CAUSAL INFERENCE IN EDUCATIONAL SYSTEMS: A GRAPHICAL MODELING APPROACH

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ABSTRACT

Educational systems have traditionally been evaluated using cross-sectional studies, namely, examining a pretest, posttest, and single intervention. Although this is a popular approach, it does not model valuable information such as confounding variables, feedback to students, and other real-world deviations of studies from ideal conditions. Moreover, learning inherently is a sequential process and should involve a sequence of interventions. In this paper, we propose various experimental and quasi-experimental designs for educational systems and quantify them using the graphical model and directed acyclic graph (DAG) language. We discuss the applications and limitations of each method in education. Furthermore, we propose to model the education system as time-varying treatments, confounders, and time-varying treatments-confounders feedback. We show that if we control for a sufficient set of confounders and use appropriate inference techniques such as the inverse probability of treatment weighting (IPTW) or g-formula, we can close the backdoor paths and derive the unbiased causal estimate of joint interventions on the outcome. Finally, we compare the g-formula and IPTW performance and discuss the pros and cons of using each method.

Keywords Causal Inference · Confounder · Directed Acyclic Graph · Education · Experimental Design · G-Formula · IPTW · Observational Study · Quasi-Experimental Design

1 Introduction

Educational systems have traditionally been evaluated using cross-sectional studies, namely, examining a pretest and posttest, and single intervention. Although this is a popular approach, it does not model valuable information such as confounding variables, feedback to students, and other real world deviations of studies from ideal conditions. Moreover, learning inherently is a sequential process and should involve a sequence of interventions. Nowadays, a large volume of educational data is available, allowing researchers to develop more intelligent and more complex modeling and inference algorithms.

Considerable research such as [1], [2], and [3] has been done in designing intelligent tutoring systems (ITS) in education. ITS is computer software that dynamically evaluates student knowledge and provides feedback. Some work such as [4] and [5] has been done in modeling student knowledge using hidden Markov model (HMM) and dynamic Bayesian networks (DBN). In [4], the authors used the HMM to predict student performance in an educational video game. They showed that student trajectories throughout the game can be strongly predictive of posttest scores. Some work such as [6], [7], and [8] has been done in the knowledge tracing, Bayesian knowledge tracing, and deep knowledge tracing. In

[8] the authors used a recurrent neural network (RNN) for deep learning knowledge tracing to model student learning. Although these frameworks are popular in education, they will not model time-varying confounders and factors that can influence student learning.

Potential outcome (PO) is a well-known causal inference framework in literature. Neyman first proposed PO for randomized experiments, and later on, it was extended to observational and non-randomized experiments by Donald Rubin. Causal DAG and structural causal model (SCM) developed by Judea Pearl [9] is another popular causal inference framework that uses directed graphs to model real world phenomena. Directed graphs have been used in many domains [10], [11], [12], and [13]. In [13], the authors used dynamic Bayesian networks to generate synthetic time series data. In this paper, first, we use causal DAG and graphical modeling frameworks to quantify the experimental and quasi-experimental studies. We study some well-known experimental and quasi-experimental designs in education and discuss their limitations in terms of threats to internal and external validities mentioned by [14]. Second, we propose more intelligent experimental designs by collecting richer data from students by including questions on potential confounders in the diagnostic test and instructing the TAs and instructors to inquire during office hours about potential confounders. Third, we propose to model the education system as time-varying treatments, confounders, and time-varying treatments-confounders feedback. We show that if we control for a sufficient set of confounders and use appropriate techniques such as the marginal structural model along with IPTW [15] or g-formula, which is the extension of Pearl's backdoor criterion, we can close a sufficient set of backdoor paths and derive the unbiased causal estimate of joint interventions on the outcome.

The rest of this paper is organized as follows. Section 2 reviews background materials used in other sections. Section 3 describes the experimental designs in education. Section 4 describes the non-randomized designs with a single intervention in education. Section 5 describes the non-randomized studies with multiple interventions where we propose time-varying treatment-confounder feedback and many other models in education. Section 6 presents the conclusion.
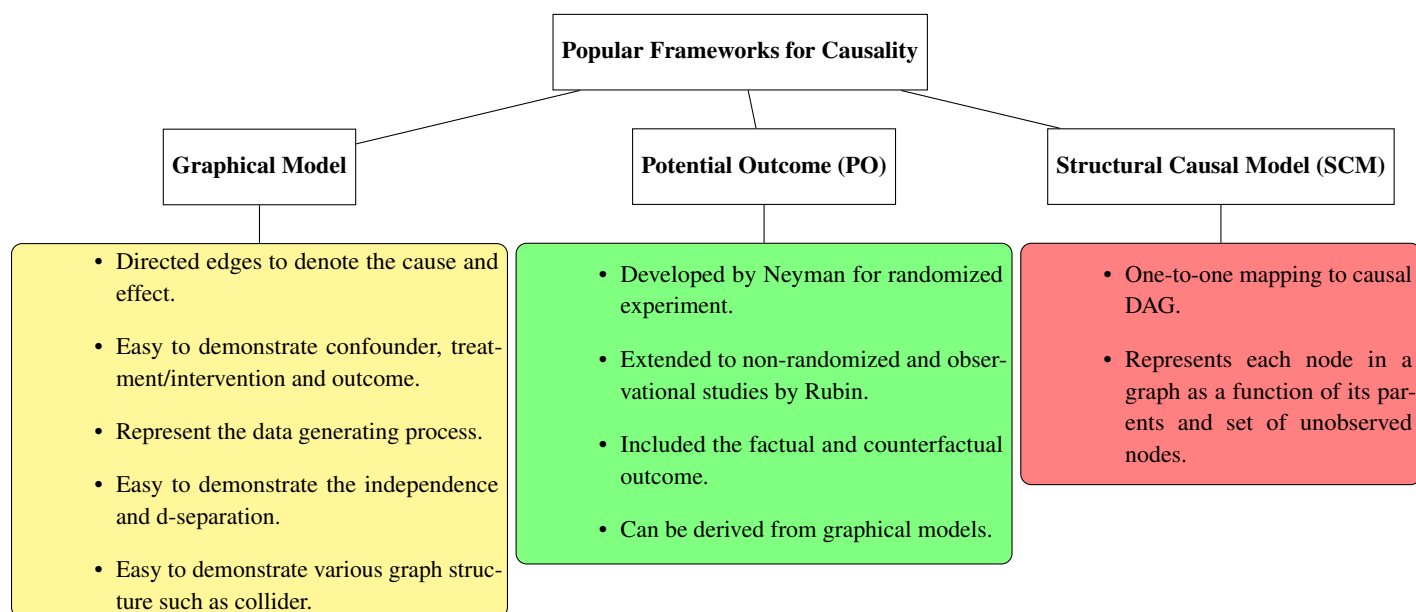
2   Background

Figure 1: Well-known Frameworks for Causality

Level (Symbol)	Typical Activity	Typical Questions
1. Association $P(y\|x)$	Seeing	What is? How would seeing X change my belief in Y?
2. Intervention $P(y\|do(x), z)$	Doing Intervening	What if? What if I do X?
3. Counterfactuals $P(y_x\|x', y')$	Imagining, Retrospection	Why? Was it X that caused Y? What if I had acted differently?

Figure 2: Pearl Ladder of Causation

2.1 Pearl Ladder of Causation

The Following are the rungs in Pearl's hierarchical ladder of causation:

- **Association**: This is the first layer in the ladder of causation corresponding to the first row of figure 2. This is where most of the current statistical literature lies. An example of association or correlation is the conditional probability of $P(y|x)$, which means the probability of obtaining y given we observe or see x.

- **Intervention**: This is the second layer in ladder of causation corresponding to the second row of figure 2. This is the layer responsible for the do calculus. An example of this is $P(y|do(x))$, which means the probability of obtaining y given we are forcing or intervening $X = x$.
  Note: $P(y|do(x))$ is commonly written as $P(y_x)$ (Potential outcome notation, explained later in this section).

- **Counterfactual**: This is the third layer in the ladder of causation corresponding to the third row of figure 2. The counterfactual output is always unobserved. For example, consider $x = 1$ denotes receiving the treatment and $x = 0$ denotes otherwise. Then $P(y_{x=1}|x = 0)$ is a counterfactual outcome since we are asking about the probability of outcome among the people who did not receive the treatment had we forced them to receive the treatment.

2.2 Potential Outcome (PO)

Y^x or Y_x is the outcome that would be observed if the treatment was set to X=x; in other words, it is the outcome under different possible treatment options. It consists of observed or factual and unobserved or counterfactual segments. Assume the case of binary treatments, where $X = 1$ denotes receiving the treatment and $X = 0$ denotes otherwise. $C = c$ denotes observing participants among those that are in group c. For example, in education, c can represent students who are in a specific class, have a certain GPA, etc. Then based on the definition of the PO and the counterfactual in the last section, we can define the following equations.

$$\text{Average Treatment Effect} = ATE = E[y_{x=1}] - E[y_{x=0}] \tag{1}$$

$$\text{Average Treatment Effect Among Treated} = ATT = E[y_{x=1}|X = 1] - E[y_{x=0}|X = 1] \tag{2}$$

$$\text{Average Treatment Effect Among Untreated} = ATU = E[y_{x=1}|X = 0] - E[y_{x=0}|X = 0] \tag{3}$$

$$\text{Conditional Treatment Effect} = CATE = E[y_{x=1}|C = c] - E[y_{x=0}|C = c] \tag{4}$$

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2.3 Graphical Model (Causal DAG)

In this framework, the nodes and variables are represented as vertices of the graph, and directed edges denote causal relationships between them. Besides having directed edges, there is no cycle or loop in a causal DAG. The benefit of using a DAG is to define a pictorial view of the problem, to represent the data generating process, and to demonstrate independence and d-separation clearly. For example, in figure 3, X is the treatment, Y is the outcome, and Z is the confounder (cause of both X and Y and is placed on the backdoor path between X to Y). In causal inference, we are usually interested in finding the average causal effect of treatment on outcome. If Z were not present, then the problem would be reduced to correlational studies such that causation and correlation would be the same.

In figure 3, the red path $X \leftarrow Z \rightarrow Y$ is called the backdoor path, which is a non-causal path pointing to a treatment X
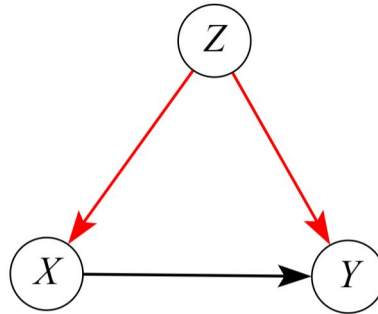

Figure 3: Graphical Representation of Treatment, Outcome and Confounder

and outcome Y. Our goal in causal inference is to block backdoor paths since they are non-causal paths and introduce bias in our analysis. To account for this backdoor path and determine unbiased causal estimate of X on Y we use the Pearl backdoor criterion as follows:

Backdoor Adjustment: If Z is a backdoor variable relative to X and Y then the causal effect of X on Y is calculated by:

$$P(Y|do(X)) = \sum_Z P(Y|X,Z)P(Z) \tag{5}$$

2.4 Structures in DAG

Figure 4 shows various independence structures on DAGs. Figures 4a and 4b are structures in which A and C are d-separated conditioned on B. However 4c denotes a collider structure, where A and C are independent but they will be dependent conditioned on B. Conditioning on B results in what is known as a collider stratification bias.

(a) $A \perp\!\!\!\perp C|B$

(b) $A \perp\!\!\!\perp C|B$
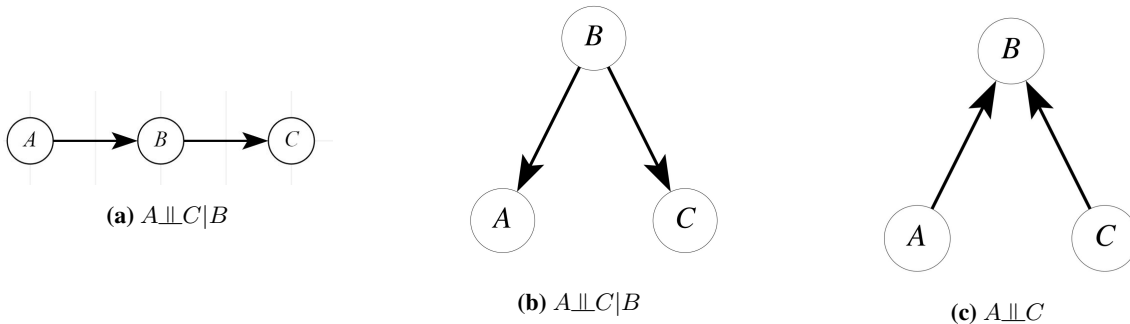
(c) $A \perp\!\!\!\perp C$

Figure 4: Various structures on a DAG

2.5 Pre-Intervention and Post-Intervention DAG

The pre-intervention DAG is a data generating process, or a DAG designed by an expert or given by the nature. The post-intervention DAG is a pre-intervention DAG after performing the intervention. This is done by removing the parents of the nodes we are doing an intervention on. For example figure 5a shows a pre-intervention DAG and 5b shows the post-intervention DAG after performing the intervention on X.

(a)

(b)

Figure 5: a) The Pre-Intervention DAG   **b)** The Post-Intervention DAG

2.6 Structural Causal Model (SCM)

The SCM is a system of structural equations relating each node in a DAG to their parents and the set of unobserved causes. It has a one-to-one mapping to graphical models. The following are the components of the SCM model:

- Variables/Nodes.
- Unobserved cause of nodes.
- Functions mapping each node to their parents.

For example, in figure 5, the following equations demonstrate the SCM for the pre (the left equation) and post (the right equation) intervention DAGs.

$$Y = f_Y(U_Y) \qquad\qquad Y = f_Y(U_Y) \tag{6}$$
$$X = f_X(Y, U_X) \qquad\qquad X = x \tag{7}$$
$$Z = f_Z(X, Y, U_Z) \qquad\qquad Z = f_Z(x, Y, U_Z) \tag{8}$$
$$U_X \perp\!\!\!\perp U_Y \perp\!\!\!\perp U_Z \tag{9}$$

2.7 Identifiability Condition

- Well-Specified Variables.
  - All variables should be properly defined.
  - The treatment, outcome and measured confounders should be correctly specified.
- Positivity.
  - No deterministic treatment within levels of the confounder. For example, in figure 3, for positivity to hold we need:

$$P(X = x | Z = z) > 0 \qquad \text{If} \quad P(Z = z) > 0 \tag{10}$$

If the equation 10 is violated then $E(Y|X, Z)$ is not defined. This is because:

$$E(Y|X, Z) = \sum_y y \, p(y|X, Z) = \sum_y y \frac{p(y, x, z)}{p(x, z)} = \sum_y y \frac{p(y, x, z)}{p(x|z)p(z)} \tag{11}$$

since the numerator of equation 11 will be zero.

- Consistency.

  - For those who receive $X = x$ then $Y_x = y$.
  - For those who receive $X = x^\star$ then $Y_{x^\star} = y$.
    For example using consistency, $E[Y_x]$ can be written as follows:

$$E(Y_x) = \sum_x E(Y_x|X = x)p(x) = E(Y_x|X = x)p(x) + E(Y_x|X = x^\star)p(x^\star) \tag{12}$$

$$= E(Y|X = x)p(x) + E(Y_x|X = x^\star)p(x^\star) \tag{13}$$

- Conditional Exchangeability.

  - Conditional exchangeability states that the potential outcome is independent of the treatment given the sufficient set of confounders. Mathematically this is represented as follows:

$$Y_X \perp\!\!\!\perp X | Z \tag{14}$$

    Conditional exchangeability states if you account for sufficient confounders then treatment and control groups are the same.

  - Z in equation 14 is sufficient for controlling any non-causal path.
  - It states that the treated and untreated groups are similar conditioned on the set Z.

- Defining Sources of Bias.

  - No measurement error.
  - No selection bias (loss-to-follow-up, etc).

- Well-specified Model.
  Define a valid statistical model for inference and testing. Make sure the statistical model can match the problem requirements in terms of amount of data, number of parameters, nature of data (whether data is cross-sectional or longitudinal), etc.

2.8 Internal and External Validities

External validity is the ability to generalize the findings to other populations and settings. Lack of external validity suggests that the outcome cannot be related to other people or contexts outside the current study. For example, consider studies where the participants are all male or students who all have perfect GPA, then the findings cannot be generalized to the entire population.
Table 1 summarizes some well-known threats to external validities.

Table 1: Threats to External Validity

Threats	Definition
Participation Characteristics	Participants should be a good representative (sample) of entire population.
Experiment Setting	How similar is the experiment setting to real-world.
Timing	Study over the past might not be the same as now.

Internal validity is defined as the degree to which the causal relationship between dependent and independent variables can be established. Internal validity is trying to answer the following questions: Is the independent variable the only cause of the change in the dependent variable? Have we considered all the confounders in the study? Table 2 indicates some well-known threats to internal validity according to [14].

Table 2: Threats to Internal Validity

Threats	Definition
Ambiguous Temporal Precedence	Lack of clarity between which variable occurs first.
Regression	Problem of regression to the mean.
Confounding Variables	Something other than treatment is causing the change in outcome.
History	External events that affect how participants responds.
Maturation	Natural process in which participants change during the course of study.
Instrumentation	An instrument or a measure in a study change over the course of study .
Testing/Practice Effects	Repetition of test to enhance the performance.
Attrition/Mortality	Participants dropping out during the course of study.

3 Experimental Designs in Education

Experimental designs are designs where the researcher has complete control over the study. Their main characteristic is randomization, where the participants are assigned to control and treatment groups randomly. Perhaps the most well-known type of experimental design is the randomized controlled trial (RCT). It assigns the participants to the control and the treatment groups randomly. Hence, it will remove the confounding variables from the study. The ideal RCT (full compliance) is considered the gold standard in medicine; however, in practice, participants might leave the study or do not comply with the treatment, which creates a bias in the study. Figure 6a demonstrates a candidate DAG for the RCT Under the non-compliance where R is the instrument and denotes the randomization to receive the treatment, X is whether you actually receive the treatment, L and W are the confounders (They may be observed or unobserved), and Y is the outcome. For example, in education, X can be either solving some problems, reading sections of a book, or getting a referral to an expert; L and W are either the observed or unobserved confounders such as family issues, socioeconomic status, prior knowledge and stress, and psychological problems. Y denotes the outcome of interest, for example, the exam score. In figure 6a X is a collider, hence conditioning on X will open the non-causal path $R \rightarrow X \leftarrow L \rightarrow Y$ and $R \rightarrow X \leftarrow W \rightarrow Y$.

Figure 6b shows the same DAG but an ideal RCT, where the effect of confounding variables is removed. In figure 6b, R and X are the same, since if you are randomized to receive the treatment, then you will receive the treatment (Full-compliance)

However, even if the RCT is done with full compliance, it still suffers from the following limitations:

- It is expensive. In education usually funding is limited, which makes the RCT not practical as a tool for widespread use.

- It requires careful planning and correct strategy to keep participants in the study and perform the correct randomization.

- It is unfeasible and unethical in many situations. For example, you may not be able to force students talk to the TA or visit a counselor.

Sequentially Multiple Assignment Randomized Trial (SMART) [16] is another type of experimental design that consists of randomization at each stage. Figure 7 shows a proposed SMART design in education, where initially students are randomly assigned to three groups based on interventions they receive:1- Students who are recommended to solve some problems (SP), 2- Students are recommended to talk to the TA (TT). 3 - Students who are recommended to talk to a counselor (TC). Students then continue with the intervention for some duration, such as five weeks, and are assessed after that. Those who respond to treatment, meaning that treatment is effective for them, continue with this treatment, and those who are non-respondent will be randomized again to students who receive the augmented treatments, namely combining the previous treatment with the new one and students who receive the previous treatment with the higher intensity. This process may continue until a certain mastery level for all students is achieved or until the duration of study is over. Figure 7 shows a two-stage SMART design with three treatment options at the first stage.
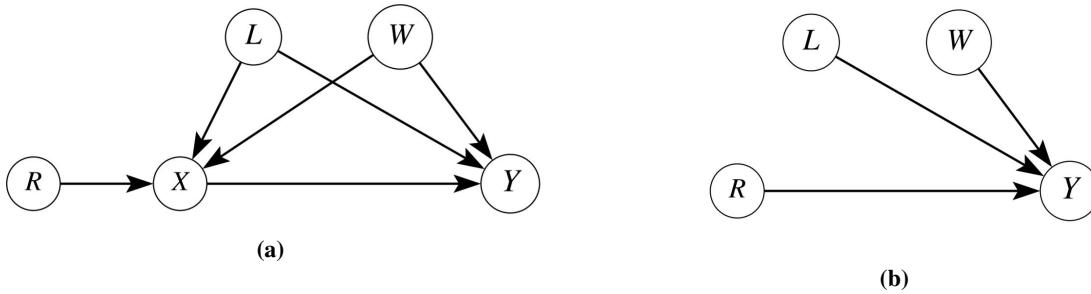
Figure 6: a) DAG for a RCT with non-compliance. b) DAG for RCT with full compliance
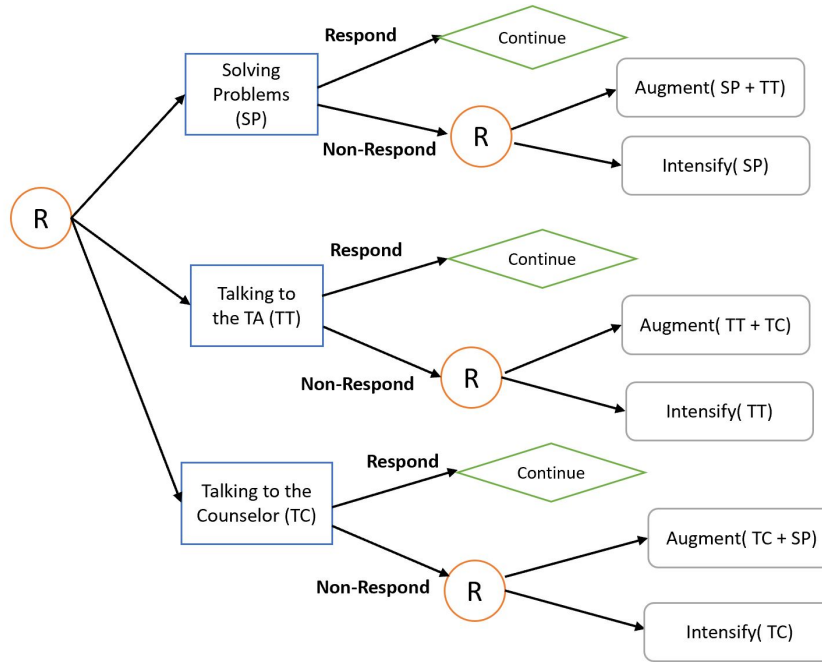

Figure 7: SMART data collection design in education

Since SMART is a multi-stage randomization design, then similar to the RCT, it is costly and requires a careful planning. Therefore it may not be a practical option for educational design. In the next sections, we will discuss non-randomized (quasi-experimental) designs in education.

4 Non-randomized Designs with a Single Intervention

Non-randomized or quasi-experimental designs are designs used when the randomized experiment is not possible due to ethical, timing, or cost restrictions. They are sometimes also referred to as observational studies. [17].
Figure 8 represents some well-known quasi-experimental designs in literature:
 In [14], the authors introduced a four levels of hierarchy for quasi-experimental designs demonstrated in table 3. In Table 3, D is the best design, followed by C, B and A. Next, we discuss the use of designs in table 3 in educations.
Note: The common notation is to use X for intervention, O for measurement (observation), and the NR for non-randomized assignment.The position from left to right determines the temporal order.
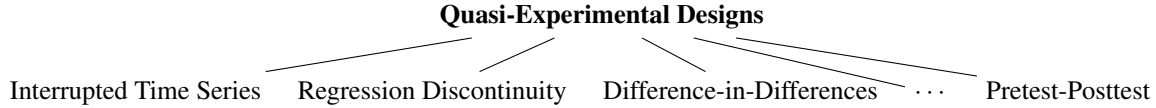
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Quasi-Experimental Designs

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Interrupted Time Series   Regression Discontinuity   Difference-in-Differences $\cdots$ Pretest-Posttest

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Figure 8: Some Well-Known Quasi-Experimental Designs

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Table 3: Hierarchy of Quasi-Experimental Design

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Type of Experiment
A. Design with no control groups.
B. Design with control groups but no pretest.
C. Design with control groups and pretest
D. Design for time series data.

4.1 Designs with no control groups

1. **The One group posttest design:**

$$X \qquad O_1$$

Figure 9 shows an example of a DAG for this scenario. X is a treatment or an intervention, O_1 is an outcome or the posttest, W_1 is observed, and U_1 and U_2 are unobserved confounders. For example, suppose a researcher is interested in finding the causal effect of doing some problem sets in a textbook on student learning by taking a final exam or posttest immediately after the study ends. Then X is doing the problem sets, and the O_1 is the final exam. This design is subject to many threats to internal validities such as history since other events besides the treatment might cause the change in exam score. Confounding variables such as prior knowledge of the topic, intelligence, family, and psychological problems (unobserved confounder) might cause a change in outcome. Lack of pretest does not let us query students' prior knowledge and the absence of a control group, makes it difficult to determine if treatment is the only cause of change in the outcome. To better understand the problem with this design, consider the DAG in figure 9; ideally, we would like to find the direct causal effect of X on O_1; however, the $X \leftarrow W_1 \rightarrow O_1$ is a non-causal backdoor path, which should be closed. But, even blocking the backdoor path through W_1 still is not sufficient due to the backdoor path via unobserved confounders.

To better understand the problem with this design consider the following simulation. The DAG for the data generating process is the same as figure 9. The treatment and all the confounders (observed and unobserved) are Bernoulli random variables and the outcome O_1 is normally distributed with mean to be the linear combination of X, U_1, U_2 and W_1 and the standard deviation is 0.6.

Simulation Setup

$$
\begin{aligned}
N &= 500 \\
P(W_1 = 1) &= 0.8 \\
P(U_1 = 1) &= 0.4 \\
P(U_2 = 1) &= 0.6 \\
P(X = 1) &= 0.30 + 0.2U_1 + 0.5U_2 - 0.3W_1 \\
\mu &= 5 + 7X + 4W_1 - 2U_1 - 2U_2 \\
\sigma &= 0.6 \qquad O_1 \sim N(\mu, \sigma^2)
\end{aligned}
$$

Table 4 presents simulation results for this design. According to this table, the causal effect of X on O_1 will be biased if we do not control for unobserved confounders.
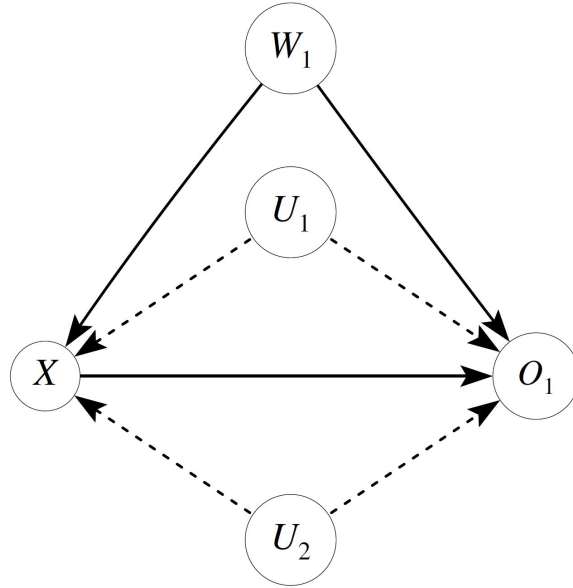
Figure 9: DAGs for a single posttest design

Table 4: Simulation Result for a design with a single intervention

Control for No Confounders		
Coefficient	True	Estimate
Intercept	5.0	7.13
X	7.0	5.04
Control for W_1		
Intercept	5.0	3.88
X	7.0	5.58
Control for W_1 and U_1		
Intercept	5.0	4.27
X	7.0	6.55
Control for W_1, U_1 and U_2		
Intercept	5.0	4.99
X	7.0	7.03

2. **The One-Group Pretest-Posttest Design:**

$$O_1 \qquad X \qquad O_2$$

Figure 10a is a candidate DAG for this case, where the outcome O_2 is the posttest score (It can be a continuous or discrete quantity). U_1 and U_2 are unobserved confounders and can refer to psychological and family problems. W_1 represents any observed confounder. This is similar to the DAG in figure 9. This is because, in causal inference, we study the causal effect of intervention/treatment on the outcome and the pretest-posttest design has similar characteristics as one-group posttest design after the intervention. Including a pretest, however, can provide some information about the student's prior knowledge, which in turn helps us to control for some confounders compared to the first design. However, repeating the same problems in the consecutive exams results in testing/practice effects that threatens internal validity. This design is also susceptible to other threads to internal validities such as maturation and history. This is because participants may drop out during the study (for example, after taking the pretest). This is sometimes referred to as a loss-to-follow-up, which causes the selection bias. This design also suffers from regression

to the mean since if there is only one observation before the intervention (the pretest), then the score in the pretest can be due to chance. Therefore, the pretest would be a very weak counterfactual had the treatment never happened. Figure 10b is another candidate DAG, which includes the pretest O_1 and L_1 as a covariate causing students to drop from the study. C_1 is a binary variable that represents the censoring where $C_1 = 0$ students are not censored and continue the study.
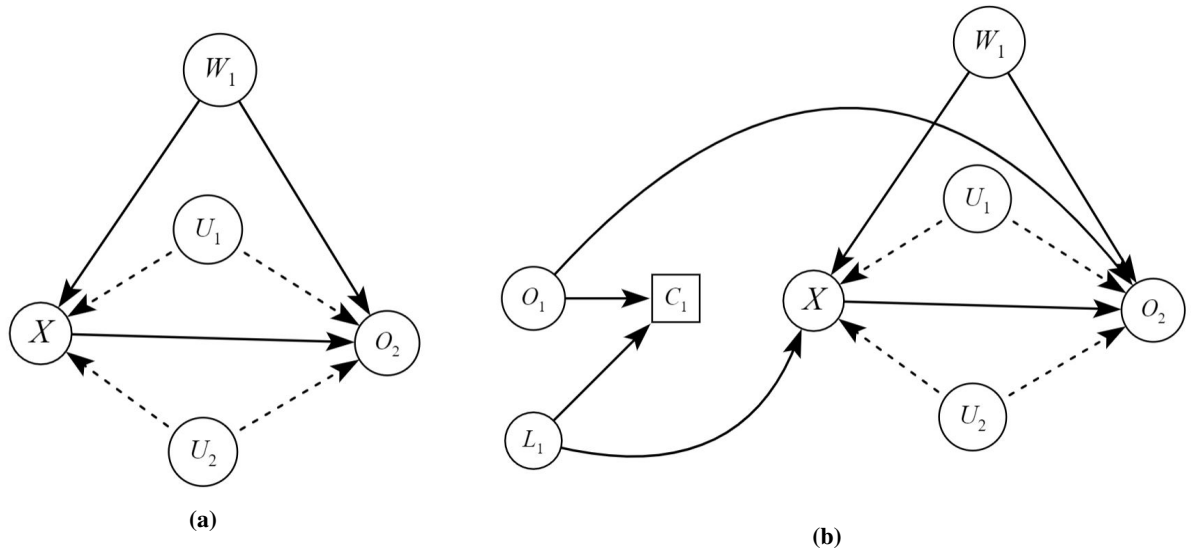

(a)

(b)

Figure 10: DAGs for a pretest-posttest Design

4.2 Designs with a control group but no pretest

3. **Posttest-Only Design With Nonequivalent Groups:**

NR		X	O_1
NR			O_1

This is similar to a one-group posttest design with the difference of adding a control group. The control group is supposed to provide a counterfactual outcome for the treated group had they not received the treatment. The DAG for this is demonstrated in figure 11 and is similar to the DAG in figure 9 with the inclusion of variables Z. Z is a variable indicating the treatment assignment. It determines the conditions on how to assign the participants to treatment and control groups, such as choosing some classrooms for the treatment and some others for the control group. One major problem with the nonequivalent design is the selection bias since the participants are not assigned randomly. Therefore, the participants in the control and treatment groups do not share identical characteristics. For example, assume participants in treatment groups are from more educated family backgrounds, or they are academically stronger candidates. Therefore they perform better in the posttest even under the null treatment effect.

4.3 Designs with both control groups and pretests

4. **The Pretest-Posttest Design with Nonequivalent Groups:**

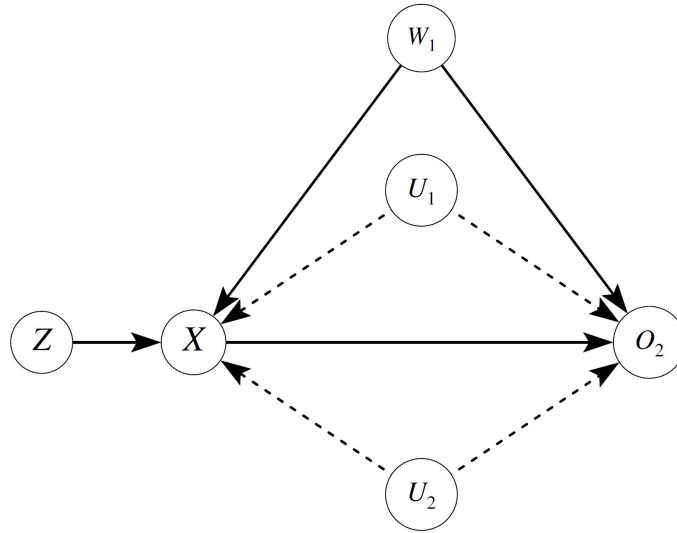| NR		O_1	X	O_2
NR		O_1		O_2

11

Figure 11: The DAG for Posttest-Only Design With Nonequivalent Groups

This is a common design in the educational systems. Students are divided into treatment and control groups based on some factors, such as the classroom. The DAG after the intervention for this design is similar to figure 11. The control and pretest provide a counterfactual for the treated group and help to remove some of the threats to the internal validities. However some flaws with this design are as follows: 1- A single intervention may not be sufficient to influence the outcome (posttest). 2- Since the participants are not randomly assigned, the treatment and control groups may have very different characteristics (confounder). 3- A single observation before and after the intervention is not enough to draw any conclusion about the participants' mastery of the subject and is prone to regression to the mean threats. 4- Since there is a single intervention then there is no opportunity to provide feedback to students.

4.4 Design for time series data

5. **Interrupted Time series Design**

$$O_1 \qquad O_2 \qquad ... \qquad O_n \qquad X \qquad O_{n+1} \qquad . . . \qquad O_{2n}$$

Interrupted time series (ITS) design consists of repeated measurements over time with an intervention, or a shock is introduced at a certain time. It is considered the most powerful design in the hierarchy of quasi-experimental designs. This is because it can rule out many threats to internal validity such as regression to the mean since many measurements are taken before and after the intervention; therefore, the probability that the result is only due to chance is very slim. However, history and instrumentation are still considered as treats to internal validities since due to administrative change, the measure for student learning can change through time. Attrition is a big threat for this design since the loss-to-follow-up can happen after each measurement. Therefore the number of participants from the beginning to the end of the study can be very different. Figure 12 shows a candidate DAG for this design for four measurements (2-time points before and 2-time points after the intervention). The $O_1, O_2, . . ., O_4$ denote the measurements, $L_1, L_2, . . ., L_4$ are the time-varying covariates such as family, financial or prerequisite problems that influence the outcome and treatment. It is important to note that figure 12 is a simplified DAG for most real-world phenomena.

Interrupted time series (ITS) is a type of design used when a randomized experiment is either too expensive or impractical. ITS measures the change in intercept and slope of the time series before and after the intervention. It comprises two distinct periods, namely the pre-intervention and the post-intervention, and may include the control group. There are two major methods to model the ITS design. The segmented regression by far is the most widely used one, in which a linear regression model is fit for before and after intervention with a dummy variable representing the
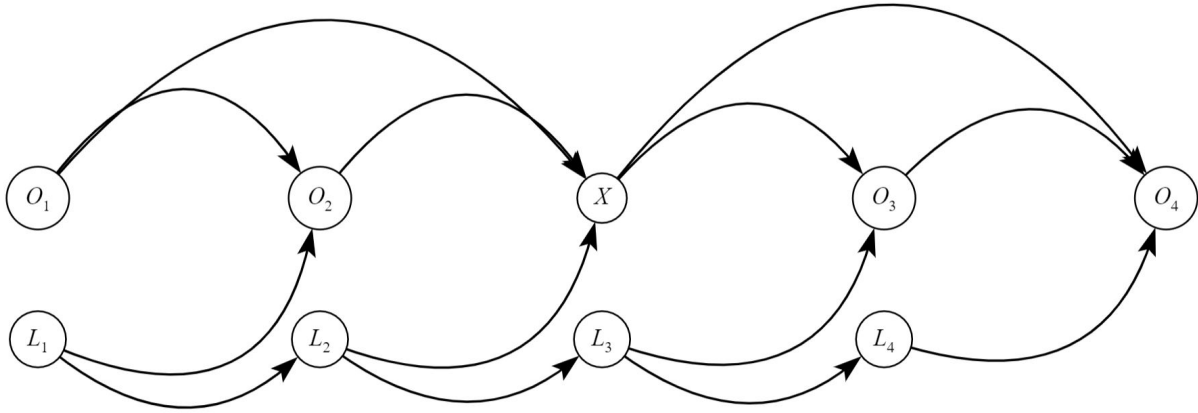
Figure 12: A DAG for the Interrupted Time Series Design

pre and post-intervention phase. This model assumes a linear trend before and after intervention time [18], [19],and [20]. The segmented regression technique assumes the residual term to be normally distributed, but if residuals are autocorrelated, then autoregressive integrated moving average (ARIMA) can be used to model the ITS [18], [21], and [22]. This method works by first building the ARIMA model of the time series using only the pre-intervention series. Then apply the model for the complete time series data and add the intervention as a dummy variable, where 1 is coded as the post-intervention phase, and 0 is for the baseline period. ARIMA only handles the univariate time series data. ITS can be used in education to evaluate the influence of a single intervention introduced in a semester as a form of a study skill or measure the influence of adding an online textbook.

4.5 Regression Discontinuity (RD)

C	O_1	X	O_2
C	O_1		O_2
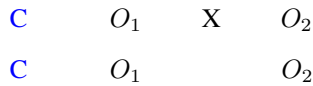
RD is another type of quasi-experimental design used when there is some kind of criterion (threshold) that must be met before participating in the intervention. This threshold identifies the eligibility for participation in the program.
It is a type of pretest-posttest comparison group, where participants are assigned to treatment and comparison groups based on the threshold or cutoff on a pretest. This can be represented mathematically as follows: A is called the running variable and C is the threshold. If A is above the threshold then you receive the intervention.

$$X = \begin{cases} 1 & A \geq C \\ 0 & A < C \end{cases}$$

Figure 13 represents a candidate DAG for RD design. According to this figure, A, L, and U are confounders (U is an unobserved confounder). and $X \leftarrow A \leftarrow L \rightarrow O$, $X \leftarrow A \leftarrow U \rightarrow O$, and $X \leftarrow A \rightarrow O$ are the backdoor paths. Therefore, one way to obtain the unbiased estimate of X on O is to control for A. In RD, we consider the effect of the intervention on the outcome when the running variable (A) is near the threshold (condition on A). Hence, by doing this, we will close all the backdoor paths.
In education, A can be a pretest, X is the intervention or treatment such as talking to a TA or a tutor, O is the posttest, L is the observed confounder such as the prerequisite knowledge, U is the unobserved confounder such as family background, and C is the threshold on the pretest for participation in the program. In RD, we study the influence of the intervention on the posttest score for those participants whose pretest score is near the threshold.
To analyze the RD for the above problem in education, let A denote the pretest score,O denote the posttest score, and Z

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be a binary dummy variable indicating the treatment or control group. Then select the participants whose pretest score is just below and above the threshold, and regress the posttest score on the pretest and other variables.

$$O = \beta_0 + \beta_1 A + \beta_2 Z + \beta_3 AZ + \epsilon \tag{15}$$

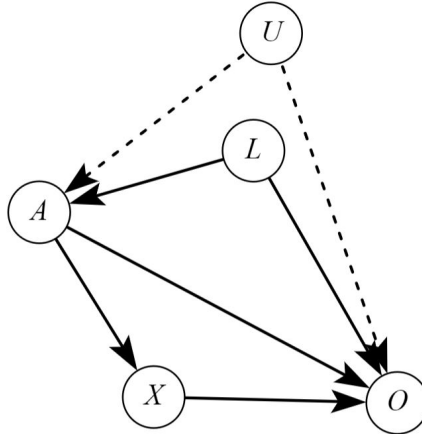The β_3 coefficient is the causal effect of A on O.

Figure 13: A DAG for Regression Discontinuity Design

Other popular quasi-experimental designs are Difference-in-differences (DID) and instrumental variable (IV) that can provide unbiased causal estimates when a randomized experiment is not possible [23],[24], [25],[26], [27], and [28]. Although quasi-experimental designs such as ITS, RD, DID, and IV provide unbiased causal estimates for many real-world phenomena, they may not be suitable options for education since 1- They mainly model a single intervention yet, learning is more effective using a sequence of interventions. 2- They do not model time-varying treatment and confounder feedback; therefore, they do not incorporate any mechanisms to reduce the negative effect of confounders through timely intervention.

Our objective is to have a tractable approach for the actual educational system by accounting for all the potential confounders and design suitable interventions to reduce the negative influence of confounders through time. Thus, in the next section, we discuss observational studies in education and introduce techniques to overcome limitations of quasi-experimental designs with a single intervention.

5 Non-randomized Designs with Multiple Interventions in Education

Before discussing non-randomized designs with time varying interventions, We first define parameters and notation used in this section:

- $X_1, X_2, \ldots X_T$ are time varying treatments, such as:
  - Reading chapter of book.
  - Solving some problems.
  - Talking to the TA/instructor/counselor.
- $L_1, L_2, \ldots L_T$ are observed and $U_1, U_2, \ldots U_T$ are unobserved time varying confounders, such as:
  - Prior knowledge of subject.
  - Psychological/stress problems.
- O is the outcome. such as exam score.
- N is the total number of students.

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The **Objective** is to find the causal effect of joint interventions $X_1, X_2, \ldots, X_T$ on O. The Joint effect of interventions is defined as a collection of direct effects unmediated by other interventions [29]. For example, joint effect of interventions X_1 and X_2 on O in figure 14 consists of $X_1 \rightarrow O$ and $X_2 \rightarrow O$. In this section, we consider the following three cases, which characterize scenarios happening in real-world educational systems.
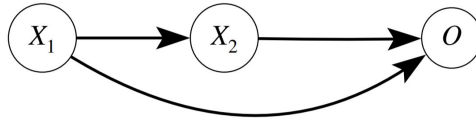

Figure 14: Joint Effect of X_1 and X_2 on O

1. Time-Varying Treatment and Confounder with no unmeasured confounders.

2. Time-Varying Treatment and Confounder with unmeasured confounders.

3. Time-Varying Treatment-Confounder feedback.

5.1 Time Varying Treatments and Confounders With No Unmeasured Confounders

Figure 15 presents an example of this scenario, where X_1, X_2 and X_3 are referred to as time-varying treatments/interventions. They can represent solving problems, talking to the TA, and talking to the counselor. L_1, L_2, and L_3 are observed confounders such as student background knowledge through time. O is the posttest exam. We use the outcome regression model to find the average causal effect of joint intervention X_1, X_2, and X_3 on O. The outcome regression measures the correlation coefficients but not necessarily the causal coefficients. Therefore we need to investigate if the correlation and causation are the same in equation 16. Controlling for L_1, L_2 and L_3 will close all the backdoors such as $X_1 \leftarrow L_1 \rightarrow O$, $X_2 \leftarrow L_2 \rightarrow O$, and $X_3 \leftarrow L_3 \rightarrow O$. Furthermore controlling for X_1, X_2 and X_3 will block the mediated and all other backdoor paths such as $X_2 \leftarrow X_1 \rightarrow O$. Hence the outcome regression in equation 16 results in unbiased estimates of causal coefficients. In equation 16, the multiplication terms are interaction terms, for example $X_1 X_2$ suggests that the causal effect of X_1 on O is different for different values of X_2.
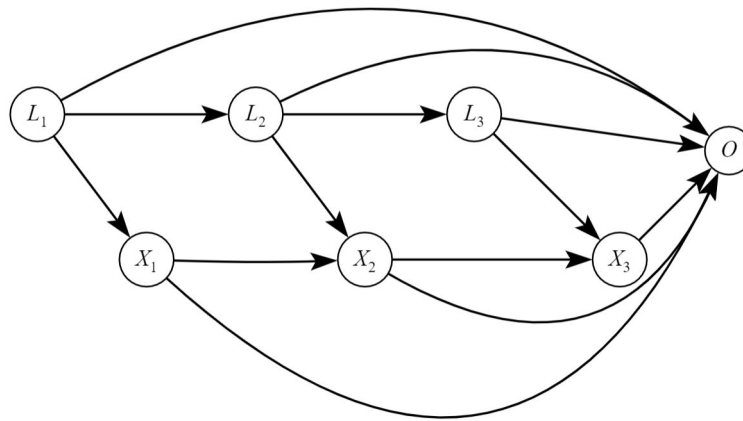

Figure 15: Time Varying Treatment and Confounder for three time points

$$E[O|X_1, X_2, X_3, L_1, L_2, L_3] = \alpha_0 + \alpha_1 X_1 + \alpha_2 X_2 + \alpha_3 X_3 + \alpha_4 X_1 X_2 +$$
$$\alpha_5 X_1 X_3 + \alpha_6 X_2 X_3 + \alpha_7 X_1 X_2 X_3 + \alpha_8 L_1 + \alpha_9 L_2 + \alpha_{10} L_3 \qquad (16)$$

The following is a simulation setup for this case:

$$
\begin{aligned}
N &= 500 \\
P(L_1 = 1) &= 0.5 \\
P(X_1 = 1) &= 0.2 + 0.4L_1 \\
P(L_2 = 1) &= 0.2 + 0.6L_1 \\
P(X_2 = 1) &= 0.3 + 0.5X_1 + 0.2L_2 \\
P(L_3 = 1) &= 0.3 + 0.5L_2 \\
P(X_3 = 1) &= 0.3 + 0.4X_2 + 0.1L_3 \\
\mu &= 0.2 + 2X_1 + 6X_3 + 5X_2 + X_1X_2 + X_1X_3 + X_2X_3 + 4L_1 + 4L_2 + 3L_3 \\
\sigma &= 0.2 \quad O \sim N(\mu, \sigma^2)
\end{aligned}
$$

N represents number of cases, confounders and treatments are all binary variables and the posttest is normally distributed. Table 5 shows the simulation result for this scenario. It indicates all the coefficients except $X_1X_2X_3$ are significant for P-value of 0.05. Therefore the coefficient of $X_1X_2X_3$ may be assumed to be zero and the regression model estimates all coefficients correctly. Table 5 only includes the coefficients for treatments and their interaction terms and not any information about the confounders. Table 6 shows the 95% confidence interval (CI) for the coefficients using bootstrapping. According to this table the true values for all the coefficients lie within the 95% confidence interval.

Table 5: Simulation Result for Time-Varying Treatments, Confounders with No Unmeasured Confounders

Coefficient	True	Estimate	P-Value
Intercept	0.2	0.17	0.0008
X_1	2	1.90	$2x10^{-16}$
X_2	5	4.98	$2x10^{-16}$
X_3	6	6.02	$2x10^{-16}$
X_1X_2	1	0.91	$2x10^{-7}$
X_1X_3	1	1.34	$9.6x10^{-7}$
X_2X_3	1	0.90	$1.9x10^{-7}$
$X_1X_2X_3$	0	-0.091	0.81

Table 6: Simulation Result for Time-Varying Treatments, Confounders with No Unmeasured Confounders

Coefficient	True	95% CI
Intercept	0.2	$(0.0702, 0.2826)$
X_1	2	$(1.710, 2.302)$
X_2	5	$(4.875, 5.336)$
X_3	6	$(5.826, 6.180)$
X_1X_2	1	$(0.4688, 1.2230)$
X_1X_3	1	$(0.755, 1.918)$
X_2X_3	1	$(0.5542, 1.1484)$
$X_1X_2X_3$	0	$(-0.8461, 0.4669)$

The results in this section suggest that if all the confounders are measured properly, then the unbiased causal effect of joint interventions on the outcome can be estimated accurately. Next, we investigate what would happen if there are unmeasured confounders.

5.2 Time Varying Treatments and Confounders With Unmeasured Confounders

Figure 16 presents an example of this scenario, where X_1, X_2 and X_3 are referred to as time varying treatments/interventions. Similar to the figure 15 interventions can represent solving problems, talking to the TA, and talking to the counselor. L_1, L_2 and L_3 are observed confounders such as student background knowledge through time. U_1, U_2 and U_3 are unobserved confounders such as family and psychological problems. O is the posttest exam. To find the average causal effect of joint intervention X_1, X_2, and X_3 on O, we use the same regression model as in previous case with all observed confounders. We need to investigate if the correlation and causation are the same in equation 17. Controlling for L_1, L_2 and L_3 will close backdoors such as $X_1 \leftarrow L_1 \rightarrow O$, $X_2 \leftarrow L_2 \rightarrow O$, and $X_3 \leftarrow L_3 \rightarrow O$. Furthermore controlling for X_1, X_2 and X_3 will block the mediated and all other backdoor paths such as $X_1 \leftarrow X_2 \rightarrow O$. However any backdoors through unobserved confounders such as $X_1 \leftarrow U_1 \rightarrow O$ and $X_2 \leftarrow U_2 \rightarrow O$ remain open. Hence the outcome regression in equation 17 results in biased estimate of causal coefficients. Note equations 16 and 17 are the same since unobserved variables cannot be included in the regression model.
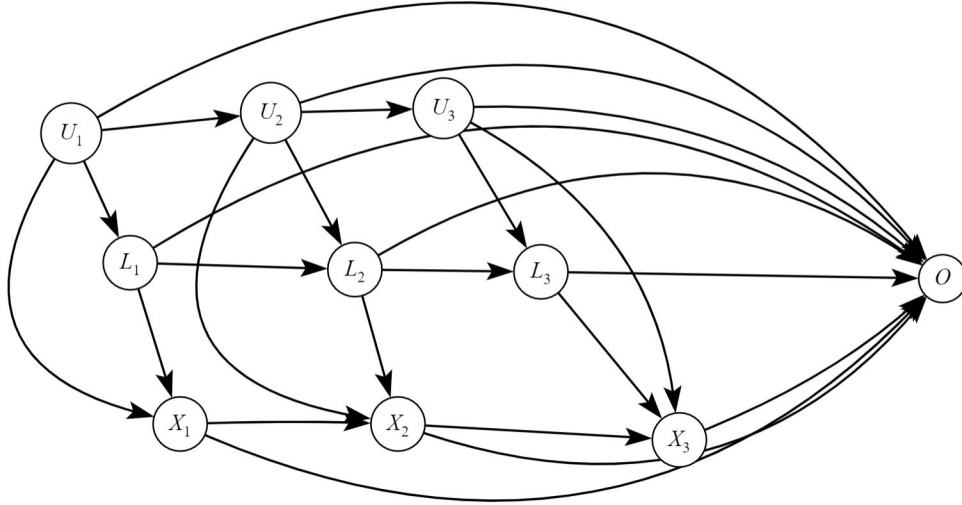

Figure 16: Time Varying Treatment and Confounder for three time points
with Unobserved Confounders

$$E[O|X_1, X_2, X_3, L_1, L_2, L_3] = \alpha_0 + \alpha_1 X_1 + \alpha_2 X_2 + \alpha_3 X_3 + \alpha_4 X_1 X_2 +$$
$$\alpha_5 X_1 X_3 + \alpha_6 X_2 X_3 + \alpha_7 X_1 X_2 X_3 + \alpha_8 L_1 + \alpha_9 L_2 + \alpha_{10} L_3 \quad (17)$$

The following is a simulation setup for this case:

$N = 500$

$P(U_1 = 1) = 0.6$ $\qquad\qquad$ $P(U_2 = 1) = 0.6 + 0.3 U_1$

$P(U_3 = 1) = 0.4 + 0.3 U_2$ $\qquad\qquad$ $P(L_1 = 1) = 0.5 + 0.4 U_1$

$P(X_1 = 1) = 0.2 + 0.4 L_1 + 0.3 U_1$ $\qquad\qquad$ $P(L_2 = 1) = 0.2 + 0.6 L_1 + 0.1 U_2$

$P(X_2 = 1) = 0.3 + 0.2 X_1 + 0.2 L_2 + 0.2 U_2$ $\qquad\qquad$ $P(L_3 = 1) = 0.3 + 0.5 L_2 + 0.1 U_3$

$P(X_3 = 1) = 0.1 + 0.4 X_2 + 0.2 L_3 + 0.2 U_3$

$\mu = 4 L_1 + 4 L_2 + 3 L_3 + 5 X_2 + 2 X_1 + 6 X_3 + X_1 X_2$

$X_1 X_3 + X_2 X_3 + 2 U_1 + 3 U_2 + U_3 + 0.2$

$\sigma = 0.2 \qquad O \sim N(\mu, \sigma^2)$

Table 7 shows the simulation result for this case. According to this table the coefficients of X_1X_2, X_1X_3, X_2X_3, and $X_1X_2X_3$ are not statistically significant for P-value of 0.05. This is not correct since we know the coefficients of X_1X_2, X_1X_3, X_2X_3 should not be zero. Table 8 shows the 95% confidence interval (CI) for the coefficients using bootstrapping. According to this table, the 95% confidence interval for the intercept, X_2 and X_3 do not contain the true values. Moreover, the confidence interval, in this case, is wider than the confidence interval when all the confounders are observed.

Table 7: Simulation Result for Time-Varying Treatments
, Confounders with Unmeasured Confounders

Coefficient	True	Estimate	P-Value
Intercept	0.2	1.92	10^{-11}
X_1	2	2.97	$2x10^{-11}$
X_2	5	6.15	$2x10^{-16}$
X_3	6	7.01	$2x10^{-16}$
X_1X_2	1	1.24	0.056
X_1X_3	1	0.55	0.42
X_2X_3	1	0.16	0.80
$X_1X_2X_3$	0	0.37	0.67

Table 8: Simulation Result for Time-Varying Treatments
, Confounders with Unmeasured Confounders

Coefficient	True	95% CI
Intercept	0.2	$(1.844, 2.967)$
X_1	2	$(1.657, 3.638)$
X_2	5	$(5.19, 7.062)$
X_3	6	$(5.486, 8.419)$
X_1X_2	1	$(-0.828, 3.084)$
X_1X_3	1	$(-0.212, 3.917)$
X_2X_3	1	$(-1.4414, 2.6802)$
$X_1X_2X_3$	0	$(-3.3632, 1.8823)$

The accuracy of the estimated coefficient depends on the influence of unobserved confounders. As an example, consider the following two case studies where the simulation setup is the same as before except the mean of the outcome μ varies as a function of unobserved confounders.

Case I : **Unobserved confounders have small influence on the outcome**:

$$\mu_1 = 4L_1 + 4L_2 + 3L_3 + 5X_2 + 2X_1 + 6X_3 + X_1X_2 + X_1X_3 + X_2X_3 + 0.1U_1 + 0.2U_2 + 0.3U_3 + 0.2$$

$$\sigma_1 = 0.2$$

$$O_1 \sim N(\mu_1, \sigma_1^2)$$

Case II : **Unobserved confounders have huge influence on the outcome**:

$$\mu_2 = 4L_1 + 4L_2 + 3L_3 + 5X_2 + 2X_1 + 6X_3 + X_1X_2 + X_1X_3 + X_2X_3 + 8U_1 + 9U_2 + 10U_3 + 0.2$$

$$\sigma_2 = 0.2$$

$$O_2 \sim N(\mu_2, \sigma_2^2)$$

Table 9 summarizes the result for these two case studies and clearly demonstrates the negative effect of unobserved confounders on the estimated coefficients. Under case II, the estimated coefficients are far from the true coefficients, and this is due to the large influence of open backdoor paths.

Table 9: Simulation Result for Time-Varying Treatments, Confounders with Unmeasured Confounders (Comparison of Case I and II)

Coefficient	True	Estimate (Case I)	Estimate (Case II)
Intercept	0.2	0.41	8.97
X_1	2	2.08	7.17
X_2	5	5.12	7.91
X_3	6	5.84	10.05
X_1X_2	1	1.22	0.64
X_1X_3	1	1.34	1.98
X_2X_3	1	1.84	0.04
$X_1X_2X_3$	0	0.44	-1.58

The results in this section suggest that open backdoor paths due to unobserved confounders can cause bias in the estimation of the causal coefficients. The amount of bias depends on how large is the influence of the unobserved confounders. This is another important conclusion in designing more intelligent and individualized educational systems that we need to measure and control for as many confounders as we can. It is true that no matter how many confounding variables we measure, there are still some unobserved confounders; however, if we can close for the confounding variables that might plausibly have a large influence on the outcome, then we reduce the bias in the causal coefficients significantly (refer to the case I and case II). In the next section, we discuss time-varying treatments, confounders feedback.

5.3 Time Varying Treatments and Confounders Feedback

Figure 17 presents an example of this scenario, where X_1, X_2 and X_3 are referred to as time-varying treatments/interventions. Similar to the previous two sections. interventions can represent solving problems, talking to the TA, and talking to the counselor. L_1, L_2, and L_3 are observed confounders such as student background knowledge through time. O is the posttest exam. We assume we control for a sufficient set of confounders, and there are no significant unobserved confounders. The difference between this DAG and the DAG in figure 15 is that there are arrows from treatments back to confounders. The motivation behind this design is as follows: assume some students are suffering from a psychological problem, we refer them to the counselor and are hoping through the counselor intervention we reduce the negative effect of the confounder. Therefore a more intelligent design requires feedback from treatments to interventions. To find the average causal effect of joint intervention X_1,X_2, and X_3 on O, we start again by the same regression model as in previous cases with all observed confounders. We need to investigate if the correlation and causation are the same in equation 18 as in other cases. Controlling for L_1, L_2 and L_3 will close backdoors such as $X_1 \leftarrow L_1 \rightarrow O$, $X_2 \leftarrow L_2 \rightarrow O$, and $X_3 \leftarrow L_3 \rightarrow O$. Furthermore controlling for X_1, X_2 and X_3 will block the mediated and all other backdoor paths such as $X_2 \leftarrow X_1 \rightarrow O$. However controlling for L_2 will close the $X_1 \rightarrow L_2 \rightarrow O$ path, which is a causal path (neither mediated nor a backdoor paths), the same way controlling for L_3, will block the causal path $X_2 \rightarrow L_3 \rightarrow O$. Therefore we conclude that the coefficients of X_2 and X_3 are biased and outcome regression produces an unbiased coefficient for X_1.

$$E[O|X_1, X_2, X_3, L_1, L_2, L_3] = \alpha_0 + \alpha_1 X_1 + \alpha_2 X_2 + \alpha_3 X_3 + \alpha_4 X_1 X_2 +$$
$$\alpha_5 X_1 X_3 + \alpha_6 X_2 X_3 + \alpha_7 X_1 X_2 X_3 + \alpha_8 L_1 + \alpha_9 L_2 + \alpha_{10} L_3 \qquad (18)$$

To find the unbiased causal estimates of joint interventions on the outcome in the case of time-varying treatments, confounders feedback, we use g-formula and the inverse probability of treatment weighting (IPTW). Before discussing how to do inference using g-formula and IPTW, we will briefly discuss each one in the next section.
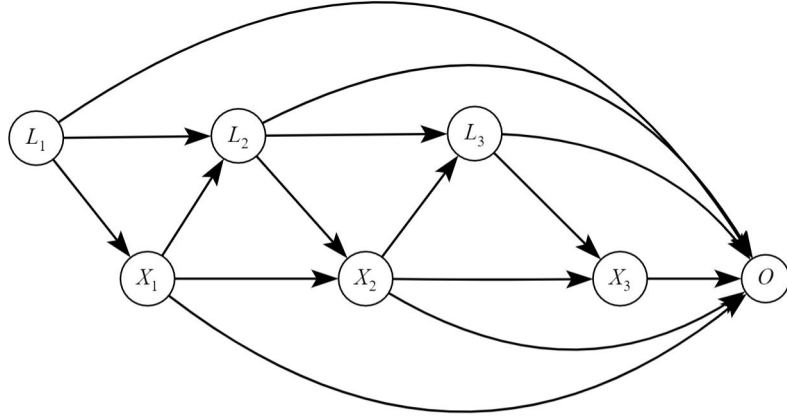
Figure 17: Time Varying Treatments and Confounders Feedback

5.3.1   G-formula and Inverse Probability of Treatment Weighting

G-formula is an extended version of the backdoor criterion or standardization discussed before in the time-varying treatments and confounders setting. Mathematically it is represented as follows:

$$E[O_{\bar{x}}] = \sum_{\bar{l}} E[O|\bar{X} = \bar{x}, \bar{L} = \bar{l}] \prod_{t=0}^{t=T} P(L_t = l_t | \bar{X}_{t-1} = \bar{x}_{t-1}, \bar{L}_{t-1} = \bar{l}_{t-1}) \tag{19}$$

where the $\bar{X}$ and $\bar{L}$ represent the treatments and confounders history, T is the number of time steps or the number of interventions. This model requires estimating the conditional expectation and conditional probability from data. Hence it is sensitive to model misspecification and requires correct models for conditional expectation and conditional probabilities.

IPTW is a method to weigh each observation by the inverse of the probability of treatment received. IPTW creates what is called the pseudo-population, which is a confounder-free population. Therefore IPTW is trying to emulate a randomized experiment. The IPTW weights for the subject i is defined as follows [29]:

$$W_i = \frac{1}{\prod_{t=0}^{t=T} P_{X_t|\bar{X}_{t-1},\bar{L}_t}(X_{t,i}, \bar{X}_{t-1,i}, \bar{L}_t, i)} \tag{20}$$

$$SW_i = \frac{\prod_{t=0}^{t=T} P_{X_t|\bar{X}_{t-1}}(X_{t,i}|X_{t-1,i})}{\prod_{t=0}^{t=T} P_{X_{t,i}|\bar{X}_{t-1,i},\bar{L}_t}(X_{t,i}, \bar{X}_{t-1,i}, \bar{L}_t, i)} \tag{21}$$

The SW_i is referred to as the stabilized weight. It results in a more stable estimator if the denominator of W_i is close to zero. However, a better way to express the IPTW weights is through a causal DAG. The denominator of IPTW weights (It is the same for W_i and SW_i) is the probability distribution of the treatments in the pre-intervention DAG since the treatment at the current time point is conditioned on treatment history and the confounder history. The numerator of the SW weight is the probability distribution on the post-intervention DAG since it is independent of confounders; hence the connections from confounders to treatments are removed. Therefore the stabilized weight can be represented as follows:

$$IPTW_{SW} = \frac{\text{Distribution of Treatment on Post-intervention DAG}}{\text{Distribution of Treatment on pre-intervention DAG}} \tag{22}$$

IPTW is used to estimate the parameters of marginal structural model (MSM) [15], [30], and [31]. MSM, as the name suggests, is a marginal model, namely not conditioned on any confounding variables. It is a model for calculating the mean potential outcome for the entire population. To see how g-formula and IPTW can solve the time-varying treatments and confounders feedback problems, consider the following simulation setup corresponding to the DAG in figure 17 with the difference that we assume the first treatment X_1 is assigned randomly; hence there is no confounder

connected to the X_1:

$$N = 500$$
$$P(X_1 = 1) = 0.6$$
$$P(L_2 = 1) = 0.1 + 0.6X_1$$
$$P(X_2 = 1) = 0.3 + 0.7L_1 - 0.25X_1$$
$$P(L_3 = 1) = 0.2 + 0.3L_2 + 0.3X_2$$
$$P(X_3 = 1) = 0.1 + 0.3L_3 + 0.1X_2$$
$$\mu = 5 + 4X_1 + 5X_2 + 6X_3 + 10X_1X_2 + 11X_1X_3 + 12X_2X_3 - 8L_1 - 9L_2$$
$$\sigma = 0.2 \qquad O \sim N(\mu, \sigma^2)$$

As it was discussed, the coefficients of X_2 and X_3 are not causal; therefore, to find the true causal coefficients we proceed as follows:

$$E[Y_{X_1X_2X_3}] = \sum_{L_2}\sum_{L_3} E[Y_{X_1X_2X_3}|L_2, L_3] = \sum_{L_2}\sum_{L_3} 5 + 4X_1 + 5X_2 + 6X_3 + 10X_1X_2 + 11X_1X_3 + \quad (23)$$
$$12X_2X_3 - 8L_1 - 9L_2$$
$$E[Y_{X_1X_2X_3}] = 2.13 - 2.42X_1 + 2.3X_2 + 6X_3 + 10X_1X_2 + 11X_1X_3 + 12X_2X_3 \quad (24)$$

As equation 24 shows the true value of the intercept and the coefficients for X_1 and X_2 are different from the original model while other coefficients stay the same. The stabilized weight according to equation 22 is:

$$IPTW_{SW} = \frac{P(X_2)P(X_3)}{P(X_2|X_1, L_2)P(X_3|X_2, L_3)} \quad (25)$$

Table 10 compares the 95% CI for the outcome regression and IPTW methods. According to this table, the outcome regression estimate is biased for the coefficients of X_1, X_2, and the intercept term, while IPTW estimates are unbiased for all the coefficients. This clearly indicates that traditional methods such as outcome regression produce biased estimates in the case of treatment and confounder feedback. Table 11 compares the outcome regression, MSM(IPTW) and non-parametric g-formula. This table represents that both IPTW and g-formula can handle time-varying treatments and confounders feedback.

We conclude this section by comparing the IPTW and g-formula: They both can handle time-varying treatments, confounders feedback. They both rely on positivity and conditional exchangeability assumptions (no unmeasured confounders). g-formula is more prone and sensitive to model misspecifications since it requires a correct model for conditional expectation and conditional probability. Non-parametric g-formula becomes very tedious to calculate if the dimension of treatments and confounders increases. Therefore, we believe IPTW is more suitable for educational studies.

Table 10: Simulation Results for Time Varying Treatments and Confounders Feedback: Comparison of Outcome Regression and MSM in terms of 95% CI

Coefficient	True	Regression 95% CI	MSM (IPTW) 95% CI
Intercept	2.13	(4.92, 5.02)	(1.910, 3.843)
X_1	-2.42	(3.97, 4.11)	(-3.351, -0.089)
X_2	2.3	(4.965, 5.120)	(-1.0192, 2.3798)
X_3	6.0	(5.816, 6.092)	(4.56, 6.90)
X_1X_2	10.0	(9.823, 10.047)	(7.861, 10.604)
X_1X_3	11.0	(10.87, 11.16)	(7.00, 12.06)
X_2X_3	12.0	(11.78, 12.14)	(9.56, 13.38)
$X_1X_2X_3$	0	(-0.1191, 0.2794)	(-1.80, 3.04)

Table 11: Simulation Results for Time Varying Treatments and Confounders Feedback:
Comparison of Outcome Regression, MSM and G-formula

Coefficient	True	Regression	MSM(IPTW)	G-formula
Intercept	2.13	5.02	3.09	1.80
X_1	-2.42	4.00	-1.85	-2.14
X_2	2.3	4.98	1.94	1.81
X_3	6.0	5.98	5.80	5.96
$X_1 X_2$	10.0	10.04	8.65	9.84
$X_1 X_3$	11.0	11.04	11.22	11.01
$X_2 X_3$	12.0	12.05	12.43	12.31
$X_1 X_2 X_3$	0	0.09	0.47	0.12

6 Conclusion

In this paper, we proposed various experimental and quasi-experimental designs for educational systems and quantify them using the graphical model and directed acyclic graph (DAG) language. We proposed to model the education system as time-varying treatments, confounders, and time-varying treatments-confounders feedback and derive unbiased causal estimates for the coefficients in fully observed and partially observed confounders. We showed that if we do not control for a sufficient set of confounders, then the causal estimate is biased. We showed that a more intelligent educational system contains feedback between treatments and confounders and provides techniques such as IPTW and g-formula to derive unbiased estimates of the coefficients.

Our model can be implemented in a standard college course by having a pretest exam before starting the course to measure students' prerequisite knowledge and including questions about student family, stress, emotional problems, and other factors such as how much time they can spend for the course per week. The pretest score can determine the initial treatment and study plan for students. Then students continue with the intervention and are tested weekly through quizzes to find the best subsequent interventions, such as talking to the TA, instructor, or the counselor depending on the problem they have. We ask the TAs, instructors, and counselors to ask questions during the meeting with students to collect information about the potential confounders and then try to control for these confounders in our analysis. We then have students take an exam (like a midterm or final exam) and follow the methods outlined in this article to evaluate the effectiveness of the joint interventions on the exam score.

References

[1] John R Anderson, C Franklin Boyle, and Brian J Reiser. Intelligent tutoring systems. *Science*, 228(4698):456–462, 1985.

[2] Hyacinth S Nwana. Intelligent tutoring systems: an overview. *Artificial Intelligence Review*, 4(4):251–277, 1990.

[3] Christina Conati. Intelligent tutoring systems: New challenges and directions. In *Twenty-First International Joint Conference on Artificial Intelligence*, 2009.

[4] Manie Tadayon and Gregory J Pottie. Predicting student performance in an educational game using a hidden markov model. *IEEE Transactions on Education*, 2020.

[5] Cristina Carmona, Gladys Castillo, and Eva Millán. Designing a dynamic bayesian network for modeling students' learning styles. In *2008 eighth IEEE international conference on advanced learning technologies*, pages 346–350. IEEE, 2008.

[6] Michael V Yudelson, Kenneth R Koedinger, and Geoffrey J Gordon. Individualized bayesian knowledge tracing models. In *International conference on artificial intelligence in education*, pages 171–180. Springer, 2013.

[7] Albert T Corbett and John R Anderson. Knowledge tracing: Modeling the acquisition of procedural knowledge. *User modeling and user-adapted interaction*, 4(4):253–278, 1994.

[8] Chris Piech, Jonathan Spencer, Jonathan Huang, Surya Ganguli, Mehran Sahami, Leonidas Guibas, and Jascha Sohl-Dickstein. Deep knowledge tracing. *arXiv preprint arXiv:1506.05908*, 2015.

[9] Judea Pearl. *Causality*. Cambridge university press, 2009.

[10] Michel Bessani, Julio AD Massignan, Talysson MO Santos, João BA London Jr, and Carlos D Maciel. Multiple households very short-term load forecasting using bayesian networks. *Electric Power Systems Research*, 189:106733, 2020.

[11] Yi Zuo and Eisuke Kita. Stock price forecast using bayesian network. *Expert Systems with Applications*, 39(8):6729–6737, 2012.

[12] Catriona M Queen and Casper J Albers. Intervention and causality: forecasting traffic flows using a dynamic bayesian network. *Journal of the American Statistical Association*, 104(486):669–681, 2009.

[13] Manie Tadayon and Greg Pottie. tsbngen: A python library to generate time series data from an arbitrary dynamic bayesian network structure. *arXiv preprint arXiv:2009.04595*, 2020.

[14] Thomas D Cook, Donald Thomas Campbell, and William Shadish. *Experimental and quasi-experimental designs for generalized causal inference*. Houghton Mifflin Boston, MA, 2002.

[15] James M Robins, Miguel Angel Hernan, and Babette Brumback. Marginal structural models and causal inference in epidemiology, 2000.

[16] Linda M Collins, Susan A Murphy, and Victor Strecher. The multiphase optimization strategy (most) and the sequential multiple assignment randomized trial (smart): new methods for more potent ehealth interventions. *American journal of preventive medicine*, 32(5):S112–S118, 2007.

[17] Paul R Rosenbaum. Observational study. *Encyclopedia of statistics in behavioral science*, 2005.

[18] Simon L Turner, Andrew B Forbes, Amalia Karahalios, Monica Taljaard, and Joanne E McKenzie. Evaluation of statistical methods used in the analysis of interrupted time series studies: a simulation study. *medRxiv*, 2020.

[19] Anita K Wagner, Stephen B Soumerai, Fang Zhang, and Dennis Ross-Degnan. Segmented regression analysis of interrupted time series studies in medication use research. *Journal of clinical pharmacy and therapeutics*, 27(4):299–309, 2002.

[20] Monica Taljaard, Joanne E McKenzie, Craig R Ramsay, and Jeremy M Grimshaw. The use of segmented regression in analysing interrupted time series studies: an example in pre-hospital ambulance care. *Implementation Science*, 9(1):1–4, 2014.

[21] Andrea L Schaffer, Timothy A Dobbins, and Sallie-Anne Pearson. Interrupted time series analysis using autoregressive integrated moving average (arima) models: a guide for evaluating large-scale health interventions. *BMC medical research methodology*, 21(1):1–12, 2021.

[22] JOHN M Gottman, Gene V Glass, and TR Kratochwill. Analysis of interrupted time-series experiments. *Single subject research: Strategies for evaluating change*, pages 197–234, 1978.

[23] Charles C Branas, Rose A Cheney, John M MacDonald, Vicky W Tam, Tara D Jackson, and Thomas R Ten Have. A difference-in-differences analysis of health, safety, and greening vacant urban space. *American journal of epidemiology*, 174(11):1296–1306, 2011.

[24] Stephen G Donald and Kevin Lang. Inference with difference-in-differences and other panel data. *The review of Economics and Statistics*, 89(2):221–233, 2007.

[25] Justin B Dimick and Andrew M Ryan. Methods for evaluating changes in health care policy: the difference-in-differences approach. *Jama*, 312(22):2401–2402, 2014.

[26] Edwin P Martens, Wiebe R Pestman, Anthonius de Boer, Svetlana V Belitser, and Olaf H Klungel. Instrumental variables: application and limitations. *Epidemiology*, pages 260–267, 2006.

[27] Joshua D Angrist, Guido W Imbens, and Donald B Rubin. Identification of causal effects using instrumental variables. *Journal of the American statistical Association*, 91(434):444–455, 1996.

[28] Sander Greenland. An introduction to instrumental variables for epidemiologists. *International journal of epidemiology*, 29(4):722–729, 2000.

[29] Rhian M Daniel, SN Cousens, BL De Stavola, Michael G Kenward, and JAC Sterne. Methods for dealing with time-dependent confounding. *Statistics in medicine*, 32(9):1584–1618, 2013.

[30] Tyler J VanderWeele. Marginal structural models for the estimation of direct and indirect effects. *Epidemiology*, pages 18–26, 2009.

[31] Stephen R Cole and Miguel A Hernán. Constructing inverse probability weights for marginal structural models. *American journal of epidemiology*, 168(6):656–664, 2008.