A Two-Step Robust Estimation Approach for Inferring Within-person Relations in Longitudinal Design: Tutorial and Simulations

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Abstract

Psychological researchers have shown an interest in disaggregating within-person variability from between-person differences. Especially for inferring reciprocal relations among variables at the within-person level, applications of the random-intercept cross-lagged panel model (RI-CLPM) with stable trait factors has increased rapidly. This paper provides a tutorial, simulation, and illustrative example of another recent approach proposed by Usami (2022a). This approach consists of a two-step procedure: within-person variability scores (WPVS) for each person, which are disaggregated from the stable traits of that person, are calculated using structural equation modeling, and causal parameters are then estimated via a potential outcome approach, such as by using structural nested mean models (SNMMs). This method assumes a data-generating process similar to that in RI-CLPM, and has several advantages: (i) the flexible inclusion of curvilinear and interaction effects for WPVS as latent variables, (ii) more accurate estimates of causal parameters for reciprocal relations can be obtained under certain conditions owing to them being doubly robust, even if unobserved time-varying confounders and model misspecifications exist, and (iii) the risk of obtaining improper solutions is minimized. After explaining the data-generating process and the analysis procedure using the R package DTRreg for SNMMs, estimation performances are compared with RI-CLPM through large-scale simulations. We show that the proposed approach works well in many conditions if longitudinal data with $T \geq 4$ are available, and that the accuracy increases as $T$ becomes larger. An analytic example using data regarding sleep habits and mental health statuses from the Tokyo Teen Cohort (TTC) study is also provided.

Keywords: within-person relation, longitudinal data, structural equation modeling, structural nested mean model, causal inference
1 INTRODUCTION

When analyzing relations among variables in data, psychological researchers differentiate between within-group (unit-level) relations and between-group (group-level) relations, and between within-person relations and between-person relations. Particularly in longitudinal design, researchers have shown an interest in inferring within-person relations: how changes in one variable influence another for the same person. Within-person relations may exhibit statistically different (or even opposite) tendencies from between-person relations, and this is one reason that statistical inference for disaggregating within- and between-person relations has long been a concern in psychology. On the other hand, estimands that are defined at the within-person level have been less common in the causal inference literature (Lüdtke & Robitzsch, 2021).

Multilevel modeling (Hoffman, 2014; Wang & Maxwell, 2015) and structural equation modeling (SEM) (Hamaker, Kuiper, & Grasman, 2015) are two statistical methods that have been popular for investigating within-person relations. The SEM approach might be advantageous if researchers wish to (i) include common factors in measurement models for multiple indicators, (ii) assume measurement errors for measurements with imperfect reliability, (iii) treat multiple outcomes to evaluate reciprocal (or mutual) relations, or (iv) use model fit indices to evaluate how the model implied mean and covariance structure can reproduce the observed mean vector and covariance matrix. After the critique of applying a cross-lagged panel model (CLPM) to infer reciprocal relations at the within-person level (Hamaker et al., 2015), applications of random-intercept CLPM (RI-CLPM), which includes common factors called stable trait factors to control for between-person differences, have been growing rapidly. It has been empirically shown that model choice of CLPM or RI-CLPM can be critical in terms of the signs, statistical significance, and magnitudes of key parameter estimates (i.e., cross-lagged parameters) for reciprocal relations (e.g., Orth, Clark, Donnellan, & Robins, 2021; Usami, Murayama, & Hamaker, 2019). RI-CLPM is a
useful analytic option, but various kinds of (SEM-based) statistical models are available for examining reciprocal relations, and model choice is still an ongoing issue (see Andersen, 2022; Lucas, 2022; Lüdtke & Robitzsch, 2021; Usami, 2021,2022ab; Usami, Murayama, et al., 2019 and later discussion).

Along with these increasing applications and theoretical interests, Usami (2022a) aimed to synthesize the SEM-based approach traditionally used in psychology and the potential outcome approaches used in epidemiology, in order to enable flexible and robust inference of within-person relations. This method consists of a two-step procedure that assumes the similar data-generating process (DGP) as assumed in the RI-CLPM. In this approach, the \textit{within-person variability scores} (WPVS) for all participants, which are disaggregated from their stable trait factor scores, are first calculated in each variable using SEM. Then, causal parameters modeled at the within-person level are estimated using a potential outcomes approach with the calculated WPVS, such as marginal structural models (MSMs; Robins, 1999; Robins, Hernán, & Brumback, 2000) or structural nested (mean) models (SNMMs; Robins, 1989, 1992). Usami (2022a) originally assumed a situation where researchers sought to investigate the unidirectional relation between variables, but it is straightforward to extend the approach to reciprocal relations. For brevity, we will refer to this two-step estimation approach as the TS method.

The TS method has some advantages over RI-CLPM. If researchers assume a DGP that includes stable trait factors, then they need to model the relations among WPVS as latent variables. This corresponds to the fact that RI-CLPM is sometimes classified as a deviation model rather than an observation model (e.g., Andersen, 2022). However, in the standard SEM as covariance structure analysis, estimating curvilinear (e.g., quadratic) effects and interaction effects for latent variables can be challenging in terms of implementation, especially if multiple indicators are not available for each variable at every time point (e.g., Kenny & Judd, 1984; Klein & Moosbrugger, 2000; Kline, 2016 pp.437-444; Marsh, Wen, & Hau, 2006). Even if multiple indicators are available, identification may require strong
parameter constraints, and implementation could be very difficult, especially for cases in which researchers wish to include interaction effects between variables from different time points (e.g., $X_3^*Y_1^*$ for variables $X^*$ at $t = 3$ and $Y^*$ at $t = 1$ at the within-person level). Bayesian approach is a useful alternative (e.g., Lee, 2007; Ozkok et al., 2022), but plausible specifications of priors and computation time can be challenging. For these reasons, usual applications of RI-CLPM do not assume curvilinear and interaction effects of variables at the within-person level (WPVS). On the other hand, in TS method, such effects can be introduced in a straightforward manner because the calculated WPVS are directly used for causal parameter estimation.

Another potential advantage of the TS method is the manner in which it accounts for time-varying confounders from the view of causal inference. RI-CLPM (and other SEM-based models) was originally proposed to uncover reciprocal relations between focal variables at the within-person level ($X^*$ and $Y^*$), and it does not directly account for time-varying confounders. One reasonable procedure to account for observed time-varying confounders in RI-CLPM is to simply include them in the lagged regression (i.e., structural models) for reciprocal relations like ANCOVA. However, along with the potential aforementioned restrictions in modeling curvilinear and interaction effects for observed time-varying confounders, this approach requires researchers to correctly specify all structural models at each time point, and parameters might be seriously biased if researchers miss some unobserved time-varying confounders. On the other hand, as we will illustrate, a more accurate estimate of causal parameters can be obtained under certain conditions using the TS method, especially with (linear) SNMMs (we call these TS-SNMMs later for brevity), even if unobserved time-varying confounders and model misspecifications relating to observed confounders exist.

The TS method is also potentially advantageous for handling improper solutions. Though this risk of these occurring in RI-CLPM is empirically known to be smaller than in some SEM-based approaches, it can still frequently produce improper solutions (e.g., the non-
Hessian matrix of trait factor scores and negative variances), especially when either the sample size or the number of time points is small (Orth et al., 2021; Usami, Todo, & Murayama, 2019). The TS method takes a two-step estimation approach, so not all parameters are simultaneously estimated as in SEM-based approaches. This can minimize the risk of obtaining improper solutions caused by sample fluctuations and model misspecifications.

For these reasons, the TS method can be considered as a viable alternative of RICLPM (and its variants) for inferring within-person (reciprocal) relations, especially when researchers wish to account for time-varying confounders. Comparing estimation results between these approaches could also be useful as a kind of sensitivity analysis. However, MSMs and SNMMs have been developed in epidemiology and we have seen few applications of these methods in psychology, while useful introductions and applications of MSMs are increasing (e.g., VanderWeele, Hawkley, Thisted & Cacioppo, 2011). Actual applications of SNMMs have also been infrequent in other disciplines because of limited software availability and tutorials (Vansteelandt & Joffe, 2014), as well as its typically strongly theoretical presentation and challenging implementation (Wallace, Moodie & Stephens, 2017a). SNMs, however, are suitable and robust for handling violation of the usual assumptions of no unobserved confounders and sequential ignorability (Robins, 1999; Robins & Hernan, 2009; Vansteelandt & Joffe, 2014). Only a brief introduction of MSMs and SNMMs was provided in Usami (2022a), and there are not many applied researchers in psychology who are capable of applying such novel methods.

On a related note, another important limitation of Usami (2022a) is that their estimation performance was assessed under restricted scenarios of model (mis)specifications with limited conditions for the number of time points and the magnitudes of WPVS and stable trait factor (co)variances. WPVS need to be calculated in the first step of the TS method, which may result in less accurate causal estimates, especially when the number of time points is limited under a misspecified measurement model. One of the primary motivations for performing the simulation in Usami (2022a) was to compare the performance of the TS
method (which aims to conduct true score centering [e.g., Asparouhov & Muthén, 2018] based on stable trait factors) over different centering methods (i.e., observed person-mean centering). Therefore, how the estimation performances (e.g., the bias of causal parameters and the frequency of improper solutions) compare between the TS method and RI-CLPM is still an open question.

In this paper, we provide a tutorial, simulation, and illustrative example of how to use the TS method to infer within-person (reciprocal) relations, focusing especially on using TS-SNMMs to estimate causal parameters by utilizing the R package DTRreg (Wallace et al., 2017ab), which was not used in Usami (2022a). We demonstrate in large-scale simulations that TS-SNMMs can flexibly and accurately estimate causal parameters due to them being doubly robust, even if unobserved time-varying confounders and model misspecifications exist. We also show that TS-SNMMs work well in many conditions if longitudinal data with $T \geq 4$ are available, while accuracy increases as $T$ becomes larger. The R code for TS-SNMMs is available in a supplementary document to make the method more accessible for applied researchers.

The remainder of this paper is organized as follows. In Section 2 we start our discussion by introducing assumed DGPs and the definition of causal effects, while appropriately referring to Usami (2022a). Readers familiar with these may skip this (sub)section. TS-SNMMs are introduced in Section 3, and simulations are provided in Section 4. Section 5 describes an empirical application of TS-SNMMs using data from the Tokyo Teen Cohort (TTC) study. A summary is provided in the final section along with a discussion of our future research agenda.
2 ASSUMED DATA-GENERATING PROCESS AND DEFINITIONS OF CAUSAL EFFECT

2.1 Data-generating process

We suppose that data are generated at fixed time points and let $X_{it}$ and $Y_{it}$ denote focal (continuous) variables at $t$ ($t = 1, \ldots, T$) for person $i$: researchers wish to infer their within-person relation. Also, let $L_{it}$ be the observed time-varying confounders. We assume a single confounder here for explanation purposes. Suppose that a time-varying confounder has three characteristics: it is independently associated with future focal variables as well as future confounders, and it is affected by earlier focal variables and confounders.

Figure 1 is a directed acyclic graph (DAG) that expresses the causal relations among variables in the assumed DGP of $T = 4$. Because we suppose the presence of stable trait factors in DGP, as in RI-CLPM, this DAG includes stable trait factors $I$ as time-invariant factors. This DAG is similar to the one presented in Usami (2022a, Figure 1b), but it now assumes that $X_{it}$, $Y_{it}$, and $L_{it}$ are measured at the same time, and thus, no direct causal relation is assumed among them within each time point. This setting is analogous to the path diagrams of the SEM-based statistical models (e.g., RI-CLPM). Each solid single-headed arrow represents a direct causal relation, and a dashed double-headed arrow indicates the covarying relations due to unobserved confounding. Dashed circles are used to express latent variables. To keep the illustration simple, here we temporarily assume first-order linear lagged effects of variables.

By the properties of stable trait factors (specifically that the difference between the expected value of measurement of each person and the temporal group mean is invariant over time; see Usami, 2022a), these factors have zero means ($E(I^{(Y)}) = E(I^{(A)}) = E(I^{(L)}) = 0$) and additively influence respective measurements. For the same reason, the values of the coefficients corresponding to the paths from these factors to the measurements are...
restricted to one.

In this DAG, directed edges from WPVS, which are expressed by variable names with an asterisk (e.g., $Y^*_3$), are drawn to the corresponding measurements. Directed edges are assumed between WPVS as (time-varying) latent variables rather than between measurements. Without loss of generality, we assume that WPVS for all variables have zero means.

It is important to note that WPVS are also assumed to be uncorrelated with stable trait factors (we will revisit this issue in the Discussion). As a result, like RI-CLPM, stable trait factors $I$ have only direct effects on measurements, and each measurement can be decomposed into a (linear) sum of time-invariant (stable traits) and time-varying factors (i.e., WPVS) that are mutually uncorrelated.

Under the linear causal DAG model, the DGP can be represented by the following equations, which correspond to RI-CLPM that includes $L$:

$$Y_{it} = \mu^{(Y)}_t + I^{(Y)}_i + Y^*_it, \quad X_{it} = \mu^{(X)}_t + I^{(X)}_i + X^*_it, \quad L_{it} = \mu^{(L)}_t + I^{(L)}_i + L^*_it$$

for $t \geq 1$, and

$$Y^*_it = \alpha^{(Y)}_i Y^*_{i(t-1)} + \beta^{(Y)}_i X^*_{i(t-1)} + \gamma^{(Y)}_i L^*_{i(t-1)} + d^{(Y)}_{it}$$
$$X^*_it = \alpha^{(X)}_i Y^*_{i(t-1)} + \beta^{(X)}_i X^*_{i(t-1)} + \gamma^{(X)}_i L^*_{i(t-1)} + d^{(X)}_{it}$$
$$L^*_it = \alpha^{(L)}_i Y^*_{i(t-1)} + \beta^{(L)}_i X^*_{i(t-1)} + \gamma^{(L)}_i L^*_{i(t-1)} + d^{(L)}_{it}$$

for $t \geq 2$. $\mu$ and $d$ denote the temporal group means and residual terms, respectively, and they are omitted in the DAG representation. As noted, stable trait factors are assumed to be uncorrelated with WPVS. For example,

$$Cov(I^{(Y)}_i, Y^*_it) = 0, \quad Cov(I^{(X)}_i, X^*_it) = 0, \quad Cov(I^{(Y)}_i, L^*_it) = 0$$

for a stable trait factor of a variable $Y$ ($I^{(Y)}_i$).

As implied in Equation (1), under these specifications, $Y^*_it$, $X^*_it$, and $L^*_it$ represent temporal deviations from the expected score for person $i$ at $t$ (i.e., $\mu^{(Y)}_t + I^{(Y)}_i$, $\mu^{(X)}_t + I^{(X)}_i$, $\mu^{(L)}_t + I^{(L)}_i$).
and $\mu_t^{(L)} + I_t^{(L)}$), whereas the stable trait factors represent stable between-person differences over time. To put it another way, WPVS can be characterized as the difference between a measurement and its expected value for each person at each time point (see Usami (2022a) for further explanation). Additionally, (co)variances of measurements at $t$ can be expressed as the sum of those of stable trait factor scores and WPVS. For a variable $Y$,

$$\text{Cov}(Y_{it}, Y_{i't}) = \text{Cov}(Y_{it}^*, Y_{i't}^*) + \text{Var}(I_t^{(Y)}). \quad (4)$$

In RI-CLPM, the initial deviations (WPVS) are modeled as exogeneous variables, and their variances and covariances are estimated.

RI-CLPM can identify the parameters from data with $T \geq 3$. If one is interested in inferring first-order lagged effects of $X^*$ on $Y^*$ ($X_{t-1}^* \rightarrow Y_t^*$), $\beta_t^{(Y)}$ in Equation (2), which is called a cross-lagged parameter, is key.

### 2.2 Definition of causal effects at the within-person level

Below we assume a similar causal DAG model to that in Figure 1. However, unlike the previous subsection, we relax some assumptions about WPVS, specifically that higher-order, curvilinear (e.g., quadratic), and interaction effects for WPVS can exist. The current focus is on evaluating the within-person relation between variables, that is, how one variable (e.g., $X_{t-1}^*$) influences another variable (e.g., $Y_t^*$) and vice versa at the within-person level. Stable trait factors and WPVS are mutually uncorrelated, and each measurement can be characterized as a collider in causal DAG (i.e., it is causally influenced by stable trait factors and WPVS; Usami, 2022a), so causal effects of WPVS can be defined independently from stable trait factors. However, modeling stable trait factors through measurements is still required for estimating causal parameters because WPVS are latent variables (Usami, 2022a). Below we first explain the definition of causal effects of $X^*$ (as predictor) on $Y^*$ (as outcome).

We use overbars $\bar{Y}_t^* = \{Y_1^*, \ldots, Y_t^*\}$ to denote the history of $Y^*$ through $t$ and underbars
\( Y_t^\ast = \{Y_t^\ast, \ldots, Y_T^\ast\} \) to denote the future of this variable. Let \( Y_{it}^\ast \Xi_{i(t-1)}^{1,\ldots,T} \) (\( t = 1, \ldots, T \)) denote the WPVS for the outcome that would take at time \( t \) for person \( i \) if this person had a history of predictors \( X^\ast \) at the within-person level \( \tilde{X}_{i(t-1)}^\ast = \{X_{i1}, \ldots, X_{i(t-1)}^\ast\} \) through \( t - 1 \). \( Y_{it}^\ast \Xi_{i(t-1)} \) is a potential outcome, which we connect to WPVS by the consistency assumption

\[
Y_{it}^\ast = Y_{it}^{\ast \Xi_{i(t-1)}^{1,\ldots,T}}
\]

if \( \tilde{X}_{i(t-1)}^\ast = \tilde{x}_{i(t-1)}^\ast \); otherwise, \( Y_{it}^{\ast \Xi_{i(t-1)}^{1,\ldots,T}} \) is counterfactual. Note that \( Y_{it}^\ast \) is unobservable, and thus can be predicted in the first step of the TS method, while potential outcomes for measurements (i.e., \( Y_{it} \)) are considered in the standard potential outcome approach.

In the potential outcome approach, causal effect refers to the contrast between potential outcomes under different levels of treatments/predictors (i.e., \( X^\ast \)). The average causal effect on \( Y_{it}^\ast \) when \( X_{i(t-1)}^\ast \) increases one unit from the reference value \( x_{i(t-1)}^r \) at time \( t - 1 \) can be expressed as

\[
E(Y_{it}^{\ast \Xi_{i(t-2)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T}} + 1 - Y_{it}^{\ast \Xi_{i(t-2)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T}}) = E(Y_{it}^{\ast \Xi_{i(t-2)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T}}) - E(Y_{it}^{\ast \Xi_{i(t-2)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T}}).
\]

The standard assumption of no unobserved confounders (or sequential ignorability) indicates that

\[
\underbrace{Y_{it}^\ast \Xi_{i(t-2)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T} \Xi_{i(t-1)}^{1,\ldots,T}}_{(\tilde{x}_{i(t-2)},0)} \perp X_{i(t-1)}^\ast | \tilde{Y}_{i(t-1)}^\ast, \tilde{L}_{i(t-1)}^\ast, \tilde{X}_{i(t-2)}^\ast = \tilde{x}_{i(t-2)}^\ast.
\]

Here, \((\tilde{x}_{i(t-2)},0)\) is the counterfactual history, that is, the history that agrees with \( \tilde{x}_{i(t-2)}^\ast \) through time \( t - 2 \) and is zero thereafter. Equation (7) indicates that the potential outcome at time \( t \) (were the person receives \( X_{i(t-1)}^\ast = 0 \)) is independent of \( X_{i(t-1)}^\ast \), given the observed confounders in past time \((\tilde{X}_{i(t-2)}^\ast, \tilde{Y}_{i(t-1)}^\ast, \tilde{L}_{i(t-1)}^\ast)^\ast\). Along with the assumed causal DAG above, as well as the consistency and sequential ignorability, we impose the stable

\footnote{Though \( \tilde{Y}_{i(t-1)}^\ast \) and \( \tilde{X}_{i(t-2)}^\ast \) are the outcome and predictors, they can be characterized as confounders when it causes \( Y_{it}^\ast \), and if it either causes or associates with \( X_{i(t-1)}^\ast \).}
unit treatment value assumption (SUTVA; no unmodeled spillovers, e.g., Hong, 2015) and assumptions of positivity (i.e., the probability of taking each level of predictor ($X^*$) conditional on past variables is greater than zero) and modularity (i.e., mechanisms that are not directly targeted by treatment/predictors are not altered). Under these assumptions, the average causal effect in Equation (6) can be expressed by the difference in conditional means given by information on observed confounders as

$$E(Y_{it}^* | x_{i(t-2)}^r, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) - E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*)$$

(8)

In other words, the average causal effect can be evaluated from the difference in conditional means of $Y^*_i$ between persons who receive $X^*_{i(t-1)} = x^*_{i(t-1)} + 1$ (i.e., levels that are $x^*_{i(t-1)} + 1$ larger than their expected scores $\mu^{(X)}_{i(t-1)} + I_{i(t-1)}^X$) and $X^*_{i(t-1)} = x^*_{i(t-1)}$, given information on the observed confounders’ history.

Similarly, the average joint effects for a sequence of predictors $\tilde{X}_{i(t-1)}^*$ on $Y^*_i$ when they commonly increase one unit from the reference values $\tilde{x}^*_{i(t-1)}$ can be generally expressed as

$$E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) - E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*) = E(Y_{it}^* | \tilde{Y}^*_{i(t-1)}, \tilde{L}_{i(t-1)}, \tilde{X}_{i(t-2)}^*; x_{i(t-1)}^r, X_{i(t-1)}^*)$$

(9)

Suppose $T = 3$, and that the DGP can be represented by linear and first-order regression models, as in Equations (1) and (2) (assuming no interaction effects for WPVS). Then, a conditional mean $E(Y_{i3}^* | \tilde{Y}_{i2}^*, \tilde{L}_{i2}, \tilde{X}_{i2}^*; x_{i2}^r)$ at $t = 3$ can be expressed as a linear (weighted)
tracing the two paths start at \( x_{i1}^* \) and \( x_{i2}^* \):

\[
E(\alpha_3(Y_{i2}^*) + \beta_3 x_{i2}^* + \gamma_3 L_{i2}^* + d_{i3}^*)
= \alpha_3(\omega_2 E(Y_{i1}^*) + \beta_2 x_{i1}^* + \gamma_2 E(L_{i1}^*)) + \beta_3(\gamma_3(\omega_2 E(Y_{i1}^*) + \beta_2 x_{i1}^* + \gamma_2 E(L_{i1}^*))
= \alpha_3(\omega_2 E(Y_{i1}^*) + \gamma_2 E(L_{i1}^*)) + \beta_3(\gamma_3(\omega_2 E(Y_{i1}^*) + \gamma_2 E(L_{i1}^*))
+ \beta_2 \gamma_2 x_{i2}^* + \beta_3 \gamma_2 x_{i2}^*
= \beta_3 x_{i2}^* + \beta_3 x_{i2}^*.
\]

The first term in the third line becomes zero because WPVS have zero means. From this result, the joint effects of \( X_{i1}^* \) and \( X_{i2}^* \) when commonly increasing one unit from the reference values \( x_{i1}^{rr} \) and \( x_{i2}^{rr} \) become

\[
(\beta_{31} x_{i1}^{rr} + 1) + \beta_{32} (x_{i2}^{rr} + 1) - \beta_{31} x_{i1}^{rr} + \beta_{32} x_{i2}^{rr} = \beta_{31} + \beta_{32}.
\]

Note that \( \beta_{31} = \alpha_3 \beta_2 + \gamma_3 \beta_2 \) (Y); the effect of \( X_{i1}^* \) on \( Y_{i2}^* \) can also be evaluated by tracing the two paths \( X_{i1}^* \rightarrow Y_{i2}^* \rightarrow Y_{i3}^* \) (\( = \alpha_3 \beta_2 \)) and \( X_{i1}^* \rightarrow L_{i2}^* \rightarrow Y_{i3}^* \) (\( = \gamma_3 \beta_2 \)) that start at \( X_{i1}^* \) and end at \( Y_{i3}^* \), shown in Figure 1.

Likewise, when the effect of \( X_{i1}^* \) on \( Y_{i2}^* \) is a focus, \( E(Y_{i2}^* | Y_{i1}^*, L_{i1}^*, X_{i1}^* = x_{i1}^*) \) at \( t = 2 \) can be expressed as

\[
E(\alpha_2(Y_{i2}^*) + \beta_2 x_{i2}^* + \gamma_2 L_{i2}^*) = \alpha_2(\omega_2 E(Y_{i1}^*)) + \gamma_2 E(L_{i1}^*)) + \beta_2 x_{i2}^* = \beta_{21} x_{i1}^*.
\]

and thus the causal effect of \( X_{i1}^* \) when increasing one unit from the reference value \( x_{i1}^{rr} \) at the within-person level becomes \( \beta_{21} = \beta_{2}(Y) \), which is equivalent to the cross-lagged parameter in Equation (2). Note that because stable trait factors are not associated with the WPVS in the assumed DGP, the effects of \( X^* \) on \( Y^* \) can be numerically equivalent to those on \( Y \) (i.e., measurements) (Usami, 2022a).

Compared with other disciplines, such as epidemiology, investigations of joint effects in psychology have been limited in applications of SEM-based approaches (e.g., RI-CLPM).
where only the cross-lagged parameters gather attention from researchers. However, useful information can be gained by interpreting the effects of time-varying treatments/predictors (e.g., how a change in $X_{i(t-2)}$ at two previous time points influences $Y_{i(t)}$, fixing $X_{i(t-1)}$).

In investigating reciprocal relations, the causal effect of $Y^*$ on $X^*$ is also a focus. In a similar manner, the average causal effect of $Y_{i(t-1)}^*$ on $X_{i(t)}^*$ when $Y_{i(t-1)}^*$ increases one unit from the reference value $y_{i(t-1)}^*$ at $t-1$ can be expressed as

$$E(X_{i(t)}^* y_{i(t-1)}^* + 1 - X_{i(t)}^* y_{i(t-1)}^*)$$

(13)

The average joint effects of a sequence of $Y_{i(t-1)}^*$ on $X_{i(t)}^*$ when they commonly increase one unit from the reference values $y_{i(t-1)}^*$ can be expressed as

$$E(X_{i(t)}^* y_{i(t-1)}^* + 1) - E(X_{i(t)}^* y_{i(t-1)}^*)$$

(14)

We can now summarize the assumptions for identifying causal parameters in the TS method: (i) measurements ($T \geq 3$) are expressed by a linear sum of stable trait factors and WPVS that are mutually uncorrelated, (ii) WPVS are expressed by functions of those in past time, (iii) consistency, (iv) no unobserved confounders, (v) SUTVA, (vi) positivity, and (vii) modularity. The first assumption is unique to the TS method and RI-CLPM, and we will revisit this issue in the Discussion.

Regarding the second assumption, if the DGP can be represented by linear equations, such as in Equations (1) and (2) (assuming no interaction effects for WPVS), then RI-CLPM (that includes $L$) can identify causal parameters. However, modeling curvilinear (e.g., quadratic) and interaction effects for WPVS as latent variables can be challenging in RI-CLPM. Furthermore, all structural models (as in Equation (2)) should be correctly specified, which might be very restrictive. The risk of obtaining improper solutions can also be an obstacle when applying RI-CLPM. TS-SNMMs can overcome these potential problems: they can provide a more flexible and robust inferential framework toward model
misspecifications, resulting in more accurate estimates of causal parameters with a lower risk of improper solutions.

3 INTRODUCTION OF THE TS METHOD

In TS-SNMMs, WPVS are first calculated for each variable by SEM that only models measurement parts with stable trait factors. Causal parameters in the structural model are then estimated by SNMMs using calculated WPVS. Usami (2022a) argues that this kind of two-step approach has some strengths compared with simultaneous estimation, such as (i) minimizing the risk of potential confounding in interpreting the estimation results when either the measurement or the structural model is misspecified, (ii) having a greater feasibility for analyses (because common factors or unit effects are not explicitly modeled in SNMMs), and (iii) having less risk of obtaining improper solutions. Regarding the first point, when a simultaneous estimation procedure like RI-CLPM is used, misspecification in the structural models at the within-person level may greatly affect parameter estimates in the measurement model ((co)variances of stable factors and WPVS), and vice versa.

3.1 Step 1: Estimation of measurement models and prediction of WPVS

The first step is subdivided into two sub-steps: (i) specification and estimation of the measurement models and (ii) prediction of WPVS.

3.1.1 Specification and estimations of measurement models

The RI-CLPM and TS approaches commonly assume measurement models like Equation (1). This equation can be viewed as a model similar to the factor analysis model, which includes a single common factor $I$ (whose factor loadings are all one) and a unique
factor in the form of WPVS. In vector notation, this equation for outcome $Y$ becomes

$$Y_i = \mu^{(Y)} + I_i^{(Y)}1_T + Y_i^*; \quad (15)$$

where $\mu^{(Y)}$ is a $T \times 1$ mean vector, $E(I_i^{(Y)}) = 0$, $Var(I_i^{(Y)}) = \phi_i^2$, and $1_T$ denotes the $T \times 1$ vector whose elements are all one. $Y_i^*$ is a $T \times 1$ vector of WPVS, and $E(Y_i^*) = 0$ and $Cov(I_i^{(Y)}, Y_i^*) = 0$. We denote as $\Psi_{(Y)}$ a $T \times T$ variance-covariance matrix of WPVS. This implies that the variance-covariance matrix of $Y$ (denoted as $\Sigma_{(Y)}$) is of the form

$$\Sigma_{(Y)} = \phi_i^2 1_T1_T^T + \Psi_{(Y)}.$$

Unlike the standard factor analysis model, $\Psi_{(Y)}$ has a dependence structure and is not diagonal. Therefore, in using SEM to estimate the parameters in Equation (15), some structure, such as the autoregressive (AR) structure, must be specified in $\Psi_{(Y)}$ to enable model identification. The specified model can then be diagnosed via model fit indices.

Similarly, we also set measurement models for the other variables $X$ and $L$ separately in this sub-step, then estimate parameters for the mean vectors ($\mu^{(X)}$ and $\mu^{(L)}$), stable trait factor variances ($\phi_i^2_{(X)}$ and $\phi_i^2_{(L)}$), and variance-covariance matrices of WPVS ($\Psi_{(X)}$ and $\Psi_{(L)}$).

3.1.2 Predicting WPVS

Let $Z_i = (Y_i^t, X_i^t, L_i^t)^t$ and $Z_i^* = (Y_i^{*t}, X_i^{*t}, L_i^{*t})^t$ be vectors of measurements and WPVS, respectively, and let $\mu = (\mu^{(Y)t}, \mu^{(X)t}, \mu^{(L)t})^t$ be a mean vector. Also, let $\Sigma$ and $\Psi$ be covariance matrices for measurements $Z_i$ and WPVS $Z_i^*$.

We consider linear prediction of WPVS $\hat{Z}_i^*$ under the condition that $\Sigma$ and $\Psi$ are known. Consider a $3T \times 3T$ weight matrix $W$ that provides WPVS from measurements

$$\hat{Z}_i^* = W^t(Z_i - \mu), \quad (16)$$

satisfying the relation

$$E(\hat{Z}_i^* \hat{Z}_i^{*t}) = W^t E[(Z_i - \mu)(Z_i - \mu)^t]W = W^t \Sigma W = \Psi. \quad (17)$$
Unlike standard applications of factor analysis, we are interested in predicting WPVS (unique factor scores) rather than stable trait factor (common factor) scores. With this point in mind, the $W$ that can minimize the risk function defined as the trace of a residual covariance matrix (i.e., the mean squared error $\text{MSE}(\hat{Z}_i^* - Z_i^*)^T(\hat{Z}_i^* - Z_i^*)$) and also satisfy the relation in Equation (17) can be directly obtained by the so-called (linear) correlation preserving predictor (e.g., ten Berge, Krijnen, Wansbeek, & Shapiro, 1999 pp.317)

$$W^t = \Psi^{1/2}(\Psi^{3/2}S^{1/2})^{-1/2}\Psi^{3/2}S^{-1}. \quad (18)$$

Here, for a positive (semi)definite matrix $C$, we denote as $C^{1/2}$ the positive (semi)definite matrix whose square equals $C$. Matrices $C^{-1/2}$ and $C^{3/2}$ are the inverse (if it exists) and third power of $C^{1/2}$, respectively.

We use the sample means $\bar{Z}$ and the unbiased variance-covariance matrix $S$ as estimators of $\mu$ and $\Sigma$. As implied from the relation in Equation (4), we use the estimated stable trait factor variances to estimate $\Psi$ as

$$\hat{\Psi} = S - \hat{\Phi} \otimes 1_T 1_T^T, \quad (19)$$

where $\hat{\Phi}$ is an estimator of a $3 \times 3$ stable trait factor variance-covariance matrix $\Phi$:

$$\hat{\Phi} = \begin{pmatrix} \hat{\phi}_{(Y)}^2 & \hat{\phi}_{(Y,X)} & \hat{\phi}_{(Y,L)} \\ \hat{\phi}_{(Y,X)} & \hat{\phi}_{(X)}^2 & \hat{\phi}_{(X,L)} \\ \hat{\phi}_{(Y,L)} & \hat{\phi}_{(X,L)} & \hat{\phi}_{(L)}^2 \end{pmatrix}. \quad (20)$$

Since stable trait factor covariances are not estimated in the previous sub-step, covariances between calculated linear correlation preserving predictors from each variable are used (see Usami (2022a) for further details). From Equations (16) and (18)–(20), we can calculate WPVS $\hat{Z}_i^*$ without specifying the structural models among variables at the within-person level, successfully maintaining independence from the next step.
3.2 Applying SNMMs

SNMMs were developed in epidemiology to estimate the causal effects of a sequence of time-varying treatments/predictors in longitudinal studies, with effectively handling violation of no unobserved confounders \((U)\) under the assumed causal DAG where \(U\) influences both the outcomes \(Y\) and the observed confounders \(L\) (Robins & Hernán, 2009; Vansteelandt & Joffe, 2014). For notational simplicity, we use the symbols \(Y^*, X^*, L^*\) below to denote WPVS that are calculated in the first step.

For explanation purposes, we assume that one is interested in evaluating the joint effects of \(X^*_{t-1}\) on the outcome of the last time point \((Y_T^*)\), while allowing its curvilinear (e.g., quadratic) and interaction effects. SNMMs simulate the sequential removal of the effect (called blip) that \(X^*_{t-1}\) has on \(Y_T^*\), after having removed the effects of all subsequent treatments/predictors. More specifically, SNMMs models a blip in \(X^*_t\) on \(Y_T^*\) while holding all future treatments/predictors at \(t' \geq t\) fixed at a reference level 0 (i.e., the level that is equal to the expected scores of a person in the current context).

Linear SNMMs parameterize contrasts of \(Y^*_i, x^*_i(t-1)\) and \(Y^*_i, x^*_i(t-2); 0\) conditionally on confounder histories through \(t-1\) \((t = 2, \ldots, T)\) as

\[
E(Y^*_i, x^*_i(t-1) - Y^*_i, x^*_i(t-2); 0 | Y^*_i, x^*_i(t-1) = \bar{y}^*_i(t-1), \bar{L}^*_i(t-1), \bar{X}^*_i(t-1) = \bar{x}^*_i(t-1)) = h_t(\bar{y}^*_i(t-1), \bar{L}^*_i(t-1), \bar{x}^*_i(t-1); \tau),
\]

where \(h_t(\bar{y}^*_i(t-1), \bar{L}^*_i(t-1), \bar{x}^*_i(t-1); \tau)\) is a known function with parameter vector \(\tau\) (Vansteelandt & Joffe, 2014).

Suppose we assume a DGP similar to the one in Figure 1 that has not only the first-order (linear) effect of \(X^*\) but also its interaction effect with \(L^*\) for blip. In the empirical
example using the data for $T = 4$, a linear SNMM may be given by

$$
E(Y^*_i^{x_1, x_2, x_3} - Y^*_i^{x_1, x_2, 0}) | \bar{Y}^*_i = \bar{y}^*_i, \bar{L}^*_i = \bar{l}^*_i, \bar{X}^*_i = \bar{x}^*_i, \beta_3 = (\beta_3 + \gamma_3 l^*_i)x^*_i.
$$

$$
E(Y^*_i^{x_1, x_2, 0} - Y^*_i^{x_1, 0, 0}) | \bar{Y}^*_i = \bar{y}^*_i, \bar{L}^*_i = \bar{l}^*_i, \bar{X}^*_i = \bar{x}^*_i, \beta_2 = (\beta_2 + \gamma_2 l^*_i)x^*_i.
$$

$$
E(Y^*_i^{x_1, 0, 0} - Y^*_i^{0, 0, 0}) | \bar{Y}^*_i = \bar{y}^*_i, \bar{L}^*_i = l^*_i, \bar{X}^*_i = x^*_i, \beta_1 = (\beta_1 + \gamma_1 l^*_i)x^*_i. \tag{22}
$$

Here, the first equation models the effect of $X^*_i$ on $Y^*_i$, the second models the effect of $X^*_i$ on $Y^*_i$, and the third models the effect of $X^*_i$ on $Y^*_i$. This is just one simple example, but another specification, say $(\beta_3 + \gamma_3 l^*_i + \gamma_3 l^*_i + \gamma_3 l^*_i)X^*_i$, could be possible to express the interaction effects between $X^*_i$ and the past confounders. From Equation (22), the (conditional) average joint effects of $X^*_i$ on $Y^*_i$ when increasing one unit from the reference values in each predictor become

$$
\beta_3 + \gamma_2 l^*_i + \beta_2 + \gamma_2 l^*_i + \beta_1 + \gamma_1 l^*_i. \tag{23}
$$

If no interaction effects are assumed, this becomes

$$
\beta_{41} + \beta_{42} + \beta_{43}. \tag{24}
$$

SNMMs consider a transformation $U^*_{i(t-1)}(\tau) \ (t = 2, \ldots, T)$, the mean value of which is equal to the mean that would be observed if treatments/predictors were stopped from time $t - 1$ onward, in the sense that

$$
E(U^*_{i(t-1)}(\tau) | \bar{Y}^*_i, \bar{L}^*_i, \bar{X}^*_i, X^*_i) = \bar{X}^*_i(\tau), \tag{25}
$$

$$
E(Y^*_i X^*_i T | \bar{Y}^*_i, \bar{L}^*_i, \bar{X}^*_i, X^*_i) = \bar{X}^*_i(T), \tag{26}
$$

Here, $U^*_{i(t-1)}(\tau)$ is a vector with components $Y^*_i - \sum_{k=t-1}^{T-1} h_k(Y^*_i, \bar{L}^*_i, \bar{X}^*_i; \tau)$. For instance, in the current example for $T = 4$,

$$
U^*_{i3}(\tau) = Y^*_i - (\beta_3 + \gamma_3 l^*_i)X^*_i;
$$

$$
U^*_{i2}(\tau) = Y^*_i - (\beta_3 + \gamma_3 l^*_i)X^*_i - (\beta_2 + \gamma_2 l^*_i)X^*_i;
$$

$$
U^*_{i1}(\tau) = Y^*_i - (\beta_3 + \gamma_3 l^*_i)X^*_i - (\beta_2 + \gamma_2 l^*_i)X^*_i - (\beta_1 + \gamma_1 l^*_i)X^*_i. \tag{26}
$$
The assumption of no unobserved confounders (Equation (7)) together with the identity (Equation (25)) implies that

\[
E(U_{i(t-1)}^*(\tau)|\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-1)}^*) = E(U_{i(t-1)}^*(\tau)|\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-2)}^*),
\]

indicating the conditional independence between \(X_{i(t-1)}^*\) and \(U_{i(t-1)}^*\). In the current example for \(T = 4\), this indicates

\[
U_{i1}^* \perp X_{i1}^*|Y_{i1}^*, L_{i1}^*, \quad U_{i2}^* \perp X_{i2}^*|\tilde{Y}_{i2}^*, \tilde{L}_{i2}^*, X_{i1}^*, \quad U_{i3}^* \perp X_{i3}^*|\tilde{Y}_{i3}^*, \tilde{L}_{i3}^*, \tilde{X}_{i2}^*.
\]

The parameters \(\tau\) can be estimated from these moment conditions by solving the estimating equation \(E_N[f(\tau; \hat{\eta}; \hat{\kappa})] = 0\), where \(E_N\) denotes the empirical average function:

\[
f(\tau; \hat{\eta}; \hat{\kappa}) = \sum_{t=2}^{T}(d_{t-1}(\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-1)}^*) - E(d_{t-1}(\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-1)}^*)|\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-2)}^*))\circ
\]

\[
[U_{i(t-1)}^*(\tau) - E(U_{i(t-1)}^*(\tau)|\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-2)}^*)],
\]

where

\[
d_{t-1}(\tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-1)}^*) = E \left[ \frac{\partial U_{i(t-1)}^*(\tau)}{\partial \tau_{i(t-1)}} \left| \tilde{Y}_{i(t-1)}^*, \tilde{L}_{i(t-1)}^*, \tilde{X}_{i(t-1)}^* \right. \right].
\]

Here, \(\tau_{i(t-1)}\) denotes the elements in \(\tau\) that are relevant to the assumption of conditional independence (Equation (28)) at \(t - 1\). For instance, in the current example for \(T = 4\), we can apply Equation (26) to obtain

\[
d_3(\tilde{Y}_{i3}^*, \tilde{L}_{i3}^*, \tilde{X}_{i3}^*) = E \left[ \frac{\partial U_{i3}^*(\tau)}{\partial \beta_3} \left| \tilde{Y}_{i3}^*, \tilde{L}_{i3}^*, \tilde{X}_{i3}^* \right. \right] = \left( \frac{\partial U_{i3}^*(\tau)}{\partial \beta_3}, \frac{\partial U_{i3}^*(\tau)}{\partial \gamma_3} \right)^t
\]

\[
= \left( -X_{i3}^*, \beta_3 + \gamma_3 L_{i3}^* X_{i3}^* \right)^t
\]

\[
d_2(\tilde{Y}_{i2}^*, \tilde{L}_{i2}^*, \tilde{X}_{i2}^*) = \left( \frac{\partial U_{i2}^*(\tau)}{\partial \beta_2}, \frac{\partial U_{i2}^*(\tau)}{\partial \gamma_2} \right)^t = (-X_{i2}^*, -L_{i2}^* X_{i2}^*)^t
\]

\[
d_1(\tilde{Y}_{i1}^*, \tilde{L}_{i1}^*, X_{i1}^*) = \left( \frac{\partial U_{i1}^*(\tau)}{\partial \beta_1}, \frac{\partial U_{i1}^*(\tau)}{\partial \gamma_1} \right)^t = (-X_{i1}^*, -L_{i1}^* X_{i1}^*)^t.
\]
This estimating equation essentially sets the sum across the time points of the conditional covariances between $U_{it(t-1)}(\tau)$ and the function $d_{t-1}(\bar{Y}_{it(t-1)}, \bar{L}_{it(t-1)}, \bar{X}_{it(t-1)}^*)$, given confounders, to zero. More specifically, in this example, the elements in Equation (29) essentially become

$$[X_{i3}^* - E(X_{i3}^*|\bar{Y}_{i3}^*, \bar{L}_{i3}^*, \bar{X}_{i2}^*)] \times [U_{i3}^*(\tau) - E(U_{i3}^*(\tau)|\bar{Y}_{i3}^*, \bar{L}_{i3}^*, \bar{X}_{i2}^*)]$$

$$[L_{i3}^*X_{i3}^* - L_{i3}^*E(X_{i3}^*|\bar{Y}_{i3}^*, \bar{L}_{i3}^*, \bar{X}_{i2}^*)] \times [U_{i3}^*(\tau) - E(U_{i3}^*(\tau)|\bar{Y}_{i3}^*, \bar{L}_{i3}^*, \bar{X}_{i2}^*)]$$

$$[X_{i2}^* - E(X_{i2}^*|\bar{Y}_{i2}^*, \bar{L}_{i2}^*, \bar{X}_{i1}^*)] \times [U_{i2}^*(\tau) - E(U_{i2}^*(\tau)|\bar{Y}_{i2}^*, \bar{L}_{i2}^*, \bar{X}_{i1}^*)]$$

$$[L_{i2}^*X_{i2}^* - L_{i2}^*E(X_{i2}^*|\bar{Y}_{i2}^*, \bar{L}_{i2}^*, \bar{X}_{i1}^*)] \times [U_{i2}^*(\tau) - E(U_{i2}^*(\tau)|\bar{Y}_{i2}^*, \bar{L}_{i2}^*, \bar{X}_{i1}^*)]$$

$$[X_{i1}^* - E(X_{i1}^*|L_{i1}^*, L_{i1}^*)] \times [U_{i1}^*(\tau) - E(U_{i1}^*(\tau)|Y_{i1}^*, L_{i1}^*)]$$

$$[L_{i1}^*X_{i1}^* - L_{i1}^*E(X_{i1}^*|Y_{i1}^*, L_{i1}^*)] \times [U_{i1}^*(\tau) - E(U_{i1}^*(\tau)|Y_{i1}^*, L_{i1}^*)].$$

If homoscedasticity of the conditional variance $U_{it(t-1)}^*(\tau)$ given confounders is satisfied, the local semiparametric efficiency under the SNMM is attained by choosing $d_{t-1}(\bar{Y}_{it(t-1)}, \bar{L}_{it(t-1)}, \bar{X}_{it(t-1)}^*)$, as in Equation (30) (Vansteelandt & Joffe, 2014).

As seen in Equation (32), solving the estimating equation (29) requires a **treatment model** $\mathcal{A}$ for the treatment/predictor $X_{it(t-1)}^*$: $f(X_{it(t-1)}^*|\bar{Y}_{it(t-1)}^*, \bar{L}_{it(t-1)}^*, \bar{X}_{it(t-2)}^*; \eta)$. It also requires a **treatment-free model** $\mathcal{B}$ (Wallace et al., 2017b) for the conditional mean of $U_{it(t-1)}^*(\tau)$, namely, $f(U_{it(t-1)}^*(\tau)|\bar{Y}_{it(t-1)}^*, \bar{L}_{it(t-1)}^*, \bar{X}_{it(t-2)}^*; \kappa)$. Notably, when the parameters $\eta$ and $\kappa$ are variation-independent, the estimator that solves Equation (29) (called $G$-estimator), obtained by substituting $\eta$ and $\kappa$ with consistent estimators, are doubly robust (Robins & Rotnitzky, 2001, cited from Vansteelandt & Joffe, 2014), meaning that estimates of causal parameters are consistent when either model $\mathcal{A}$ or model $\mathcal{B}$ is correctly specified.

Though we have supposed situations with joint effects of $\bar{X}_3^*$ on $Y_4^*$, the effects of $X_{t-1}^*$ on $Y_t^*$ for $t < 4$ can also be modeled and estimated in a similar manner. Likewise, the opposite relation for the effects of $\bar{Y}_{t-1}^*$ on $X_t^*$ can also be investigated with a separate procedure.

To summarize, in the second step of using (linear) SNMMs, we first specify the blip
model (e.g., Equation (22)) for time-varying treatments/predictors that may have curvilinear and interaction effects (e.g., $X_{t-1}^2$ and $X_{t-1}^* L_{t-1}^*$). We then consider a transformation $U_{i(t-1)}^*(\tau)$ (e.g., Equation (26)), and the parameters for blip ($\tau$) are estimated based on moment conditions implied by conditional independence (e.g., Equation (28)). When estimating the parameters, the treatment model $A$ and the treatment-free model $B$ need to be specified based on the assumed DGP, but SNMMs have the property of being doubly robust. We further explain below the specifications of these models using the R package DTRreg (Wallace et al., 2017b).

### 3.3 SNMMs via the R package DTRreg

The framework of SNMMs is usually presented using estimation equations like Equation (29). However, Wallace et al (2017a) explained that the same calculation may in fact be conducted using a relatively straightforward series of matrix equations (see Wallace & Moodie, 2015, and the Web Appendix provided by Wallace et al., 2017a for more details). More specifically, the final stage (i.e., $t = T$) blip parameters are estimated first, before working backwards until every stage at $t$ has been analyzed. By working recursively, we are able to calculate each potential outcome by plugging in all future blip parameters (the Web Appendix in Wallace et al., 2017a).

Suppose we assume a DGP similar to that in Figure 1, and first-order (linear) effects of $X^*$ and its interaction effect with $L^*$ are assumed for the blip model in $T = 4$, as in Equation (22). $\beta_3$ and $\gamma_3$ (i.e., the effects associated with $X_{i3}^*$ on $Y_{i4}^*$) are first estimated using the moment condition induced by the conditional independence between $X_{i3}^*$ and $U_{i3}^*$ (Equation (28)). After plugging in these estimates, $\beta_2$ and $\gamma_2$ are estimated next based on the conditional independence between $X_{i2}^*$ and $U_{i2}^*$. Finally, using the estimates obtained so far, $\beta_1$ and $\gamma_1$ are estimated based on the conditional independence between $X_{i1}^*$ and $U_{i1}^*$.

---

2[http://links.lww.com/EDE/B134](http://links.lww.com/EDE/B134)
Even if we take this matrix equations approach, specifications of the treatment model $A$ and the treatment-free model $B$ are required. These models are expressed in the R package DTRreg as `treat.mod` and `tf.mod`, respectively.

In the current example, from the assumed DGP $A$ can be specified as an (linear) AR(1) model. If a researcher is interested in the joint effect of $X_3^*$ on $Y_4^*$, `treat.mod` may be specified in the R package DTRreg as

```r
treat.mod <- list(X1~1, X2~X1+L1+Y1, X3~X2+L2+Y2).
```

Note that `X1~1` indicates that only an intercept is included because $X_1^*$ is now treated as an exogenous variable. In $B$, because the outcomes $Y^*$ are modeled by the similar (linear) AR(1) model (with interaction effects between $X^*$ and $L^*$) in the assumed DGP, if $X_3^* = 0$, then the conditional means of $Y_3^*$ used for $U_3(\tau)$ (given $\bar{Y}_3^*, \bar{L}_3^*$, and $\bar{X}_3^*$) can be expressed by a linear (weighted) sum of $Y_3^*$ and $L_3^*$. Next, if $X_2^* = X_3^* = 0$, then the conditional means of $Y_4^*$ used for $U_2(\tau)$ (given $\bar{Y}_2^*, \bar{L}_2^*$, and $\bar{X}_1^*$) becomes a linear (weighted) sum of $Y_2^*$ and $L_2^*$. Likewise, if $\bar{X}_3^* = 0$, then the conditional means of $Y_4^*$ used for $U_1(\tau)$ (given $Y_1^*$ and $L_1^*$) becomes a linear (weighted) sum of $Y_1^*$ and $L_1^*$. Thus, `tf.mod` can be specified in the R package DTRreg as

```r
tf.mod <- list(~Y1+L1, ~Y2+L2, ~Y3+L3).
```

Note that one can allow curvilinear and interaction effects for WPVS in $A$ and $B$, but this can be challenging when using RI-CLPM as covariance structure analysis.

In addition to these, the blip model needs to be specified in the R package DTRreg. Since the interaction effect between $X^*$ and $L^*$ is now assumed, the blip model (expressed as `blip.mod`) can be expressed as

```r
blip.mod <- list(~L1, ~L2, ~L3).
```

If no interaction effect is assumed, then the specification becomes

```r
blip.mod <- list(~1, ~1, ~1).
```
The causal parameters $\tau$ can now be estimated in the R package DTRreg by three models specified so far:

```r
mod<-DTRreg(Y4, blip.mod, treat.mod, tf.mod, var.est="sandwich", type="alt"),
```

where the option `var.est="sandwich"` specifies the robust (or sandwich) variance estimator obtained using standard estimating equation theory (Web Appendix in Wallace et al., 2017a; see Hardin & Hilbe, 2013 pp.30-34 for further details). Though nuisance parameters $\eta$ and $\kappa$ need to be accounted for to obtain better estimates of standard errors, Wallace et al. (2017a) explained that sandwich estimators that ignore the nuisance parameter estimation typically perform as well as the bootstrap or the nuisance parameter corrected standard error. The package DTRreg also offers a variety of more complex options, including bootstrap, and it can be specified by a combination of `var.est="boot"` and `B=n` for the number of bootstrap replications (Wallace et al., 2017b).

Using the command `type="alt"`, `summary(mod)` and `coef(mod)` return summaries and blip parameter estimates in a fashion similar to more familiar commands like `lm` and `glm` (Web Appendix in Wallace et al., 2017a). Additionally, the package DTRreg will automatically ignore any persons with missing data (thereby carrying out a complete-cases analysis), but if the option `missing = "ipcw"` is specified, then the inverse probability of censored weights (e.g., Hernan & Robins, 2021) is used. The probability of censoring is estimated via logistic regression on the full covariate history up to that point (Web Appendix in Wallace et al., 2017a).

## 4 SIMULATIONS

There are two main goals in the simulations. The first is to investigate the performance of TS-SNMMs under various data-generating conditions in which data are generated by a linear sum of stable trait factors and WPVS, as in RI-CLPM. In the first simulation, we
assume there are no misspecifications in the structural models. The second is to demonstrate the robustness of TS-SNMMs and to compare its performance to RI-CLPM under the presence of unobserved time-varying confounders $U^*$ (that influence outcomes $Y^*$ and observed time-varying confounders $L^*$) and model misspecifications caused by observed time-varying confounders $L^*$ (whose direct second-order effect on the outcome is missing) in the structural model. Second and third simulations are performed for this purpose.

Throughout the simulation, we suppose a situation where researchers want to evaluate the joint effects of $X_{i(T-2)}^*$ and $X_{i(T-1)}^*$ (we do not focus on the effects from $X_{i(T-3)}^*$) on $Y_{iT}^*$, and vice versa for the reciprocal relation. For simplicity, we also assume that the interaction effects of predictors with observed confounders are not present.

### 4.1 Scenario 1: No misspecifications in the structural models

#### 4.1.1 Method

Three stable trait factors are first generated by the multivariate normal:

$$
\begin{pmatrix}
I_t^{(Y)}
I_t^{(X)}
I_t^{(L)}
\end{pmatrix} = MN \begin{pmatrix}
0 & 0 & 0
0 & \phi^2 & \phi^2_rB & \phi^2_rB
0 & \phi^2_rB & \phi^2 & \phi^2_rB
\end{pmatrix},
$$

(33)

where $\phi^2$ is a stable trait factor variance and $r_B$ is a correlation between factors. The initial WPVS are generated independently from the stable trait factors as

$$
\begin{pmatrix}
Y_{i1}^*
X_{i1}^*
L_{i1}^*
\end{pmatrix} = MN \begin{pmatrix}
0 & 0 & 0
(1-\phi^2) & (1-\phi^2)r_W & (1-\phi^2)r_W
(1-\phi^2)r_W & (1-\phi^2) & (1-\phi^2)r_W
\end{pmatrix},
$$

(34)

where $r_W$ is a correlation among the initial WPVS. Then, the WPVS at successive times are sequentially generated via a first-order linear regression model with time-invariant co-
the correlations between initial WPVS to \( \phi \) \( 20 \) \( 60 \); where temporal group means are set to zero at each time point for each variable.

Like RI-CLPM, measurements are then generated as 

\[
E(Y_{i3}^{*\hat{y}_{i2}}) = E(0.30Y_{i2}^{*} + 0.20L_{i2}^{*}) + 0.20x_{i2}^{*} = (0.06x_{i1}^{*} + 0.04x_{i1}^{*}) + 0.20x_{i2}^{*} = 0.10x_{i1}^{*} + 0.20x_{i2}^{*}
\]

\[
E(X_{i3}^{*\hat{y}_{i2}}) = 0.10y_{i1}^{*} + 0.20y_{i2}^{*}.
\]

More generally, the joint effects of \( X_{i(t-2)}^{*} \) and \( X_{i(t-1)}^{*} \) (with \( \hat{X}_{i(t-3)}^{*} \) not manipulated) on \( Y_{it}^{*} \) can be evaluated as 

\[
E(Y_{it}^{*X_{i(t-2)}^{*}X_{i(t-1)}^{*}}) = 0.10x_{i(t-2)}^{*} + 0.20x_{i(t-1)}^{*}.
\]

Similarly, 

\[
E(X_{it}^{*X_{i(t-2)}^{*}X_{i(t-1)}^{*}}) = 0.10y_{i(t-2)}^{*} + 0.20y_{i(t-1)}^{*}.
\]

Therefore, accurately estimating these four coefficients \((0.10,0.20,0.10,0.20)^t\) for within-person reciprocal relations is a shared goal between TS-SNMMs and RI-CLPM.

The variance of the normal residual \( d \) for each variable was set so that ratio of the variance of WPVS at time \( t \) (e.g., \( \text{var}(Y_{it}^{*}) \) for \( Y \)) to that at time \( t = 1 \) (e.g., \( \text{var}(Y_{i1}^{*}) \)) becomes \( \text{var}(Y_{it}^{*})/\text{var}(Y_{i1}^{*}) = \frac{T-t+(t-1)\omega}{T-1} \). More specifically, when \( t = T \), this ratio becomes \( \omega \) (i.e., \( \omega = \text{var}(Y_{iT}^{*})/\text{var}(Y_{i1}^{*}) \)). When \( \omega = 1 \), the variance of WPVS is constant over time. Note that residual variances were controlled over time in the simulations of Usami (2022a).

Like RI-CLPM, measurements are then generated as 

\[
Y_{it} = I_{i}^{(Y)} + Y_{it}^{*}, \quad X_{it} = I_{i}^{(X)} + X_{it}^{*}, \quad L_{it} = I_{i}^{(L)} + L_{it}^{*},
\]

where temporal group means are set to zero at each time point for each variable.

In this simulation, we systematically changed the total number of persons to \( N = 200,600,1000 \), the number of time points to \( T = 3,4,6,9 \), the variances of stable trait factors to \( \phi^2 = 0.1,0.4,0.7 \), the correlations between stable trait factors to \( r_B = 0.1,0.3,0.5 \), the correlations between initial WPVS to \( r_w = 0.1,0.3,0.5 \), and the ratio of the variance
of WPVS at time $t = T$ to that at time $t = 1$ to $\omega = 1, 3, 5$. Some major differences from the simulations in Usami (2022a) are that more varied specifications of $T$ and the manipulations of $r_B$, $r_W$, and $\omega$. By crossing these factors, we generated 200 simulation data for each combination of factors. Under each simulation condition, we calculated the bias and root mean squared error (RMSE) of estimates from TS-SNMMs and RI-CLPM (which assumes first-order lagged regressions that include $L^*$).

In the first step of TS-SNMMs, a model that assumes a linear AR(1) structure with time-varying autoregressive coefficients and residual variances was specified for the WPVS in each variable when $T = 3$. The models with similar AR(2) and AR(3) structures were also specified in the $T = 4$ and $T = 6, 9$ conditions, respectively. Note that in the current DGP, a true measurement model for each variable has an AR($T - 1$) structure in WPVS, but such a measurement model cannot be identified. Therefore, the measurement model is more or less misspecified in the current setting. In the second step, both the treatment model $A$ and the treatment-free models $B$ were correctly specified. For example, in $T = 9$ (the joint effects of $X_7^*$ and $X_8^*$ on $Y_9^*$), three models are specified in the package DTRreg as

```r
treat.mod <- list(X7~X6+L6+Y6, X8~X7+L7+Y7)
tf.mod <- list(~Y7+L7, ~Y8+L8)
blip.mod <- list(~1, ~1).
```

Estimation results are discarded when improper solutions because of out-of-range parameter estimates (e.g., negative variance or the singular Hessian matrix for trait factors) occur when applying either TS-SNMMs or RI-CLPM. The simulation was conducted in R using the package lavaan (Rosseel, 2012) to estimate parameters with MLE in RI-CLPM and in measurement models for the first step of TS-SNMMs. The package DTRreg was used in the second step of TS-SNMMs. The simulation code is available in the Online Supplemental Material.
4.1.2 Results

Figure 2 shows biases of causal parameter estimates from the TS-SNMMs (with an AR(1) measurement model in the first step) and RI-CLPM. Because similar tendencies were observed, we provide results for the effects of $X^*$ on $Y^*$ (the sizes of the first-order effect from $X_{T-1}^*$ and the second-order effect from $X_{T-2}^*$ are 0.20 and 0.10, respectively), and those of $Y^*$ on $X^*$ are provided in the Online Supplemental Material (Figures S10-S11). Also, the impacts of $N$ and $r_W$ were not relatively large in terms of biases, especially in the second-order effect estimates, which exhibited a larger variability than the first-order effect estimates (see Table S1 of the ANOVA results, where calculated biases are set as outcomes). Here, we provide results from $N = 1000$ and $r_W = 0.3$, and we confirm that whole conclusions are not influenced by differences in $N$ and $r_W$ (Figures S2-S5).

Since the data were generated by the process assumed in RI-CLPM, RI-CLPM can accurately estimate the parameters. However, since improper solutions often arise, especially when $N$ is small or $\phi^2 = 0.1$ (i.e., small true stable trait variances cause negative estimates), small (negative) biases occur in RI-CLPM as a result of discarding estimates with improper solutions. The magnitudes of RMSEs were similar between TS-SNMMs and RI-CLPM (Figures S1,S6-S9), so we will focus particularly on the results of biases below.

TS-SNMMs show biases in some conditions because of the misspecified measurement model in the first step. However, these biases become smaller as $T$ becomes larger and $\phi^2$ becomes smaller. When $T$ is as small as $T = 3$, TS-SNMMs show large biases in many conditions (e.g., the relative biases over true effects exceed 10%), so even though TS-SNMMs can be used in $T = 3$ for some conditions, such as $\phi^2 = 0.1$, it is generally advisable not to do so. On the other hand, when $T \geq 4$, biases were within 10% of the relative biases over true effects in most conditions, the exceptions being when trait factor correlations were small and the proportions of variances explained by within-person fluctuations (i.e., WPVS) were kept small over time (i.e., large $\phi^2 = 0.4, 0.7$ and small $r_B = 0.1$ and $\omega = 1$).
The overall results become more accurate when $T \geq 6$.

When an AR(2) measurement model was used in TS-SNMMs for $T = 4$, the positive biases observed in some conditions were mitigated, but the overall differences were almost ignorable (see Figures S12-S13 in the Online Supplemental Material). The same tendencies were observed when either AR(2) or AR(3) measurement models were used in $T \geq 6$. Therefore, the choice of AR order in the measurement model does not have a large impact on biases, at least in the current parameters setting.

Table 1 provides the proportions of improper solutions calculated when a total of 200 proper solutions were obtained in each condition. It shows that improper solutions very often arise in RI-CLPM, especially when $\phi^2$ is small, but also that the sizes of both $N$ and $T$ have large impacts. RI-CLPM produces improper solutions more frequently than TS-SNMMs in all conditions. This difference results from the fact that TS-SNMMs calculate WPVS from the measurement model of each variable, while RI-CLPM estimates structural models and measurement models for all variables simultaneously. With the exception of $\phi^2 = 0.1$, improper solutions arise in less than 1% of the simulations in TS-SNMMs when $T \geq 4$. When $T = 4, 6$ and $\phi^2=0.1$, improper solutions arise in more than 15% in all conditions, even if $N$ becomes larger in RI-CLPM. In cases of small $N$, the risk of improper solutions in RI-CLPM becomes an issue if either $T$ is small ($T \leq 4$) or $\phi^2$ is small ($\phi^2=0.1$).

4.2 Scenario 2: Unobserved confounders that influence observed time-varying confounders and outcomes

4.2.1 Method

We consider the DGP in which an unobserved time-varying confounder $U^*$ influences both the observed time-varying confounders $L^*$ and the outcomes $Y^*$. In this scenario, controlling for $L^*$ (as a collider) to infer the causal effects of $X^*$ on $Y^*$ introduces bias through $U^*$ on the path from $X^*$ to $Y^*$ (i.e., $X^*_{T-2} \rightarrow L^*_T \leftarrow U^*_{T-2} \rightarrow U^*_T \rightarrow Y^*_T$). This is known as
collider bias, and this DGP is in line with the one typically assumed in the epidemiological literature on MSMs and SNMMs (e.g., Robins & Hernán, 2009; Vansteelandt & Joffe, 2014). Such a DGP might be assumed when researchers perform a sequential randomized trial and when the treatment model can be correctly specified (i.e., one is sure how the treatments/predictors $X^*$ can be explained by other observed variables, but the outcomes $Y^*$ and the observed confounders $L^*$ may be influenced by unobserved confounders $U^*$).

For this DGP, the AR(1) process is assumed in the model for $U^*$ as $U^*_{i(t-1)} = 0.7U^*_{i(t-1)} + d^{(U)}_{it}$, and the variance of the residual $d^{(U)}_{it}$ is manipulated according to $\omega$. Y* and $L^*$ are then generated as

$$Y^*_{it} = 0.30Y^*_{i(t-1)} + 0.20X^*_{i(t-1)} + 0.20L^*_{i(t-1)} + 0.30U^*_{i(t-1)} + d^{(Y)}_{it}$$

$$L^*_{it} = 0.20Y^*_{i(t-1)} + 0.20X^*_{i(t-1)} + 0.30L^*_{i(t-1)} + 0.30U^*_{i(t-1)} + d^{(L)}_{it}, \quad (38)$$

while $X^*$ is generated as in the previous simulation. Both the treatment model $A$ and the treatment-free model $B$ were specified as in the previous simulation, and we compared the estimation performances of TS-SNMMs and RI-CLPM that lacked $U^*$. Namely, in TS-SNMMs $B$ is misspecified in inferring effects from $X^*$ on $Y^*$, whereas $A$ is still correctly specified. Thus, TS-SNMMs can take doubly robust property.

4.2.2 Results

Figure 3 shows the biases of estimates for the effects of $X^*$ on $Y^*$ in TS-SNMMs (that assume an AR(1) measurement model in the first step) and RI-CLPM. Because the impacts of $N$ and $r_W$ on the results were again smaller in terms of biases, we provide the results for $N = 1000$ and $r_W = 0.3$. We also confirm again that the choice of AR order in the measurement model does not have a large impact on biases in TS-SNMMs (Figure S14). From Figure 3, we can see that TS-SNMMs show a similar amount of biases in each condition to those in the previous simulation. Namely, with the exception of the specific conditions shown above (large $\phi^2 = 0.4, 0.7$ and small $r_B = 0.1$ and $\omega = 1$), the biases were
small in $T = 4$, and the overall results become more accurate as $T$ became larger.

On the other hand, notable positive biases were observed in RI-CLPM for the second-order effect of $X^*$. With the exception of the conditions specified above, the biases became larger in RI-CLPM than those in TS-SNMMs in $T \geq 6$, and RI-CLPM exhibited more than a 10% level of relative biases over the true effects in all conditions. This demonstrates that estimates from RI-CLPM can be seriously biased by unobserved confounders (that influences both observed confounders and outcomes), while TS-SNMMs are robust to this influence. Note that because the first-order effect is not influenced by collider bias in the current DGP, except for the conditions specified above, estimation performances are competitive between methods.

These methods are also competitive in estimates for the second-order effects of $Y^*$ on $X^*$, but they commonly show small amounts of biases (Figure S15) because in TS-SNMMs both $A$ and $B$ are misspecified in the current DGP. More specifically, the model for $Y^*_{T-2}$ is influenced by the previous $U^*$, and so too is $X^*_T$ via the causal path $U^*_{T-2} \rightarrow L^*_{T-1} \rightarrow X^*_T$. Therefore, in the current DGP, TS-SNMMs can more accurately estimate the effects of $X^*$ on $Y^*$ than RI-CLPM, but not the effects of $Y^*$ on $X^*$.

As shown in Table S2, similar tendencies were observed for the frequency of improper solutions as in the previous simulation. RI-CLPM very often produces improper solutions: at least 30% of all cases when $\phi^2$ is as small as 0.1. Even for larger $\phi^2$, when $T$ and $N$ are as small as $T = 4,6$ and $N = 200$, the observed proportions were around 10%. On the other hand, the risk of obtaining improper solutions in TS-SNMMs was much smaller than RI-CLPM.

One might consider that when such unobserved confounders exist, one can avoid referring to such biased estimation results from RI-CLPM by diagnosing some model fit indices. However, in the current scenario, 98.06% of the estimation results from RI-CLPM showed an SRMR lower than .05. Likewise, 99.97% and 97.23% of the results from RI-CLPM showed a CFI larger than .95 and an RMSEA lower than .05, respectively (see Table S3).
These proportions exceeded 70–80% on average, even when $T$ and $N$ were small. Therefore, it would be very difficult to detect collider biases caused by $U^*$ through these model fit indices alone.

### 4.3 Scenario 3: Ignored direct higher-order effects of observed time-varying confounders on outcomes

#### 4.3.1 Method

In this scenario, we assume that there are ignored direct higher-order effects of $L^*$ on $Y^*$. Although a first-order lagged effect is often assumed when applying the (RI-)CLPM, an ignored (direct) higher-order effect leads to biased causal estimates unless it is zero, and assessing the number of order is a major task in inferring within-person relations. However, as the numbers of time points and observed time-varying confounders increase, this task becomes more complex, and the risk of misspecifications can increase.

In this scenario, $Y^*$ is generated as

$$ Y_{it}^* = 0.30Y_{i(t-1)}^* + 0.20X_{i(t-1)}^* + 0.20L_{i(t-1)}^* + 0.20L_{i(t-2)}^* + d_{it}^{(Y)}, \quad (39) $$

while $X^*$ and $L^*$ are generated similarly as in the first simulation. Because $L_{i(t-2)}^*$ influences both $X_{i(T-1)}^*$ and $Y_{T}^*$, it is obviously a confounder in evaluating the relation between $X_{i(T-1)}^*$ and $Y_{T}^*$, and we expect ignoring $L_{i(T-2)}^*$ in structural models for $Y_{T}^*$ leads to biased causal estimates in RI-CLPM. Both the $\mathcal{A}$ and $\mathcal{B}$ models were specified as in the first simulation, and we compared the estimation results between TS-SNMMs and RI-CLPM that did not include $L_{i(t-2)}^*$. Namely, $\mathcal{B}$ was misspecified, but $\mathcal{A}$ was still correctly specified when estimating the effects of $X^*$ on $Y^*$ in TS-SNMMs.
4.3.2 Results

Figure 4 shows the biases of causal effect estimates of $X^*$ on $Y^*$ in TS-SNMMs (that assume an AR(1) measurement model in the first step) and RI-CLPM. Since the impacts of $N$ and $r_W$ on the results were again smaller in terms of biases, we provide here the results when $N = 1000$ and $r_W = 0.3$. We also confirmed again that the choice of AR order in the measurement model does not have a large impact on biases in TS-SNMMs (Figure S16). From Figure 4, we can see that TS-SNMMs show a similar amount of biases in each condition as the first simulation (i.e., Figure 2). Therefore, with the exception of the conditions mentioned above, the biases were small in $T \geq 4$, but the overall results became more accurate when $T \geq 6$.

Notably, both the first- and second-order effects have large positive biases (that exceed a 10% level of relative biases over true effects) in RI-CLPM, and the amounts of biases in RI-CLPM exceed those in TS-SNMMs in most conditions when $T \geq 4$. This clearly indicates that causal estimates from RI-CLPM can be seriously biased by model misspecification in the form of omitted direct higher-order lagged effects of $L^*$ in the structural models, even if there are no other variables as unobserved confounders $U^*$ in the DGP. On the other hand, estimates for the opposite relation ($Y^*$ on $X^*$) were similar to those in Figure 2 for both methods due to there being no ignored direct higher-order effects in the structural model of $X^*$ (Figure S17).

As shown in Table S4, similar tendencies were observed for the frequency of improper solutions as in the previous simulations. RI-CLPM often produces improper solutions when $\phi^2$ is as small as 0.1. Even for larger $\phi^2$, when $T$ and $N$ are small as $T = 4$ and $N = 200$, the observed proportions were more than 10%.

Like the previous simulation, we evaluated how model fit indices work to avoid referring to estimation results from RI-CLPM. However, 94.63% of the estimation results from RI-CLPM (that ignore direct second-order lagged effects) showed an SRMR lower than .05.
Likewise, 99.09% and 87.06% showed a CFI larger than .95 and an RMSEA lower than .05, respectively (see Table S5). These proportions exceeded 30-50% on average, even when $T$ and $N$ were small. Therefore, it seems very difficult to detect estimation biases caused by misspecification in the form of omitted direct higher-order lagged effects of $L^*$ in the structural model by using these model fit indices.

We have demonstrated that TS-SNMMs can be effectively used in many conditions where longitudinal data with $T \geq 4$ are available, and that more accurate causal estimates can be obtained under some situations even if unobserved time-varying confounders and model misspecifications exist, with a lower risk of obtaining improper solutions compared with RI-CLPM.

## 5 EMPIRICAL APPLICATION

In this section, we describe an empirical application of TS-SNMMs using data from the Tokyo Teen Cohort (TTC) study (Ando et al., 2019), which was also used in Usami (2022a) with $T = 3$ waves. We assume a similar causal DAG model to that in Figure 1. Namely, we assume that measurements are expressed by the linear sum of stable trait factors and WPVS. TTC is a longitudinal cohort study for investigating the psychological and physical development of ($N=3,171$) adolescents in the Tokyo metropolitan area, and data for which have been gathered in $T = 4$ waves: from 2012 to 2015 (age 10), from 2014 to 2017 (age 12), from 2017 to 2019 (age 14), and from 2020 to 2022 (age 16).

The focus of this analysis is on the reciprocal relations between sleep duration ($X^*$) and depressive symptoms ($Y^*$), and we estimate the effects that past sleep duration ($X^*$) at ages 10, 12, and 14 has on later depressive symptoms ($Y^*$) at age 16 (measured by the Short Mood and Feelings Questionnaire; SMFQ, Angold et al., 1995), and vice versa, based on the $T = 4$ waves. Several epidemiological studies have suggested a relationship between sleep habits (sleep duration, bedtime, and bedtime regularity) and mental health status
(depression and anxiety) in adolescents, and their reciprocal relations were investigated by Matamura et al. (2014). However, this study did not account for unit effects (i.e. stable traits) in sleep duration and depressive symptoms, and the relation at the within-person level was also not investigated. In inferring the effects of sleep duration on depressive symptoms, the statistical control of other sleep habits like bedtime may be key. However, as illustrated in the previous simulation, controlling for observed confounders \( L^* \) can introduce collider bias via unobserved confounders \( U^* \) that influence both \( L^* \) and the outcome \( Y^* \). In this example, some life habits and the home environment (e.g., discipline from parents, engagement in clubs/extracurricular activities in school) might affect the level of depressive symptoms and bedtime. Therefore, the use of TS-SNMMs can be considered a reasonable way to robustly estimate the causal effects of sleep duration over time.

The SMFQ consists of 13 items that assess depressive symptoms (0: not true, 1: sometimes true, 2: true) related to feelings and actions over the preceding two weeks. Higher SMFQ scores suggest more severe symptoms. Sleep duration in hours was measured by the question “How long do you usually sleep on weekdays?” Bedtime was used as observed confounders (\( L^* \)), which was measured by the question “When do you usually go to bed on weekdays?” In the present example, we focus on \( N = 1,294 \) adolescents who consistently responded to these items during four waves. Descriptive statistics of these variables are available in Table S6.

In the first step, we estimated the measurement model that assumes an AR(1) structure with time-varying autoregressive coefficients and residual variances in WPVS by MLE, and WPVS were calculated for each variable. In the second step, we used the calculated WPVS to estimate the joint effect of sleep duration at ages 10, 12, and 14 on depressive symptoms at age 16 (i.e., the joint effect of \( X_1^*, X_2^*, X_3^* \) on \( Y_4^* \)), and vice versa. Models \( A \) and \( B \) were both specified based on first-order linear regression models (see the specifications in the package DTRreg shown in Subsection 3.3). For the blip model, we specified one that assumes interaction effects between predictors and observed confounders (e.g., \( X_3^* L_3^* \) and
$X_3^*Y_3^*$ on $Y_4^*$). For comparison, we also applied RI-CLPM assuming AR(1) regressions that include $L^*$ (i.e., Equation (2)).

We confirmed that the first step did not produce improper solutions for each variable, and that the specified AR(1) measurement model shows good fit in terms of the model fit indices. Table S7 summarizes the model fit indices for each variable. Proportions of the variances in measurements attributable to estimated stable trait factors at an initial time (like the $\phi^2$ manipulated in the previous simulations) were calculated as 24.3%, 18.0%, and 27.2% for depressive symptoms, sleep duration, and bedtime, respectively. The ratio of variance in WPVS at time $t = 4$ to that at time $t = 1$ ($\omega$) was 2.30, 1.45, and 2.22 for depressive symptoms, sleep duration, and bedtime, respectively. The amounts of biases in TS-SNMMs were pragmatically small in the previous simulations under the conditions for the calculated $\phi^2$ and $\omega$.

Table 2 gives the estimated joint effect for reciprocal relations in TS-SNMMs (that assume interaction effects) and RI-CLPM, respectively. Bootstrap was used in RI-CLPM to estimate standard errors.

As seen in Table 2, TS-SNMMs reveal that sleep duration at age 14 ($X_3^*$) shows a statistically significant effect for decreasing later depressive symptoms at age 16 ($Y_4^*$) at the within-person level ($\hat{\beta}_{y3} = -0.305$, 95%CI [-0.595, -0.016], $p < .05$: 1 hour longer sleep at age 14 decreases the SMFQ score by 0.305 points at age 16), while the main effects for age 12 ($X_2^*$) and 10 ($X_1^*$) do not. A statistically significant interaction effect between sleep duration and depressive symptoms at age 12 ($X_2^*Y_2^*$) was observed ($\hat{\gamma}_{y2} = -0.173$, 95%CI [-0.331, -0.016], $p < .05$: adolescents with higher depressive symptoms at age 12 take larger effects on later depressive symptoms at age 14 from having more sleep), while such interaction effects were not observed at age 14 ($X_4^*Y_4^*$). On the other hand, depressive symptoms at age 12 ($Y_2^*$) show a small, but statistically significant, main effect at increasing sleep durations at age 16 ($X_4^*$) ($\hat{\beta}_{x2} = 0.018$ [60 x 0.018 = 1.08min increase], 95%CI [0.003, 0.032], $p < .05$), while the main effects from ages 14 ($Y_4^*$) and 10 ($Y_1^*$) do not. On the
whole, the results were unchanged when using AR(2) measurement models for each variable (Table S8). Similar positive effects of sleep duration on later depressive symptoms were found in a previous study (Matamura et al., 2014). Usami (2022a), which mainly focused on a clinical group comprising \( N = 416 \) adolescents with SMFQ scores of 6 or higher during the study, also demonstrated that increased sleep duration has positive effects on later depressive symptoms.

RI-CLPM produced similar results to those from TS-SNMMs in that the effects of sleep duration at ages 12 \((X_2^*)\) and 10 \((X_1^*)\) were not significant, but sleep duration at 14 years old \((X_3^*)\) showed a significant effect for decreasing later depressive symptoms \((\hat{\beta}_4^{(Y)} = -0.901, 95\%\text{CI} \ [-1.533, \ -0.282], p < .05)\). Compared with TS-SNMMs, this point estimate is smaller and indicates that a longer sleep duration has a more positive effect on later depressive symptoms. On the other hand, depressive symptoms did not exhibit any statistically significant effects on later sleep duration. The TTC study is now gathering new data for a fifth wave, and further investigations with larger \( T \) are desired.

As illustrated in this example, the statistical significance, sign, and magnitude of estimates of causal effects might change depending on how the effects are modeled and how the parameters are estimated. TS-SNMMs that can flexibly and robustly estimate parameters can be considered as potential alternatives of RI-CLPM (and its variants), and comparing estimation results between these approaches could also be useful as a sensitivity analysis.

6 DISCUSSION

In this paper, we provided a tutorial, simulation, and illustrative example of TS-SNMMs to infer within-person (reciprocal) relations. This method assumes a DGP similar to RI-CLPM, but we have shown through our simulations and the illustrative example that TS-SNMMs have several advantages over RI-CLPM: (i) the flexible inclusion of curvilinear (e.g., quadratic) and interaction effects of WPVS as latent variables, (ii) more accurate estimates
of causal parameters can be obtained under certain conditions due to them being doubly robust, even if unobserved time-varying confounders and model misspecifications exist, and (iii) the risk of obtaining improper solutions is minimized. We showed in simulations that TS-SNMMs work well in many conditions if longitudinal data with $T \geq 4$ are available, and more accurate estimates can be obtained if $T$ becomes larger.

In psychology and related disciplines, the use of RI-CLPM has rapidly increased in the last few years. Although researchers often wish to look at within-person (reciprocal) relations from the perspective of causal inference, there are potential limitations in traditional SEM-based approaches like RI-CLPM when it comes to accounting for time-varying confounders (which relate to the ability to flexibly and robustly estimate causal parameters), and the risk of obtaining improper solutions can present a challenge, especially when the sample size is small. TS-SNMMs can be used as an alternative over RI-CLPM (and its variants), especially when data with $T \geq 4$ are available and when improper solutions occur in RI-CLPM. However, the purpose of this paper is not to completely denounce the use of RI-CLPM. Comparing estimation results between these methods can be useful as a sensitivity analysis, especially when either $T$ is small or researchers are concerned with the presence of unobserved time-varying confounders and model misspecifications.

There are other scenarios in which biased estimates can arise in RI-CLPM while TS-SNMMs perform well. One is the case where some model misspecifications exist in the structural model for the observed confounders $L^*$. Even if researchers are sure that observed confounders were appropriately controlled for, the model for $L^*$ might be misspecified for some reasons (e.g., a misspecified functional form and/or omitted variables). In the standard SEM approach as covariance structure analysis, one needs to specify the (linear) model for $L^*$, even if the main focus is the inference of relations between the focal variables $X^*$ and $Y^*$. For this reason, model misspecification in $L^*$ can cause biases in the causal parameters when applying RI-CLPM. Future investigations should study how a misspecification in $L^*$ can cause biased causal estimates in RI-CLPM depending on the DGP and
model specifications, and how effectively TS-SNMMs can mitigate this problem.

However, TS-SNMMs are also prone to possible pitfalls that practitioners should be aware of. The number of time points $T$ is an especially critical aspect. Most research aimed at inferring reciprocal (within-person) relations has used longitudinal data with $T = 2, 3$ (e.g., Usami, Todo et al., 2019), and imprecise predictions of WPVS can seriously degrade the estimation performance in TS-SNMMs. However, in this case, the risk of producing seriously biased estimates can increase even in RI-CLPM if model misspecifications exist.

Correct specification of the measurement model can be challenging. Fortunately, both current simulations and Usami (2022) have shown that even (misspecified) models with a time-varying AR(1) structure for WPVS produce accurate estimates, and that the choice of order has little influence on estimation performance. However, further investigations of estimation performance that account for various misspecifications in measurement models are still required, and comparisons with other recent estimation approaches (e.g., Du & Bentler, 2021; Du, Bentler & Rosseel, 2022) for parameters in measurement models, especially for data with non-normal or small $N$, will also be an important topic for future studies.

Another important but still unresolved issue is how to establish the correct (or even a plausible) DAG model, or how one can validate the incorporation of time-invariant factors, such as stable trait factors, to infer within-person relations. To identify the causal parameters, we assumed that measurements are expressed by the linear sum of stable trait factor scores and WPVS, as in RI-CLPM. Usami, Murayama et al. (2019) explained that there are two primary ways to statistically control for time-invariant factors as individual differences: using stable trait factors included in RI-CLPM that have only direct effects on measurements and are uncorrelated with within-person processes (i.e., WPVS), and using the *accumulating factors* included in several other statistical models (e.g., Bollen & Brand, 2010), which have both direct and indirect effects on measurements. In these models, accumulating factors are modeled with lagged regressions rather than being modeled separately.
from them (see Usami 2022a for further details and the corresponding DGP).

The discussion of issues surrounding the appropriate model choice and the potential
difference of inferential results among statistical models when inferring within-person
relations is ongoing, and it continues to gather attention from quantitative researchers in
psychology (e.g., Andersen, 2022; Lucas, 2022; Lüdtke & Robitzsch, 2021; Usami, 2022ab).
Though our attention in this paper was on TS-SNMMs that assume stable traits factors in
DGP, if an uncorrelated assumption of between- and within-person processes is violated,
then both RI-CLPM and TS-SNMMs can produce seriously biased estimates. One can
resort to substantial theory to choose the model (e.g., Shehata et al., 2021; who argued
that some statistical models that include stable trait factors, such as a constrained ver-

tion of RI-CLPM, are well suited to model between- and within-person components when
capturing maintenance effects in communication research). However, in many cases, re-
searchers do not exactly know the true DGP and how time-invariant factors (if they exist)
influence measurements (e.g., linearly or nonlinearly, directly or indirectly, or both), and
unambiguous specification of the theoretically derived expected relations for variables is
quite challenging in practical applications (e.g., Curran & Bauer, 2011; Usami, 2022a).
Further discussion accounting for empirical investigations and the sensitivity of results will
be required in the future (Usami, 2022a). The extensions of TS-SNMMs that account
for accumulating factors (i.e., correlated within-person and between-person processes) and
measurement errors, along with developing a package to further increase the feasibility of
TS-SNMMs, are also important future research goals.
REFERENCES


Table 1. Proportion of improper solutions in each condition.

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$\varphi^2$, (proportion of) stable trait factor variances at $t=1$; $T$, the number of time points; $N$, sample size; RI-CLPM, random intercept cross-lagged panel model; TS-SNMMs, two step estimation approach with structural nested mean model.
Table 2. Joint effect estimates for reciprocal relations in TS-SNMMs and the RI-CLPM (N=1,294)

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<td>0.092</td>
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<tr>
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Bold font indicates statistical significance. Sleep, sleep duration; SMFQ, Short Mood and Feelings Questionnaire; SE, standard error.
Figure 1. The linear causal diagrams (DAGs) for data-generating process in which stable trait factors are included. Solid single-headed arrows (directed edges) are labeled with path coefficients that quantify direct causal effects. A dashed double-headed arrow (bidirected edge) represents a correlation due to an unobserved common cause. Stable trait factors are represented in dashed circles, indicating that these are latent variables.
\( \varphi^2 \), (proportion of) stable trait factor variances at \( t=1 \); \( T \), the number of time points; \( r_{g} \), correlation between stable trait factors; \( \omega \), ratio of variance of within-person variability scores at \( t=T \) to that at \( t=1 \); RI-CLPM, random intercept cross-lagged panel model; TS-SNMMs, two-step estimation approach with structural nested mean model.

Figure 2. Averages of estimates and biases for the joint effects of X* on Y* in TS-SNMMs and the RI-CLPM.
\( \varphi^2 \), (proportion of) stable trait factor variances at \( t=1 \); \( T \), the number of time points; \( r_{B} \), correlation between stable trait factors; \( \omega \), ratio of variance of within-person variability scores at \( t=T \) to that at \( t=1 \); RI-CLPM, random intercept cross-lagged panel model; TS-SNMMs, two-step estimation approach with structural nested mean model.

Figure 3. Averages of estimates and biases for the joint effects of \( X^* \) on \( Y^* \) in TS-SNMMs and the RI-CLPM when there exist unobserved confounders \( U^* \) that influence the observed time-varying confounders \( L^* \) and the outcomes \( Y^* \).
\( \varphi^2 \), (proportion of) stable trait factor variances at \( t=1 \); \( T \), the number of time points; \( r_{gb} \), correlation between stable trait factors; \( \omega \), ratio of variance of within-person variability scores at \( t=T \) to that at \( t=1 \); RI-CLPM, random intercept cross-lagged panel model; TS-SNMMs, two-step estimation approach with structural nested mean model.

Figure 4. Averages of estimates and biases for the joint effects of \( X^* \) on \( Y^* \) in TS-SNMMs and the RI-CLPM when there exist ignored direct higher-order effects of observed time-varying confounders \( L^* \) on outcomes \( Y^* \).
Online Supplemental Material

Tables ................................................................. p. 2
Figures ................................................................. p. 11
*R code for the simulation is provided in a separate file.
Table S1. Results of ANOVA where calculated biases are set as outcomes.

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<tr>
<td>( T \times N \times \varphi^2 \times r_W )</td>
<td>24 0.00060 0.00002 0.001 0.0005 0.00002 0.000</td>
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<tr>
<td>( T \times N \times r_B \times r_W )</td>
<td>24 0.00059 0.00002 0.001 0.0031 0.00013 0.001</td>
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<td>( T \times \varphi^2 \times r_B \times r_W )</td>
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<tr>
<td>( T \times N \times \varphi^2 \times \omega )</td>
<td>24 0.00321 0.00013 0.007 0.0069 0.00029 0.003</td>
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<tr>
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<td></td>
</tr>
<tr>
<td>( T \times \varphi^2 \times r_B \times \omega )</td>
<td>24 0.00157 0.00007 0.004 0.0158 0.00066 0.007</td>
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<tr>
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<tr>
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<td>( T \times \varphi^2 \times r_W \times \omega )</td>
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<tr>
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<tr>
<td>( T \times N \times r_B \times \omega )</td>
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</tr>
<tr>
<td>( N \times r_B \times r_W \times \omega )</td>
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</tr>
<tr>
<td>( \varphi^2 \times r_B \times r_W \times \omega )</td>
<td>16 0.00063 0.00004 0.001 0.0005 0.00003 0.000</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>( T \times N \times \varphi^2 \times r_B \times r_W )</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( T \times N \times \varphi^2 \times r_B \times \omega )</td>
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<td></td>
</tr>
<tr>
<td>( T \times N \times \varphi^2 \times r_W \times \omega )</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( T \times N \times r_B \times r_W \times \omega )</td>
<td>48 0.00196 0.00004 0.005 0.0065 0.00014 0.003</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( T \times \varphi^2 \times r_B \times r_W \times \omega )</td>
<td>48 0.00135 0.00003 0.003 0.0031 0.00006 0.001</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>( N \times \varphi^2 \times r_B \times r_W \times \omega )</td>
<td>32 0.00111 0.00003 0.003 0.0027 0.00009 0.001</td>
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</tr>
<tr>
<td>( T \times N \times \varphi^2 \times r_B \times r_W \times \omega )</td>
<td>96 0.00273 0.00003 0.006 0.0103 0.00011 0.005</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>971</strong> 0.43226 2.1121</td>
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Table S2. Proportions of improper solutions in each condition (second simulation).

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<th>N=1000</th>
</tr>
</thead>
<tbody>
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<td>$\phi^2=0.1$</td>
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<td>TS-SNMMs</td>
<td>RI-CLPM</td>
</tr>
<tr>
<td>$T=3$</td>
<td>85.59%</td>
<td>14.75%</td>
<td>76.63%</td>
</tr>
<tr>
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<td>1.90%</td>
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<td>0.40%</td>
<td>46.52%</td>
</tr>
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<td>43.33%</td>
<td>2.34%</td>
<td>23.14%</td>
</tr>
<tr>
<td>$\phi^2=0.4$</td>
<td>RI-CLPM</td>
<td>TS-SNMMs</td>
<td>RI-CLPM</td>
</tr>
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<td>$T=3$</td>
<td>13.26%</td>
<td>0.05%</td>
<td>4.52%</td>
</tr>
<tr>
<td>$T=4$</td>
<td>6.44%</td>
<td>0.00%</td>
<td>1.68%</td>
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<tr>
<td>$T=6$</td>
<td>31.89%</td>
<td>9.46%</td>
<td>13.93%</td>
</tr>
<tr>
<td>$T=9$</td>
<td>5.67%</td>
<td>0.61%</td>
<td>0.56%</td>
</tr>
<tr>
<td>$\phi^2=0.7$</td>
<td>RI-CLPM</td>
<td>TS-SNMMs</td>
<td>RI-CLPM</td>
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<td>1.32%</td>
<td>0.07%</td>
<td>0.22%</td>
</tr>
<tr>
<td>$T=4$</td>
<td>1.32%</td>
<td>0.07%</td>
<td>0.22%</td>
</tr>
<tr>
<td>$T=6$</td>
<td>1.32%</td>
<td>0.07%</td>
<td>0.22%</td>
</tr>
<tr>
<td>$T=9$</td>
<td>1.32%</td>
<td>0.07%</td>
<td>0.22%</td>
</tr>
</tbody>
</table>

RI-CLPM, random intercept cross-lagged panel model; TS-SNMMs, two-step estimation approach with a structural nested mean model.
Table S3. Proportions of observed values that exceeded prespecified criteria for model fit indices (second simulation).

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<th>$T = 6$ SRMR</th>
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<th>$T = 9$ SRMR</th>
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<tbody>
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<td>&gt;.95</td>
<td>&lt;.05</td>
<td>&gt;.95</td>
<td>&lt;.05</td>
</tr>
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<td>$\varphi^2 = 0.1$</td>
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<td></td>
<td></td>
</tr>
<tr>
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<td>99.80%</td>
<td>80.24%</td>
<td>99.89%</td>
<td>99.74%</td>
<td>93.04%</td>
<td>98.07%</td>
</tr>
<tr>
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<td>100.00%</td>
<td>94.11%</td>
<td>100.00%</td>
<td>100.00%</td>
<td>99.98%</td>
<td>100.00%</td>
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<tr>
<td>N=1000</td>
<td>100.00%</td>
<td>100.00%</td>
<td>98.11%</td>
<td>100.00%</td>
<td>100.00%</td>
<td>100.00%</td>
<td>100.00%</td>
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<tr>
<td>$\varphi^2 = 0.4$</td>
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<td>93.67%</td>
<td>97.11%</td>
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<td>100.00%</td>
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<td>100.00%</td>
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<td>98.37%</td>
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<td>100.00%</td>
<td>100.00%</td>
<td>100.00%</td>
</tr>
<tr>
<td>$\varphi^2 = 0.7$</td>
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<td></td>
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<td>83.19%</td>
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<td>100.00%</td>
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</tr>
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</table>

CFI, comparative fit index; RMSEA, root mean square error of approximation; SRMR, standardized root mean square residual.
Table S4. Proportions of improper solutions in each condition (third simulation).

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<td>TS-SNMMs</td>
<td>RI-CLPM</td>
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<td>17.05%</td>
<td>79.12%</td>
</tr>
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<td>$\varphi^2=0.4$</td>
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<td>$\varphi^2=0.7$</td>
<td>26.26%</td>
<td>6.62%</td>
<td>11.61%</td>
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</table>

RI-CLPM, random intercept cross-lagged panel model; TS-SNMMs, two-step estimation approach with a structural nested mean model.
Table S5. Proportions of observed values that exceeded prespecified criteria for model fit indices (third simulation).

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<td>SRMR</td>
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<td>SRMR</td>
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<td>&gt;.95</td>
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<tr>
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<td>96.94%</td>
<td>69.81%</td>
<td>87.06%</td>
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<td>100.00%</td>
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<td>100.00%</td>
<td>99.94%</td>
<td>77.94%</td>
<td>100.00%</td>
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<tr>
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<td>80.15%</td>
<td>100.00%</td>
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</table>

CFI, comparative fit index; RMSEA, root mean square error of approximation; SRMR, standardized root mean square residual.
Table S6. Descriptive statistics.

(a) Mean and standard deviation

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<th>SD10</th>
<th>SMFQ10</th>
<th>BT12</th>
<th>SD12</th>
<th>SMFQ12</th>
<th>BT14</th>
<th>SD14</th>
<th>SMFQ14</th>
<th>BT16</th>
<th>SD16</th>
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<td>3.67</td>
<td>23.10</td>
<td>7.65</td>
<td>3.67</td>
<td>23.72</td>
<td>7.01</td>
<td>3.66</td>
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<tr>
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<td>0.73</td>
<td>0.74</td>
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<td>0.81</td>
<td>0.86</td>
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(b) Variances, covariances, and correlations

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<th>SMFQ10</th>
<th>BT12</th>
<th>SD12</th>
<th>SMFQ12</th>
<th>BT14</th>
<th>SD14</th>
<th>SMFQ14</th>
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<th>SD16</th>
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<td>-0.28</td>
<td>0.09</td>
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<td>-0.16</td>
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<td>0.55</td>
<td>-0.04</td>
<td>-0.22</td>
<td>0.34</td>
<td>-0.05</td>
<td>-0.14</td>
<td>0.17</td>
<td>-0.06</td>
</tr>
<tr>
<td>SMFQ10</td>
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<td>0.00</td>
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<td>-0.19</td>
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<td>0.02</td>
<td>-0.42</td>
<td>0.55</td>
<td>-0.06</td>
<td>-0.32</td>
<td>0.48</td>
<td>-0.08</td>
<td>-0.16</td>
<td>0.22</td>
<td>-0.11</td>
</tr>
<tr>
<td>SMFQ12</td>
<td>0.16</td>
<td>-0.10</td>
<td>7.87</td>
<td>0.23</td>
<td>-0.19</td>
<td>18.51</td>
<td>0.05</td>
<td>0.00</td>
<td>0.36</td>
<td>0.02</td>
<td>0.06</td>
<td>0.29</td>
</tr>
<tr>
<td>GB14</td>
<td>0.19</td>
<td>-0.11</td>
<td>0.16</td>
<td>0.28</td>
<td>-0.19</td>
<td>0.16</td>
<td>0.65</td>
<td>-0.67</td>
<td>0.14</td>
<td>0.47</td>
<td>-0.32</td>
<td>0.12</td>
</tr>
<tr>
<td>SD14</td>
<td>-0.15</td>
<td>0.18</td>
<td>0.13</td>
<td>-0.25</td>
<td>0.30</td>
<td>0.00</td>
<td>-0.47</td>
<td>0.74</td>
<td>-0.08</td>
<td>-0.28</td>
<td>0.43</td>
<td>-0.12</td>
</tr>
<tr>
<td>SMFQ14</td>
<td>0.26</td>
<td>-0.15</td>
<td>5.39</td>
<td>0.34</td>
<td>-0.27</td>
<td>7.12</td>
<td>0.53</td>
<td>-0.29</td>
<td>20.58</td>
<td>0.11</td>
<td>-0.02</td>
<td>0.44</td>
</tr>
<tr>
<td>GB16</td>
<td>0.13</td>
<td>-0.08</td>
<td>0.09</td>
<td>0.16</td>
<td>-0.10</td>
<td>0.06</td>
<td>0.33</td>
<td>-0.20</td>
<td>0.42</td>
<td>0.75</td>
<td>-0.60</td>
<td>0.13</td>
</tr>
<tr>
<td>SD16</td>
<td>-0.09</td>
<td>0.09</td>
<td>0.19</td>
<td>-0.12</td>
<td>0.15</td>
<td>0.21</td>
<td>-0.23</td>
<td>0.33</td>
<td>-0.06</td>
<td>-0.47</td>
<td>0.81</td>
<td>-0.07</td>
</tr>
<tr>
<td>SMFQ16</td>
<td>0.32</td>
<td>-0.19</td>
<td>4.05</td>
<td>0.47</td>
<td>-0.42</td>
<td>6.38</td>
<td>0.49</td>
<td>-0.52</td>
<td>10.33</td>
<td>0.59</td>
<td>-0.31</td>
<td>26.92</td>
</tr>
</tbody>
</table>

* BT, bedtime; SD, sleep duration; SMFQ, Short Mood and Feelings Questionnaire.

Variances are in the diagonal elements, covariances are in the lower-left elements, and correlations are in the upper-right elements.
Table S7. Fit indices of the AR(1) measurement model for each variable.

<table>
<thead>
<tr>
<th></th>
<th>Bedtime</th>
<th>Sleep duration</th>
<th>SMFQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of parameters</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>df</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>chi-square</td>
<td>2.507</td>
<td>7.325</td>
<td>7.788</td>
</tr>
<tr>
<td>p-value</td>
<td>0.285</td>
<td>0.026</td>
<td>0.02</td>
</tr>
<tr>
<td>CFI</td>
<td>1</td>
<td>0.995</td>
<td>0.992</td>
</tr>
<tr>
<td>RMSEA</td>
<td>0.014 [0.000, 0.059]</td>
<td>0.045 [0.014, 0.083]</td>
<td>0.047 [0.016, 0.084]</td>
</tr>
<tr>
<td>SRMR</td>
<td>0.011</td>
<td>0.018</td>
<td>0.018</td>
</tr>
</tbody>
</table>

Model fits are perfect for the AR(2) measurement model.
Table S8. Estimates from TS-SNMMs with AR(2) measurement models ($N=1,294$).

<table>
<thead>
<tr>
<th>TS-SNMMs</th>
<th>Sleep → SMFQ</th>
<th>SMFQ → Sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep 3 → SMFQ 4</td>
<td>-0.290</td>
<td>0.003</td>
</tr>
<tr>
<td>Sleep 3 × Bedtime 3 → SMFQ 4</td>
<td>0.033</td>
<td>0.016</td>
</tr>
<tr>
<td>Sleep 3 × SMFQ 3 → SMFQ 4</td>
<td>-0.027</td>
<td>0.008</td>
</tr>
<tr>
<td>Sleep 2 → SMFQ 4</td>
<td>-0.198</td>
<td>0.015</td>
</tr>
<tr>
<td>Sleep 2 × Bedtime 2 → SMFQ 4</td>
<td>-0.655</td>
<td>0.009</td>
</tr>
<tr>
<td>Sleep 2 × SMFQ 2 → SMFQ 4</td>
<td>-0.161</td>
<td>0.015</td>
</tr>
<tr>
<td>Sleep 1 → SMFQ 4</td>
<td>0.382</td>
<td>0.015</td>
</tr>
<tr>
<td>Sleep 1 × Bedtime 1 → SMFQ 4</td>
<td>0.223</td>
<td>0.009</td>
</tr>
<tr>
<td>Sleep 1 × SMFQ 1 → SMFQ 4</td>
<td>0.101</td>
<td>0.007</td>
</tr>
<tr>
<td>SMFQ 3 → Sleep 4</td>
<td>-0.009</td>
<td>0.015</td>
</tr>
<tr>
<td>SMFQ 3 × Bedtime 3 → Sleep 4</td>
<td>0.003</td>
<td>0.004</td>
</tr>
<tr>
<td>SMFQ 3 × Sleep 3 → Sleep 4</td>
<td>0.003</td>
<td>0.004</td>
</tr>
<tr>
<td>SMFQ 2 → Sleep 4</td>
<td>0.050</td>
<td>0.007</td>
</tr>
<tr>
<td>SMFQ 2 × Bedtime 2 → Sleep 4</td>
<td>0.043</td>
<td>0.016</td>
</tr>
<tr>
<td>SMFQ 1 → Sleep 4</td>
<td>0.004</td>
<td>0.007</td>
</tr>
<tr>
<td>SMFQ 1 × Bedtime 1 → Sleep 4</td>
<td>0.050</td>
<td>0.004</td>
</tr>
<tr>
<td>SMFQ 1 × Sleep 1 → Sleep 4</td>
<td>0.043</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Bold font indicates statistical significance.
Figure S1. RMSEs of estimates for the joint effects of \( X^* \) on \( Y^* \) in TS-SNMMs and RI-CLPM. 
\[ (N=1000 \text{ and } r_W = 0.3) \]
Figure S2. Averages of estimates and biases for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM. 

($N=200$ and $r_W = 0.3$)
Figure S3. Averages of estimates and biases for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM.

\( (N=600 \text{ and } r_W = 0.3) \)
Figure S4. Averages of estimates and biases for the joint effects of $X^*$ on $Y^*$ in TS-SNMMs and RI-CLPM.

($N=1000$ and $r_W = 0.1$)
Figure S5. Averages of estimates and biases for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM.

\(N=1000\) and \(r_w = 0.5\)
Figure S6. RMSEs of estimates for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM.

\( N=200 \) and \( r_W = 0.3 \)
Figure S7. RMSEs of estimates for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM.

(N=600 and \( r_W = 0.3 \))
Figure S8. RMSEs of estimates for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM. 
\( (N=1000 \text{ and } r_W = 0.1) \)
Figure S9. RMSEs of estimates for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM.

\( (N=1000 \text{ and } r_W = 0.5) \)
Figure S10. Averages of estimates and biases for the joint effects of $Y^*$ on $X^*$ in TS-SNMMs and RI-CLPM.

($N=1000$ and $r_W = 0.3$)
Figure S11. RMSEs of estimates for the joint effects of $Y^*$ on $X^*$ in TS-SNMMs and RI-CLPM. 

($N=1000$ and $r_W = 0.3$)
Figure S12. Averages of estimates and biases for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM.

(AR(2) measurement model; N=1000 and \( r_W = 0.3 \))
Figure S13. RMSEs of estimates for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM. (AR(2) measurement model; N=1000 and \( r_W = 0.3 \))
Figure S14. Averages of estimates and biases for the joint effects of $X^*$ on $Y^*$ in TS-SNMMs and RI-CLPM when there exist unobserved confounders $U^*$ that influence observed time-varying confounders $L^*$ and outcomes $Y^*$ (AR(2) measurement model; $N=1000$ and $r_{\mu}=0.3$).
Figure S15. Averages of estimates and biases for the joint effects of $Y^*$ on $X^*$ in TS-SNMMs and RI-CLPM when there exist unobserved confounders $U^*$ that influence observed time-varying confounders $L^*$ and outcomes $Y^*$ ($N=1000$ and $r_{xy}=0.3$).
Figure S16. Averages of estimates and biases for the joint effects of X* on Y* in TS-SNMMs and RI-CLPM when there exist ignored direct higher-order effects of observed time-varying confounders L* on outcomes Y* (AR(2) measurement model; N=1000 and $r_{WY}=0.3$).
Figure S17. Averages of estimates and biases for the joint effects of Y* on X* in TS-SNMMs and RI-CLPM when there exist ignored direct higher-order effects of observed time-varying confounders L* on outcomes Y* \( (N=1000\) and \( r_w = 0.3 \)).