



The Relationship between Posttraumatic Stress Disorder, Interpersonal Sensitivity and Specific Distress Symptoms: the Role of Cognitive Emotion Regulation

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Abstract

This study examined 1) the relationship between posttraumatic stress disorder (PTSD) from past trauma, interpersonal sensitivity and psychiatric co-morbidity, and 2) whether cognitive emotion regulation strategies would mediate the impact of PTSD on specific distress outcomes. Four hundred seventy-five Kazakh students ($F = 336$, $M = 139$) participated in the study and completed a demographic page, Posttraumatic Stress Diagnostic Scale for DSM-5, General Health Questionnaire-28, Interpersonal Sensitivity Measure and Cognitive Emotion Regulation Questionnaire. The results showed that 71% reported that they had experienced at least one trauma throughout their lifespan, of whom 39% met the criteria for full-PTSD. Controlling for age and university majors, PTSD was associated with interpersonal sensitivity and psychiatric co-morbidity. Cognitive emotion regulation strategies were correlated with specific distress outcomes. Whilst positive reappraisal and refocusing on planning were associated with interpersonal sensitivity, self-blame and putting the trauma into perspective were associated with psychiatric co-morbidity. Self-blame mediated the impact of PTSD on psychiatric co-morbidity. To conclude, trauma can heighten levels of sensitivity in interpersonal interaction and psychological symptoms. Having specific thoughts about the trauma can impact on specific psychological reactions. Blaming oneself for the trauma can influence its impact on the severity of psychological symptoms.

Keywords PTSD · Interpersonal sensitivity · Cognitive emotion regulation

Introduction

Interpersonal sensitivity is defined as an excessive awareness of or sensitivity to the behaviour and feelings of others. Individuals with a high level of interpersonal sensitivity tend to be

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preoccupied with social interaction, excessively sensitive to perceived criticism or rejection during the interaction and vigilant to others' behaviour and mood [1]. Posttraumatic stress disorder (PTSD), alongside other psychological symptoms such as anxiety, depression, somatization, dissociation, and aggression, has been associated with interpersonal sensitivity [2–17]. This association is persistent over time [3, 5, 11] and individuals who have met the full-PTSD criteria or experienced multiple-victimization tend to report higher levels of interpersonal sensitivity and psychiatric co-morbidity than individuals with partial PTSD [10, 17–19].

Little is known regarding whether cognitive emotion regulation might influence the relationship between PTSD, interpersonal sensitivity and psychiatric co-morbidity. Cognitive emotion regulation emphasizes the importance of regulating emotions through specific thoughts or cognitions, i.e. what people think about specifically after the trauma. One might, for example, think about blaming oneself (self-blame) or others (other-blame) for the trauma, exaggerating the terror (catastrophizing) or downgrading the severity of it (putting into perspective), giving it a positive meaning or seeing it as a growth experience (positive reappraisal), or resigning oneself to what has happened (acceptance). One might think about the feelings and thoughts associated with the trauma (rumination), positive interpretations (positive refocusing) or what steps to take to manage it (planning) [20].

Whilst some cognitive emotion regulation strategies are adaptive, some are maladaptive [21]. Negative cognitions about the self, the world, self-blame and catastrophizing, for example, have been associated with the PTSD symptom of negative alterations in cognitions and mood; catastrophizing is also associated with the re-experiencing symptoms [22]. Rumination tends to be reduced through treatment [23]. On the other hand, positive-reappraisal and putting-into-perspective strategies have been shown to facilitate natural recovery from PTSD among motor vehicle accident survivors [23].

In essence, cognitive emotion regulation is a way of managing the emotionally distressing information of a trauma. Trauma can create drastic changes in the way people perceive themselves, pre-empting considerable emotional distress. To prevent exhaustion, emotional regulation strategies are employed to inhibit or regulate the flow of traumatic information to a tolerable extent [24]. In turn, they impact on mental health outcomes [25]. In other words, cognitive emotion regulation strategies can act as mediators, mitigating the effects of trauma onto psychological symptoms. This has been demonstrated for the cognitive emotion regulation strategies of catastrophizing [26] and rumination of negative emotions [27].

According to the cognitive specificity hypothesis [28], since thoughts or cognitive processes are paramount in cognitive emotion regulation, one would speculate that they would have effects on specific mental health outcomes. That is, adopting certain cognitive emotion regulation strategies (i.e. having certain thoughts about oneself, others or the trauma) to regulate trauma distress would impact on, for example, either interpersonal sensitivity or general psychological symptoms. Several trauma studies have in fact supported this speculation [29–31]. A meta-analysis has also concluded that the relationship between emotion regulation strategies and psychological symptoms would vary depending on their typologies [25].

Focusing on a group of university students from Kazakhstan, the current study aimed to examine 1) the relationship between PTSD from past trauma, interpersonal sensitivity and psychiatric co-morbidity, and 2) whether cognitive emotion regulation strategies would mediate the relationship between PTSD and specific distress outcomes. Guided by the preceding literature, we hypothesized that PTSD would be associated with interpersonal sensitivity and psychiatric co-morbidity. Different cognitive emotion regulation strategies would mediate the

impact of PTSD on specific outcomes, i.e. either interpersonal sensitivity or psychiatric co-morbidity.

Methods

Procedure

University students were recruited by posting advertisements on student hall of residence and in classes taught by the authors from Karaganda State University. On the advertisements, the purpose and a hyperlink to the research were given with inclusion criteria: 1) students aged over 18, and 2) Kazakh in ethnic origin. Using a snowball recruitment method, students who completed the online survey were encouraged to pass the hyperlink onto friends using social networking media such as Facebook and SMS. The online survey opened with a page stating that the research was entirely voluntary and anonymous, that data would be kept confidential and that participants were entitled to exit from the research at any point without giving a reason. The online survey comprised the questionnaires described in the measures section below. Since the Cognitive Emotion Regulation Questionnaire measured a range of strategies used to regulate emotions related to the trauma, those who did not experience any trauma were asked to complete a questionnaire focusing on how their emotions pertaining to daily stress were regulated. The ethics committee at Karaganda State University granted approval for the research.

Measures

A demographic page was used to collect information on gender, age, marital status, ethnicity, student status (full or part time), university major and the academic year at the time of the research.

Posttraumatic Stress Diagnostic Scale for DSM-5 (PDS-5) [32] was used to assess students' self-report on traumatic events and PTSD symptoms. The first part provides a list of traumatic events (e.g. natural disaster, accident) that participants need to select from and, if more than one, the event which has affected them the most. The second part consists of 20 trauma reactions, each rated on a 5-point Likert scale from 0 (not at all) to 4 (6 or more times a week/severe). Students were instructed to rate each item to indicate the severity of a particular symptom during the past month. Posttraumatic Stress Diagnostic Scale demonstrated excellent internal consistency ($\alpha = 0.95$), test-retest reliability ($r = 0.90$) and good convergent validity with the PTSD Checklist—Specific Version ($r = 0.90$) and the PTSD Symptom Scale—Interview Version for DSM-5 (PSSI-5; $r = 0.85$). Based on the current sample, the Cronbach's α for the total score was 0.96.

General Health Questionnaire-28 (GHQ-28) [33] was used to measure levels of somatic symptoms, anxiety and insomnia, psychosocial dysfunction, and depression among students using a 4-point Likert scale (0 = better than usual to 3 = much worse than usual). The questionnaire has excellent reliabilities with Cronbach's α ranging from 0.90–0.95 [34]. The current study revealed good reliability for the total score (Cronbach's $\alpha = 0.91$).

Interpersonal Sensitivity Measure (ISM) [1] generates five subscales concerning interpersonal sensitivity: interpersonal awareness, need for approval, separation anxiety, timidity and fragile inner self. Students rated 36 statements on a 4-point Likert scale (4 = very like me to

1 = very unlike me). The coefficient α for the total of interpersonal sensitivity was 0.86 for the student sample in the original study. Data based on the current study yielded the α of 0.85.

Cognitive Emotion Regulation Questionnaire (CERQ) [20] aims to measure what people think after a stressful event. It generates five adaptive (acceptance, positive refocusing, refocusing on planning, positive reappraisal, and putting into perspective) and four maladaptive (self-blame, rumination, catastrophizing, and blaming others) strategies. Students rated 18 items on a 5-point Likert scale (1 = almost never to 5 = almost always). Alpha reliabilities ranged from 0.67 to 0.81. Based on the current sample, α scores ranged from 0.63 to 0.88.

Data Analysis

Descriptive statistics were used to record students' demographic information. *T* tests were used to compare PTSD and no-PTSD groups in terms of levels of psychiatric co-morbidity, cognitive emotion regulation strategies and interpersonal sensitivity. Bonferroni correction was also used to reduce the likelihood for Type I error. Correlation coefficients were used to identify links between demographic variables and distress outcomes. Controlling for these demographic variables, PROCESS was used to examine mediational effects [35]. It provides alternatives to the causal steps approach [36] which has been criticized heavily in recent years [37]. In PROCESS, bias-corrected bootstrapping was used to generate confidence intervals which addressed the problem of power resulting from the asymmetric and non-normal sampling distributions of an indirect effect [38]. The bootstrapping sampling ($n = 1000$) distributions of the indirect effects were produced by selecting a sample of cases from the complete data set and calculating the indirect effects in the resamples. Point estimates and confidence intervals (95%) were estimated for the indirect effects. When zero was not contained in the confidence interval, point estimates of indirect effects were considered significant. Expectation Maximization (EM) algorithm [39] was used to replace the missing data. In the current study, less than 5% of responses were missing due to participants omitting questionnaire items. Regression imputation has been shown to be a valid method in dealing with missing data [40].

Results

Four hundred and seventy-five Kazakh students ($F = 336$, $M = 139$) responded to the online questionnaire. On average, they were 20 years old (mean = 19.90, $SD = 1.17$) and the majority were single (93%). Almost all (99%) were studying full-time for an undergraduate degree (98%) mainly in sciences (55%). Most were in the first three years of their studies (year 1 = 25%, year 2 = 39% and year 3 = 28%). A large proportion (71%) reported that they had experienced trauma, of whom 65% had experienced only one in their lifespan and the rest between 2 and 4. The most common event was personal assault (both physical and sexual) (35%), followed by child abuse (14%) and accident (10%). Using the diagnostic criteria of PDS-5, 39% ($n = 131$) of those who experienced trauma in the past met the criteria for full-PTSD.

Compared to the no-PTSD group, the PTSD group reported significantly higher levels of interpersonal sensitivity, psychiatric co-morbid symptoms and cognitive emotion regulation

strategies except positive reappraisal, positive refocusing and timidity. These results were based on the α level of 0.003 after Bonferroni correction to reduce the likelihood for Type I error (Table 1). Prior to the PROCESS analysis, bivariate analysis was used to establish whether demographic variables related to distress outcomes, since “victim variables” have been shown to influence distress outcomes [41, 42]. Since all were Kazakh in ethnicity, mostly single and studying an undergraduate degree full time, these variables were not entered into bivariate analysis. Age, gender, university major (dummy variable: science vs social sciences/humanities), academic year (dummy variable: junior: years 1 and 2, senior: years 3 and 4) and the number of traumas were subject to correlational analysis including point biserial correlations (r_{bp}). The results showed that age and university major were significantly correlated with interpersonal sensitivity (age: $r = -0.19$, $p < 0.001$; majors: $r_{bp} = -0.19$, $p < 0.001$) and psychiatric co-morbidity (age: $r = -0.14$, $p < 0.05$; majors: $r_{bp} = -0.30$, $p < 0.000$). They were entered into PROCESS analysis as co-variables.

Focusing on interpersonal sensitivity as the outcome variable, a significant direct effect of PTSD on interpersonal sensitivity was found (Effect = 0.14, ES = 0.04, $t = 2.90$, $p < 0.01$, LLCI: 0.046, ULCI: 0.239). PTSD and refocusing on planning were positively associated with the outcome, whilst positive reappraisal was negatively associated with it. No cognitive emotion regulation strategies mediated the impact of PTSD onto interpersonal sensitivity (see Table 2). Turning to psychiatric co-morbidity as the outcome variable, there was also a significant direct effect of PTSD on psychiatric co-morbidity (Effect = 0.16, ES = 0.04, $t = 3.76$, $p < 0.00$, LLCI: 0.080, ULCI: 0.256). PTSD, self-blame and putting the trauma into perspective were positively associated with the outcome. Self-blame was the only variable mediating the impact of PTSD on psychiatric co-morbidity (see Table 3).

Table 1 Means and standard deviations of psychiatric co-morbidity, cognitive emotion regulation strategies and interpersonal sensitivity

	PTSD		No-PTSD		t	Cohen's d
	Mean	SD	Mean	SD		
Somatic problems	16.05	3.76	12.95	3.67	7.31	0.83 ^b
Anxiety	16.16	4.24	12.60	4.09	7.49	0.85 ^b
Social dysfunction	15.64	3.10	14.08	1.91	5.06	0.60 ^b
Depression	14.52	4.58	10.39	3.06	8.88	1.06 ^b
Acceptance	6.68	3.03	5.48	3.34	3.27	0.37 ^b
Rumination	3.82	2.18	2.84	1.68	4.23	0.50 ^b
Positive reappraisal	6.84	2.76	6.32	2.69	1.66	0.19 ^c
Self-blame	5.23	2.26	4.40	2.47	2.99	0.35 ^a
Other-blame	3.40	1.70	2.54	1.28	4.78	0.57 ^b
Catastrophizing	4.66	2.50	2.87	1.65	7.01	0.84 ^b
Positive refocusing	6.50	2.75	7.33	2.73	-2.62	0.30 ^c
Refocusing on planning	6.51	2.59	5.57	2.06	3.36	0.40 ^b
Putting into perspective	6.31	2.70	5.00	2.05	4.58	0.54 ^b
Interpersonal awareness	18.73	3.65	15.49	3.51	7.95	0.90 ^b
Need for approval	23.27	3.58	24.51	3.57	-3.01	0.34 ^a
Separation anxiety	20.62	3.92	18.39	3.02	5.42	0.63 ^b
Timidity	21.06	4.14	20.17	3.78	1.97	0.22 ^c
Fragile self	11.48	3.43	8.20	2.86	8.91	1.03 ^b

$a = p < 0.003$; $b = p < 0.001$; $c = ns$

Discussion

The current study examined the relationship between PTSD from past trauma, interpersonal sensitivity and psychiatric co-morbidity, and the mediational effects of cognitive emotion regulation strategies on the relationship between PTSD and specific distress outcomes. In line with the first hypothesis, PTSD was associated with interpersonal sensitivity and psychiatric co-morbidity. The second hypothesis was partially supported in that although different cognitive emotion regulation strategies were associated with specific distress outcomes (positive reappraisal and refocusing on planning with interpersonal sensitivity; self-blame and putting the trauma into perspective with psychiatric co-morbidity), self-blame was the only item mediating the impact of PTSD onto psychiatric co-morbidity.

In line with literature, elevated PTSD was associated with increased interpersonal sensitivity and psychiatric co-morbidity e.g. [4, 11, 12, 14]. However, the number of traumas did not relate to distress outcomes, contrary to the literature emphasising the role of multiple-victimization [10, 17–19]. This could have been a sampling issue in that 65% of our samples experienced only one trauma.

Further analysis revealed that, with the exception of timidity, PTSD was correlated with all interpersonal sensitivity domains especially interpersonal awareness ($r=0.25$), and fragile inner self ($r=0.30$) at the α level of 0.001. These results might reflect the characteristics of a posttraumatic self. Trauma can affect the self-structure, ego-structure and identity processes of victims and thereby generate self-dissolution, feelings of separation, discontinuity, fragmentation and characteristics of a fragile inner-self, leading to reconfigurations of one's internal structural components. Trauma can also change self-monitoring processes including alteration in personal awareness, interpersonal sensitivity, hyperreactivity, as well as general sensitivity in social reaction. Sensitivity in perceiving abandonment by others has also been reported [43–45]. Interpersonal sensitivity problems co-existed with psychiatric co-morbid symptoms which was not surprising given that PTSD is not a discrete psychological syndrome but often expressed through other psychological symptoms [46].

Trauma can affect emotion regulation, another constituent of a posttraumatic self [44, 45] for which the current study provided further support. In line with the cognitive

Table 2 Direct and indirect effects of X (PTSD) on Y (Interpersonal sensitivity) with cognitive emotion regulation strategies as mediators

	Coeff	SE	t	LLCI	ULCI	Effect	Boot SE	Boot LLCI	Boot ULCI
PTSD	0.14	0.04	2.90 ^b	0.04	0.23	–	–	–	–
Acceptance	0.58	0.33	1.74 ^c	–0.07	1.25	–0.00	0.00	–0.02	0.01
Rumination	–0.86	0.49	–1.74 ^c	–1.84	0.10	–0.02	0.02	–0.07	0.00
Positive reappraisal	–1.06	0.49	–2.16 ^a	–2.02	–0.09	0.03	0.02	–0.00	0.08
Self-blame	0.00	0.37	0.01 ^c	–0.73	0.74	0.00	0.01	–0.02	0.02
Other-blame	0.25	0.52	0.47 ^c	–0.78	1.28	0.00	0.01	–0.02	0.04
Catastrophizing	0.42	0.48	0.88 ^c	–0.52	1.38	0.02	0.02	–0.03	0.07
Positive refocusing	–0.30	0.38	–0.79 ^c	–1.06	0.45	0.01	0.02	–0.03	0.07
Refocusing on planning	1.84	0.50	3.64 ^b	0.84	2.84	–0.01	0.01	–0.04	0.02
Putting into perspective	0.63	0.39	1.59 ^c	–0.14	1.41	–0.00	0.00	–0.01	0.01

$a = p < 0.05$; $b = p < 0.01$; $c = ns$

Table 3 Direct and indirect effects of X (PTSD) on Y (psychiatric co-morbidity) with cognitive emotion regulation strategies as mediators

	Coeff	SE	t	LLCI	ULCI	Effect	Boot SE	Boot LLCI	Boot ULCI
PTSD	0.16	0.04	3.76 ^a	0.08	0.25	–	–	–	–
Acceptance	–0.02	0.28	–0.08 ^b	–0.59	0.54	0.00	0.00	–0.01	0.00
Rumination	0.21	0.42	0.50 ^b	–0.62	1.06	0.00	0.02	–0.03	0.05
Positive reappraisal	–0.69	0.41	–1.65 ^b	–1.51	0.13	0.02	0.01	–0.00	0.06
Self-blame	1.16	0.32	3.54 ^a	0.51	1.80	0.04	0.01	0.01	0.08
Other-blame	–0.14	0.45	–0.31 ^b	–1.04	0.75	–0.00	0.01	–0.03	0.02
Catastrophizing	0.24	0.42	0.59 ^b	–0.58	1.08	0.01	0.02	–0.04	0.07
Positive refocusing	–0.58	0.33	–1.76 ^b	–1.23	0.06	0.03	0.02	–0.00	0.07
Refocusing on planning	–0.20	0.44	–0.46 ^b	–1.07	0.66	0.00	0.00	–0.01	0.01
Putting into perspective	1.47	0.34	4.26 ^a	0.79	2.15	0.00	0.01	–0.02	0.04

$a = p < 0.01$; $b = ns$

specificity hypothesis [28], content of thoughts characterized by positive reappraisal, refocusing on planning, self-blame and putting the trauma into perspective in this study related to specific mental health symptoms, thereby echoing existing literature [29–31]. Whilst the former two cognitive emotion regulation strategies were related to interpersonal sensitivity, the latter two related to general psychological disorder symptoms. Not one cognitive emotion regulation strategy was identified as a generic vulnerability strategy generating pervasive effects on different mental health domains.

Specifically, increased positive reappraisal buffered against the effect of interpersonal sensitivity rather than psychiatric co-morbid symptoms. This contradicted existing literature depicting the adaptive nature of this cognitive emotion regulation strategy for psychological disorder symptoms [23]. Also, positive reappraisal characteristics bear similarities to posttraumatic growth characteristics in that students in this study reported, to different degrees, that they had learned from their traumas (67%) and become a stronger person as a result (85%). These growth characteristics have been shown in literature to impact particularly on psychiatric co-morbid symptoms [47] although this has not been demonstrated in this study.

Somewhat unexpectedly, refocusing on planning, which is often considered an adaptive cognitive emotion regulation, was correlated with increased interpersonal sensitivity. When students thought about different ways of changing the trauma-related situation or a plan of how they could best deal with it, they tended to increase sensitivity. One possible explanation is that re-focusing on planning does not automatically translate into actual behaviour especially when the trauma is out of reach in terms of the possibility of change [48]. To refocus on planning would likely lead to continuous thoughts about what to do whilst knowing that actual changes would not likely occur. This feeling of entrapment or helplessness has been associated with psychological outcomes [49] manifested, in the current study, through heightened sensitivity particularly in social interaction with others.

In line with literature [22], self-blame was associated with increased psychiatric co-morbid symptoms. Self-blame implies a sense of regret. Over 60% of students reported different degrees of feeling responsible for what had happened to them or believing that the cause of the trauma must lie within themselves. They might have reflected on, examined and ruminated on their own behaviour and developed self-judgement. Such judgement

consequently led to trapped regretful moods which then contributed to depression, anxiety, somatization and poor psychological well-being [50, 51].

Playing down the seriousness of the trauma (putting the trauma into perspective) did not seem to buffer against psychological distress, supporting some of the findings in literature (e.g. [52]. Arguably, this was a form of emotion-focused coping in which students distanced themselves from the distress by, for example, making themselves believe that it had not been too bad compared to other events or that there were worse things in life. However, emotion-focused coping tends to be endorsed by those who have high levels of PTSD and anxiety [53, 54]. Patients who had a high level of PTSD following life threatening illness [30, 55], for example, tended to use a great deal of emotion-focused coping and at the same time report elevated psychiatric symptom severity. On the other hand, reduced emotion-focused coping is associated with reduced psychiatric symptoms and general psychological distress [56].

Apart from self-blame, cognitive emotion regulation strategies did not mediate the impact of PTSD onto distress outcomes. Contrary to literature, catastrophizing [26] and rumination of negative emotions [27] have not been found as mediators. In other words, PTSD and cognitive emotion regulation strategies mostly affected interpersonal sensitivity or general psychological disorder symptoms directly. They generated additive rather than mediational effects. Nevertheless, the idea that trauma can exacerbate psychiatric co-morbid symptoms through changes in emotional regulation was not entirely refuted. Trauma was indeed related to increased psychiatric co-morbidity through changes in emotional regulation but a specific kind, namely, self-blame. Seemingly, blaming oneself for the trauma has a unique role to play in mitigating the impact of trauma onto psychiatric co-morbid symptoms. Perhaps this group of students expressed trauma reactions by internalizing the reasons or consequences of the trauma [57].

Several limitations of the research need to be acknowledged. The effect of cumulative trauma [10, 19] onto distress outcomes needs to be investigated further. Effort should have been made to increase the sample size for individuals who have experienced more than one trauma. Secondly, the cultural characteristics among these Kazakh students should have been examined. These additional data could have provided information on the results on cognitive emotional regulation and distress outcomes. This study was based on a cross-sectional design which yielded bias in mediational analysis due to the lack of temporal precedence [58]. Our interpretation of these results should focus primarily on indirect effects (i.e. the structural relationship of the model) rather than causality inference [59].

To conclude, following trauma, sensitivity in interacting with others can be heightened and psychological symptoms increased among students. Psychological symptoms are related to specific thought processes about the trauma. Additionally, students who blame themselves for trauma can influence the direct impact of the trauma onto the severity of psychological symptoms.

Compliance with Ethical Standards

Conflict of Interest All authors declare that they have no conflicts of interest.

Ethical Approval All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all participants included in the study.

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