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RESEARCH ARTICLE

AGGRESSIVE BEHAVIOR WILEY

# Modeling state- and trait-level associations between aggression, somatic symptoms, substance use, and distress tolerance

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## Abstract

The current study examined the state- and trait-level associations of psychological and physical aggression to somatic symptoms, and alcohol and drug use and tested the influence of distress tolerance on these associations, while controlling for stress, sex, and minority status. A naturalistic observation was used to collect data with a sample of 245 college students at three time points with 2 weeks apart. Random-intercept cross-lagged panel models were used to disaggregate within-person effects (autoregressive and cross-lagged effects) from the between-person (latent trait-level) associations. The findings revealed that there were autoregressive effects of psychological aggression between Time 1 (T1) and Time 2 (T2) and of physical aggression between T1 and T2. There was a bidirectional association between psychological aggression and somatic symptoms at T2 and Time 3 (T3), in which T2 psychological aggression predicted T3 somatic symptoms and verse vera. T1 drug use predicted T2 physical aggression, which in turn predicted T3 somatic symptoms, indicating physical aggression being a mediator between earlier drug use and later somatic symptoms. Distress tolerance was negatively associated with psychological aggression and somatic symptoms, respectively, and such an influence did not differ across time occasions. The findings indicated the importance of incorporating physical health in the prevention and intervention of psychological aggression. Clinicians may also consider including psychological aggression in the screening of somatic symptoms or physical health. Empirical-supported therapy components for enhancing distress tolerance may help mitigate psychological aggression and somatic symptoms.

KEYWORDS

aggression, distress tolerance, latent trait-level correlations, random-intercept cross-lagged panel modeling, somatic symptoms, substance use

## 1 | INTRODUCTION

Aggression includes two major forms, psychological (also referred to as relational aggression) and physical aggression (Anderson et al., 1995) and often arises in the context of interpersonal conflicts, wherein people adopt psychological or physical aggression to manage conflicts when people perceive the goals, values, or beliefs being frustrated by others (Chapman & Gillespie, 2019; De Dreu & Beersma, 2005). Two of the influential aggression theories are the $I^3$ theory (Finkel et al., 2012) and the General Aggression Model (GAM) (Anderson & Bushman, 2002). The $I^3$ theory posits that aggression is the outcome of a moderating pathway, comprised of instigating triggers (e.g., provocation), impelling forces (e.g., certain personality traits that determine how an individual would respond to an instigating trigger), and inhibiting forces (e.g., self-control, social support). Thus, impelling forces or inhibiting forces may moderate the response to an instigating trigger). The GAM highlights that internal representations of past experiences play an important role in aggression and asserts that individuals who are more susceptible to a negative event would experience a greater degree of emotional arousal and hold more distorted attributions, which in turn lead to a higher risk of aggression. Collectively, these two theories indicate that strong negative emotions, lowered inhibition, distorted cognition, and certain personality traits contribute to aggression.

Empirical literature shows that aggression is expressed with a variety of negative emotions (Lazarus & Folkman, 1987). These negative emotions and concomitant psychological strain have a detrimental effect on health and exacerbate internalizing and externalizing symptoms (Ezell et al., 2023). For college students, aggression often is co-morbid with several negative social outcomes and personal symptoms, such as interpersonal difficulties, social adjustment, learning outcomes, loneliness, and sadness (Thomas, 2019). Thus, understanding aggression and its relation to comorbid behavioral and psychological difficulties is a critical and relevant topic for college students. In this context, the current study focused on three under-investigated domains, including somatic symptoms, substance (alcohol and drug) use, and distress tolerance, and examined the less-understood associations between aggression and these variables among a sample of college students. In addition, these three domains were pertinent to the important constructs included in the $I^3$ and the GAM theories, which were referred to in the following sections.

### 1.1 | Aggression and somatic symptoms

Somatic symptoms are somatic complaints (e.g., chest pain, heart pain) or unpleasant physical sensation (e.g., nausea) that are not part of diagnosed medical conditions (Kirmayer & Looper, 2007). While most of the extant literature on somatic symptoms has been drawn from clinical samples, a growing number of studies have shown that aggression is associated with somatic symptoms among nonclinical populations. For example, a study with a sample of college students has found psychological aggression is positively related to somatic symptoms, the relationship of which is amplified among those with more difficulties in emotion regulation; however, physical aggression does not predict somatic symptoms (Yang, 2020). The relation between aggression and somatic symptoms can also be extrapolated from the literature on aggression-related emotions (such as anger and hostility) and somatic symptoms. Maladaptive anger and anger suppression are associated with health issues, such as cardiovascular disease and pain-related complaints (Bruehl et al., 2012; Smith et al., 2004). Kirmayer and colleagues (2007) present an integrative, iterative model of somatic symptoms in which individual perception and cognition, personality, and social factors contribute to and amplify somatic experiences, which in turn reinforce the risk of negative cognitive processes and behavioral tendencies. Consistent with the GAM of aggression, this process reflects a self-perpetuating and exacerbating fashion that underlies the relations between somatic symptoms and related factors. For example, individuals with high levels of irritability and maladaptive anger may have biased attention to physiological disturbance and thus be more susceptible to experiencing somatic symptoms. Somatic symptoms may interfere with psychological functioning and, thus, heighten the intensity and valence of negative emotional arousal. This would in turn result in an increased risk of aggression. Therefore, there might be a reciprocal relation between aggression and somatic symptoms.

### 1.2 | Aggression and substance use

Psychopharmacological research on the acute effect of substances shows that substances can alter the communications between neurotransmitters and functions of neurotransmitters, which in turn impair the reward circuit, emotion regulation functioning, and cognitive functioning (NIDA, 2022). Behavioral symptoms resulting from the impairment in emotion regulation and cognitive functioning may include aggression. According to the disinhibition hypothesis, alcohol can lower the capacity to inhibit certain behaviors, which leads to an increased risk of aggressive behaviors (Källmén & Gustafson, 1998). Cannabis has been found to link to physical aggression among youth and young adults, even after controlling for sociodemographic factors and different study designs (Dellazizzo et al., 2020). Likewise, cocaine and other stimulants (e.g., Phencyclidine or phenylcyclohexyl piperidine, amphetamines) have been shown to lead to aggression (Bey & Patel, 2007; Stuart et al., 2008; Vaughn et al., 2015). While opiates may exert a sedating effect, opiate cravings can trigger a dysphoric state (Kakko et al., 2019), which may lead to a heightened risk of aggression. Taken together, alcohol and drugs can alter the psychomotor system which results in more aggressive acts, distort attention to selective, salient environmental cues, and hinder the internal inhibition and decision-making systems that control aggressive impulses. Aligned with the $I^3$ theory and the GAM of aggression, lowered inhibition and enhanced attention to negative cues lead to a propensity to engage in aggressive behaviors (Anderson & Bokor, 2012; Giancola et al., 2010; Hoaken & Stewart, 2003).

Several empirical and review studies have shown a bidirectional, dynamic association among factors associated with substance use (e.g., (Mason & Windle, 2002; McCabe et al., 2023). Some of these studies can shed light on understanding the relationship between aggression and substance use. For example, with 4-panel data from 1218 high schoolers, Mason and Windle (2002) have revealed a

reciprocal relation between polysubstance use and delinquency (e.g., aggression) in male students but not in female students. Another study with college students has found that impulsivity (i.e., positive urgency) increases the risk of alcohol use, which in turn exacerbates impulsivity (Kaiser et al., 2016). Correlation studies have showed that greater alcohol consumption is associated with more aggression in both male and female participants (Crane et al., 2017). Daily dairy data have shown that alcohol use is associated with both verbal and physical aggression among community couples (Testa & Derrick, 2014). Longitudinal studies with self-report data have shown that aggression is one of the key risk factors to earlier substance use initiation (Ernst et al., 2006; Iacono et al., 2008; Jester et al., 2008; Mathias et al., 2015). In this sense, substance use serves as a coping mechanism for addressing negative emotions that are concomitant to or induced by aggression. However, it is important to mention the literature primarily focused on physical aggression. The findings have been primarily drawn on cross-sectional data or longitudinal data collected with large time differences that reflect stable patterns. Thus, it is important to understand acute and stable correlations between aggression and substance use simultaneously.

## 1.3 | Distress tolerance

The literature consistently indicates common liability factors, such as personality traits, account for or contribute to the associations pertinent to undesired behaviors and psychopathology (e.g., Ernst et al., 2006; Tomlinson et al., 2016). Distress tolerance is the perceived capacity to withstand negative emotional or other aversive states (e.g., physical discomfort) and the behavioral capacity of withstanding distressing internal states elicited by some type of stressor (Zvolensky et al., 2010). Distress tolerance impacts multiple forms of psychopathology and can be conceptualized as a transdiagnostic risk factor underlying several pathological symptoms, such as anxiety (Michel et al., 2016), depression (Lass & Winer, 2020), and alcohol use (Howell et al., 2010). A dearth of research has been conducted to examine the association between distress tolerance and aggression. The potential association between distress tolerance and aggression may be inferred from the literature on distress tolerance and borderline personality disorder. The literature shows that the symptoms of borderline personality disorder (e.g., self-injury, suicidal attempts, and substance use) are dysfunctional attempts to address emotional distress (Linehan, 2014). Borderline personality disorder symptoms are associated with anger, physical aggression, and violence (Newhill et al., 2012; Sansone & Sansone, 2012; Scott et al., 2017). Because distress tolerance is a key factor underlying the symptoms of borderline personality disorder, it is possible that distress tolerance is associated with aggression.

There are some studies reporting the influence of distress tolerance on various forms of psychopathology. A meta-analysis study has revealed medium-to-large negative associations of distress tolerance to substance use, disordered eating behavior, borderline personality disorder, and impulsivity-type personality, respectively (Mattingley et al., 2022). The meta-analysis study also suggests that low distress tolerance could increase the risk of undesirable behaviors that function as mechanisms to cope with emotional distress. Research on college students' nonsuicidal self-harm has revealed that distress tolerance might mitigate the association between risk factors and nonsuicidal self-harm. High rumination and low distress tolerance predict nonsuicidal self-harm; strong affect intensity and low distress tolerance predict nonsuicidal self-harm (Slabbert et al., 2018). While distress tolerance has been shown to be negatively associated with substance use, regardless of the type of substances (Mattingley et al., 2022), most of the research is contextualized in co-commodity with both substance use and other psychopathology (e.g., eating disorder, borderline personality disorder) (Mattingley et al., 2022). Laboratory-based studies examining the association of substance use to distress tolerance have shown that people who actively use substances are more quickly to discontinue a task inducing uncomfortable sensation (e.g., anxiety); the findings apply to people who use alcohol and drugs (Daughters et al., 2005). However, it is less clear if the influence of distress tolerance on substance use endures over time or may be subject to state-level variables.

Much less is known to which degree the influence of distress tolerance can be extended to other forms of dysfunctioning (such as somatic symptoms). The limited literature indicates that distress tolerance is associated with less panic disorder (Leyro et al., 2010; Trépanier et al., 2022). Because distress tolerance is related to the degree to which individuals can withstand uncomfortable physical sensations (Schmidt et al., 2006), those with high distress tolerance may report fewer somatic symptoms. Given the theoretical relevance, empirical data are needed to clarify the association between distress tolerance and somatic symptoms.

## 1.4 | Current study

There were several research gaps. First, even though the previous studies conceptually supported bidirectional relations of aggression to somatic symptoms and substance use, respectively, much extant research used cross-sectional designs which only assessed concurrent relationships or, of the studies utilizing longitudinal designs, the overwhelming majority adopted methodologies precluding an examination of bidirectional associations of the relevant variables. Second, although a small number of studies adopted longitudinal designs, these studies did not differentiate between the within-person and between-person components in delineating the reciprocal associations between these constructs. One of the widely adopted approaches in the literature was using the cross-lagged panel model (CLPM) to estimate autoregressive and cross-lagged effects and infer reciprocal relations and temporal causality between repeatedly measured variables. However, the CLPM does not account for the part of stability derived from the trait-like, time-invariant nature of the constructs; as a result, the autoregressive and cross-lagged parameters may include spurious conclusions regarding the mechanism underlying variables that are assessed longitudinally (Hamaker et al., 2015). That is, the autoregressive and cross-lagged effects estimated by the CLPM are actually comprised of within-person

changes that are derived from temporality (i.e., autoregressive and lagged effects) and between-person stability that is time invariant. Thus, alternative analytic models are needed to disaggregate these two components so that the autoregressive and lagged effects on the within-person dynamics (such as the foci of this study) can be exclusively modeled. Third, while common liability factors, such as personality traits, underlie associations between aggression, somatic symptoms, and substance use, it is little known to which degree distress tolerance serves as a transdiagnostic risk factor to a variety of symptoms depicted in this study.

Building upon the need to address the aforementioned research gaps, the current study adopted a novel statistical method—random-intercept cross-lagged panel models (RI-CLPM) to identify stable, trait-like differences between persons and the lagged relations pertaining exclusively to within-person fluctuations (Hamaker et al., 2015). Expanding upon the CLPM, RI-CLPM integrates the multilevel data structure and structural equation modeling to account for stable, trait-like differences between units (e.g., individuals, dyads, families, etc.), such that the autoregressive and lagged relations pertain exclusively to within-unit fluctuations (Hamaker et al., 2015). Specifically, RI-CLPM models distinguish the between-subject variation (time-invariant or trait-like) and within-person (time-variant or state-like) variance by creating one latent variable for each construct across assessment time points, with factor loadings constrained to one (Hamaker et al., 2015). Applied in the current study, the RI-CLPM would yield the covariance between variables at the between-person level (e.g., how individuals with varying degrees of aggression report different levels of somatic symptoms) and variances (e.g., how individuals' aggression fluctuates over time) and covariance at the within-person level (e.g., how within-person changes in aggression are related to the within-person changes in somatic symptoms) (Keijsers, 2015). In addition, the RI-CLP models can examine if the influence of time-invariant variables (such as distress tolerance) varies across different time points (Mulder & Hamaker, 2021). Because somatic symptoms might be correlated with substance use (Stewart & Conrod, 2008), the current study also included the association between somatic symptoms and substance use. Because perceived stress is associated with aggression, substance use, and somatic symptoms, stress has been included in the current study as a time-varying covariate. The literature suggests sex and racial differences in aggression and substance(Mason & Windle, 2002); thus, the current study also included sex and race as the time-invariant covariates. It was hypothesized that (1) aggression (psychological and physical aggression respectively) would be positively associated with substance use (alcohol use and drug use, respectively) at both state- and trait-levels, while controlling for other variables; (2) aggression would be positively associated with somatic symptoms at both state- and trait-levels, while controlling for other variables; (3) substance use would be positively associated with somatic symptoms, after controlling for other variables; and (4) distress tolerance would be associated with aggression, substance use, and somatic symptoms at the trait level (see Figure 1). The RI-CLPM also allowed for testing

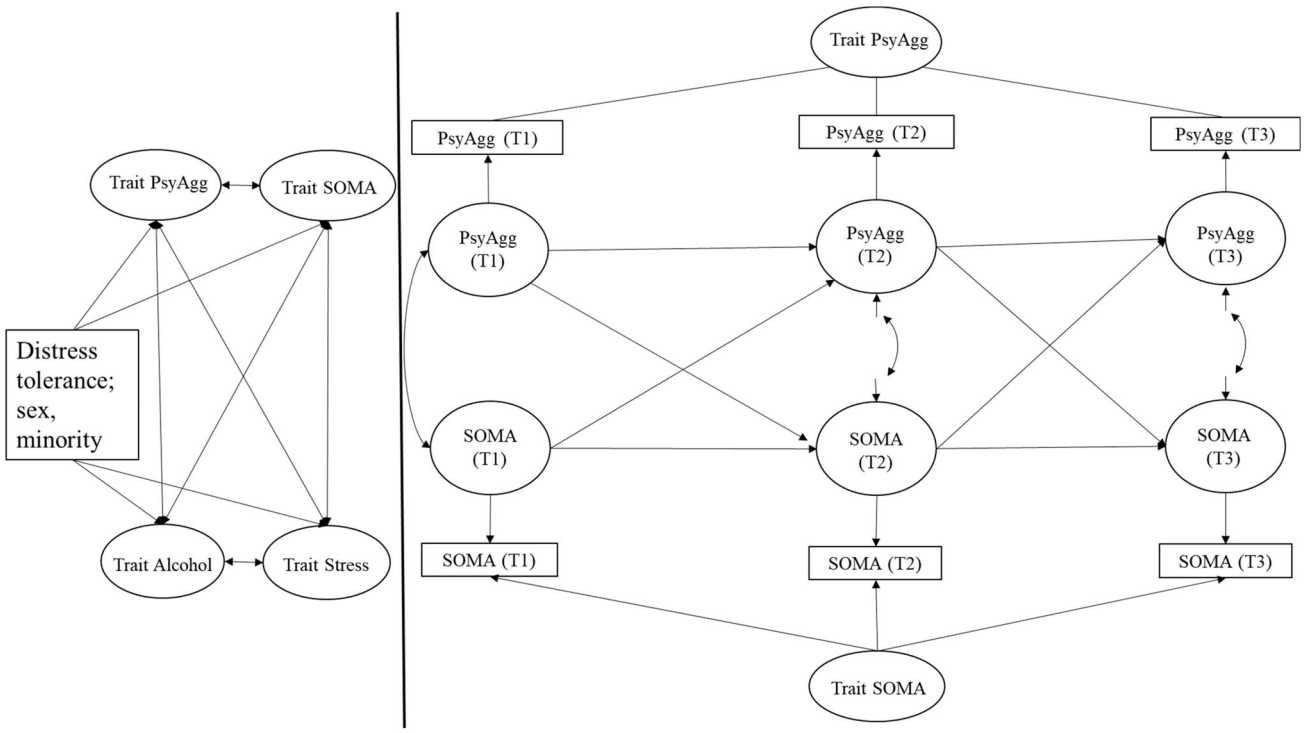


**FIGURE 1** Hypothesized model relating distress tolerance, psychological aggression, somatic symptoms, alcohol use (not including), while controlling for stress, sex, and minority (Model 1). Left panel depicts between-person latent trait correlations. Sex and minority are two time-invariant covariates. Right panel depicts within-person correlations. For simplicity, the within-person model only depicts the within-person correlations between psychological aggression and somatic symptoms. PsyAgg, psychological aggression; SOMA, somatic symptoms.

whether the influence of time-invariant variable distress tolerance on state-like variables varied across different time points. Thus, RI-CLPMs were adopted to examine if distress tolerance influenced variables directly or if the influence of distress tolerance occurred indirectly through the random intercept (Mulder & Hamaker, 2021).

## 2 | METHODS

### 2.1 | Participants

Two hundred and forty-five adult college students (69% were females; 5% were Hispanic) were recruited from a Southern university. Students were from intro-level psychology courses and learned of the study via the psychology department participant pool system and/or class announcements. Participants received course credit for participation. The data were collected between the fall of 2018 and the summer of 2019. The study was approved by the author's university Institutional Review Board. Informed consent was obtained from all individual participants included in the study before data collection. The majority of them were freshmen (64%) or sophomores (23%); the remaining were junior (8%) or senior students (5%). The mean age was 20.4 (*SD* = 2.4, ranging from 18 to 37). Self-reported race of the sample was 55% Caucasian, 35% African American, 6% more than one race, 3% Asian, 1% others.

### 2.2 | Procedure

The current study adopted a naturalistic, longitudinal design to assess participants' demographic characteristics and state-like and trait-like variables at baseline/Time 1 (T1), Time 2 (T2), and Time 3 (T3). Baseline surveys were completed in a research lab. Participants were reached out via email after 2 weeks and 4 weeks, respectively, and were invited to participate in online surveys. Demographic information and distress tolerance were assessed at baseline. The rest of the variables were assessed at each time point of data collection.

### 2.3 | Measures

**Aggression** was assessed with eight-item psychological aggression and the 12-item physical aggression subscales of the Revised Conflict Tactics Scale (CTS) (Straus et al., 1996). Participants were asked to indicate whether (0 = no, 1 = yes) they had perpetrated psychological and physical aggression toward someone in the last 2 weeks (including the current day). Sample questions for psychological aggression included "I threatened to hit or throw something at someone." A sample question for physical aggression was "I slapped someone." The CTS has good internal reliability ($\alpha$ = .79 for psychological aggression; $\alpha$ = .86 for physical aggression) (Straus et al., 1996). The internal reliability was good for the current sample ($\alpha \geq .73$ for the psychological aggression subscale across three assessments; $\alpha \geq .77$ for the physical aggression subscale across three assessments). The sum of items in each scale was used in data analysis. The data of physical aggression were transformed into a binary variable (1 = yes, 0 = no) due to extreme skewness (ranging 3.91–5.47 across three time points) and kurtosis (ranging 17.12–30.11 across three time points).

**Somatic symptoms** were assessed with the 7-item Somatization subscale of the Brief Symptom Checklist (Derogatis, 1993). Participants were asked to report how often symptoms (i.e., "faintness," "pains in heart/chest," "nausea," "trouble getting breath," "hot/cold spells," "numbness," "weakness of body") have been experienced within the last 2 weeks (including the current day) on a five-point Likert scale ranging from 0 (*not at all*) to 4 (*extremely*). The scale had good reliability in the current sample ($\alpha \geq .71$ across three assessments). The sum of the items was used in data analysis.

**Substance use** included alcohol and drug use. Alcohol use was measured with two items asking about (a) typical frequency and (b) typical quantity of alcohol consumption per day during the last 2 weeks (Jenzer et al., 2021). The frequency item was: "How often did you drink during the last two weeks? (0 = I did not drink at all, 1 = about once, 2 = two or twice a week, 3 = three to four times a week, 4 = nearly every day, 5 = once a day or more)." The quantity item was: "Think of the occasion during the last 2 weeks when you had something to drink. How many drinks did you usually have on any one occasion (response options ranging from 0 to 30)?" Alcohol use was created by multiplying the responses on these two items. Because of data skewness (2.72–2.86 across three time points) and kurtosis (8.83–12.14 across time points), the log algorithm was used to transform the data (thereafter skewness ranging from 0.39 to 0.98; kurtosis ranging from −1.25 to −0.53). Drug use was assessed with one item (i.e., How many times, in the past 2 weeks, have you engaged in illicit drug use, such as marijuana use, hard drug use (e.g., methamphetamines, cocaine, crack), inhalant use, injecting drug use, and misuse of prescription drugs (e.g., any use not specifically prescribed by a doctor). Response options include 0 = never, 1 = once or twice, 2 = 3–5 times, 3 = 6–10 times, and 4 = more than 10 times. Due to severe skewness (2.47–2.95) and kurtosis (5.42–8.61 across three time points), the log algorithm was used for data transformation but did not yield normally distributed data. Thus, the inverse function was used for data transformation, which yielded skewness of 1.37–1.80 and kurtosis of 0.19–1.01. Then, the data were multiplied by −1 to facilitate interpretation.

**Distress tolerance** was assessed using the Distress Tolerance Scale (Simons & Gaher, 2005). Individuals were asked to think of times when people feel distressed or upset and rate to which degree they agree with each of the 15 items using a 5-point Likert scale (1 = *strongly agree*, 5 = *strongly disagree*). An example item is: "When I feel distressed or upset, all I can think about is how bad I feel." The scale demonstrated good reliability in the literature (e.g., $\alpha \geq .85$; Simons & Gaher, 2005) and the current study ($\alpha$ = .86).

**Perceived stress** was assessed with the 14-item self-report Perceived Stress Scale (Cohen & Williamson, 1988), which is widely used to assess the degree to which situations in one's life are

appraised as stressful. Participants rated the frequency of their feelings (e.g., "How often have you felt nervous and stressed") and thoughts (e.g., "How often have you been able to control irritations in your life") about life events and situations over the previous 2 weeks using a five-point scale ranging from (0) *Never* to (4) *Very Often*. The internal reliability was good in the current sample ($\alpha \geq .73$).

## 2.4 | Analytic plan

Descriptives are presented in Table 1. The RI-CLPM models were conducted with Mplus 8.4 to test the hypothesized relations between two types of aggression (psychological and physical aggression, respectively), and somatic symptoms, substance use (alcohol use, drug use, respectively), and distress tolerance, resulting in four sets of RI-CLP analyses across three assessment occasions. Time-invariant covariates, sex (0 = male, 1 = female) and minority status (0 = no, 1 = yes), and time-variant covariate perceived stress were included in each RI-CLP model. Model fit indices were reported in Table 2. Following the instruction by Mulder and Hamaker (2021), the current study also compared two sets of models (unconstrained vs. constrained) to examine whether the influence of distress tolerance on the variables differed across occasions. The unconstrained models tested different influences of distress tolerance on latent variables across different time points (parameters are freely estimated). The constrained models examined the tenability of equal magnitude of influences across occasions by imposing constraints on the model. The comparison between nested models indicated that the constrained models were better fit (see Table 2). Thus, the effects of distress tolerance on substance use, psychological and physical aggression, and somatic symptoms were constrained to be same at each time point. Preliminary analyses examined the patterns of missing data and revealed that missingness at T2 and T3, respectively, was not associated with any variables ($p \geq .15$), with the exception of the T2 drug use variable which was associated with missing at T3 ($p = .02$). Thus, it was considered as missing at random because an observed variable was associated with the missingness and that variable was included in the analytic models. Missing data were handled using full information maximum likelihood estimation (FIML) estimation. FIML utilizes all available information and relies on missing at random assumption (allowing missingness to be conditioned on all variables included in this model), making it ideal for

**TABLE 1** Descriptives of the key variables.

| | Time 1 ($N = 245$) | | Time 2 ($N = 218$) | | Time 3 ($N = 204$) | |
|---|---|---|---|---|---|---|
| | Mean | *SD* | Mean | *SD* | Mean | *SD* |
| Psychological aggression | 1.34 | 2.28 | 1.21 | 2.39 | 1.03 | 2.26 |
| Physical aggression | 0.29 | 0.95 | 0.33 | 1.18 | 0.22 | 0.84 |
| Somatic symptoms | 1.89 | 0.32 | 1.84 | 0.41 | 1.77 | 0.35 |
| Daily number of drinks | 2.34 | 3.04 | 1.59 | 2.53 | 1.46 | 2.27 |
| Alcohol use | 7.56 | 12.31 | 4.61 | 8.52 | 4.28 | 7.99 |
| Distress tolerance | 3.09 | 0.61 | - | - | - | - |
| Stress | 2.92 | 0.24 | 2.85 | 0.28 | 2.79 | 0.34 |
| **Substance use** | ***n*** | ***%*** | ***n*** | ***%*** | ***n*** | ***%*** |
| Alcohol use | | | | | | |
| None | 101 | 41 | 124 | 57 | 118 | 58 |
| About once | 63 | 26 | 41 | 19 | 48 | 24 |
| Two or twice a week | 44 | 18 | 42 | 19 | 28 | 14 |
| Three to four times a week | 32 | 13 | 7 | 3 | 6 | 3 |
| Nearly every day | 4 | 2 | 3 | 1 | 4 | 2 |
| Once a day or more | 1 | 1 | 0 | 0 | 0 | 0 |
| Drug use | | | | | | |
| Never | 184 | 75 | 177 | 81 | 163 | 80 |
| Once or twice | 35 | 14 | 2 | 11 | 27 | 13 |
| 3–5 times | 10 | 4 | 7 | 3 | 9 | 4 |
| 6–10 times | 3 | 1 | 5 | 2 | 4 | 2 |
| More than 10 times | 13 | 5 | 5 | 2 | 1 | 1 |

**TABLE 2** Model fit indices.

| | χ2 | *df* | RMESA | 90% CI RMESA | Proba RMSEA ≤ 0.05 | CFI | TLI | SRMR | AIC | BIC | SBIC |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Model 1* | | | | | | | | | | | |
| Model without time-invariant covariates | 26.75 | 14 | 0.061 | 0.023, 0.096 | 0.272 | 0.987 | 0.930 | 0.032 | 4812 | 5120 | 4841 |
| Full unconstrained model | 131.11 | 16 | 0.171 | 0.145, 0.199 | 0.000 | 0.892 | 0.311 | 0.074 | 4891 | 5276 | 4927 |
| Full model with constraint loadings | 96.44 | 30 | 0.149 | 0.121, 0.178 | 0.000 | 0.915 | 0.424 | 0.086 | 4381 | 4769 | 4417 |
| *Model 2* | | | | | | | | | | | |
| Model without time-invariant covariates | 14.33 | 14 | 0.010 | 0.000, 0.063 | 0.860 | 1.000 | 0.998 | 0.028 | 2822 | 3131 | 2852 |
| Full unconstrained model | 124.63 | 16 | 0.166 | 0.140, 0.194 | 0.000 | 0.889 | 0.293 | 0.068 | 2903 | 3288 | 2939 |
| Full model with constraint loadings | 37.05 | 30 | 0.031 | 0.000, 0.060 | 0.838 | 0.994 | 0.978 | 0.031 | 3754 | 4090 | 3785 |
| *Model 3* | | | | | | | | | | | |
| Model without time-invariant covariates | 14.45 | 14 | 0.011 | 0.000, 0.063 | 0.855 | 1.000 | 0.998 | 0.024 | 3758 | 4066 | 3787 |
| Full unconstrained model | 124.22 | 16 | 0.166 | 0.140, 0.194 | 0.000 | 0.902 | 0.374 | 0.073 | 3869 | 4254 | 3905 |
| Full model with constraint loadings | 39.63 | 30 | 0.036 | 0.000, 0.064 | 0.767 | 0.990 | 0.967 | 0.036 | 2790 | 3125 | 2821 |
| *Model 4* | | | | | | | | | | | |
| Model without time-invariant covariates | 6.50 | 14 | 0.000 | 0.000, 0.997 | 0.997 | 1.000 | 1.000 | 0.020 | 1742 | 2040 | 1771 |
| Full unconstrained model | 120.86 | 16 | 0.164 | 0.137, 0.191 | 0.000 | 0.900 | 0.362 | 0.068 | 1852 | 2237 | 1888 |
| Full model with constraint loadings | 32.28 | 30 | 0.018 | 0.000, 0.052 | 0.932 | 0.998 | 0.993 | 0.030 | 1735 | 2071 | 1767 |

Abbreviations: AIC, akaike information criterion; BIC, bayesian information criterion; CFI, comparative fit index; CI, confidence interval; *df*, degrees of freedom; RMSEA, the root-mean-square error of approximation; SBIC, structural bayesian information criterion; SRMR, the standardized root-mean-square residual; TLI, Tucker–Lewis Index.

longitudinal research with repeated measures (Enders & Bandalos, 2001). The factor loadings of the time-specific measures on the latent trait variables are included in the supplementary file. A power analysis for structural equation modeling demonstrated that the sample ($N = 245$, $\alpha = .05$) had adequate power (0.80–0.95) to detect small to medium effect sizes in the models (Wang & Rhemtulla, 2021).

## 3 | RESULTS

The regression coefficients of the state-level autoregressive and cross-lagged associations are presented in Table 3. Specifically, Model 1 depicts the regression coefficients for the associations between psychological aggression, somatic symptoms, and alcohol use, while controlling for other variables. Model 2 describes the associations between psychological aggression, somatic symptoms, and drug use, while controlling for other variables. Likewise, the regression coefficients of such associations between physical aggression, somatic symptoms, and substance use, while controlling for other variables are presented in Models 3 and 4 in Table 3.

### 3.1 | Autoregressive and cross-lagged associations

With regard to autoregressive associations, none of the autoregressive associations were significant in Model 1. Model 2 revealed that T1 psychological aggression predicted T2 psychological aggression ($p = .04$), after controlling for drug use and other variables. Both Model 3 and Model 4 indicated that a higher level of T1 physical aggression predicted more T2 physical aggression ($p < .001$) after controlling for other variables. Low levels of T1 stress predicted more T2 stress in Models 2–4 ($p < .05$).

Models 1 and 2 revealed that T2 psychological aggression predicted T3 somatic symptoms ($\beta = .22–.26$, $p \leq .05$); T2 somatic symptoms predicted T3 psychological aggression ($\beta = .35–.36$, $p \leq .03$). Model 4 revealed that T1 drug use predicted T2 physical aggression ($\beta = .24$, $p = .01$) and T2 physical aggression predicted T3 somatic symptoms ($\beta = .31$, $p < .001$). In addition, all models indicated that T1 somatic symptoms predicted Time 2 stress ($\beta = .38–.47$, $p \leq .05$). The findings indicated that there was a reciprocal relation between psychological aggression and somatic symptoms on later occasions. In addition, T1 drug use predicted T2 physical aggression, which in turn predicted T3 somatic symptoms. There was a unidirectional effect of somatic symptoms on stress on earlier occasions.

**TABLE 3** Autoregressive and cross-lagged regression paths.

| Regression paths | T1 → T2 | | | | T2 → T3 | | | |
|---|---|---|---|---|---|---|---|---|
| | *β* | *SE* | *p* Value | 95% CI | *β* | *SE* | *p* Value | 95% CI |
| *Model 1* | | | | | | | | |
| Autoregressive paths | | | | | | | | |
| Alc → Alc | .25 | 0.14 | .07 | [−0.024, 0.515] | .10 | 0.20 | .62 | [−0.293, 0.495] |
| PsyAgg → PsyAgg | .19 | 0.12 | .12 | [−0.049, 0.422] | −.20 | 0.18 | .26 | [−0.538, 0.148] |
| SOMA → SOMA | .14 | 0.14 | .31 | [−0.131, 0.410] | .15 | 0.13 | .26 | [−0.109, 0.403] |
| Stress → Stress | .52 | 0.27 | .06 | [−1.047, 0.010] | .09 | 0.12 | .45 | [−0.143, 0.324] |
| Cross-lagged paths | | | | | | | | |
| Alc → PsyAgg | −.02 | 0.11 | .90 | [−0.238, 0.208] | .01 | 0.19 | .98 | [−0.368, 0.384] |
| Alc → SOMA | .07 | 0.12 | .54 | [−0.161, 0.306] | .13 | 0.13 | .32 | [−0.125, 0.384] |
| Alc → Stress | .06 | 0.19 | .74 | [−0.305, 0.431] | −.02 | 0.13 | .87 | [−0.284, 0.239] |
| PsyAgg → Alc | −.07 | 0.13 | .62 | [−0.321, 0.191] | .23 | 0.15 | .14 | [−0.072, 0.531] |
| PsyAgg → SOMA | .11 | 0.12 | .36 | [−0.124, 0.342] | **.22** | **0.11** | **.05** | **[−0.004, 0.442]** |
| PsyAgg → Stress | .12 | 0.17 | .49 | [−0.218, 0.456] | .03 | 0.12 | .82 | [−0.203, 0.256] |
| SOMA → Alc | .13 | 0.13 | .32 | [−0.122, 0.377] | .01 | 0.15 | .94 | [−0.273, 0.296] |
| SOMA → PsyAgg | .13 | 0.12 | .28 | [−0.103, 0.356] | **.35** | **0.16** | **.02** | **[0.046, 0.653]** |
| SOMA → Stress | **.39** | **0.19** | **.04** | **[0.015, 0.758]** | .19 | 0.13 | .13 | [−0.056, 0.439] |
| Stress → Alc | −.09 | 0.16 | .58 | [−0.393, 0.219] | −.12 | 0.12 | .32 | [−0.353, 0.115] |
| Stress → PsyAgg | −.08 | 0.14 | .56 | [−0.360, 0.194] | −.08 | 0.14 | .55 | [−0.354, 0.188] |
| Stress → SOMA | −.11 | 0.16 | .47 | [−0.416, 0.191] | .09 | 0.11 | .43 | −0.129, 0.300] |
| *Model 2* | | | | | | | | |
| Autoregressive paths | | | | | | | | |
| Drugs → Drugs | −.03 | 0.16 | .84 | [−0.349, 0.284] | .02 | 0.23 | .93 | [−0.433, 0.476] |
| PsyAgg → PsyAgg | **.23** | **0.11** | **.04** | **[0.015, 0.448]** | −.07 | 0.15 | .64 | [−0.369, 0.226] |
| SOMA → SOMA | .13 | 0.14 | .35 | [−0.139, 0.394] | .10 | 0.16 | .53 | [−0.206, 0.404] |
| Stress → Stress | **.60** | **0.27** | **.03** | **[−1.126, −0.070]** | .04 | 0.13 | .76 | [−0.210, 0.286] |
| Cross-lagged paths | | | | | | | | |
| Drugs → PsyAgg | .03 | 0.11 | .76 | [−0.188, 0.257] | −.05 | 0.18 | .78 | [−0.402, 0.303] |
| Drugs → SOMA | .09 | 0.12 | .46 | [−0.148, 0.328] | .06 | 0.16 | .71 | [−0.250, 0.368] |
| Drugs → Stress | .20 | 0.17 | .25 | [−0.140, 0.533] | −.16 | 0.16 | .30 | [−0.474, 0.147] |
| PsyAgg → Drugs | −.02 | 0.14 | .91 | [−0.286, 0.256] | .20 | 0.16 | .19 | [−0.102, 0.506] |
| PsyAgg → SOMA | .09 | 0.11 | .41 | [−0.130, 0.318] | **.26** | **0.11** | **.02** | **[0.045, 0.481]** |
| PsyAgg → Stress | .01 | 0.17 | .97 | [−0.326, 0.339] | .05 | 0.12 | .66 | [−0.181, 0.286] |
| SOMA → Drugs | .01 | 0.15 | .96 | [−0.288, 0.303] | −.05 | 0.20 | .81 | [−0.436, 0.341] |
| SOMA → PsyAgg | .13 | 0.11 | .26 | [−0.096, 0.350] | **.36** | **0.17** | **.03** | **[0.030, 0.683]** |
| SOMA → Stress | **.38** | **0.19** | **.05** | **[0.004, 0.748]** | .26 | 0.16 | .09 | [−0.042, 0.565] |
| Stress → Drugs | .004 | 0.19 | .98 | [−0.367, 0.376] | .11 | 0.15 | .46 | [−0.188, 0.414] |
| Stress → PsyAgg | −.11 | 0.14 | .44 | [−0.386, 0.169] | −.10 | 0.13 | .42 | [−0.357, 0.149] |
| Stress → SOMA | −.13 | 0.16 | .40 | [−0.443, 0.175] | .09 | 0.12 | .46 | [−0.141, 0.313] |

(Continues)

**TABLE 3** (Continued)

| Regression paths | T1 → T2 | | | | T2 → T3 | | | |
|---|---|---|---|---|---|---|---|---|
| | *β* | *SE* | *p* Value | 95% CI | *β* | *SE* | *p* Value | 95% CI |
| *Model 3* | | | | | | | | |
| Autoregressive paths | | | | | | | | |
| Alc → Alc | .22 | 0.13 | .08 | [−0.029, 0.475] | .04 | 0.22 | .86 | [−0.384, 0.459] |
| PhyAgg → PhyAgg | **.30** | **0.09** | **.00** | **[0.122, 0.481]** | .10 | 0.12 | .42 | [−0.142, 0.337] |
| SOMA → SOMA | .17 | 0.13 | .21 | [−0.092, 0.425] | .15 | 0.13 | .24 | [−0.100, 0.394] |
| Stress → Stress | **.65** | **0.31** | **.04** | [−1.264, −0.040] | .08 | 0.11 | .46 | [−0.138, 0.306] |
| Cross-lagged paths | | | | | | | | |
| Alc → PhyAgg | .14 | 0.09 | .15 | [−0.049, 0.321] | −.04 | 0.14 | .75 | [−0.320, 0.231] |
| Alc → SOMA | .07 | 0.11 | .51 | [−0.141, 0.287] | .12 | 0.13 | .36 | [−0.136, 0.375] |
| Alc → Stress | .16 | 0.18 | .39 | [−0.201, 0.512] | .002 | 0.13 | .99 | [−0.255, 0.259] |
| PhyAgg → Alc | .10 | 0.11 | .38 | [−0.121, 0.318] | .10 | 0.15 | .53 | [−0.205, 0.398] |
| PhyAgg → SOMA | .14 | 0.10 | .19 | [−0.066, 0.342] | **.31** | **0.11** | **.00** | **[0.105, 9.521]** |
| PhyAgg → Stress | −.17 | 0.20 | .38 | [−0.559, 0.211] | .14 | 0.11 | .21 | [−0.080, 0.357] |
| SOMA → Alc | .11 | 0.13 | .41 | [−0.147, 0.357] | .01 | 0.16 | .93 | [−0.304, 0.331] |
| SOMA → PhyAgg | .12 | 0.10 | .26 | [−0.087, 0.316] | .02 | 0.12 | .87 | [−0.222, 0.264] |
| SOMA → Stress | **.47** | **0.21** | **.02** | **[0.063, 0.875]** | .20 | 0.12 | .11 | [−0.042, 0.442] |
| Stress → Alc | −.04 | 0.17 | .81 | [−0.370, 0.290] | −.06 | 0.13 | .62 | [−0.314, 0.187] |
| Stress → PhyAgg | .05 | 0.14 | .70 | [−0.215, 0.320] | .05 | 0.10 | .63 | [−0.152, 0.250] |
| Stress → SOMA | −.08 | 0.16 | .63 | [−0.401, 0.243] | .13 | 0.10 | .22 | [−0.074, 0.324] |
| *Model 4* | | | | | | | | |
| Autoregressive paths | | | | | | | | |
| Drugs → Drugs | .05 | 0.15 | .76 | [−0.254, 0.347] | .08 | 0.21 | .73 | [−0.335, 0.485] |
| PhyAgg → PhyAgg | **.35** | **0.09** | **.00** | **[0.185, 0.523]** | .14 | 0.12 | .26 | [−0.100, 0.375] |
| SOMA → SOMA | .15 | 0.13 | .24 | [−0.101, 0.400] | .08 | 0.15 | .62 | [−0.222, 0.371] |
| Stress → Stress | **.68** | **0.30** | **.05** | **[−1.267, −0.086]** | .06 | 0.12 | .62 | [−0.172, 0.289] |
| Cross-lagged paths | | | | | | | | |
| Drugs → PhyAgg | **.24** | **0.10** | **.01** | **[0.056, 0.430]** | −.17 | 0.15 | .23 | [−0.458, 0.111] |
| Drugs → SOMA | .16 | 0.12 | .18 | [−0.071, 0.383] | .13 | 0.15 | .41 | [−0.171, 0.421] |
| Drugs → Stress | .21 | 0.18 | .25 | [−0.146, 0.572] | −.14 | 0.15 | .35 | [−0.433, 0.154] |
| PhyAgg → Drugs | −.003 | 0.13 | .98 | [−0.260, 0.253] | .27 | 0.14 | .06 | [−0.013, 0.551] |
| PhyAgg → SOMA | .14 | 0.10 | .18 | [−0.064, 0.340] | **.31** | **0.10** | **.00** | **[0.108, 0.516]** |
| PhyAgg → Stress | −.15 | 0.19 | .45 | [−0.523, 0.233] | .17 | 0.11 | .12 | [−0.044, 0.388] |
| SOMA → Drugs | .07 | 0.14 | .62 | [−0.209, 0.348] | −.04 | 0.18 | .85 | [−0.389,−0.319] |
| SOMA → PhyAgg | .09 | 0.10 | .35 | [−0.099, 0.281] | .10 | 0.14 | .48 | [−0.180, 0.384] |
| SOMA → Stress | **.42** | **0.20** | **.03** | **[0.032, 0.812]** | .26 | 0.15 | .08 | [−0.033, 0.546] |
| Stress → Drugs | −.01 | 0.19 | .98 | [−0.379, 0.368] | .16 | 0.13 | .24 | [−0.103, 0.418] |
| Stress → PhyAgg | .05 | 0.13 | .71 | [−0.206, 0.301] | .01 | 0.11 | .93 | [−0.197, 0.215] |
| Stress → SOMA | −.11 | 0.16 | .52 | [−0.425, 0.213] | .15 | 0.11 | .18 | [−0.066, 0.359] |

*Note*: Bold values are statistically significant.

Abbreviations: Alc, alcohol use; CI, confidence interval; PhyAgg, physical aggression; PsyAgg, psychological aggression; SOMA, somatic symptoms.

**TABLE 4** Correlations among between-subjects variables and correlations between latent state variables.

| Random-intercept correlations depicting correlations of between-person variables | | | | Correlations between latent state variables | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | T1 | | | T2 | | | T3 | | |
| Model 1 | Alc | PsyAgg | SOMA | Alc | PsyAgg | SOMA | Alc | PsyAgg | SOMA | Alc | PsyAgg | SOMA |
| PsyAgg | 0.29* | | | −0.10 | | | 0.18 | | | 0.06 | | |
| SOMA | 0.13 | 0.32** | | 0.06 | 0.09 | | −0.06 | 0.17 | | 0.12 | 0.26* | |
| Stress | 0.22 | 0.21* | 0.47*** | 0.03 | 0.10 | 0.30* | −0.17 | 0.02 | 0.44** | −0.12 | −0.04 | 0.37*** |
| Model 2 | Drugs | PsyAgg | SOMA | Drugs | PsyAgg | SOMA | Drugs | PsyAgg | SOMA | Drugs | PsyAgg | SOMA |
| PsyAgg | 0.22* | | | 0.08 | | | 0.18 | | | 0.07 | | |
| SOMA | 0.19 | **0.31**** | | 0.03 | 0.09 | | 0.40** | 0.17 | | 0.21 | 0.27* | |
| Stress | 0.21* | **0.24*** | **0.49***** | 0.11 | 0.03 | 0.27 | −0.02 | −0.02 | 0.44* | −0.05 | −0.01 | 0.37*** |
| Model 3 | Alcohol | PhyAgg | SOMA | Alc | PhyAgg | SOMA | Alc | PhyAgg | SOMA | Alc | PhyAgg | SOMA |
| PhyAgg | 0.11 | | | 0.06 | | | 0.34* | | | 0.00 | | |
| SOMA | 0.10 | 0.26 | | 0.09 | 0.11 | | −0.07 | 0.11 | | 0.19 | 0.18 | |
| Stress | 0.16 | **0.38**** | **0.46***** | 0.09 | −0.11 | 0.31* | −0.09 | −0.04 | 0.56* | −0.08 | 0.10 | 0.35*** |
| Model 4 | Drugs | PhyAgg | SOMA | Drugs | PhyAgg | SOMA | Drugs | PhyAgg | SOMA | Drugs | PhyAgg | SOMA |
| PhyAgg | 0.25 | | | −0.09 | | | 0.31* | | | 0.24* | | |
| SOMA | 0.13 | 0.31 | | 0.08 | 0.08 | | 0.44*** | 0.08 | | 0.25* | 0.17 | |
| Stress | 0.19 | **0.41**** | **0.46***** | 0.12 | −0.11 | 0.28 | 0.00 | −0.08 | 0.52* | −0.04 | 0.09 | 0.36*** |

*Note*: Bold values are statistically significant.

Abbreviations: Alc, alcohol use; PhyAgg, physical aggression; PsyAgg, psychological aggression; SOMA, somatic symptoms.

*$p < .05$; **$p \leq .01$; ***$p \leq .001$.

## 3.2 | Within-wave correlations

Table 4 presents correlations between time-specific latent state variables. The covariance depicts whether individuals with higher residuals for one latent state variable also had higher residuals for another state variable. At T1, somatic symptoms were associated with stress (*rs* = .30–.31, $p < .05$). T2 somatic symptoms were positively associated with T2 drug use (*rs* = .40–.44, $p < .01$). T2 physical aggression was positively associated with T2 alcohol use $r = .34$, $p = .02$) and T2 drug use, ($r = .31$, $p = .02$) respectively. T3 psychological aggression was positively associated with T3 somatic symptoms (*rs* = .26–.27, $p < .05$). T3 drug use was positive associated with T3 physical aggression ($r = .24$, $p = .03$) and T3 somatic symptoms ($r = .25$, $p = .02$), respectively. Somatic symptoms were associated with stress at T2 and T3 (*rs* = .35–.56, $p < .05$).

## 3.3 | Correlations between trait-like latent constructs (Random Intercepts)

Correlations between random intercepts are the associations between the mean levels of variables, which reflect the latent trait-level correlations (see Table 4). The medium-to-high magnitude of the correlations between variables across time points indicates that individuals with a higher mean level of psychological aggression (i.e., who had more psychological aggression across three time points) tended to experience a greater degree of somatic symptoms ($r = .31–.32$, $p < .01$) across three time points. This reflects trait-like correlations. Likewise, the mean level of psychological aggression was positively associated with the mean levels of alcohol use ($r = .29$, $p = .05$), drug use ($r = .22$, $p = .04$), and stress ($r = .21–.24$, $p < .05$), respectively. Trait-like physical aggression was only associated with trait-like stress ($r = .38–.41$, $p < .01$). However, the mean level of physical aggression was not significantly associated with the mean level of other variables.

## 3.4 | The effect of distress tolerance

The fit of the constrained and unconstrained models indicated that the magnitude of the influence of distress tolerance on aggression, somatic symptoms, and substance use was equivalent across time points. The regression estimates describing the associations between distress tolerance and the latent constructs are reported in Table 5. The findings across four models partially supported the hypotheses. In Models 1 and 2, distress tolerance was negatively associated with psychological aggression ($\beta = -.20$ to $-.19$, $p = .01$), somatic symptoms ($\beta = -.43$ to $.41$, $p < .001$), and perceived stress ($\beta = -.50$ to $-.49$,

**TABLE 5** Correlations between time-invariant predictors and trait-level latent variables.

| | Distress tolerance | Female | Minority |
|---|---|---|---|
| Model 1 | | | |
| PsyAgg | **−0.19**** | 0.07 | 0.11 |
| SOMA | **−0.41***** | **0.19**** | 0.04 |
| Alcohol | −0.11 | −0.09 | −0.35 |
| Stress | **−0.50***** | **0.18**** | **0.14*** |
| Model 2 | | | |
| PsyAgg | **−0.20**** | 0.07 | 0.11 |
| SOMA | **−0.41***** | **0.18*** | 0.05 |
| Drugs | −0.03 | −0.004 | −0.02 |
| Stress | **−0.49***** | **0.18**** | **0.14*** |
| Model 3 | | | |
| PhyAgg | −0.13 | 0.001 | **0.25*** |
| SOMA | **−0.43***** | **0.18*** | 0.06 |
| Alcohol | −0.10 | −0.11 | **−0.34***** |
| Stress | **−0.49***** | **0.18**** | **0.14*** |
| Model 4 | | | |
| PhyAgg | −0.15 | 0.01 | 0.25 |
| SOMA | **−0.43***** | **0.17*** | 0.06 |
| Drugs | −0.03 | 0.02 | 0.00 |
| Stress | **−0.49***** | **0.18**** | **0.14*** |

*Note*: Bold values are statistically significant.

Abbreviations: PhyAgg, physical aggression; PsyAgg, psychological aggression; SOMA, somatic symptoms.

*$p < .05$; **$p \leq .01$; ***$p \leq .001$.

$p < .001$). However, in Models 3 and 4, distress tolerance was not associated with physical aggression, alcohol use, or drug use. Females reported higher trait-like somatic symptoms ($\beta = .17–.19$, $p < .05$) and stress compared to males ($\beta = .18$, $p = .002$). Minority students reported higher levels of alcohol use ($p < .001$) and stress than did white students.

### 3.5 | Sensitivity analysis

Multilevel modeling was used to examine the partial correlations among within-person variables, after controlling for between-persons variables, comprised of distress tolerance, female, and minority, as a comparison to the cross-lagged effects revealed in the RI-CLPM analyses. The results (see Supporting Information: Table 2) revealed that psychological aggression was positively associated with physical aggression, $\beta = .52$, $p < .001$, and distress tolerance $\beta = -.23$, $p = .03$. Minority was the only significant predictor of physical aggression, $\beta = .96$, $p = .02$. Somatic symptoms were associated with drug use, $\beta = .12$, $p < .001$, stress, $p = .41$, $p < .001$, and distress tolerance, $\beta = -.22$, $p < .001$. Alcohol use was associated with drug use, $\beta = .15$, $p < .001$, and minority, $\beta = -.25$, $p < .001$. Stress was associated with distress tolerance, $\beta = -.27$, $p < .001$, female, $\beta = .17$, $p = .003$, minority, $\beta = .11$, $p = .04$.

## 4 | DISCUSSION

The present study adopted a naturalistic, longitudinal design to examine (1) the reciprocal relationships between each form of aggression, somatic symptoms, and alcohol and drug use and (2) the effect of distress tolerance on these relationships, while controlling for perceived stress, sex, and minority. A significant strength of this study was the methodological and analytical features of a RI-CLPM design. The three-panel cross-lagged component of the RI-CLPM design disaggregated within-person (state-like) dynamic processes and between-person (trait-like) associations and provided strong inferential evidence for examining reciprocal relations between the studied variables. The within-person dynamic processes captured the correlations between variables of interest within the same person across different time points, whereas between-person associations reflected the general patterns of relationships between cross-sectional variables. The within-person dynamic processes included the autoregressive (the value of a current variable predicted by the same construct assessed at a previous time T1; e.g., T2 psychological aggression being predicted by T1 psychological aggression) and cross-lagged relations (the value of a current variable predicted by a correlated, yet different construct assessed at a previous time; e.g., T2 psychological aggression being predicted by T1 drug use) among three time points as well as within-wave within-person correlations. The between-person correlations included correlations between mean levels of variables across three time points and the association of distress tolerance to these variables.

The autoregressive effect was found in both psychological and physical aggression between T1 and T2. T1 psychological aggression predicted T2 psychological aggression. Likewise, T1 physical aggression predicted T2 physical aggression. These significant autoregressive effects indicated a carry-over effect of psychological and physical aggression on earlier occasions. The autoregressive paths were not significant at later time points, providing little evidence that fluctuations in state psychological aggression endured over time. Therefore, psychological aggression and physical aggression were also likely explained by a more stable trait-like component (e.g., distress tolerance for psychological aggression, as discussed later).

With regard to cross-lagged effects, psychological aggression, and somatic symptoms had a reciprocal, temporal effect with each other at a later lagged time period (i.e., T2–T3). Somatic symptoms were a temporal predictor of stress; however, the temporal influence of which did not carry over to a later time point (from T2 to T3). T1 drug use predicted T2 physical aggression, which in turn predicted T3 somatic symptoms. This finding supported the mediational effect of physical aggression on the association between drug use and somatic symptoms. The cross-lagged effect between T1 drug use and T2

physical aggression was consistent with the previous findings on the positive association between drug use and aggression (Hoaken & Stewart, 2003; Roozen et al., 2011; Stuart et al., 2008). According to the $I^3$ theory of aggression, inhibiting factors moderate the association between an instigating factor and aggression propensity (Finkel et al., 2012). To this end, drug use lowers inhibition and, therefore, lead to aggression. Regarding within-wave correlations, T2 somatic symptoms were positively associated with T2 drug use. T2 physical aggression was positively associated with T2 alcohol use and T2 drug use, respectively. T3 psychological aggression was positively associated with T3 somatic symptoms. Somatic symptoms were associated with stress at all times. The *innovations* or *impulses* (Falkenström et al., 2022) indicated that there were other systematic changes accounted for the aforementioned within-wave correlations between aggression, somatic symptoms, and substance use between T2 and T3; these changes cannot be explained by autoregression or cross-lagged effects. That is, there were other unobserved, third variables (e.g., neuroticism) that may explain the associations of physical aggression to alcohol and drug use at T2, the association between somatic symptoms and drug use at T2, and the association between psychological aggression and somatic symptoms at T3.

Regarding the trait-like relationships (analogous to associations between cross-sectional variables), psychological aggression was associated with all variables, in the form of positively correlated random intercepts. Individuals who reported higher severity on one factor (e.g., psychological aggression) tended to report greater severity on another factor (e.g., alcohol use). In contrast, physical aggression was only associated with stress. Given that psychological aggression was associated with a wider range of psychopathology than physical aggression, psychological aggression may be more damaging to psychological and physical health than physical aggression. This underscores the necessity of obtaining a nuanced understanding of different forms of aggression, instead of treating them as a broad category as in most studies.

The study revealed that distress tolerance was negatively associated with psychological aggression, somatic symptoms, and stress and the influence did not differ across time points. The findings suggest that low distress tolerance is a risk factor to verbalizing aggressive behaviors and experiencing somatic symptoms across times. This finding was consistent with the literature that low distress tolerance was associated with various psychopathologies (Mattingley et al., 2022). The finding mounts to the literature suggesting that distress tolerance is considered as a transdiagnostic risk and maintenance factor to many forms of psychopathological and behavioral symptoms. The finding also adds to the literature by showing such an influence being stable across times. In addition, the findings could be explained by the GAM of aggression (Anderson & Bushman, 2002). People with low distress tolerance may misattribute an event and, thus, be more likely to experience negative emotions and adopt aggression. Distress tolerance was not associated with alcohol use, drug use, or physical aggression. In the current study, distress tolerance was operationalized as a capacity to shield from experiencing emotional distress; thus, it could have a stronger, conceptual connection with psychological dysfunctioning, compared to behavioral symptoms. With alcohol use being conceptualized as a coping mechanism, the current study did not find cross-lagged and between-person associations between aggression and alcohol use. A lack of significant relations might be due to the perceived norms of drinking among college students, wherein alcohol consumption primarily serves for socialization purposes (Borsari & Carey, 2003).

The results of the sensitivity analyses showed that somatic symptoms were positively correlated with somatic symptoms, which was consistent with the results of the RI-CLPM analyses. However, the association between psychological aggression and somatic symptoms as well as that between physical aggression and somatic symptoms were not significant in the multilevel modeling. Nor was the association between physical aggression and drug use in the multilevel modeling. The different results speak to the different functions of multilevel modeling and RI-CLPM, in which multilevel modeling examines the within-person correlations at the same time point, whereas RI-CLPM decomposes the within-person correlations into within-person concurrent correlations (e.g., the association between aggression and somatic symptoms at T2 and T3, respectively) and within-person cross-lagged effects (e.g., the association between T2 aggression and T3 somatic symptoms).

## 5 | LIMITATIONS AND FUTURE DIRECTIONS

There are several limitations. First, the participants were predominantly female, emerging adults, the findings may not be able to extend to other populations, such as mature adults, who may respond differently to situational triggers and, thus, lead to different embodiments of psychopathology. Relatedly, the current study revealed sex and racial differences in some key variables (e.g., sex differences in somatic symptoms, and racial differences in physical aggression). It would be important to understand to which extent the findings can apply to other populations and examine the moderation effect of sex and race. Second, although interpersonal stress was controlled as a covariate, additional proximal (e.g., violence exposure, social network) or distal environmental (e.g., childhood experiences) factors can contribute to the symptoms of aggression, somatic complaints, and substance use. Furthermore, real-time contextual triggers might also heighten the risk of aggression and related symptoms. Relatedly, the current study adopted self-report measures, the findings of which may be impacted by recall bias and social desirability. People may underreport (e.g., lack of recollection) or over-report (e.g., due to an association with negative affect) the somatic symptoms. Individuals may tend to underreport the prevalence of aggression and substance use, due to social desirability. Future research might consider expanding to factors at interindividual levels as well as extending the duration of data collection and adopting ecologically valid methodologies that allow for testing reciprocal relations between individual and environmental variables. Third, the current study did not differentiate different types of drugs. Given the diverse psycho-pharmacological effects of substances and different prevalence rates of

drugs (e.g., marijuana is popular among young adults), it would be important to examine if the cross-lagged association between drug use and physical aggression can be replicated in different types of drugs. Fourth, some of the differences between the current findings and the literature may be due to different analytic designs and differing lags between time points. Other statistical methods, such as stable trait, autoregressive trait, state models, or latent curve models with structured residuals, might provide additional information that enhances the understanding of intricate relationships between these variables. Fifth, the current study observed a bidirectional relationship between psychological aggression and somatic symptoms at later assessment occasions. It might be interesting to examine if such a reciprocal effect is acute or can be extended beyond 2 weeks. The findings may benefit from more extended longitudinal designs, in conjunction with different lags between time points, to clarify if the observed bidirectional associations between psychological aggression and somatic symptoms could extend to a longer period of time or lagging period. Sixth, given the relatively low rates of physical aggression (22%–33%) and drug use (19%–25%), this study may have been underpowered to detect small associations between these low-rate events and other variables. Seventh, given the within-wave, within-person correlations (e.g., T2 physical aggression and T2 somatic symptoms), future research is needed to explore potential variables (e.g., a sports event night for correlation between physical aggression and alcohol use at T2; poor sleep quality for correlation between psychological aggression and somatic symptoms at T3) underlying the innovations or impulses that are uniquely captured by the RI-CLPM models. Eighth, the current did not account for the influence of personality traits on aggression, somatic symptoms, and substance use. For example, neuroticism has been shown to predict somatic complaints and substance use (Denovan et al., 2019; Turiano et al., 2012). Future research may consider incorporating personality traits in a dynamic model to identify to which degree a personality trait accounts for the within-person correlations. Likewise, additional factors may contribute to both aggression and substance use, which could moderate the associations between the constructs revealed in the current study. For example, these hypothesized relations may be strengthened by exposure to intimate partner violence or affiliation with fraternity or sorority organizations. Ninth, the current study only included cisgender participants. However, given the high prevalence of aggression and substance use among sexual minority groups (e.g., Anderson et al., 2022; Rosner et al., 2021), it would be important to test the hypothesized relations within sexual minority people.

## 6 | IMPLICATIONS

The current study has several implications for understanding and reducing aggression, somatic symptoms, and substance use among college students. First, building upon the evidence speaking to the reciprocal relation between psychological aggression and somatic symptoms, clinicians may consider integrating physical health into prevention and intervention for aggression and including interpersonal conflict and aggression as part of screening for somatic symptoms. A short checklist of psychological and physical aggression might help clinicians to identify specific intervention needs. Relatedly, mental health programs may consider including psychological and physical aggression as an important set of risk factors when evaluating college students' well-being and identifying specific intervention needs. In addition, colleges may consider delivering educational programs to raise public awareness of the potential influence of psychological aggression on health and well-being. Because of the association substance use and physical aggression, social contexts wherein substance use is more prevalent (such as weekends, game nights, fraternity or sorority events) may warrant more prevention and harm-reduction efforts. Colleges may also capitalize on these social events to publicize the opportunities of relevant educational programs. Given psychological aggression correlating with alcohol use at the trait and state levels, skill training on how to de-escalate interpersonal conflict may need to be incorporated into school-wide efforts regarding alcohol prevention. Given the stable influence of distress tolerance on psychological aggression and somatic symptoms, empirically supported therapies, such as dialectical behavioral therapy (e.g., self-soothing, managing extreme arousal), that target distress tolerance could potentially mitigate the risk of aggression and somatic symptoms (Harned et al., 2014).

## DATA AVAILABILITY STATEMENT

Data and Mplus syntax can be accessed here in Open Science Framework via https://osf.io/b9scm/?view_only=26dfe63ee9b94f1b9b338f91988493cd.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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