Journal of Anxiety Disorders 76 (2020) 102319

Contents lists available at ScienceDirect

Journal of Anxiety Disorders

journal homepage: www.elsevier.com/locate/janxdis

Test of the dynamic interplay between *DSM-5* PTSD symptom clusters in children and adolescents

Gen Li [a,b], Li Wang [a,b,*], Chengqi Cao [a,b], Ruojiao Fang [a,b], Chen Chen [a,b], Xue Qiao [a,b], Haibo Yang [c], David Forbes [d], Jon D. Elhai [e]

[a] Laboratory for Traumatic Stress Studies, CAS Key Laboratory of Mental Health, Institute of Psychology, Chinese Academy of Sciences, Beijing, China
[b] Department of Psychology, University of Chinese Academy of Sciences, Beijing, China
[c] Academy of Psychology and Behavior, Tianjin Normal University, Tianjin, China
[d] Department of Psychiatry, Phoenix Australia – Centre for Posttraumatic Mental Health, University of Melbourne, Carlton, Australia
[e] Department of Psychology, and Department of Psychiatry, University of Toledo, Toledo, USA

ARTICLE INFO

Keywords:
Posttraumatic stress symptom
Structural equation modeling
Longitudinal
DSM-5
Child

ABSTRACT

Background: Revealing the dynamic interplay between posttraumatic stress disorder (PTSD) symptom clusters has always been an important topic in traumatic stress studies. Based on longitudinal studies, different hypotheses have been proposed to explain PTSD symptom dynamics. But currently, no study have been conducted to test these hypotheses in children and adolescents.

Methods: Data were derived from a longitudinal study of child and adolescent traumatic event survivors of an explosion accident (N = 659). *DSM-5* PTSD symptoms was measured by the PTSD Checklist-5 (PCL-5) at 4, 8, and 13 months after the disaster. Latent difference score (LDS) modeling was used to evaluate the dynamic interplay between clusters.

Results: The results of LDS model indicated that intrusion level positively predicted subsequent rate of increase for hyperarousal ($p = .008$) and negative changes in cognitions and mood symptoms ($p = .036$). Also, intrusion level trended to positively predict subsequent increase rate of avoidance symptoms ($p = .059$).

Conclusions: This study expands previous knowledge of dynamic relations between symptom clusters during the maintenance and fluctuation of child and adolescent PTSD symptoms. By using new methodology, this study provided novel evidence for the hypothesis that intrusion symptom plays an important role in maintaining other PTSD symptoms.

1. Introduction

Posttraumatic stress disorder (PTSD) is a trauma and stress-related mental disorder with high clinical heterogeneity (Galatzer-Levy & Bryant, 2013). According to meta-analyses, the prevalence of PTSD in trauma-exposed children and adolescents ranged from 11 % to 20 % (Alisic et al., 2014). Studies on child and adolescent PTSD course found that PTSD prevalence and symptom severity showed moderate declines, particularly across the first 3 months after trauma exposure (Hiller et al., 2016). There was less evidence of further change after 6 months, which means children would not recover from PTSD symptoms beyond this point without intervention. As reviewed before, chronic trajectory of PTSD is common in disaster-exposed children and adolescents, ranging from 3.9%–38.0% (Lai et al., 2017). Identifying factors which maintains PTSD symptoms and prevent normal recovery from the acute phase after trauma exposure is necessary for the understanding and intervention of chronic PTSD in young population.

As described in *DSM-5* (American Psychiatric Association, 2013), PTSD contains 20 symptoms, mapping onto four distinct symptom clusters: intrusion (In), avoidance (Av), negative alterations in cognitions and mood (NACM), and alterations in arousal and reactivity (AAR). It has been well-recognized that these symptom clusters are not "created equal" (e.g., Schell, Marshall, & Jaycox, 2004): each symptom cluster has a distinct developmental course in the aftermath of trauma exposure. The dynamic relationships between these symptom clusters over time shape the longitudinal course of the overall disorder (Solberg, Birkeland, Blix, Hansen, & Heir, 2016). Therefore, revealing these dynamic interplay patterns could help us to understand the maintenance of

* Corresponding author at: Institute of Psychology, Chinese Academy of Sciences, 16 Lincui Road, Beijing 100101, China.
E-mail address: wangli1@psych.ac.cn (L. Wang).

https://doi.org/10.1016/j.janxdis.2020.102319
Received 5 April 2020; Received in revised form 14 September 2020; Accepted 21 September 2020
Available online 28 September 2020

on-going PTSD symptom after trauma exposure.

Till now, all the studies on PTSD symptom dynamics were conducted in adult samples. Most studies have highlighted hyperarousal symptom as a dominant symptom cluster shaping subsequent maintenance of PTSD symptoms. The initial study conducted in survivors of community violence found hyperarousal symptom severity significantly predicted the symptom levels of intrusion, avoidance and numbing at a subsequent time point, but was not generally influenced by them (Schell et al., 2004). The results were replicated in another longitudinal study of orofacial injury survivors (Marshall, Schell, Glynn, & Shetty, 2006), and in a 20-year follow-up study of war veterans (Solomon, Horesh, & Ein-Dor, 2009). Basing on these findings, a 'psychological engine' model was proposed to describe the role of hyperarousal in maintaining PTSD symptom levels. In this model, the hyperarousal symptom was hypothesized as the driving force of PTSD by providing the platform on which other symptom clusters subsequently appear (Solomon et al., 2009). The model has got empirical support from a more recent study of veteran. In this study, hyperarousal level was reported as a stable predictor of subsequent fluctuations in the 3 other PTSD symptom clusters at subsequent 2-week intervals (Doron-LaMarca et al., 2015). Moreover, it was reported in disaster survivors that different components of hyperarousal play different roles in PTSD dynamics. Anxious arousal symptoms, measured by hypervigilance and exaggerated startle response, may primarily drive intrusion symptoms. Meanwhile, dysphoric arousal symptoms, measured by sleep disturbance and difficulty concentrating, may primarily drive numbing symptoms over 3–8 years after the disaster (Pietrzak et al., 2014).

In addition, there are increasing evidence showing intrusion symptom as a key factor of PTSD maintenance (e.g. Maples-Keller, Price, Rauch, Gerardi, & Rothbaum, 2017; Solberg et al., 2016). Intrusion at 10 months after direct exposure to a bombing was reported to predict levels of all PTSD symptom clusters at 22 months (Solberg et al., 2016). Results from psychotherapy research also suggest that intrusion demonstrated significant effects on other PTSD symptoms clusters across pretreatment, mid-treatment and posttreatment (Maples-Keller et al., 2017). These findings were in accordance with the cognitive processing model of PTSD (Creamer, Burgess, & Pattison, 1992). According to the model, the presence of intrusive thoughts indicates activation of the memory network, which further produces a state of hyperarousal, negative affective and cognitive responses and contribute to the maintenance of PTSD.

Several limitations of these studies should be noted. First, all existing findings were based on the *DSM-III* or *DSM-IV* criteria for PTSD. The *DSM-5* made several significant modifications to the symptom criteria of PTSD, and reorganized the symptoms from three symptom clusters to four. To our knowledge, no studies have reported the dynamic interplay between *DSM-5* PTSD symptom clusters to date. Reinvestigation to the question using the new criteria is clearly needed. Furthermore, all these studies used cross-lagged models, which is a commonly used statistical method to uncover interplay between multiple variables from longitudinal datasets. The cross-lagged model was focused on the relationships between symptom levels. Changes, as one concept of most interest in longitudinal studies, are not represented in the model. Therefore, it was suggested that any casual inference of cross-lagged results should be treated with caution as because the model does not allow for systematic growth components (Grimm & Ram, 2018).

Despite of the limitations, these findings from adult samples provided important reference for the understanding of child and adolescent PTSD dynamics, and would help to develop two different hypotheses for child PTSD. The first one postulates that hyperarousal symptoms may play a predominant role in maintenance of other PTSD symptoms. The alternate hypothesis is that intrusion symptoms might be the causal factor that maintain other symptoms. Neither of these two hypotheses have been tested in children and adolescents. To test these hypotheses and extend our knowledge on child PTSD psychopathology, we conducted the current study. *DSM-5* PTSD symptoms were measured at 4, 8 and 13 months after trauma exposure in a disaster-exposed child and adolescent sample. The *DSM-5* criteria and advanced statistical models were used to address the aforementioned methodological limitations of previous studies.

2. Method

2.1. Participants and procedures

The Binhai District of Tianjin City in China was hit by a series of severe chemical explosions in August 2015. One hundred and seventy three people were killed and 797 were injured in this disaster. The participants in the current study were recruited from a primary school and a middle school located nearest to the blast area. All students above third-grade level were initially included in the study. Students who did not confirm personally experience the explosion were excluded. Of the sample of 836 students who took part in the first survey, 637 (76.2 %) participants reported at least one type of explosion-related trauma exposure, and 162 (19.4 %) participants reported more than four types. For more detailed information of this sample, see Wang et al. (2015).

The surveys were conducted by classroom groups. All the participants completed self-reported questionnaires with assistance from trained research assistants and school teachers after being introduced to the aim of the study. Written informed consent was obtained from the participants and their guardians. The study protocol was reviewed and approved by the Institutional Review Board of the institute. The first survey was conducted at four months after the disaster, and two follow-up surveys took place at eight and thirteen months after the disaster, respectively. For the current purpose, the analytic sample was restricted to those participants who took part in all three surveys ($N = 659$, 78.8 %). Those participants who were excluded (N = 177) showed no significant differences in gender, trauma exposure to the explosion and PTSD total symptom severity at time point 1 (all $p > .05$) but were significantly older ($t = 5.79$, $df = 834$, $p < .001$, Cohen's $d = 0.40$), when compared with those who were included in our study.

2.2. Measures

PTSD symptoms was assessed using the PTSD Checklist for *DSM-5* (PCL-5; Weathers et al., 2013). The PCL-5 was developed as a self-report checklist of 20 *DSM-5* PTSD symptoms. Each item is rated on a 5-point Likert scale reflecting severity from 0 (not at all) to 4 (extremely) during the past month. All the participants were instructed to rate their PTSD symptoms specifically based on the explosion. The Chinese version of PCL-5 was adapted by translation and back translation, and has demonstrated good psychometric properties in traumatized Chinese children and adolescents (Cao, Wang, Cao, Zhang, & Elhai, 2017; Li et al., 2019; Wang et al., 2015). Cronbach's α were .86–.90 for intrusion subscale, .84–.87 for avoidance subscale, .81–.91 for NACM subscale and .83–.89 for AAR subscale in three waves of the survey.

2.3. Data analysis

Structural Equation Modeling (SEM) was used to model level changes and dynamic influences between PTSD symptom clusters, using Mplus 7.0. Dependent variable scores were treated as continuous variables. To provide estimates robust to non-normal distribution and missing values, the models were evaluated by full information maximum likelihood estimation with robust standard errors (MLR, Muthén & Muthén, 2007). Excellent fit is evidenced by the root mean square error of approximation (RMSEA) $\leq .06$, standardized root mean square residual (SRMR) $\leq .08$, comparative fit index (CFI) $\geq .95$ and Tucker-Lewis index (TLI) $\geq .95$ as suggested by Hu and Bentler (1999). To compare nested models, the corrected scaled χ^2 difference test was used (Satorra & Bentler, 2001).

To model dynamic influences across symptom clusters, a

multivariate latent difference score (LDS) growth model was used. The model is an extension of the latent growth curve and cross-lagged models by containing the rate of change in the model (McArdle, 2009). The model would help to explain individual differences in trajectory features over time by estimating effects from predictors to the rate of changes (Grimm, 2007). In current study, four symptom clusters were simultaneously contained in the LDS model as previous reported (Ghisletta & Lindenberger, 2005). We modeled three sources of change rates for each symptom cluster. The first source was a constant component. The second source was the effect from the symptom cluster level (i.e. lagged effect). The third source was the influence from other symptom clusters (i.e. cross-variable effect). As suggested previously (Grimm, 2007), lagged effects across the two intervals were set as equal for model simplicity and increased statistical power, as were cross-variable effects. To test our hypotheses, the model was estimated in a confirmative manner. Cross effects were modeled only from intrusion and hyperarousal symptoms. In order to compare the contribution to symptom maintenance, cross-symptom effects from intrusion and hyperarousal to other PTSD symptoms were simultaneously estimated in a single LDS model. The cross-sectional correlations between the symptoms were also modeled as suggested (Grimm, 2007).

3. Results

3.1. Descriptive analyses

According to the *DSM-5* diagnostic algorithm of at least one intrusion symptom, one avoidance symptom, two negative alterations in cognitions and mood symptoms, and two alterations in arousal and reactivity symptoms of at least moderate (2 or higher) severity, 4.9 %, 3.3 % and 3.0 % of participants were screened as current probable PTSD cases at follow-up assessments, respectively. The severity of each symptom cluster over time is shown in Table 1. Repeated measures ANOVA on each symptom cluster revealed main effects of time on all the symptom cluster severity (all $P < 0.001$), which reflects the decreasing trend over time.

3.2. Latent difference score growth analysis

The LDS model showed excellent fit to the data (S–B $\chi^2(25, N = 659) = 8.67$, $p = .99$, RMSEA $< .001$, CFI $= 1.00$, TLI $= 1.00$, SRMR $= .017$). Detailed parameter estimation results of the model are shown in Table 2 and Table 3. Unstandardized path coefficients for the final LDS model are presented in Fig. 1 for better interpretability of the model. For avoidance, negative mood/cognition and hyperarousal symptoms, the rate of change for each interval was negatively predicted by their symptom levels at the start of the interval (e.g. the rate of change between the first follow-up and the second follow-up was significantly predicted by symptom levels at the first follow-up), known as the lagged effects (all $p < .01$). The lagged effects were not significant for intrusion symptoms. In terms of cross-variable effects, the symptom level of intrusion at the start of the interval positively predicted the rate of hyperarousal ($p = .008$) and negative changes in cognitions and mood ($p = .036$) symptom changes for each interval. Intrusion level also trended to positively predict subsequent increase in avoidance symptoms ($p = .059$). Effects of hyperarousal level to other symptoms were not significant (all $p > .05$).

Table 1
Descriptive statistics for demographics and symptom cluster severity over time.

	4 months	8 months	13 months
Boys	340 (51.6 %)		
Age	12.3 (2.3)		
Trauma exposure	2.1 (1.7)		
Intrusion	3.0 (3.8)	2.0 (3.4)	1.5 (3.1)
Avoidance	1.0 (1.7)	0.7 (1.5)	0.5 (1.5)
Negative alterations in cognitions and mood	1.9 (3.5)	1.9 (3.8)	1.3 (3.7)
Alterations in arousal and reactivity	3.4 (4.4)	3.0 (4.3)	2.2 (4.2)

Note. $N = 659$.

Table 2
Multivariate LDS model parameter estimates.

	IN	AV	NACM	AAR
Parameter estimates				
Mean initial level	2.959 (0.152)*	0.996 (0.073)*	1.835 (0.147)*	3.331 (0.181)*
Mean constant change	3.243 (1.588)*	0.859 (0.424)*	3.533 (1.534)*	4.194 (1.705)*
Variance of initial level	11.362 (1.318)*	2.654 (0.329)*	5.424 (1.374)*	12.378 (1.517)*
Variance of constant change	16.716 (14.666)	1.714 (1.123)	27.401 (16.794)	33.931 (21.415)
Cross-lagged effects				
$l_{IN}\rightarrow\Delta$	0.106 (0.836)	0.251 (0.133)	0.861 (0.410)*	1.433 (0.541)*
$l_{AAR}\rightarrow\Delta$	–1.346 (0.816)	–0.293 (0.214)	–0.961 (0.735)	–2.637 (0.811)*
$l_{NACM}\rightarrow\Delta$	_	_	–1.683 (0.440)*	_
$l_{AV}\rightarrow\Delta$	_	–2.511 (0.740)*	_	_

Note. $N = 659$; IN = intrusion; AV = avoidance; NACM = negative alterations in cognitions and mood; AAR = alterations in arousal and reactivity; $l_{IN}\rightarrow\Delta$= latent cross-lagged effect of intrusion level on latent change scores; values in parentheses are standard errors.
* $p < 0.05$.

4. Discussion

The current study evaluated the natural course and dynamic interplay between DSM-5 PTSD symptom clusters within 13 months after trauma exposure in a child and adolescent sample exposed to an explosion accident. The results of latent difference score growth analysis indicated that higher severity levels of avoidance, negative mood/cognition and hyperarousal symptom clusters could predict lower subsequent change score (i.e. greater decrease) in their respective symptom levels at the next time point, and higher intrusion symptom levels predicted higher subsequent change score (i.e. lower rates of recovery) for hyperarousal symptoms. These findings provide new insights into the relationship between PTSD symptom clusters during symptom development and clusters.

Generally, all PTSD symptom cluster levels decreased over time, which could be interpreted as spontaneous recovery of posttraumatic reactions. LDS analyses found for most symptom clusters, higher symptom severity was related to a higher rate of subsequently decrease for most symptom clusters. As the symptom levels decrease over time, the rates of symptom recovery slowed down, resulting in nonlinear changes in symptom levels. This response pattern was in consistency with the acute stress trajectory identified in a previous study of PTSD symptoms after exposure to fire disasters (van Loey, van de Schoot, & Faber, 2012). A meta-analysis of child and adolescent PTSD course following trauma also revealed similar findings that although PTSD prevalence and symptom severity showed moderate declines, particularly across the first 3 months, there was less evidence of further change after 6 months (Hiller et al., 2016).

Results from the current study provided not only important empirical support for the hypothesis that intrusion symptoms are the causal factor that maintain other symptoms, but also evidence against the hyperarousal hypothesis. We found direct effects of intrusion severity on the rate of changes in subsequent hyperarousal, avoidance and negative changes in cognitions/mood symptoms, which is consistent with some

Table 3
Covariance and standard errors from LDS model.

	IN_0	IN_1	AV_0	AV_1	$NACM_0$	$NACM_1$	AAR_0	AAR_1
IN_0	_							
IN_1	9.25 (4.32)*	_						
AV_0	4.30 (0.57)*	3.80 (1.87)*	_					
AV_1	3.13 (1.23)*	5.07 (3.73)	1.43 (0.52)*	_				
$NACM_0$	5.81 (1.01)*	8.97 (4.36)*	2.97 (1.15)*	2.97 (1.15)*	_			
$NACM_1$	12.24 (4.46)*	19.18 (15.22)	4.17 (1.99)*	5.86 (4.01)	15.81 (6.23)*	_		
AAR_0	9.76 (1.22)*	13.55 (6.47)*	4.01 (0.55)*	4.14 (1.73)*	7.24 (1.20)*	15.81 (6.23)*	_	
AAR_1	8.49 (4.93)*	22.42 (17.47)	3.87 (2.11)	6.39 (4.63)	11.63 (4.60)*	26.85 (18.43)	17.75 (6.77)*	_

Note. $N = 659$; IN = intrusion; AV = avoidance; NACM = negative alterations in cognitions and mood; AAR = alterations in arousal and reactivity; IN_0= initial level of intrusion; IN_1 = constant changes of intrusion; values in parentheses are standard errors.
* $p < 0.05$.

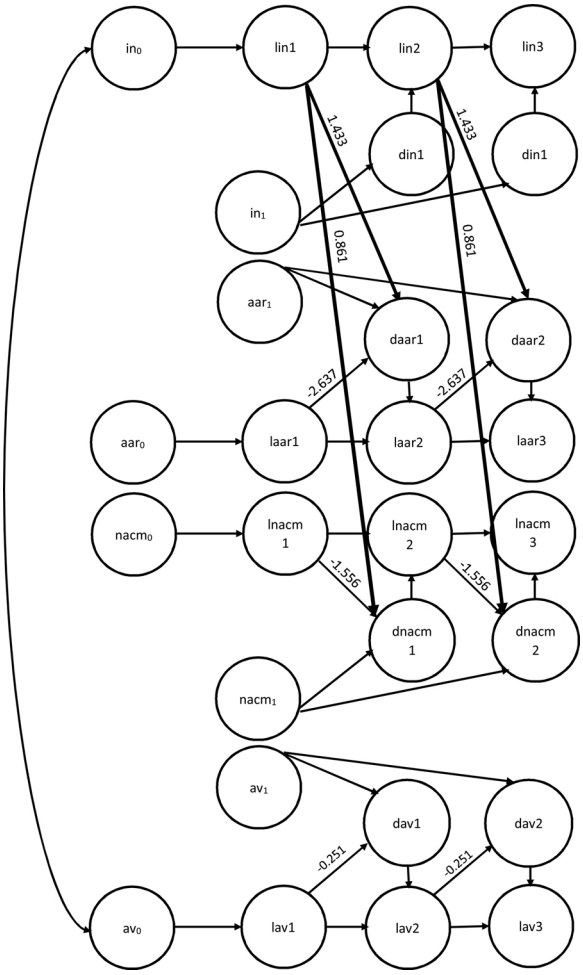

Fig. 1. Multivariable LDS model for DSM-5 symptom clusters. *Note.* N = 659. in = intrusion; av = avoidance; nacm = negative alterations in cognitions and mood; aar = alterations in arousal and reactivity. Coefficients reflect unstandardized path coefficients. All the paths shown in the figure are significant at $p < .05$. Paths in bold represent the cross effect. Correlation paths and residual terms were not specified in the diagram. Observed variables are not depicted in this diagram. Paths coefficients not marked on the paths were set as 1.

previous findings (e.g. Doron-LaMarca et al., 2015; Solberg et al., 2016). Specifically, those participants with higher intrusion symptom severity recovered more slowly from other PTSD symptoms, which suggests intrusion may play an important role in the maintenance and chronicity of overall PTSD symptoms. Additionally, we found no effects of hyperarousal symptoms on other PTSD symptom clusters. As mentioned above, some previous studies using cross-lagged models reported significant influence of hyperarousal to other PTSD symptoms, including intrusion (e.g. Marshall et al., 2006; Schell et al., 2004; Solomon et al., 2009), while other findings highlighted the role of intrusion symptoms (e.g. Maples-Keller et al., 2017; Solberg et al., 2016). Findings on the dynamics of PTSD symptoms are still inconsistent. One possible explanation for the inconsistent results could be the statistical model used in previous studies. Cross-lagged models have been widely used to analyze time-series data and draw dynamic inferences. By including the difference and change concepts, latent difference score models goes further from the cross-lagged model and thus provide a more flexible and precise framework for causal-dynamic questions (McArdle, 2009). Reanalysis of existing time-series datasets using a latent change model could potentially provide more precise insight into the dynamics between PTSD symptom dimensions and in fact redress some of the variability in findings. The difference in clinical phenotypes between child and adult PTSD also could explain some of the finding variances here. The current study is the first one to test PTSD symptom dynamics in a child and adolescent sample. Whether interplay between symptom clusters differs between patients in different developmental period still need to be further clarified.

Our current investigation of interplay between PTSD symptom clusters has some important theoretical and clinical implications. First, it expands the extant knowledge on the dynamic relationships between PTSD symptom clusters by conducting analyses to evaluate rate of changes in symptom severity. The results provide empirical support for theories which highlight intrusion and trauma-related memory in the pathology of PTSD (Creamer et al., 1992; Ehlers & Clark, 2000; Rubin, Berntsen, & Bohni, 2008). As described in the cognitive processing model of PTSD (Creamer et al., 1992), intrusion is the first-stage of reaction to processing of the traumatic memory network. The presence of intrusive thoughts indicates activation of the memory network, which further produces a state of hyperarousal, negative affective and cognitive responses. PTSD's avoidance symptoms are then coping strategies activated in response to the discomfort caused by the intrusion stage. The current findings are generally congruent with the proposal of the cognitive processing model. The effects from intrusion to hyperarousal and negative cognition/mood were stronger because they are directly driven by intrusive thoughts. Avoidance were triggered by intrusion afterwards, so their association was weaker. Considering the high heterogeneity within the intrusion clusters, more precise investigations on a symptom level would be helpful to explain which intrusive symptoms are doing most of the driving for other PTSD symptoms, which will really enhance our understanding of posttraumatic psychopathology. Second, our results highlight the importance of studies that examine the biological mechanisms underlying intrusion symptoms. A recent genome-wide association study of PTSD intrusion symptoms identified several meaningful regions in the genome related to the symptom clusters (Gelernter et al., 2019). Further studies uncovering the genetic and neuroscientific underpinning these symptom clusters would considerably enhance our understanding of the pathophysiology of PTSD. Last but not least, considering the effect of intrusion to

hyperarousal maintenance, psychotherapy and pharmacotherapy targeting on intrusion and trauma-related memories (e.g. prolonged exposure therapy, the eye movement desensitization and reprocessing, β-adrenergic receptor blocker propranolol) would be effective to treat those patients suffering from long-lasting hyperarousal states, and therefore improve quality of life.

This study had several limitations. First, PTSD symptoms were assessed by a self-report questionnaire. Results from studies using clinical interviews are still required to verify the robustness of results. Second, our surveys were conducted between 4 and 13 months after trauma exposure and therefore we could not investigate dynamics in the most acute phase of PTSD symptom trajectory. It was reported intrusion may increase the hyperarousal through mediation of avoidance symptoms during the first 30 days after trauma exposure (Price, Legrand, Brier, Gratton, & Skalka, 2019). Third, our participants were Chinese children and adolescents exposed to an explosion accident. The generalizability of the results needs to be tested in additional studies using samples from other cultural backgrounds and experienced different trauma types.

5. Conclusion

The current study is the first longitudinal study to investigate the dynamic influences between *DSM-5* PTSD symptom clusters, and also the first study of children and adolescent PTSD symptom dynamics. The findings support our hypothesis that intrusion symptoms are involved in the maintenance and chronicity of other PTSD symptoms, which provides support for the prominent role of intrusion in child PTSD dynamics, and extends our current knowledge on the development and maintenance of posttraumatic psychopathology.

Declaration of Competing Interest

All the authors declare no conflicts of interest.

Acknowledgements

This study was partially supported by the Key Project of the National Social Science Foundation of China (No. 20ZDA079), the National Natural Science Foundation of China (No. 31471004, 31971020), the Key Project of Research Base of Humanities and Social Sciences of Ministry of Education (No. 16JJD190006), and the Key Research Program of the Chinese Academy of Sciences (No. ZDRW-XH-2019-4).

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (fifth edition). Washington, DC: American Psychiatric Publishing.

Alisic, E., Zalta, A. K., Van Wesel, F., Larsen, S. E., Hafstad, G. S., Hassanpour, K., & Smid, G. E. (2014). Rates of post-traumatic stress disorder in trauma-exposed children and adolescents: Meta-analysis. *British Journal of Psychiatry, 204*(5), 335–340.

Cao, X., Wang, L., Cao, C., Zhang, J., & Elhai, J. D. (2017). DSM-5 posttraumatic stress disorder symptom structure in disaster-exposed adolescents: Stability across gender and relation to behavioral problems. *Journal of Abnormal Child Psychology, 45*(4), 803–814. https://doi.org/10.1007/s10802-016-0193-1

Creamer, M., Burgess, P., & Pattison, P. (1992). Reactions to trauma: A cognitive processing model. *Journal of Abnormal Psychology, 101*, 452–459.

Doron-LaMarca, S., Niles, B. L., King, D. W., King, L. A., Kaiser, A. P., & Lyons, M. J. (2015). Temporal associations among chronic PTSD symptoms in U.S. Combat veterans. *Journal of Traumatic Stress, 28*, 410–417. https://doi.org/10.1002/jts

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behavior Research and Therapy, 38*, 319–345. https://doi.org/10.1016/S0005-7967(99)00123-0

Galatzer-Levy, I. R., & Bryant, R. A. (2013). 636,120 ways to have posttraumatic stress disorder. *Perspectives on Psychological Science, 8*, 651–662. https://doi.org/10.1177/1745691613504115

Gelernter, J., Sun, N., Polimanti, R., Pietrzak, R., Levey, D. F., Bryois, J., … Stein, M. B. (2019). Genome-wide association study of post-traumatic stress disorder reexperiencing symptoms in &165,000 US veterans. *Nature Neuroscience, 22*, 1394–1401. https://doi.org/10.1038/s41593-019-0447-7

Ghisletta, P., & Lindenberger, U. (2005). Exploring structural dynamics within and between sensory and intellectual functioning in old and very old age: Longitudinal evidence from the Berlin Aging Study. *Intelligence, 33*, 555–587. https://doi.org/10.1016/j.intell.2005.07.002

Grimm, K. J. (2007). Multivariate longitudinal methods for studying developmental relationships between depression and academic achievement. *International Journal of Behavioral Development, 31*, 328–339. https://doi.org/10.1177/0165025407077754

Grimm, K. J., & Ram, N. (2018). Latent growth and dynamic structural equation models. *Annual Review of Clinical Psychology, 14*, 55–89.

Hiller, R. M., Meiser-stedman, R., Fearon, P., Lobo, S., Mckinnon, A., Fraser, A., & Halligan, S. L. (2016). Research Review: Changes in the prevalence and symptom severity of child post-traumatic stress disorder in the year following trauma — a meta-analytic study. *Journal of Child Psychology and Psychiatry, 8*, 884–898.

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6*, 1–55. https://doi.org/10.1080/10705519909540118

Lai, B. S., Lewis, R., Livings, M. S., Greca, A. M. L., & Esnard, A. M. (2017). Posttraumatic stress symptom trajectories among children after disaster exposure: A review. *Journal of Traumatic Stress, 30*, 571–582.

Li, J., Zhang, W., Chen, W., Yuan, H., Zhang, S., Tian, M., … Qu, Z. (2019). Applications of the Chinese version of the primary care PTSD screen for DSM-5 (PC-PTSD-5) for children. *Journal of Affective Disorders, 254*, 109–114. https://doi.org/10.1016/j.jad.2019.05.021

Maples-Keller, J. L., Price, M., Rauch, S., Gerardi, M., & Rothbaum, B. O. (2017). Investigating relationships between PTSD symptom clusters within virtual reality exposure therapy for OEF/OIF veterans. *Behavior Therapy, 48*, 147–155. https://doi.org/10.1016/j.beth.2016.02.011

Marshall, G. N., Schell, T. L., Glynn, S. M., & Shetty, V. (2006). The role of hyperarousal in the manifestation of posttraumatic psychological distress following injury. *Journal of Abnormal Psychology, 115*, 624–628. https://doi.org/10.1037/0021-843X.115.3.624

McArdle, J. J. (2009). Latent variable modeling of differences and changes with longitudinal data. *Annual Review of Psychology, 60*, 577–605. https://doi.org/10.1146/annurev.psych.60.110707.163612

Muthén, L. K., & Muthén, B. O. (2007). *Mplus User's Guide. Seventh Edition*. Los Angeles, CA: Muthén & Muthén.

Pietrzak, R. H., Feder, A., Schechter, C. B., Singh, R., Cancelmo, L., Bromet, E. J., Katz, C. L., Reissman, D. B., Ozbay, F., Sharma, V., Crane, M., Harrison, D., Herbert, R., Levin, S. M., Luft, B. J., Moline, J. M., Stellman, J. M., Udasin, I. G., El-Gabalawy, R., & Southwick, S. M. (2014). Dimensional structure and course of post-traumatic stress symptomatology in World Trade Center responders. *Psychological Medicine, 44*(10), 2085–2098.

Price, M., Legrand, A. C., Brier, Z. M. F., Gratton, J., & Skalka, C. (2019). The short-term dynamics of posttraumatic stress disorder symptoms during the acute posttrauma period. *Depression and Anxiety*. https://doi.org/10.1002/da.22976. in press.

Rubin, D. C., Berntsen, D., & Bohni, M. K. (2008). A memory-based model of posttraumatic stress disorder: Evaluating basic assumptions underlying the PTSD diagnosis. *Psychological Review, 115*, 985–1011. https://doi.org/10.1037/a0013397

Satorra, A., & Bentler, P. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika, 66*, 507–514.

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology, 113*, 189–197. https://doi.org/10.1037/0021-843X.113.2.189

Solberg, O., Birkeland, M. S., Blix, I., Hansen, M. B., & Heir, T. (2016). Towards an exposure-dependent model of post-traumatic stress: Longitudinal course of post-traumatic stress symptomatology and functional impairment after the 2011 Oslo bombing. *Psychological Medicine, 46*, 3241–3254. https://doi.org/10.1017/S0033291716001860

Solomon, Z., Horesh, D., & Ein-Dor, T. (2009). The longitudinal course of posttraumatic stress disorder symptom clusters among war veterans. *The Journal of Clinical Psychiatry, 70*, 837–843. https://doi.org/10.4088/JCP.08m04347

van Loey, N. E., van de Schoot, R., & Faber, A. W. (2012). Posttraumatic stress symptoms after exposure to two fire disasters: Comparative study. *PloS One, 7*, e41532. https://doi.org/10.1371/journal.pone.0041532

Wang, L., Zhang, L., Armour, C., Cao, C., Qing, Y., Zhang, J., … Fan, G. (2015). Assessing the underlying dimensionality of DSM-5 PTSD symptoms in Chinese adolescents surviving the 2008 Wenchuan earthquake. *Journal of Anxiety Disorders, 31*, 90–97. https://doi.org/10.1016/j.janxdis.2015.02.006

Weathers, F. W., Litz, B. T., Keane, T. M., Palmieri, P. A., Marx, B. P., & Schnurr, P. P. (2013). *The PTSD Checklist for DSM–5 (PCL-5)*. Boston, MA: National Center for PTSD.