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Master's thesis

Prolonged Grief, Depressive, and Post-Traumatic Stress Symptoms: Random-Intercept Cross-Lagged Panel Analyses

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Abstract

Prolonged grief symptoms co-occur with depressive and, post-traumatic stress (PTS) symptoms, but the temporal relationships between prolonged grief symptoms and other post-loss symptomatology are not well understood. Insights into the temporal relationships can inform treatments for bereaved adults. To clarify these relationships, 307 bereaved adults within their first bereavement year (78% female) completed questionnaires to assess prolonged grief, depressive, and PTS symptoms at five timepoints at 1.5-month intervals. Random-intercept cross-lagged panel modeling was utilized to inspect reciprocal relationships between prolonged grief, depressive, and PTS symptoms. Higher prolonged grief symptoms than usual predicted other psychopathology symptoms and vice versa in all bivariate models. In a model including prolonged grief, depressive, and PTS symptoms, within-person fluctuations in grief levels close after the loss predicted other post-loss symptoms but not vice versa. Most variance could be attributed to stable differences between individuals. Future research should examine the clinical utility of targeting acute grief reactions to prevent the emergence of other post-loss symptomatology.

Keywords: prolonged grief symptoms; comorbidity; co-occurrence; longitudinal relationship; posttraumatic stress symptoms; random-intercept cross-lagged analyses

Prolonged Grief, Depressive, and Posttraumatic Stress Symptoms: Random-Intercept Cross-Lagged Panel Analyses

A substantial minority of bereaved individuals experience an intense and enduring grief reaction known as prolonged grief. Similar but distinct diagnoses characterized by such grief reactions have been added to ICD-11 and DSM-5-TR as prolonged grief disorder (American Psychiatric Association, 2022; Eisma et al., 2022; World Health Organization, 2018). PGD is characterized by persistent yearning for the deceased and/or preoccupation with the deceased, combined with symptoms indicative of emotional pain. Approximately 10 percent of bereaved adults are estimated to be at risk of developing prolonged grief (Lundorff et al., 2017). Prolonged grief symptoms have been associated with various adverse physical and mental health outcomes such as sleep disturbances (de Lang et al., 2023), cardiac health problems (Pini et al., 2015), and reduced quality of life (Eisma & Schmitt, 2024).

Alongside prolonged grief, various comorbid mental health problems, including depression and post-traumatic stress (PTS), may emerge following bereavement (e.g., Eisma et al., 2019; Komischke-Konnerup et al., 2021; Lenferink et al., 2017; Wen et al., 2023). Prolonged grief shares descriptive characteristics with neighboring disorders, such as depression and PTSD, including negative emotions (e.g. sadness) and avoidance of reminders of negative life events, respectively. However, the core feature of prolonged grief is separation distress (i.e., yearning for the deceased and preoccupation with the loss), differentiating it from these neighboring disorders (Maercker & Znoj, 2010). Moreover, factor analyses and latent class analyses have relatively consistently distinguished prolonged grief symptoms from depressive and PTS symptoms (e.g., Eisma et al., 2019; Grafiadeli et al., 2022; Kokou-Kpolou et al., 2021; Lenferink et al., 2017; Spuij et al., 2012; for a review see: Heeke et al., 2023).

While the similarities, differences, and comorbidity between prolonged grief and neighboring disorders have received ample scientific attention, less is known about their temporal relationships. Understanding these relationships is clinically relevant as it can help guide treatment strategies for severely distressed bereaved adults. For instance, if symptoms of prolonged grief predict subsequent symptoms of depression and/or PTS, it may be advisable to prioritize the treatment of severe grief reactions early in the therapeutic process. Prolonged grief symptoms may contribute to the emergence of other post-loss pathologies, as specific prolonged grief symptoms could serve as mechanisms explaining the exacerbation of other symptoms (Janshen & Eisma, 2024). For example, loss-related avoidance in PGD may generalize to other forms of avoidance in PTSD, while difficulty moving on with life may lead to inactivity worsening depressive symptomatology.

A recent systematic review of eight longitudinal studies demonstrated that research on the temporal relationships of prolonged grief with depressive and/or PTS symptoms shows considerable methodological heterogeneity (Janshen & Eisma, 2024). Most studies to date have applied traditional cross-lagged panel modeling (CLPM) and showed that PG symptoms more consistently predict other post-loss pathology across time than vice versa (Djelantik et al., 2018; Lenferink et al., 2019; Wen et al., 2022; Yan et al., 2021). However, traditional CLPM has faced criticism for not separating within- and between-person effects. Since it is reasonable to assume that most psychological constructs exhibit some degree of time-invariant stability indicative of a trait-like characteristic, the autoregressive and cross-lagged effects will not reflect the actual within-person effect but are contaminated by the stable between-person effect. Random-intercept cross-lagged panel modeling (RI-CLPM), addresses this problem by separating within- and between-person variance, and therefore presents a more robust alternative to the traditional CLPM (Hamaker et al., 2015).

The two studies to date that inspected temporal relationships of prolonged grief symptoms with other post-loss symptomatology using RI-CLPM did not find consistent results (Glad et al., 2022; Komischke-Konnerup et al., 2023). In the first study, the temporal relationship between prolonged grief symptoms and PTS symptoms was examined in individuals who experienced bereavement due to the Utøya massacre (Glad et al., 2022). The findings revealed that higher PTS symptoms at 14-15 months post-loss were predictive of an increase in prolonged grief symptoms at 30-32 months post-loss. However, the reverse relationship was not observed and no other significant cross-lagged effects were identified in the study. In the second study, the temporal relationships between prolonged grief and depressive and PTS symptoms were examined in a sample of Danish adults bereaved of a parent or spouse (Komischke-Konnerup et al., 2023). The findings revealed that early grief symptoms predicted other post-loss psychopathology symptoms and not vice versa whereas at later waves the relationships between different types of psychopathology symptoms became more intertwined.

Taken together, two themes emerge that require further investigation. Firstly, the temporal relationships appear to differ in traumatized and non-traumatized samples. In the traumatized sample, within-person fluctuations of PTS symptoms predicted within-person fluctuation in prolonged grief symptoms but not vice versa (Glad et al., 2022), while in the non-traumatized sample, within-person fluctuations in prolonged grief, depressive, and PTS symptoms were interrelated (Komischke-Konnerup et al., 2023) complementing the idea that after experiencing a violent loss, intense PTS symptoms can disrupt the grieving process, potentially leading to prolonged grief symptoms (Nakajima et al., 2012). Secondly, the time passed since the loss occurred appears to matter since acute grief reactions shortly after the loss were predictive of other post-loss symptomatology. This, in turn, aligns with the notion

that specific prolonged grief symptoms could serve as mechanisms explaining the exacerbation of other symptoms (Janshen & Eisma, 2024).

Consequently, the aim of the current study is to contribute to the body of literature by investigating the temporal relationship between prolonged grief and symptoms of depression and PTS using RI-CLPM. More specifically, we will investigate how within-person fluctuations in prolonged grief symptoms relate to within-person fluctuations in depressive and PTS symptoms over time in a sample that experienced predominantly non-violent losses using five-wave longitudinal survey data. By using RI-CLPM, it is possible to separate within-person from between-person effects. This approach provides unique insights that could inform treatment strategies for individuals experiencing severe distress after a loss. For instance, if within-person fluctuations in acute grief shortly after a loss predict fluctuations in other post-loss symptoms, it might be beneficial to address grief symptoms early in treatment. In line with prior results (Janshen & Eisma, 2024), we hypothesize that higher prolonged grief symptoms than usual will more consistently predict higher symptoms of depression and PTS than usual than vice versa.

Method

Procedure and Participants

The present data was collected as part of the Utrecht Longitudinal Study on Adjustment to Loss, a research project investigating the cognitive and behavioral processes associated with emotional challenges following bereavement. Participants were recruited through announcements posted on various internet platforms and websites dedicated to grief and bereavement care. These announcements, disseminated by nonprofit bereavement care organizations and funeral companies, outlined the project objectives and invited adults aged 18 and older who were recently bereaved of a relative or friend to participate. Upon expressing interest through an online application form, participants received a personalized

login code and access to a secure website with detailed study information. After providing online informed consent, participants completed the baseline questionnaire. Those who had experienced a loss within the past year were asked if they would be willing to participate in additional questionnaires. Participants who agreed to this were sent automated e-mails containing links to online follow-up questionnaires, repeated 11 times at 6-week intervals. The follow-up questionnaires were accessible for seven days after the email invitation to maintain consistent intervals between completions. No additional reminders were sent, and participants did not receive monetary incentives for questionnaire completion.

Recruitment spanned from early 2012 to late 2020, during which 2,104 individuals completed application forms. Of these, 1,170 (56%) completed the baseline questionnaires. Individuals who experienced a loss in the previous year were invited to participate in the follow-up questionnaires ($N = 426$). Of the 458 participants who agreed to participate in the longitudinal study, the majority of participants did not consistently complete the surveys across the study waves. There was a substantial number of individuals who filled out only the baseline assessment ($n = 151$). Due to the larger numbers of missing values in later waves, we decided a priori to base our analysis on the first five waves and exclude participants who filled out only the baseline measurement. This reduced missingness patterns substantially and enabled a more effective way of handling missing data in the main analysis. This resulted in a final sample size of $N = 307$. Of these 307 participants, most participants showed intermittent participation, with instances of completing surveys in certain waves followed by periods of absence and then returning to complete surveys again for subsequent waves. In total, 123 (40 %) participants filled out all five measurement waves. The sample size across waves ranged from 208 (70%) to 307 (100 %). The majority of participants were female, middle-aged adults (78% female; mean age: 54.85 years ($SD = 13.62$) who experienced a loss on average

4.43 (*SD* = 2.74) months ago at baseline. The majority experienced a non-violent loss (88%) of a partner (49%). See Table 1 for all sample characteristics.

Table 1

Sample Characteristics (N = 307)

Gender, *n* (%)	
Female	237 (78)
Age in years, *M* (*SD*)	40.84 (17.37)
Level of education[a], *n* (%)	
College or university	173 (56)
Other than college or university	134 (44)
The deceased was, *n* (%)	
Partner	151 (49)
Child	27 (9)
Sibling	17 (5)
Parent	92 (30)
Other[b]	20 (7)
Time since the loss in months, *M* (*SD*)	30.65 (30.71)
Cause of death[c], *n* (%)	
Nonviolent loss	269 (88)
Violent loss	38 (12)
The loss was, *n* (%)	
Expected	160 (52)
Unexpected	112 (36)
Both or neither	35 (12)

Note. [a] Higher education = college and university, other = primary school, high school, and household school, vocational education, or participant who did not specify their education level; [b] Other = other second or third-degree relatives (65%), multiple losses (5%), friends (30%); [c] Nonviolent loss = natural deaths, Violent loss = accident, murder and suicides.

Measures

The questionnaires mentioned below were selected from a larger battery of questionnaires. Below, we describe the questionnaires relevant to our research aims.

Sociodemographic and Loss-Related Characteristics

To asses sociodemographic characteristics (i.e., age, gender, and level of education) and loss-related characteristics (i.e., relationship to the deceased, time since the loss in months, cause of death, and expectedness of the loss) a costum-designed questionnaire was administered.

Prolonged Grief Symptoms

Prolonged grief symptoms were assessed with 11 items of the Prolonged Grief Disorder Scale (PGD Scale; Boelen, 2012). This scale covers prolonged grief symptoms in line with the diagnostic proposal of PGD by Prigerson et al. (2009). Participants rated the frequency of symptom occurrence over the past month on a 5-point scale, ranging from 1 (*never*) to 5 (*always*). An unweighted sum score representing grief severity was calculated. The reliability of the PGD Scale was excellent (α =.92) at baseline (range across all study waves: α = .92 – .95).

Depressive Symptoms

Depressive symptoms were measured using the 7-item depression subscale of the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983; Dutch version: Spinhoven et al., 1997). Participants rated the extent of their experienced symptoms in the past week on a 4-point scale ranging from 1 to 4. An example item is “I feel as if I am slowed down”. The unweighted sum score of the depression subscale was calculated. The reliability was excellent (α =. 92) at baseline (range across all study waves: α = .88 - .93).

Post-Traumatic Stress Symptoms

Posttraumatic stress symptoms were measured using the PTSD Symptom Scale Self-Report version (PSS-SR; Foa et al., 1997; Dutch version: Engelhard et al., 2007), a 17-item assessment aligned with criteria from the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). Participants reported the frequency of experiencing symptoms during the preceding month on a 4-point scale, ranging from 1 (*not at all*) to 4

(*five/more times per week/almost always*). An example item of the PSS-SR is "How often did you have unpleasant dreams or nightmares about the death of your loved one?". The unweighted sum score of all items was calculated. The internal consistency of the PSS-SR was good ($\alpha = .89$) at baseline (range across all study waves: $\alpha = .88$ - $.90$).

Statistical Analysis

All analyses were performed using R software (R Core Team, 2021). First, the missing data was inspected. We investigated if there were differences between completers of the first five waves and non-completers (i.e. those who did not fill in one or more waves). They were compared on the main variables (i.e., prolonged grief, depressive, and PTS symptoms) and background characteristics (i.e. gender, age, level of education, cause of death, relationship to the deceased, time since the loss, cause of death, expectedness of the loss). Bivariate relationships between the main variables were also inspected as well as the univariate distributions and standard estimators of location and spread of the main variables, for exploratory purposes. Next, we calculated the intra-class correlation (ICC) to inspect the amount of variability between and within clusters.

After data cleaning and preliminary analyses, we utilized RI-CLPMs to analyze our data. We built three RI-CLPMs: one including PG symptoms and depressive symptoms, one including PG symptoms and PTS symptoms, and one including all three symptom types. The analysis was carried out using the "lavaan" package. The models were estimated using full information maximum likelihood estimation (i.e., FIML) to account for missing data, and model fit was evaluated following Kline's minimum set of reported fit statistics (i.e., $\chi 2$, RMSEA with a 90% CI, CFI, SRMR; Kline, 2016). The size of the standardized beta coefficients was interpreted using Cohen's guidelines for effect sizes of correlations (small effect: $\beta = .10$ - $.29$, moderate effect: $\beta = .30$ - $.49$, large effect: $\beta \geq 0.50$; cf. Cohen, 1988).

The model specification aligned with the specification of Hamaker (2015). Two latent variables representing the random intercepts were created. Both intercepts were correlated because they are exogenous variables. A constant was included in the model to add the mean structure. The observed variables prolonged grief symptoms, depressive, and PTS symptoms at T1 – T5 represented the total scores of the symptom measures per wave. Scores of all waves were regressed on the random intercepts while constraining the factor loadings to one allowing the intercept to vary across individuals. The observed total scores of the symptom measures served as single indicators for the latent constructs of the symptoms (i.e., the path was fixed to one and the measurement error was fixed to zero). Next, the auto-regressive and cross-lagged effects were freely estimated. The disturbance terms of the latent variables were correlated because it is plausible to assume that unknown causes of these variables are related. The model specification of the last model was extended by one variable.

Next, we employed a model-trimming strategy to test whether constraining autoregressive, cross-lagged paths, (residual) (co-)variances in the within-person part, and grand means over time to be equal across waves would improve the model fit using chi-square difference tests. In case constraining the effects to be equal improves model fit, this would imply that the underlying relationships at the within-person level observed in the data are consistent and stable over time. The unconstrained RICLPM model served as the base model in which the model with constraints was nested.

Lastly, we conducted sensitivity analyses to inspect the effect of influential points on our findings. The data was checked for implausible values by visual inspection. Furthermore, we checked for multivariate outliers using Mahalanobis distance (critical values: $\alpha = .001$, $\chi^2(9) = 27.887$, $\chi^2(7) = 36.12$) and residual vs. leverage plots. Cases that were flagged as influential points in these plots were excluded from the sensitivity analyses. In case the findings from the sensitivity analyses diverged from the findings of the original analyses (i.e.,

changes that allow for interpretations different from the original analysis), the findings of the sensitivity analyses were reported.

Results

Data Inspection

Descriptive Analyses

Generally, the symptom levels appeared to decrease over time. Zero-order correlations between different types of psychopathology symptoms were inspected to examine the bivariate relationships. There were strong positive associations between the different types of symptomatology (range: r = .63 – .85; see Appendix A). This implies that prolonged grief, depressive, and PTS symptoms are concurrently and longitudinally positively related. Individuals with intermittent participation (non-completers; n = 184) were younger than individuals who consistently filled out all five waves (completers; n = 123; $t(281.12) = -2.67$, p = .008, 95% CI [-7.12, -1.07]). Completers and non-completers did not differ significantly in prolonged grief, depressive, and PTS symptom levels and other recorded background variables. The ICCs were high for prolonged grief, depressive, and PTS symptoms, respectively (ICC = .79, .80, .78). This indicates that a significant portion of the variance observed in our data stems from differences between individuals, suggesting the presence of stable trait-like characteristics. However, there remains a notable amount of variability within individuals across different waves. Given these findings, we proceeded with the main analyses.

Model Diagnostics

To ensure that the assumption of multivariate normality holds for our main analysis, we used full information maximum likelihood estimation (FIML) which is a model-based approach for data with missing values that enables us to use all the available information (Kline, 2016) and therefore, enables us to use data from the full sample.

Next, the multicollinearity between predictors was inspected using the VIF. There was no indication of high multicollinearity between predictors exceeding the traditional threshold of 10 (range: VIF = 2.29 – 4.13). However, given the large correlations between predictors, full information likelihood estimation (FIML) was utilized which has been demonstrated to be an effective estimator of data with multicollinearity (Agunbiade & Iyaniwura, 2010).

No unusual observations were identified via visual inspection of the data. Using Mahalanobis distance (critical values: $\alpha = .001$, $\chi^2(9) = 27.887$, $\chi^2(7) = 36.12$) we did not identify any influential points. In total, five influential points were identified using residual vs. leverage plots. Thus, sensitivity analyses were conducted. The exclusion of the influential points led to significant changes in the findings in the second and third RI-CLPM (i.e., changes that warrant a different interpretation compared to the original analysis). Therefore, the findings of the sensitivity analyses are reported ($n = 302$; analysis including outliers in Appendix B).

Main Analysis

Longitudinal Associations of Prolonged Grief and Depressive Symptoms

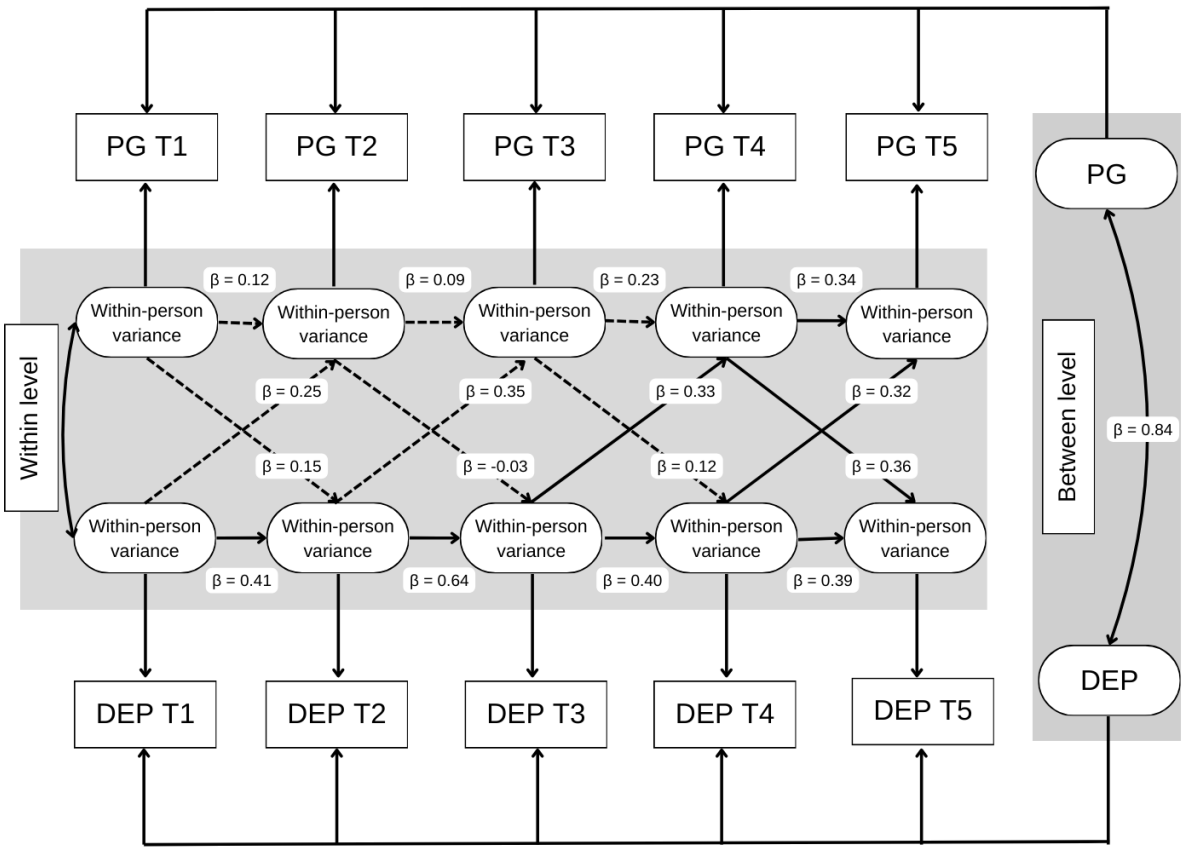
The first RI-CLPM assessed the longitudinal associations between prolonged grief and depressive symptoms (see Figure 1). The model with constrained autoregressive, cross-lagged effects, time-invariant (residual) (co-)variances in the within-person part, and constrained grand means over time fitted significantly worse compared to the model without constraints ($\chi 2_{dif} = 151.84$, $df_{dif.} = 29$, $p < .001$), suggesting that the constraints imposed in the model likely oversimplify the data structure and fail to capture important variations that the unconstrained model can accommodate. The model fit of the unconstrained model was good ($\chi 2 = 25.55$, df = 21, $p = .224$; RMSEA = 0.027, CI [0.000; 0.058]; CFI = 0.999; SRMR = 0.022).

On the between-person level, there was a significant relationship between the random intercepts of prolonged grief and depressive symptoms (β = .84, $p < .001$; large effect), suggesting that, on average, individuals with higher trait-like prolonged grief symptoms also experienced higher trait-like depressive symptoms.

On the within-person level, there was an autoregressive effect of prolonged grief symptoms from T4 to T5 (β = .34, $p = .003$; moderate effect), suggesting that higher prolonged grief symptoms than usual at T4 were related to higher prolonged grief symptoms at T5. Furthermore, there were significant autoregressive effects of depressive symptoms from T1 to T2 (β = 0.41, $p < .001$; moderate effect), T2 to T3 (β = 0.64, $p < .001$; large effect), T3 to T4 (β = 0.40, $p = .002$; moderate effect), and T4 to T5 (β = 0.39, $p = .002$; moderate effect). This suggests that higher depressive symptoms than usual at earlier time points were consistently linked to higher depressive symptoms than usual at the next time point. Furthermore, significant cross-lagged effect effects were found. Higher depressive symptoms than usual at T3 predicted higher prolonged grief symptoms than usual at T4 (β = 0.33, $p = .003$; moderate effect) but not vice versa. Furthermore, higher prolonged grief symptoms than usual at T4 predicted higher depressive symptoms than usual at T5 (β = 0.36, $p = .001$; moderate effect) and vice versa (β = 0.32, $p = .007$; moderate effect). No other cross-lagged effects emerged. Thus, prolonged grief and depressive symptoms became increasingly intertwined over time. Lastly, we found significant positive associations between the disturbance terms of the prolonged grief and depressive symptoms at all waves (all p's ≤ .009) indicating that unknown unmeasured causes of prolonged grief and depressive symptoms are linked.

Figure 1

RI-CLPM Including Prolonged Grief and Depressive Symptoms



Note. For clarity the mean structure and the disturbance terms in the within level are not depicted.

Longitudinal Associations of Prolonged Grief and Post-Traumatic Stress Symptoms

The second RI-CLPM assessed the longitudinal associations between prolonged grief and PTS symptoms (see Figure 2). The model with constrained autoregressive, cross-lagged effects, time-invariant (residual) (co-)variances in the within-person part, and constrained grand means over time fitted significantly worse compared to the model without constraints ($\chi 2_{dif} = 216.88$, $df_{dif.} = 29$, $p < .001$), suggesting that the constraints imposed in the constrained model likely fail to capture important variations that the unconstrained model can account for. The model fit of the unconstrained model was good ($\chi 2 = 34.678$, df = 21, $p = .031$; RMSEA = 0.046, CI [0.014; 0.073]; CFI = 0.996; SRMR = 0.037).

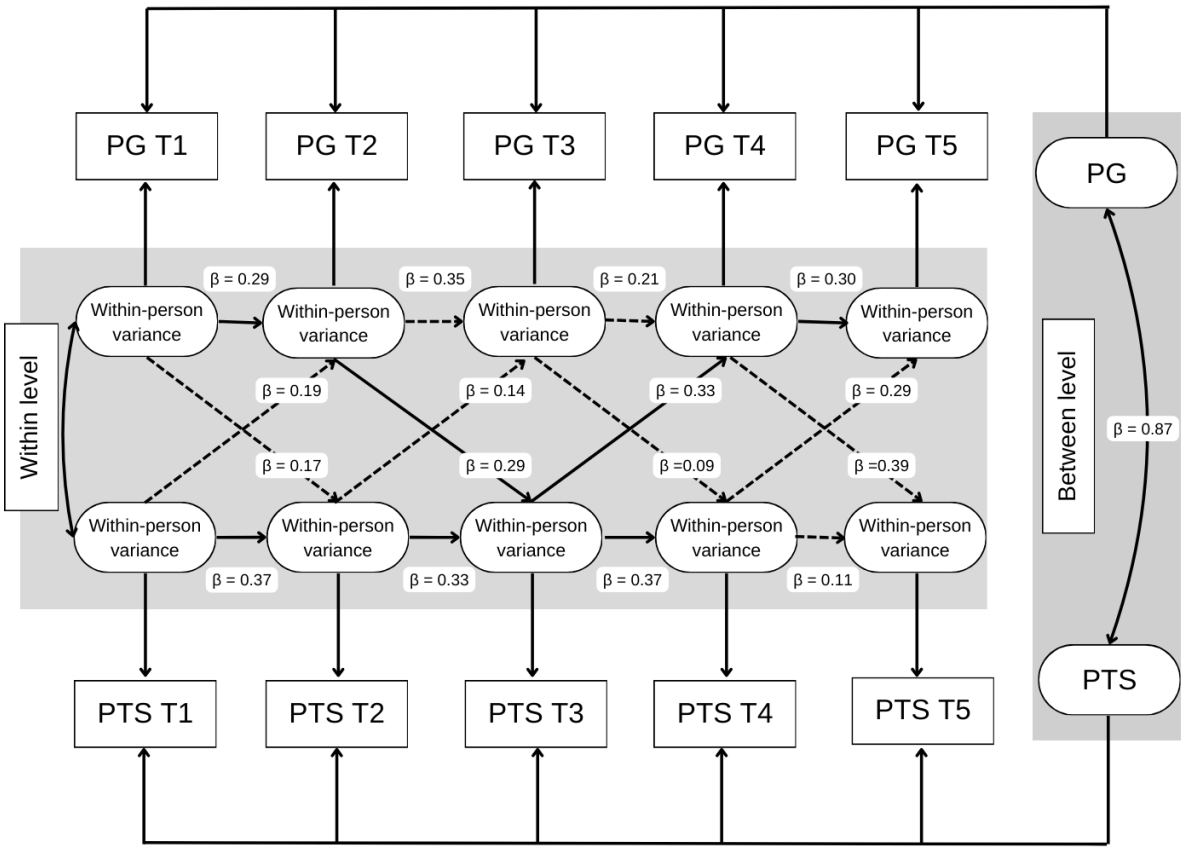
On the between-person level, there was a significant relationship between the random intercepts of prolonged grief and PTS symptoms (β = .87, $p < .001$; large effect), suggesting

that, on average, individuals with higher trait-like prolonged grief symptoms also experienced higher trait-like PTS symptoms.

On the within-person level, there were significant autoregressive effects of prolonged grief symptoms from T1 to T2 (β = .29, $p < .038$; small effect) and T4 to T5 (β = .30, $p < .040$; moderate effect), which suggests that higher prolonged grief symptoms than usual at T1 and T4 were linked to higher PTS symptoms than usual at T2 and T5, respectively. Furthermore, there were significant autoregressive effects of PTS symptoms from T1 to T2 (β = .37, $p < .001$; moderate effect), T2 to T3 (β = .33, $p = .016$; moderate effect), and T3 to T4 (β = .37, $p = .022$; moderate effect), which suggests that higher PTS symptoms than usual at earlier time points were linked to higher PTS symptoms than usual at the next time point. Furthermore, significant cross-lagged effects were found. Higher prolonged grief symptoms than usual at T2 predicted higher PTS symptoms than usual at T3 (β = .29, $p = .032$; small effect). Higher PTS symptoms than usual at T3 predicted higher prolonged grief symptoms than usual at T4 (β = .33, $p = .022$; moderate effect) but not vice versa. Thus, prolonged grief and PTS symptoms were also bidirectionally related, yet not consistently so. Lastly, we found significant positive associations between the disturbance terms of the prolonged grief and depressive symptoms at all waves (all p's $\leq .002$) implying that the unknown unmeasured causes of prolonged grief and PTS symptoms are related.

Figure 2

RI-CLPM Including Prolonged Grief and PTS Symptoms



Note. For clarity the mean structure and the disturbance terms in the within level are not depicted.

Longitudinal Associations of Prolonged Grief, Depressive, and Post-Traumatic Stress Symptoms

The third RI-CLPM inspected the longitudinal associations between prolonged grief, depressive, and PTS symptoms simultaneously (see Figure 3). The model with constrained autoregressive, cross-lagged effects, time-invariant (residual) (co-)variances in the within-person part, and constrained grand means over time, fitted significantly worse compared to the model without constraints ($\chi 2_{dif} = 262.77$, $df_{dif.} = 57$, $p < .001$), suggesting that the constrained effects are not stable but vary over time. The model fit of the unconstrained model was good ($\chi 2 = 61.256$, df = 48, $p = .095$; RMSEA = 0.030, CI [0.000; 0.051]; CFI =0.997; SRMR = 0.036).

On the between-person level, there was a significant relationship between the random intercepts of prolonged grief and depressive symptoms ($\beta = .54$, $p < .001$; large effect), prolonged grief, and PTS symptoms ($\beta = .88$, $p < .001$; large effect), and depressive and PTS symptoms ($\beta = .82$, $p < .001$; large effect), suggesting that, on average, individuals with higher trait-like prolonged grief symptoms also experienced higher trait-like depressive and PTS symptoms. Moreover, individuals with higher trait-like depressive symptoms, on average, also experienced higher trait-like PTS symptoms.

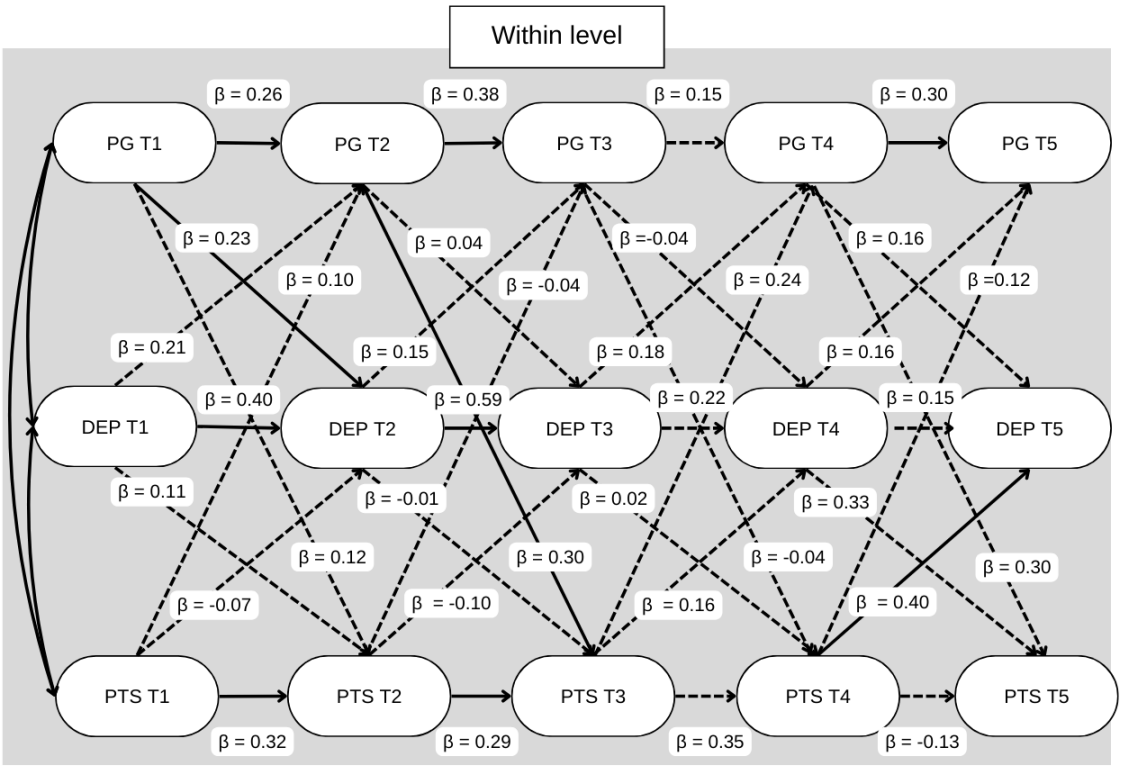
On the within-person level, there were significant autoregressive effects of prolonged grief symptoms from T1 to T2 ($\beta = .26$, $p = .043$; small effect), T2 to T3 ($\beta = .37$, $p = .049$; moderate effect), and T4 to T5 ($\beta = .30$, $p = .048$; moderate effect), which suggests that higher prolonged grief symptoms than usual at earlier time points were linked to higher prolonged grief symptoms than usual at the next time point. Furthermore, there were significant autoregressive effects of depressive symptoms from T1 to T2 ($\beta = .40$, $p < .001$; moderate effect), and T2 to T3 ($\beta = .59$, $p < .001$; large effect), which suggests that higher depressive symptoms than usual at T1 and T2 predicted higher depressive symptoms than usual at T2 and T3, respectively. Moreover, there were significant autoregressive effects of PTS symptoms from T1 to T2 ($\beta = .32$, $p = .001$; moderate effect), and T2 to T3 ($\beta = .29$, $p = .043$; small effect) suggesting that higher PTS symptoms than usual at T1 and T2 predict higher PTS symptoms at T2 and T3, respectively.

Furthermore, significant cross-lagged effects were found. Higher prolonged grief symptoms than usual at T1 predicted higher depressive symptoms than usual at T2 ($\beta = .23$, $p = .046$; small effect) and higher prolonged grief symptoms than usual at T2 predicted higher PTS symptoms at T3 ($\beta = .30$, $p = .034$; moderate effect). Thus, there were directional effects in which higher prolonged grief symptoms than usual were related to higher depressive and PTS symptoms than usual in later waves. Fluctuations in depressive and PTS symptoms were

not significantly related in this model. Lastly, we found significant positive associations between the disturbance terms of the prolonged grief, depressive, and PTS symptoms at all waves (all *p's* ≤ .003) except between disturbance terms of prolonged grief and depressive symptoms at wave three ($p = .111$) implying that in most instances the unknown unmeasured causes of prolonged grief, depressive and PTS symptoms are related.

Figure 3

RI-CLPM Including Prolonged Grief, Depressive, and PTS Symptoms



Note. For clarity the mean structure, the between level, single indicators of the latent variables, and the disturbance terms in the within level are not depicted.

Discussion

The current study aimed to investigate the temporal relationships of prolonged grief symptoms with symptoms of depression and PTS using RI-CLPM. This approach enables the distinction of trait-like (between-person) effects from state-like (within-person) effects. Thus, one can infer on the within-person level whether a higher score than usual in one variable,

predicts a higher score than usual in another variable at the next time point. Our findings demonstrated that: (a) within-person fluctuations in prolonged grief and depressive symptoms were bi-directionally related, (b) within-person fluctuations in prolonged grief and PTS symptoms were bi-directionally related, and (c) in a complex model including prolonged grief, depressive, and PTS symptoms within-person changes in prolonged grief symptoms predicted within-person changes in depressive and PTS symptoms but not vice versa. Between-person differences explained more variance than within-person differences. No effects were consistent across all time points.

The first model assessed the longitudinal relationships of within-person fluctuations in prolonged grief and depressive symptoms. Higher depressive symptoms than usual at T3 (*M* time since loss = 7.43 months) predicted higher prolonged grief symptoms than usual at T4 (*M* time since loss = 8.93 months) and higher prolonged grief symptoms than usual at T4 predicted higher depressive symptoms than usual at T5 (*M* Time since the loss = 10.43 months) and vice versa. There are no previous studies solely examining the reciprocal relationships between within-person fluctuations of prolonged grief symptoms and depressive symptoms using RI-CLPM. These findings appear to demonstrate that over time effects of prolonged grief and depressive symptoms become increasingly intertwined implying that these symptoms are closely related and likely share core mechanisms that mutually influence the emergence of these different symptoms (Komischke-Konnerup et al., 2023).

The second model assessed the longitudinal relationship of within-person fluctuations in prolonged grief and PTS symptoms. Our findings demonstrated that higher prolonged grief symptoms than usual at T2 (*M* Time since the loss = 5.93) predicted higher PTS symptoms than usual at T3 (*M* Time since the loss = 7.43), but not vice versa. Furthermore, higher PTS symptoms than usual at T3 (*M* Time since the loss = 7.43) predicted higher prolonged grief symptoms than usual at T4 (*M* Time since the loss = 8.93) but not vice versa. Therefore,

within-person fluctuations in prolonged grief and PTS symptoms appear intertwined aligning with the notion that these symptom patterns are interrelated and probably share fundamental mechanisms that promote the development of each type of symptom (Komischke-Konnerup et al., 2023). One previous study examined the reciprocal relationships between within-person fluctuations of prolonged grief symptoms and PTS symptoms (Glad et al., 2022). In a three-wave study, they found that higher PTS symptoms than usual at 14-15 months post-loss predicted higher prolonged grief symptoms at 30-32 months post-loss (Glad et al., 2022), but no effects in the opposite direction. While they only reported an effect from PTS symptoms on prolonged grief symptoms, we also found an effect in the reverse direction. The findings may differ due to multiple reasons, including diverging timeframes (i.e., this study: five time-points ranging from an average of 4.43 to 10.34 months post-loss; Glad et al. (2022): three time-points ranging from 4-5 to 30-32 months post-loss), and diverging sample characteristics (this study: predominantly non-violent losses, Glad et al. (2022): traumatic losses). The latter appears particularly important, as severe posttraumatic stress has been theorized to interfere with the processing of the death of a loved one (Nakajima et al., 2012).

The final model examined the longitudinal relationship of within-person fluctuations in prolonged grief, depressive, and PTS symptoms. The findings suggest that higher prolonged grief symptoms than usual at T1 (*M* Time since the loss = 4.43) predicted higher depressive symptoms than usual at T2 (*M* Time since the loss = 5.93) and higher prolonged grief symptoms than usual at T2 predicted higher PTS symptoms at T3 (*M* Time since the loss = 7.43). The findings partially align with results from a previous study (Komischke-Konnerup et al., 2023). In both studies, no consistent within-person effects were identified. Furthermore, in line with the previous study, we also found that acute grief levels close after the loss of a loved one were a significant predictor of within-person fluctuations of other post-loss symptomatology. However, in contrast to Komischke-Konnerup et al. (2023), we

did not find cross-lagged effects from depressive and PTS symptoms to prolonged grief symptoms at any of the time points. Thus, they found that prolonged grief affects other symptom types and all become increasingly intertwined over time while we observe that acute prolonged grief levels are a unique determinant of depressive and PTS symptoms, but not the other way around. This aligns with Janshen and Eisma (2024) who concluded that existing cross-lagged panel model studies indicate that prolonged grief symptoms appear a risk factor for the development of depressive and post-traumatic stress symptoms.

In the simpler models (i.e., including two variables) within-person changes in prolonged grief symptoms were also predicted by within-person changes in depressive and PTS symptoms. Thus, the effects appeared to be more intertwined which complement the results of latent class analysis studies that found a mixed class of prolonged grief, depressive, and PTS symptoms (Boelen et al., 2016; Djelantik et al., 2017; Kokou-Kpolou et al., 2021; Lenferink et al., 2017; for a review see: Heeke et al., 2023). The intertwined nature of post-loss symptoms also aligns with the notion of fundamental mechanisms that mutually affect the development of each symptom type (Komischke-Konnerup et al., 2023). For example, avoidance strategies and maladaptive cognitions, which are considered core mechanisms in prolonged grief, depression, and post-traumatic stress (Beck, 1987; Boelen et al., 2006; Ehlers & Clark, 2000), are associated with prolonged grief, depressive, and PTS symptoms in bereaved individuals (e.g., Boelen, 2021; Treml et al., 2021). However, when including all three variables in a model, the existing relationships from within-person fluctuations in depressive and PTS symptoms to prolonged grief symptoms were non-significant, suggesting that the inclusion of depressive or PTS symptoms accounted for variance that was originally explained by the other variable. This indicates that the variance in prolonged grief symptoms initially explained by either depressive or PTS symptoms alone was shared between the two, reducing their unique contributions when combined in the model. It appears, however, that

the variance explained by within-person fluctuations in prolonged grief symptoms is not strongly affected by the inclusion of another variable implying that within-person fluctuations in prolonged grief symptoms explained variance that is not shared with depressive and PTS symptoms. This aligns with the theoretical notion that prolonged grief symptoms may contribute to the emergence of other post-loss pathologies, as specific prolonged grief symptoms (e.g., loss-related avoidance, inactivity) could serve as mechanisms explaining the emergence of symptoms of other post-loss symptomatology (Janshen & Eisma, 2024).

Notably, we found a strong relationship between stable between-person differences in prolonged grief, depressive, and PTS symptoms. This indicates that participants who reported higher prolonged grief symptoms across waves also reported higher depressive and PTS symptomatology across waves. This complements findings from previous studies using RI-CLPM (Glad et al., 2022; Komischke-Konnerup et al., 2023) and research that demonstrated high correlations and cooccurrence between these symptoms (e.g., Djelantik et al., 2020; Eisma et al., 2019; Komischke-Konnerup et al., 2021, 2023; Lenferink et al., 2017). It also aligns with research adopting a network approach to prolonged grief (Komischke-Konnerup et al., 2023; for an overview see: Robinaugh et al., 2022). This perspective suggests that the development and maintenance of psychopathology are influenced by causal interactions between symptoms. Rather than stemming from a single cause, psychopathology is theorized to emerge from a complex network of symptoms that can perpetuate and exacerbate each other (Borsboom, 2017). It appears that while fluctuations in prolonged grief symptoms do occasionally predict other post-loss symptomatology, high stable levels of prolonged grief symptoms often co-occur with severe high depressive and PTS symptomatology.

These findings align with the allostatic load theory in stress research that posits that chronic exposure to stress rather than occasional spikes in stress and the body's frequent adjustment to these stressors (allostasis) can lead to wear and tear on the body and mind,

known as allostatic overload. Over time, this can contribute to various health problems including mental disorders (McEwen, 1993; for a review see: Guidi et al., 2020). Thus, constantly experiencing high levels of prolonged grief symptoms could be more harmful than occasionally experiencing higher prolonged grief symptoms than usual. However, within-person fluctuations in post-loss symptoms did predict within-person fluctuations in other post-loss symptoms on some occasions and should therefore not be neglected. Higher symptom levels than usual can be triggered by a variety of causes (e.g., certain life events, anniversary of death, holidays, family events, stress) and are likely highly idiosyncratic.

Our findings have implications for clinical practice. Specifically, considering that high acute grief appears to be a determinant of other symptomatologies as demonstrated relatively consistently (Janshen & Eisma, in press; Komischke-Konnerup et al., 2023), acute high grief after the loss can addressed effectively in treatment to prevent the emergence of other post-loss symptomatology (e.g., Litz et al., 2014; Reitsma et al., 2023). Second, in light of the findings that stable high levels of all types of symptomatology (i.e., traumatic grief; Rando, 2013) are strongly related, our findings emphasize the importance of addressing shared mechanisms (e.g., maladaptive cognitions, and maladaptive avoidance strategies) in treatment (i.e., transdiagnostic treatment; e.g., Komischke-Konnerup et al., 2023).

Strengths of this study include a sample of recently bereaved individuals, the use of five measurement points, and RI-CLPM to inspect temporal relationships between within-person fluctuations in prolonged grief, depressive, and PTS symptoms. Furthermore, in contrast to previous studies (Glad et al., 2022; Komischke-Konnerup et al., 2023), models were run including two variables and also three variables to gain deeper insight into the interrelations of these variables.

Despite these strengths, some limitations warrant mention. First, the majority of our sample was female and highly educated which is a common issue in bereavement research

(Eisma & Stroebe, 2021). Second, the measurement instruments were outdated and did not represent the current diagnostic criteria for prolonged grief and participants were assessed using self-report questionnaires instead of clinical interviews (Eisma, 2023). Thus, we can only inspect and draw conclusions about symptom levels and not make any statements regarding the diagnostic status of our participants (Stroebe et al., 2024). Third, from a statistical perspective, individuals' time since the loss at the baseline assessment varied. This unmatched time-axis likely increased variance at the within-level which leads to increased standard errors, lower power, and more uncertainty. Fourth, the relatively large amount of missing data, and a relatively modest sample size could potentially lead to imprecise estimates and more uncertainty. Furthermore, we did not include measurement error in this analysis (i.e., we assumed perfect measurement). While this is commonly the case using traditional CLPM and RI-CLPM, this can lead to biased estimates (Mulder & Hamaker, 2020). Lastly, we have no qualitative information about the life circumstances of the participants that could help us understand what triggered higher symptoms than usual that were predictive of within-person fluctuations at later time points. This could aid in understanding the inconsistent patterns at the within-person level that have been found in this and previous studies.

Future research should aim to replicate the analyses of the current study using clinical samples with a larger proportion of men and lower-educated individuals. Furthermore, future research should aim to utilize validated measurement instruments that align with the current conceptualization of prolonged grief in the ICD-11 and DSM-5-TR (e.g., Lenferink et al., 2022; O'Connor et al., 2023) and conduct clinical interviews to gain information about the diagnostic status of the participants (Stroebe et al., 2024). Next, the time since the loss should be aligned at baseline to reduce the variance due to a different time-axis and thereby standard errors. Future research should aim to optimize participant recruitment and employ retention

strategies to avoid participant drop-out thereby maximizing the sample size and reducing the amount of missing data. Moreover, future research should aim to model measurement error instead of assuming perfect measurement (e.g., by manually fixing the measurement error; see Bollen, 1989). Lastly, qualitative and single-subject studies are necessary to gain a deeper understanding of what causes within-person symptom changes.

Notwithstanding these limitations, this study provided unique insights into the reciprocal relationship between prolonged grief, depressive, and PTS symptoms. Higher prolonged grief symptoms than usual were predictive of other post-loss symptomatology in all three models and vice versa. In a model including prolonged grief, depressive, and PTS symptoms, within-person fluctuations in prolonged grief predicted other post-loss symptomatology but not vice versa. Most variance was explained by stable between-person differences. No consistent pattern emerged when inspecting the within-person fluctuations in prolonged grief, depressive, and PTS symptoms. Generally, relationships between within-person fluctuations in these variables appear intertwined. Future research should aim to replicate this study in a larger and more diverse sample with little missing data and assess prolonged grief symptoms using updated validated measurement instruments and clinical interviews. To inform clinical practice, future research should elucidate whether targeting acute grief reactions shortly after the loss might prevent the emergence of other post-loss symptoms.

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Appendix A

Table A1

Zero-order Correlations between Main Variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	*M* (*SD*)
(1) PG 1	-															38.25 (11.93)
(2) PG 2	0.86	-	.													37.23 (11.79)
(3) PG 3	0.84	0.89	-													35.46 (11.64)
(4) PG 4	0.79	0.85	0.89	-												33.64 (12.02)
(5) PG 5	0.77	0.81	0.87	0.89	-											32.52 (12.10)
(6) DEP 1	0.77	0.73	0.70	0.67	0.68	-										13.92 (5.18)
(7) DEP 2	0.74	0.78	0.73	0.71	0.69	0.88	-									14.09 (5.10)
(8) DEP 3	0.67	0.70	0.75	0.73	0.75	0.84	0.91	-								13.39 (5.01)
(9) DEP 4	0.67	0.68	0.72	0.80	0.78	0.79	0.82	0.87	-							12.86 (5.08)

(10) DEP 5	0.64	0.68	0.69	0.75	0.80	0.78	0.80	0.86	0.91	-						12.70 (4.88)
(11) PTS 1	0.80	0.72	0.71	0.67	0.71	0.73	0.68	0.65	0.63	0.65	-					34.41 (9.96)
(12) PTS 2	0.74	0.78	0.75	0.76	0.71	0.69	0.75	0.70	0.67	0.65	0.83	-				32.07 (9.38)
(13) PTS 3	0.73	0.77	0.83	0.79	0.79	0.70	0.72	0.77	0.71	0.70	0.80	0.86	-			30.90 (9.12)
(14) PTS 4	0.70	0.71	0.78	0.85	0.81	0.67	0.67	0.71	0.80	0.77	0.75	0.82	0.88	-		29.61 (9.03)
(15) PTS 5	0.70	0.67	0.76	0.78	0.84	0.66	0.66	0.76	0.77	0.79	0.76	0.76	0.85	0.89	-	29.20 (8.41)

Note. All correlations are significant ($p < .05$); PG = Prolonged grief symptoms; DEP = Depressive symptoms; PTS = Posttraumatic stress symptoms

Appendix B

Main Analysis Including Outliers

Longitudinal Associations of Prolonged Grief and Depressive Symptoms

The first RI-CLPM inspected the longitudinal associations between prolonged grief and depressive symptoms (see Figure B1). The model with constrained autoregressive, cross-lagged effects, time-invariant (residual) (co-)variances in the within-person part, and constrained grand means over time fitted significantly worse compared to the model without constraints ($\chi 2_{dif}$ =162.37, $df_{dif.}$ = 29, $p < .001$), suggesting that the constraints imposed in the constrained model likely oversimplify the data structure and fail to capture important variations that the unconstrained model can accommodate. The model fit of the unconstrained model was good ($\chi 2$ = 30.691, df = 21, p = .079; RMSEA = 0.039, CI [0.000; 0.067]; CFI = 0.997; SRMR = 0.023).

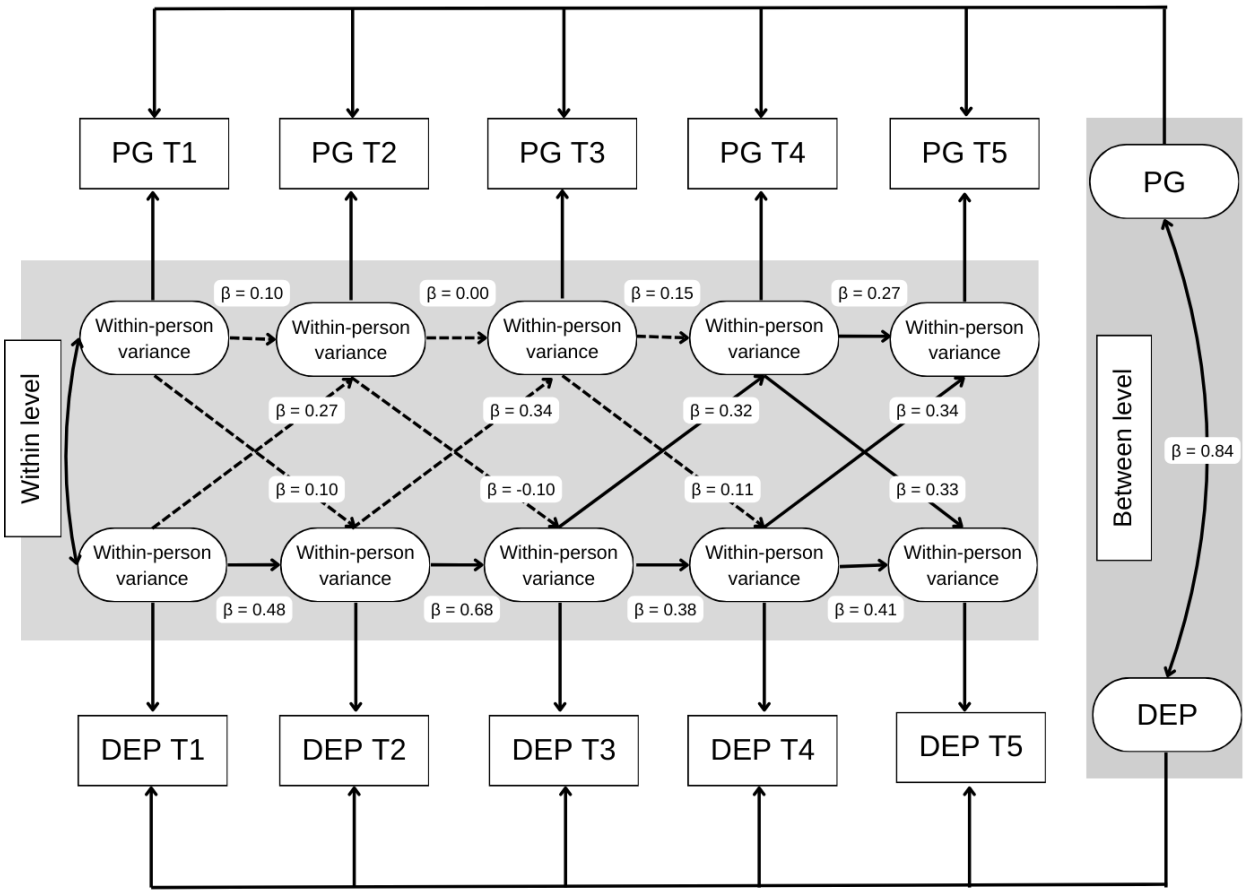
On the between-person level, there was a significant relationship between the random intercepts of prolonged grief and depressive symptoms (β = .84, $p < .001$; large effect), suggesting that, on average, individuals with higher trait-like prolonged grief symptoms also experienced higher trait-like depressive symptoms.

On the within-person level, there was an autoregressive effect of prolonged grief symptoms from T4 to T5 (β = .27, p = .038; small effect), suggesting that higher prolonged grief symptoms than usual at T1 were related to higher prolonged grief symptoms at T5. Furthermore, there were significant autoregressive effects of depressive symptoms from T1 to T2 (β = .48, $p < .001$; moderate effect), T2 to T3 (β = .68, $p < .001$; large effect), T3 to T4 (β = .38, p = .003; moderate effect), and T4 to T5 (β = .41, p = .001; moderate effect). This suggests that higher depressive symptoms than usual at earlier time points were consistently linked to higher depressive symptoms than usual at the next time point. Furthermore, significant cross-lagged effect effects were found. Higher prolonged grief symptoms than

usual at T4 predicted higher depressive symptoms than usual at T5 (β = 0.33, *p* = .003; moderate effect) and vice versa (β = 0.34, *p* = .007; moderate effects). Higher depressive symptoms than usual at T3 predicted higher prolonged grief symptoms than usual at T4 (β = 0.32, *p* = .009; moderate effect) but not vice versa. Thus, prolonged grief and depressive symptoms were bidirectionally related. Lastly, we found significant positive associations between the disturbance terms of the prolonged grief and depressive symptoms at all waves (all *p's* ≤ .002) except wave three (*p* = .002) indicating that the unknown unmeasured causes of prolonged grief and depressive symptoms are linked.

Figure B1

RI-CLPM Including Prolonged Grief and Depressive Symptoms



Note. For clarity the mean structure and the disturbance terms in the within level are not depicted.

Longitudinal Associations of Prolonged Grief and Post-Traumatic Stress Symptoms

The second RI-CLPM inspected the longitudinal associations between prolonged grief and PTS symptoms (see Figure B2). The model with constrained autoregressive, cross-lagged effects, time-invariant (residual) (co-)variances in the within-person part, and constrained grand means over time fitted significantly worse compared to the model without constraints ($\chi 2_{dif}$ =227.11, $df_{dif.}$ = 29, $p < .001$), suggesting that the constraints imposed in the constrained model likely fail to capture important variations that the unconstrained model can accommodate. The model fit of the unconstrained model was good ($\chi 2$ =39.82, df = 21, p = .008; RMSEA = 0.054, CI [0.027; 0.079]; CFI = 0.994; SRMR = 0.035).

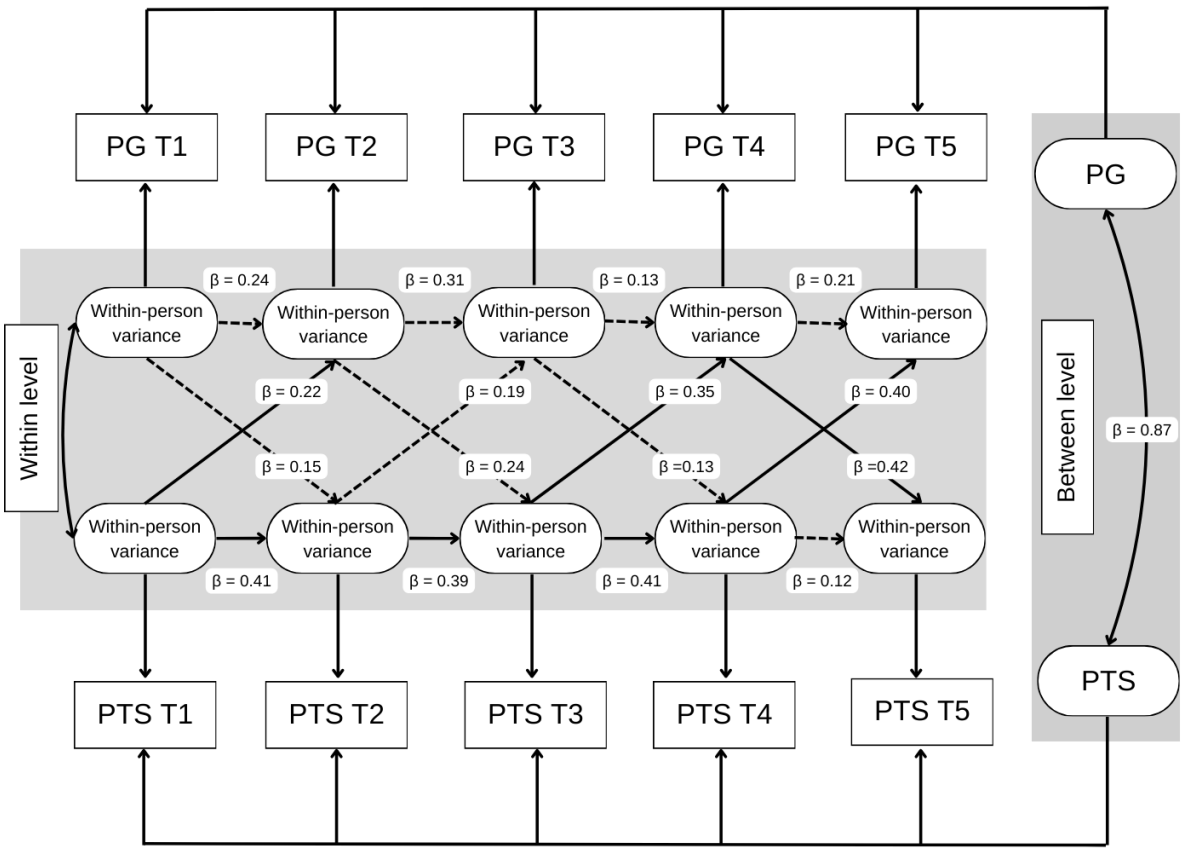
On the between-person level, there was a significant relationship between the random intercepts of prolonged grief and PTS symptoms (β = .87, $p < .001$; large effect), suggesting that, on average, individuals with higher trait-like prolonged grief symptoms also experienced higher trait-like PTS symptoms.

On the within-person level, there were significant autoregressive effects of PTS symptoms from waves T1 to T2 (β = .41, $p < .001$; moderate effect), T2 to T3 (β = .39, p = .002; moderate effect), and T3 to T4 (β = .41, p = .005; moderate effect), which suggests that higher PTS symptoms than usual at earlier time points were linked to higher PTS symptoms than usual at the next time point. Furthermore, significant cross-lagged effects were found. Higher prolonged grief symptoms than usual at T4 predicted higher PTS symptoms than usual at T5 (β = .42, p = .049; small effect). Higher PTS symptoms than usual at T1 predicted higher prolonged grief symptoms than usual at T2 (β = .22, p = .039; small effect). Higher PTS symptoms than usual at T3 predicted higher prolonged grief symptoms than usual at T4 (β = .35, p = .013; moderate effect) but not vice versa. Higher PTS symptoms than usual at T4 predicted higher prolonged grief symptoms than usual at T5 (β = .40, p = .017; moderate effect) but not vice versa. Thus, there were bidirectional effects in which

higher prolonged grief symptoms than usual were related to higher PTS symptoms than usual and vice versa. Lastly, we found significant positive associations between the disturbance terms of the prolonged grief and depressive symptoms at all waves (all *p's* ≤.001) implying that the unknown unmeasured causes of prolonged grief and PTS symptoms are related.

Figure B2

RI-CLPM Including Prolonged Grief and PTS Symptoms



Note. For clarity the mean structure and the disturbance terms in the within level are not depicted.

Longitudinal Associations of Prolonged Grief, Depressive, and Post-Traumatic Stress Symptoms

The third RI-CLPM inspected the longitudinal associations between prolonged grief, depressive, and PTS symptoms (see Figure B3). The model with constrained autoregressive, cross-lagged effects, time-invariant (residual) (co-)variances in the within-person part, and

constrained grand means over time, fitted significantly worse compared to the model without constraints ($\chi 2_{dif}$ =257.49, $df_{dif.}$ = 57, $p < .001$), suggesting that the constrained effects vary over time. The model fit of the unconstrained model was good ($\chi 2$ = 69.107, df = 48, p = .025; RMSEA = 0.038, CI [0.014; 0.057]; CFI =0.995; SRMR = 0.033).

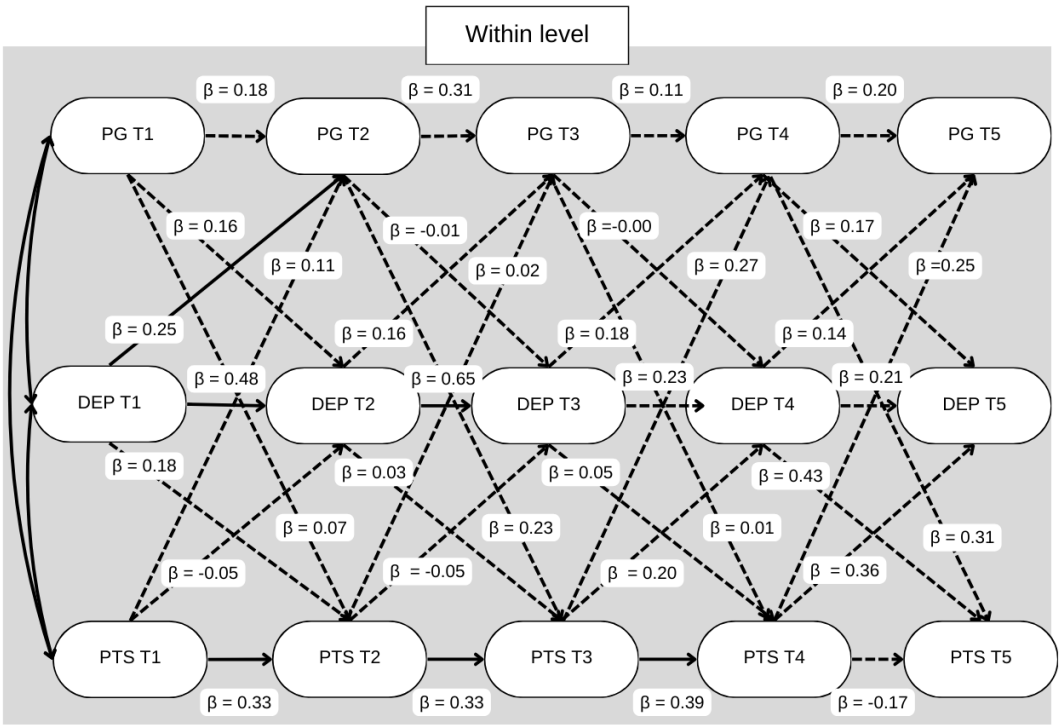
On the between-person level, there was a significant relationship between the random intercepts of prolonged grief and depressive symptoms (β = .84, $p < .001$; large effect), prolonged grief, and PTS symptoms (β = .87, $p < .001$; large effect), and depressive and PTS symptoms (β = .82, $p < .001$; large effect), suggesting that, on average, individuals with higher trait-like prolonged grief symptoms also experienced higher trait-like depressive and PTS symptoms. Moreover, individuals with higher trait-like depressive symptoms, on average, also experienced higher trait-like PTS symptoms.

On the within-person level, there were significant autoregressive effects of depressive symptoms from T1 to T2 (β = .48, $p < .001$; moderate effect), and T2 to T3 (β = .65, $p < .001$; large effect), which suggests that higher depressive symptoms than usual at earlier time points were linked to higher depressive symptoms than usual at the next time point. There were significant autoregressive effects of PTS symptoms from T1 to T2 (β = .33, $p = .001$; moderate effect), T2 to T3 (β = .33, $p = .022$; moderate effect), and T3 to T4 (β = .39, $p = .023$; moderate effect). Furthermore, a significant cross-lagged effect was found. Higher depressive symptoms than usual at T1 predicted higher prolonged grief symptoms than usual at T2 (β = .25, $p = .041$; small effect) but not vice versa. There were no significant autoregressive or cross-lagged effects from prolonged grief symptoms to the other variables and no significant cross-lagged effects from PTS symptoms to the other variables at any time point. Thus, there were directional effects in which higher depressive symptoms than usual were related to higher prolonged grief symptoms than usual at one wave but not vice versa. Fluctuations in prolonged grief and PTS symptoms were not related in this model. Lastly, we

found significant positive associations between the disturbance terms of the prolonged grief, depressive, and PTS symptoms at all waves (all *p's* ≤ .003) except between disturbance terms of prolonged grief and depressive symptoms at wave three ($p = .117$) implying that in most instances the unknown unmeasured causes of prolonged grief, depressive and PTS symptoms are related.

Figure B3

RI-CLPM Including Prolonged Grief, Depressive, and PTS Symptoms



Note. For clarity the mean structure, the between level, single indicators of the latent variables, and the disturbance terms in the within level are not depicted.