

# **Estimation of Effects of Endogenous Time-Varying Covariates: A Comparison Of Multilevel Linear Modeling and Generalized Estimating Equations**

Research Report

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# 1 Introduction

Across a wide range of disciplines, researchers analyze clustered longitudinal, observational data to investigate prospective causal relationships between variables. When analyzing such data, psychological researchers most commonly use the multilevel linear model<sup>1</sup> (MLM, [Bauer & Sterba, 2011](#)), which—in the context of longitudinal data analysis—partitions observed variance into stable between-person differences and within-person fluctuations ([Hamaker & Muthén, 2020](#)). In the application of the MLM, time invariant and time-varying covariates, the latter measured repeatedly over time, are often available. The inclusion of covariates is a common strategy to improve parameter precision and address bias introduced by (time-varying) confounders ([Daniel et al., 2013](#); [Wodtke, 2020](#)). Nevertheless, it is well-known that this approach is not universally beneficial, as conditioning on variables like colliders within the causal pathway can distort treatment effect estimates ([Elwert & Winship, 2014](#)).

Dating back to the work of Pepe and Anderson (1994), it has been well-established that including endogenous time-varying covariates—those directly or indirectly influenced by prior exposure/treatment or outcome—in longitudinal studies without treatment can result in biased estimates of treatment effects. Despite its significance, this issue has received little attention in psychological research. Building on this foundation, a recent paper by Qian et al. (2020) examined the suitability of MLM for estimating the causal effect of a time-varying exposure or treatment. Specifically, they focused on settings where the exposure is randomly assigned at each occasion within individuals. Such randomized exposures may include, for example, prompts delivered through push notifications to remind participants of cognitive or mindfulness-based strategies ([Nahum-Shani et al., 2021](#); [Walton et al., 2018](#)). While random assignment with a constant probability might seem sufficient to identify (the presence and absence of) causal effects, Qian et al. (2020) showed that model fitting issues and parameter bias can arise when

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<sup>1</sup>The MLM is known by various names, including: linear mixed model, hierarchical linear model, random-effect model and mixed-effects model.

a *time-varying endogenous covariate* is present.

However, due to a divide between the disciplines that employ the MLM, such critiques appear to have largely failed to reach the applied researcher in psychology. One specific reason might be that the technical jargon in other disciplines makes it difficult for researchers to recognize when and how these issues emerge. Therefore, this report aims to understand why Qian et al. (2020) found biased estimates of the treatment effect for some generative models containing endogenous covariates and not for others; and to explain this issue to an audience of psychologists. To achieve this aim, the study will first use graphical diagrams to evaluate relevant criteria, ensuring accessibility and rigor through a reliance on graphical rules rather than algebra. Next, data simulations, based on the original scenarios of Qian et al. (2020) as well as additional scenarios, will be conducted to pinpoint the issue and to assess whether these criteria effectively identify bias. Accordingly, the following research question will be addressed: *When does the inclusion of endogenous variables in multilevel linear models result in biased estimates of the treatment effect?*

## 2 Methods

To obtain a better understanding of the issue exposed by Qian et al. (2020), two methods were employed. First, graphical methods were used provide insight into the presence and extent of bias with potential violation of criteria: (a) path diagrams were used to evaluate the conditional independence assumption (Qian et al., 2020) and (b) directed acyclic graphs (DAGs) were used to evaluate the backdoor criterion (Pearl, 1988, 2009). Second, a simulation study was performed to reproduce the results for the generative models (GMs) from Qian et al. (2020) and to further isolate the issue using additional GMs. In this simulation, bias in the treatment effect was quantified using analytical multilevel models identical to the generative models.

## 2.1 Data Generation

We consider 2 generative models (GMs) from Qian et al. (2020), one (GM A) being a special case of the general model (GM G) where bias was detected. To further isolate the source of bias, we introduce two additional special cases, labeled GM B and C. Table 1 summarizes the differences between the generative models. Compared to the general model G, GM A is not directly determined by the random intercept  $b_{i0}$ ; GM B is does not have a random slope  $b_{i2}$  for treatment; and GM C does not have a fixed interaction effect  $\beta_1$  between covariate and treatment.

Table 1: Generative Models: Summary of Differences

Generative	Name in Qian et	dependency $b_{i0}$	random slope	
Model	al. (2020)	and $X_{it}$	treatment $b_{i2}$	interaction $\beta_1$
G(eneral)	3	✓	✓	✓
A	1	×	✓	✓
B	NA	✓	×	✓
C	NA	✓	✓	×

The details of the generative models are described below. We follow the symbol notation of Qian et al. (2020) to allow for direct comparison, but rewrite the equations into within- and between-person models (see Raudenbush & Bryk, 2002; Schoot, 2017).

### 2.1.1 Generative Model G

Following the original notation of Qian et al. (2020), the outcome of GM G was generated according to the following model:

$$Y_{it+1} = \alpha_0 + \alpha_1 X_{it} + b_{i0} + A_{it}(\beta_0 + \beta_1 X_{it} + b_{i2}) + \epsilon_{it+1}$$

where  $Y_{it+1}$  is the outcome at time  $t + 1$ ,  $X_{it}$  is the covariate at time  $t$ ,  $A_{it}$  is the treatment

at time  $t$ ,  $b_{i0}$  is the random intercept,  $b_{i2}$  is the random slope for the treatment, and  $\epsilon_{it+1}$  is the error term. We may rewrite this model into the repeated-observations or within-person model in the following steps:

$$\begin{aligned}
Y_{it+1} &= \alpha_0 + \alpha_1 X_{it} + b_{i0} + A_{it}(\beta_0 + \beta_1 X_{it} + b_{i2}) + \epsilon_{it+1} \\
&= \alpha_0 + \alpha_1 X_{it} + b_{i0} + \beta_0 A_{it} + \beta_1 A_{it} X_{it} + A_{it} b_{i2} + \epsilon_{it+1} \\
&= \alpha_0 + b_{i0} + \alpha_1 X_{it} + \beta_0 A_{it} + A_{it} b_{i2} + \beta_1 A_{it} X_{it} + \epsilon_{it+1} \\
&= (\alpha_0 + b_{i0}) + \alpha_1 X_{it} + (\beta_0 + b_{i2}) A_{it} + \beta_1 A_{it} X_{it} + \epsilon_{it+1} \\
&= \pi_{0i} + \pi_{1i} X_{it} + \pi_{2i} A_{it} + \pi_{3i} A_{it} X_{it} + \epsilon_{it+1}.
\end{aligned}$$

with the person-level or between-person model (level 2):

$$\begin{aligned}
\pi_{0i} &= \alpha_0 + b_{i0}, \quad \text{where } b_{i0} \sim \mathcal{N}(0, \sigma_{b0}^2), \\
\pi_{1i} &= \alpha_1, \\
\pi_{2i} &= \beta_0 + b_{i2}, \quad \text{where } b_{i2} \sim \mathcal{N}(0, \sigma_{b2}^2), \\
\pi_{3i} &= \beta_1.
\end{aligned}$$

We model fixed effects  $\alpha_0$ ,  $\alpha_1$ ,  $\beta_0$ , and  $\beta_1$  as constants across individuals, while random effects  $b_{i0}$  and  $b_{i2}$  capture individual-specific deviations. Specifically,  $b_{i0}$  represents deviations from the population intercept  $\alpha_0$ , and  $b_{i2}$  represents deviations from the population slope  $\beta_0$ . A higher  $b_{i0}$  indicates a higher initial outcome, while a higher  $b_{i2}$  indicates a stronger treatment effect. Following Qian et al. (2020), the random effects  $b_{i0}$  and  $b_{i2}$  are modeled independent of each other.

The covariate is generated as:

$$X_{it} = \begin{cases} b_{i0} + \epsilon_{X_{it}}, & \text{if } t = 1, \\ b_{i0} + Y_{it} + \epsilon_{X_{it}}, & \text{if } t \geq 2, \end{cases} \quad \text{where } \epsilon_{X_{it}} \sim \mathcal{N}(0, 1)$$

The randomization probability of treatment  $p_t = P(A_{it} = 1 \mid H_{it})$  is constant at  $1/2$ . Thus,  $A_{it} \sim \text{Bernoulli}(0.5)$  for  $i = 1, \dots, N$  and  $t = 1, \dots, T$ . In other words, for every given person  $i$  and every timepoint  $t$ , the probability that treatment is assigned is equivalent to a fair coinflip. The exogenous noise is  $\epsilon_{it+1} \sim \mathcal{N}(0, \sigma_\epsilon^2)$ .

Figure 1a shows the path diagram for the first couple observations of GM G.

### 2.1.2 Generative Model A

GM A is a special case of GM G, where the effect of the random intercept  $b_{i0}$  on the covariate  $X_{it}$  is set to zero. This results in a model where the covariate  $X_{it}$  is not directly determined by the random intercept  $b_{i0}$  (see Figure 1b). Instead, the endogenous covariate  $X_{it}$  equals the previous outcome  $Y_{it}$  plus some random noise:

$$X_{it} = \begin{cases} \epsilon_{X_{it}}, & \text{if } t = 1, \\ Y_{it} + \epsilon_{X_{it}}, & \text{if } t \geq 2, \end{cases} \quad \text{where } \epsilon_{X_{it}} \sim \mathcal{N}(0, 1)$$

### 2.1.3 Generative Model B

GM B is a special case of GM G, where the variance for the random slope  $b_{i2}$  is set to zero:  $\sigma_{b_2}^2 = 0$ . Consequently, the random slope  $b_{i2}$  for the treatment  $A_{it}$  is removed (see Figure 1c). While the within-person model is the same as GM G, there is a slight alteration in the between-person model:

$$\pi_{2i} = \beta_0.$$

The single equation model then becomes:

$$Y_{it+1} = (\alpha_0 + b_{i0}) + \alpha_1 X_{it} + \beta_0 A_{it} + \beta_1 A_{it} X_{it} + \epsilon_{it+1}$$

### 2.1.4 Generative Model C

GM C is a special case of GM G, where the fixed interaction parameter  $\beta_1 = 0$ , which implies the removal of the interaction term  $\beta_1 A_{it} X_{it}$  (see Figure 1d). This, in turn, removed  $\pi_{3i}$ , thereby creating a discrepancy in within-person model of GM C and GM G:

$$Y_{it+1} = \pi_{0i} + \pi_{1i} X_{it} + \pi_{2i} A_{it} + \epsilon_{it+1}.$$

Nevertheless, the between-person model of  $\pi_{0i}$ ,  $\pi_{1i}$  and  $\pi_{2i}$  remains the same as GM G. The single equation model then becomes:

$$Y_{it+1} = \alpha_0 + \alpha_1 X_{it} + b_{i0} + A_{it}(\beta_0 + b_{i2}) + \epsilon_{it+1}.$$

### 2.1.5 Parameter Values

The following parameter values were adapted from Qian et al. (2020):

$$\alpha_0 = -2, \quad \alpha_1 = -0.3, \quad \beta_0 = 1, \quad \beta_1 = 0.3,$$

$$\sigma_{b0}^2 = 4, \quad \sigma_{b2}^2 = 1, \quad \sigma_{\epsilon}^2 = 1.$$

## 2.2 Data Analysis

In the simulation study, we evaluated the performance of the models across a total of 24 different settings, each replicated 1,000 times, by systematically varying the following factors:

- **Generative Models (GM):** G, A, B, C
- **Number of timepoints (T):** 10, 30
- **Sample size (N):** 30, 100, 200

All data generation and estimation was performed in R, version 4.4.2 (Team, 2024). After the generation of data generation for any given setting, analytical multilevel linear models were fit that are equivalent to each of the respective data-generating models. To fit the standard MLM, the `lmer` function from the R-package `lme4` (Bates et al., 2015) was employed with restricted maximum likelihood estimation.

### 3 Results

#### 3.1 Conditional Independence and Path Diagrams

The first criterion to evaluate the presence of bias in the treatment effect estimates is the *conditional independence assumption*. According to Qian et al. (2020), this assumption should identify whether estimators of the treatment effect are consistent and unbiased under randomized treatment assignment. The conditional independence assumption states that the covariate at time  $t$  ( $X_{it}$ ) should be independent of the individual’s random effects ( $b_{i0}$  and  $b_{i1}$ ) once we account for their history of covariates ( $H_{it-1}$ ), previous treatments ( $A_{it-1}$ ), and prior outcomes ( $Y_{it}$ ). This is implied from the following notation:

$$X_{it} \perp (b_{i0}, b_{i1}) \mid H_{it-1}, A_{it-1}, Y_{it}.$$

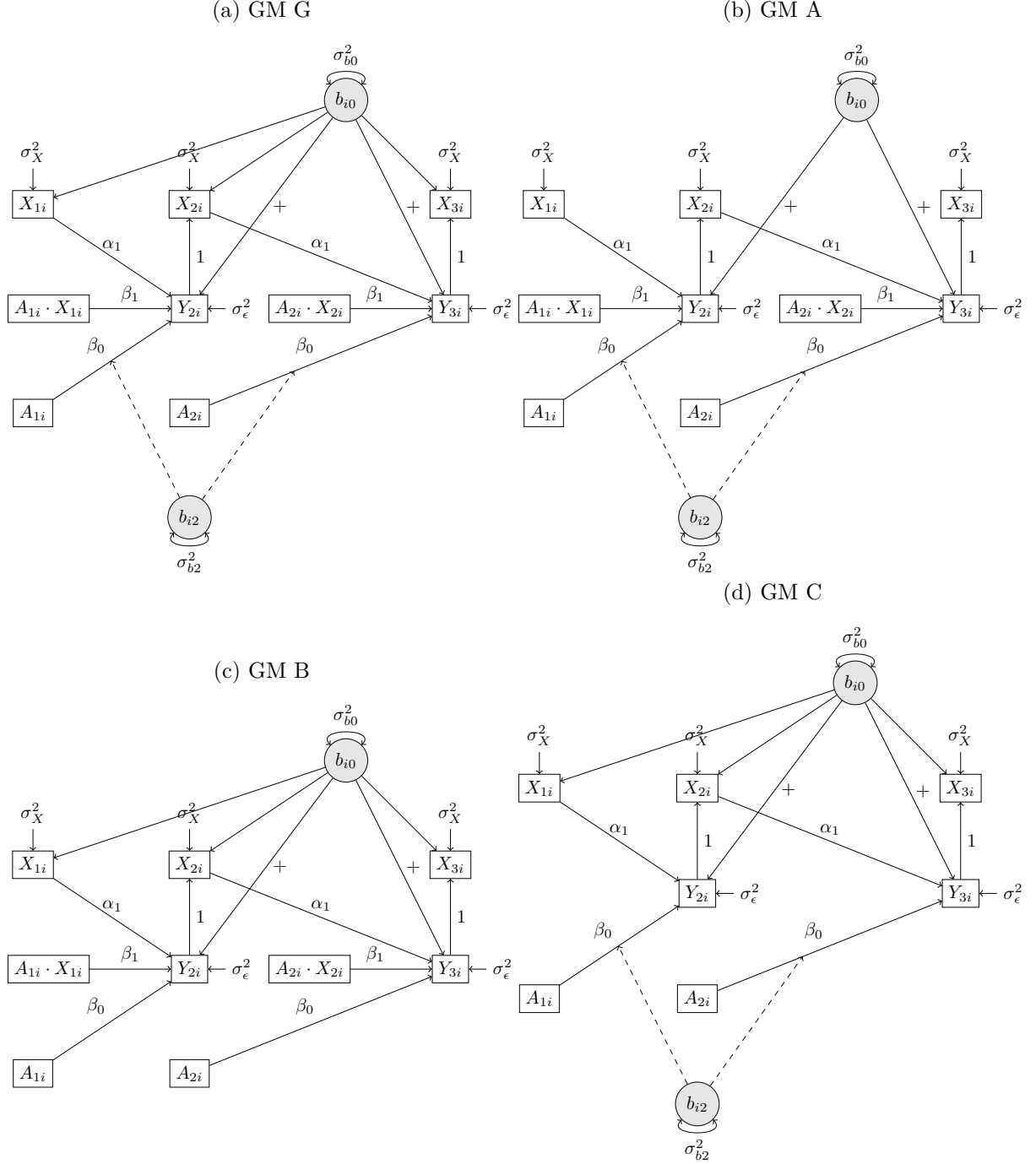
where  $b_{i0}$  and  $b_{i1}$  represent the random intercept and random slope(s), respectively, and  $H_{it-1}$  comprises all observations of that covariate before timepoint  $t$ . This assumption allows for  $X_{it}$  to be influenced by earlier variables (e.g., outcomes or treatments) but not directly by unobserved individual characteristics (i.e., random effects). If the included endogenous covariates are only affected by prior outcomes and treatments, the assumption is automatically satisfied. However, as Qian et al. (2020) highlights, ensuring this assumption holds requires careful consideration of theory and domain knowledge.

To clarify the application of the conditional independence assumption, we pair the equations



of the generative models (GMs) with path diagrams (Duncan, 1966; Wright, 1934) illustrating the first three timepoints ( $t$ ) for each model (see Figure 1).

Figure 1: Path Diagrams for Generative Models G, A, B and C ( $t = 1, 2, 3$ )



*Note.* Random effects are represented by grey circles, observed variables by squares and relationships across variables by arrows, where dashed lines are reserved for random slopes.

Let’s begin with the general model, GM G (Figure 1a), which Qian et al. (2020) identified as prone to bias. In GM G, the covariate  $X_{it}$  is directly influenced by unobserved individual factors (represented by the random effects,  $b_{i0}$ ). Consequently, conditioning on prior variables, such as the outcome at the previous timepoint  $Y_{it}$ , does not fully block or eliminate the influence of these unobserved factors. As a result,  $X_{it}$  remains dependent on the random effects, violating the assumption that  $X_{it}$  should be independent of these unobserved factors once we account for prior variables. This violation of the conditional independence assumption explains the biased estimates of the treatment effect observed in GM G, as identified by Qian et al. (2020).

In contrast, GM A, a special case of GM G where no bias was found by Qian et al. (2020), removes the direct link between  $X_{it}$  and the random effects  $b_{i0}$ . In this case,  $X_{it}$  is simply the previous outcome  $Y_{it}$  plus some random noise. While there remains an indirect connection between  $X_{it}$  and  $b_{i0}$  through  $Y_{it}$ , conditioning on  $Y_{it}$  effectively “breaks the link” between  $X_{it}$  and the random effects, satisfying the conditional independence assumption. This explains the unbiased treatment effect estimates found by Qian et al. (2020).

For the additional special cases, GM B and GM C, the direct link between the random effects and  $X_{it}$  remains, as in GM G. As a result, these models also violate the conditional independence assumption, leading to biased estimates of the treatment effect.

In summary, GM G, B, and C violate the conditional independence assumption, which suggests that we would expect biased treatment effect estimates for these models. In contrast, GM A satisfies the assumption, supporting unbiased estimates of the treatment effect.

### 3.2 Backdoor Criterion and DAGs

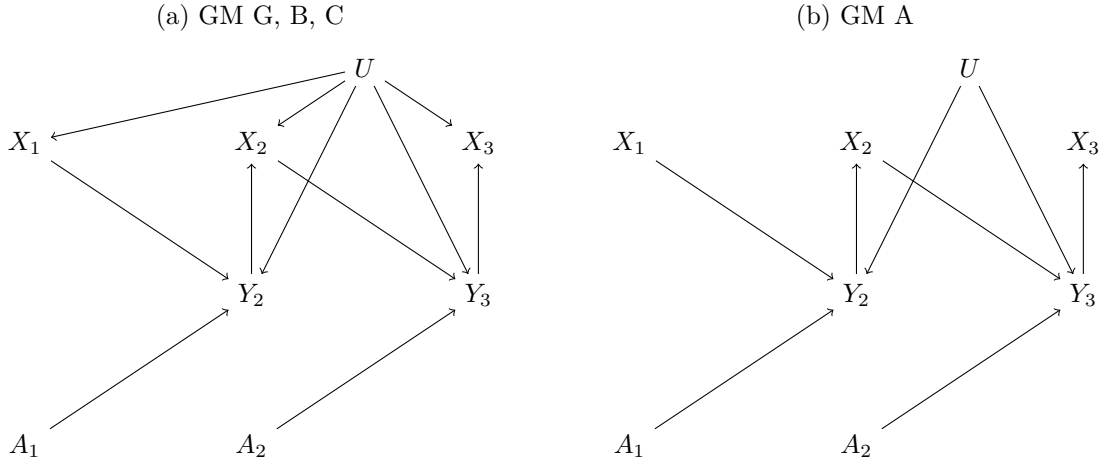
The second criterion for evaluating the presence of bias in treatment effect estimates is the *backdoor criterion* (Pearl, 1988, 2009). This criterion provides a partial solution to the classical problem in causal inference: *causal effects* cannot be directly observed and must instead be inferred from observed associations, which often represent a mixture of causal effects and various

undesirable non-causal, or *spurious*, components (Holland, 1986). When it is possible, under ideal conditions (e.g., no measurement error, infinite sample size), to isolate the causal effect from a combination of causal and spurious components, the causal effect is said to be identified. According to the backdoor criterion (Pearl, 1988, 2009), causal effects can be identified by blocking non-causal paths through conditioning on appropriate variables (e.g., controlling or matching). For instance, a causal effect of exposure on outcome can be identified by including in the analysis (i.e., controlling for) all relevant confounders—common causes that would otherwise induce spurious relationships. However, if spurious paths remain unblocked due to unmeasured variables or measurement error, the treatment and outcome remain linked via backdoor paths, leading to biased estimates of the treatment effect (Kim & Steiner, 2021).

To detect the presence of such backdoor paths, directed acyclic graphs (DAGs) (Pearl, 1995, 2009) are invaluable tools. DAGs generalize conventional linear path diagrams (Duncan, 1966; Wright, 1934) and operate in a fully nonparametric framework. Unlike traditional path diagrams, DAGs make no assumptions about distributional properties (e.g., multivariate normality) or functional forms (e.g., linearity). Instead, they encode qualitative causal assumptions about the data-generating process in the population. Arrows connecting nodes indicate direct causal effects, which may vary in magnitude across individuals (effect heterogeneity) or depend on the values of other variables (effect interaction or modification) (Elwert & Winship, 2014). Notably, random slopes from random-effects models and interaction effects are not explicitly represented in DAGs, which precludes their use for evaluating the conditional independence assumption.

Using the direct causal effects specified in each generative model (GM), we can formulate DAGs for the first three observations, representing the random disturbance  $b_{0i}$  as the node  $U$  (e.g., Kim & Steiner, 2021, see Figure 2). These diagrams confirm that random slopes and fixed interaction effects are absent. Indeed, this absence explains why the DAGs for GMs G, B, and C are equivalent.

Figure 2: DAGs for Generative Models G, A, B and C ( $t = 1, 2, 3$ )



*Note.* The red arrows show the biased backdoor path(s) in the treatment effect (before controlling for  $X_{it}$ ).

We now apply the backdoor criterion to these DAGs to assess potential bias in the treatment effect. For all GMs, there are no backdoor paths in the treatment effect  $A_t \rightarrow Y_{t+1}$ , as  $A_t$  lacks any parent nodes. Consequently, covariate  $X_t$  need not be controlled to obtain an unbiased total effect. Importantly, including  $X_{it}$  does not introduce identification issues, as it is neither a mediator (i.e., on the pathway from  $A_t$  to  $Y_{t+1}$ ) nor a collider (i.e., a common effect of  $A_t$  and  $Y_{t+1}$ ). Therefore, according to the backdoor criterion, controlling for  $X_{it}$  should not result in biased estimates of the treatment effect in any of the generative models.

### 3.3 Simulation Study

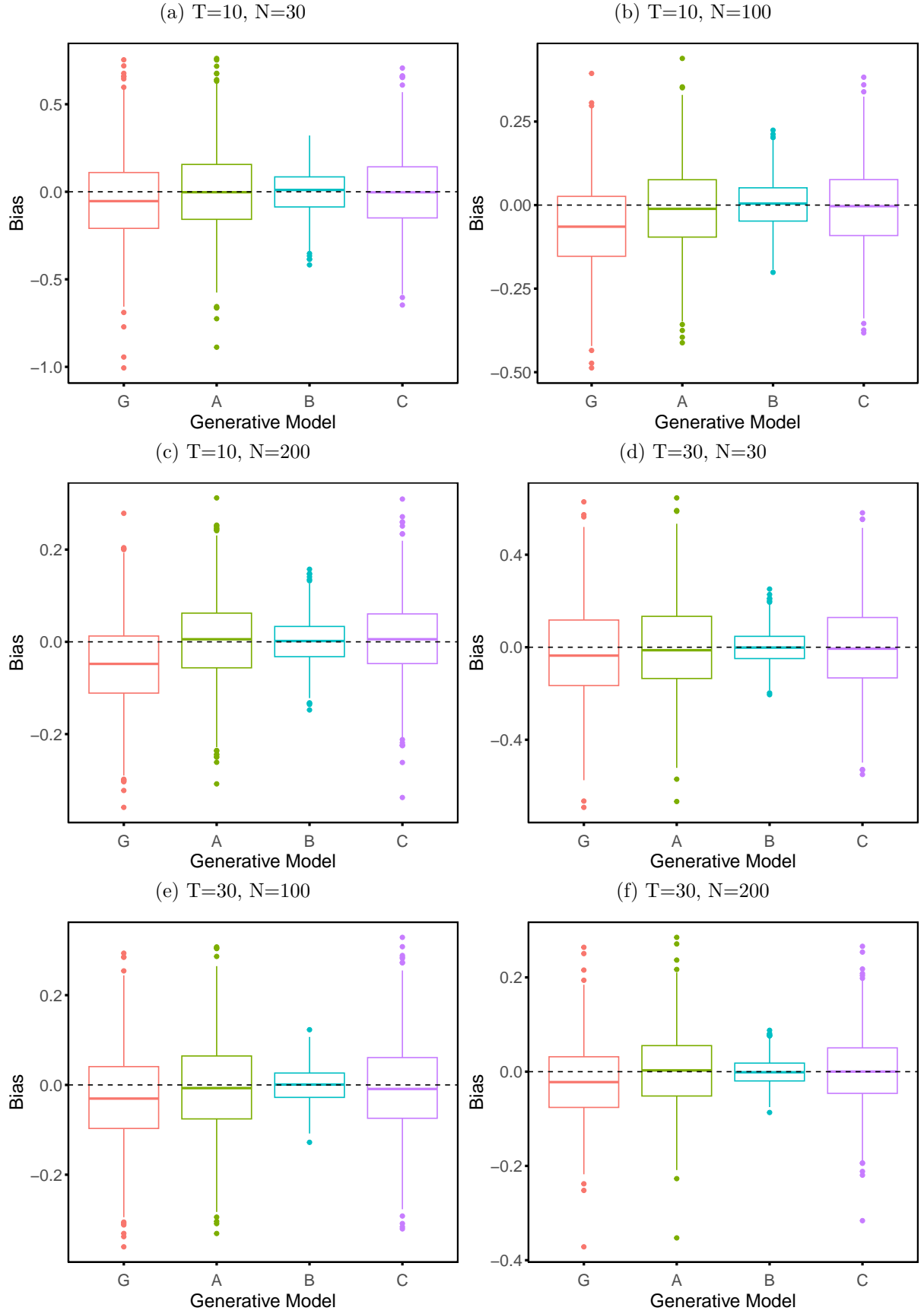
Table 2 and Figure 3 present the simulation results for each of the generative models. The  $\beta_0$  bias in Table 2 refers to  $\bar{\hat{\beta}}_0 - \beta_0$ , representing the difference between the mean of the estimated parameter values  $\bar{\hat{\beta}}_0$  and the prespecified treatment effect  $\beta_0 = 1$ . Thus, an absolute bias of 0.05 implies a 5% relative bias.

Table 2: Treatment effect bias for Generative Models G, A, B and C over 1000 replications

GM	T	N	$\beta_0$		SR
			Bias	SD	
G	10	30	-0.052	0.245	0.999
		100	-0.064	0.134	1.000
		200	-0.051	0.096	1.000
	30	30	-0.024	0.206	0.997
		100	-0.030	0.108	0.996
		200	-0.023	0.080	0.997
A	10	30	0.000	0.238	0.998
		100	-0.012	0.129	1.000
		200	0.003	0.093	0.999
	30	30	-0.001	0.203	0.998
		100	-0.007	0.107	0.996
		200	0.001	0.079	0.996
B	10	30	0.000	0.126	1.000
		100	0.004	0.073	1.000
		200	0.002	0.048	1.000
	30	30	-0.001	0.071	1.000
		100	0.000	0.040	1.000
		200	0.000	0.028	1.000
C	10	30	0.001	0.217	0.999
		100	-0.008	0.121	1.000
		200	0.005	0.087	1.000
	30	30	0.000	0.193	1.000
		100	-0.008	0.103	0.997
		200	0.001	0.075	0.999

*Note.* GM: generative model. T: number of timepoints. N: sample size. SD: standard deviation of estimates across replications. SR: model fitting success rate. Bias:  $\hat{\hat{\beta}}_0 - \beta_0$ , which represents the difference between the mean of the estimated parameter values  $\hat{\hat{\beta}}_0$  and the prespecified treatment effect  $\beta_0 = 1$

Figure 3: Estimation bias for the fixed treatment effect  $\beta_0$  of each generative model for different combinations of sample size  $N$  and number of timepoints  $T$  over 1000 simulation replications



In this reproduction of Qian et al. (2020), the overall pattern was consistent with the original study (see Figure 3): we observed substantial absolute bias ranging from 0.02 to 0.06 for the most general generative model (GM G), and much smaller bias of  $\leq 0.015$  for GM A. These results align with expectations based on the conditional independence assumption, which predicts that the treatment effect would be unbiased for GM A and biased for GM G. However, the findings contradict the backdoor criterion, which predicts no bias for any of the generative models (GMs). Notably, we found greater treatment effect bias for GM A than reported by Qian et al. (2020), with a maximum bias of  $-0.012$  in the scenario with  $T = 10$  and  $N = 100$  (see Table 2), compared to  $-0.003$  found by Qian et al. (2020) for the same scenario. For GM G, the bias was most pronounced in this scenario as well, reaching  $-0.064$ . Furthermore, the size of the bias decreased as the number of time points increased.

For the two additional special cases of GM G, namely GM B and GM C, we observed even smaller absolute bias than for GM A:  $\leq 0.010$  for GM C and  $\leq 0.005$  for GM B (see Table 2). These findings align with the backdoor criterion’s prediction of no bias but contradict the expectations based on the conditional independence assumption, which suggests the presence of bias. Additionally, GM B exhibited the smallest absolute bias overall and showed much smaller variability across simulation replications compared to all other GMs. In contrast, the remaining models exhibited comparable levels of variability (see Figure 3 and Table 2).

In summary, these findings suggest that once we modify the most general model G by removing either the dependency of the random intercept on the covariate (GM A), the random slope  $b_{i2}$  (GM B), or the interaction term  $\beta_1$  (GM C), the bias either disappears or becomes negligible. However, neither the backdoor criterion nor the conditional independence assumption provided consistent predictions of treatment effect bias across all models.

## 4 Discussion

This report employed both graphical methods and data simulations to understand and explain the issue of endogenous covariates.

While Qian et al. (2020) show that GM3 is the only model with bias in the treatment effect, the backdoor criterion failed to identify this bias, as there is no backdoor path in the treatment effect. This may be explained by the fact that the classic DAG does not impose restrictions based on (a) the random slopes and (b) interaction effects.

The research question pertained to the extent of treatment effect bias of generative models GM B and C that are special cases of GM G. First, we reproduced the findings by Qian et al. (2020) who found consistent estimators for GM1 and inconsistent ones for GM3. Using additional generative models, we found that bias became indiscernable when removing from GM3 either the dependency between the random intercept and covariate (GM1), the random slope for treatment (GM3A) or the interaction effect (GM3B). This finding is in sharp contrast to the suggestion of the conditional independence assumption that the treatment effect would be biased for GM3, 3A and 3B.

The current research report leaves several avenues unexplored. First, it is unclear whether the simulation findings pertaining the generative models in Qian et al. (2020) and here generalize to other generative models. For instance, we found here that removal of a random slope or interaction from GM3 got rid of most if not all of the treatment effect bias. Thus, it is important to establish how this generalizes, so that practical recommendations can be formulated. This is particularly important, since while violations of model assumptions are never desired, the robustness against and the practical implications of a violation is what matters. Second, it is unclear how exactly the divide between the literatures pertaining to the focus of the MLM on different centering methods and within- and between-person interpretations and the focus of the GEE on marginal and conditional interpretations may be bridged. Consequently, future research could assess the implications of centering methods in MLMs on the extent to which



the marginal interpretation of MLM breaks down. Third, we found that the classical DAG may not be sufficient to identify bias in the treatment effect for GM3, especially due to its lack of specification of interaction effects. Concerns regarding the use of Pearl’s backdoor criterion in situations with interaction effects have been voiced by several people (see Weinberg (2007); Attia et al. (2022)). Future research could explore to what extent proposed extensions of the DAG may be useful in identifying bias in the treatment effect for GM3. Finally, it may be interesting to investigate the implications of endogenous covariates in MLMs for other types of longitudinal data analysis methods, such as dynamic structural equation modelling (DSEM; a widely used framework in the social sciences based on MLM).

Third, since the issue extends to all longitudinal data analysis methods according to Diggle (2002), in future research it may be interesting to investigate the implications of endogenous covariates in MLMs for other types of longitudinal data analysis methods, such as dynamic structural equation modelling (DSEM; a widely used framework in the social sciences based on MLM).

**to recognize and understand precisely when and why endogenous covariates may trouble an empirical investigation.**

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