Causal Graphs in Choice Modelling: The What, Why, and How

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Abstract

In this chapter, we focus on using causal graphs to make causal inferences in choice modelling contexts. We address a longstanding disconnect whereby choice modellers have not adopted techniques from causal inference researchers, even when trying to make causal inferences. To bridge this disconnect, we conduct simulation studies to first demonstrate the need for paying close attention to causal graphs in choice modelling. We then present new guidelines, methods, and perspectives for the construction and validation of causal graphs, complete with empirical examples. Next we give examples and direction for using one's causal graphs to make causal inferences in common choice modelling scenarios, including those with latent confounding. Finally, we also provide extensive literature reviews of testing and discovery methods for causal graphs. At the chapter's conclusion, choice modellers should have a much clearer understanding of (or references to find out about) why they should use causal graphs, what graphs to use for their dataset, and how they can use a given causal graph to complete their causal inferences.

1 Introduction

In transportation, we often build models for policy evaluation. Specifically, we often use behavioral models to evaluate the impact of external interventions on travel outcomes. These evaluations are explicitly causal: we are interested in how the system reacts to *interventions*. Yet, when these models are developed, causality is often not considered. Furthermore, when causal concepts are accounted for, the process is done implicitly without a formal framework.

This chapter aims to fill this gap. Specifically, we focus on addressing the existing disconnect between the fields of travel demand modelling and causal inference. The chapter is motivated by the current lack of use of methods and findings from the causal inference literature in travel demand modelling and choice modelling more broadly.

While the field of transportation demand modelling could benefit greatly from incorporating causal inference techniques, there are barriers that have slowed this integration. These barriers stem from the difference between the types of problems transportation demand modellers deal with and those that are typically studied in the causal inference literature. Perhaps the fundamental difference is that demand modellers are typically trying to forecast the impacts of policies that haven't been implemented or seen before. Forecasting the effects of unseen interventions requires additional work and a change to the typical causal modelling workflow. In particular, we must translate a given policy (treatment) into a set of characteristics and variables that exist in the data and system at hand. (For a more thorough discussion of this and other barriers, please refer to Brathwaite and Walker (2018a).)

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While these barriers complicate the efforts of demand modellers, there is still a lot to gain from incorporating causal inference techniques where appropriate and from contributing to the causal inference literature where it's lacking.

This topic is even more relevant now because of the significant and recent boost in the causal inference literature, both in the potential outcomes and the causal graphical modelling frameworks. Leveraging these latest advances, the goal of this chapter is to empirically illustrate a workflow for approaching transportation demand modelling problems from a causal perspective. We will draw heavily on the use of directed acyclic graphs (DAGs) formalized by Pearl (2000) as a means of representing the modeller's knowledge and assumptions about a given problem. The chapter will provide an overview of DAGs, the consequences of ignoring them when making causal inferences, and their testable assumptions/implications. We then present convenient methods of testing those assumptions, and we show how to transition from isolated tests to complete inference of causal graphs from existing data. Throughout, we demonstrate our graphical framework through simulations and empirical examples. These examples highlight the benefits of our proposed approach versus traditional methods that eschew causal inference techniques, and they provide guidance to researchers and practitioners that wish to use such methods in their own work.

The last part of this chapter deals with the more complicated issue of latent confounding, where an unobserved variable confounds two or more variables in the causal graph. Confounding creates variations in the outcome variable that correlate with but are not caused by the treatment variables. These spurious correlations bias the estimated causal effects if nothing is done to account for them. In Section 7, we focus on a recent technique by Wang and Blei (2019) for addressing unobserved confounding when collecting additional data is not feasible. Finally, in Section 8, we briefly discuss the many subsequent and important steps that are necessary for making and benefiting from one's causal inferences.

2 Perils of disregard

As stated in Section 1, transportation demand models are used to evaluate the impact of policies on a certain transportation demand related outcome. As an example, consider the proposals from the following fictitious scenarios:

- Based on input from the public, the Department of Transportation (DOT) is considering implementing a new parking policy. This proposed policy would change prices and restrict availability to discourage individuals from parking in the central business district. The DOT and its constituents believe that this policy will encourage people to use more active transportation modes (e.g., walking, biking, etc.) and cause them to drive less.

- A certain DOT is considering constructing a streetcar (trolley, tram) line in a low/mid-income area in its jurisdiction. Citing examples from other cities and countries, the DOT claims that the new streetcar line will create more transit oriented developments and increase economic activity in the areas surrounding the proposed project.

Thinking more closely about these proposals, it is clear that they assume a causal relationship between the proposed project/policy and the desired goals. The DOT in question analyzed data, concluding that such a policy or project would cause the desired output and achieve the desired goal. In the presented scenarios, the DOT claims that implementing the new parking policy will cause an increase the share of active transportation modes in the central business district and that constructing the proposed streetcar line will cause more transit oriented developments and economic activity.

Policymakers base their analyses and conclusions on hypotheses or beliefs of how the world operates. In other words, the data analysis is based on specific beliefs about the data generating process. However, policymakers often do not present these beliefs in a clear and concise manner. As a result, these proposals maintain an obscure representation of how the policy or project will achieve their desired goals.

DAGs allow one to clearly encode their assumptions about the data generating process and the problem at hand. Researchers and practitioners have made use of DAGs in fields ranging from medicine and epidemiology (Shrier and Platt (2008); Sung (2012)) to economics (White and Lu (2011)) and have found them to be practical.

Likewise, DAGs could prove useful in addressing transportation policy questions. Brathwaite and Walker (2018a) have proposed a framework illustrating how practitioners and researchers can use DAGs to answer such transportation modelling questions in a causal context. However, Brathwaite and Walker (2018a) did not show an empirical application of their framework and how it results in different conclusions when compared to traditional modelling approaches.

In this section, we will present an example illustrating the importance of using DAGs in transportation demand modelling. Specifically, we will illustrate how different assumptions about the data generating process result in different conclusions, even while assuming the same outcome model. To make our point, we build upon Brathwaite and Walker (2018a). Here, we present an empirical exercise using a simplified transportation modelling problem. Before going any further, we note that this example is illustrative, and it does not reflect all complexities in a typical transportation choice modelling problem. Indeed, it is not our primary goal in this section to recover the causal effect of the proposed intervention. Instead, we are most interested in showing how different DAGs would result in different conclusions about the effect of the proposed intervention.

Let us assume that a company wants to reduce its workforce carbon footprint by moving its employees closer to their campus. We would like to forecast how such an intervention would change the share of employees driving to work. We model this travel mode choice problem based on a dataset from Brathwaite and Walker (2018b). This dataset is based on the 2012 California Household Travel Survey, and it contains approximately 4000 home-based school or work tours made by approximately 3850 individuals in the California Bay area. The dataset includes eight travel modes. For our illustrative purposes, we focus on the following car-centric modes:

- Drive Alone: The individual uses a private vehicle to make the trip
- Shared Ride 2: The individual shares an automobile ride with one more individual
- Shared Ride 3+: The individual shares an automobile ride with two or more individuals

Readers interested in a more detailed description of the dataset can refer to Brathwaite and Walker (2018b). For the purposes of this exercise, we treat the multinomial logit model (MNL) defined in Brathwaite and Walker (2018b) as the true outcome generating model.

In this model, the systematic utility equations of the car-centric modes defined above are specified as follows:

$$\begin{aligned}\text{Utility (Drive Alone)} = {} & \beta_{\text{travel\_time}} \times \text{Travel\_Time} + \beta_{\text{cost\_per\_distance\_drive\_alone}} \times \text{Cost\_per\_Distance}_{\text{da}} \\ & + \beta_{\text{autos}} \times \text{Number\_of\_Autos}\end{aligned} \tag{1}$$

$$\begin{aligned}\text{Utility (Shared Ride 2)} = {} & ASC_{\text{shared\_ride\_2}} + \beta_{\text{time\_drive}} \times \text{Travel\_Time} \\ & + \beta_{\text{cost\_per\_distance\_shared\_ride\_2}} \times \text{Cost\_per\_Distance}_{\text{sr2}} + \beta_{\text{autos}} \times \text{Number\_of\_Autos} \\ & + \beta_{\text{cross\_bay}} \times \text{Cross\_Bay} + \beta_{\text{hh\_size}} \times \text{Household\_Size} \\ & + \beta_{\text{n\_kids\_hh}} \times \text{Number\_of\_kids}\end{aligned} \tag{2}$$

$$\begin{aligned}\text{Utility (Shared Ride 3+)} = {} & ASC_{\text{sr3+}} + \beta_{\text{time\_drive}} \times \text{Travel\_Time} \\ & + \beta_{\text{cost\_per\_distance\_sr3+}} \times \text{Cost\_per\_Distance}_{\text{sr3+}} + \beta_{\text{autos}} \times \text{Number\_of\_Autos} \\ & + \beta_{\text{cross\_bay}} \times \text{Cross\_Bay} + \beta_{\text{hh\_size}} \times \text{Household\_Size} \\ & + \beta_{\text{n\_kids\_hh}} \times \text{Number\_of\_kids}\end{aligned} \tag{3}$$

Note that, since we consider this model to be the true outcome model, there are no latent variables we need to account for. Below is a description of the key variables included in the model:

- Total Travel Distance: the total travel distance for individual i and mode j, for all available modes for individual i during trip t of tour l.

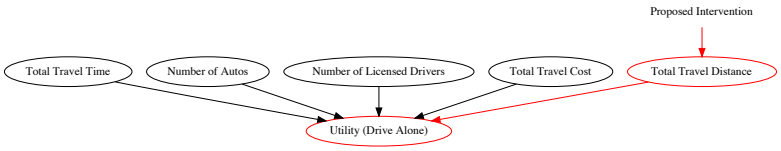

Figure 1: Causal Graph with Independent Covariates

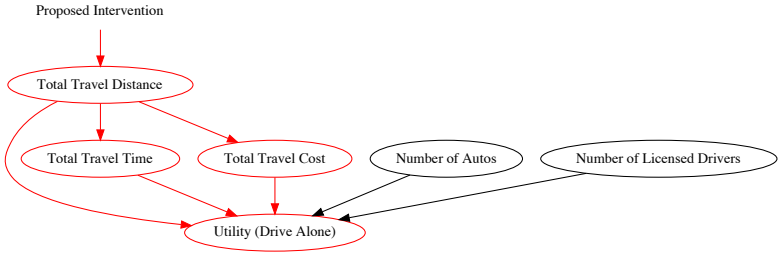

Figure 2: Causal Graph for the Drive Alone Utility Function

- Total Travel Cost: the travel cost in dollars for individual i and mode j, for all available modes for individual i during trip t of tour l.
- Total travel time: the travel time in minutes for individual i and mode j, for all available modes for individual i during trip t tour l.
- Number of Autos: the number of automobiles owned by individual i's household.
- Number of Licensed Drivers: is the number of licensed drives in individual i's household.
- Number of Kids: the number of kids in individual i's household.
- Cross-bay trip: a binary variable indicating whether the trip t in tour l for individual i is a cross-bay trip.

Figure 1 illustrates the DAG where all explanatory variables in each utility equation are marginally independent. This DAG is equivalent to what an analyst assumes when they only update the variables directly impacted by a given policy, without considering the dependencies between the explanatory variables. To illustrate the problem with this approach, consider the case where the "true" data generating process that reflects the dependencies between the covariates is as shown in Figures 2 through 4. Under this generative model, intervening on one variable would also result in changes to other variables that are dependent on it. The goal of our simulation exercise is to show that ignoring the true generative model of the data (Figures 2 through 4) can result in arbitrarily biased treatment effects, even if the analyst knows the true outcome model (Equations 1 through **??**).

To achieve this goal, we follow these simulation steps, using the same outcome model defined in Equations 1 to **??**:

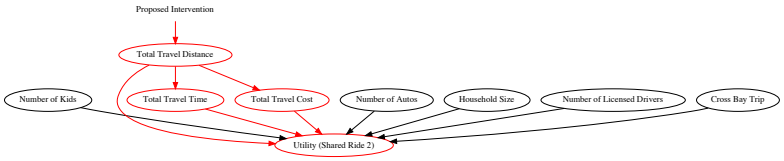

Figure 3: Causal Graph for the Shared Ride 2 Utility Function

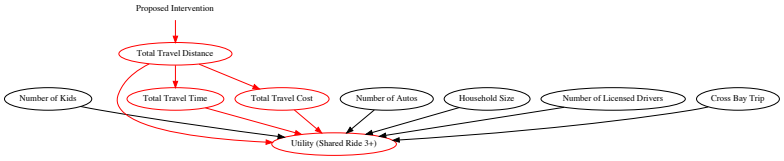

Figure 4: Causal Graph for the Shared Ride 3+ Utility Function

- Simulate data from the DAGs shown in Figure 2 through Figure 4.
- In one scenario, only modify the travel distance variable in all graphs to emulate a company's decision to move its employees closer to campus. This is similar to assuming the DAG in Figure 1 as the true data generating model.
- In the other scenario, modify the travel distance variable in all utility graphs, as well as all the variables that are dependent on it based on the DAGs in Figure 2 through Figure 4. This case is meant to reflect the "correct" approach needed to quantify the effects of the company's policy.
- Predict the probabilities of choosing car-centric modes before and after modifying the data to emulate the proposed intervention under each of the two scenarios above, and compute the differences in mode choice probabilities.

Readers interested in exploring the details of our simulation exercise can refer to our GitHub repository (Brathwaite et al., 2020).

We then plot histograms of the computed differences between the average probability of an individual in our sample choosing a car centric mode before and after implementing a policy or intervention aimed at reducing travel distance. Recall that these differences are plotted under the assumptions that the outcome model is the same in both scenarios, and the only difference between the two scenarios is the set of variables assumed to be affected by the proposed intervention. This difference in the set of variables affected by the proposed intervention is a result of the two different causal graphs representing each scenario (Figures 1 - 4). Figure 5 highlights the resulting bias between the estimated probability of an average individual choosing a car centric mode. The histograms show that the distribution of inferred treatment effects based on the "true" causal graph includes both positive and negative treatment effects. With low but non-trivial probability, using the wrong causal graph in one's analyses could result in not only wrong magnitudes of the treatment effect of interest but also the wrong sign. This plotted discrepancy shows the importance of using causal graphs in the estimation of treatment effects. Of course, the importance of a correct causal graph does not and should not take away from the importance of correctly specified outcome choice models.

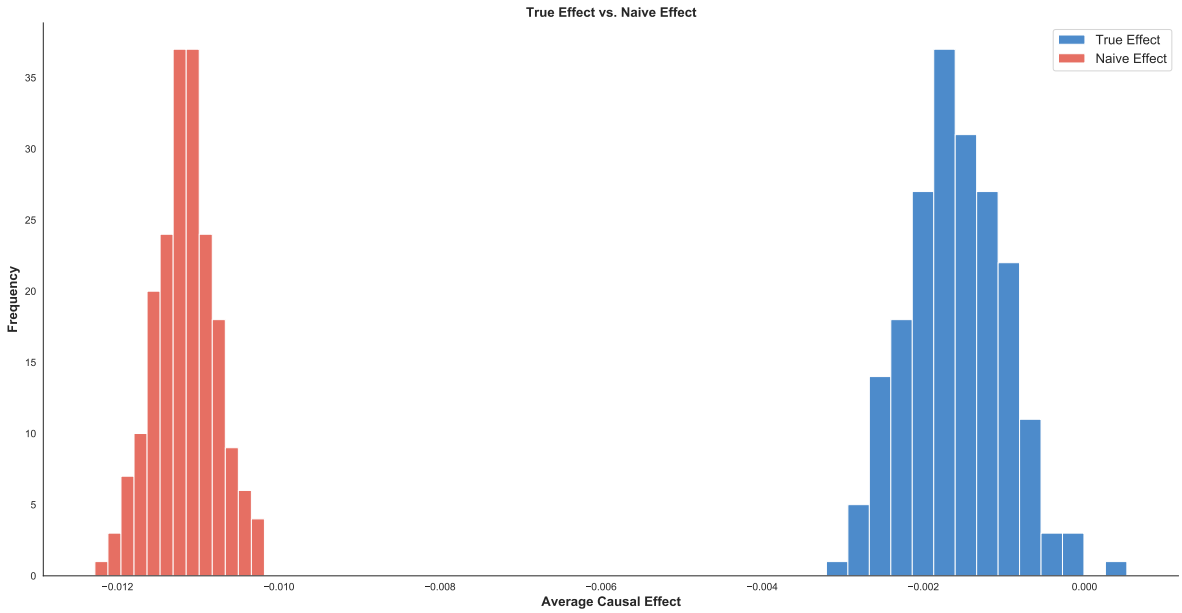

Figure 5: Histograms of the probability of choosing Car Centric Modes under Different Data Generating processes.

We have shown in this simulation exercise how causal graphs can be helpful in avoiding biased causal effect inferences. Specifically, two situations highlight this fact; having mediating or confounding variables relative to the variable being intervened on in our outcome model. In the case of mediating variables relative to the node the treatment intervenes on, we need to be careful about constructing our treatment effect estimator. In particular, we need to ensure that we appropriately use total effect estimators instead of natural direct effect estimators. Enacting a certain treatment shows its effect through the mediating variable. Therefore, when "changing" the value of the intervention node, we need to make sure the values and distributions of any downstream nodes reflect this change. For more information on mediating mechanisms, please refer to work by Pearl (2012) and references therein. In the case of confounding variables, we need to consider the treatment assignment mechanism when building choice models. As (Hahn et al., 2020) show, one way to account for the treatment assignment mechanism is to estimate the propensity score in our sample and adjust for that in our outcome model.

In contrast to the example shown in this section, the data generating process might not be easily distinguishable in the majority of situations, mainly due to the complexity of the real world. Therefore, constructing a causal graph that represents the data generating process as much as possible is not an easy task. To prepare readers to create causal graphs, the next section will provide a brief overview of them and their history in choice modelling. Then, Section 4 explores this topic and includes detailed guidance on how to build causal graphs representing the researchers beliefs about the data generating process. Section 5 follows up with guidance on how to test one's causal graphs against one's data.

3 Overview of Causal Graphs

The previous section, 2, motivated why the knowledge encoded in one's DAG is important. This section gives a high level overview of what DAGs are and provides references for more thorough readings on the subject. Causal diagrams and causal graphical models have been introduced by Pearl (1995) as a powerful tool for causal inference, especially in observational studies. Perhaps one of the most important and useful features of causal graphs when dealing with causal inference problems is the clear illustration of the causal relationships between the variables. While a formal introduction to the topic of DAGs is beyond the scope of this chapter, here we focus specifically on illustrating the power of DAGs to represent and encode complex causal relationships between variables in an intuitive and clear manner. Interested readers can refer to Pearl

(2000) for a thorough introduction.

Consider the causal graph represented in Figure 6. Suppose we're interested in the effect of a treatment Z on Y. What the graph in Figure 6 implies is that Z is independent of Y(Z) given X. For more information on how to semantically parse a causal graph as just done, see Pearl (2009, Ch 11.1). Continuing with interpretation, in causal jargon, we say the mechanism by which the treatment Z was assigned is ignorable, once we control for covariates X. In such situations, it is sufficient to control for X to obtain an unbiased estimate of the causal effect of Z on Y.

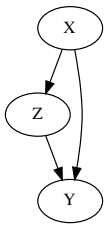

Figure 6: Simple DAG of the causal relationships between X, Y and Z

The graph in Figure 6 can also be summarized by the following set of structural equations:

$$Z = f_Z(X, \epsilon_Z)$$

$$Y(z) = f_Y(X, z, \epsilon_Y)$$

Now consider the case where there exists another latent confounding variable, U, which also affects both the treatment Z, as well as the outcome, Y. Figure 7 illustrates this assumption in DAG form, and the equations below are the structural equation equivalent:

$$Z = f_Z(X, U, \epsilon_Z)$$

$$Y(z) = f_Y(X, U, z, \epsilon_Y)$$

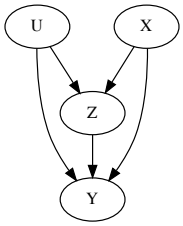

Figure 7: Simple DAG of the causal relationships between X, Y, Z and confounder U

Figure 7 shows an indirect connection between Z and Y that goes through U. Therefore, even if we condition on X, omitting U will yield biased results of the relationship between Z and Y, due to the variation in both Z and Y caused by U. The structural equations also show that the ignorability assumption of Z does not hold if we only control for X, and we risk obtaining biased estimates of the causal effect of Z on Y if we fail to account for U, sometimes referred to as a common cause. While the set of structural equations and the DAGs illustrate the same points in theory, the clarity of structural equations scales poorly with a problem's

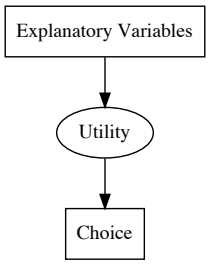

Figure 8: Archetypical RUM causal diagram

complexity. As the number of covariates in one's causal system increases, a graphical representation of the relationship between those covariates quickly becomes more intuitive and easier to follow. More importantly, graphically illustrating our assumptions eases the communication of these assumptions to the reader. This communication is key to avoiding the misuse of our models in real-world applications. For example, consider travel demand modelling applications. Here, the communication advantage of DAGs is of particular relevance, as travel demand models typically involve many variables with complex interdependencies.

Another benefit of DAGs is that they come with testable implications. Incorporating these tests in any causal analysis adds robustness and defensibility to one's study. In section 5, we discuss these implications further, and we illustrate how to tests for them in one's analysis.

Lastly, it is important to note that the graphical approach to causality focuses primarily on issues of identification of causal effects, as opposed to issues of estimation and inference. That is, given a DAG that encodes an analyst's knowledge and assumptions about the data generation process of the problem at hand, one can determine algorithmically whether a causal effect of interest can be identified. As such, we emphasize that DAGs are great tools for a modeller to encode their assumptions about a problem. Later on, we will use more familiar statistical tools to actually infer/estimate our causal effects of interest.

3.1 Prior Uses of Causal Graphs in Choice Modelling

In our last subsection, we reviewed the basics of causal graphs: what are they and why are they useful? We targeted that subsection at choice modellers who are unfamiliar with these tools. However, readers should be aware of the history of causal graphs in choice modelling. Indeed, choice modellers have used causal graphs (in limited fashion) for years. Here are three examples to illustrate our point. First, consider the case of Random Utility Maximization (RUM) models.

For decades, choice modellers have used stylized DAGs to depict RUM models. In particular, these diagrams illustrate an assumed choice-making (i.e., causal) process. As an example, see Figure 8, reproduced from Figure 1 of Ben-Akiva et al. (2002). Ben-Akiva et al. draw a two-part process. First, they assume that explanatory variables cause unobserved utilities. Then, they assume that the unobserved utilities cause the choice.

Note that although RUM diagrams adequately show the choice process, we still call them stylized. Specifically, these diagrams lack detail about the relationships between explanatory variables. When speaking collectively, we cannot tell if one explanatory variable causes another. Unfortunately, as shown in Section 2, such knowledge is crucial. Without more detailed causal knowledge, our inferences may be inconsistent and arbitrarily bad.

Besides RUM models, causal graphs appear in the literature on Integrated Choice and Latent Variable (ICLV) models. An example of an ICLV model is in Figure 9, based on Figure 5 of Ben-Akiva et al. (2002). As with RUM models, ICLV causal graphs represent assumptions about the choice process. Here concern typically centers around unobserved mediators. These unobserved variables cause the outcome and are caused by one's observed covariates. Omitting such mediators leads to models that misrepresent the assumed choice process. As a result, researchers care deeply about ICLV models that avoid these behavioral

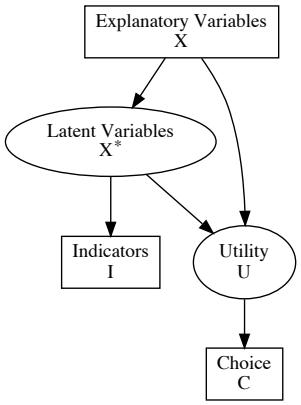

Figure 9: Archetypical ICLV causal diagram

misrepresentations.

Finally, consider activity based travel demand models (ABM). These models often come with a causal diagram that depicts the interrelations between the outcomes in the model. E.g., household location choice, destination choice, travel mode choice, departure time choice, route choice etc. For example, see Figure 10, from Bradley et al. (2010, Fig. 2). The purpose of these graphs is to explain the structure of the entire system of outcome models.

In particular, ABM diagrams highlight two sets of researcher assumptions. They detail the researcher's beliefs about which choices, i.e. outcomes, precede others. For instance, work location choice preceding auto-ownership choice. ABM diagrams also detail which downstream choices partially cause which preceding ones. For example, considerations about one's travel mode choice influences one's daily activity choices. This happens even though the activity choices precede the travel mode choices.

Two main features distinguish RUM, ICLV, and ABM graphs from the causal graphs of Pearl (1995). First, causal graphs in choice modelling traditionally ignore relations between the explanatory variables. Typically, choice modelling causal graphs show all explanatory variables together as a monolith. The relationships between explanatory variables is often not specified. Even worse, researchers may tacitly treat the variables as if they are jointly independent. In the language used by a large group of causal inference scholars, econometric causal diagrams ignore the treatment assignment mechanism.

Secondly, causal graphs in choice modelling papers are purely didactic. They convey how choice modellers perceive the world and the choice generation process. However, they are seldom treated as a model themselves. In particular, choice modellers rarely test the predictions of causal graphs. In doing so, we underuse causal implications such as statistical independence. Worse, we possibly violate these implications with great frequency. Such actions ignore the efforts from causal inference in computer science. There, researchers stress using their data to test the implications of their causal graphs.

In conclusion, choice modellers have long made use of causal graphs. In select contexts, causal graphs convey causal assumptions about choice processes. Thus far, however, our field has underutilized these tools. We seldom use causal graphs to encode our assumptions about how our explanatory variables came to be. Moreover, we do not routinely test causal graphs against empirical choice data.

These two issues are opportunities for choice modelling to gain from the insights and methods of causal inference scholars. The rest of the chapter will focus on the following three topics. How we can construct causal diagrams that pay attention to explanatory and outcome variables? How we can test a given causal graph against one's data? How can we deal with "real-world" graphs featuring unobserved confounding?

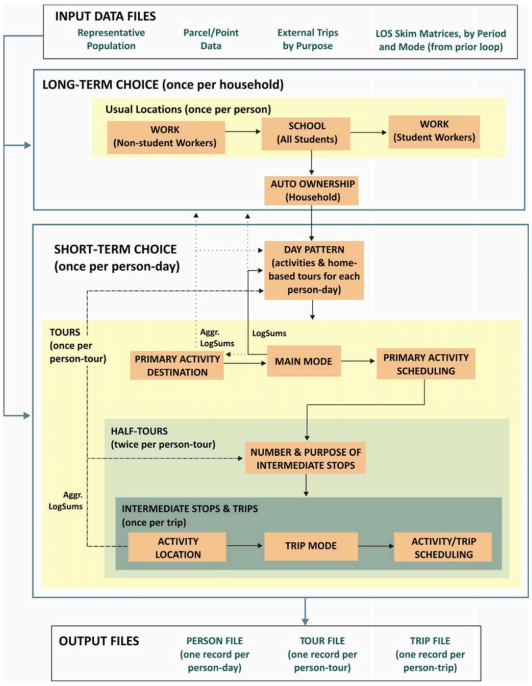

Figure 10: Archetypical causal diagram for activity-based models

4 (Initial) Causal Graph Construction

Construction of an initial causal graph typically proceeds as follows. At a high level, we

1. adopt a population perspective,
2. brainstorm all variables that we think affect the system that generates our observations,
3. remove any variables that could cause bias in our causal inferences,
4. connect all variables in our graph according to our a-priori beliefs about causal relations amongst them
5. consider how the graph structure may differ between individuals and subgroups within the population.

The following paragraphs describe these steps in detail.

To begin, we adopt the position of a researcher concerned about population level relationships. This means we will think through what is a likely generative model for all individuals. Later in this section, we will devote time to thinking about how subgroups and individual heterogeneity may affect our causal graphs.

Now, we add our first variable(s) to our graph, the outcome variable(s) of interest in our problem. Note that we should consider relations and dependencies between these outcomes, and we should draw these onto our graphs. Such inter-outcome dependencies may be of great relevance or even focus. Recall, for example, the case of activity-based modellers that was mentioned in Section 3.1. Similarly, medical researchers with data on multiple health measures or companies with multiple business metrics may all be interested in how the outcomes cause each other.

After adding our outcomes to our graph, we list all the variables we believe to cause them. We refer to these influencing variables as our initial explanatory variables.

Next, we iterate through these initial explanatory variables. For each current explanatory variable in the iteration, we think of variables that may modify the effect of the current explanatory variable on the outcome(s) of interest. We refer to these variables as effect modifiers[1]. Note that some effect modifiers may be a part of our list of initial explanatory variables. For any effect modifiers that we think of, outside of the list of initial explanatory variables, we add them to our causal graph.

Overall, modifiers are important because our treatment effects systematically vary with them. Accordingly, if we better understand when our treatments will be effective, then we can better target them. For instance, imagine that a region-wide lockdown reduces the 14-day rolling average of new COVID-19 cases by X% (on average). Of course, we know that a lockdown's effectiveness is modified by the percentage of workers who must continue going out to work. If most residents in an area are essential workers, then a lockdown will be less effective there, as compared with other locales. We might wish to target other interventions for that region, as a replacement or supplement for the lockdown. Targeting aside, knowledge of modifiers is also crucial to generalizing treatment effect inferences from one population to another. To credibly transport our inferences, we must know what variables cause the treatment effects to differ between populations, and we must know how the distributions of those variables differs across populations (Pearl and Bareinboim, 2014). In general, see Zheng et al. (2018) for a thorough introduction to moderation, its differences from other variables in one's causal graph, and for instructions on how to find moderators through data analysis alone.

After adding explanatory and effect modifying variables to the graph, we turn our attention to mediating variables. A mediating variable is one through which an explanatory variable influences our outcome(s) of interest. Such variables have multiple uses. Under certain instances of confounding, mediators enable the "front-door" criterion to identify one's causal effect (Glynn and Kashin, 2018; Bellemare and Bloem, 2019; Gupta et al., 2020). Similarly, subject to particular causal assumptions, mediating variables permit inference on long-term outcomes of a selected intervention, given only its short-term proxies (Athey et al., 2019; Yang et al., 2020).

To find these mediators, we again iterate through each explanatory variable. On each iteration, we brainstorm variables along paths of influence from our explanatory variable to our outcome. For instance,

[1]Note, effect modifiers and confounders are easily confused. Both variables cause the outcome. The difference is that effect modifiers do not cause the explanatory / treatment variables. Confounders do. For discussion and classification of the different types of effect modification, based on one's causal graph, see VanderWeele and Robins (2007)

consider how the presence of a bike lane influences bicycle mode choice. We hypothesize that an individual's subjective perception of safety is the primary (or sole) mediator through which bicycle lane presence influences mode choice. Accordingly, we add subjective perception of safety to our causal graph for travel mode choice.

After considering the variables above, we turn our attention to variables that complicate our analyses. To begin with, we think of confounding variables. The process is similar to how we generated effect modifying variables. We iterate through each of the explanatory, mediating, and effect modifying variables, thinking specifically of any variables that both cause the current variable in the iteration and cause the outcome variable(s). We call these variables, which cause our outcome and current variables in the iteration, confounding variables (Elwert, 2013; Greenland et al., 1999). As an example, consider a person's attitude towards environmental conservation. This attitude may cause both that individual's observed distance to their workplace (another explanatory variable) and that individual's choice of travel mode. Both in this example and in general, we should add such confounding variables to our causal graph.

Next, we consider the effects of selection. As noted by Greenland (2020), all datasets have a causal graph that implicitly conditions on a selection node. I.e., we only analyze data that has been selected to be a part of our dataset. We should therefore consider how all of the other nodes in our causal graph relate to the selection node. In particular, will we suffer any selection bias due to the outcomes influencing whether an observation is selected for inclusion in our dataset? Selection bias, if present, can cause our estimated causal effects to differ greatly from their population counterparts. This stems from systematic differences between the observations that have been selected into our dataset and the observations in our population of interest. For more details, see Heckman (1979) and Hernán et al. (2004) as canonical references.

Another universally implied yet only implicitly described element of one's causal graph is the prior data and code that led to one's dataset (Greenland, 2020, Pg.7). Presumably, prior data and potentially code-enabled-analysis influenced the sample design that led to your dataset. Perhaps some data transformations and code to implement those transformations was used to convert a raw dataset into the dataset being used for causal inference. And at all times, one uses computer programs to compute your reported results. In each case, the prior data is variable that influences your current data, and your code is computational (sub)graph that is implicit in your causal graph. These elements should perhaps be made explicit, and their influence on your causal effect estimates should definitely be assessed and reported.

Next, we should explicitly consider the role of time, even in research that may be cross-sectional due to the data that is available to us or due to the problem itself. In reality, how do we think our system evolves over time? If we consider multiple observations of a given decision maker, how does that decision maker's observed variables at time t partially cause future variables important to the context or outcome(s) for that decision maker at time $t' > t$? How do the actions of a decision maker i at time t partially cause the future context or outcomes of a decision maker j? We should add explicit nodes to our graph, subscripted or denoted by time, to show the cross-time causal relationships in our system. For in-depth discussion of time-related causal inference topics, see papers such as Gill and Robins (2001), Eichler (2007), and Peters et al. (2013). Please note that the literature on this topic is vast, and the cited authors are not at all exhaustive or representative of all papers in this space. Interested readers are encouraged to perform further literature searches on their own.

Similarly, we will frequently want to consider the role of space. In the context of choice modelling, this includes questions such as "how does a decision maker's existence in a particular geographical area shape their choices?" For example, consider multinational corporations where the business operates differently across state borders. Here, the borders associated with space directly causes a difference in the causal graph of how these businesses deliver their goods and services to customers. In other instances, one can consider space as a noisy proxy for unobserved confounders, such as cultural attitudes of a region's inhabitants (Paciorek, 2010). Rooted in the causal problem of unobserved confounding, this results in known statistical issues for choice modellers such as spatial correlation of model residuals (Fleming, 2004). Fortunately, some progress has been made in dealing with such problems. For general discussion of recent techniques in causal inference for spatial-causal modelling to deal with such issues, see Osama et al. (2019).

At this point, we have added to our causal graph all the outcome, explanatory, effect modifying, mediating, confounding, selection, data/code, time-indexed, and space-indexed variables that we believe are relevant for our problem. However, many of these variables may be disconnected nodes, i.e., singletons in the graph. We now focus on pruning nodes from this graph, before drawing our final hypothesized connections. In particular, we focus on pruning "post-outcome" variables that are not part of the causal graph for future

time periods or other observations. The reason for this is that conditioning on such post-outcome variables would bias our causal effect estimates. Our pruning exception is the selection variable which we have no choice but to condition on. It may unfortunately be a post-outcome.

To remove the problematic variables, we iterate through each of the non-outcome variables in our graph, and we assess whether each variable is actually a result of the outcome (perhaps in combination with other variables in our graph). These post-outcome variables temporally follow the outcome variable(s) but do not cause variables in the causal graph for other observations. We remove all such post-outcome variables from our graph.

Now is a good time to step back and consider what other researchers have thought about our problem. Specifically, we should conduct a literature review to see how other researchers have conceptualized the topic that we are working on. Have they included variables that we have not? Were those variables related our outcomes of interest? If so, should we add these variables to our causal graph? How should these variables enter our graph? Do the included variables of other researchers suggest the existence of confounders in their work that we should include in our graph? Have other researchers ascribed differing roles to our graph's current variables than we have? For example, have other researchers judged a variable to be a confounder, when we solely thought of the variable as an effect modifier? As we answer these questions, we should critically examine the evidence for these alternative decisions to see if we should also reconsider how we're judging our variables.

Finally, we need to connect the variables in our graph.

1. Draw direct arrows from our explanatory variables, confounders, and effect modifiers to the outcomes.
2. Draw arrows from the explanatory variables to the mediators, and then draw arrows from the mediators to the outcomes.
3. Draw arrows from the confounders to the explanatory variables and mediators that they may cause.
4. Draw arrows from the variables in time t to the variables that they cause in time $t + 1$.
5. Draw arrows from the variables that cause one's location in space to spatial variable nodes, and draw arrows to variables that are caused by one's location in space.
6. Draw arrows from all other nodes in one's graph to the selection node, based on which variables cause inclusion in one's dataset.

After drawing in all arrows, we should now have a fully connected causal graph. Pause. Take a moment to look over the graph to ensure there are no remaining singletons and that we have not drawn any spurious connections. Then, take a moment to celebrate. Drawing a project's first causal graph is hard work!

After celebrating, take a moment to pursue the following graph editing exercises. First, think about how the graph might differ across sub-populations. What sub-populations, if any, exist in your population of interest? Are there any causal relationships that should, or should not, not exist for a given sub-population? For instance, are the outcomes in some sub-populations independent of a given explanatory variable? Can you think of any inverted causal relationships that are specific to this sub-population? (I.e., for a given sub-population, does $B \rightarrow A$ instead of $A \rightarrow B$?) As explained in Druzdzel and Díez (2003, Sec. 4), add these sub-populations to one's initial causal graph via a "selection node," or if this is not clear enough, draw modified causal graphs for each sub-population of interest. Now, one can actually relax. This concludes the "purely mental" drafting of one's causal graph. In the next section, we'll look at testing this graph against data, and making any edits deemed empirically necessary.

5 Testing of Causal Graphs

5.1 Testing observable assumptions

In the last section, we reviewed a process for creating an initial causal graph using expert opinion. Critically, after drafting a causal graph, we should test it against our available data. Doing so will add robustness and credibility to our final conclusions. This is important because, if our graph captures inaccurate assumptions

about the data generating process, then we have no reason to think that our conclusions from using the graph will be accurate.

To test our causal graphs against data, we will first test the implications of our graph that involve observable variables only. We will defer the task of testing implications that involve unobserved / latent variables to later in this subsection. For now, recall our discussion in Section 3 about the two basic implications of causal graphs: marginal independence and conditional independence. In both cases, direct testing of marginal or conditional independence amongst nodes in the causal graph may be difficult. Indeed, there are no direct tests of conditional independence that can detect all types of dependence, especially for continuous variables (Bergsma, 2004; Shah et al., 2020).

As a result of this hardness, there are a myriad of research efforts aimed at testing conditional independence. These efforts rely on additional assumptions about the variables or the test statistic itself. Some researchers create tests under the assumption that one has access to an approximation of the conditional distribution of $X \mid Z$ (Candès et al., 2018; Berrett et al., 2019). Other researchers designed conditional independence tests for general cases, assuming smoothness of the underlying data distributions and assuming accurate estimation of the distribution of the test statistic under the null hypothesis of conditional independence (e.g. Zhang et al. (2012); Strobl et al. (2019)).

In this chapter, we will take an easier and less decisive route to testing independence. If a pair of variables have conditionally or marginally independent distributions, then their statistical moments will also be conditionally or marginally independent. Accordingly, we will not test for marginal or conditional independence in distribution. We will instead perform a more tractable test for marginal or conditional independence in means. If the variables in question are not conditionally or marginally independent in their means, then we know they are not independent in their distributions. Conversely, even if a set of variables are marginally or conditionally independent in their means, this **does not** imply that the variables are independent in distribution. Mean independence simply provides justification for placing greater belief in the variables being distributionally independent.

This approach of indirectly assessing distributional independence by testing mean independence is not new. The following papers have all proposed and implemented such an idea: Burkart and Király (2017); Chalupka et al. (2018); Inácio et al. (2019). For conditional independencies, the crux of the approach is to predict Y based on X and Z. Then, compare against a prediction of Y based on a resampled value of X and the original Z. If Y is mean-independent of X given Z, i.e. $E[Y \mid X, Z] = E[Y \mid Z]$, then the predictive power of a model with resampled X should resemble the predictive power of a model with the original X. After all, in both cases, the conditional expectation of Y is independent of our X values (real or resampled). When assessing marginal independencies, we removes Z from the models for the expectation of Y and proceed as described.

Note that as with the case of testing distributional independence, testing mean independence still requires researchers to make choices. First, we have to select models for $E[Y \mid X, Z]$ and $E[Y \mid Z]$, respectively, for testing conditional and marginal mean-independence. Second, we have to choose the performance statistic (e.g. R^2, log-likelihood, etc.) to compare these models. Lastly, we also have to select a resampling method. In particular, how (if at all) will our resampling strategy account for the possible dependence between X and Z?

For our demonstration, we made the following choices. First, we used linear regressions to model $E[Y \mid X, Z]$ and $E[Y \mid Z]$. Second, we used R^2 as our test statistic for judging the regressions' predictive performances. Third, we resampled X without replacement, keeping the length of the resampled vector equal to the length of the original vector. In other words, we permuted X. Finally, we visualized our tests by encoding our observed test-statistic as a vertical line and by plotting the kernel density estimate of our test statistic's distribution.

Our rationales for these choices are as follows. In our dataset, most of our explanatory variables were continuous (at least in theory). Accordingly, R^2 seemed a sensible performance metric for a model of the conditional expectation of a continuous random variable.

In contrast to our choice of performance metric, we chose our conditional expectation models and resampling methods through empirical testing. In particular, we created simulations to assess our mean-independence testing procedure. We assessed the performance of our mean-independence testing procedures using simulations where $Y \leftarrow Z \rightarrow X$ and X either did or did not cause Y.

Of particular importance were our simulations under the null hypothesis where X was conditionally

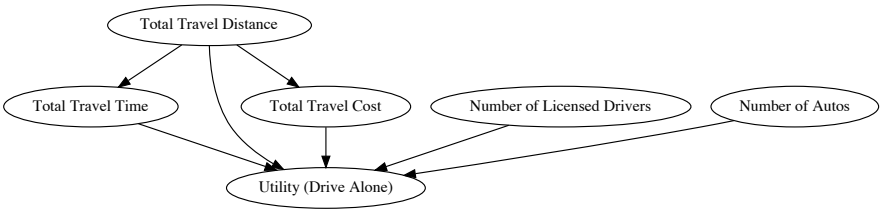

Figure 11: Expository causal graph of drive alone utility

independent of Y. Our initial simulations used random forests as our conditional expectation models and permutations as resampling methods. Random forests are a non-parametric method that would allow us to have less fear of model misspecification, and permutations are easy to implement. However, under the null hypothesis, the random forest-based tests resulted in non-uniform p-values. Such non-uniform p-values makes it harder to a-priori reject a false model (Gelman et al., 2013). When we switched from the combination of random forests and permutations to linear regressions and permutations, our p-values turned out to be empirically, uniformly distributed. Moreover, we still retained high statistical power.

We do not claim that these choices for assessing mean independence will always be appropriate. Indeed, one should assess one's tests on simulated data that resembles one's real data. For our dataset and simulations though, the combination of linear regressions, permutations, and R^2 resulted in adequate tests of marginal and conditional mean-independence.

5.2 Demonstration

To demonstrate the testing procedures described above, we used the causal graph in Figure 11. This causal graph shows a set of hypothesized causal relationships between variables thought to contribute to the utility of the drive-alone travel alternative in our dataset. As drawn, this graph encodes multiple marginal and conditional independence assumptions. Luckily, tools such as Daggity (Textor et al., 2016) can infer all independencies based on one's graph. For didactic purposes, however, we focused our attention on two particular independence assumptions.

First, we tested the assumption of marginal independence between the number of licensed drivers and the number of automobiles in a household. A-priori, we assign low probability to this independence. Generally, we expect the number of automobiles to be positively related to the number of licensed drivers in a household. Secondly, we tested the assumption that travel cost was independent of travel time, conditional on travel distance. Unlike the previous independence assertion, this conditional independence is a-priori more credible. In both cases, we will test our assumptions using the previous subsection's procedures.

In particular, Figure 12 shows the results of using permutation, linear regression, and R^2 to test the hypothesis of marginal independence between the number of automobiles and the number of licensed drivers in a household. The empirical p-value of 0 confirms that the observed data is unlikely given the null-hypothesis of marginal, mean-independence. More specifically, when regressing the number of licensed drivers in a household on the number of cars in that household, one achieves an R^2 near 0.4. In contrast, when permuting the number of cars in the household and re-estimating the regression, the distribution of p-values concentrates around 0. This plot visualizes the fact that—through the lens of our chosen test statistic (R^2), linear regression model, and permutation-based resampling strategy—data generated under an assumption of marginal mean-independence does not "look like" the observed data. Accordingly, we should consider the weaker assumption of marginal dependence. This relaxation may contain data-generating assumptions that better reflect our observations.

In Figure 13, we have an analogous visualization of a conditional independence test. Here, we test the

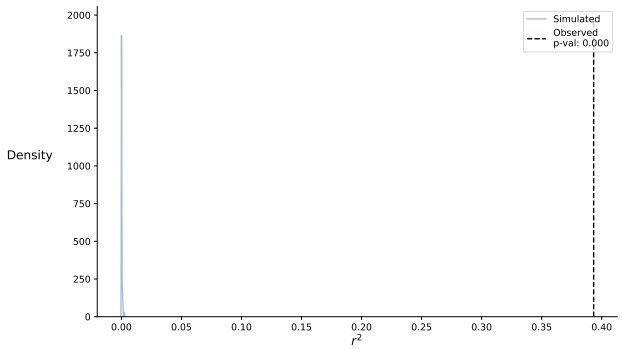

Figure 12: Marginal independence test results for the number of cars and licensed drivers in a household

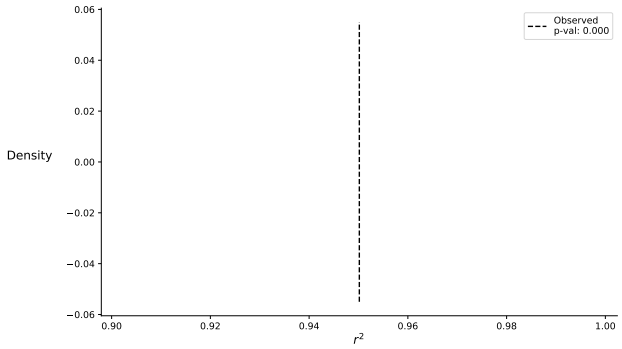

Figure 13: Conditional independence test results for travel time and travel cost given travel distance

hypothesis that travel time is mean-independent of travel cost, conditional on travel distance. To execute this test, we model the conditional expectation of travel time as a linear function of travel cost and travel distance. As with the marginal independence test results, the R^2 of the model using the observed values of travel cost are greater than the model's R^2 using any simulated datasets. In fact, the simulated R^2 values are so tightly distributed between 0.9371 and 0.9373 that the distribution is not visible in relation to the observed value of 0.95. Again, this means that the R^2 using observed data is unlikely given our method of sampling from the null distribution of R^2 given conditional, mean-independence of travel time and travel cost.

As before, these results suggest conditional dependence between our variables of interest. In particular, we should investigate how travel time and travel cost relate, conditional on travel distance. Why might this be the case? Does travel time cause travel cost when driving alone? Does travel cost cause travel time while driving alone? Does some other set of variables (potentially unmeasured) cause both travel time and travel cost?

Thinking through these questions, we can immediately think of latent variables that cause both travel time, travel cost, and the choice, even after conditioning on one's travel distance. For example, consider whether one drives alone over the San Francisco Bay Bridge. If one crosses the bridge, then traffic delays will likely increase one's travel time. Moreover, if one crosses the bridge, then one's travel cost is higher due to tolls that one must pay. And finally, if one takes a toll lane to get across the bridge faster, then one also pays a higher price. Overall, intuitive explanations exist for conditional dependence between travel time and travel cost. These explanations suggest particular relationships to analyze and particular variables, such as bay bridge crossings, to include in one's mode choice (i.e. outcome) model.

5.3 Testing assumptions involving latent variables

Now that we have described testing with observable variables, we can more easily describe conditional independence tests that involve unobserved (i.e., latent) variables. Indeed, when dealing with observational

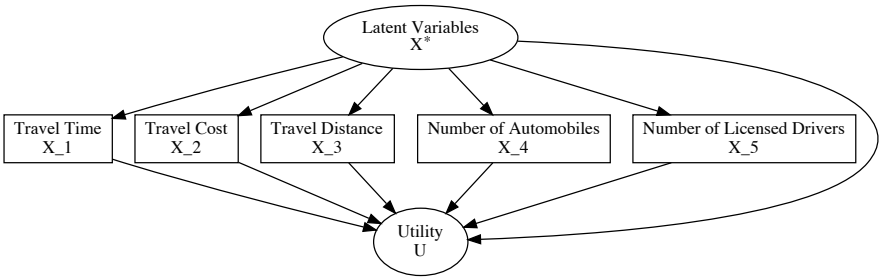

Figure 14: Causal graph from applying the deconfounder algorithm (Wang and Blei, 2019) to our dataset.

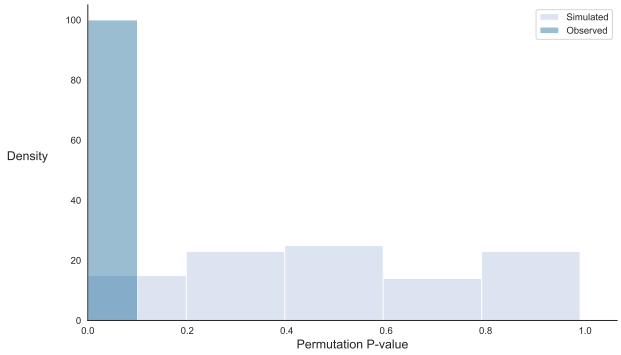

Figure 15: Results of testing that the number of drivers is independent of the number of automobiles in the household, conditional on the latent variable.

data, we will frequently find ourselves not having observed all variables that are of interest. Nevertheless, we still wish to test whether our data contradicts our graph. One way to directly extend our conditional independence testing to account for latent variables is to adopt a missing data perspective and impute the latent variables from a prior distribution. In particular, we can generalize our previous tests as follows.

First, we can consider expanding our test. Instead of performing one test with a set of observed X, observed Y and observed Z, we perform tests of observed X, observed Y, and imputed Z. This recasts the randomness underlying the null-distribution in our original test statistic of R^2 as a function of our permutation of Y and our imputation of Z. Here, we impute Z by sampling from the prior or posterior distribution of Z, depending on whether we're testing independencies before or after performing inference on our model's parameters. Moreover, we'll now compute this test's p-value by averaging over the permutations and imputations. Specifically, our p-value will be

$$E_{\text{samples, permutations}}\left[\mathbb{I}\left\{R^2\left(X, Y, Z_{\text{sampled}}\right) < R^2\left(X_{\text{sampled}}, Y^{\text{permuted}}_{\text{sampled}}, Z_{\text{sampled}}\right)\right\}\right] \tag{4}$$

where $\mathbb{I}$ represents the indicator function that equals one if the condition inside its braces is true and zero otherwise. For reference, this is the same as the p-value for test statistics (or discrepancies) defined in Gelman et al. (1996, Eq. 7).

Lastly, note that our "observed" test statistic is itself a random variable: it depends on the imputed values of Z. Because we now have a distribution of observed test statistics, we change our visualization method. Instead of plotting a single line versus a distribution, we now plot two distributions against one another. We first plot the distribution of "observed" test statistics that we computed using the observed X, observed

Y and imputed Z values. Then, we plot the distribution of "sampled" test statistics using prior samples of (X, Y, Z) as a reference. Here, we have one value per imputed vector Z in both the observed and sampled distributions. However, for the distribution of "sampled" test statistics, we marginalize over permutations of Y since this distribution represents the null hypothesis of conditional or marginal independence.

That last paragraph may have been confusing, so we'll walk through an example. Here, we apply the deconfounder algorithm of Wang and Blei (2019) to our data. In particular, Figure 14 shows the deconfounder's assumed causal graph. Note, we describe this application more thoroughly in Section 7. For now, we present the graph to highlight the assumptions that we will test. Specifically, we'll examine the assumption that the observed number of licensed drivers in a household is independent of the observed number of automobiles in that household, conditional on the latent variable X^*.

Figure 15 shows the result of following the aforementioned testing procedures for assumptions involving latent variables. From the Figure, we see that the distribution of "observed" test statistics clusters around zero while the distribution of "sampled" test statistics is closer to uniform. This result highlights the fact that (generally) there is "no free lunch": our approach to testing assumptions involving latent variables has its drawbacks. In particular, these tests are sensitive to assumptions about the joint prior distribution, $P_{\text{prior}}(X, Y, Z)$.

If, as in this case[2], the observed data (X, Y) is unlikely under the joint prior distribution $P_{\text{prior}}(X, Y, Z)$ then the conditional independence test is likely to fail. As always, the failing test indicates that the observed data is unlike the simulated data used to make the reference distribution. Unfortunately, we are unsure of how much dissimilarity comes from conditional independence violations. The observed data can differ distributionally from the simulated data in many ways. This highlights the need for extensive prior predictive checking of one's assumed joint prior, $P_{\text{prior}}(X, Y, Z)$, *before* using conditional independence tests on causal graphs with latent variables. Specifically, we want our marginal priors $P_{\text{prior}}(X^*) = \int P_{\text{prior}}(X^* \mid Z^*) P(Z^*) \partial Z^*$ to reasonably well represent the observed X (and the same for $P_{\text{prior}}(Y^*)$ and Y). Then, any remaining discrepancies between our observed data and our simulated data can be mainly attributed to their differing conditional independence properties.

5.4 Additional techniques

The methods presented in this section test independence assertions using one's dataset. While perhaps the most accessible strategy for testing one's causal graph, other techniques apply as well. For instance, Pitchforth and Mengersen (2013) proposed a checklist of qualitative questions for one's causal graph. Answering these questions should increase the trustworthiness of one's graph. Alternatively, there are other quantitative tests of one's causal graph that were not explored in this section.

For instance, causal graphs encode assumptions about the number of independent variables in one's data. The independent variables are the parentless-nodes in one's graph. Crucially, each graph assumes a particular number of such parentless-nodes. To test this assumption we first estimate our data's "intrinsic dimension." Then, we test whether the intrinsic dimension equals the number of parentless-nodes in our graph. For more information on estimating the intrinsic dimension of a dataset, see Camastra and Staiano (2016); Song et al. (2019). Additionally, see Chenwei et al. (2019) for an extension of this idea when we cannot rule out unobserved confounding.

Another empirical implication of one's causal graph is the existence of so-called "vanishing tetrads" (Spearman, 1904). This term signifies that the difference between the product of two particular pairs of covariances must be zero. As stated, this implication of one's causal graph is hard to intuitively understand. However, one can graphically determine the existence of vanishing tetrads and determine which variables are part of these tetrads. Then, one can estimate the necessary covariances and test to see if their difference of products is indeed unlikely to be zero. Such a test is yet another way to empirically determine whether one's graph is incompatible with one's dataset. For the original theorems proving that tetrads can be graphically identified and characterized, see Shafer et al. (1996) and references therein. For a more detailed and intuitive explanation of the graphical criterion for vanishing tetrads, see Thoemmes et al. (2018).

[2]Details of the prior predictive checks that show prior-data mismatch are not shown due to space constraints. Please see `https://github.com/hassan-obeid/tr_b_causal_2020/blob/master/notebooks/final/_04-tb-testing-your-causal-graph.ipynb`

Finally, we note that there are still a whole host of other techniques for testing one's causal graphs. Many of these remaining techniques are useful when one's causal graph contains unobserved (i.e., latent) variables. On one hand, we can use "triad constraint" tests that test independence between "pseudo-residual" values and one's explanatory variables (Cai et al., 2019). Results from these tests are useful for judging how unobserved variables in our graph relate to each other and to our observed variables.

Relatedly, one can make use of constraints on entropies of our observed variables instead of independencies. The idea is that differing latent variable graphs imply differing entropies in our observed variables. Accordingly, we test for these entropies and constraints. For more information and examples, see the literature about:

- inequality constraints, e.g. Tian and Pearl (2002); Kang and Tian (2006); Ver Steeg and Galstyan (2011)
- information inequalities, e.g. Chaves et al. (2014a)
- entropic inequalities, e.g. Chaves et al. (2014b)
- the inflation technique, e.g. Wolfe et al. (2019); Navascués and Wolfe (2020)

6 Causal Discovery

Our previous section detailed a method for testing independence assumptions of one's causal graph. As presented, this strategy requires one to first have a causal graph. Indeed, this requirement is why Section 4 provides instructions on how to construct an initial causal graph using expert opinion.

Our unstated presumption is that we will test our proposed causal graph. If any of our tests fail, we will then revise the graph until its assumptions appear defensible. Essentially, we postulate, test, and edit our causal graph until the data conform to the graph's assumptions. If this iterative discovery process sounds tedious, that is because it can be!

In this section, we will discuss how we can avoid such repetitive, manual graph editing and testing. Specifically, this section describes causal discovery: algorithmically inferring our causal graph from data. Here, we detail why causal discovery is important; we provide a brief overview of the main concepts in causal discovery; and we show the results of using causal discovery algorithms on the dataset described in Section 2. At the end, we provide references to some further topics at the intersection of causal discovery and experimentation.

6.1 Why use causal discovery?

As a topic of study, causal discovery is important for numerous reasons. Three of these reasons are the following. First, causal discovery promotes robustness and understanding of our causal effect estimates. For example, we can use causal discovery to understand how our prior beliefs affect our inferences. To do so, we could compare our inferred causal effects under expert-opinion versus data-driven causal graphs.

Secondly, causal discovery helps inspire the creation and editing of expert-opinion graphs. If we have not already created our own causal graphs, then we will find that it's typically easier to edit discovered graphs than to create from scratch. Alternatively, graphs found via causal discovery can spark revisions if we have already created our own graphs. This is especially likely when the discovered graphs feature different causal relations than are present in our own graph. Thirdly, causal discovery algorithms can aid characterization of posterior uncertainty in one's causal graph and causal effect estimates. Each causal graph discovered from one's data represents an alternative way of understanding the world, and we can quantify the probability of each of these causal models representing our data generating process.

Let's begin with robustness. Here, one way of reducing the probability of incorrect inferences is to give oneself multiple chances to be correct. In particular, the Section 5 tested, expert-opinion based graph was never meant to be the stopping point in one's exploration of possible causal relationships. Instead, we advocate using multiple graphs to help us understand our causal effect estimates.

Specifically, our effect estimates are dependent on our causal graphs. To assess dependence strength, we can compare our effect estimates under different causal graphs. For instance, we can use a discovered

graph versus our expert-opinion graph. Any graph-induced differences reflect and characterize structural sensitivity in our causal effect inferences.

Beyond increasing our understanding of our estimates, causal discovery algorithms can help us create causal graphs based on expert-opinion. In particular, criticizing causal graphs is easier than creating them.

As noted by Pearl (1995, p. 708), "every pair of nodes in the graph waves its own warning flag in front of the modeller's eyes: 'Have you neglected an arrow or a dashed arc?" Additionally, the presence of directed causal relations is vividly placed before one's eyes for immediate criticism (e.g., is $X \to Y$ plausible?). Similarly, undirected or bi-directed edges between variables highlight causal ambiguity. Such call-outs invite analysts to resolve the question of directionality in the relationship. And conversely, we may learn from causal links (i.e. arrows) that are present in the graphs output from our causal discovery algorithm that we initially overlooked. By contrasting and criticizing alternative graphs, we clarify the strengths and deficiencies of our own point of view. Then, once we've identified elements that we think should or should not be present in a causal graph for our dataset, we can amend our hand-crafted graph to meet these requirements.

Lastly, causal discovery can help us characterize our posterior uncertainty about the data-generating causal graph. They enable approximation of the posterior distribution over causal graphs, in at least two ways. One approach is to use a weighted likelihood bootstrap approximation (Newton and Raftery, 1994) to the posterior distribution over graphs. In this approach, one would first sample a vector of sample weights for the likelihood terms. Then, one would use those weights in one's causal discovery algorithm to produce a single 'sample' from the posterior approximation.

Alternatively, we could use yet another randomize-then-optimize (Bardsley et al., 2014; Orabona et al., 2014) approach to sampling from an approximate posterior distribution. Here, one would sample from a prior on entries of the matrix representation of a causal graph. Semantically, we sample constraints such as "$X \to Y$ (MUST | MUST NOT) be in the causal graph". We "sample" from the approximate posterior by running the causal discovery algorithm on the original dataset, with the inclusion of these randomly generated constraints. And, of course, we can consider hybrids of these two posterior approximation schemes.

With these posterior approximation methods, we can generate causal graphs for all the purposes mentioned above:

- for criticism and inspiration,
- for distributional analyses of the causal graphs, and
- for distributional analyses of one's causal effect estimates, conditional on each sampled graph. I.e., how certain are we of any one causal graph?

6.2 Overview of causal discovery algorithms

This subsection presents a non-exhaustive overview of causal discovery algorithms. For conciseness, an exhaustive review of causal discovery algorithms is out of the scope of the article. Crucially, we rely heavily on review papers such as Glymour et al. (2019) and Spirtes and Zhang (2016) to fill in our gaps.

Overall, there are three classes of causal discovery algorithms. One class attempts to directly infer the marginal and conditional independencies in one's causal system. That is, this class of algorithms identifies a so-called Markov Equivalence Class (MEC) of graphs. Considering the discussion of independence testing in Section 5, this class of algorithms is perhaps best understood as repeated and systematic independence tests. These causal discovery algorithms are constraint-based algorithms, because the observed independencies represent constraints that define the space of plausible causal graphs for the dataset. Common constraint-based algorithms include the Peter-Clarke (PC) algorithm and the Fast Causal Inference (FCI) algorithm (Glymour et al., 2001). The PC algorithm assumes no unobserved confounding variables, whereas the FCI allows for (and sometimes infers) the presence of such unobserved confounders.

The second class of algorithms are score-based algorithms. These techniques proceed sequentially, in pairs of variables. For each considered pair, an edge may be added, removed, or reversed in direction. These changes are made greedily, so long as our scoring criterion improves. Most commonly, the scoring criterion is the Bayesian Information Criterion for the joint prediction of all variables in our dataset (Malinsky and Danks, 2018). However, newer scoring criterions have been created with wider applicability than the BIC. See Huang et al. (2018) for details.

To begin using a score-based algorithm, we start with a fully disconnected graph. To this graph, we add directed causal relationships to increase the score of the generative model that corresponds to our graph. Once we cannot improve the score this way anymore, we have found the graph structure that maximizes the score on our data. From this graph, we then prune as many directed causal relationships as possible without harming our score. In the end, the algorithms return the resulting set of graphs that retain maximal score. Common score-based algorithms include the Greedy Equivalence Search (GES) algorithm (Chickering, 2002). Typically these methods operate under stricter assumptions than constraint-based methods. Nonetheless, combinations of score and constraint-based ideas have outperformed methods from either class alone (Glymour et al., 2019).

Lastly, the third class of algorithms estimates Functional Causal Models (FCM) (Goudet et al., 2018) for each of the variables in our system. The defining characteristic in such algorithms is that they exploit asymmetries in the residuals of models for the hypothesized relationships of $X \to Y$, $Y \to X$. In particular, the residuals will be independent of the hypothesized cause in only one of the two potential models. As noted in the description of such algorithms, they are especially useful for discovering causal relationships between pairs of variables. In other words, discovery methods based on FCMs enable orientation of a graph's undirected or bi-directed edges. By orienting these edges, FCMs reduce ambiguity over whether $X \to Y$ or $Y \to X$. This orienting capability can be usefully combined with the previous causal discovery algorithms that infer classes of graphs with equivalent marginal and conditional independencies. And of course, extensions of these techniques exist for inference in the presence of unobserved confounding (Goudet et al., 2018, Sec. 6) and for learning an entire causal graph as opposed to dealing solely with variable pairs (Zheng et al., 2020).

6.3 An application of causal discovery

To demonstrate the methods in this section, we chose to use the simplest causal discovery algorithm: the PC algorithm (Glymour et al., 2001). For practitioners who may reasonably start with the simplest method available and increase methodological complexity as needed, this should be an illuminating starting point. As noted in Section 5.2, we suspect that our causal graph for the drive alone utility of our dataset contains unobserved confounding. Given that the PC algorithm assumes that we are free of unobserved confounding, our example allows us to observe how the algorithm behaves under violation of its assumptions. Does the algorithm fail gracefully and point towards violations of its assumptions, or does it return incorrect results with certainty?

To begin, it helps to understand the PC algorithm's general procedure. It uses all our observed variables to infer a skeleton of a causal graph. That is, the algorithm attempts to infer an undirected graph that denotes which variables relate to which other variables, ignoring the directionality of causation between them. Then, after inferring a skeleton, the algorithm attempts to orient the undirected edges as much as possible. Optimistically, we end up with a fully directed acyclic graph. In other cases, we recover a mix of directed and undirected edges, denoting a MEC of graphs with the same independence properties. In the final case, we recover an undirected causal graph. This corresponds to the situation where we cannot infer which objects cause which other objects. We merely know that given sets of objects relate to each other, not why these relationships exist.

With these basics understood, we can now present the results of using the PC algorithm to infer the causal graph for the variables present in Figure 11. For reference, the variables (travel time, travel cost, travel distance, number of automobiles in one's household, and the number of licensed drivers in one's household) are all thought to influence the utility of commuting by driving-alone. We care about the causal relationships between those explanatory variables because intervening on any one of these variables may cause downstream impacts on another explanatory variable. As shown in Section 2, to make accurate causal inferences, we need to know and account for these differing pathways of influence on one's utility and mode choice. Causal discovery helps us discover these differing pathways of influence by generating plausible causal graphs for our datasets.

Figure 16 shows the final result of applying the PC algorithm to the variables in our drive-alone utility function. Two main differences exist between this graph and the example graph shown in Figure 11. First, the number of licensed drivers is not independent of the number of automobiles in the household. The graph discovered via the PC algorithm depicts the number of automobiles in one's household as a function of two

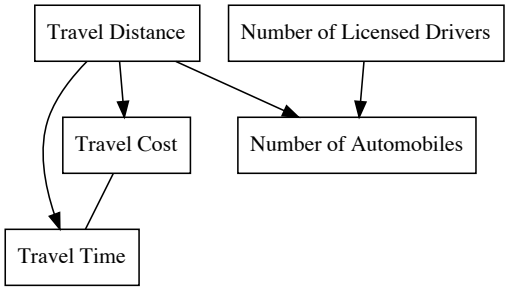

Figure 16: Result of using the PC algorithm on variables in the drive-alone utility

variables: the number of licensed drivers that one has in one's household and how far one has to travel to work or to school. The second major difference is the presence of an undirected edge between travel time and travel cost. The discovered graph labels travel time and travel cost as dependent, even after conditioning on travel distance.

As described in Section 5.2, independence testing foreshadows (indeed, determines) both of these results. Marginal independence testing showed us that the number of licensed drivers in a household and the number of automobiles in that household are not independent. Likewise, conditional independence testing revealed that travel cost is not independent of travel time, conditional on travel distance. Far from being a surprising congruence, we should expect this alignment: conditional and marginal independence testing results are used to generate the graph returned by the PC algorithm.

Relative to manual independence testing, what do we gain from using causal discovery algorithms? Critically, we gain at least three benefits from causal discovery algorithms. First, we gain insight into the dependence structure of our variables. For instance, we recover that the number of licensed drivers causes the number of automobiles, as opposed to the reverse. Secondly, we gain a greater sense of uncertainty in our causal graphs. For instance, we can bootstrap our data and look at the distribution of inferred causal graphs. Moreover, discovered graphs with undirected edges express uncertainty about what-causes-what. Disclosing and marginalizing over structural uncertainty of this kind is rare in choice modelling, as far as we know. Lastly, we gain confidence in our results. Through automated tests, we ensure our results are not based on untested or implausible causal assumptions.

6.4 Experimental extensions

As just demonstrated, causal discovery algorithms take in data and output causal graphs. So far, we described causal discovery algorithms in the context of observational data, where there is no random assignment to treatment. However, this setting has its limitations. Indeed, limitations on a variable's observability lead to causal discovery algorithms sometimes returning graphs with undirected edges, representing latent confounding. To overcome issues of confounding, there are causal discovery algorithms that make use of experimental data. In particular, one should think of three kinds of experiments: planned, unplanned, and natural.

Planned experiments are what we typically think of as experiments. These include A/B tests in technology companies, medical trials for drug certification, randomized controlled trials by economists and social scientists, etc. Unplanned experiments are instances of true randomization that were not pre-meditated for experimentation. For instance, technology companies commonly use random allocation (e.g., 'np.random.choice' in Python) throughout their code bases, outside of formal experiments. These are often placeholders or temporary measures, but each such instance is an experiment that allocates some individuals to one treatment and some to another.

Finally, natural experiments are instances where our system is subject to non-random interventions. Sometimes, these natural experiments take the form of a policy that we apply uniformly across our system.

For example, imagine a country-wide change in immigration policy that affects the supply of employees into the country, when one is analyzing firm success. In other instances, the interventions affect specific individuals. For instance, rounding prices up or down to the nearest dollar allocates people with a "true price" of $2.51 to a $3.00 treatment, and it allocates people with a "true price" of $2.49 to a $2.00 treatment. This treatment allocation is not random, but perhaps there are ways it is "as good as random" thus permitting us to treat it similarly to a formal experiment.

In making use of planned, unplanned, and natural experiments, there are roughly two types of causal discovery algorithms. One class of causal discovery algorithm takes a causal graph with unresolved ambiguities and outputs an experimentation plan whose resulting data will permit resolution of the causal graph. For algorithms of this kind, see work by Ghassami et al. (2018), the work cited therein, and related literature. The second class of algorithm takes available experimental and observational data and tries to best combine these sources of information to construct a causal graph that is valid for all datasets. For references and guidance in this vein, see work such as Tian and Pearl (2013); Peters et al. (2016); Ghassami et al. (2017); Zhang et al. (2017); Kallus et al. (2018).

Finally, to complement this sub-section's discussion about using existing experimental data, see Section 8. There, we discuss using planned experiments for proactive data acquisition efforts to help us infer our system's causal graph. In essence, planning experiments to get the data we need, as opposed to merely doing our best with the data we have.

7 Latent Confounding

This section focuses on latent confounding in causal inference problems. As mentioned in Section 4, latent confounders affect the treatment assignment of our causal variables of interest, as well as the outcome. While more complicated, this setup is the more realistic and typical case faced by demand modellers. We first go over a few examples of confounding in transportation analyses and explain the challenges that come with such cases. Next we will briefly review a few approaches to dealing with latent confounding.

Then we focus specifically the recent de-confounder technique of Wang and Blei (2019). In particular, we conduct a case study and simulation using the deconfounder. Using data from Brathwaite and Walker (2018b), our case study shows how directed acyclic graphs can help clarify one's reasoning regarding the number of confounders, the assumptions for which variables are confounded, and the models needed to estimate the causal effects. Additionally, we use a simplified simulation scenario to investigate the usefulness and pitfalls of the deconfounder approach for generating accurate model estimates.

7.1 Examples of confounding

Confounding occurs when a certain (confounding) variable induces variations in both the outcome as well as the treatment (policy) variables of interest, creating correlation between the treatment and the outcome that is not caused by the treatment variable itself. When the confounding variable (or variables) is observed, we can control for the confounding effect, and there exists many methods in the literature for how to do that, including post-stratification, multiple regression, propensity score methods, g-computation, targeted maximum likelihood estimation, etc. It is when the confounding variable is unobserved that the problem becomes significantly more challenging.

For an illustration, imagine we're interested in the effect of adding a bike lane on the mode share of bicycling in a given neighborhood. To estimate this effect, we might develop a disaggregate mode choice model with a dummy variable for whether a bike lane exists between an individual's trip origin and destination. We might then wish to treat the coefficient on this variable as the causal effect of adding a bike lane on the log-odds of choosing to bike. The problem with this strategy is that individuals may self-select to live in an area where bicycle infrastructure exists because they have a preference for commuting by bike. In other words, if we define an additional variable to encode a person's latent, inherent preference for biking, then this variable determines both a person's likelihood for living in an area with existing bicycle infrastructure and whether that person chooses to bike. This latent variable is subsequently expected to lack "balance" across the treatment and control groups: those who live near a bicycle lane are expected to have higher preference for biking than those who don't. Accordingly, if we neglect the latent confounder as a source of

variation in both our treatment and outcome variables, then we risk biasing our treatment effect estimates of interest.

7.2 Latent Variables

Latent confounders are a special case of latent variables, i.e. unobserved variables. These include both psychological constructs and physically missing observations. Psychological constructs include measurements about happiness, lifestyle, economic expectation, morale, among many others. Physically missing observations include measurement errors (such as inexact measurements of key variables, data entry errors, or measurement inaccuracy), missing data, survey item non-response, etc.

To cope with these latent variables, modellers make parameterized assumptions about how the latent variables relate to the observed variables. Then, these parameters are inferred alongside the latent variables and used to estimate causal effects of interventions. Examples of such latent variable models include Integrated Choice and Latent Variable (ICLV), Generalized Linear Latent and Mixed Models (GLMM; including mixed logit models), Factor Analysis Models, and Latent Class Models. For specific examples of these models being used to address latent confounding, see Louizos et al. (2017) and Perrakis et al. (2019) for factor analysis and latent class modelling examples, respectively.

From a choice modelling perspective, recall from Section 3.1 that ICLV models have been used for decades. The same is true of GLMMs / mixed-logit models. These models address primarily address issues of latent mediation. For mixed logit models this is evident in specifications where observable variables such as socio-demographics (e.g. income) cause an unobservable preferences (e.g. price sensitivity) that then influences the choice. For ICLVs, recall Figure 9 in Section 3.1. Despite their primary focus on mediation, these models dovetail with latent confounding in the following instances.

Consider socio-demographic variables such as age. Age may be thought to influence a latent attitude of environmental friendliness. This latent attitude may then influence both whether one lives near bicycle infrastructure and what travel mode one chooses. In this case, the latent variable of environmental friendliness is a latent mediator of age and the travel choice. Simultaneously, the latent variable is a latent confounder of bicycle infrastructure presence and travel choice.

Here, failing to account for the latent attitudes may bias our observational study if we're interested in the effect on bicycle mode share from adding a bike lane. However, we could use an ICLV model to both address latent mediation and latent confounding. For estimation, ICLV models usually rely on collecting indicator/proxy data to infer the levels of latent variable for individuals in a certain sample. Unfortunately, the collection of this data is not always easy or even feasible. For example, there may be sensitivity of individuals towards some attitudinal questions, or we may not be able to reach the individuals to conduct additional surveys with them. This makes it difficult for researchers to collect necessary information that allows for controlling for unobserved confounders in models.

7.3 The deconfounder algorithm

One recent method that has been proposed to deal with the problem of latent confounding without the need to collect additional data is the deconfounder algorithm by Wang and Blei (2019). The method attempts to control for the confounding variable by estimating a "substitute confounder": a set of variables that once controlled for, renders all variation in the treatment variables of interest exogenous. The process of applying the method can be stated simply. It proceeds as follows:

- First, estimate the substitute confounder using any good latent variable model the modeller chooses. The authors suggest estimating a factor model with k factors on the set of covariates the modeller is interested in.
- Second, check the factor model's accuracy using posterior predictive checks.
- Once a sufficiently accurate latent variable model is recovered, use it to estimate an expected value of the latent variable for each observation, and control for this value in the outcome model, alongside the treatment variables and other covariates of interest.

One main assumption of the deconfounder algorithm is that the data at hand should only have multi-cause confounders, thus the title of the paper, "The blessings of multiple causes." In other words, this method works when all unobserved confounders affect multiple of the observed causes (or treatment variables) of interest, alongside the outcome. This assumption is weaker than the standard ignorability assumption, which requires the absence of both single cause and multi-cause confounders for accurate causal inferences.

7.4 Case study: Simulation

The purpose of this section is to investigate the effectiveness of the deconfounder algorithm (Wang and Blei, 2019) in adjusting for unobserved confounding. We use simulated mode choice data where travel distance linearly confounds both travel time and travel cost. We then mask the travel distance data and treat it as an unobserved variable.

We estimate three models:

- Model 1: A multinomial logit with the correct original specification, except we omit the travel distance variable in the specification without trying to adjust for it. This model represents the worst case scenario where a modeller ignores, or is unaware of, unobserved confounding.
- Model 2: We use the deconfounder algorithm to try to recover the confounder (travel distance). In this method, we use all the variables in each mode's utility to recover that mode's confounder. This is in line with the approach taken in Wang and Blei (2019), where they use all the of observed variables in the factor model to recover a substitute confounder.
- Model 3: We use the deconfounder algorithm to try to recover the confounder (travel distance), but this time, we only use travel time and cost in the factor model, instead of all the variables in the utility specification of each mode. By only using what we know are confounded variables to recover the substitute confounder, our goal is to analyze whether we can improve the accuracy of this approach by adopting a stronger prior on which variables are confounded. This can be in the form of building and testing candidate causal graphs that try to illustrate this confounding.

We compare both the coefficient estimates on travel time and cost from each of those three models to the coefficient estimates obtained from using the actual travel distance values. These are shown in Figure 17. We also compare the distribution of the recovered substitute confounder under each of models 2 and 3 to the true confounder. The main finding of this exercise is the following: using the true variables believed to be confounded (i.e. method 3 where only travel time and cost are used to recover the confounder) leads to a better recovery of the true confounder. Figure 18 and 19 show a QQ plot of the true and recovered confounders under models 2 and 3 respectively. Looking at those figures, we see that the distribution of the recovered substitute confounder under method 3 is closer to that of the true confounder than method 2. This suggests that it may be better to run the deconfounder algorithm based on a hypothesized causal graph, rather than just running it on all the observed covariates. Please refer to Section 4 for how to build plausible causal graphs.

Additionally, and perhaps most importantly, the effectiveness of the deconfounder algorithm is very sensitive to small errors and misfits in the recovered confounder. This can be seen in three ways. First, although method 3 returns a relatively good fit of the true confounder (based on the QQ plot), Figure 17 shows that the adjusted coefficients on travel time and cost do not exhibit any significant reduction in the bias resulting from omitting the true confounder. Second, the confidence intervals on these coefficients in models 2 and 3 do not cover the estimated coefficient using the true confounders. I.e., the deconfounder-powered coefficient estimates are certain in their incorrectness. Third, the coefficients on the recovered confounders have p-values that are insignificant and orders of magnitude higher than the significant and low p-values on the coefficients estimated with the true confounder (travel distance). This means that despite having visibly good confounder recovery as shown in the QQ-plot, the recovery was not close enough to result in equivalent statistical inferences. This need for stringent confounder recovery raises questions about the usefulness of the deconfounder algorithm in practice.

Limitations notwithstanding, it is important to point out that sensitivity to small errors and misfits is not only a by-product of the deconfounder algorithm itself. In fact, it is one of the built in characteristic of

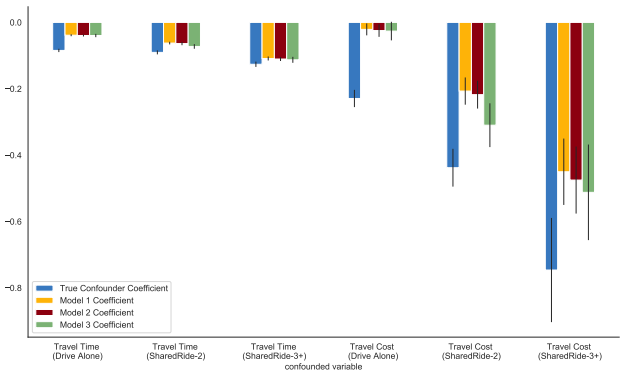

Figure 17: Bias in coefficient estimates on the confounded variables

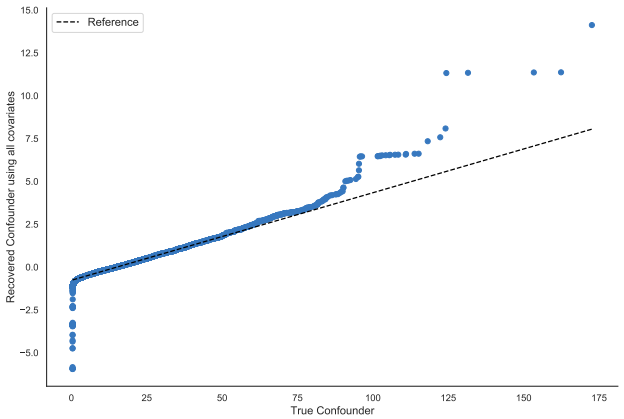

Figure 18: QQ plot of true confounder against recovered confounder using all covariates

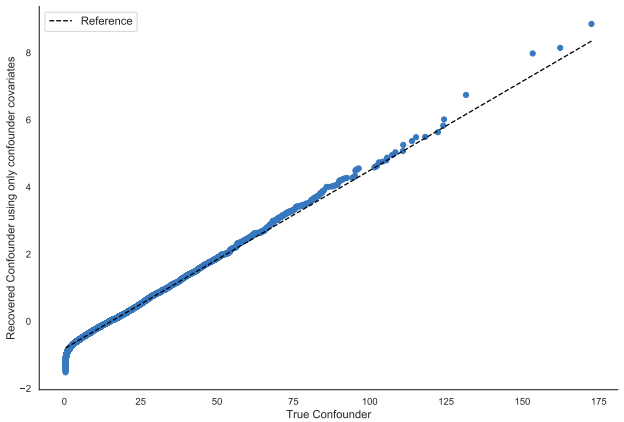

Figure 19: QQ plot of true confounder against recovered confounder using all covariates

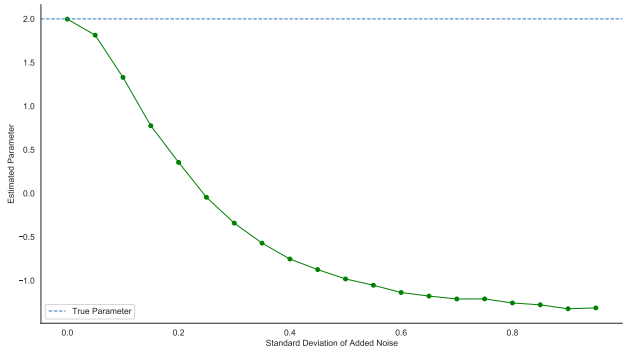

Figure 20: Sensitivity of causal estimates to random errors in the confounder variable

omitted variable bias, and perhaps more broadly, the problem of error-in-variables in regression. To illustrate this, suppose we actually observe the true confounder variable, but with some white, random Gaussian noise. Figure 20 shows how the bias in the parameter of interest increases quickly as a function of the standard deviation of the random noise. This emphasizes the difficulty of recovering unbiased estimates in the presence latent confounders, and highlights potential limitations with methods that attempt to recover a substitute confounder to control for.

7.5 Alternatives

Given the algorithmic difficulties with accurately inferring latent confounders, we encourage analysts to investigate and experiment with alternative methods of coping with unobserved confounding. In particular, we advise researchers to conduct sensitivity analyses and to compute bounds for their treatment effects of interest. Sensitivity analyses estimate the strength of relationship between a latent confounder and one's treatment variables that is needed to invalidate one's results. Audiences are then able to judge whether it is plausible for a confounder of that strength to exist. For more information about and demonstration of this technique for dealing with latent confounding, see Rosenbaum and Rubin (1983), Liu et al. (2013), and Jung et al. (2020). For related literature and guidance on bounding one's treatment effects in the presence of latent confounding, see Manski (1990), Richardson et al. (2014), and Geiger et al. (2014). The crux of this research is that even with unobserved confounding, we can derive credible bounds for our treatment effects. This enables us to avoid the difficulties of trying to provide precise point estimates via accurate reconstruction of the latent confounders.

8 Discussion

In this chapter, we've focused on why causal graphs are important, how to create them, how to test them, and on how to use them in applied problems with latent confounding. Our latent confounding example showed that statistical inference of our models' parameters may still be a challenge, even with a correct causal graph. Such challenges lead us to the following set of post-graph-construction topics:

- model estimation
- model checking
- experimental design
- experiment analysis
- decision analysis

To us, each item above is important for getting credible results from our analysis and for maximizing our interventions' benefits. Accordingly, even though we will neglect details due to space and time constraints, we briefly discuss these topics below.

Perhaps most obviously, after creating and testing a causal graph, we will use it to estimate the effects of our interventions. To compute our effect estimates, we will need to evaluate statements such as the probability of a particular node taking a given value, conditional on the values of that node's parents. These probabilities will come from our estimated models, so model estimation is critically important to our effect estimation. Thankfully, this is the part of causal inference that choice *modellers* are most familiar with. For instance, Kostic et al. (2020) first use the PC and GES causal discovery algorithms to generate a causal graph, then they test the graph qualitatively, and finally they estimate models corresponding to this causal graph. As another example, Garrido et al. (2020) start from where we end: at a known (or selected) causal graph. They then use neural network density estimators to model the necessary probabilities for estimating one's causal effects of interest.

Next, after estimating the models for our causal graph but before interpreting or using our results, we should check our entire system of models. Here, there are multiple, complementary ways of performing these diagnostics. We can check our models separately, jointly, or in subsets. For thoroughness' sake, we can even perform all these checks instead of one kind.

From a disaggregate perspective, we can consider a sequential application of model checking exercises, one per estimated model. Ideally, each model checking process will include the use of visual diagnostics, as (for example) described in Brathwaite (2018). Alternatively, we can check subsets of models together instead of checking one model at a time. For example, Tran et al. (2016) jointly check their models for all variables that their intervention will set (i.e. the treatment assignment variables), and then they separately check their outcome variable models. Finally, we can check all our models jointly by defining global diagnostic measures over all nodes in our causal graph. See Williamson et al. (2013) for a demonstration.

Following model diagnostics, we are ready to use our models and causal graph to inform real interventions. These interventions can come in two kind: an experiment or a "full-scale" implementation of one's policy. If our intervention is experimental, then we are likely interested in one of two aims. We either want to decide between one or more treatment options, or we want to learn about our system, though not necessarily to make a decision. In both cases, however, we pay great attention to the design of our experiments.

When experimenting to make decisions, such as whether to launch a given treatment or not, we pay extra attention to the size of our experiment. Specifically, we want our experiment's sample size to be large enough such that after we update our beliefs using the experimental data, that we have at least our minimum desired probability of making the correct decision. The decision can be to declare the effect of a treatment statistically different from zero, but more frequently, the decision will be more fundamental such as "implement treatment A." For thorough explanations of how to conceptualize and design experiments in a Bayesian, model-based setting, see Chaloner and Verdinelli (1995) and Wang et al. (2002). For examples and guidance on how to use one's causal graph structure to guide the general design of one's experiment, beyond sample size, see Madrigal et al. (2007). There, the structure of one's causal graph is used to inform general design decisions such as the clustered allocation of individuals to treatment, and the experimental design is itself analyzed graphically. Additionally, note the relations to reinforcement learning where an agent has to perform experiments in order to discover the action/intervention that will maximize her expected, counterfactual reward. In this context, Lee and Bareinboim (2018) have shown that designing our experiments without guidance from one's causal model is generally suboptimal, and that we can achieve optimality by leveraging our causal graph to design our experimentation plan.

Now, let's transition from experimentation for decision making to consider experimentation for learning. Imagine that we are at a transportation network company and that we are running a pricing experiment to learn about price elasticities of our customers. Here, there is no immediate decision being made, but we learn about an edge in our causal graph: the edge from price of a trip (treatment) to purchase of the ride (outcome). In other cases, we may experiment to learn not just about the strength of an edge, but about the presence of edges and the structure of the graph more generally. For example, we may wish to remove residual ambiguity from a causal discovery process that outputs a Markov Equivalence Class of graphs instead of a single causal DAG. In these situations, we are interested in optimally designing an experiment (or series of experiments) to learn a causal graph (or its properties). We are further interested in how we can leverage potentially multiple experimental datasets to improve our causal graphs. For a review of the literature on experimentation for learning and construction/refinement of a preliminary causal graph, see Hyttinen et al. (2013) and Kalisch and Bühlmann (2014, Sec. 3.1.2). For more recent approaches in this vein, see works such as Triantafillou and Tsamardinos (2015); Kocaoglu et al. (2017); Brouillard et al. (2020); Rantanen

et al. (2020).

Finally, after we have run any experiments that we are interested in, we still must decide how to intervene in our population. Three major types of questions come to mind immediately:

1. Should we launch the treatment(s) at all?
2. Should we launch the treatments to everyone?
3. How should the treatments be dispersed/implemented?

After updating our posterior beliefs with the experimental data, we'll want to analyze and come to a conclusion about launching our treatment(s). In particular, we will wish to determine the expected distribution of impacts under each alternative decision. Here, Manski (2019) should provide the basic idioms of thought and pointers to the larger literature on treatment choice from a decision theoretic perspective.

Next, if we've decided to launch the treatments, we come to the question of who should receive the treatment(s)? Everyone? A select few? Are there certain subgroups that should receive the treatment but not others? These questions fundamentally revolve around the level and nature of heterogeneity in treatment effects. We will, with good reason, want to search for evidence of heterogeneity and characterize it if found. In doing so, we should consult articles such as Pearl (2017) and Webster-Clark et al. (2020) for guidance on how to perform one's subgroup analysis in light of one's causal graph. This should help us avoid drawing incorrect conclusions or misinterpreting our analyses.

Thirdly, we will need to answer logistical questions about the levels and the frequency of treatment. With regard to choosing the levels of (possibly continuous and multiple) treatments, recent work on causal Bayesian optimization represents the state of the art in this area (Aglietti et al., 2020). Moreover, the entire field of reinforcement learning focuses on running experiments and learning from past observations to determine the treatment arms/levels that will maximize one's reward (however we define it). Accordingly, we stand to gain much by consulting the work on and principles from causal reinforcement learning (c.f. Bareinboim et al. (2015)) when choosing our optimal treatment plan.

9 Conclusion

Travel demand problems aim at forecasting the impact of proposed project and policies. These problems are causal in nature. To date, choice modellers have not routinized the use of causal inference techniques in the field of travel demand modelling. Brathwaite and Walker (2018a) documented this disconnect and presented an initial framework for addressing it.

In this chapter, we built upon their framework. We highlighted the importance of using causal graphs and causal inference methods in transportation demand modelling efforts and in choice modelling more generally.

We then presented a "selection on observables" simulation showing the importance of the data generating process in the estimation of treatment effects from external interventions. We showed, using correctly specified outcome models, that incorrect assumptions about one's data generating processes could lead to bias in estimates of one's treatment effect of interest.

To help avoid such pitfalls, we then discussed how using causal graphs effectively illustrate one's assumed, data generating process. We went beyond sole reliance on existing graphs, and we presented a process for constructing causal graphs based on expert opinion of the problem at hand. To ensure these opinions are empirically supported, we also presented methods for testing the implications encoded in a proposed causal graph. These implications might be either observed or latent, and implications of both kinds may be present at once.

Going further, we reviewed causal discovery methods for algorithmically creating causal graphs from data as opposed to depending solely on expert-opinion graphs. We described how causal discovery is helpful for discerning relationships between one's covariates. Then, we briefly presented several causal discovery algorithms. Following the review, we demonstrated one of these causal discovery algorithms, the PC algorithm, on a simple example. We showed that causal discovery methods help us test independencies within our data, resolve ambiguities in our graphs, and characterize the uncertainty in our graph inferences and treatment effect estimates.

Moving from inference to usage of causal graphs, we presented examples of latent confounding within transportation demand modelling. First, we highlighted some current methods for addressing latent confounding such as ICLV models. Noting their restrictive requirements for supplemental indicator data, we then presented a recently developed algorithm by Wang and Blei (2019) that works with just one's choice data. We used this model on the same example illustrated in the selection on observables simulations, and we highlighted instances where it aided recovery of a confounder in one's causal graph. We then showed that while the deconfounder algorithm might be a promising step in the right direction, it still has some notable deficiencies. Specifically, we showed that the deconfounder algorithm can be impractically sensitive to small errors in one's inferences.

Given these issues, we pointed out alternatives to the deconfounder that one may wish to investigate when dealing with latent confounding in one's analyses. Further, we reviewed and referenced the literature on the many downstream issues to be considered in order to maximize the benefit from one's causal inference activities.

Overall, we hope that this chapter has provided enough evidence to convince choice modellers that using causal graphs in their analyses is important and useful. Ideally, readers will step outside of their traditional analytical frameworks and use techniques presented across the causal inference literature. In doing so, we hope our chapter provides the necessary introductory knowledge (and references) on how to:

- construct causal graphs that fit their specific contexts,
- test the robustness and credibility of produced causal graphs against available data,
- make use of causal discovery techniques, and
- characterize and assess one's options for dealing with latent variables in one's causal graph.

If not already apparent, incorporating causal graphs into our work will help us all more clearly outline our assumptions and conduct more credible analyses.

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