and  $\lambda_{11}^Y$ —the probabilities that  $K = 1$  regardless of  $X$ , and that  $Y = 1$  regardless of  $K$ , with flat distributions on all other lower-level effects. With  $\lambda_{11}^K$ ,  $\lambda_{11}^Y$  both close to 0,  $K$  becomes a ‘smoking gun’ test for the proposition that  $X$  has a positive effect on  $Y$  ( $b$  type).

### 7.3 Assessing the possibility of probative value from a graph

As we have argued, causal queries can be expressed as collections of combinations of nodal types (i.e., as collections of causal types) in a causal model. A nodal type is itself represented as an unobservable node in a model — as a  $\theta^j$  pointing into node  $j$ . Thus, causal inference in this framework is

*the use of observable nodes on a causal graph to assess the value of one or more unobserved nodes on a causal graph.* Placing our queries on the graph together with the observable nodes has the important advantage of allowing us to graphically identify the possibilities for learning about these queries: that is, to say which observable nodes are potentially informative about a given query.

To think through the logic of potential probative value, it is useful to distinguish among three different features of the world, as represented in our causal model: there are the things we want to learn about; the things we have already observed; and the things we could observe. As notation going forward, we let:

- $\mathcal{Q}$  denote the collection of  $\theta^j$  nodes that define our *query*;  $\mathcal{Q}$  cannot be directly observed so that its values must be inferred;
- $\mathcal{W}$  denote a set of previously observed nodes in the causal model; and
- $\mathcal{K}$  denote a set of additional variables—clues—that we have not yet observed but could observe.

Now suppose that we seek to design a research project to investigate a causal question. How should the study be designed? Given that there are some features of the world that we have already observed, which additional clues should we seek to collect to shed new light on our question? In terms of the above notation, what we need to figure out is whether a given  $\mathcal{K}$  might be informative about—might provide additional leverage on— $\mathcal{Q}$  given the prior observation of  $\mathcal{W}$ .

To ask whether one variable (or set of variables) is informative about another is to ask whether the two (sets of) variables are, on average, *correlated* with one another, given whatever we already know. Likewise, if two variables' distributions are fully *independent* of one another (conditional on what else we have observed), then knowing the value of one variable can provide no new information about the value of the other.

Thus, asking whether a set of clues,  $\mathcal{K}$ , is informative about  $\mathcal{Q}$  given the prior observation of  $\mathcal{W}$ , is equivalent to asking whether  $\mathcal{K}$  and  $\mathcal{Q}$  are conditionally independent given  $\mathcal{W}$ . That is,  $\mathcal{K}$  can be informative about  $\mathcal{Q}$  given  $\mathcal{W}$  only if  $\mathcal{K}$  and  $\mathcal{Q}$  are *not* conditionally independent of one another given  $\mathcal{W}$ .

As our discussion of conditional independence in Chapter 2 implies, as long as we have built  $\mathcal{Q}$ ,  $\mathcal{K}$ , and  $\mathcal{W}$  into our causal model of the phenomenon of interest, we can answer this kind of question by inspecting the structure of the model's DAG. In particular, what we need to go looking for are relationships of *d-separation*. The following proposition, with only the names of the variable sets altered, is from Pearl (2009) (Proposition 1.2.4):

**Proposition 1:** If sets  $\mathcal{Q}$  and  $\mathcal{K}$  are *d-separated* by  $\mathcal{W}$  in a DAG,  $\mathcal{G}$ , then  $\mathcal{Q}$  is independent of  $\mathcal{K}$  conditional on  $\mathcal{W}$  in every distribution compatible with  $\mathcal{G}$ . Conversely, if  $\mathcal{Q}$  and  $\mathcal{K}$  are *not d-separated* by  $\mathcal{W}$  in DAG  $\mathcal{G}$ , then  $\mathcal{Q}$  and  $\mathcal{K}$  are dependent conditional on  $\mathcal{W}$  in at least one distribution compatible with DAG  $\mathcal{G}$ .

We begin with a causal graph and a set of nodes on the graph ( $W$ ) that we have already observed. Given what we have already observed, *a collection of clue nodes,  $\mathcal{K}$ , will be uninformative about the query nodes,  $\mathcal{Q}$ , if  $\mathcal{K}$  is d-separated from  $\mathcal{Q}$  by  $\mathcal{W}$  on the graph.* (Equivalently,  $\mathcal{K}$ , will be uninformative about  $\mathcal{Q}$ , given that we have already observed  $\mathcal{W}$ , if  $\mathcal{K}$  and  $\mathcal{Q}$  are conditionally independent given  $\mathcal{W}$ .) When  $\mathcal{W}$  *d-separates*  $\mathcal{K}$  from  $\mathcal{Q}$ , this means that what we have already observed *already* captures all information that the clues might yield about our query. On the other hand, if  $\mathcal{K}$  and  $\mathcal{Q}$  are *d-connected* (i.e., not *d-separated*) by  $W$ , then  $K$  is *possibly* informative about  $Q$ .  $K$  is not *d-separated* from  $Q$  by  $W$ .<sup>6</sup> Note, moreover, that under quite general conditions (referred to in the literature as the *faithfulness* of a probability distribution) then there are at least *some* values of  $\mathcal{W}$  for which  $\mathcal{K}$  *will* be informative about  $\mathcal{Q}$ .<sup>7</sup>

Let us examine Proposition 1 in practice. We begin with the simplest case possible, and then move on to more complex models.

The very simplest probabilistic causal graph, shown in Figure 7.3, has  $X$

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<sup>6</sup>This proposition is almost coextensive with the definition of a DAG. A DAG is a particular kind of dependency model (“graphoid”) that is a summary of a collection of “independency statements”, ( $I$ ), over distinct subsets of  $\mathcal{V}$  (Pearl and Verma 1987), where  $I(\mathcal{Q}, \mathcal{W}, \mathcal{K})$  means “we learn nothing about  $\mathcal{Q}$  from  $\mathcal{K}$  if we already know  $\mathcal{W}$ ”. More formally:  $I(\mathcal{K}, \mathcal{W}, \mathcal{Q}) \leftrightarrow P(\mathcal{K}, \mathcal{Q}|\mathcal{W}) = P(\mathcal{K}|\mathcal{W})P(\mathcal{Q}|\mathcal{W})$ . A Directed Acyclic Graph Dependency model is one where the set of independencies corresponds exactly to the relations that satisfy *d-separation* (Pearl and Verma 1987, p376). Thus on DAG  $\mathcal{G}$ ,  $I(\mathcal{K}, \mathcal{W}, \mathcal{Q})_{\mathcal{G}}$  implies that  $\mathcal{K}$  and  $\mathcal{Q}$  are *d-separated* by  $\mathcal{W}$ .

<sup>7</sup>Put differently, there will not be any conditional independencies that are *not* captured in the DAG.

influencing  $Y$ , with  $X$  determined by a coin flip. If we want to know  $X$ 's effect on  $Y$ , this query is defined solely in terms of  $Y$ 's nodal type,  $\theta^Y$ . To help us conceptualize the more general point about informativeness for queries, we relabel  $\theta^Y$  as  $Q$  to emphasize the fact that this node represents our query.

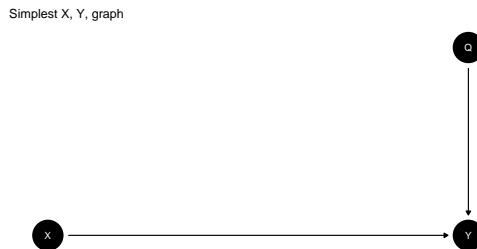


Figure 7.3: A simple causal setup in which the effect of  $X$  on  $Y$  in a given case depends on the case's response type for  $Y$ .

Let us assume that we have observed nothing yet in this case and then ask what clue(s) might be informative about  $Q$ , the node of interest. The other two nodes in the graph are  $X$  and  $Y$ : these are thus the possible clues that we might go looking for in our effort to learn about  $Q$  (i.e., they are the possible members of  $\mathcal{K}$ ).

First, can we learn about  $Q$  by observing  $X$ ? We can answer this question by asking whether  $X$  is  $d$ -connected to  $Q$  on the graph given what we have already observed (which is nothing). We can see visually that there is no active path from  $X$  to  $Q$ : the only path between  $X$  and  $Q$  is blocked by colliding arrow heads. Thus,  $X$  and  $Q$  are  $d$ -separated, meaning that  $X$  will not be informative about  $Q$ : observing the value that a causal variable takes on in a case—having seen nothing else in the case—tells us nothing whatsoever about that variable's effect on the outcome. If we want to know whether a case is of a type in which the presence of natural resources would cause civil war, for instance, observing only that the case has natural resources does not help answer the question.

$\rightarrow$

What, then, if we instead were to observe only  $Y$ ? Is  $Y$   $d$ -connected to  $Q$  given what we have already observed (which, again, is nothing)? It is: the arrow from  $Q$  to  $Y$  is an active path. Observing only the *outcome* in a

case does tell us something about causal effects. Returning to the natural resources and civil war example, observing only that a country has had a civil war is informative about the case's causal type (the value of  $Q$ ). In particular, it rules out the possibility that this is a case in which nothing could cause a civil war: that is, it excludes  $\theta_{00}^Y$  (i.e.,  $c$ -type) as a possible value of  $Q$ .

Suppose now, having observed  $Y$ , that we were to consider also observing  $X$ . Would we learn anything further about  $Q$  from doing so? We have already seen that observing  $X$  alone yields no information about  $Q$  because the two nodes are unconditionally  $d$ -separated, the path between them blocked by the colliding arrowheads at  $Y$ . However, as we have seen, observing a collider variable (or one of its descendants) *unblocks* the flow of information, generating relations of conditional dependence across the colliding arrowheads. Here,  $X$  and  $Q$  are  $d$ -connected by  $Y$ : thus, if we have *already* observed  $Y$ , then observing  $X$  does confer additional information about  $Q$ . Knowing only that a country has natural resources tells us nothing about those resources' effect on civil war in that country. But if we already know that the country has a civil war, then learning that the country has natural resources helps narrow down the case's possible response types. Having already used the observation of  $Y = 1$  to rule out the possibility of  $\theta_{00}^Y$ , observing  $X = 1$  *together with*  $Y = 1$  allows us to additionally rule out the possibility that natural resources *prevent* civil war, i.e., that  $Q = \theta_{01}^Y$ .<sup>8</sup>

Finally, what if we observe  $X$  first and are considering whether to seek information about  $Y$ ? Would doing so be informative?  $X$  does not  $d$ -separate  $Q$  from  $Y$ ; thus, observing  $Y$  will be informative about  $Q$ . In fact, observing  $Y$  if we have already seen  $X$  is *more* informative than observing  $Y$  alone. The reasoning follows the logic of collision discussed just above. If we observe  $Y$  having already seen  $X$ , not only do we reap the information about  $Q$  provided by  $Y$ 's correlation with  $Q$ ; we simultaneously open up the path between  $X$  and  $Q$ , learning additionally from the conditional dependence between  $X$  and  $Q$  given  $Y$ .

We put Proposition 1 to work in a slightly more complex set of models in Figure 7.4. Here we investigate the informativeness of a clue that is neither  $X$  nor  $Y$ . Each graph in Figure 7.4 has four variables:  $X$ ;  $Y$ ; a possible clue,  $K$ ; and a node,  $Q$ , representing the query (which we might also naturally think

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<sup>8</sup>That is, we can rule out that the case is an  $a$  type, or one with a negative causal effect.

of as  $\theta^Y$ ). We draw all 34 possible graphs with variables  $X$ ,  $Y$ ,  $K$ , and  $Q$  for causal models in which (a) all variables are connected to at least one other variable, (b)  $X$  causes  $Y$  either directly or indirectly, and (c)  $Q$  is a direct cause of  $Y$  but is not caused by any other variable in the model and is thus exogenous. The title of each panel reports  $K$ 's conditional informativeness using principles of  $d$ -separation: it tells us when  $K$  is possibly informative about  $Q$  depending on whether  $X$ ,  $Y$ , both or none are observed.<sup>9</sup>

The results show us not just what kinds of variables can be informative about a case's response-type but also what combinations of observations yield leverage on case-level causal effects. A number of features of the graphs are worth highlighting:

- **Clues at many stages.** Process tracing has focused a great deal on observations that lie “along the path” between suspected causes and outcomes. What we see in Figure 7.4, however, is that observations at many different locations in a causal model can be informative about causal effects. We see here that  $K$  can be informative when it is pre-treatment (causally prior to  $X$ —e.g. panel (3)), post-treatment but pre-outcome (that is, “between”  $X$  and  $Y$  as, e.g., in panel (20)), an auxiliary effect of  $X$  that itself has no effect on  $Y$  (e.g., in panel (19)), post-outcome (after  $Y$ —e.g., in panel (15)), or a joint effect of both the suspected cause and the outcome (e.g., panel (31)).
- **Mediator Clues.** While clues that lie in between  $X$  and  $Y$  may be informative, they can only be informative under certain conditions. For instance, when a clue serves *only* as a mediator in our model (i.e., its only linkages are being caused by  $X$  and being affected by  $Y$ ) and  $Q$  only affects  $Y$ , as in panels (20) and (21), the clue is only informative about  $Q$  if we have also observed the outcome,  $Y$ . Of course, this condition may commonly be met—qualitative researchers usually engage in retrospective research and learn the outcome of the cases they are studying early on—but it is nonetheless worth noting why it matters: in this setup,  $K$  is unconditionally  $d$ -separated from  $Q$  by the collision at  $Y$ ; it is only by observing  $Y$  (the collider) that the path between  $K$

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<sup>9</sup>Note the “possibly” can be dropped under the assumption that the underlying probability model is “stable” (Pearl 2009, section 2.9.1) and with the interpretation that  $K$  is informative about  $Q$  for some, but not necessarily all, values of  $W$ .

### 7.3. ASSESSING THE POSSIBILITY OF PROBATIVE VALUE FROM A GRAPH 165

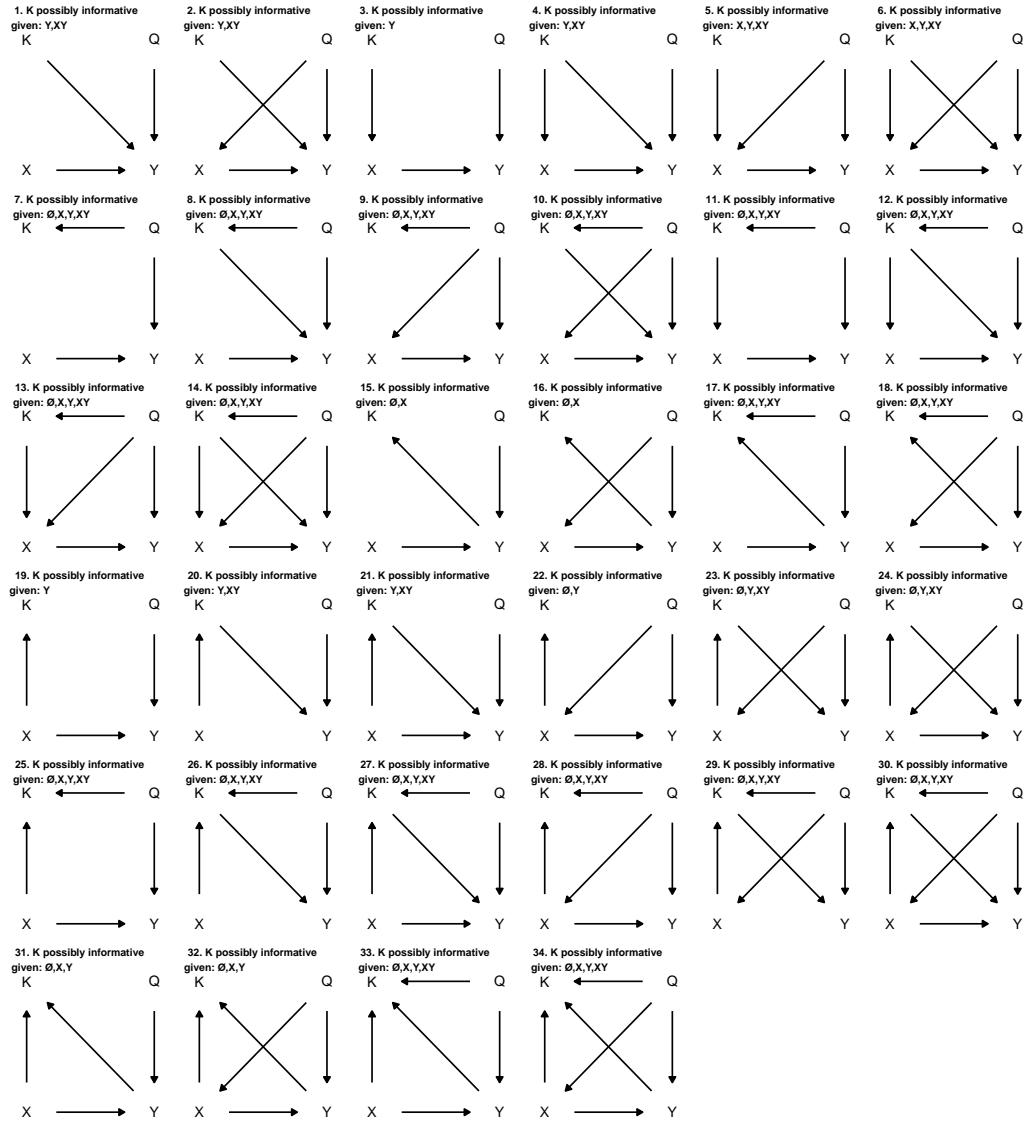


Figure 7.4: All connected directed acyclic graphs over  $X, Y, K, Q$ , in which  $Q$  is an exogenous variable that directly causes  $Y$ , and  $X$  is a direct or indirect cause of  $Y$ . The title of each graph indicates the conditions under which  $K$  can be informative about (i.e., is not  $d$ -separated from)  $Q$ , given the prior observation of  $X, Y$ , both, or neither (...).

and  $Q$  becomes unblocked. (As we saw above, the very same is true for observing  $X$ ; it is only when we know  $Y$  that  $X$  is informative about  $Q$ .)

In short, observations along causal paths are more helpful in identifying causal effects to the extent that we have measured the outcome. Importantly, this is not the same as saying that mediator clues are *only* informative about causal effects where we have observed the outcome. Observing  $Y$  is necessary for the mediator to be informative about a  $Q$  term that is connected only to  $Y$ . Observing a mediator without the outcome, however, could still be informative about the overall effect of  $X$  on  $Y$  by providing leverage on how the mediator responds to  $X$ , which is itself informative about  $X$ 's effect on  $Y$  via the mediator.<sup>10</sup> Moreover, observing the mediator could be informative without the observation of  $Y$  if, for instance,  $Q$  also points into  $K$  itself or into a cause of  $K$ . As we discuss below, the clue then is informative as a “symptom” of the case’s response type, generating learning that does not hinge on observing the outcome.

- **Symptoms as clues.** Some clues may themselves be affected by  $Q$ : that is to say, they may be symptoms of the same conditions that determine causal effects in a case. For instance, in our illustrative model involving government survival, government sensitivity functions as a response-type variable for the effect of a free press ( $X$ ) on government removal ( $Y$ ): a free press only generates government removal when the government is non-sensitive to public opinion. Sensitivity to public opinion thus represents our query variable,  $Q$ , if we seek to learn whether a free press causes government removal in a case. While it may not be possible to observe or otherwise measure the government’s sensitivity, there may be *consequences* of government sensitivity that are observable: for instance, whether government officials regularly consult with civil-society actors on policy issues. While consultations would not be part of the causal chain generating the free press’s effect, observing consultations (or the lack of them) would be informative about that effect because consultations are a symptom of the same conditions that enable the effect.

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<sup>10</sup>In other words, the clue would then be providing leverage on a response-type variable pointing into the mediator itself.

We see that  $K$  is a child or descendant of  $Q$  in several of the graphs in Figure 7.4:  $Q$  directly causes  $K$  in panels (7) through (14), (17), (18), (25)-(30), (33), and (34);  $Q$  causes  $(K)$  only indirectly through  $X$  in panels (22) through (24);  $Q$  causes  $(K)$  only indirectly through  $Y$  in panels (15), (16), and (31); and  $Q$  causes  $K$  only indirectly through  $X$  and through  $Y$  in panel (32). We can then use the principle of  $d$ -separation to figure out when the symptom clue is potentially informative, given what we have already observed. It is easy to see that  $K$  is potentially informative, no matter what we have already observed, if  $K$  is directly affected by  $Q$ ; there is nothing we could observe that would block the  $Q \rightarrow K$  path. Thus,  $Q$ 's “symptom” can, in this setup, contain information about type above and beyond that contained in the  $X$  and  $Y$  values. However, where  $Q$  affects  $K$  only through some other variable, observing that other variable renders  $K$  uninformative by blocking the  $Q$ -to- $K$  path. For instance, where  $Q$  affects  $K$  indirectly through  $X$ , once we observe  $X$ , we already have all the information about  $Q$  that would be contained in  $K$ .

- **Surrogates as clues.** Clues may be consequences of the outcome, as in graphs (15) and (16). If  $K$  is a consequence *only* of  $Y$ , then it will contain no new information about  $Q$  where  $Y$  is already known. However, in situations where the outcome has not been observed,  $K$  can act as a “surrogate” for the outcome and thus yield leverage on  $Q$  (Frangakis and Rubin (2002)). A researcher might, for instance, seek to understand causal effects on an outcome that is difficult to directly observe: consider, for instance, studies that seek to explain ideational change. Ideas themselves, the  $Y$  in such studies, are not directly observable. However, their consequences—such as statements by actors or policy decisions—will be observable and can thus serve as informative surrogates for the outcome of interest.

Clues may similarly serve as surrogates of a cause, as in graphs (19) and (22). Here  $X$  causes  $K$ , but  $K$  plays no role in the causal process generating  $Y$ .  $K$  is of no help if we can directly measure  $X$  since the latter  $d$ -separates  $K$  from  $Q$ . But if an explanatory variable cannot be directly measured—consider, e.g., ideas or preferences as causes—then its consequences, including those that have no relationship to the outcome of interest, can provide leverage on the case-level causal effect.

Clues can also be a consequence of both our suspected cause and the outcome of interest, thus serving as what we might call “double surrogates,” as in panels (31) and (32). Here  $X$  is a direct cause of  $Y$ , and  $K$  is a joint product of  $X$  and  $Y$ . A double surrogate can be informative as long as we have not already observed both  $X$  and  $Y$ . Where data on either  $X$  or  $Y$  are missing, there is an open path between  $K$  and  $Q$ . If we have already observed both, however, then there is nothing left to be learned from  $K$ .

- **Instruments as clues.** Clues that are causally prior to an explanatory variable, and have no other effect on the outcome, can sometimes be informative. Consider, for instance, graph (3). Here  $K$  is the only cause of  $X$ . It can thus serve as a proxy. If we have seen  $X$ , then  $X$  blocks the path between  $K$  and  $Q$ , and so  $K$  is unhelpful.  $K$  can be informative, though, if we have *not* observed  $X$ . Note that informativeness here still requires that we observe  $Y$ . Since  $Y$  is a collider for  $Q$  and the  $K \rightarrow X \rightarrow$  chain, we need to observe  $Y$  in order to  $d$ -connect  $K$  to  $Q$ .

A rather different setup appears in graph (5), where both  $K$  and  $Q$  cause  $X$ . Now the conditions for  $K$ ’s informativeness are broader. Observing  $X$  still makes  $K$  uninformative as a proxy for  $X$  itself. However, because  $X$  is a collider for  $K$  and  $Q$ , observing  $X$  *opens up* a path from  $K$  to  $Q$ , rendering a dependency between them. Still, we have to observe at least one of  $X$  or  $Y$  for the instrument to be informative here. This is because both of  $K$ ’s paths to  $Q$  run through a collision that we need to unblock by observing the collider. For one path, the collider is  $X$ ; for the other path, the collider is  $Y$ .<sup>11</sup>

Other patterns involving instrumentation are also imaginable, though not graphed here. For example, we might have a causal structure that combines instrumentation and surrogacy. Suppose that  $X$  is affected by  $Q$  and by an unobservable variable  $\theta_X$ ; and that  $\theta_X$  has an observable consequence,  $K$ .

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<sup>11</sup>As a simple example one might imagine a system in which  $X = K$  if  $q \in a, b$  and  $X = 1 - K$  if  $q \in c, d$ . Then if we observe, say,  $X = Y = K = 1$ , we can infer that  $q = b$ . Another way to think about what is happening in graph (5) is that  $K$  is providing information about the *assignment process*. In this graph, the causal effect ( $Y$ ’s potential outcomes, determined by  $Q$ ) is also a partial determinant of the assignment of cases to values on  $X$ . In terms of cross-case correlational inference, then, we would think of this as a situation of confounding. Observing another cause of  $X$ , then, allows us to more fully characterize the process of assignment.

Then  $K$ , though not a cause of  $X$ , is a “surrogate instrument” (Hernán and Robins, 2006) as it is a descendant of an unobserved instrument,  $U$ , and thus allows us to extract inferences similar to those that we could draw from a true instrument.

- **Confounders as clues.** In several of the graphs,  $K$  is a confounder in that it is a direct cause of both  $X$  and  $Y$  (panels (4), (6), (12), and (14)). Let us focus on graph (4), which isolates  $K$ ’s role as a confounder. Here  $K$  can be informative via two possible paths. First, if  $X$  is not observed but  $Y$  is, then  $K$  is  $d$ -connected to  $Q$  along the path  $K \rightarrow X \rightarrow Y \leftarrow Q$ .  $K$  is in this sense serving as a proxy for  $X$ , with its path to  $Q$  opened up by the observation of the collider,  $Y$ . Second, with  $Y$  observed,  $K$  can provide information on  $Q$  via the more direct collision,  $K \rightarrow Y \leftarrow Q$ . If  $X$  is observed, then the first path is blocked, but the second still remains active. As with any pre-outcome variable, for a confounder clue to provide purchase on  $Y$ ’s response type,  $Y$  itself must be observed.

In a sense, then, the role of confounders as clues in case-level inference is the mirror image of the role of confounders as covariates in cross-case correlational inference. In a correlational inferential framework, controlling for a variable in  $K$ ’s position in graph (5) renders the  $X, Y$  correlation (which we assume to be observed) informative about  $X$ ’s average causal effect. When we use confounders as evidence in within-case inference, it is our observations of other variables that determine how informative the confounder *itself* will be about  $X$ ’s causal effect.

It is important to be precise about the kinds of claims that one can make from graphs like those in Figure {fig:34graphs}. The graphs in this figure allow us to identify informativeness about an unobserved node  $Q$  that is a parent of  $Y$ . This setup does not, however, capture all ways in which clues can be informative about the causal effect of  $X$  on  $Y$  or about other causal queries of interest. For instance, as noted above, even if a clue is uninformative about a  $Q$  node pointing into  $Y$ , it may still help establish whether  $X$  causes  $Y$ : the statement that  $X$  causes  $Y$  will for some graphs be a statement about a *collection* of nodes that form the set of query variables  $\mathcal{Q}$ . This is the case, for instance, in any graph of the form  $X \rightarrow M \rightarrow Y$ , where we are interested not just in  $Y$ ’s response to  $M$  (the mediator) but also in  $M$ ’s response to

$X$ . Of interest, thus, are not just a  $Q^Y$  response-type node pointing into  $Y$  but also a  $Q^M$  response-type node that is a parent of  $M$ . Observations that provide leverage on either  $Q$  term will thus aid an inference about the overall causal effect. A clue  $K$  that is  $d$ -separated from  $Q^Y$  may nevertheless be informative about  $X$ 's effect on  $Y$  if it is not  $d$ -separated from  $Q^M$ ; this opens up a broader range of variables as informative clues.

Additionally, as our discussion in Chapter 2 makes clear, queries other than the case-level causal effect—such as average causal effects, actual causes, and causal paths—involve particular features of context: particular sets of exogenous nodes as members of our query set,  $\mathcal{Q}$ . Thus, even for the same causal model, informativeness will be defined differently for each causal question that we seek to address. The broader point is that we can identify what kinds of observations may address our query if we can place that query on a causal graph and then assess the graph for relationships of  $d$ -separation and -connection.

Further, we emphasize that a DAG can only tell us when a clue *may* be informative (conditional some prior observation):  $d$ -connectedness is necessary but not sufficient for informativeness. This fact derives directly from the rules for drawing a causal graph: the absence of an arrow between two variables implies that they are *not* directly causally related, while the presence of an arrow does not imply that they always are. As we saw in our analysis of the government-removal example in Chapter 2, whether variables connected to one another by arrows in the original DAG were in fact linked by a causal effect depended on the context. Likewise, whether a clue  $K$  is in fact informative may depend on particular values of  $\mathcal{W}$ —the variables that have already been observed. As a simple example, let  $q = k_1 w + (1 - w)k_2$ , where  $W$  is a variable that we have already observed and  $K_1$  and  $K_2$  are clues that we might choose to observe next. Here, if  $w = 1$  then learning  $K_1$  will be informative about  $Q$ , and learning  $K_2$  will not; but if  $w = 0$ , then  $K_1$  will be uninformative (and  $K_2$  informative).

In general, then, graphical analysis alone can help us exclude unhelpful research designs, given our prior observations and a fairly minimal set of prior beliefs about causal linkages. This is no small feat. But identifying those empirical strategies that will yield the *greatest* leverage requires engaging more deeply with our causal model, as we show in detail in our discussion of clue-selection in Chapter ??.

## 7.4 Principles of learning

While `CausalQueries` can implement process-tracing inference for us, it is helpful for researchers to be able to reason their way through what is happening “under the hood.” We provide here some core principles and intuitions for thinking through the features of models and queries that influence whether and how much we can learn from within-case observations.

### 7.4.1 A DAG alone does not guarantee probative value for a single case

A DAG puts qualitative structure on causal relations but quantitative implications depend on the beliefs over causal types. In general, learning from new data requires that, conditional on known data, the probability of a new data pattern is different depending on whether or not the query is true. With flat priors this condition may not hold for many queries of interest.

To illustrate, suppose that we are interested in whether  $X$  caused  $Y$  and we posit a simple  $X \rightarrow M \rightarrow Y$  model with flat priors over  $\theta^M$  and  $\theta^Y$ . Now we would like to conduct process tracing and observe  $M$  to tell us about the effect of  $X$  on  $Y$  in a case with  $X = Y = 1$  case.

Does the observation of  $M$  provide leverage on whether  $X = 1$  caused  $Y = 1$ ?

It does not. We can learn nothing about  $X$ ’s effect on  $Y$  from observing  $M$ .

To see why, consider that there are two causal types that will satisfy the query,  $X = 1$  caused  $Y = 1$ . Those are the types  $\theta_1^X \theta_{01}^M \theta_{01}^Y$  and  $\theta_1^X \theta_{10}^M \theta_{10}^Y$ : either linked positive effects or linked negative effects could generate an overall positive effect of  $X$  on  $Y$ . Moreover, with flat priors over nodal types, these causal types are equally likely. Now, think about what we would conclude if we collected process data and observed  $M = 1$  in the  $X = Y = 1$  case. This observation would rule out various ways in which  $X$  did not cause  $Y$  but it also rules out one way in which the query could be satisfied: the causal type with linked negative effects. And what if we observed, instead,  $M = 0$ ? This would have similar implications, this time ruling out the other way in which the query could be satisfied: linked positive effects. Intuitively we would update the same way no matter what we find, which means we must not be updating at all.

More formally, conditional on observing  $X = 1, Y = 1$  our prior that  $X$  caused  $Y$  is:

$$\frac{\lambda_{01}^M \lambda_{01}^Y + \lambda_{10}^M \lambda_{10}^Y}{(\lambda_{01}^M + \lambda_{11}^M)(\lambda_{01}^Y + \lambda_{11}^Y) + (\lambda_{10}^M + \lambda_{00}^M)(\lambda_{10}^Y + \lambda_{11}^Y)}$$

Our posterior on observing  $M = 1$  is:

$$\frac{\lambda_{01}^M \lambda_{01}^Y}{(\lambda_{01}^M + \lambda_{11}^M)(\lambda_{01}^Y + \lambda_{11}^Y)}$$

it is easy to see these are equal with flat priors ( $\lambda_{ab}^j = \lambda^{j*}$  for all  $a, b$ ). What we can see from the comparison is that when we observe data we rule out half the types consistent with the data (denominator) but also rule out half the types consistent with the query *and* data (numerator) .

It is worth noting however that informative priors on *either*  $\theta^M$  or  $\theta^Y$ , would help here.

More generally, what we need at the level of priors depends on the query. Suppose that we start with the model,  $X \rightarrow M \rightarrow Y$ , and formulate the following query: does  $X$  have a positive effect on  $Y$  that runs through a chain of positive effects via  $M$ ? We can learn about this query without any informative priors over nodal types because of the way in which the query itself restricts the type space. Since the query is not satisfied if negative mediating effects are operating, we will update to probability 0 on the query for any observation that violates  $X = M = Y$ , and we will update upwards on the query for any observation of  $X = M = Y$ .

### 7.4.2 Learning requires uncertainty

While case-level inference from within-case evidence requires informative priors about nodal types, there is also such a thing as *too much* information – or, put differently, as insufficient uncertainty about causal relations. Suppose, for instance, that our beliefs are such that  $X$  always has a positive effect on  $M$  in an  $X \rightarrow M \rightarrow Y$  model. Consider, further, that we already know that  $X = 1$  in a case. In that situation, nothing can be learned by observing  $M$  since the prior observation of  $X$  already reveals  $M$ 's value given our prior beliefs.

To take a less extreme example, suppose that our priors put a *very high probability* on  $X$ 's having a positive effect on  $M$  and that, again, we already know that  $X = 1$  in a case. In that situation, we should *expect* to learn very little from observing  $M$  since we believe that we are very likely to see  $M = 1$ , given that we already know  $X = 1$ . It is true that our beliefs will shift *if* we look for  $M$  and find the unexpected value of  $M = 0$ . But because that data-realization is highly unlikely, we should expect the learning from observing  $M$  to be minimal.

We address the concept of expected learning more systematically in Chapters ?? and ??, but our general point here is that, we will learn more from process-tracing evidence, to the extent that (a) we know enough about causal relations in a domain to know how to make causal sense of the evidence we find, but (b) we do not know so much that that evidence can be largely predicted from what we have already observed.

### 7.4.3 The more specific the query the more difficult it is to gain leverage

It is difficult to get empirical leverage on very highly queries.

To illustrate, suppose that we start with the two-path model,  $X \rightarrow M \rightarrow Y \leftarrow X$ , and formulate the following query: does  $X$  have a positive effect on  $Y$  that runs through a chain of positive effects via  $M$ ? Suppose that we begin with flat priors over all nodal types. Intuitively, this seems like exactly the kind of question for which an observation of  $M$  is the perfect empirical strategy. And that intuition is, in a sense, correct: we can indeed learn about the query by observing  $M$ . Seeing  $M = 1$  in an  $X = Y = 1$  case, for instance, would be evidence consistent with the query while seeing  $M = 0$  in that same case would be inconsistent with the query.

Yet, we will only learn very modestly about the query from this observation. The reason is that the query itself has a very low prior probability. It may, in fact, not be obvious at first glance just how unlikely our query is in our priors. At first glance, it looks as though all we are asking is whether there exist positive effects running through one of the two causal paths in the model. However, consider the joint nodal-type probabilities implied by the query. First, the query requires  $X$  to have a positive effect on  $M$ , to which our priors give only a 25% chance. In addition, the query puts a very narrow

constraint on  $Y$ 's possible nodal types: to satisfy the query,  $Y$  must have a nodal type in which  $M$  has a positive effect on  $Y$  when  $X$  does not change, and in which  $X$  does not have a positive effect on  $Y$  unless  $M$  changes from 0 to 1. This pair of conditions is met by only 2 of  $Y$ 's 16 nodal types, implying a 12.5% chance. The prior on the query itself is, thus,  $0.25 \times 0.125 = 0.03125$ . Thus, while observing  $M = 0$  takes the probability of the query down to 0%, we started out very close to 0%! Observing  $M = 1$  results in only a small uptick, to about 6% because there remain many type combinations consistent with  $M=1$  but that do not fit through the needle-eye of this query.

In sum, what seems intuitively like a simple question will sometimes be a very unlikely query. What makes this query so unlikely is the interplay between the query itself and the model. The model allows for a *wide* range of causal combinations (e.g.,  $Y$ 's 16 nodal types), spreading prior weight thinly across those many possibilities, while the query zeroes in on a couple of *particular* combinations that, in our priors, each have very low probability.

#### 7.4.4 Population-level uncertainty does not alter case-level causal inference

In the procedure described for process tracing in this chapter (and different to what we introduce in Chapter 8) we assume that  $\lambda$  is known and we do not place uncertainty around it.

This might appear somewhat heroic, but in fact for single case inference it is without loss of generality. The expected inferences we would make for any query accounting for priors is the same as the inferences we if we use the expectation only.

To see this, let  $\pi_j$  denote the probability of observing causal type  $j$  and  $p(D)$  te probability of observing data realization  $D$ . Say that  $j \in D$  if type  $j$  produces data type  $D$  and say  $j \in E$  if causal type  $j$  is an element of the query set of interest. The posterior on a query  $E$  given data  $D$  given prior over  $\pi$ ,  $p(\pi)$  is:

$$\Pr(E|D) = \int_{\pi} \frac{\sum_{j \in E \cap D} \pi_j}{\sum_{j \in D} \pi_j} f(\pi) d\pi$$

However, since for any  $\pi$ ,  $\sum_{j \in D} \pi_j = p(D)$  we have:

$$\Pr(E|D) = \int_{\pi} \sum_{j \in E \cap D} \pi_j f(\pi) d\pi / p(D) = \sum_{j \in E \cap D} \bar{\pi}_j / p(D)$$

For instance in an  $X \rightarrow Y$  model, if we observe  $X = Y = 1$  then  $D$  consists of causal types  $D = (\theta_1^X, \theta_{01}^Y), (\theta_1^X, \theta_{11}^Y)$  and the query set for “ $X$  has a positive effect on  $Y$ ” consists of  $E = (\theta_1^X, \theta_{01}^Y), (\theta_0^X, \theta_{01}^Y)$ . Let  $\pi_1, \pi_2$  denote the priors on the two elements of  $D$ . We then have:

$$\Pr(E|D) = \frac{\pi_1}{\pi_1 + \pi_2}$$



# Chapter 8

## Process Tracing Application: Inequality and Democracy

In this chapter, we demonstrate how causal-model-based process-tracing works using real data. We undertake this illustration on a substantive issue that has been of central interest to students of comparative politics for decades: the causes of democratization. As the literature and range of arguments about democratization are vast, we focus on just a piece of the debate—specifically on causal claims about the relationship between economic inequality and democratization, with particular attention to the work of Boix (2003), Acemoglu and Robinson (2005), and Haggard and Kaufman (2012). In this chapter, we demonstrate process tracing with causal models, while in a later chapter we demonstrate the integration of process-tracing with correlational analysis. Our focus in this chapter is on using process tracing to assess *the case-level causal effect of inequality on democracy*.

### 8.1 Inequality and Democratization: The Debate

Sociologists, economists, and political scientists have long theorized and empirically examined the relationship between inequality and democracy (e.g., Dahl (1973), Bollen and Jackman (1985), Acemoglu and Robinson (2005),

Boix (2003), Ansell and Samuels (2014)). In recent years, the work of Boix (2003), Acemoglu and Robinson (2005), and Ansell and Samuels (2014) represent major theoretical advances in specifying when and how inequality might generate transitions to democracy (as well as its persistence, which we bracket here). The first and third of these books also provide large-n cross-national and historical tests of their theories' key correlational predictions. Haggard and Kaufman (2012), moreover, derive causal process observations from a large number of "Third Wave" cases of democratization in order to examine these theories' claims about the centrality of distributional issues to regime change. We provide a very condensed summary of the core logic of Boix (2003) and Acemoglu and Robinson (2005) before seeking to translate that logic into a causal model for the purposes of process tracing, using a transformed version of Haggard and Kaufman's causal-process data.

We briefly summarize the core logics of and differences among these three sets of arguments here, bracketing many of their moving parts to focus on the basic theorized relationship between inequality and democracy. Both Boix's and Acemoglu and Robinson's theories operate within a Meltzer-Richard (Meltzer and Richard (1981)) framework in which, in a democracy, the median voter sets the level of taxation-and-transfer and, since mean income is higher than median income, benefit from and vote for a positive tax rate, implying redistribution from rich to poor. The poorer the median voter, the more redistribution she will prefer. Democracy, with its poorer median voter, thus implies greater redistribution than (rightwing) authoritarianism—a better material position from the poor at the expense of the rich elite. Thus, in each of these approaches, struggles over political regimes are conflicts over the distribution of material resources.

In Boix's model, the poor generally prefer democracy for its material benefits. When they mobilize to demand regime change, the rich face a choice as to whether to repress or concede, and they are more likely to repress as inequality is higher since, all else equal, they have more to lose from democracy. Thus, with the poor always preferring democracy over rightwing authoritarianism, inequality reduces the prospects for democratization.

In Acemoglu and Robinson's model, inequality simultaneously affects the expected net gains to democracy for both rich and poor. At low levels of inequality, democracy is relatively unthreatening to the elite, as in Boix, but likewise of little benefit to the poor. Since regime change is costly, the poor

do not mobilize for democracy when inequality is low, and democratization does not occur. At high levels of inequality, democracy is of great benefit to the poor but has high expected costs for the elite; thus, democratization does not occur because the elite repress popular demands for regime change. In Acemoglu and Robinson's model, democracy emerges only when inequality is at middling levels: high enough for the poor to demand it and low enough for the rich to be willing to concede it.

Ansell and Samuels, finally, extend the distributive politics of regime change in two key ways. First, they allow for a two-sector economy, with a governing elite comprising the landed aristocracy and an urban industrial elite excluded from political power under authoritarian institutions. Total inequality in the economy is a function of inequality in the landed sector, inequality in the industrial sector, and the relative size of each. Second, authoritarian (landed) elites can tax the industrial bourgeoisie, thus giving the industrial elite an incentive to seek constraints on autocratic rule. Third, in Ansell and Samuels' model, rising industrial inequality means a rising industrial elite, generating a larger gap between them and industrial workers, though the industrial masses are richer than the peasantry. A number of results follow, of which we highlight just a couple. Rising land inequality reduces the likelihood of bourgeois rebellion by giving the landed elite greater repressive capacities and increasing their expected losses under democracy. As industrial inequality rises, however, the industrial elite have more to lose to confiscatory taxation and thus greater incentive to push for partial democracy (in which they have the ability to constrain the government, though the poor remain politically excluded) as well as greater resources with which to mobilize and achieve it. Full democracy, brought on by joint mass and bourgeois rebellion, is most likely as the industrial sector grows in relative size, giving the urban masses more to lose to autocratic expropriation and more resources with which to mobilize and rebel.

These three theoretical frameworks thus posit rather differing relationships between inequality and democracy. Taking these theoretical logics as forms of background knowledge, we would consider it possible that inequality reduces the likelihood of democracy or that it increases the likelihood of democracy. Yet one feature that all three theories have in common is a claim that distributional grievances drive demands for regime change. Moreover, in both Boix and Acemoglu and Robinson, less economically advantaged groups are, all else equal, more likely to demand democracy the worse their relative eco-

nomic position. Ansell and Samuels' model, on the other hand, suggests that relative deprivation may cut both ways: while poorer groups may have more to gain from redistribution under democracy, better-off groups have more to fear from confiscatory taxation under autocracy. In all three frameworks, *mobilization* by groups with material grievances is critical to transitions to democracy: elites do not voluntarily cede power.

In their qualitative analysis of “Third Wave” democratizations, Haggard and Kaufman point to additional factors, aside from inequality, that may generate transitions. Drawing on previous work on 20th century democratic transitions (e.g., Huntington (1993), Linz and Stepan (1996)), they pay particular attention to international pressures to democratize and to elite defections.

## 8.2 A Structural Causal Model

We now need to express this background knowledge in the form of a structural causal model. Suppose that we are interested in the case-level causal effect of inequality on democratization of a previously autocratic political system. Suppose further, to simplify the illustration, that we conceptualize both variables in binary terms: inequality is either high or low, and democratization either occurs or does not occur. This means that we want to know, for a given case of interest, whether high inequality (as opposed to low inequality) causes democracy to emerge, prevents democracy from emerging, or has no effect (i.e., with democratization either occurring or not occurring independent of inequality). We can represent this query in the simple, high-level causal model shown in Figure 8.1. Here, the question, “What is the causal effect of high inequality on democratization in this case?” is equivalent to asking what the value of  $\theta^D$  is in the case, where the possible values are  $\theta_{00}^D$ ,  $\theta_{01}^D$ ,  $\theta_{10}^D$ , and  $\theta_{11}^D$ . We assume here that the case’s nodal type,  $\theta^D$ , is not itself observable, and thus we are in the position of having to make inferences about it.

Drawing on the grammar of causal graphs discussed in Chapter 2, we can already identify possibilities for learning about  $\theta^D$  from the other nodes represented in this high-level graph. Merely observing the level of inequality in a case will tell us nothing since  $I$  is not  $d$ -connected to  $\theta^D$  if we have observed nothing else. On the other hand, only observing the outcome—regime type—in a case *can* give us information about  $\theta^D$  since  $D$  is  $d$ -connected

to  $\theta^D$ . For instance, if we observe  $D = 1$  (that a case democratized), then we can immediately rule out  $\theta_{00}^D$  as a value of  $\theta^D$  since this type does not permit democratization to occur. Further, conditional on observing  $D$ ,  $I$  is now  $d$ -connected to  $\theta^D$ : in other words, having observed the outcome, we can additionally learn about the case's type from observing the status of the causal variable. For example, if  $D = 1$ , then observing  $I = 1$  allows us additionally to rule out the value  $\theta_{10}^D$  (a negative causal effect).

Now, observing just  $I$  and  $D$  alone will always leave two nodal types in contention. For instance, seeing  $I = D = 1$  (the case had high inequality and democratized) would leave us unsure whether high inequality caused the democratization in this case ( $\theta^D = \theta_{01}^D$ ) or the democratization would have happened anyway ( $\theta^D = \theta_{11}^D$ ). This is a limitation of  $X, Y$  data that we refer to in Humphreys and Jacobs (2015) as the “fundamental problem of type ambiguity.” Note that this does not mean that we will be left indifferent between the two remaining types. Learning from  $X, Y$  data alone—narrowing the types down to two—can be quite significant, depending on our priors over the distribution of types. For example, if we previously believed that a  $\theta_{00}^D$  type (cases in which democracy will never occur, regardless of inequality) was much more likely than a  $\theta_{11}^D$  type (democracy will always occur, regardless of inequality) and that positive and negative effects of inequality were about equally likely, then ruling out the  $\theta_{00}^D$  and  $\theta_{10}^D$  values for a case will shift us toward the belief that inequality caused democratization in the case. This is because we are ruling out both a negative effect and the type of null effect that we had considered the most likely, leaving a null effect that we consider relatively unlikely.

Nonetheless, we can increase the prospects for learning by *theorizing* the relationship between inequality and democratization. Given causal logics and empirical findings in the existing literature, we can say more than is contained in Figure 8.1 about the possible structure of the causal linkages between inequality and democratization. And we can embed this prior knowledge of the possible causal relations in this domain in a lower-level model that is consistent with the high-level model that most simply represents our query.

If we were to seek to fully capture them, the models developed by Boix, Acemoglu and Robinson, and Ansell and Samuels would, each individually, suggest causal graphs with a large number of nodes and edges connecting them. Representing all variables and relationships jointly contained in these

A high-level model of democratization

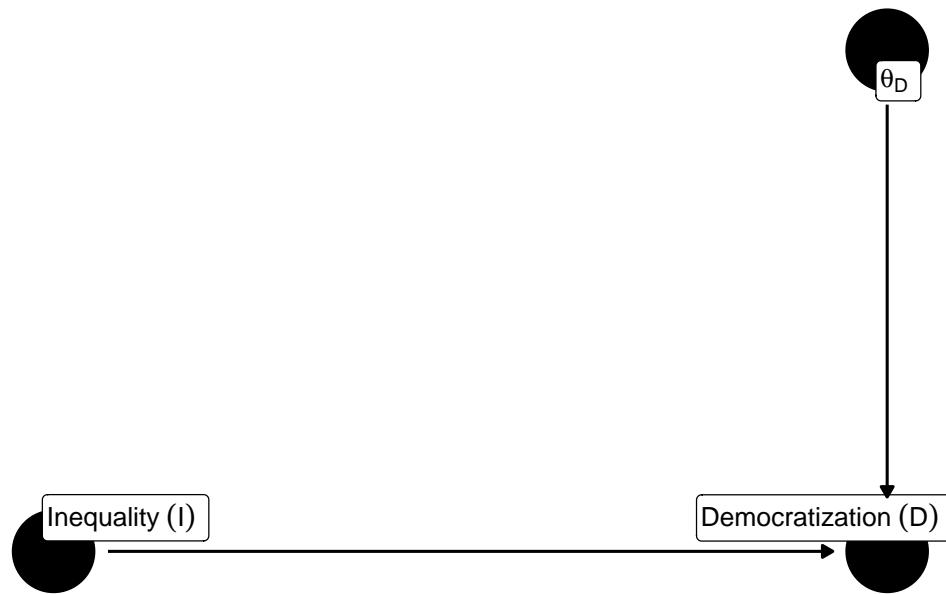


Figure 8.1: Simple democracy, inequality model

three models would take an extremely complex graph. Yet there is no need to go down to the lowest possible level—to generate the *most* detailed graph—in order to increase our empirical leverage on the problem.

We represent in Figure 8.2 one possible lower-level model consistent with our high-level model. Drawing on causal logics in the existing literature, we unpack the nodes in the high-level model in two ways:

1. We interpose a mediator between inequality and democratization: mobilization ( $M$ ) by economically disadvantaged groups expressing material grievances.  $M$  is a function of both  $I$  and of its own response-type variable,  $\theta^M$ , which defines its response to  $I$ . In inserting this mediator, we have extracted  $\theta^M$  from  $\theta^D$ , pulling out that part of  $D$ 's response to  $I$  that depends on  $M$ 's response to  $I$ .
2. We specify a second influence on democratization, international pressure ( $P$ ). Like  $\theta^M$ ,  $P$  has also been extracted from  $\theta^D$ ; it represents that part of  $D$ 's response to  $I$  that is conditioned by international pressures.

```
## Warning: Unknown or uninitialised column:
## `segment.alpha`.

## Warning: Unknown or uninitialised column:
## `segment.alpha`.

## Warning: Unknown or uninitialised column:
## `segment.alpha`.
```

In representing the causal dependencies in this graph, we allow for inequality to have (in the language of mediation analysis) both an “indirect” effect on democratization via mobilization and a “direct” effect. The arrow running directly from  $I$  to  $D$  allows for effects of inequality on democratization beyond any effects running via mobilization of the poor, including effects that might run in the opposite direction. (For instance, it is possible that inequality has a positive effect on democratization via mobilization but a negative effect via any number of processes that are not explicitly specified in the model.)

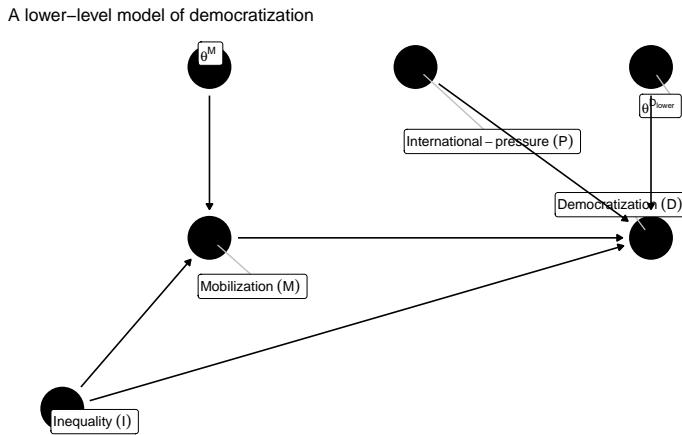


Figure 8.2: A lower-level model of democratization in which inequality may affect regime type both directly and through mobilization of the lower classes, and international pressure may also affect regime type.

The graph also implies that there is no confounding: since there is no arrow running from another variable in the graph to  $I$ ,  $I$  is modeled as exogenous.

The lower-level graph thus has two exogenous, response-type nodes that will be relevant to assessing causal effects:  $\theta^M$  and  $\theta^{D_{lower}}$ .  $\theta^M$ , capturing  $I$ 's effect on  $M$ , ranges across the usual four values for a single-cause, binary setup:  $\theta_{00}^M, \theta_{01}^M, \theta_{10}^M$ , and  $\theta_{11}^M$ .

$\theta^{D_{lower}}$  is considerably more complicated, however, because this node represents  $D$ 's response to three causal variables:  $I$ ,  $M$ , and  $P$ . One way to put this is that the values of  $\theta^{D_{lower}}$  indicate how inequality's direct effect will depend on mobilization (and vice-versa), conditional on whether or not there is international pressure. We need more complex notation than that introduced in Chapter 5 in order to represent the possible response types here.

The result is  $2^8 = 256$  possible response types for  $D$ . With 4 response types for  $M$ , we thus have 1024 possible combinations of causal effects between named variables in the lower-level graph. How do these lower-level response types map onto the higher-level response types that are of interest? In other words, which combinations of lower-level types represent a positive, negative, or zero causal effect of inequality on democratization?

To define a causal effect of  $I$  in this setup, we need to define the “joint effect” of two variables as being the effect of changing both variables simultaneously (in the same direction, unless otherwise specified). Thus, the joint effect of  $I$  and  $M$  on  $D$  is positive if changing both  $I$  and  $M$  from 0 to 1 changes  $D$  from 0 to 1. We can likewise refer to the joint effect of an increase in one variable and a decrease in another. Given this definition, a positive causal effect of inequality on democratization emerges for any of the following three sets of lower-level response patterns:

1. **Linked positive mediated effects.**  $I$  has a positive effect on  $M$ ; and  $I$  and  $M$  have a *joint* positive effect on  $D$  when  $P$  takes on whatever value it takes on in the case.
2. **Linked negative mediated effects**  $I$  has a negative effect on  $M$ ; and  $I$  and  $M$  have a *joint* negative effect on  $D$  when  $P$  takes on whatever value it takes on in the case.
3. **Positive direct effect**  $I$  has no effect on  $M$  and  $I$  has a positive effect on  $D$  at whatever value  $M$  is fixed at and whatever value  $P$  takes on in the case.

If we start out with a case in which inequality is high and democratization has not occurred (or inequality is low and democratization *has* occurred), we will be interested in the possibility of a negative causal effect. A negative causal effect of inequality on democratization emerges for any of the following three sets of lower-level response patterns:

4. **Positive, then negative mediated effects**  $I$  has a positive effect on  $M$ ; and  $I$  and  $M$  have a *joint* negative effect on  $D$  when  $P$  takes on whatever value it takes on in the case.
5. **Negative, then joint negative mediated effects**  $I$  has a negative effect on  $M$ ; and jointly increasing  $I$  while decreasing  $M$  generates a decrease in  $D$  when  $P$  takes on whatever value it takes on in the case.
6. **Negative direct effects**  $I$  has no effect on  $M$  and  $I$  has a negative effect on  $D$  at whatever value  $M$  is fixed at and whatever value  $P$  takes on in the case.

Finally, all other response patterns yield *no* effect of inequality on democratization.

Thus, for a case in which  $I = D = 1$ , our query amounts to assessing the probability that  $\theta^M$  and  $\theta_{lower}^D$  jointly take on values falling into conditions 1, 2, or 3. And for a case in which  $I \neq D$ , where we entertain the possibility of a negative effect, our query is an assessment of the probability of conditions 4, 5, and 6.

### 8.2.1 Forming Priors

We now need to express prior beliefs about the probability distribution from which values of  $\theta^M$  and  $\theta_{lower}^D$  are drawn. We place structure on this problem by drawing a set of beliefs about the likelihood or monotonicity of effects and interactions among variables from the theories in Boix, Acemoglu and Robinson, and Ansell and Samuels. As a heuristic device, we weight more heavily those propositions that are more widely shared across the three works than those that are consistent with only one of the frameworks. We intend this part of the exercise to be merely illustrative of how one might go about forming priors from an existing base of knowledge; there are undoubtedly other ways in which one could do so from the inequality and democracy literature.

Specifically, the belief that we embed in our priors about  $\theta^M$  is:

- **Monotonicity of  $I$ 's effect on  $M$ :** In Acemoglu and Robinson, inequality should generally increase the chances of—and, in Boix, should never prevent—mobilization by the poor. Only in Ansell and Samuels' model does inequality have a partial downward effect on the poor's demand for democracy insofar as improved material welfare for the poor increases the chances of autocratic expropriation; and this effect is countervailed by the greater redistributive gains that the poor will enjoy under democracy as inequality rises.<sup>1</sup> Consistent with the weight of prior theory on this effect, in our initial run of the analysis, we rule

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<sup>1</sup>In addition, as the industrial bourgeoisie become richer, which increases the Gini, this group faces a greater risk of autocratic expropriation. If we consider the rising bourgeoisie's mobilization to be mobilization by a materially disadvantaged group, then this constitutes an additional positive effect of inequality on mobilization.

out negative effects of  $I$  on  $M$ . We are indifferent in our priors between positive and null effects and between the two types of null effects (mobilization always occurring or never occurring, regardless of the level of inequality). We thus set our prior on  $\theta^M$  as:  $p(\theta^M = \theta_{10}^M) = 0.0$ ,  $p(\theta^M = \theta_{00}^M) = 0.25$ ,  $p(\theta^M = \theta_{11}^M) = 0.25$ , and  $p(\theta^M = \theta_{01}^M) = 0.5$ . We relax this monotonicity assumption, to account for the Ansell and Samuels logic, in a second run of the analysis.

For our prior on democracy's responses to inequality, mobilization, and international pressure ( $\theta_{lower}^D$ ), we extract the following beliefs from the literature:

- **Monotonicity of direct  $I$  effect: no positive effect:** In none of the three theories does inequality promote democratization via a pathway *other than* via the poor's rising demand for it. In all three theories, inequality has a distinct negative effect on democratization via an increase in the elite's expected losses under democracy and thus its willingness to repress. In Ansell and Samuels, the distribution of resources also affects the probability of success of rebellion; thus higher inequality also reduces the prospects for democratization by strengthening the elite's hold on power. We thus set a zero prior probability on all types in which  $I$ 's direct effect on  $D$  is positive for any value of  $P$ .
- **Monotonicity of  $M$ 's effect: no negative effect:** In none of the three theories does mobilization reduce the prospects of democratization. We thus set a zero probability on all types in which  $M$ 's effect on  $D$  is negative at any value of  $I$  or  $P$ .
- **Monotonicity of  $P$ 's effect: no negative effect:** While international pressures are only discussed in Haggard and Kaufman's study, none of the studies considers the possibility that international pressures to democratize might prevent democratization that would otherwise have occurred. We thus set a zero probability on all types in which  $P$ 's effect is negative at any value of  $I$  or  $M$ .

In all, this reduces the number of nodal types for  $D$  from 256 to just 20.

For all remaining, allowable types, we set flat priors.

In remaining 20 allowable types can involve a rich range of interactions between international pressure, inequality, and mobilization, including::

1. Types for which  $P$  has no moderating effect
2. Types for which  $P = 1$  creates an “opportunity” for  $X$  to have an effect that it does not have at  $P = 0$ ; at  $P = 1$  and  $X = 0$ ,  $D$  takes on the value it does when  $X = 0$  and  $X$  has an effect, but does not take on this value when  $P = 0$  and  $X = 0$
3. Types for which  $P = 1$  is a causal “complement” to  $X$ , allowing  $X$  to have an effect it did not have at  $P = 0$ ; at  $P = 1$  and  $X = 1$ ,  $D$  takes on the value it does when  $X = 1$  and  $X$  has an effect, but does not take on this value when  $P = 0$  and  $X = 1$
4. Types for which  $P = 1$  “substitutes” for  $X$ , generating the outcome that  $X = 1$  was necessary to generate at  $P = 0$ ; at  $P = 1$  and  $X = 0$ ,  $D$  takes on the value it does when  $X = 1$  and  $X$  has an effect, but does not take on this value when  $P = 0$  and  $X = 0$
5. Types for which  $P$  “eliminates”  $X$ ’s effect, preventing  $X = 1$  from generating the outcome it generates when  $P = 0$ ; at  $P = 1$  and  $X = 1$ ,  $D$  does not take on the value it does when  $X = 1$  and  $X$  has an effect, but does take on this value when  $P = 0$  and  $X = 1$

Since  $P$  conditions the effect of  $I$ , we must also establish a prior on the distribution of  $P$ . In this analysis, we set the prior probability of  $P = 1$  to 0.5, implying that before seeing the data we think that international pressures to democratize are present half the time.

## 8.3 Results

We can now choose nodes in addition to  $I$  and  $D$  to observe from the lower-level model. Recall that our query is about the joint values of  $\theta^M$  and  $\theta^{D_{lower}}$ . By the logic  $d$ –separation, we can immediately see that both  $M$  and  $P$  may be informative about these nodes when  $D$  has already been observed. Conditional on  $D$ , both  $M$  and  $P$  are  $d$ –connected to both  $\theta^M$  and  $\theta^{D_{lower}}$ . Let us see what we learn, then, if we search for either mobilization of the lower classes or international pressure or both, and find either clue either present or absent.

We consider four distinct situations, corresponding to four possible combinations of inequality and democratization values that we might be starting with. In each situation, the nature of the query changes. Where we start with a case with low inequality and no democratization, asking if inequality caused the outcome is to ask if the lack of inequality caused the lack of democratization. Where we have high inequality and no democratization, we want to know if democratization was prevented by high inequality (as high inequality does in Boix’s account). For cases in which democratization occurred, we want to know whether the lack or presence of inequality (whichever was the case) generated the democratization.

Inference is done by applying Bayes rule to the observed data given the priors. Different “causal types” are consistent or inconsistent with possible data observations. Conversely the observation of data lets us shift weight towards causal types that are consistent with the data and away from those that are not. As a simple illustration if we observe  $D = 1$  then we would shift weight from types for which  $D$  is always 0, given the observed data, to types for which  $D$  can be 1 given the observed data.

### 8.3.1 Inferences for cases with observed democratization

We first turn to cases in which democratization has occurred—the category of cases that Haggard and Kaufman examine.

For these cases we use data from Haggard and Kaufman (2012) to show the inferences we would draw using this procedure and the actual observations made for a set of 8 cases.

Haggard and Kaufman consider only cases that democratized, so all cases in this table have the value  $D = 1$ . We show here how confident we would be that the level inequality caused democratization if (a) we observed only the cause and effect ( $I$  and  $D$ ); (b) we additionally observed either the level of mobilization by disadvantaged classes or the level of international pressure; and (c) if we observed both, in addition to  $I$  and  $D$ . Note that countries labels are marked in the “full data” cells in the lower right quadrant, but their corresponding partial data cells can be read by moving to the left column or the top row (or to the top left cell for the case with no clue data).

In coding countries' level of inequality, we rely on Haggard and Kaufman's codings using the Gini coefficient from the Texas Inequality dataset. In selecting cases of democratization, we use the codings in Cheibub et al. (2010), one of two measures used by Haggard and Kaufman. Our codings of the  $M$  and  $P$  clues come from close readings of the country-specific transition accounts in Haggard et al. (2012), the publicly shared qualitative dataset associated with Haggard and Kaufman (2012). We code  $M$  as 1 where the transition account refers to anti-government or anti-regime political mobilization by economically disadvantaged groups, and as 0 otherwise. For  $P$ , we code  $P = 0$  if international pressures to democratize are not mentioned in the transition account. The main estimates refer to analyses with only qualitative, monotonicity restrictions on our priors. We also show in square brackets the estimates if we allow for a negative effect of inequality on mobilization but believe it to be relatively unlikely.

### 8.3.1.1 $I = 0, D = 1$ : Low inequality democracies

In a case that had low inequality and democratized, did low inequality cause democratization, as Boix's thesis would suggest? Looking at the first set of cases in Table 8.1, did Mexico, Albania, Taiwan, and Nicaragua democratize because they had relatively low inequality? Based only on observing the level of inequality and the outcome of democratization, we would place a 0 probability on inequality having been a cause. What can we learn, then, from our two clues?

We are looking here for a negative effect of  $I$  on  $D$ , which in our model can only run via a direct effect, not through mobilization. Thus, the learning from  $M$  is limited for the same reason as in an  $I = 1, D = 0$  case. And  $M$  is modestly informative as a moderator for the same reasons and in the same direction, with observing mobilization generally reducing our confidence in inequality's negative effect relative to observing no mobilization. In our four cases, if we observe the level of mobilization, our confidence that inequality mattered goes up slightly (to 0) in Mexico and Taiwan, where mobilization did not occur, and goes down slightly in Albania and Nicaragua (to 0) where mobilization did occur.

Looking for the international pressure clue is, however, highly informative, though the effect runs in the opposite direction as in an  $I = 1, D = 0$  case.

Table 8.1: Four cases with low inequality and democratization. Question of interest: Was low inequality a cause of democracy? Table shows posterior beliefs for different data for four cases given information on  $M$  or  $P$ . Data from Haggard and Kaufman (2012). Analyses here use priors assuming only monotonic effects.

Case	M: Mobilization?	P: Pressure?	No clues	M only	P only	M and P
Mexico (2000)	0	0	0.438	0.475	0.615	0.667
Taiwan (1996)	0	1	0.438	0.475	0.34	0.393
Albania (1991)	1	0	0.438	0.394	0.615	0.571
Nicaragua (1984)	1	1	0.438	0.394	0.34	0.263

It is observing the absence of international pressure that makes us more confident in low inequality's effect. Since democratization *did* occur, the presence of international pressure makes it less likely for low inequality to have generated the outcome since international pressure could have generated democratization by itself. Once we bring this second clue into the analysis, Mexico and Taiwan sharply part ways: seeing no international pressure in Mexico, we are now much more confident that inequality mattered for the Mexican transition (1); seeing international pressure in Taiwan, we are now substantially less confident that inequality mattered to the Taiwanese transition (0). Similarly, observing  $P$  sharply differentiates the Albanian and Nicaraguan cases: seeing no international pressure in the Albanian transition considerably boosts our confidence in inequality's causal role there (0), while observing international pressure in the Nicaraguan transition strongly undermines our belief in an inequality effect there (1).

### 8.3.1.2 $I = 1, D = 1$ : High inequality democracies

Where we see both high inequality and democratization, the question is whether high inequality caused democratization via a positive effect. Considering the second set of cases in Table 8.2, did high inequality cause Mongolia, Sierra Leone, Paraguay, and Malawi to democratize?

Observing only the level of inequality and the democratization outcome, we would have fairly low confidence that inequality mattered, with a posterior on that effect of 1. Let us see what we can learn if we also observe the level

Table 8.2: Four cases with high inequality and democratization. Question of interest: Was high inequality a cause of democratization? Table shows posterior beliefs for different data for 4 cases given information on  $M$  or  $P$ . Data from Haggard and Kaufman (2012). Analyses here use priors assuming only monotonic effects.

Case	M: Mobilization?	P: Pressure?	No clues	M only	P only	M and P
Mongolia (1990)	0	0	0.128	0	0.231	0
Paraguay (1989)	0	1	0.128	0	0.088	0
Sierra Leone (1996)	1	0	0.128	0.15	0.231	0.25
Malawi (1994)	1	1	0.128	0.15	0.088	0.107

of mobilization and international pressure.

As in an  $I = 0, D = 0$  case,  $M$  can now be highly informative since this positive effect has to run through mobilization. Here it is the observation of a lack of mobilization that is most telling: high inequality cannot have caused democratization, given our model, if inequality did not cause mobilization to occur. There is no point in looking for international pressure since doing so will have no effect on our beliefs. Thus, when we observe no mobilization by the lower classes in Mongolia and Paraguay, we can be certain (given our model) that high inequality did *not* cause democratization in these cases. Moreover, this result does not change if we also go and look for international pressure: neither seeing pressure nor seeing its absence shifts our posterior away from 1.

If we do see mobilization, on the other hand—as in Sierra Leone and Malawi—we are slightly more confident that high inequality was the cause of democratization (1). Moreover, if we first see  $M = 1$ , then observing international pressure can add much more information; and it substantially differentiates our conclusions about the causes of Sierra Leone’s and Malawi’s transitions. Just as in an  $I=0, D=1$  case, it is the absence of international pressure that leaves the most “space” for inequality to have generated the democratization outcome. When we see the absence of pressure in Sierra Leone, our confidence that high inequality was a cause of the transition increases to 0; seeing pressure present in Malawi reduces our confidence in inequality’s effect to 1.

Table 8.3: No inequality and No democratization: Was no inequality a cause of no democratization? Analyses here use priors assuming only monotonic effects.

	P	M	posterior
I0P0M0D0	0	0	0.107
I0P1M0D0	1	0	0.250
I0P0M1D0	0	1	0.000
I0P1M1D0	1	1	0.000
I0M0D0		0	0.150
I0M1D0		1	0.000
I0P0D0	0		0.088
I0P1D0	1		0.231
I0D0			0.128

### 8.3.2 Cases with incomplete data

We next first causal relations for cases that did not democratize. These cases are not included in Haggard and Kaufman (2012) but our model nevertheless characterizes our beliefs for these cases also.

The results for cases that did not democratize (at the time in question) are presented in Table 8.3 and Table 8.4. Each table shows, for one kind of case, our posterior beliefs on the causal responsibility of  $I$  for the outcome for different search strategies.

#### 8.3.2.1 $I = 0, D = 0$ : Non democracy with low inequality

To begin with  $I = 0, D = 0$  cases, did the lack of inequality cause the lack of democratization (as, for instance, at the lefthand end of the Acemoglu and Robinson inverted  $U$ -curve)?

We start out, based on the  $I$  and  $D$  values and our model, believing that there is a 0.107 chance that low inequality prevented democratization. We then see that our beliefs shift most dramatically if we go looking for mobilization and find that it was present. The reason is that any positive effect of  $I$  on  $D$  has to run through the pathway mediated by  $M$  because we have excluded a positive direct effect of  $I$  on  $D$  in our priors. Moreover, since we do not allow

$I$  to have a negative effect on  $M$ , observing  $M = 1$  when  $I = 0$  must mean that  $I$  has no effect on  $M$  on this case, and thus  $I$  cannot have a positive effect on  $D$  (regardless also of what we find if we look for  $P$ ). If we do *not* observe mobilization when we look for it, we now think it is somewhat more likely that  $I = 0$  caused  $D = 0$  since it is still possible that high inequality *could* cause mobilization.

We also see that observing whether there is international pressure has a substantial effect on our beliefs. When we observe  $M = 1$  (or don't look for  $M$  at all), the presence of international pressure increases the likelihood that low inequality prevented democratization. Intuitively, this is because international pressure, on average across types, has a positive effect on democratization; so pressure's presence creates a greater opportunity for low inequality to counteract international pressure's effect and prevent democratization from occurring that otherwise would have (if there had been high inequality and the resulting mobilization).

### 8.3.2.2 $I = 1, D = 0$ : Non democracy with high inequality

In cases with high inequality and no democratization, the question is whether high inequality prevented democratization via a negative effect, as theorized by Boix. That negative effect has to have operated via inequality's direct effect on democratization since our monotonicity restrictions allow only positive effects via mobilization. Here, the consequence of observing  $P$  is similar to what we see in the  $I = 0, D = 0$  case: seeing international pressure greatly increases our confidence that high inequality prevented democratization, while seeing no international pressure moderately reduces that confidence. There is, returning to the same intuition, more opportunity for high inequality to exert a negative effect on democratization when international pressures are present, pushing toward democratization.

Here, however, looking for  $M$  has more modest effect than it does in an  $I = 0, D = 0$  case. This is because we learn less about the indirect pathway from  $I$  to  $D$  by observing  $M$ : as we have said, we already know from seeing high inequality and no democratization (and under our monotonicity assumptions) that any effect could not have run through the presence or absence of mobilization.

However,  $M$  provides some information because it, like  $P$ , acts as *moderator*

Table 8.4: Inequality and No democratization: Was inequality a cause of no democratization? Analyses here use priors assuming only monotonic effects.

	P	M	posterior
I1P0M0D0	0	0	0.263
I1P1M0D0	1	0	0.571
I1P0M1D0	0	1	0.393
I1P1M1D0	1	1	0.667
I1M0D0		0	0.394
I1M1D0		1	0.475
I1P0D0	0		0.340
I1P1D0	1		0.615
I1D0			0.438

for  $I$ 's direct effect on  $D$  (since  $M$  is also pointing into  $D$ ). As we know, learning about moderators tells us something about (a) the rules governing a case's response to its context (i.e., its response type) and (b) the context it is in. Thus, in the first instance, observing  $M$  together with  $I$  and  $D$  helps us eliminate types inconsistent with these three data points. For instance, if we see  $M = 0$ , then we eliminate any type in which  $D$  is 0, regardless of  $P$ 's value, when  $M = 0$  and  $I = 1$ . Second, we learn from observing  $M$  about the value of  $M$  under which  $D$  will be responding to  $I$ . Now, because  $M$  is itself potentially affected by  $I$ , the learning here is somewhat complicated. What we learn most directly from observing  $M$  is *the effect of I on M* in this case. If we observe  $M = 1$ , then we know that  $I$  has no effect on  $M$  in this case; whereas if we observe  $M = 0$ ,  $I$  might or might not have a positive effect on  $M$ . Learning about this  $I \rightarrow M$  effect then allows us to form a belief about how likely  $M$  would be to be 0 or 1 if  $I$  changed from 0 to 1; that is, it allows us to learn about the context under which  $D$  would be responding to this change in  $I$  (would mobilization be occurring or not)? This belief, in turn, allows us to form a belief about how  $D$  will respond to  $I$  given our posterior beliefs across the possible types that the case is.

The net effect, assuming that we have not observed  $P$ , is a small upward effect in our confidence that inequality mattered if we see no mobilization, and a small downward effect if we see mobilization. Interestingly, if we *do* observe  $P$ , the effect of observing  $M$  reverses: observing mobilization increases our

confidence in inequality's effect, while observing no mobilization reduces it.

## 8.4 Theory dependence

Haggard and Kaufman set out to use causal process observations to test inequality-based theories of democratization against the experiences of “Third Wave” democratizations. Their principal test is to examine whether they see evidence of distributive conflict in the process of democratization, defined largely as the presence or absence of mobilization prior to the transition. They secondarily look for other possible causes, specifically international pressure and splits in the elite.

In interpreting the evidence, Haggard and Kaufman generally treat the absence of mobilization as evidence against inequality-based theories of democratization as a whole (p. 7). They also see the *presence* of distributive mobilization in cases with high inequality and democratization as evidence against the causal role of inequality (p. 7). These inferences, however, seem only loosely connected to the logic of the causal theories under examination. Haggard and Kaufman express concern that inequality-oriented arguments point to “cross-cutting effects” (p. 1) of inequality, but do not systematically work through the implications of these multiple pathways for empirical strategy. Our analysis suggests that a systematic engagement with the underlying models can shift that interpretation considerably. Under the model we have formulated, where inequality is *high*, the absence of mobilization in a country that democratized is indeed damning to the notion that inequality mattered. However, where inequality is *low*—precisely the situation in which Boix’s theory predicts that we will see democratization—things are more complicated. If we assume that inequality cannot prevent mobilization, then observing no mobilization does not work against the claim that inequality mattered for the transition; indeed, it slightly supports it, at least given what we think is a plausible model-representation of arguments in the literature. Observing the absence of inequality in such a case, however, can undercut an inequality-based explanation if (and only if) we believe it is possible that inequality might prevent mobilization that would otherwise have occurred. Further, in cases with high inequality and democratization, it is the *absence* of mobilization by the lower classes that would least consistent with the claim that inequality mattered. Observing mobilization, in contrast,

pushes in favor of an inequality-based explanation.

Moreover, it is striking that Haggard and Kaufman lean principally on a mediator clue, turning to evidence of international pressure and elite splits (moderators, or alternative causes) largely as secondary clues to identify “ambiguous” cases. As we have shown, under a plausible model given prior theory, it is the moderator clue that is likely to be much more informative.

Of course, the model that we have written down is only one possible interpretation of existing theoretical knowledge. It is very possible that Haggard and Kaufman and other scholars in this domain hold beliefs that diverge from those encoded in our working model. The larger point, however, is that our process tracing inferences will inevitably *depend*—and could depend greatly—on our background knowledge of the domain under examination. Moreover, formalizing that knowledge as causal model can help ensure that we are taking that prior knowledge systematically into account—that the inferences we draw from new data are consistent with the knowledge that we bring to the table.

The analysis also has insights regarding case selection. Haggard and Kaufman justify their choice of only  $D = 1$  cases as a strategy “designed to test a particular theory and thus rests on identification of the causal mechanism leading to regime change” (p. 4). Ultimately, however, the authors seem centrally concerned with assessing whether inequality, as opposed to something else, played a key causal role in generating the outcome. As the results above demonstrate, however, there is nothing special about the  $D = 1$  cases in generating leverage on this question. The tables for  $D = 0$  show that, given the model, the same clues can shift beliefs about as much for  $D = 0$  as for  $D = 1$  cases. We leave a more detailed discussion of this kind of issue in model-based case-selection for Chapter ??.

Finally we emphasize that all of the inference in this chapter depends on a model that is constrained by theoretical insights but not one that is trained by data. Although we are able to make many inferences using this model, given the characteristics of a case of interest, we have no empirical grounds to justify these inferences. In Chapter 10 we show how this model can be trained with broader data from multiple cases and in Chapter ?? we illustrate how the model itself can be put into question.



# Chapter 9

## Integrated inferences

In this chapter we generalize the model developed in Chapter 7 to research situations in which we have data on multiple cases.

We start with a conceptual point: the structure introduced in Chapter 6 for single-case analysis can be used *as is* for multi-case analysis. Thus, the conceptual work for mixed methods inference from models has been done already. Our goal for the rest of the chapter is thus more technical than conceptual—to show how to shift focus beyond sample level queries and to exploit assumptions regarding independence across cases to generate simpler models of causal processes that affect many units. As we do so, we provide microfoundations for the models in Chapter 8 (as with those in Humphreys and Jacobs (2015)) with the probative value of clues derivable from a causal structure and data rather than provided directly by researchers.

### 9.1 Sample inference

Conceptualized correctly, there is no deep difference between the logic of inference used in single-case and in multi-case studies. This is not because any single “case” can be disaggregated into many “cases,” thereby allowing for large  $n$  analysis on small problems (King et al., 1994). Rather, the opposite: fundamentally, model-based inference always involves comparing *a* pattern of data with the logic of the model. Studies with multiple cases can, in fact, be conceptualized as single-case studies: we always draw our inferences from

a single *collection* of clues, whether those clues have come from one or from many units.

In practice, when we move from a causal model with one observation to a causal model with multiple observations, we can use the structure we introduced in Chapter 7 but simply replace nodes that have a single value (i.e., scalars) with nodes containing multiple values (i.e., vectors) drawn from multiple cases. We then make inferences about causal relations between nodes from seeing the values of those nodes' (or other nodes') vectors.

To illustrate, consider the following situation. Suppose that our model includes a binary treatment  $X$  that is assigned to 1 with probability 0.5; an outcome,  $Y$ ; and a third “clue” variable,  $K$ , all observable. We posit an unobserved variable  $\theta^Y$ , representing  $Y$ 's nodal type, with  $\theta^Y$  taking on values in  $\{a, b, c, d\}$  with equal probability. (We interpret the types in  $\{a, b, c, d\}$  as defined in Section 2.1.) In addition to pointing into  $Y$ , moreover,  $\theta^Y$  affects  $K$ . In particular,  $K = 1$  whenever  $X$  has an effect on  $Y$ , while  $K = 1$  with a 50% probability otherwise. In other words, our clue  $K$  is informative about  $\theta^Y$ , a unit's nodal type for  $Y$ . As familiar from Chapters 7 and 8, when we observe  $K$  in a case we can update on causal effects within the case since that  $K$  value will have different likelihoods under different values of  $\theta^Y$ .

So far, we have described the problem at the unit level. Let's now consider a two-case setup. We do this by exchanging scalar nodes for vectors:

- We have a treatment node,  $X$ , that can take on one of four values,  $(0, 0), (0, 1), (1, 0), (1, 1)$  with equal probability.
- $\theta^Y$  is now a vector with two elements that can take on one of 16 values  $(a, a), (a, b), \dots (d, d)$  as determined by  $\lambda_\theta$ . We might imagine a uniform distribution over these 16 elements.
- $Y$  is a vector that is generated by  $\theta^Y$  and  $X$  in the obvious way (e.g.,  $X = (0, 0), \theta^Y = (a, b)$  generates outcomes  $Y = (1, 0)$ )
- The vector  $K$  has the same domain as  $X$  and  $Y$ , and element  $K[j] = 1$  if  $\theta^Y[j] = b$ .

Now, consider a causal estimand. In a single-case setup, we might ask whether  $X$  has an effect on  $Y$  in the case. For a multi-case setup, we might ask what the Sample Average Treatment Effect,  $\tau$ , is. Note a subtle difference in the nature of the answers we seek in these two situations. In the

first (single-case) instance, our estimand is binary—of the form: “is the case a  $b$  type?”—and our answer is a probability. In the multi-case estimation of the sample average treatment effect (“SATE”), our estimand is categorical and our answer is a probability distribution: we are asking “what is the probability that  $\tau$  is 0?,” “what is the probability that  $\tau$  is .5?”, and so on.

While the estimand shifts, we can use the tools introduced for single-case process tracing in Chapters 7 and 8 to analyze this (superficially) multi-case study. To begin, our prior on the probability that  $\tau = 1$  is the prior that  $X$  has a positive effect on  $Y$  in both cases, that is, that  $\theta^Y = (b, b)$ : just 1 in 16.

Now, suppose that we observe that, for both units,  $X = 1$  and  $Y = 1$ . This data pattern is consistent only with four possible  $\theta$  vectors:  $(b, b), (d, d), (b, d), (d, b)$ . Moreover each of these four is equally likely to produce the data pattern we see). So our belief that  $\tau = 1$  now shifts from 1 in 16 to 1 in 4. Next, suppose that we further observe the data pattern  $\mathbf{K} = (1, 1)$ . The probability of this pattern for  $\Theta$  vector  $(b, b)$  ( $\tau = 1$ ) is 1. And for the type vectors  $(d, d), (b, d), (d, b)$ , the probability of this  $\mathbf{K}$  pattern is .25, .5, and .5, respectively. Applying Bayes’ rule, our updated belief that  $\tau = 1$  is then  $1/(1 + .25 + .5 + .5) = 4/9$ .

We can similarly figure out the posterior probability on any possible value of  $\tau$  and so build up a full posterior distribution. And we can do so given any  $\mathbf{K}$  pattern (i.e.,  $\mathbf{K}$  realization) across the cases. Thus, if we observe the data pattern  $\mathbf{K} = (0, 1)$ , the probability of this pattern for type vector  $(b, b)$  ( $\tau = 1$ ) is 0. For the type vectors  $(d, d), (b, d), (d, b)$  it is .25, 0, .5, respectively. The table below represents the posterior distribution over a set of discrete treatment effect values given different  $K$  patterns observed.

$X$ pattern	$Y$ pattern	$K$ pattern	$\tau = -1$	$\tau = -.5$	$\tau = 0$	$\tau = .5$	$\tau = 1$
(1,1)	(1,1)	(1,1)	0	0	1/9	4/9	4/9
(1,1)	(1,1)	(1,0)	0	0	1/3	2/3	0
(1,1)	(1,1)	(0,0)	0	0	1	0	0

The conceptual point is that the general logic of inference with multiple units is the same as that with one unit. In both situations, we work out the likelihood of any given data *pattern* for each possible set of values of model parameters and update our beliefs about those parameters. And, from our

posterior distribution over model parameters (e.g.,  $\Theta^Y$ ), we then derive a posterior distribution over the possible answers to our query (e.g., values of  $\tau$ ).<sup>1</sup>

## 9.2 General queries

Although the core conceptual logic is the same for multi-case and single-case inference, going forward, we operationalize these problems somewhat differently.

For the remainder of this chapter, and for the rest of the book, when we focus on multi-case studies, we will set our sights primarily on models that describe general processes. Rather than seeking to understand the average effect in a set of cases, we seek to understand the causal relations that gave rise to the set of cases. From these we sometimes draw inferences to cases but in general our models will involve queries pitched in general terms.

There are two reasons for this. The first is that we are interested in learning across cases: To figure out how what we see in one case provides insight for what is happening in another. We do this by using data on some cases to update our beliefs about a general model that we think is of relevance for other cases. Thus we seek to learn about a general model. The second reason is more practical. If we can think of units as draws from a large population, and then invoke independence assumptions across types, then we can greatly reduce complexity by analyzing problems at the unit level rather than at the population level. In the 2-case example above, the vector  $\theta^Y$  could take on any of 16 values  $((a, a), (a, b), \dots (d, d))$ . At the case level, however, the node  $\theta^Y$  can take on only 4 values  $(\{a, b, c, d\})$ , yet we can learn about each case's  $\theta^Y$  value from data drawn from all the cases. Thinking about it this way simplifies the problem by greatly reducing the parameter space, but it is not free. It requires invoking the assumption that (potential) outcomes across units do not depend on each other. If we cannot stand by that assumption, then we will need to build independence failures into our models.

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<sup>1</sup>Representing node values in vector forms like this allows for vector-level mappings that imply more complex dependencies between units. For instance we might imagine instead that we observe  $K = 1$  if and only if  $\theta^Y = (b, b)$ , in which case observation of  $K$  lets us distinguish between  $\tau = 1$  and  $\tau = .5$  but not between  $\tau = .5$  and  $\tau = 0$ .

Taking this step, the procedure we now use in the mixed methods works as follows.

### 9.2.1 Set up

1. **A DAG.** As for process tracing, we begin with a graphical causal model specifying possible causal linkages between nodes. Our “chain” model for instance has DAG:  $X \rightarrow M \rightarrow Y$ .
2. **Nodal types.** Just as in process tracing, the DAG and variable ranges define the set of possible nodal types in the model—the possible ways in which each variable is assigned (if exogenous) or determined by its parents (if endogenous). For the  $X \rightarrow M \rightarrow Y$  model there are 2 types for  $\theta^X$ , 4 for  $\theta^M$ , and 4 for  $\theta^Y$ .
3. **Causal types.** A full set of nodal types gives rise to a full set of causal types, encompassing all possible combinations of nodal types across all nodes in the model. We let  $\theta$  denote an arbitrary causal type. For a  $X \rightarrow M \rightarrow Y$  model, one possible causal type would be  $\theta = (\theta_1^X, \theta_{01}^M, \theta_{01}^Y)$ .
4. **Parameters.** As before, we use  $\lambda^V$  to denote the probabilities of  $\theta^V$  for a given node,  $V$ . Recall that in process tracing, we sought to learn about  $\theta$  and our priors were given by  $\lambda$ . When we shift to multi-case inference,  $\lambda$  becomes the parameter that we want to learn about: we seek to learn about the probability of different types arising in a population (or the *shares* of types in a large population).
5. **Priors.** In the process tracing setup, we treat  $\lambda$  as given: we do not seek to learn about  $\lambda$ , and uncertainty over  $\lambda$  plays no role. When we get to observe data on multiple cases, however, we have the opportunity to learn *both* about the cases at hand *and* about the population. Moreover, our level of uncertainty about population-level parameters will shape our inferences. We thus want our parameters (the  $\lambda$ ’s) to be drawn from a prior *distribution* — a distribution that expresses our uncertainty and over which we can update once we see the data. While different distributions may be appropriate to the task in general, uncertainty over proportions (of cases, events, etc.) falling into a set of

discrete categories is usefully described by a Dirichlet distribution, as discussed in Chapter 5. Recall that the parameters of a Dirichlet distribution (the  $\alpha$ 's) can be thought of as conveying both the relative expected proportions in each category and our degree of uncertainty.

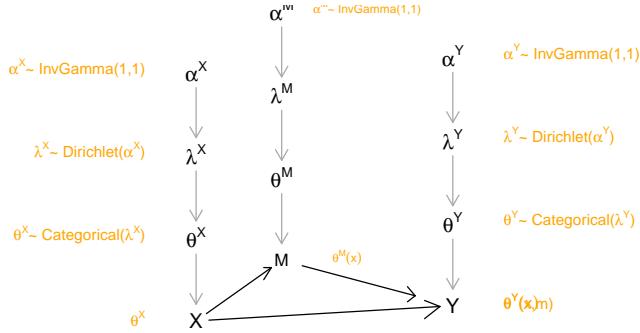


Figure 9.1: Types, parameters, and priors

### 9.2.2 Inference

Inference then works by figuring out the probability of the data given different possible parameter vectors,  $\lambda$ s, and then applying Bayes' rule. In practice we proceed as follows.

**Distributions over causal types.** We first need characterize our beliefs about causal types given any possible parameter vector  $\lambda$ . Imagine a draw of one possible value of  $\lambda$  from the prior. This  $\lambda$  vector implies a set of nodal type shares for all nodes. That set of nodal type shares implies, in turn, a distribution over *causal* types ( $\theta$ ). For instance, the probability of causal type  $\theta = (\theta_1^X, \theta_{01}^Y, \theta_{01}^M)$  is simply  $p(\theta|\lambda) = \lambda_1^X \lambda_{01}^M \lambda_{01}^Y$ . More generally:

$$p(\theta|\lambda) = \prod_{k,v:\theta_k^v \in \theta} \lambda_k^v$$

**Event probabilities.** Each causal type in turn implies a single data realization, or data type. For instance  $\theta = (\theta_1^X, \theta_{01}^M, \theta_{01}^Y)$  implies data  $X = 1, M = 1, Y = 1$ . Let  $D(\theta)$  denote the data type implied by causal type  $\theta$ . A single data type, however, may be implied by multiple causal types. We use  $\Theta(d)$  to denote the set of causal types that imply a given data type:

$$\Theta(d) : \{\theta | D(\theta) = d\}$$

The probability of a given data type  $d$ , is then:

$$w_d = \sum_{\theta \in \Theta(d)} p(\theta | \lambda)$$

And we use  $\mathbf{w}$  to denote the vector of event probabilities over all data types.

To illustrate, a data type  $d = (X = 1, M = 1, Y = 1)$  is consistent with four different causal types in the  $X \rightarrow M \rightarrow Y$  model:  $\Theta(d) = \{(\theta_0^X, \theta_{01}^M, \theta_{01}^Y), (\theta_0^X, \theta_{11}^M, \theta_{01}^Y), (\theta_0^X, \theta_{01}^M, \theta_{11}^Y), (\theta_0^X, \theta_{11}^M, \theta_{11}^Y)\}$ . The probability of the data type is then calculated by summing up the probabilities of each causal type that implies the event:  $w_{111} := \lambda_1^X (\lambda_{01}^M + \lambda_{11}^M) (\lambda_{01}^Y + \lambda_{11}^Y)$ .

In practice, calculating the full  $\mathbf{w}$  vector is made easier by the construction of a “parameter matrix” and an “ambiguity matrix”, just as for process tracing, that tells us which causal types are consistent with a particular data type.

We use Tables 9.2 and 9.3 to illustrate how to calculate the event probability for each data type for a given parameter vector  $\lambda$ . Starting with data type  $X = 0, Y = 0$  (first column of the ambiguity matrix), we see that the consistent causal types are  $(\theta_0^X, \theta_{00}^Y)$  and  $(\theta_0^X, \theta_{01}^Y)$ , in rows 1 and 4. We then turn to columns 1 and 4 of the parameter matrix to read off the probability of each of these causal types—in each case given by the probability of the nodal types that it is formed out of. This gives  $.4 \times .3$  and  $.4 \times .2$  giving a total probability of 0.2 for the  $X = 0, Y = 0$  event. All four event probabilities, for the four data types, are then calculated in the same way.

In practice we do this all using matrix operations.

**Likelihood.** Now that we know the probability of observing each data pattern in a *single* case given  $\lambda$ , we can use these event probabilities to aggregate up to the likelihood of observing a data pattern across multiple cases (given

Table 9.2: An ambiguity matrix for a simple  $X \rightarrow Y$  model (with no unobserved confounding). Rows are causal types, columns are data types.

	X0Y0	X1Y0	X0Y1	X1Y1
X0Y00	1	0	0	0
X1Y00	0	1	0	0
X0Y10	0	0	1	0
X1Y10	0	1	0	0
X0Y01	1	0	0	0
X1Y01	0	0	0	1
X0Y11	0	0	1	0
X1Y11	0	0	0	1

Table 9.3: A parameter matrix for a simple  $X \rightarrow Y$  model (with no unobserved confounding), indicating a single draw of  $\lambda$  values from the prior distribution.

	X0.Y00	X1.Y00	X0.Y10	X1.Y10	X0.Y01	X1.Y01	X0.Y11	X1.Y11	\$\lambda\$
X.0	1	0	1	0	1	0	1	0	
X.1	0	1	0	1	0	1	0	1	
Y.00	1	1	0	0	0	0	0	0	
Y.10	0	0	1	1	0	0	0	0	
Y.01	0	0	0	0	1	1	0	0	
Y.11	0	0	0	0	0	0	1	1	

$\lambda$ ). For this aggregation, we make use of an independence assumption: that each unit is independently drawn from a common distribution. Doing so lets us move from a categorical distribution that gives the probability that a single case has a particular data type to a *multinomial* distribution that gives the probability of seeing an arbitrary data pattern across any number of cases.

Specifically, with discrete variables, we can think of a given multiple-case data pattern simply as a set of counts across categories. For, say,  $X, Y$  data, we will observe a certain number of  $X = 0, Y = 0$  cases (which we notate as  $n_{00}$ ), a certain number of  $X = 1, Y = 0$  cases ( $n_{10}$ ), a certain number of  $X = 0, Y = 1$  cases ( $n_{01}$ ), and a certain number of  $X = 1, Y = 1$  cases ( $n_{11}$ ). A data pattern, given a particular set of variables observed (a search strategy), thus has a multinomial distribution. The likelihood of a data pattern under a given search strategy, in turn, takes the form of a multinomial distribution conditional on the number of cases observed,  $n$ , and the probability of each data type, given a  $\lambda$  draw. More formally, we write:

$$d \sim \text{Multinomial}(n, w(\lambda))$$

To illustrate, assume now that we have a 3-node model, with  $X, Y$ , and  $M$  all binary. Let  $\mathbf{n}_{XYM}$  denote an 8-element vector recording the number of cases in a sample displaying each possible combination of  $X, Y, M$  data, thus:  $\mathbf{D} = \mathbf{n}_{XYM} := (n_{000}, n_{001}, n_{100}, \dots, n_{111})$ . The elements of  $\mathbf{n}_{XYM}$  sum to  $n$ , the total number of cases studied. Likewise, let the event probabilities for data types given  $\lambda$  be registered in a vector,  $\mathbf{w}_{XYM} = (w_{000}, w_{001}, w_{100}, \dots, w_{111})$ . The likelihood of a data pattern,  $\mathbf{D}$  is then:

$$p(d|\lambda) = \text{Multinom}\left(n_{XYM} | \sum n_{XYM}, w_{XYM}(\lambda)\right)$$

In other words, the likelihood of observing a particular data pattern given  $\lambda$  is given by the corresponding value of the multinomial distribution given the data probabilities.

4. **Estimation.** We now have all the components for updating on  $\lambda$ . Applying Bayes rule (see Chapter 5), we have:

Table 9.4: An illustration of a posterior distribution for a  $X \rightarrow M \rightarrow Y$  model. Each row is a draw from  $p(\lambda|d)$ . Such a posterior would typically have thousands of rows and capture the full joint posterior distribution over all parameters.

X.0	X.1	M.00	M.10	M.01	M.11	Y.00	Y.10	Y.01	Y.11
0.47	0.53	0.21	0.07	0.17	0.55	0.20	0.23	0.15	0.41
0.68	0.32	0.02	0.41	0.38	0.19	0.12	0.20	0.07	0.61
0.33	0.67	0.16	0.45	0.27	0.12	0.08	0.02	0.81	0.09
0.68	0.32	0.15	0.10	0.70	0.05	0.03	0.07	0.00	0.90
0.17	0.83	0.02	0.11	0.64	0.22	0.44	0.06	0.30	0.20
0.83	0.17	0.16	0.08	0.02	0.73	0.49	0.28	0.12	0.11

$$p(\lambda|d) = \frac{p(d|\lambda)p(\lambda)}{\int_{\lambda'} p(d|\lambda')p(\lambda')}$$

In the `CausalQueries` package this updating is implemented in `stan`, and the result of the updating is a data frame that contains a collection of draws from the posterior distribution for  $\lambda$ . Table 9.4 illustrates what such a data frame might look like for an  $X \rightarrow M \rightarrow Y$  model. Each row represents a single draw from  $p(\lambda|d)$ . The 10 columns represent shares for each of the 10 nodal types in the model, under each  $\lambda$  draw.

## 5. Querying.

Once we have generated a posterior distribution for  $\lambda$ , we can then query that distribution. The simplest queries relate to values of  $\lambda$ . For instance, if we are interested in the probability that  $M$  has a positive effect on  $Y$ , given an updated  $X \rightarrow M \rightarrow Y$  model, we want to know about the distribution of  $\lambda_{01}^M$ . This distribution can be read directly from column 9 ( $Y.01$ ) of Table 9.4. More complex queries can all be described as summaries of combinations of these columns. For instance, the query, “What is the average effect of  $M$  on  $Y$ ” is a question about the distribution of  $\lambda_{01}^M - \lambda_{10}^M$ , which is given by the difference between columns 9 and 8 of the table. Still more complex queries may require keeping some nodes constant while varying others, yet all of these can be calculated as summaries of the combinations of columns of the posterior distribution, following the rules described in Chapter 4.

Table 9.5: Inferences on a chain model given different amounts of data (all on the diagonal, with  $X=0, Y=0$  or  $X=1, Y=1$ ). Columns 1-4 are shares of a, b, c, d causal types (as described in Chapter 2); columns 5 - 8 show  $\tau_{ij}$ —average effects of  $i$  on  $j$ ; the last column shows the probability of causation: the probability that  $X$  caused  $Y$  in a  $X = Y = 1$  case.

Data	a	b	c	d	$\tau_{XM}$	$\tau_{MY}$	$\tau_{XY}$
No data	0.13	0.13	0.37	0.38	0.00	-0.01	0.00
2 cases X, Y data only	0.12	0.14	0.37	0.37	0.00	0.00	0.02
2 cases, X, M, Y data	0.12	0.16	0.36	0.36	0.20	0.20	0.04
10 cases: X, Y data only	0.11	0.27	0.31	0.31	0.00	0.00	0.17
10 cases: X, M, Y data	0.09	0.44	0.23	0.23	0.59	0.59	0.35

Table 9.5 shows examples of a full mapping from data to posteriors. We begin with a simple chain model of the form  $X \rightarrow M \rightarrow Y$  with flat priors over nodal types and report inferences on a set of queries (columns) for difference data types (rows).

### 9.2.3 Wrinkles

The procedure we described works in the same way for a very wide class of causal models. More attention is needed for special cases in which there is confounding, complex sampling, or sample estimands.

#### 9.2.3.1 Unobserved confounding.

When there is unobserved confounding, we need parameter sets that allow for a joint distribution over nodal types. Unobserved confounding, put simply, means that there is confounding across nodes that is not captured by nodes and edges represented on the DAG. More formally, in the absence of unobserved confounding, we can treat the distribution of nodal types for a given node as independent of the distribution of nodal types for every other node. Unobserved confounding means that we believe that nodal types may be correlated across nodes. Thus, for instance, we might believe that those units assigned to  $M = 1$  have different potential outcomes for  $Y$  than those assigned to  $M = 0$  – i.e., that the probability of  $M = 1$  is correlated with

whether or not  $M$  has an effect on  $Y$ . To allow for such a correlation, we have to allow  $\theta^M$  and  $\theta^Y$  to have a joint distribution. There are different ways to do this in practice, but a simple approach is to split the parameter set corresponding to the  $Y$  node into two: we specify one distribution for  $\theta^Y$  when  $M = 0$  and a separate distribution for  $\theta^Y$  when  $M = 1$ . For each of these parameter sets, we specify two  $\alpha$  parameters representing our priors. We can draw  $\lambda$  values for these conditional nodal types from the resulting Dirichlet distributions, as above, and can then calculate causal type probabilities in the usual way. Note that if we do this in an  $X \rightarrow M \rightarrow Y$  model, we have one 2-dimensional Dirichlet distribution corresponding to  $X$ , one 4-dimensional Dirichlet distribution corresponding to  $M$ , and two 4 dimensional distributions corresponding to  $Y$ . In all, with  $1+3+3+3$  degrees of freedom: exactly the number needed to represent a joint distribution over all  $\theta^X, \theta^M, \theta^Y$  combinations.

In the figure below we represent this confounding by indicating parameters values  $\lambda_{MY}$  that determine the joint distribution over  $\theta_M$  and  $\theta_Y$ .

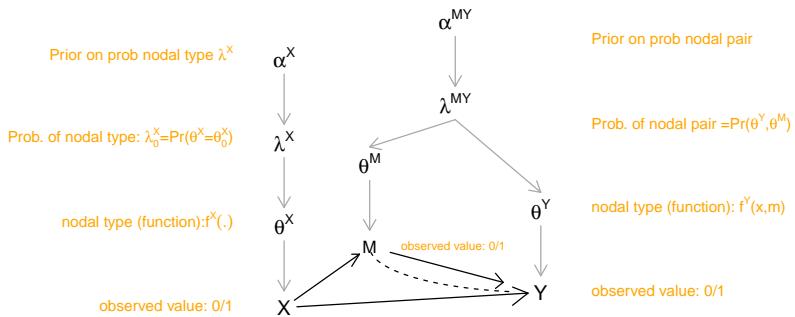


Figure 9.2: Types, parameters, and priors, with confounding

### 9.2.3.2 Sampling and the likelihood principle

In constructing a likelihood function, we sometimes need to take the sampling strategy into account. Sometimes however we can ignore the sampling procedure if we can invoke the “likelihood principle”—the principle that the relevant information for inference is contained in the likelihood.

To see the likelihood principle in operation, consider the following *conditional* data strategy: we collect data on  $X$  and  $Y$  in 2 cases, and we then measure  $M$  in any case in which we observe  $X = 1, Y = 1$ .

We draw data and end up with one case with  $X = Y = 0$  ( $M$  not observed) and one case with  $X = 1, M = 0, Y = 1$  ( $M$  measured, following the strategy).

One way to think of the event probabilities is to think of a set of 5 possible events, as described in table below:

data type:	prob:
$X1M0Y1$	$\lambda_1^X(\lambda_{00}^M + \lambda_{10}^M)(\lambda_{11}^Y + \lambda_{10}^Y)$
$X1M1Y1$	$\lambda_1^X(\lambda_{11}^M + \lambda_{01}^M)(\lambda_{11}^Y + \lambda_{01}^Y)$
$X0Y0$	$\lambda_0^X(\lambda_{00}^M + \lambda_{01}^M)(\lambda_{00}^Y + \lambda_{01}^Y) + \lambda_0^X(\lambda_{10}^M + \lambda_{11}^M)(\lambda_{00}^Y + \lambda_{10}^Y)$
$X0Y1$	$\lambda_0^X(\lambda_{00}^M + \lambda_{01}^M)(\lambda_{10}^Y + \lambda_{11}^Y) + \lambda_0^X(\lambda_{10}^M + \lambda_{11}^M)(\lambda_{01}^Y + \lambda_{11}^Y)$
$X1Y0$	$\lambda_1^X(\lambda_{00}^M + \lambda_{10}^M)(\lambda_{00}^Y + \lambda_{01}^Y) + \lambda_1^X(\lambda_{01}^M + \lambda_{11}^M)(\lambda_{00}^Y + \lambda_{10}^Y)$

In this conditional strategy view (draw  $X, Y$  first and then draw  $M$  based on what you find) we have

- $2P(X = 0, Y = 0)P(X = 1, Y = 1)P(M = 0|X = 1, Y = 1)$

The two observations could however also be thought of as coming from a simple multinomial draw from the five event types in the table above. Call this the single multinomial view.

In the single multinomial view we have the probability of seeing data with  $X = Y = 0$  in one case and  $X = 1, M = 0, Y = 1$  in another is:

- $2P(X = 0, Y = 0)P(X = 1, M = 0, Y = 1)$

But since  $P(X = 1, Y = 1)P(M = 0|X = 1, Y = 1) = P(M = 0|X = 1, Y = 1)$  these two expressions are the same “up to a constant” and so the inferences we make will be the same under both views.

Consider now a third strategy in which rather than conditioning  $X = Y = 1$  to examine  $M$ , one of the two cases were chosen at random to observe  $M$  and it just so happened to be a case with  $X = Y = 1$ . Or another strategy in which for each data point researchers randomly determined whether to gather data on  $M$  or not. In all of these cases the probability of observing the data we do in fact observe has the same basic form, albeit with possibly different constants.

In other words, these details of sampling can be ignored.

Other sampling procedures do have to be taken into account however, in particular, sampling—or more generally missingness—that is related to potential outcomes. As the simplest illustration consider a model in which  $X \rightarrow Y$ , but data is only recorded in cases in which  $Y = 1$ . Then a naive implementation of our procedure would infer that  $Y = 1$  regardless of  $X$  and so  $X$  has no effect on  $Y$ . The problem here is that the likelihood is not taking account of the process through which cases enter our data. In this case the correct likelihood would make use of event probabilities of the form:

$$\begin{aligned} x_d &= \sum_{\theta \in \Theta(d)} p(\theta|\lambda) \\ x_d &= \sum_{\theta \in \Theta(d)} p(\theta|\lambda) \end{aligned}$$

Let  $D^*$  denote the set of data types involving  $Y=1$ . Then:

$$w_d = \begin{cases} 0 & \text{if } d \notin D^* \\ \frac{x_d}{\sum_{d' \in D^*} x_{d'}} & \text{otherwise} \end{cases}$$

While this kind of sampling can be handled relatively easily (it is implemented also in the **CausalQueries** package) the general principle holds that sampling (missingness) that is related to potential outcomes is a part of the data generating process and needs to be taken into account in the likelihood. For strategies to address non random sampling by blocking, see Bareinboim and Pearl (2016).

### 9.2.3.3 Case inference following population updating

We are often in situations in which we observe patterns in  $n$  units and then seek to make an inference about one or more of the  $n$  cases conditional on *both* the case level data and the broader patterns in the full data.

Divide cases into set  $S^0, S^1$  where  $S^0$  is the set for which we wish to make case level inferences and  $S^1$  is the collection of other cases for which we have data.

In such cases should one use the data from  $S^0$  when updating on population estimands or rather update using  $S^1$  only and use information on  $S^1$  for the case level inferences only?

The surprising answer is that it is possible to do both, though exactly how queries are calculated depends on the method used.

Let  $\Lambda$  denote a collection of possible population parameters with typical element  $\lambda^i$ . Let  $p$  denote a distribution over  $\Lambda$  (after updating on data from set  $S^1$ ), with typical element  $\lambda^i$ . Let  $X$  denote possible data for cases in  $S^0$  with realization  $x$ .

Let  $d^i$  denote the probability of observing data  $X = x$  for a case (or set of cases) given  $\lambda^i$ .

Let  $\tau^{j|x}$  denote a query of interest—where the query is conditional in the sense that it relates to cases with data  $x$ . An example might be: what is the effect of  $X$  on  $Y$  in a case in which  $M = 1$  and  $Y = 1$ . Let  $q_j^i$  denote the probability that  $\tau^{j|x} = \tau_j^{j|x}$  when  $\lambda = \lambda^i$  for a case with data  $X = x$ . Note  $q_j^i$  can be written  $z_j^i/d^i$  where  $z_j^i = \Pr(\tau^{j|x} = \tau_j^{j|x}, X = x | \lambda^i)$ .

To illustrate say in an  $X \rightarrow Y$  model we were interested the effect of  $X$  on  $Y$  in a case with  $X = 1, Y = 1$ . Then  $d^i = (\lambda^i)_1^X((\lambda^i)_{01}^Y + (\lambda^i)_{11}^Y)$  is the probability of observing  $((X = 1, Y = 1))$ . Then for query  $\tau_j^{j|x} = 1$  (did  $X$  cause  $Y$ ) we have  $z_j^i = (\lambda^i)_1^X((\lambda^i)_{01}^Y)$ , and so the probability of this query for this case given  $\lambda^i$  is:  $q_j^i = \frac{(\lambda^i)_{01}^Y}{(\lambda^i)_{01}^Y + (\lambda^i)_{11}^Y}$

The posterior on  $\tau^{S^0}$  for the cases in  $S^0$  that provide data  $x$ , is then:

$$\Pr(\tau^{j|x} = \tau_j^{j|x}) = \frac{p^i z_j^i}{\sum_k p^k d^k}$$

This can be calculated from the prior  $p$  (that is the distribution on  $\Theta$  after updating on cases in  $S^1$  only).

Notice however that (a) the *posterior* distribution on  $\lambda^i$  given observation of  $x$  in the  $S^0$  set is  $\frac{p^i d^i}{\sum_k p^k d^k}$  and (b)  $p^i z_j^i = p^i d^i q^i$ . It follows that this quantity can also be interpreted as the posterior mean of  $q^i$ , after observing both  $S^0$  and  $S^1$ .

We therefore have two approaches to calculating these sample quantities: either take the posterior mean (posterior to  $S^0$  and  $S^1$ ), over the distribution of  $\lambda$  of the conditional probability of the estimand given the case data in  $S^0$ , or take the expected probability of  $\tau$  given the prior (after observing  $S^1$  only) and condition on the probability of the case level data in  $S^0$ .

### 9.3 Mixed methods

As can be seen already from our discussion of sampling, we do not need data on all nodes in order to implement the procedure. If we have data on only some of the nodes in a model, we follow the same basic logic as with partial process-tracing data. In calculating the probability of a pattern of partial data, we use all columns (data types) in the ambiguity matrix that are consistent with the partial data.

So, for instance, if we have an  $X \rightarrow Y$  model but observe only  $Y = 1$ , then we would retain both the  $X = 0, Y = 1$  column and the  $X = 1, Y = 1$  column. We then calculate the probability of this data type by summing causal-type probabilities for all causal types that can produce *either*  $X = 0, Y = 1$  *or*  $X = 1, Y = 1$ .

What if our data have been collected via a mixture of search strategies? Suppose, for instance, that we have collected  $X, Y$  data for a set of cases, and have additionally collected data on  $M$  for a random subset of these. We can think of this mixed strategy as akin to conducting quantitative analysis on a large sample while conducting in-depth process tracing on part of the large- $N$  sample. We can then summarize our data in two vectors, an 8-element  $n_{XYM}$  vector  $((n_{000}, n_{001}, \dots, n_{111})$  for the cases with process-tracing ( $M$ ) observations, and a 4-element vector  $n_{XY*} = (n_{00*}, n_{10*}, n_{01*}, n_{11*})$  for the partial data on those cases on which we did not conduct process tracing.

Likewise, we now have two sets of data probabilities: an 8-element vector for the set of cases with complete data,  $w_{XYM}$ , and a 4-element vector for those with partial data,  $w_{XY*}$ .

Let  $n$  denote the total number of cases examined, and  $k$  the number for which we have data on  $M$ . Assuming that each observed case represents an independent, random draw from the population, we can form the likelihood function as a *product* of multinomial distributions, one representing the complete-data (process-traced) cases and one representing those with only  $X, Y$  data:

$$\Pr(\mathcal{D}|\theta) = \text{Multinom}(n_{XY*}|n - k, w_{XY*}) \times \text{Multinom}(n_{XYM}|k, w_{XYM})$$

The generalization is straightforward. Say that a strategy is a set of nodes on which data is gathered on  $n_s$  units. For example data may be gathered through three strategies:  $n_1$  units for which data is gathered on nodes  $V_1$  only,  $n_2$  units for which data is gathered on nodes  $V_2$  only, and  $n_3$  units for which data is gathered on nodes  $V_3$  only. The observed number of units for each data type under each data strategy is  $m_s$  and the event probabilities are  $w_s$ . The likelihood is:

$$L = \prod_s \text{multinomial}(m_s|n_s, w_s)$$

## 9.4 Considerations

In this last section we consider six implications and extensions of this approach.

### 9.4.1 Probative value can be derived from a causal structure plus data

In Chapter 7, we discussed the fact that a DAG by itself is insufficient to generate learning about causal effects from data on a single case; we also need informative prior beliefs about population-level shares of nodal types.

When working with multiple cases, however, we *can* learn about causal relations when starting with nothing more than the DAG and data. In particular, we can simultaneously learn about case-level queries and justify our inferences from population-level data patterns.

For instance, in an  $X \rightarrow M \rightarrow Y$  model, even if we start with flat priors over  $M$ 's nodal types, observing a correlation (or no correlation) between  $X$  and  $M$  across multiple cases provides information about  $X$ 's effect on  $M$ . Simply, a stronger, positive (negative)  $X, M$  correlation implies a stronger positive (negative) effect of  $X$  on  $M$ . In turn, a stronger  $X, M$  correlation implies a stronger effect of  $X$  on  $Y$  since, under this model, that effect has to run through an effect of  $X$  on  $M$ .

What's more, data from multiple cases can *provide* probative value for within-case inference. Suppose, for the  $X \rightarrow M \rightarrow Y$  model, that we start with flat priors over all nodal types. As discussed in Chapter 7, observing  $M$  in a single case cannot be informative about  $X$ 's effect on  $Y$  in that case. If we have no idea of the direction of the intermediate causal effects, then we have no idea which value of  $M$  is more consistent with an  $X \rightarrow M$  effect or with an  $M \rightarrow Y$  effect. But suppose that we first observe data on  $X$  and  $M$  for a group of cases and find a strong positive correlation between the two variables. We now update to a belief that any effect of  $X$  on  $M$  is more likely to be positive than negative. Now, let's say we look at one of our other cases in which  $X = 1$  and  $Y = 1$  and want to know if  $X = 1$  caused  $Y = 1$ . Knowing now that any such effect would most likely have operated via a positive  $X \rightarrow M$  effect means that observing  $M$  will be informative: seeing  $M = 1$  in this case will be more consistent with an  $X \rightarrow Y$  effect than will  $M = 0$ . The same logic, of course, also holds for observing cross-case correlations between  $M$  and  $Y$ .

Our ability to draw probative value from cross-case data will depend on the causal model we start with. For instance, if our model allows  $X$  also to have a direct effect on  $Y$ , our ability to learn from  $M$  will be more limited. We explore this issue in much greater detail in Chapter ??.

#### 9.4.2 Learning without identification

Some causal queries are *identified* while others are not. When a query is identified, each true value for the query is associated with a unique data

distribution given infinite data. Thus, as we gather more and more data, our posterior on the query should converge on the true value. When a query is not identified, multiple true values of the query will be associated with the same data distribution given infinite data. With a non-identified query, our posterior will never converge on a unique value regardless of how much data we collect since multiple answers will be equally consistent with the data. A key advantage of causal model framework, however, is that we can *learn* about queries that are not identified.

We can illustrate the difference between identified and non-identified causal questions by comparing an *ATE* query to a probability of causation (*PC*) query for a simple  $X \rightarrow Y$  model. When asking about the *ATE*, we are asking about the average effect of  $X$  on  $Y$ , or the difference between  $\lambda_{01}^Y$  (the share of units with positive effects) and  $\lambda_{10}^Y$  (share with negative effects). When asking about the *PC*, we are asking, for a case with given values of  $X$  and  $Y$ , about the probability that  $X$  caused  $Y$  in that case. And a *PC* query is defined by a different set of parameters. For, say, an  $X = 1, Y = 1$  case and a  $X \rightarrow Y$  model, the probability of causation is given by just  $\lambda_{01}^Y$ .

Let us assume a “true” set of parameters, unknown to the researcher, such that  $\lambda_{01}^Y = 0.6$ ,  $\lambda_{10}^Y = 0.1$  while we set  $\lambda_{00}^Y = 0.2$  and  $\lambda_{11}^Y = 0.1$ . Thus, the true average causal effect is 0.5. We now use the parameters and the model to simulate a large amount of data ( $N = 10,000$ ). We then return to the model, set flat priors over nodal types, and update the model using the simulated data. We graph the posterior on our two queries, the *ATE* and the probability of positive causation in an  $X = 1, Y = 1$  case, in Figure 9.3.

The figure illustrates nicely the difference between an identified and non-identified query. While the *ATE* converges on the right answer, the probability of causation fails to converge even with a massive amount of data. We see instead a range of values for this query on which our updated model places roughly equal posterior probability.

Importantly, however, we see that we *do* learn about the probability of causation. Despite the lack of convergence, our posterior rules out a wide range of values. While our prior on the query was 0.5, we have correctly updated toward a range of values that includes (and happens to be fairly well centered over) the true value ( $\approx 0.86$ ).

A distinctive feature of updating a causal model is that it allows us to learn about non-identified quantities in this manner. We will end up with “ridges”

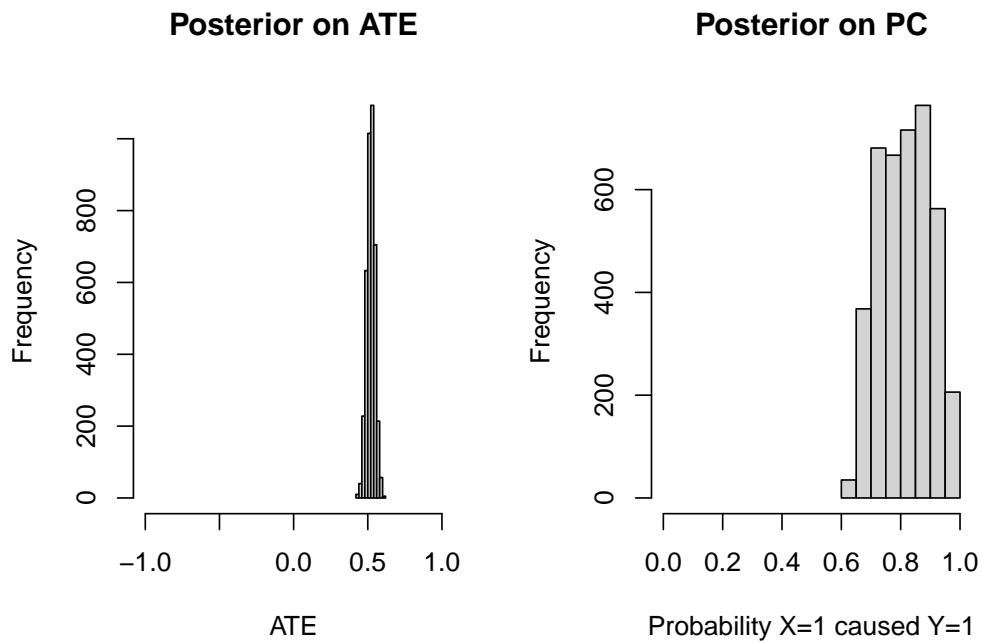


Figure 9.3: ATE is identified, PC is not identified but has informative bounds

in our posterior distributions: ranges or combinations of parameter values that are equally likely given the data. But our posterior weight can nonetheless shift toward the right answer.

At the same time, for non-identified queries, we have to be cautious about the impact of our priors. As  $N$  becomes large, the remaining curvature we see in our posteriors may simply be function of those priors. One way to inspect for this is to simulate a very large dataset and see whether variance shrinks. A second approach is to do sensitivity analyses by updating the model on the same data with different sets of priors to see how this affects the shape of the posterior.

### 9.4.3 Beyond binary data

While the setup used in this book involves only binary nodes, the approach readily generalizes to non-binary data. Moving beyond binary nodes allows for considerably greater flexibility in response functions. For instance, moving from binary to merely 3-level ordinal  $X$  and  $Y$  variables allows us to represent non-linear and even non-monotonic relationships. It also allows us pose more complex queries, such as, “What is the probability that  $Y$  is linear in  $X$ ?”, “What is the probability that  $Y$  is concave in  $X$ ?”, or “What is the probability that  $Y$  is monotonic in  $X$ ? ”

To move to non-binary measurement, we need to be able to expand the nodal-type space to accommodate the richer range of possible relations between nodes that can take on more than two possible values. Suppose, for instance, that we want to operate with variables with 4 ordinal categories. In an  $X \rightarrow Y$  model,  $Y$ ’s nodal types have to accommodate 4 possible values that  $X$  can take on, and 4 possible values that  $Y$  can take on for any value of  $X$ . This yields  $4^4 = 256$  nodal types for  $Y$  and 1024 causal types (compared to just 8 in a binary setup).

The **CausalQueries** package, set up to work most naturally with binary nodes, can be used to represent non-binary data as well. The trick, as it were, is to express integers in base-2 and then represent the integer as a series of 0’s and 1’s on multiple nodes. In base-2 counting we would represent four integer values for  $X$  (say, 0, 1, 2, 3) using 00, 01, 10, 11. If we use one binary node,  $X_1$  to represent the first digit, and a second node  $X_2$  to represent the second, we have enough information to capture the four values of  $X$ .

Table 9.7: Data from non binary model (selection of rows)

X1	X2	Y1	Y2	X	Y
0	0	0	0	0	0
1	0	1	1	2	3
1	0	1	1	2	3
1	1	1	0	3	2

Table 9.8: Posteriors on potential outcomes for non binary model

Q	Using	True value	mean	sd
Y(0)	posteriors	0	0.37	0.08
Y(1)	posteriors	1	0.98	0.07
Y(2)	posteriors	3	2.60	0.09
Y(3)	posteriors	2	2.02	0.07

The mapping then is:  $X_1 = 0, X_2 = 0$  represents  $X = 0$ ;  $X_1 = 0, X_2 = 1$  represents  $X = 1$ ;  $X_1 = 1, X_2 = 0$  represents  $X = 2$ ; and  $X_1 = 1, X_2 = 1$  represents  $X = 3$ . We construct  $Y$  in the same way. We can then represent a simple  $X \rightarrow Y$  relation as a model with two  $X$  nodes each pointing into two  $Y$  nodes:  $Y_1 \leftarrow X_1 \rightarrow Y_2, Y_1 \leftarrow X_2 \rightarrow Y_2$ . To allow for the full range of nodal types we need to allow a joint distribution over  $\theta^{X_1}$  and  $\theta^{X_2}$  and over  $\theta^{Y_1}$  and  $\theta^{Y_2}$ , which results in 3 degrees of freedom for  $X$  and 255 for  $Y$ , as required.

In the illustration below with two 4-level variables, we generate data ( $N = 100$ ) from a non-monotonic process with the following potential outcomes:  $Y(0) = 0, Y(1) = 1, Y(2) = 3, Y(3) = 2$ . We then update and report on posteriors on potential outcomes.

Data from this model looks like this:

Updating and querying is done in the usual way:

We see that the model performs well. As in the binary setup, the posterior reflects both the data and the priors. And, as usual, we have access to a full posterior distribution over all nodal types and can thus ask arbitrary queries of the updated model.

The greatest challenge posed by the move to non-binary data is computa-

tional. If  $Y$  takes on  $m$  possible values and has  $k$  parents, each taking on  $r$  possible values, we then have  $m^{r^k}$  nodal types for  $Y$ . Thus, the cost of more granular measurement is complexity – an explosion of the parameter space – as the nodal type space expands rapidly with the granularity of measurement and the number of explanatory variables. With three 3-level ordinal variables pointing into the same outcome, for instance, we have  $3^{27} = 7.6$  trillion nodal types!

We expect that, as measurement becomes more granular, researchers will want to manage the complexity by placing structure onto the possible patterns of causal effects. Structure, imposed through model restrictions, can quite rapidly tame the complexity. For some substantive problems, one form of structure we might be willing to impose is monotonicity. In a  $X \rightarrow Y$  model with 3-level variables, excluding non-monotonic effects brings down the number of nodal types from 27 to 17. Alternatively, we may have a strong reason to rule out effects in one direction: disallowing negative effects, for instance, brings us down to 10 nodal types. If we are willing to assume linearity, the number of nodal types falls further to 5.

#### 9.4.4 Measurement error

One potential application of the approach we have described in this chapter to integrating differing forms of data is to addressing the problem of measurement error. The conceptual move to address measurement error in a causal model setup is quite simple: we incorporate the error-generating process into our model.

Consider, for instance, a model in which we build in a process generating measurement error on the dependent variable.

$$X \rightarrow Y \rightarrow Y_{\text{measured}} \leftarrow \text{source of measurement error}$$

Here  $X$  has an effect on the true value of our outcome of interest,  $Y$ . The true value of  $Y$ , in turn, has an effect on the value of  $Y$  that we measure, but so too does a potential problem with our coding process. Thus, the measured value of  $Y$  is a function of both the true value and error.

To motivate the setup, imagine that we are interested in the effect of a rule restricting long-term care staff to working at a single site ( $X$ ) on outbreaks

of the novel coronavirus in long-term care facilities ( $Y$ ), defined as infections among two or more staff or residents. We do not directly observe infections, however; rather, we observe positive results of PCR tests. We also know that testing is neither comprehensive nor uniform. For some units, regular random testing is carried out on staff and residents while in others only symptomatic individuals are tested. It is the latter arrangement that potentially introduces measurement error.

If we approach the problem naively, ignoring measurement error and treating  $Y_{\text{measured}}$  as though it were identical to  $Y$ , a differences in means approach might produce attenuation bias—insofar as we are averaging between the true relationship and 0.

We can do better with a causal model, however. Without any additional data, we can update on both  $\lambda_Y$  and  $\lambda^{Y_{\text{measured}}}$ , and our posterior uncertainty would reflect uncertainty in measurement. We could go further if, for instance, we could reasonably exclude negative effects of  $Y$  on  $Y_{\text{measured}}$ . Then, if we observe (say) a negative correlation between  $X$  and  $Y_{\text{measured}}$ , we can update on the substantive effect of interest –  $\lambda^Y$  – in the direction of a larger share of negative effects: it is only *via* negative effects of  $X$  on  $Y$  that a negative correlation between  $X$  and  $Y_{\text{measured}}$  could emerge. At the same time, we learn about the measure itself as we update on  $\lambda^{Y_{\text{measured}}}$ : the negative observed correlation  $X$  and  $Y_{\text{measured}}$  is an indicator of the degree to which  $Y_{\text{measured}}$  is picking up true  $Y$ .

We can do better still if we can collect more detailed information on at least some units. One data strategy would be to invest in observing  $Y$ , the true outbreak status of each unit, for a subset of units for which we already have data on  $X$  and  $Y_{\text{measured}}$  — perhaps by implementing a random-testing protocol at a subset of facilities. Getting better measures of  $Y$  for some cases will allow us to update more directly on  $\lambda^Y$ , the true effect of  $X$  on  $Y$ , for those cases. But just as importantly, observing true  $Y$  will allow us to update on measurement *quality*,  $\lambda^{Y_{\text{measured}}}$ , and thus help us make better use of the data we have for those cases where we only observe  $Y_{\text{measured}}$ . This strategy, of course, parallels a commonly prescribed use of mixed methods, in which qualitative research takes place in a small set of units to generate more credible measures for large- $n$  analysis (see, e.g., Seawright (2016)).

In the illustration below, we posit a true average effect of  $X$  on  $Y$  of 0.6. We also posit an average “effect” of  $Y$  on measured  $Y$  of just 0.7, allowing for

Table 9.9: Inferences on effects on true  $Y$  given measurement error (true ATE = .6)

Data	Using	mean	sd
Data on $Y$ measured only	posteriors	0.64	0.09
Data on true $Y$ for 20% of units	posteriors	0.63	0.03
Data on true $Y$	posteriors	0.61	0.02

measurement error.

In this setup, with a large amount of data, we would arrive at a differences-in-means estimate of the effect of  $X$  on *measured Y* of about 0.42. Importantly, this would be the effect of  $X$  on  $Y_{\text{measured}}$  — not the effect of  $X$  on  $Y$  — but if we were not thinking about the possibility of measurement error, we would likely conflate the two, arriving at an estimate far from the true value.

We can improve on this “naive” estimate in a number of ways using a causal model, as shown in Table 9.9. First, we can do much better simply by undertaking the estimation within a causal model framework, even if we simply make use of the exact same data. We write down the following simple model  $X \rightarrow Y \rightarrow Y_{\text{measured}}$ , and we build in a monotonicity restriction that disallows negative effects of  $Y$  on  $Y_{\text{measured}}$ . As we can see from the first row in Table 9.9, our mean estimate of the *ATE* moves much closer to the true value of 0.6.

Second, we can add data by gathering measures of “true”  $Y$  for 20% of our sample. As we can see from the second row in the table, this investment in additional data does not change our posterior mean much but yields a dramatic increase in precision. In fact, as we can see by comparison to the third row, partial data on “true”  $Y$  yields an estimate that is almost the same and almost as precise as the one we would arrive it with data on “true”  $Y$  for *all* cases.

An alternative strategy might involve gathering multiple measures of  $Y$ , each with their own independent source of error. Consider the model,  $X \rightarrow Y \rightarrow Y_{\text{measured}[1]}; Y \rightarrow Y_{\text{measured}[2]}$ . Assume again a true *ATE* of  $X$  on  $Y$  of 0.6, that  $Y$  has an average effect of 0.7 on both  $Y_{\text{measured}[1]}$  and  $Y_{\text{measured}[2]}$ , and no negative effects of true  $Y$  on the measures.<sup>2</sup> In this setup, updating on

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<sup>2</sup>Importantly, this model assumes nodal types for  $Y_{\text{measured}[1]}$  and  $Y_{\text{measured}[2]}$  are in-

Table 9.10: Inferences on effects on true  $Y$  given two noisy measures (true ATE = .6)

Data	Using	mean	sd
Two noisy measures	posterioris	0.61	0.02

the true  $Y$  can be thought of as a Bayesian version of “triangulation”, or factor analysis. The results in Table 9.10 are based the same data as in the previous example but now augmented with the second noisy measure for  $Y$ .

As we can see, two noisy measures perform about as well as access to full data on the true  $Y$  (as in Table 9.9).

The main point here is that measurement error matters for inference and can be taken directly into account within a causal model framework. Confusing measured variables for variables of interest will obviously lead to false conclusions. But if measurement concerns loom large, we can respond by making them part of our model and learning about them. We have illustrated this point for simple setups, but more complex structures could be just as well envisioned, such as those where error is related to  $X$  or, more perniciously, to the effects of  $X$  on  $Y$ .

#### 9.4.5 Spillovers

A common threat to causal inference is the possibility of spillovers: a given unit’s outcome being affected by the treatment status of another (e.g., possibly neighboring) unit. We can readily set up a causal model to allow for estimation of various quantities related to spillovers.

Consider, for instance, the causal model represented in Figure ???. We consider here a cluster of 3 units across which spillovers might occur. We might imagine, for instance, a cluster of geographically proximate villages separated from other clusters such that spillovers might occur between villages within a cluster, but can be ruled out across clusters. Here  $X_i$  and  $Y_i$  represent village  $i$ ’s treatment status and outcome, respectively. The pattern of directed

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dependent of one another (no unobserved confounding), implying independent sources of measurement error in this setup.

edges indicates that each village's outcome might be affected both by its own and by its neighbors' treatment status.

We now simulate data that allow for spillovers. Specifically, while independently assigning  $X_1$  and  $X_2$  to treatment 50% of the time, we (a) set  $Y_1$  equal to  $X_1$ , meaning that Unit 1 is affected only by its own treatment status and (b) set  $Y_2$  equal to  $X_1 \times X_2$ , meaning that Unit 2 is equally affected by its own treatment status and that of its neighbor, such that  $Y_2 = 1$  only if both Unit 2 and its neighbor are assigned to treatment.

We simulate 100 observations from this data-generating process and then update a model (with flat priors over all nodal types).

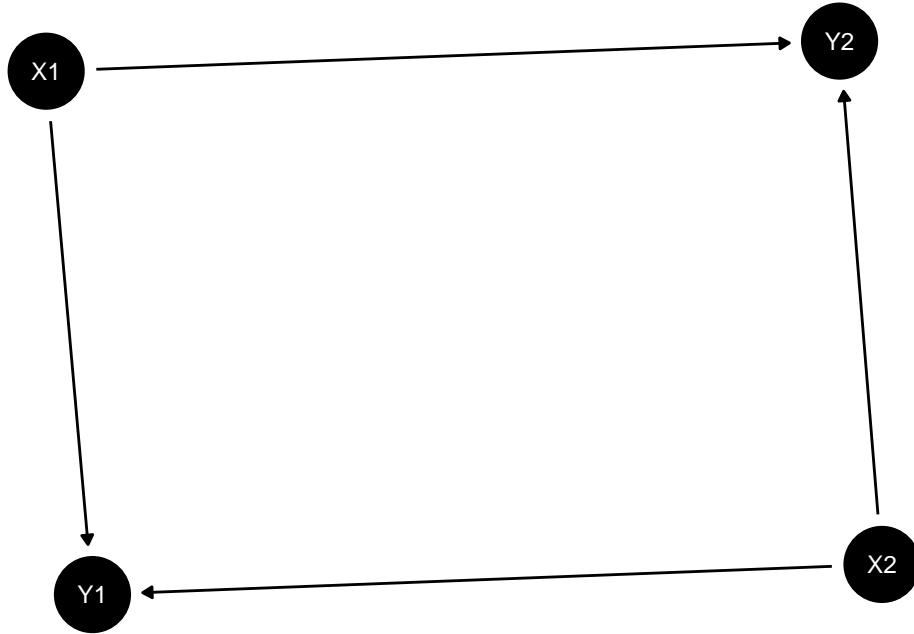
Now we can extract a number of spillover-relevant causal quantities from the updated model. First we ask: what is the average effect of exposing a unit *directly* to treatment (“only\_self\_treated”) when the neighboring unit is untreated? Under the data-generating process that we have posited, we know that this effect will be 1 for Unit 1 (which always has a positive treatment effect) and 0 for Unit 2 (which sees a positive effect of  $X_2$  only when  $X_1 = 1$ ), yielding an average across the two units of 0.5. We see that we update, given our 100 observations, from a prior of 0 to a posterior mean of 0.371, approaching the right answer.

A second question we can ask is about the spillover by itself: what is the average treatment effect for a unit of its neighbor being assigned to treatment when the unit itself is not assigned to treatment (“only\_other\_treated”)? We know that the correct answer is 0 since Unit 1 responds only to its own treatment status, and Unit 2 requires that both units be assigned to treatment to see an effect. Our posterior estimate of this effect is right on target, at 0.

We can then ask about the average effect of *any* one unit being treated, as compared to no units being treated (“one\_treated”). This is a more complex quantity. To estimate it, we have to consider what happens to the outcome in Unit 1 when only  $X_1$  shifts from control to treatment, with  $X_2$  at control (true effect is 1); what happens to Unit 1 when only  $X_2$  shifts from control to treatment, with  $X_1$  at control (true effect is 0); and the same two effects for Unit 2 (both true effects are 0). We then average across both the treatment conditions and units. We arrive at a posterior mean of 0.186, not far from the true value of 0.25.

Finally, we can ask about the average effect of both treatments going from control to treatment (“both\_treated”). The true value of this effect is 1 for both units, and the posterior has shifted quite far in the direction of this value.

Obviously, more complex setups are possible. We can also model the process in a way that allows for more learning (pooling) across units. In the present model, learning about effects for Unit 1 in a cluster tells us nothing about effects for Unit 2 in a cluster because they are set up to have completely independent nodal types. We could instead treat all units as drawn from the same population: we could represent this, for instance, in a graph with just one  $Y$  and two treatment nodes pointing into it, one for the unit’s own treatment status and one for its neighbor’s treatment status.



#### 9.4.6 Clustering

We can also represent some forms of clustering, understood as the presence of an exogenous but unobserved factor that influences outcomes for some sub-

Table 9.11: Spillovers queries

Query	Using	mean	sd
only_self_treated	posteriors	0.37	0.05
only_other_treated	posteriors	0.00	0.04
one_treated	posteriors	0.19	0.04
both_treated	posteriors	0.75	0.05

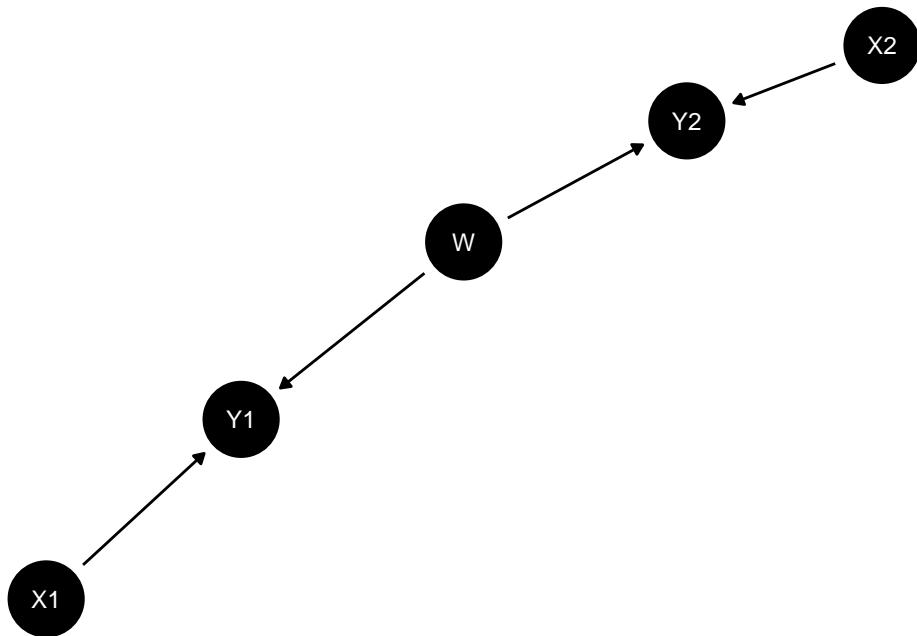
group of units. For instance, we might be interested in the effects of training on individuals' employment prospects, while recognizing that individuals living in the same neighborhood will be affected by common neighborhood-level features that we cannot directly observe.

We can capture some forms of clustering by placing the cluster-level factor on the graph.<sup>3</sup> Let us imagine that we are studying the effect of providing public health insurance coverage on health outcomes for individuals in two-adult households that have a single earner. Our units of analysis are individuals, but these units are clustered into pairs within households.

We can represent this situation via the following structural model:

---

<sup>3</sup>In this illustration the two units in each pair are treated as separate nodes rather than as repeated instances of realizations of the same node. Implicitly then the effect for one unit type (men, say) can be quite independent of the effect of another type (women, say). Indeed, here they are linked only through the unobserved variable  $W$ .



Let Unit 1 be the adult in paid employment and Unit 2 the adult not in paid employment, with  $X_i$  indicating the provision of public health insurance to Unit  $i$  and  $W$  representing an unobserved household-level factor that moderates causal effects for both units. We restrict the model such that, more specifically,  $W$  shuts off effects of health insurance for both individuals. By representing earners and non-earners separately on the graph, we allow for different effects of health insurance for these two different kinds of individuals. By representing the cluster-level factor,  $W$ , on the graph, we also allow for *learning* across types of units: seeing effects (or non-effects) for one kind of unit allows us to update on  $W$ 's value, which in turn provides information about effects for the other type of unit.

One question we can ask with this setup is: would we learn more from concentrating our observations within a smaller number of clusters or spreading them out across clusters? In Table 9.12, we show results from two different data-collection strategies. In one instance, we observe both Unit 1 and Unit 2 in two clusters. In the other instance, we observe only Unit 1 in two clusters and only Unit 2 in two clusters. In both situations, we calculate the same estimand, the average treatment effect, defined as the effect of providing

Table 9.12: Data from many pairs is more informative than the same data from fewer pairs.

Data	mean	sd
2 obs from each of 2 clusters	0.018	0.094
1 obs from each of 4 clusters	0.020	0.095

health insurance, averaged across the two unit types.



# Chapter 10

## Integrated Inferences Application: Inequality and Democracy Revisited

### 10.1 A trained model

Whereas in Chapter 8 we took the model as given and sought to draw inferences individual cases given data on those cases, the model now becomes an object that we both learn from and learn about. In essence we use the data on many cases to update our beliefs about the general model and then use this “trained” model to make inferences about cases.

Instead of positing a belief over the nodal types for a given case,  $\theta$ , we now need to posit a belief over the *distribution* of nodal types—that is, over  $\lambda$ . For instance, whereas in the simple process-tracing model we *specified* the shares of nodal types for  $M$ , we now specify a *distribution* over the nodal type shares, using a Dirichlet distribution to ensure that the shares across types are always constrained to add up to 1. We do the same, of course, for all nodes. Because we set a prior distribution over nodal types (rather than fixed proportions), we can now update on these population-level distributions as the model confronts data.

The same applies to beliefs about confounding. Recall that we allow for unobserved confounding by allowing  $\lambda$  to include beliefs about the *joint* dis-

tributions of nodal types, and we set priors on these joint distributions as well. In the application below, we focus on potential confounding in the relationship between inequality and mobilization: the possibility that inequality may be more or less likely in places where inequality would induce mobilization. Here we do not express informed prior beliefs about the direction or magnitude of such confounding; we set up the parameter matrix to allow for the possibility of confounding and set a flat prior over its direction and magnitude. We can, in turn, learn about confounding from the data.

We begin with the same basic model as we used in Chapter 8, with inequality ( $I$ ) potentially affecting democratization ( $D$ ) both through a direct pathway and through an indirect pathway mediated by mobilization ( $M$ ). International pressure ( $P$ ) is also a “parent” of democratization.

Further, we impose the same set of qualitative restrictions, ruling out a negative effect of inequality on mobilization, a direct positive effect of inequality on democratization, a negative effect of mobilization on democracy, and a negative effect of pressure on democratization. Note that this setup allows for inequality to have a positive (through mobilization) effect on democratization, a negative (direct) effect on democratization, or no effect at all.

Finally, we allow for confounding. The theoretical intuition we want to instantiate in the model is that the level of inequality could be endogenous to inequality’s effect on mobilization. In particular, in places where mobilization would pose a mobilizational threat, governments may work harder to reduce inequality. To allow for this possibility, we need to create distinct elements of  $\lambda$  representing the conditional distribution of  $I$ ’s nodal types given  $M$ ’s: one parameter for  $\theta^I$ ’s distribution when  $M$ ’s nodal type is  $\theta_{01}^M$ , and another parameter for  $\theta^I$ ’s distribution when  $M$ ’s nodal type is something else.

This model, with confounding, is represented graphically as in Figure 10.1. The possibility of confounding is represented with the bidirected edge, connecting  $I$  and  $M$ .

## 10.2 Data

As in Chapter 8, we will confront the model with data drawn from our coding of the case narratives in the Supplementary Material for Haggard and

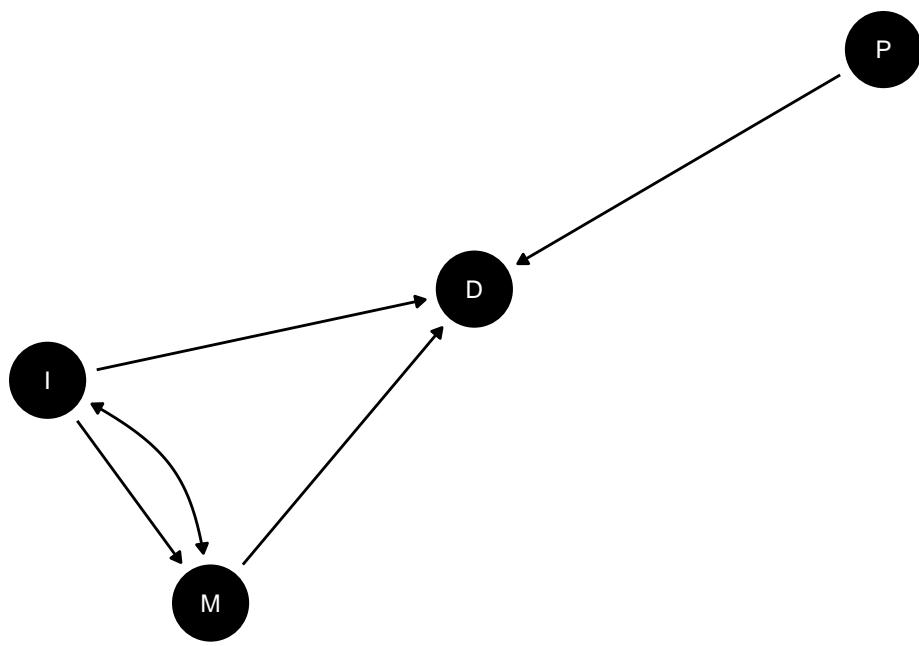


Figure 10.1: Democracy and Inequality Model

Table 10.1: Data (snapshot) derived from Haggard and Kaufman (2012)

Case	P	I	M	D
Afghanistan		1		0
Albania	0	0	1	1
Algeria		0		0
Angola		1		0
Argentina	0	0	1	1
Bangladesh	0	0	0	1

Kaufman (2012). However, rather than implementing the analysis case-by-case, we now derive leverage from the joint distribution of the data available across all cases.

Table 10.1 gives a snapshot of the data.

Note that this is not a rectangular dataset in that Haggard and Kaufman's collection of clues was conditional on the outcome,  $D = 1$ : they gathered qualitative data on the presence of international pressure and the presence of mass-mobilization *only* for those cases that democratized. This is not an uncommon case-selection principle. The analyst often reasons that more can be learned about how an outcome arises by focusing in on cases where the outcome of interest has in fact occurred. (We assess this case-selection intuition, in the context of model-based inferences, in Chapter ??.)

The raw correlations between variables is shown in Table 10.2. Some correlations are missing because, as mentioned, data on some variables were only gathered conditional on the values of others. For those quantities where we do see correlations, they are not especially strong. There is, in particular, a weak overall relationship between inequality and democratization — though, of course, this is consistent with inequality having heterogeneous effects across the sample. The strongest correlation in the data is between  $P$  and  $M$ , which are assumed to be uncorrelated in the model, though this correlation is also quite weak.

Table 10.2: Pairwise correlations in the democracy-inequality (PIMD) data.  
 P = Pressure, I = Inequality, M = Mobilization, D = Democratization

	P	I	M	D
P	1.000	0.157	-0.177	
I	0.157	1.000	0.114	-0.154
M	-0.177	0.114	1.000	
D		-0.154		1.000

## 10.3 Inference

With data and model in hand, we can now update our model to get posteriors on the distribution  $\lambda$  from which we can generate beliefs over all causal relations.

What do we find?

### 10.3.1 Did inequality *cause* democratization?

We have used the data to update on  $\lambda$ : our beliefs about the distributions of nodal types, including about their joint distributions (i.e., confounding). We first use this to make claims about types, similar to what we did in Chapter 8 but now with a model that has been trained on data.

In Table 10.3 we first ask what we would now infer about cases given democratization and low inequality and different observations on mobilization and international pressure. This table is the analogue of Table REF in Chapter 8.

In Table 10.3 we ask what we would now infer about cases given democratization and *high* inequality and different observations on mobilization and international pressure. This table is the analogue of Table REF in Chapter 8.

Table 10.3: Four cases with low inequality and democratization. Question of interest: Was low inequality a cause of democracy? Table shows posterior beliefs for different data for four cases given information on  $M$  or  $P$ . Data from Haggard and Kaufman (2012). Analyses here use a model with assumptions on monotonic effects but magnitudes of effects updated from data.

Case	M: Mobilization?	P: Pressure?	No clues	M only	P only	M and P
Mexico (2000)	0	0	0.438	0.475	0.615	0.667
Taiwan (1996)	0	1	0.438	0.475	0.34	0.393
Albania (1991)	1	0	0.438	0.394	0.615	0.571
Nicaragua (1984)	1	1	0.438	0.394	0.34	0.263

Table 10.4: Four cases with high inequality and democratization. Question of interest: Was high inequality a cause of democratization? Table shows posterior beliefs for different data for 4 cases given information on  $M$  or  $P$ . Data from Haggard and Kaufman (2012). Analyses here use a model with assumptions on monotonic effects but magnitudes of effects updated from data.r

Case	M: Mobilization?	P: Pressure?	No clues	M only	P only	M and P
Mongolia (1990)	0	0	0.128	0	0.231	0
Paraguay (1989)	0	1	0.128	0	0.088	0
Sierra Leone (1996)	1	0	0.128	0.15	0.231	0.25
Malawi (1994)	1	1	0.128	0.15	0.088	0.107

Table 10.5: No inequality and No democratization: Was no inequality a cause of no democratization? Analyses here use priors assuming only monotonic effects.

	P	M	posterior
I0P0M0D0	0	0	0.122
I0P1M0D0	1	0	0.259
I0P0M1D0	0	1	0.000
I0P1M1D0	1	1	0.000
I0M0D0		0	0.139
I0M1D0		1	0.000
I0P0D0	0		0.097
I0P1D0	1		0.233
I0D0			0.112

### 10.3.2 Did inequality *prevent* democracy?

As before we can also ask questions about causes that have not democratized – even though we have no additional data about these cases in particular.

## 10.4 From cases to population

One set of questions we can ask of the updated model is about the probability that high inequality causes democratization. We can pose this question at different levels of conditioning. For instance, we can ask:

1. **For all cases.** For what proportion of cases in the population does inequality have a positive effect on democratization?
2. **For all cases displaying a given causal state and outcome.** Looking specifically at those cases that in fact had high inequality and democratized, for what proportion was the high inequality a cause of democratization?
3. **For cases displaying a given causal state and outcome, and with additional clues present or absent.** What if we have also

Table 10.6: Inequality and No democratization: Was inequality a cause of no democratization? Analyses here use priors assuming only monotonic effects.

	P	M	posterior
I1P0M0D0	0	0	0.247
I1P1M0D0	1	0	0.565
I1P0M1D0	0	1	0.393
I1P1M1D0	1	1	0.655
I1M0D0		0	0.309
I1M1D0		1	0.424
I1P0D0	0		0.313
I1P1D0	1		0.594
I1D0			0.359

collected clues on mediating or moderating nodes? For instance, for what proportion of high-inequality, democratizing cases *with* mobilization did inequality cause the outcome? For what proportion *without* mobilization? Likewise for the presence or absence of international pressure? Importantly, comparing a given estimate with and without a given clue amounts to an assessment of the clue's probative value.

We ask **CausalQueries** now to query  $\lambda$ 's posterior distribution to generate posterior distributions for each of these quantities. We can define our queries quite simply in terms of the causal types that correspond to the effect of interest and then take the conditional probability of these. We present the code and results of these operations below and in Figure 10.2.

In figure @??fig:pimdqueries), we graph the priors for each of these queries next to the posteriors.

```
## `stat_bin()` using `bins = 30`. Pick better value
## with `binwidth`.

## Warning: Removed 12 rows containing missing values
## (geom_bar).
```

We can see that the share of cases overall in which inequality causes democratization is estimated to be very low, with a good deal of confidence. The

Table 10.7: Probability that inequality increases chances of democratization given:

Given	mean
-	0.04
$D==1 \& I==1$	0.11
$D==1 \& I==1 \& M==1$	0.16
$D==1 \& I==1 \& M==0$	0.00
$D==1 \& I==1 \& M==1 \& P==1$	0.09
$D==1 \& I==1 \& M==1 \& P==0$	0.20

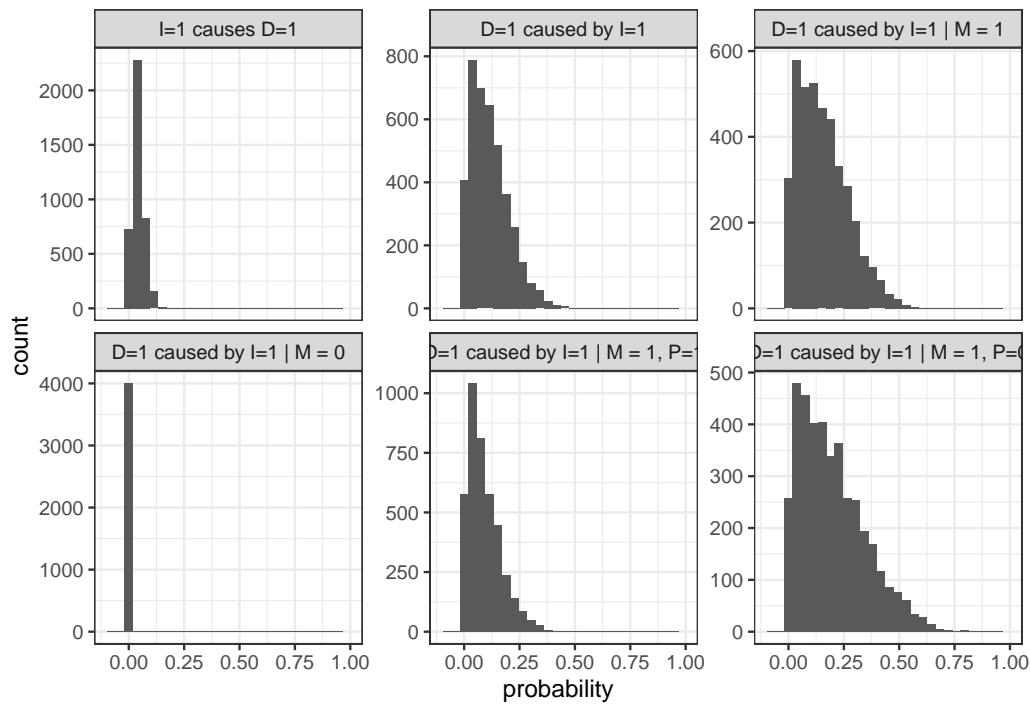


Figure 10.2: Posteriors on Causes of Democratization

proportion is considerably higher for those cases that in fact experienced high inequality and democratization. The proportion of positive causal effects is believed to be even higher for those in which mobilization occurred. Moreover, the proportion of  $I = 1, D = 1$  cases with a positive effect of inequality on democratization is even higher when an alternative cause—international pressure—is absent, though our uncertainty about this share is also very high.

We also see that the absence of mobilization tells us for certain that democratization was not caused by inequality. Interestingly, however, this result derives purely from the model restrictions, rather than from the data: under the restrictions we imposed, a positive effect of inequality can operate *only* through mobilization.

Turning now to the cases in which democratization did not occur, Figure 10.3 asks for what proportion of cases overall inequality has a negative effect on democratization; for what proportion of  $I = 1, D = 0$  cases inequality prevented democratization; and this latter query conditional on different clue realizations.

```
## `stat_bin()` using `bins = 30`. Pick better value
## with `binwidth`.

## Warning: Removed 12 rows containing missing values
## (geom_bar).
```

We see that inequality appears, overall, more commonly to prevent democratization than to cause it. We are, moreover, most confident that inequality played a preventive role in those cases in which there was mobilization and international pressure—both of which *could* have generated democratization—but still no democratization occurred.

#### 10.4.1 Contribution to case-level inference

There are two ways of thinking about the learning we derive from the above estimates. On the one hand, we can think of our estimands in population-level terms. The results in Figures 10.2 and 10.3 can be understood as

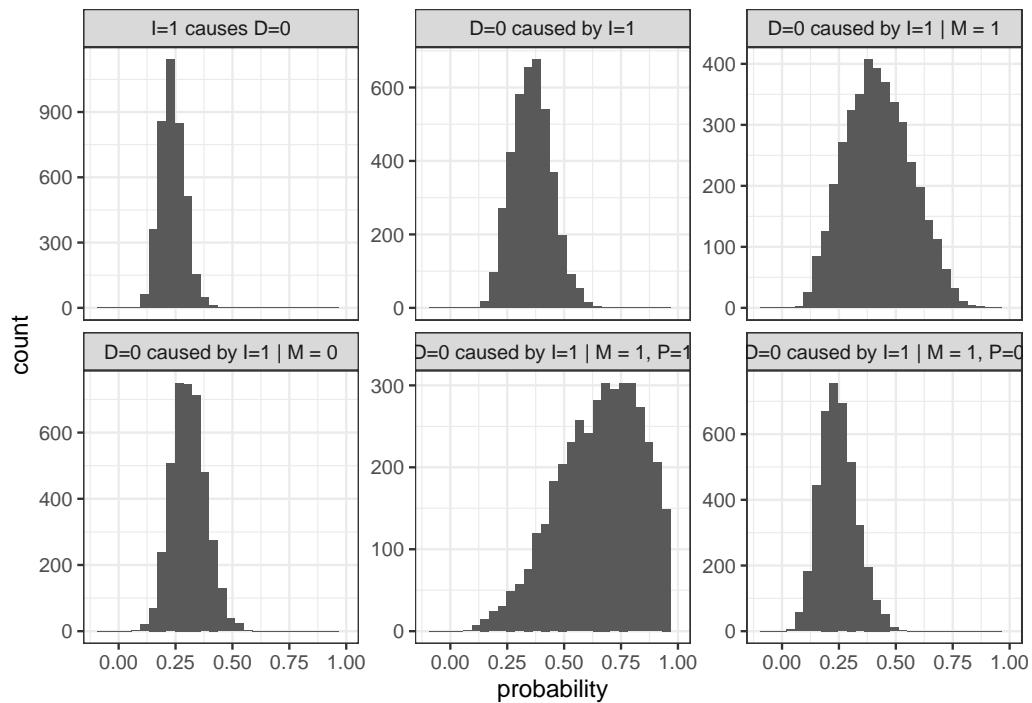


Figure 10.3: Posteriors on Causes of Democratization

our estimates of the *share* of cases in the population, with a given set of characteristics, for which a particular causal effect holds.

Yet these distributions, by the very same token, represent our beliefs about the probability that  $I$  had a positive or negative (depending on the query we are talking about) causal effect in an individual case for which we have seen a given data pattern. Thus, for instance, from Figure 10.2 we can see our posterior belief about the proportion of  $I = D = M = 1$  cases in the population for which  $I = 1$  caused  $D = 1$ . But this also tells us, for an individual case randomly selected from the population of  $I = D = M = 1$  cases, the probability that high inequality caused democratization in the case. To put the point differently, suppose we want to know whether  $I = 1$  was a cause of  $D = 1$  in a randomly selected case, so we do process tracing and observe  $M = 1$ : this same estimate answers the case-level query, telling us the probability that  $I = 1$  caused  $D = 1$  in the case at hand.

The key difference between case-level inference from mixed data and the pure process-tracing setup from Chapter 8 is that now our case-level inferences are informed by *data* from the population, rather than merely by a set of prior beliefs about the population. Consider, for instance, the difference in posteriors on the probability of a negative effect for the query for  $I = 1, D = 0$  given  $M = 0$  (mean of about 0.31) as compared to the query for  $I = 1, D = 0$  given  $M = 1$  (about 0.42). That difference in what we believe depending on  $M$ 's value represents the probative value of the  $M$  clue given the model as *updated* from the data. Of course, we have also made assumptions in building the model, including monotonicity restrictions, and we speak to their role below. But the general point is that, in mixed-data inference, we can learn empirically about the probative value of any given node, rather than drawing probative value purely from theory.

#### 10.4.2 How much do we get from the model vs. the data?

We might wonder, at the same time, how much we are in fact learning from the data, as compared to what we built into the model at the outset, including through the monotonicity restrictions that we imposed. To examine this, in Figures 10.4 and 10.5, we plot the mean (and one-standard-deviation error bars) of our prior and our posterior on the same set of queries. We see

that there is almost no shift in beliefs for the positive-effect queries, with a somewhat greater shift in means for the negative-effect queries. However, our uncertainty about negative effects shrinks some, though remains quite high.

By comparing the prior and posterior estimates given  $M = 1$  and given  $M = 0$ , we can also assess whether we have learned about  $M$ 's informativeness. We see that, in this example, we do not happen to learn anything from the data about  $M$ 's probative value: the difference in beliefs between each estimate given  $M = 1$  and that estimate given  $M = 0$  remains about the same in our posteriors as it was in our priors.

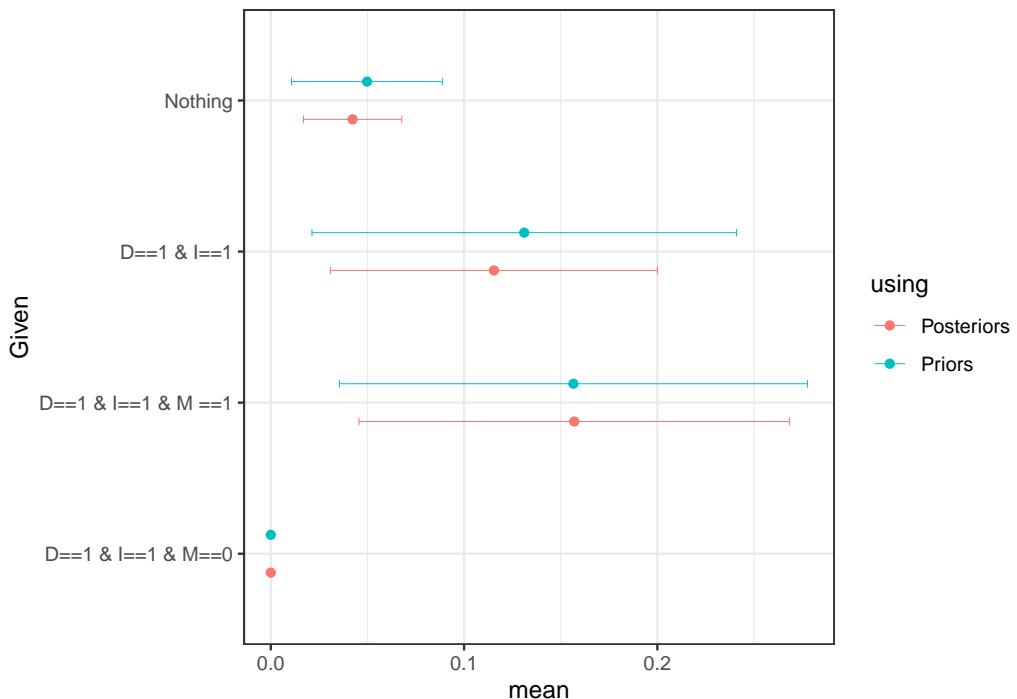


Figure 10.4: Priors and posteriors on the probability that inequality caused democratization (probability that I has a positive effect on D) given different kinds of observed case-level data. The error bars show plus or minus one standard deviation of the posterior variance.

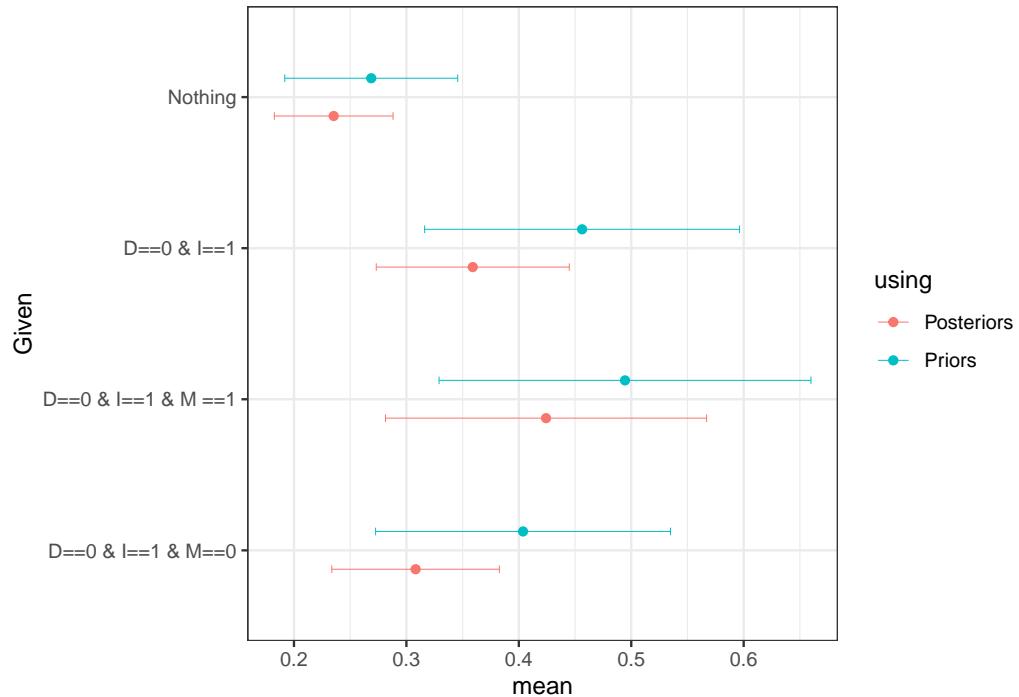


Figure 10.5: Priors and posteriors on the probability that inequality prevented democratization (probability that I has a negative effect on D) given different kinds of observed case-level data. The error bars show plus or minus one standard deviation of the respective distribution's variance.

# Chapter 11

## Mixing models

In the previous two chapters, we described one form of integration that structural causal models can enable: the systematic combination of (what we typically think of as) qualitative and quantitative evidence for the purposes of drawing population- and case-level causal inferences. One feature of the analyses we have been considering so far is that the integration is essentially “nested.” We are, for instance, integrating quantitative evidence for a large set of cases with qualitative evidence for a *subset* of those cases. We are, moreover, drawing inferences from the set of cases we observe to a population *within which* that sample of cases is situated.

In this chapter, we examine how we can use structural causal models to integrate across studies or settings that are, in a sense, more disjointed from one another: across studies that examine different causal relationships altogether; study designs that require different assumptions about exogeneity; and contexts across which the causal quantities of interest may vary.

1. **Integrating across a model** Often, individual studies in a substantive domain examine distinct segments of a broader web of causal relationships. For instance, while one study might examine the effect of  $X$  on  $Y$ , another might examine the effect of  $Z$  on  $Y$ , and yet another might examine the effect of  $Z$  on  $K$ . We show in this chapter how we can integrate across such studies in ways that yield learning that we could not achieve by taking each study on its own terms.

2. **Integrating between experimental and observational studies** One form of multi-method research that has become increasingly common is the use of both observational and experimental methods to study the same basic causal relationships. While an experiment can offer causal identification in a usually local or highly controlled setting, an observational analysis can often shed light on how the same relationships operate “in the wild,” if with greater risk of confounding. Usually, observational and experimental results are presented in parallel, as separate sources of support for a causal claim. We show how, in a causal model setup, we can use experimental and observational data *jointly* to address questions that cannot be answered when the designs are considered separately.
3. **Transporting knowledge across contexts** Researchers are sometimes in a situation in which they can identify causal quantities in a particular setting — say, from a randomized controlled trial implemented in a specific local context — but want to know how those inferences travel to other settings. Would the intervention work differently in other countries or regions? As we will explain, with an appropriately specified causal model and the right data from the original context, we can draw inferences about causal relationships in other contexts.
4. **Models in hierarchies.** Sometimes researchers learn about the same types of processes in different settings. By thinking of the processes in each setting as deriving from a family of processes, researchers can learn from observations in one setting about causal processes in another and also learn about the nature of heterogeneity between settings.

Before delving into the details of these strategies, we make one key qualification explicit: each of these approaches requires us to believe that setting-, or study-, specific causal model can be nested within a lower level, “encompassing,” model that operates across the multiple settings that we are learning from and want to draw inferences about. Encompassing models, of course, can specifically take heterogeneity across settings into account, for instance by including in the model moderators that condition the effects of interest. But we have to believe that we have indeed captured in the model any ways in which relationships vary across the set of contexts across which we are integrating evidence or transporting inferences.

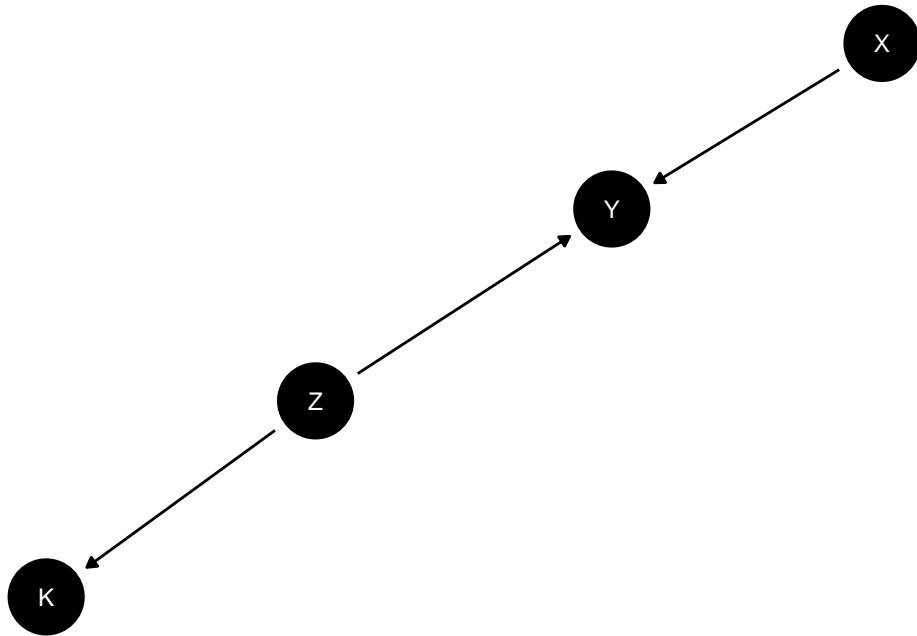
Put differently, and perhaps more positively, we see social scientists commonly seeking to transport knowledge or combine information informally across studies and settings. Often such efforts are motivated, sometimes implicitly, by an interest in or reliance on general theoretical propositions. The approaches that we describe below force the researcher to be *explicit* about the underlying causal beliefs that warrant that integration while also ensuring that the integration proceeds in a way that is logically consistent with stated beliefs.

## 11.1 A jigsaw puzzle: Integrating across a model

Generating knowledge about a causal domain often involves cumulating learning across studies that each focus in on some specific part of the domain. For instance, scholars interested in the political economy and democratization might undertake studies focused on the relationship between inequality and mass protests; studies on the role of mass mobilization in generating regime change; pathways other than mass mobilization through which inequality might affect democratization; studies of the role of international sanctions on the likelihood that autocracies will democratize; and studies of the effects of democratization on other things, such as growth or the distribution of resources.

We can think of these studies as each analyzing data on a particular part of a broader, more encompassing causal model. In an informal way, *if* findings “hold together” in a reasonably intuitive way, we might be able to piece together an impression of the overall relations among variables in this domain. Yet an informal approach becomes more difficult for complex models or data patterns and, more importantly, will leave opportunities for learning unexploited.

Consider this simple DAG, in which both  $X$  and  $Z$  are causes of  $Y$ , and  $Z$  also causes  $K$ . Now imagine three studies, all conducted in contexts in which we believe this model to hold:



1. Study 1 is an RCT in which  $X$  is randomized, with data collected on both  $Y$  and  $K$ .  $Z$  is not observed.
2. Study 2 is a factorial experiment, in which  $X$  and  $Z$  are independently randomized, allowing an examination of the joint effects of  $X$  and  $Z$  on  $Y$ .  $K$  is not observed.
3. Study 3 is an experiment randomizing  $Z$ , with only  $K$  observed as an outcome.  $X$  and  $Y$  are not observed.

Now, let's say that our primary interest is in the relationship between  $X$  and  $Y$ . Obviously, Study 1 will, with a sufficiently large sample, perform just fine in estimating the average treatment effect of  $X$  on  $Y$ . However, what if we are interested in a case-oriented query, such as the probability of causation: the probability, say,  $X = 1$  caused  $Y = 1$  in a given  $X = 1, Y = 1$  case?

We know that within-case, process-tracing clues can sometimes provide probative value on case-level estimands like the probability of causation, and we have observed  $K$  in the Study 3 cases. So what if we combine the  $X$ ,  $Y$ , and  $K$  data?

Table 11.1: The clue  $K$  uninformative in all three studies

Study	Given	mean	sd
1	$X == 1 \& Y == 1 \& K == 1$	0.652	0.107
	$X == 1 \& Y == 1 \& K == 0$	0.640	0.108
2	$X == 1 \& Y == 1 \& K == 1$	0.645	0.138
	$X == 1 \& Y == 1 \& K == 0$	0.645	0.137
3	$X == 1 \& Y == 1 \& K == 1$	0.499	0.150
	$X == 1 \& Y == 1 \& K == 0$	0.499	0.134

A simple analysis of the graph tells us that  $K$  cannot help us learn about  $Y$ 's potential outcomes since  $K$  and  $Y$  are  $d$ -separated by  $Z$ , and we have not observed  $Z$  in Study 3. We see this confirmed in Table 11.1.

In the first pair of rows, we show the results of analyses in which we have simulated data from the whole model, then updated using the Study 1 observations. We give here the posterior mean on the probability of causation for an  $X = Y = 1$  case, conditional on each possible value that  $K$  might take on. As we can see, our beliefs about the estimand remain unaffected by  $K$ 's value, meaning that it contains no information about  $X$ 's effect in the case.

We see that the same thing is true for each of the other studies. In study 2, we have not used  $K$  to update the model, and so have not learned anything from the data about  $K$ 's relationship to the other variables. Thus, we have no foundation on which to ground probative value for  $K$ . In study 3, we understand the  $Z, K$  relationship well, but know nothing quantitatively about how  $Z$  and  $X$  relate to  $Y$ . Thus, we have learned nothing from Study 3 about what observing  $K$  might tell us about the effect of  $X$  on  $Y$ .

We can do much better, however, if we combine the data and update *jointly* across all model parameters. The results are shown in Table 11.2. Updating simultaneously across the studies allows us, in a sense, to bridge across inferences. In particular, inferences from Study 2 make  $Z$  informative about  $Y$ 's potential outcomes under different values of  $X$ . Meanwhile, inferences from the data in Study 3 allow us to use information on  $K$  to update on values for  $Z$ . As we now see in rows 1 and 2, having updated the model in an integrated fashion,  $K$  now *is* informative about the probability of causation, with our posterior mean on this query changing substantially depending on the value of  $K$  that we observe in a case.

Table 11.2: Clue is informative after combining studies linking  $K$  to  $Z$  and  $Z$  to  $Y$

Given	mean	sd
$X == 1 \& Y == 1 \& K == 1$	0.79	0.08
$X == 1 \& Y == 1 \& K == 0$	0.62	0.12
$X == 1 \& Y == 1 \& K == 1 \& Z == 1$	0.84	0.08
$X == 1 \& Y == 1 \& K == 0 \& Z == 1$	0.84	0.08

Rows 3-4 highlight that the updating works through inferences on  $Z$ : we see that if  $Z$  is already known (we show this for  $Z = 1$ , but it holds for  $Z = 0$  as well), then there are no additional gains from knowledge of  $K$ .

We devote Chapter 15 to a discussion of how we justify a model. However, we note already that in this example we have an instance in which a researcher (examining a case in study 3) might wish to draw inferences using  $K$ , but she does not have anything in study 1 that justifies using  $K$  for inference. However with access to studies 2 and 3, and conditional on the overall model, she has a justification for process tracing strategy. The general principle is that weaker commitments to lower level theories —here the causal structure—can justify more fully inferences from more fully specified higher-level theories.

## 11.2 Combining observational and experimental data

Experimental studies are often understood as the “gold standard” for causal inference. This is, in particular, because of the ability of a randomized trial (given certain assumptions, such as “no spillovers”) to eliminate sources of confounding. By design, an experiment removes from the situation processes that, in nature, would generate a correlation between selection into treatment and potential outcomes. An experiment thereby allows for an unbiased estimate of the average causal effect of the treatment on the outcome.

At the same time, an interesting weakness of experimental studies is that, by dealing so effectively with selection into treatment, they limit our ability to learn about selection and its implications in the real world. Often, however,

we want to know what causal effects would be specifically for units that *would* in fact take up a treatment in a real-world, non-experimental settings. This kind of problem is studied for example by Knox et al. (2019).

Consider, for instance, a policy that would make schooling subsidies available to parents, with the aim of improving educational outcomes for children. How would we know if the policy was effective? A source of confounding in an observational setting might be that those parents who apply for and take up the subsidy might also be those who are investing more in their children's education in other ways as compared to those parents who do not apply for the subsidy. To eliminate this problem, we might design an experiment in which parents are randomly assigned to receive (or not receive) the subsidy and compare outcomes between children in the treatment and control groups. With a no-spillovers assumption, we can extract the *ATE* of the receipt of subsidies.

What this experiment cannot tell us, however, is how much the policy will boost educational outcomes outside the experiment. That is because the causal quantity of interest, for answering that question, is *not* the *ATE*: it is the average treatment effect for the *treated* (*ATT*), given real-world selection effects. That is, the policymaker wants to know what the effect of the subsidy will be for the children of parents who *select into* treatment. One could imagine the real-world *ATT* being higher than the *ATE* if, for instance, those parents who are informed and interested enough to take up the subsidy also put the subsidy to more effective use. One could also imagine the *ATT* being lower than the *ATE* is, for instance, there are diminishing marginal returns to educational investments and the self-selecting parents are already investing quite a lot.

Even outside a policy context, we may be interested in the effect of a causal condition *where* that causal condition emerges. To return to our inequality and democracy example, we may want to know what would have happened to autocracies with low inequality *if* they had had high inequality – the standard average-treatment effect question. But we might also be interested in knowing how much of a difference high inequality makes *in the kinds of cases* where high inequality tends to be occur – where the effect could be very different.

With such questions, we are in a sort of bind. The experiment cannot tell us *who* would naturally select into treatment and what the effects would

be for them. Yet an observational study faces the challenge of ruling out confounding. Ideally, we would like to be able to combine the best features of both: use an experiment to deal with confounding and use observational data to learn about those whom nature assigns to treatment.

We can achieve this form of integration with a causal model. We do so by creating a model in which random assignment is nested within a broader set of assignment processes. We plot the model in Figure 11.1

At the substantive core of this model is the  $X \rightarrow Y$  relationship. However, we give  $X$  a parent that is fully exogenous,  $Z$ , to capture a random-assignment process. We give  $X$  a second parent,  $O$ , that is confounded with  $Y$ :  $O$  here represents the observational scenario. Finally, we include a “switch” variable,  $R$ , that determines whether  $X$  is randomly assigned or not. So when  $R = 1$ ,  $X$  is determined solely by  $Z$ , with  $X = Z$ . When  $R = 0$ , we are in an observational setting, and  $X$  is determined solely by the confounded  $O$ , with  $X = O$ .

A few notes on the parameter space. Parameters allow for complete confounding between  $O$  and  $Y$ , but  $Z$  and  $Y$  are unconfounded.  $X$  has only one causal type since its job is to operate as a conveyor belt, simply inheriting the value of  $Z$  or  $O$ , depending on  $R$ .

Note also that this model assumes the exclusion restriction that entering the experimental sample ( $R$ ) is not related to  $Y$  other than through assignment of  $X$ .

Now, let us imagine true parameter values such that  $X$  has a 0.2 average effect on  $Y$ . However, the effect is different for those who are selected into treatment in an observational setting: it is positive (0.6) for cases in which  $X = 1$  under observational assignment, but negative (-0.2) for cases in which  $X = 0$  under observational assignment. (See appendix for complete specification.)

When we use the model to analyze the data, we will start with flat priors on the causal types.

The implied true values for the estimands of interest, and our priors on those estimands, are displayed in Table 11.3.

Now, we generate data from the model, using the posited “true” parameter values, and then update the model using these data.

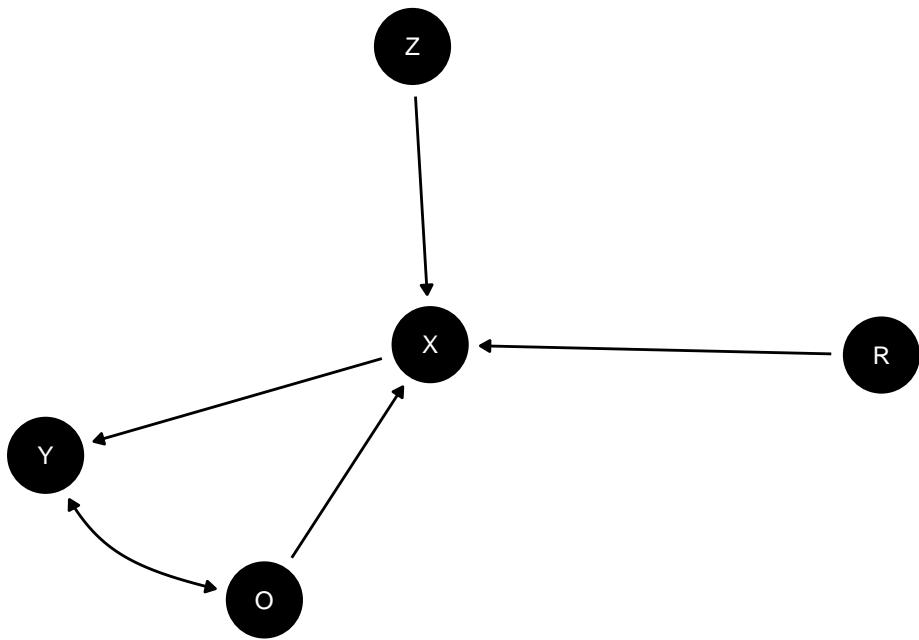


Figure 11.1: A model that nests an observational and an experimental study. The treatment  $X$  either takes on the observational value  $O$ , or the assigned values  $Z$ , depending on whether or not the case has been randomized,  $R$ .

Table 11.3: Estimands in different sites

Query	Given	Using	mean	sd
ATE	-	parameters	0.2	
ATE	-	priors	0.0	0.26
ATE	$R==0$	parameters	0.2	
ATE	$R==0$	priors	0.0	0.26
ATE	$R==1$	parameters	0.2	
ATE	$R==1$	priors	0.0	0.26

Table 11.4: Inferences on the ATE from differences in means

	Estimate	Std. Error	t value	Pr(> t )	CI Lower	CI Upper	DF
X	0.831	0.027	30.37	0	0.777	0.885	188

Table 11.5: Estimates on the ATE for observational ( $R = 0$ ) and experimental ( $R = 1$ ) set.

Query	Given	Using	Case.estimand	mean	sd
ATE	-	posteriors	FALSE	0.2028	0.0305
ATE	R==0	posteriors	FALSE	0.2028	0.0305
ATE	R==1	posteriors	FALSE	0.2028	0.0305

We begin by analyzing just the observational data (cases where  $R = 0$ ) and display the results in Table 11.4. Recall that the true average effect of  $X$  on  $Y$  is 0.2. Naive analysis of the observational data, taking a simple difference in means between the  $X = 0$  and  $X = 1$  cases, yields a strongly upwardly biased estimate of that effect, of 0.0806.

In contrast, when we use **CausalQueries** to update on the full causal model and use both the experimental and observational data, we get the much more accurate results shown in Table 11.5. Moving down the rows, we show here the estimate of the unconditional *ATE*, the estimate for the observational context ( $R = 0$ ), and the estimate for the experimental context ( $R = 1$ ). Unsurprisingly, the estimates are identical across all three settings since, in the model,  $R$  is  $d$ -separated from  $Y$  by  $X$ , which is observed. And, as we see, the posterior means are very close to the right answer of 0.2.

Since the model used both the experimental and the observational data, we might wonder from where the leverage derived: did the observational data improve our estimates of the average treatment effect, or do our inferences emerge strictly from the experimental data? In the appendix, we show results when we updating using experimental data only. Comparing the two sets of results, we find there that we do indeed get a tightening of posterior variance and a more accurate result when we use both the observational and experimental data, but the experimental data alone are quite powerful, as we should expect for an estimate of the *ATE*. The observational data do not add a great deal to an *ATE* estimate, and the gains from observational data

Table 11.6: Effects of  $X$  conditional on  $X$  for units that were randomly assigned or not. Effects of  $X$  do not depend on  $X$  in the experimental group, but they do in the observational group because of self selection.

Query	Given	Using	Case.estimand	mean	sd
ATE	$R == 1 \ \& \ X == 0$	posteriors	FALSE	0.2028	0.0305
ATE	$R == 1 \ \& \ X == 1$	posteriors	FALSE	0.2028	0.0305
ATE	$R == 0 \ \& \ X == 0$	posteriors	FALSE	-0.1831	0.0267
ATE	$R == 0 \ \& \ X == 1$	posteriors	FALSE	0.5932	0.0478

would be smaller still (and the experimental results even more accurate) if the experimental sample were larger.

However, what we can learn about uniquely from this model and the combined observational and experimental data is *heterogeneity* in effects between those that are in treatment and those that are in control *in the observational setting*. In Table 11.6, we display the results of *ATT* and *ATC* queries of the updated model. In the first two rows, we see that, in the experimental setting, the average effect of  $X$  on  $Y$  is the same on both the treated and control groups, exactly as we would expect under random assignment. In the third row, we see the estimate of  $X$ 's average effect for those assigned “by nature” to the control group in the observational setting, extracting a result close to the “true” value of  $-0.2$ . The final row shows our estimate of the treatment effect for those who are selected into treatment in the observational setting, again getting close to the answer implied by the underlying data-generating process (0.6).

We can learn nothing about the observational *ATT* or *ATC* from the experimental data alone, where there *ATT* and *ATC* are the same quantity. And in the observational data alone, we are hobbled by confounding of unknown direction and size. What the mixed model and data, in effect, are able to do is (a) learn about the *ATE* experimental data, (b) use inferences on the *ATE* to separate true effects from confounding in the observational data and thus learn about the direction and size of the confounding in those data, and (c) estimate the treatment effect for the  $X = 0$  group and for the  $X = 1$  group, respectively, in the observational data *using* knowledge about confounding in these data. By mixing the experimental and observational data, we can learn about how the treatment has affected those units that,

in the “real” world of the observational setting, selected into treatment *and* about how the treatment *would* affect those that selected into control.

The numbers in our toy example, while purely notional, can help us see why the observational  $ATT$  and  $ATC$  might be of great interest to decision makers where strong causal heterogeneity is a possibility. Based on the experimental data alone, we might conclude that the policy that makes  $X = 1$  available is a good bet, given its positive  $ATE$  (assuming, of course, that  $Y = 1$  is a valued outcome). And, of course, the observational data alone would not allow us to confidently conclude otherwise. What the integrated analysis reveals, however, is that  $X$  in fact has a *negative* mean effect on those who would be most likely to take up the treatment. The strong positive effect for the control strongly shapes the experimental results but will go unrealized in the real world. In a similar vein, these estimates can aid causal explanation. Seeing the positive  $ATE$  might lead us to infer that most of the  $X = 1, Y = 1$  cases we observe in the world are ones in which  $X = 1$  caused  $Y = 1$ . The observational  $ATT$  estimates point in a very different direction, however, indicating that these are cases in which  $X$  is least likely to have a positive effect and, thus, where  $Y = 1$  was most likely generated by some other cause.

We note that the results here relate to the LATE theorem (Angrist and Imbens, 1995). Imagine using data only on (a) the experimental group in control and (b) the observational group, some of whom are in treatment. We can conceptualize our design as one in which the observational group are “encouraged” to take up treatment, allowing us to estimate the effect for the “compliers” in the observational setting: those that self-select into treatment. Conversely, we could use data only on (a) the experimental group in treatment and (b) the observational group, some of whom are in control. This is a design in which the observational group are “encouraged” to take the control condition, allowing us to estimate the effect for the “compliers” in this group (those that self select into control).

## 11.3 Transportation of findings across contexts

In some circumstances, we study the effect of  $X$  on  $Y$  in one context (a country, region, or time period, for instance) and then want to make inferences about these effects in another context (say, another country, region, or time period). We may face the challenge that effects are heterogeneous, and that conditions that vary across contexts may be related to treatment assignment, to outcomes, and to selection into the sample. For example, we might study the relationship between inequality and democratization in low-income countries and then want to know how those effects travel to middle-income settings. However, the level of income may have implications jointly for the level of inequality and for how likely inequality is to generate regime change, meaning that causal effects uncovered in the first context cannot be assumed to operate in the second context.

This is the problem studied by Pearl and Bareinboim (2014). In particular, Pearl and Bareinboim (2014) show for which nodes data are needed in order to “licence” external claims, given a model.

We illustrate with a simple model in which an observable confounder has a different distribution across contexts. In the model drawn in Figure 11.2, *Context* determines the distribution of the confounder,  $W$ . We set a restriction such that the value of  $W$  in Context 1 is never less than the value of  $W$  in Context 0; our priors are otherwise flat over the remaining nodal types in the model.

We show priors and true values for the estimands (drawn from the parameters) in Table 11.7. We see that the incidence of  $W = 1$  is higher in Context 1 than in Context 0, both in our priors and in the “truth” posited by the assigned parameter values. The “true” *ATE* of  $X$  on  $Y$  is also higher in Context 1, though this is not reflected in our priors. The average treatment effect conditional on  $W$  is the same in both contexts, whether we work from priors or assigned parameter values, as it must be given the model. That is, in this model the *ATE* varies conditional on  $W$  — and it varies conditional *only* on  $W$ .

We now update the model using data from one context and then see if we can transport those findings to the other context. Specifically, we update using

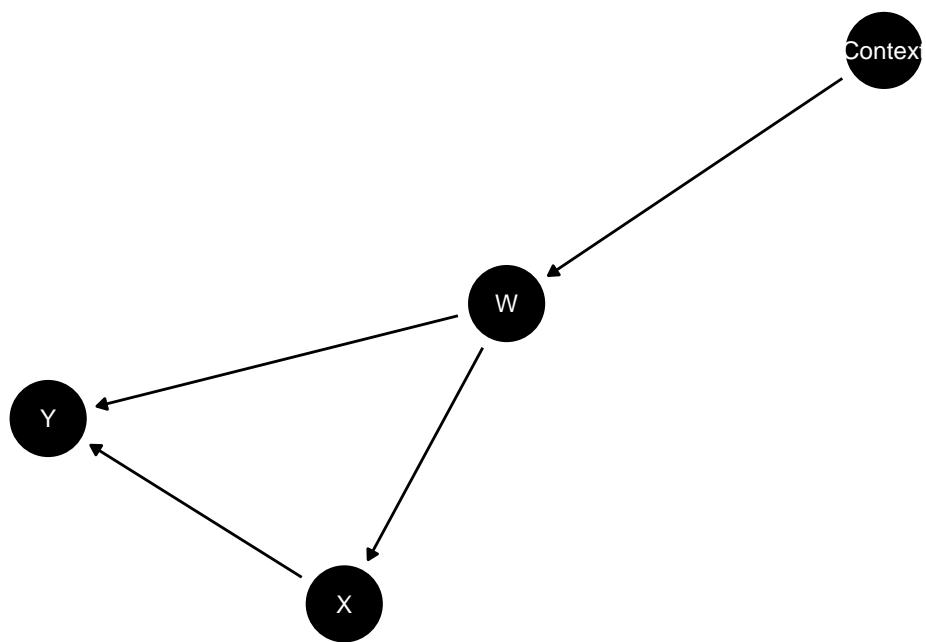


Figure 11.2: Extrapolation when confounders have different distributions across cases.

Table 11.7: Priors and true values (parameters) for three estimand: the frequency of  $W$ , the effect of  $X$  on  $Y$ , and the effect conditional on  $W = 1$

Query	Given	Using	mean	sd
Incidence	Case==0	priors	0.334	0.237
Incidence	Case==0	parameters	0.333	
Incidence	Case==1	priors	0.666	0.238
Incidence	Case==1	parameters	0.667	
ATE	Case==0	priors	0.002	0.140
ATE	Case==0	parameters	0.333	
ATE	Case==1	priors	0.003	0.142
ATE	Case==1	parameters	0.573	
CATE	Case==0	priors	0.002	0.174
CATE	Case==0	parameters	0.812	
CATE	Case==1	priors	0.002	0.174
CATE	Case==1	parameters	0.812	

data on  $X$ ,  $Y$ , and  $W$  from Context 0. We then use the updated beliefs to draw inferences about Context 1, using data *only* on  $W$  from Context 1. In Table 11.8, we show our posteriors on the queries of interest as compared to the truth, given the parameter values.

By comparing the *ATE* estimates using our posteriors and the estimates using the assigned parameter values, we see that we have done well in recovering the effects, *both* for the context we studied (i.e., in which we observed  $X$  and  $Y$ ) and for the context we did not study. We can think of the learning here as akin to post-stratification. We have learned from observing  $X$ ,  $Y$ , and  $W$  in Context 0 how  $X$ 's effect depends on  $W$ . Then we use those updated beliefs when confronted with a new value of  $W$  in Context 1 to form a belief about  $X$ 's effect in this second context. Of course, getting the right answer from this procedure depends, as always, on starting with the correct model.

We can also see, in Table 11.9, what would have happened if we had attempted to make the extrapolation to Context 1 without data on  $W$  in that context. We would get the wrong answer for Context 1, though we would also report greater posterior variance. The higher posterior variance here captures the fact that we know things could be different in Context 1, but we don't know in what way they are different.

Table 11.8: Extrapolation when two sites differ on  $W$  and  $W$  is observable in both contexts

Query	Given	Using	mean	sd
Incidence	Case==0	posteriors	0.336	0.007
Incidence	Case==0	parameters	0.333	
Incidence	Case==1	posteriors	0.661	0.007
Incidence	Case==1	parameters	0.667	
ATE	Case==0	posteriors	0.340	0.011
ATE	Case==0	parameters	0.333	
ATE	Case==1	posteriors	0.570	0.009
ATE	Case==1	parameters	0.573	
CATE	Case==0	posteriors	0.810	0.009
CATE	Case==0	parameters	0.812	
CATE	Case==1	posteriors	0.810	0.009
CATE	Case==1	parameters	0.812	

Table 11.9: Extrapolation when two contexts differ on  $W$  and  $W$  is not observable in target context

Query	Given	Using	mean	sd
Incidence	Case==0	posteriors	0.329	0.007
Incidence	Case==0	parameters	0.333	
Incidence	Case==1	posteriors	0.675	0.007
Incidence	Case==1	parameters	0.667	
ATE	Case==0	posteriors	0.319	0.011
ATE	Case==0	parameters	0.333	
ATE	Case==1	posteriors	0.572	0.009
ATE	Case==1	parameters	0.573	
CATE	Case==0	posteriors	0.811	0.009
CATE	Case==0	parameters	0.812	
CATE	Case==1	posteriors	0.811	0.009
CATE	Case==1	parameters	0.812	

## 11.4 Multilevel models, meta-analysis

A key idea in Bayesian meta-analysis is that when you analyze multiple studies together you learn not only about common processes that give rise to the different results seen in different sites, but you also learn more about each study from seeing the other studies.

A classic setup is provided in Gelman et al. (2013), in which we have access to estimates of effects and uncertainty in eight sites (schools),  $(b_j, se_j)_{j \in \{1, 2, \dots, 8\}}$ . To integrate learning across these studies we employ a “hierarchical model” that treats each  $b_j$  as a draw from distribution  $N(\beta_j, se_j)$  (and, in turn treats each  $\beta_j$  as a draw from distribution  $N(\beta, \sigma)$ ). In that setup we want to learn about the superpopulation parameters  $\beta, \sigma$ , but we also get to learn more about the study level effects  $(\beta_j)_{j \in \{1, 2, \dots, 8\}}$  by studying them jointly.

A hierarchical model like this allows us to think about the populations in our study sites as themselves drawn from a larger population (“superpopulation”) of settings. And, crucially, it allows us in turn to use data in the study sites to learn about that broader superpopulation of settings.

Although often used in the context of linear models with parameters for average causal effects, this logic works just as well with the kinds of causal models we have been using in this book.

Let’s review how our analytic setup has worked so far. At each node in a causal model, we conceptualize a given case as having a particular nodal type. The case’s nodal type is drawn from a distribution of nodal types in the population of cases from which this case has been drawn. When we do process tracing, we consider that population-level distribution to be a set of fixed shares of nodal types in the population: say, for node  $Y$ , we might believe that half the cases in the population are  $\lambda_{01}^Y$ , a quarter are  $\lambda_{00}^Y$ , and a quarter are  $\lambda_{11}^Y$ . We then use data from the case to update on the case’s nodal types (or on the combination of nodal types that correspond to some case-level query), given the population-level shares.

When we engage in population-level inference, we begin with *uncertainty* about the population-level shares of types, and we express our prior beliefs about those shares as a Dirichlet *distribution*. So, for instance, our beliefs might be centered around a  $\lambda_{01}^Y = 0.5, \lambda_{00}^Y = 0.25, \lambda_{11}^Y = 0.25$  breakdown of shares in the population; and we also express some degree of uncertainty about what the breakdown is. Now, when we analyze data on some number

of cases, we can update both on those cases' types and on our beliefs about the distribution of types in the population – perhaps shifting toward a higher share of  $\lambda_{01}^Y$ 's (and with a change in the distribution's variance).

We can also, as in the last section, build a model in which there are multiple settings, possibly differing on some population-level characteristic. Fundamentally, however, the setup in the last section still involved population-level inference in that we were assuming that the *type shares* ( $\lambda$  values) are the same across settings. The settings might differ in the value of a moderating variable, but they do not differ in the shares of cases that *would* respond in any given way to the moderator (and other causal conditions). The data allow us to update on what those common, cross-setting type proportions are.

When we build a hierarchical model, each case is still understood as being embedded within a population: our cases might be citizens, say, each embedded within a country. The key difference from population-level inference is that we now conceive of there being *multiple* populations – say, multiple countries – each drawn from a population of populations, or superpopulation. Now, we think of each population (country) as having its own set of type shares for each node. And we think of each country's type shares as being drawn from a Dirichlet distribution of type shares (for each node) that lives at the superpopulation level. Moreover, we are *uncertain* about what that distribution at the superpopulation level *is*. We uncertain around what type proportions the superpopulation-level distribution is centered, and we are uncertain about how dispersed this distribution is. While the distribution's central tendency will be related to the mean type shares for countries, its variance will determine the degree of *heterogeneity* across countries in their type shares.

To summarize, in population-level inference, we express uncertainty about the population's type shares with a Dirichlet prior, at the population level, on which we update. In the hierarchical setting, we are uncertain both about the population-level type shares and the superpopulation Dirichlet from which each node's type shares are drawn. We express our uncertainty about each superpopulation Dirichlet by positing a prior distribution over the Dirichlet's alpha parameters.

Now, when we observe data on citizens within countries, we can update our beliefs about types for the particular citizens we observe, about type shares

in the population of citizens within each country that we study, *and* on the parameters of the Dirichlet distribution from which population shares have been drawn. In updating on the last of these, we are learning not just about the countries we observe but also about those we do not directly observe.

We illustrate with a simulation using a simple  $X, Y$  model. We imagine that we are studying the  $X \rightarrow Y$  relationship in  $n$  countries. Each country has a parameter distribution drawn from common Dirichelets. We start off with flat priors over the alpha arguments of the superpopulation Dirichlets.

We assign a particular true set of superpopulation parameter values that, for the analytic exercise, is treated as unknown and that we would like to recover. In this true world, the probability of assignment to  $X = 1$  is .4, and the average treatment effect is .1. Using these true parameter values, we simulate  $X, Y$  data for  $n = 8$  countries.

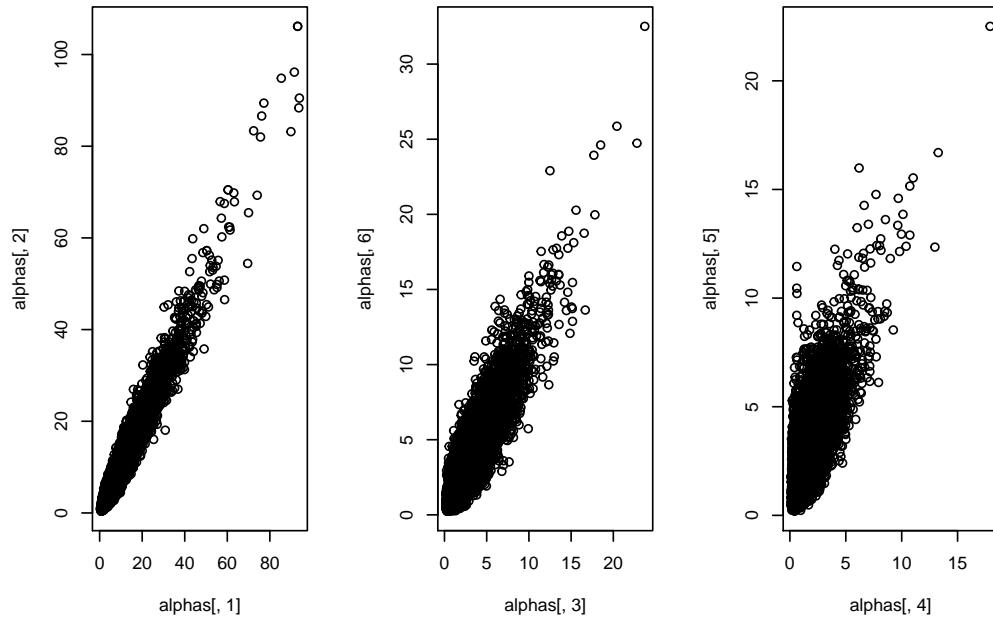


Figure 11.3: Joint distributions over alpha parameters.

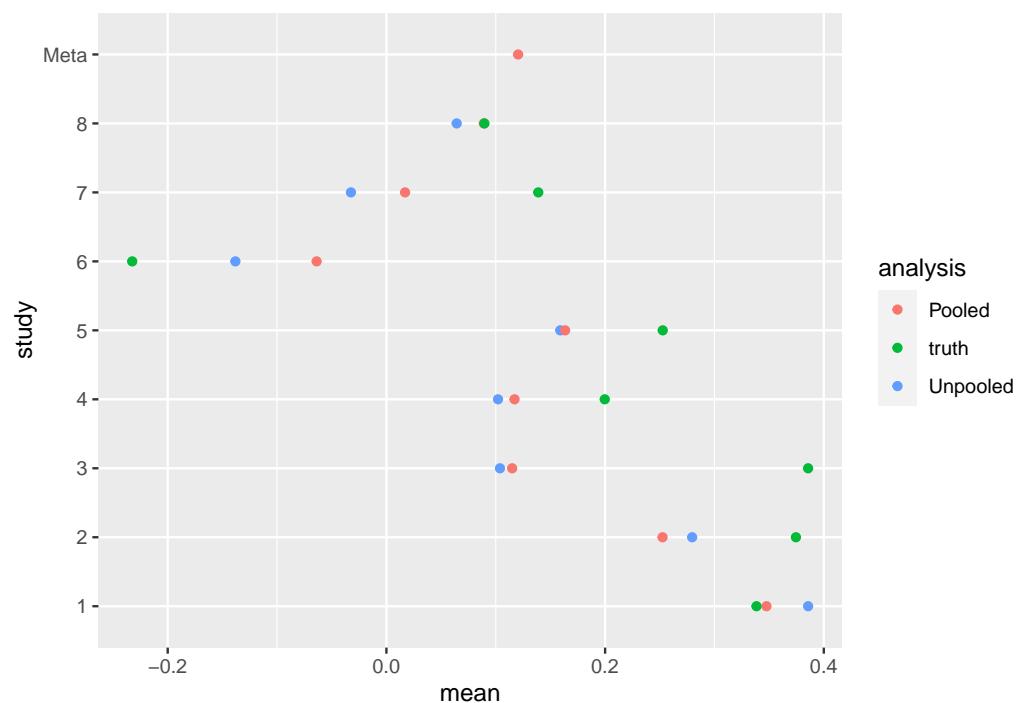
In Figure 11.3, we graph our posterior beliefs about the superpopulation parameters. We do this by plotting two alpha parameters against each other

at a time. In the first panel, we plot the alphas for  $X = 0$  and  $X = 1$ . In the next panel, we plot the alpha's corresponding to  $c$  types against those corresponding to  $d$  types. And in the third panel we plot the alpha's corresponding to  $a$  types against those corresponding to  $b$  types.

As we can see, each distribution falls roughly along a diagonal. Probability mass located further up the diagonal represents worlds in which the super-population Dirichlet distribution of type shares is relatively low in variance. Thus, the more that our posterior beliefs are concentrated toward a graph's northeast corner, the lower the heterogeneity we have inferred there to be in the relevant type shares across countries. Meanwhile, the dispersion of probability mass *away* from the diagonal represents greater posterior *uncertainty* about the heterogeneity across countries, arising from greater variance about the posterior distribution of the alphas.

We can think of a concentration parameter here that is operationalized as the sum of the  $\alpha^j$  terms for a node,  $j$ , with a higher value representing lower overall heterogeneity.

In Figure 11.4 we turn to the causal query of interest and show a comparison of three *ATE* estimates for each country: in blue, we show the unpooled estimate, or the estimate we get for each country using only data from that country; in red, we see the pooled estimates, or the estimate we get for each country using data from *all* countries to inform that country's parameter estimates; and in black, we plot the truth as posited for this simulation. As we can see, the pooled estimates are all closer to the center than the unpooled estimates: this is because we are effectively using data from all countries to discount extreme features of the data observed in a given country. Put differently, the pooled data serve the function of a prior when it comes to drawing inferences about a single country: our inference is a compromise between the data from that country and the beliefs we have formed from the pooled data. We can also see that, for most countries, the pooling helps: the regularization provided by the pooling gives us an estimate closer to the truth for most of the settings.





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