

A longitudinal study of ICD-11 PTSD and Complex PTSD in the general population of Israel

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Abstract

The ICD-11 includes two trauma disorders: Posttraumatic Stress Disorder (PTSD) and Complex PTSD (CPTSD). CPTSD is a disorder comprised of PTSD and Disturbance in Self-Organization (DSO) symptoms. Evidence supports the construct validity of PTSD and CPTSD, however, the temporal stability of these constructs has rarely been tested. This study examined the diagnostic stability of PTSD and CPTSD, and the temporal associations between PTSD and DSO symptoms over a period of one-year. Data were collected from a nationally representative sample of Israeli adults ($n = 1,003$) and one year later a random half of this sample were reassessed ($n = 543$). There were no statistically significant changes in rates of PTSD (6.7%, 5.3%) and CPTSD (4.9%, 3.7%) over time. Latent variable cross-lagged analysis indicated that PTSD and DSO symptoms were stable over time and that DSO symptoms predicted subsequent PTSD symptoms. Results suggest that ICD-11 PTSD and CPTSD are stable constructs in the general population over a period of one year. We discuss the possibility that these findings are influenced by the specific cultural context of Israel. Additionally, given the stability and influence of DSO symptoms we discuss the potential value of psychological therapies that directly address these symptoms.

Key words: trauma; PTSD; Complex PTSD; ICD-11; longitudinal; cross-lagged analysis.

Highlights

1. 11.6% (T1) and 9.0% (T2) of the general population of Israel met diagnostic requirements for ICD-11 PTSD and Complex PTSD.
2. The prevalence rates of PTSD and Complex PTSD remained stable over the course of one year.
3. DSO symptoms are persistent over time and reinforce and intensify PTSD symptoms.
4. Clinical interventions that specifically address DSO symptoms may be of value.

Author contributions

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1.1 Introduction

The 11th version of the *International Classification of Diseases* (ICD-11) includes Posttraumatic Stress Disorder (PTSD) and Complex PTSD (CPTSD) as related-but-distinct diagnoses (World Health Organization [WHO], 2018). Both disorders require exposure to an event of an extremely threatening or horrific nature for the consideration of a diagnosis, and CPTSD is more likely to occur following exposure to multiple or prolonged traumatic events, particularly those that occur during early development (WHO, 2018; Brewin et al., 2017; Cloitre et al., 2019). A PTSD diagnosis is made if at least one symptom is present from the clusters of ‘Re-experiencing in the here and now’, ‘Avoidance’, and ‘Sense of Threat’ and these symptoms are associated with impaired functioning. A CPTSD diagnosis is made if all PTSD criteria are met and at least one symptom is present from the three Disturbance in Self-Organization (DSO) clusters of ‘Affective Dysregulation’, ‘Negative Self-Concept’, and ‘Disturbed Relationships’. These symptoms must also be associated with impaired functioning.

Empirical support for the construct validity of ICD-11 PTSD and CPTSD has come from general population (Ben-Ezra et al., 2018; Cloitre et al., 2019; Karatzias et al., 2019), clinical (Hyland et al., 2017; Karatzias et al., 2016), and refugee samples (Shevlin et al., 2018; Vallières et al., 2018) from around the world. The overwhelming majority of existing evidence, however, comes cross-sectional data and consequently little is known about the temporal stability of the symptoms and diagnoses of PTSD and CPTSD. Barbano et al. (2019) reported changes in rates of (ICD-11) PTSD among survivors of non-interpersonal traumas from three weeks post-trauma, on average, to four-and-a-half months post-trauma, on average. PTSD declined from 39.7% to 14.6%, reflecting the well-evidenced natural recovery process that occurs within the first months following traumatic exposure (e.g., Galatzer-Levy et al., 2013; Hepp et al., 2008). Although

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highly informative, these findings are limited as PTSD was assessed using a proxy measure (the CAPS-IV) rather than an ICD-11 specific measure such as the International Trauma Questionnaire (ITQ; Cloitre et al., 2018) meaning that the DSO symptoms were not assessed. Thus, it was impossible to determine how many people classified as having a PTSD diagnosis actually met diagnostic criteria for CPTSD. Furthermore, because many of the survivors were first assessed less than three weeks post-trauma, it is likely that the diagnostic rate at the first was over-estimated.

In this study we measured PTSD and DSO symptoms with the ITQ at two points across a 12-month period among a nationally representative sample of the Israeli adult population. The first objective was to test if there were statistically significant changes in rates of PTSD and CPTSD over the period of one year, and the second objective was to examine the temporal associations between the PTSD and DSO symptoms. Given the lack of available data and theory regarding the temporal stability of these diagnoses and the temporal relationships between the PTSD and DSO symptoms, no hypotheses were formulated, and an exploratory approach was adopted.

2. Methods

2.1 Participants and procedures

Participants were recruited from a nationally representative internet panel of Israeli adults ($N = 130,000$). The internet panel matches demographic information provided by the Israeli Bureau of Statistics (Bodas et al., 2018). At the first assessment (T1), a nationally representative sample of 1,003 adults were recruited. The participation rate was 31%. Twelve months later (T2), approximately 50% of the original sample were re-assessed ($n = 543$). The decision to only re-assess a subset of the original sample was due to resource constraints. The participants at T2

were selected on a random basis from the original sample and were contacted by the survey company and asked to participate in a second assessments. We do not have data regarding the participation rate at T2. At both assessments, participants were contacted by the survey company via email and at T1 participants were informed that they may be contacted again in the future to participate in a second round of assessments. If participant chose to participate, they followed a link to a secure website where they were required to provide informed consent to participate before completing any measures. Ethical approval for the collection of these data was provided by the ethical review board to which the final author is affiliated. All procedures contributing to this work complied with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. Inclusion criteria were that participants were over the age of 18 at the time of initial contact and were fluent in Hebrew. Additionally, all participants were considered to be trauma-exposed as Israeli citizens live under a direct or potential threat to life due to the ongoing armed-conflict in the Middle East, and thus satisfy the typical description of trauma exposure.

The mean age of the sample at T1 was 40.57 years ($SD = 14.53$, range 18-70) and 51.7% ($n = 519$) were female. The majority of participants lived in an urban area (82.3%, $n = 825$), were in a committed relationship (70.5%, $n = 707$), were in full- or part-time employment (82.7%, $n = 830$), and had completed a university level education (68.4%, $n = 686$). Participants at T2 did not significantly differ from non-participants in terms of sex ($\chi^2 (1) = 1.62, p = .204$, $\phi = .04$), urbanicity ($\chi^2 (1) = 0.00, p = .952$, $\phi = .00$), relationship status ($\chi^2 (1) = 3.39, p = .066$, $\phi = .06$), employment status ($\chi^2 (3) = 3.45, p = .327$, $\phi = .06$), or education status ($\chi^2 (3) = 7.23, p = .065$, $\phi = .09$). Additionally, no significant differences were observed between those who participated at T2 and those who did not on the number of reported traumatic life

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events ($t(1001) = 0.81, p = .416, d = .05$), PTSD symptoms ($t(1001) = 1.36, p = .174, d = .08$), or DSO symptoms ($t(1001) = 0.51, p = .607, d = .03$).

2.2 Measures

2.2.1 Traumatic Exposure

Lifetime exposure to 18 different traumatic events was measured using a modified version of the Life Events Checklist for DSM-5 (LEC-5: Weathers et al., 2013). In this study, two additional questions were added to the list of 16 LEC-5 events in order to measure childhood physical assault and childhood sexual assault. Respondents were instructed to indicate whether each event “Happened to me”, “Witnessed it”, “Learned about it”, “Part of my job”, “Not Sure”, or “Doesn’t apply”. Responses of “Happened to me” or “Witnessed it” were used to reflect exposure to each event, and all other responses reflected non-exposure. The number of traumatic life events ranged, therefore, from 0-18. The LEC-5 also asks a respondent to identify which event was most distressing, and how long ago this event occurred. The LEC-5 was completed at T1 but not at T2.

2.2.2 ICD-11 PTSD and CPTSD

The ITQ (Cloitre et al., 2018) is a self-report measure designed to capture the ICD-11 diagnoses of PTSD and CPTSD. At both assessments, the Hebrew translation of the ITQ was used and both versions can be freely accessed here: <https://www.traumameasuresglobal.com/itq>. The psychometric properties of the Hebrew translation of the ITQ were demonstrated in a previous study based on responses from the T1 assessment of this study (Ben-Ezra et al., 2018). Consistent with findings from the English version of the ITQ (Cloitre et al., 2018; Hyland et al., 2017; Karatzias et al., 2016), the latent structure of the Hebrew translation is best represented by a higher-order model including two second-order factors (‘PTSD’ and ‘DSO’) capturing the

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covariation between the first-order factors of ‘Re-experiencing in the here and now’, ‘Avoidance’ and ‘Threat’ (PTSD), and, ‘Affective Dysregulation’, ‘Negative Self-Concept’, and ‘Disturbed Relationships’ (DSO), respectively.

The ITQ instructs participants to complete the questionnaire in relation to their most distressing traumatic event. The ITQ includes six items measuring each PTSD symptom (T1 $\alpha = .89$; T2 $\alpha = .90$) and six items measuring each DSO symptom (T1 $\alpha = .88$; T2 $\alpha = .89$). Additionally, three items measure functional impairment (in the domains of social, occupational, and other important areas of life) related to the PTSD and DSO symptoms, respectively. Individuals respond to each PTSD item in terms of how much they have been bothered by that symptom over the past month, and to each DSO item in terms of how they typically feel, think about themselves, and relate to others. All items are measured using a five-point Likert scale ranging from 0 (*Not at all*) to 4 (*Extremely*). PTSD and DSO symptoms range from 0-24 with higher scores reflecting greater symptomatology. For diagnostic purposes, a symptom was deemed to be ‘present’ based on a Likert score of ≥ 2 (*Moderately*) (Cloitre et al., 2018).

2.3 Data analysis

The proportions of people meeting diagnostic criteria for PTSD and CPTSD at T1 and T2 were compared using McNemar’s exact binomial test. Paired samples t-tests were used to compare PTSD and DSO symptoms (total and subscale scores) across the two assessments. To adjust for an increased likelihood of a Type-1 error as a result of multiple testing, a Bonferroni correction was applied ($0.05 / 8 = 0.006$). Effect sizes were calculated using *Cohen’s d* corrected for repeated measures (d_{rm}) and values $< .40$ reflect small effects, values from .40-.80 reflect medium effects, and values $> .80$ reflect large effects (Morris, 2008).

The temporal associations between the PTSD and DSO symptoms were assessed using latent variable cross-lagged analysis. The PTSD latent variable was estimated using summed scores from the Re-experiencing, Avoidance, and Sense of Threat clusters, and the DSO latent variable was estimated using summed scores from the Affective Dysregulation, Negative Self-Concept, and Disturbed Relationships clusters. Four nested models were tested (Figure 1). Model 1 included only autoregressive paths for PTSD and DSO and tested the concept that these symptoms predict themselves but not one another over time. Model 2 included only cross-lagged paths between PTSD and DSO and tested the concept that these symptoms predict each other but not themselves over time. Model 3 included autoregressive and cross-lagged paths for PTSD and DSO and tested the concept that these symptoms predict themselves and one another over time. Model 4 imposed equality constraints on the cross-lagged paths between PTSD and DSO and tested the concept that both sets of symptoms were equally predictive of one another over time.

These models were tested with the factor loadings at T1 and T2 freely estimated (Models 1a, 2a, 3a, and 4a) and with the factor loadings constrained to be equal over time (Models 1b, 2b, 3b, and 4b). Constraining the factor loadings to be equal across the two assessments tests the assumption of metric invariance. If the models with equal factor loadings fit the data as well as or better than the models with freely estimated factor loadings the assumption of metric invariance is satisfied. Satisfying the assumption of metric invariance demonstrates that the same constructs are being measured at both assessments (van de Schoot, Lugtig, & Hox, 2012). All models were adjusted for age, sex, time since exposure to one's worst trauma ('time'), and the total number of traumatic life events ('traumas'). These covariates were measured at T1 and the PTSD and DSO latent variables at T1 and T2 were regressed onto the covariates. The PTSD and DSO latent variables were free to correlate at both assessments.

The cross-lagged models were estimated using the robust maximum likelihood estimator (Yuan & Bentler, 2000) in Mplus 8.2 (Muthén & Muthén, 2017). Missing data were managed using the full information maximum likelihood function meaning that the analyses were based on all available data from the T1 assessment (Cham et al., 2017). Determination of model fit followed standard recommendations whereby satisfactory fit was indicated by a non-significant chi-square (χ^2) result; Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) values $\geq .90$; and Root-Mean Square Error of Approximation (RMSEA) and Standardized Root Mean Square Residual (SRMR) values $\leq .08$ (Hu & Bentler, 1999). The four nested models were compared using the Akaike Information Criterion (AIC) where lower values indicate superior model fit. Metric invariance was assessed in accordance with Chen's (2007, p. 501) recommendations for large sample sizes ($N > 300$) where "...a change of $\geq -.010$ in CFI, supplemented by a change of $\geq .015$ in RMSEA or a change of $\geq .030$ in SRMR would indicate noninvariance".

Figure 1 here

3. Results

3.1 Descriptive statistics

The mean number of traumatic life events was 4.35 ($SD = 3.01$) and the event most commonly identified as the most distressing was the 'unexpected death of a loved one' (23.5%). Approximately half of the participants (50.8%) experienced their most distressing trauma more than ten years ago, 37.8% experienced their trauma between one and ten years ago, and 11.4% experienced their trauma in the year prior to the first assessment.

At T1 6.7% (95% CIs = 4.2%, 8.3%) met diagnostic requirements for PTSD and 4.9% (95% CIs = 2.4%, 5.7%) for CPTSD. At T2 5.3% (95% CIs = 3.4%, 7.2%) met requirements for PTSD and 3.7% (95% CIs = 2.1%, 5.3%) for CPTSD. A McNemar's exact binomial test found

no statistically significant change in diagnostic rates of PTSD ($p = .576$) and CPTSD ($p = .851$) across the two assessments.

In order to ensure that the observed changes in the PTSD and CPTSD diagnostic rates were not due to re-sampling bias, the T1 diagnostic rates were calculated among only for those who participated at T2 ($n = 543$). The PTSD (6.3%, 95% CIs = 4.2%, 8.3%) and CPTSD (4.1%, 95% CIs = 2.4%, 5.7%) rates were extremely similar to the estimates for the full sample and the confidence intervals were identical.

The changes in the mean PTSD and DSO symptoms (as well as their constituent symptom clusters) from T1 to T2 are presented in Table 1. PTSD symptoms significantly declined; however, the magnitude of this decline was small ($d = .14$, $p < .001$). The decline in PTSD symptoms was attributable to a change in Sense of Threat symptoms specifically ($d = .16$, $p < .001$). There were no statistically significant changes in the total or subscale DSO symptoms.

Table 1 here

3.2 Cross-lagged model results

Model fit results for the cross-lagged models are presented in Table 2. The changes in CFI, RMSEA, and SRMR between the models with freely estimated factor loadings and equal factor loadings (i.e., 1a vs. 1b, 2a vs. 2b etc.) supported the assumption of metric invariance across time. Thus, the four models with equal factor loadings were subsequently compared.

Model 1b (autoregressive paths only) provided a satisfactory representation of the data whereas Model 2b (cross-lagged paths only) provided an unsatisfactory representation of the data. Model 3b (autoregressive paths and freely estimated cross-lagged paths) had a lower AIC value than Model 1b indicating an improvement in fit following the inclusion of the cross-lagged paths between PTSD and DSO. Model 4b (autoregressive paths and equal cross-lagged paths)

had a higher AIC value than Model 3b indicating that the cross-lagged paths between PTSD and DSO were not equivalent.

Table 2 here

As presented in Figure 2, the factor loadings for PTSD and DSO at T1 and T2 were all significant ($p < .001$), positive, and strong ($\lambda > .70$). The DSO autoregressive path ($\beta = .79, p < .001$) was greater than the PTSD autoregressive path ($\beta = .39, p < .001$). T1 DSO positively predicted T2 PTSD ($\beta = .19, p = .012$), and T1 PTSD negatively predicted T2 DSO ($\beta = -.18, p = .007$). The correlations between PTSD and DSO at T1 ($r = .53, p < .001$) and T2 ($r = .59, p < .001$) were strong.

Females had higher levels of PTSD ($\beta = .18, p < .001$) and DSO ($\beta = .08, p = .014$) at T1, and higher levels of PTSD ($\beta = .14, p = .001$) and DSO ($\beta = .08, p = .043$) at T2. Age was negatively associated with DSO at T1 ($\beta = -.10, p = .003$) and T2 ($\beta = -.08, p = .049$). Time passed since trauma exposure was negatively associated with T1 PTSD scores ($\beta = -.19, p < .001$) and positively associated with T2 PTSD scores ($\beta = .09, p = .025$). Exposure to a greater number of traumatic life events was positively associated with PTSD at T1 ($\beta = .35, p < .001$) and T2 ($\beta = .14, p = .004$), as well as with DSO at T1 ($\beta = .30, p < .001$) and T2 ($\beta = .10, p = .042$).

Figure 2 here

As a supplementary analysis, an additional test of scalar invariance was conducted to assess the invariance of the PTSD and DSO indicators over time, and then test for differences in the PTSD and DSO latent variable means over time. When the corresponding PTSD and DSO indicator intercepts were constrained to be equal at T1 and T2 the fit of the model was acceptable ($\chi^2(94) = 406.58, p < .001$; RMSEA=.058 (95% CIs = .052 - .063); CFI = .982; TLI = .903;

SRMR = .052). Based on Chen's (2007) guidelines for determining scalar invariance, the change in the fit statistics between the model with intercepts freely estimated and the model with the intercepts constrained equal supported the invariance of intercepts ($\Delta\text{CFI} = -.012$; $\Delta\text{RMSEA} = .002$; $\Delta\text{SRMR} = .01$). Subsequently, the PTSD and DSO latent variable means at T1 were fixed to zero and the latent variable means at T2 were estimated. The T2 mean for PTSD was significantly lower than T1 ($M = -1.10$, $SE = .25$, $p < .001$) but there was no significant difference for DSO ($M = -0.01$, $SE = .28$, $p = .959$). These estimates are consistent with the mean changes reported in Table 1.

4. Discussion

We found that 11.6% and 9.0% of the general adult population of Israel met the diagnostic requirements for ICD-11 PTSD or CPTSD at the first and second assessments, respectively. Previous studies from Israel that employed self-report measures of DSM-IV PTSD found that rates varied between 8.8% and 10.1% (see Hoffman, Diamond, & Lipsitz, 2011 for a review). This suggests that the ICD-11 criteria identify a very similar proportion of diagnostic cases as the DSM-IV within the general population. This result is consistent with findings from the United States where 7.2% of the adult population met criteria for ICD-11 PTSD or CPTSD (Cloitre et al., 2019); a prevalence rate consistent with estimates of DSM-IV (Kessler et al. 1995; Kessler et al., 2005) and DSM-5 (Goldstein et al., 2016) PTSD.

We found no statistically significant change in the rates of PTSD and CPTSD across the 12-month assessment period. This is inconsistent with the findings of Barbano et al. (2019) who reported substantial reductions in PTSD rates. These inconsistent findings are almost certainly a consequence of the differences in when participants were assessed following their trauma exposure. Unlike the recently traumatised group assessed by Barbano and colleagues, the vast

majority (88.6%) of participants in this study experienced their index trauma event more than one year prior to their assessment, with half experiencing their index trauma more than a decade prior to their assessment. Collectively, these findings suggest that recovery from (ICD-11) PTSD is common in the first months after traumatic exposure but as time passes natural recovery from PTSD and CPTSD is unlikely.

Our results showed that PTSD was slightly more common than CPTSD at both assessments, however, the confidence intervals for the two diagnoses overlapped at both assessments meaning that PTSD was not *significantly* more common than CPTSD. These findings are similar and distinct from findings from other nations. For example, in the United States, rates of PTSD (3.4%) and CPTSD (3.8%) were very similar (Cloitre et al., 2019); in Ireland, CPTSD (7.7%) was more common than PTSD (5.0%) but not significantly so (Hyland et al., 2020); and in a trauma-exposed sample from the United Kingdom, CPTSD (12.9%) was significantly more common than PTSD (5.3%). Collectively, these data indicate that CPTSD is not a rare condition in the general population and may occur at least as frequently as PTSD. Moreover, the variability in relative rates of PTSD and CPTSD across nations may be attributable to specific cultural factors within each country. The slightly higher rate of PTSD – a primary fear-based disorder – in Israel, for example, may be due to the fact that the Israeli population lives under the ongoing threat of rocket and terrorist attacks. This interpretation should be viewed cautiously in light of the fact that PTSD rates were not significantly higher than rates of CPTSD, however, recognizing the role that cultural and contextual factors play in the presentation of trauma responses may improve our understanding of these disorders and the best ways to intervene to prevent and treat them (Vallières et al., 2016).

The paired samples t-test results showed that the only symptom cluster to significantly change over time was Sense of Threat, although the decline was very small. There are several possible explanations for this change: (1) it may reflect a naturally occurring decline in threat-related symptoms, possibly driven by the small proportion of recently traumatised (i.e., in the last year) participants in the sample; (2) it may be the result of the increased safety in Israel across the study period where the number of terrorist related attacks continued its sharp decline since 2015; or (3) it may simply reflect a Type-1 error. Given that the change was so small, we caution against over-interpreting this result. The overwhelming trend was one of stability over time.

This stability of PTSD and DSO symptoms was also demonstrated by the results of the cross-lagged analyses. Specifically, the strength of the autoregressive paths for PTSD and DSO indicated that these symptoms were strongly self-reinforcing. The DSO symptoms were particularly stable over time and this is consistent with the conceptualization of DSO symptoms as ‘severe and persistent’ indicators of distress (WHO, 2018). Although stable, the PTSD and DSO symptoms did influence one another across time. Cross-sectional studies have shown these symptoms to be strongly and positively associated with one another (Brewin et al., 2017), however, the temporal effects observed in this study suggests a less straightforward relationship. The negative effect of T1 PTSD symptoms on T2 DSO symptoms was perplexing as it suggests that higher levels of PTSD predict slightly lower levels of DSO one year later. We suspected that this counterintuitive effect likely occurred because T1 DSO scores accounted for so much of the variance in T2 DSO scores (i.e., the strong autoregressive effect). As such, we examined the bivariate association between T1 PTSD and T2 DSO scores and found them to be positively correlated ($r = .35, p < .001$). Thus, we believe that the negative temporal effect from T1 PTSD symptoms to T2 DSO symptoms in the cross-lagged model was due to multicollinearity in the

model. It was telling that T1 DSO scores *positively* predicted T2 PTSD scores and we believe this effect was possible because T1 PTSD scores accounted for much less of the variance in T2 PTSD scores (i.e., the weaker autoregressive effect). A plausible interpretation of these results, therefore, is that PTSD and DSO symptoms do have some influence on one another over time; that DSO symptom likely reinforce and intensify PTSD symptoms over time; and although PTSD symptoms may have a small effect on later DSO symptoms, it is likely that once the DSO symptoms are established they remain largely self-perpetuating.

These findings have several important clinical and diagnostic implications. First, the stability of the DSO symptoms suggests that interventions other than those that have been established for the fear-based symptoms that characterise PTSD (such as exposure) may be required, or, a longer course of treatment may be beneficial (Karatzias et al., 2019). Such speculations, of course, require empirical testing. Second, PTSD symptoms are required for a diagnosis of CPTSD, however, given that these symptoms may be more likely to naturally decline than the DSO symptoms, it is conceivable that clinicians will encounter patients who have high DSO symptoms and low PTSD symptoms. This may be especially likely if the traumatic event occurred in the distant past. Such patients may be conceptualized as experiencing ‘sub-clinical CPTSD’. Such patients may benefit more from psychological interventions that specifically target and treat DSO symptoms such as Skills Training for Affective and Interpersonal Regulation (Hassija & Cloitre, 2015). Further research is required to determine how common sub-clinical cases of CPTSD are in the population, and in clinical settings, and the optimal method to treat such patients.

4.1 Limitations

These results should be interpreted with several limitations in mind. First, we could only recruit approximately half of the original sample for the follow-up assessment. While we cannot completely rule out the possibility that our results were influenced by sampling bias, we attempted to minimise this possibility by randomly selecting participants from the original sample. Second, cross-lagged panel model analysis has been criticised due to its inability to distinguish between the within- and between-person effects; a limitation that can result in biased estimates of the presence, strength, and direction of causal influences (Hamaker, Kuiper, & Grasman, 2015). This may be another reason why T1 PTSD symptoms were found to negatively predict T2 DSO symptoms. This limitation can be addressed using ‘multiple indicator random intercept cross-lagged panel modelling’, however, this approach necessitates data from a minimum of three assessments (Hamaker et al., 2015). Consequently, it was impossible to disentangle the within- and between-person effects in this study. Future research with additional follow up assessments will help to clarify the temporal associations between the PTSD and DSO symptoms. Finally, we were unable to determine if participants experienced any positive (e.g., mental health interventions) or negative (e.g., additional traumatic life events) life events during the assessments that may have influenced diagnostic rates and symptom levels at the follow up assessment.

4.2 Conclusion

In conclusion, this study provides novel evidence that PTSD and CPTSD are stable constructs at the symptom and diagnostic levels within the general population of Israel. In particular, the DSO symptoms were shown to be extremely stable across time and reinforced and intensified later PTSD symptoms. These findings are important as they suggest that there may be

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clinical value in developing evidenced-based psychological interventions to treat DSO symptoms, specifically, in addition to the 'core' PTSD symptoms (Karatzias & Cloitre, 2019).

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Table 1. Paired samples t-tests comparing mean symptom scores from Time 1 to Time 2 ($n = 543$).

		Mean	SD	t	p*	d _{rm} (95% CI)
PTSD	Time 1	4.32	5.04	3.34	.001	.14 (.02, .26)
	Time 2	3.58	4.97			
Re-experiencing	Time 1	1.21	1.69	1.75	.081	.08 (-.04, .19)
	Time 2	1.06	1.69			
Avoidance	Time 1	1.46	1.98	2.40	.017	.10 (.02, .22)
	Time 2	1.24	1.98			
Sense of threat	Time 1	1.66	2.17	3.97	< .001	.16 (.04, .28)
	Time 2	1.28	1.95			
DSO	Time 1	4.68	4.60	.43	.669	.02 (-.10, .14)
	Time 2	4.61	4.87			
Affective dysregulation	Time 1	2.18	1.79	1.75	.081	.07 (-.05, .19)
	Time 2	2.05	1.78			
Negative self-concept	Time 1	1.01	1.65	-1.33	.183	.06 (-.06, .18)
	Time 2	1.10	1.80			
Disturbed relationships	Time 1	1.50	1.93	.50	.615	.02 (-.10, .14)
	Time 2	1.46	1.99			

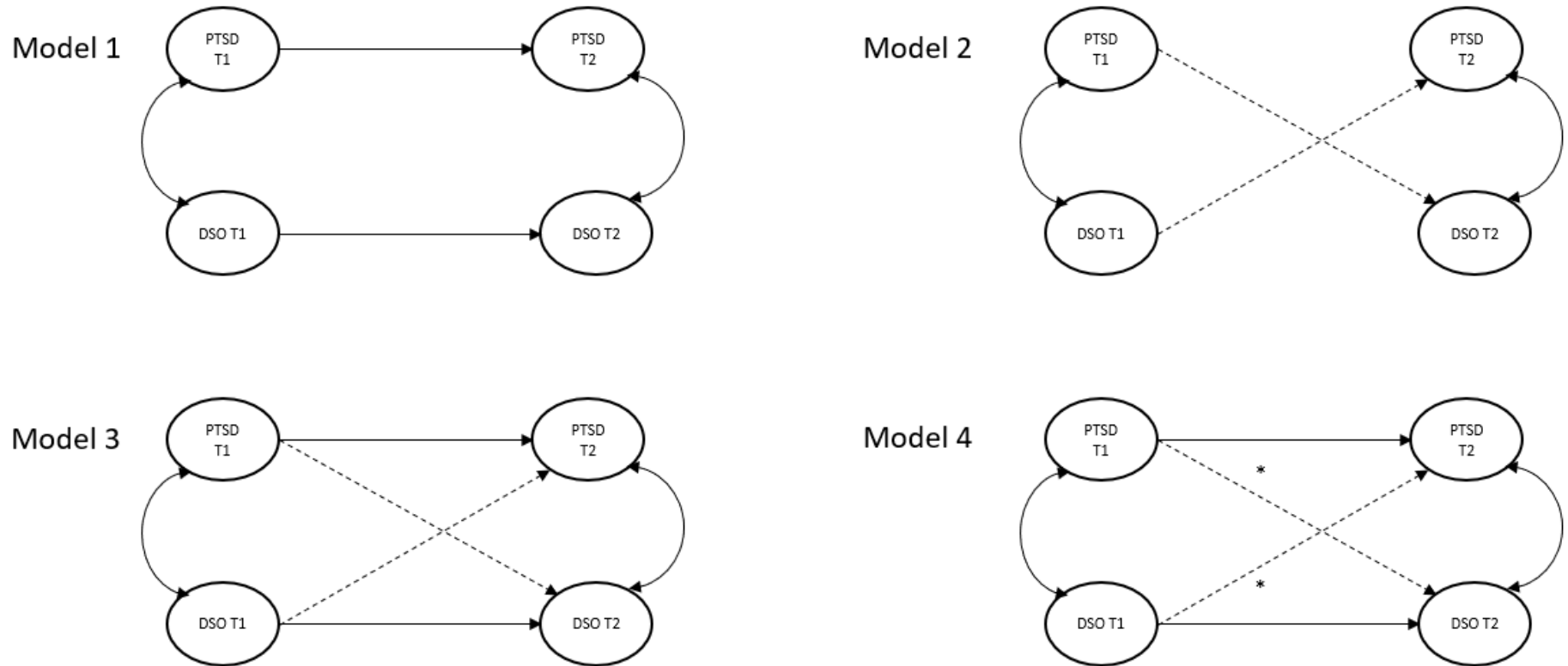
Note. All degrees of freedom = 542; SD = standard deviation; PTSD = posttraumatic stress disorder; DSO = disturbance in self-organization; p* = statistical significance result with a Bonferroni correction (alpha level = .006); d_{rm} (95% CI) = Cohen's d for repeated measures designs with 95% confidence intervals.

Table 2. Latent cross-lagged panel model results with covariates ($N = 1,003$).

	χ^2	df	CFI	TLI	RMSEA (90% CI)	SRMR	AIC
1a. Autoregressive paths only	357.35*	82	.929	.902	.058 (.052, .064)	.048	33436
1b. Autoregressive paths only	358.42*	86	.930	.907	.056 (.050, .062)	.046	33432
2a. Cross-lagged paths only	557.09*	82	.878	.830	.076 (.070, .082)	.117	33695
2b. Cross-lagged paths only	557.08*	86	.879	.840	.074 (.068, .080)	.112	33693
3a. Autoregressive and cross-lagged paths	346.03*	80	.932	.903	.058 (.051, .064)	.042	33424
3b. Autoregressive and cross-lagged paths	347.23*	84	.932	.908	.056 (.050, .062)	.040	33420
4a. Autoregressive and cross-lagged paths constrained equal	359.21*	81	.929	.900	.059 (.052, .065)	.048	33438
4b. Autoregressive and cross-lagged paths constrained equal	360.12*	85	.929	.905	.057 (.051, .063)	.046	33433

Note. Models 1a–4a = factor loadings are freely estimated; Models 1b–4b = factor loadings are constrained equal across time (test of metric invariance); χ^2 = chi-square goodness of fit statistic; * indicates χ^2 is statistically significant ($p < .001$); df = degrees of freedom; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA (90% CI) = Root-Mean-Square Error of Approximation with 90% confidence intervals; SRMR = Standardized Square Root Mean Residual; AIC = Akaike Information Criterion; Best fitting model in bold.

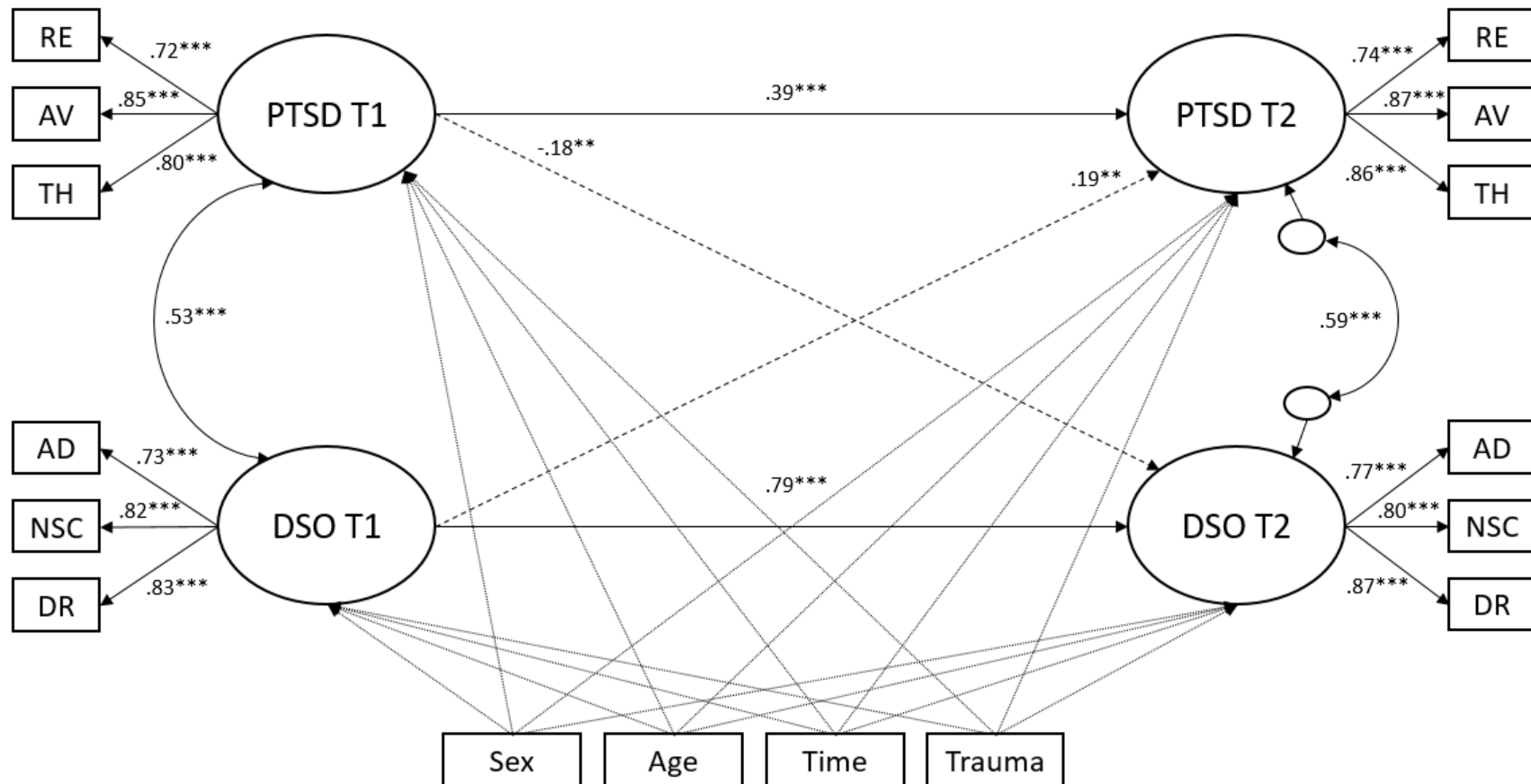
Figure 1. Simplified illustrations of the four cross-lagged panel models (Models 1b – 4b).



Note: bold lines = autoregressive paths; dashed lines = cross-lagged paths; * cross-lagged paths are constrained to be equal in Model 4.

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Figure 2. Standardized coefficients from the metric invariant cross-lagged panel model of the associations between PTSD and DSO symptoms across a 12-month period controlling for multiple covariates.



Note: PTSD = posttraumatic stress disorder; DSO = disturbances in self-organization; RE = reexperiencing in the here and now; AV = avoidance; TH = sense of threat; AD = affective dysregulation; NSC = negative self-concept; DR = disturbed relationships; statistical significance = * $p < .05$, ** $p < .01$, *** $p < .001$; bold lines = autoregressive paths; dashed lines = cross-lagged paths; dotted lines = covariate effects.