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RESEARCH ARTICLE

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Changes in intolerance of uncertainty over the course of treatment predict posttraumatic stress disorder symptoms in an inpatient sample

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Abstract

Intolerance of uncertainty (IU) is the inability to tolerate distress that arises in response to the absence of important information. The level of IU has been investigated across various psychological disorders; however, few studies have examined IU in trauma-affected samples. We aimed to investigate the relationship between IU and posttraumatic stress disorder (PTSD) across the course of treatment. Participants ($n = 106$) had a diagnosis of PTSD and were from first responder, military, and occupational injury backgrounds. Participants completed self-report questionnaires pre- and post-engagement in an inpatient group trauma-informed psychoeducation and skills (TIPS) intervention. Regression analyses indicated that decreases in overall and inhibitory IU were associated with decreases in PTSD severity overall and at the symptom cluster level. However, prospective IU was only associated with changes in the re-experiencing, avoidance, and arousal PTSD symptom clusters. Our findings are congruent with the nascent literature indicating that IU may be a maintaining factor for PTSD, suggesting clinical relevance for attendance to IU within the course of treatment.

KEYWORDS
inpatient, intolerance of uncertainty, occupational trauma, posttraumatic stress disorder, psychoeducation, treatment

1 | INTRODUCTION

Difficulty tolerating uncertainty has been implicated as a trans-diagnostic factor across a range of anxious and depressive psychopathologies (Carleton, 2016b). Intolerance of Uncertainty (IU) has been defined as '… an individual's dispositional incapacity to endure the aversive response triggered by the perceived absence of salient, key, or sufficient information, and sustained by the associated perception of uncertainty' (Carleton, 2016b, p. 31). As indicated by confirmatory factor analyses, the construct of IU is composed of two latent factors, Prospective IU and Inhibitory IU (Hong & Lee, 2015). Prospective IU focuses on cognitive distress associated with uncertainty about future events, whereas inhibitory IU emphasizes behavioural impediments in functioning arising in response to uncertainty (Carleton et al., 2007).

According to the Uncertainty and Anticipation Model of Anxiety (Grupe & Nitschke, 2013), high levels of IU are associated with overestimation of threat outcomes, increased levels of vigilance and

avoidance, impaired learning in regard to safety, and over-responsiveness to uncertainty. As such, IU could conceivably maintain the sense of threat and impending danger that characterizes Posttraumatic Stress Disorder (PTSD). In this respect, IU might be especially associated with elevated arousal and threat-related symptoms, but less so with mood and cognition symptoms, which may be less directly linked with perceived threat. An association with arousal related symptoms might in turn contribute to associations between IU and PTSD overall.

In Pole et al.'s (2008) prospective study of police during the first year of service, physiological markers of elevated levels of fear in response to low levels of threat were the strongest predictor of PTSD 1 year following spontaneous episodes of distressing work-related incidents. In a large-scale study involving personnel likely to be exposed to occupationally distressing events such as paramedics, fire fighters, and police officers, higher levels of IU were reported for personnel who screened positively for mental disorder, and, more specifically, IU was shown to account for significant variance in PTSD levels (Angehrn et al., 2020).

The extent to which IU may also be a marker of risk in PTSD is uncertain. The few studies that have investigated IU and PTSD have reported mixed findings. In an undergraduate sample of students who had experienced a traumatic event, Bardeen et al. (2013) reported no main effect between IU and traumatic stress symptoms, but there was evidence of an interaction effect with high levels of IU and worry in combination associated with traumatic stress symptoms and the arousal symptom cluster specifically. In a trauma-exposed community sample, Fetzner et al. (2013) reported that inhibitory IU accounted for significant variance in the avoidance, numbing, and arousal clusters, but not the re-experiencing cluster. However, prospective IU did not contribute to variance in any of the symptom clusters.

Amongst a prospective sample of undergraduate students who were exposed to a campus shooting (Oglesby et al., 2016), pre-event IU was positively associated with the arousal and re-experiencing clusters of PTSD. However, in a later study involving trauma-exposed individuals in the community (Oglesby et al., 2017), IU was associated with the arousal, avoidance, and numbing clusters, but not with the re-experiencing cluster when covariance of negative affect and anxiety sensitivity were accounted for. Boelen's (2019) prospective study showed that Inhibitory IU was associated with analogue traumatic stress symptoms in a student sample; however, IU as a single construct was not investigated in this study. These indeterminate outcomes highlight the need for additional research into the relationship between IU and PTSD, in order to inform clinical treatment.

A number of studies have also investigated the associations between IU and PTSD symptoms in clinical PTSD samples. Hollingsworth et al. (2018) investigated the role of IU as a mediator between depressive and posttraumatic stress symptoms in a sample of 113 African American veterans who were receiving treatment for PTSD. IU was found to mediate the relationship between depression and PTSD overall, as well as the PTSD symptom clusters of arousal, numbness, and avoidance. Consistent with Oglesby et al.'s (2017) civilian study, IU was not specifically associated with the re-experiencing symptom cluster. In their study of 191 Israeli combat veterans, which focused on moderators of moral injury, self-harm, and suicidal ideation, Zerach and Levi-Belz (2019) showed that inhibitory IU was correlated with traumatic stress symptoms.

Recently, Raines et al. (2019) conducted a study that investigated IU in a sample of veterans who were receiving treatment for PTSD. The 116 participants completed self-report measures as part of the intake process. After controlling for depression and anxiety sensitivity, IU was shown to be associated with PTSD severity overall, as well as the arousal and reactivity clusters, and avoidance symptoms. IU was not associated with re-experiencing symptoms and, in contrast to previous studies with PTSD clinical samples, was not associated with the negative mood and cognitions cluster. In additional analyses, Raines et al. (2019) also analysed the relationship between the IU subscales (prospective and inhibitory IU) and PTSD. Prospective IU was associated with overall PTSD severity and the arousal cluster only, whereas Inhibitory IU was not a unique predictor for PTSD overall or any of the symptom clusters. However, these associations were only investigated at pre-treatment, leaving open the question of whether IU has predictive value in relation to PTSD symptoms *across* treatment.

These initial studies with clinical PTSD samples provide an important platform from which IU research can be extended. The cross-sectional nature of past studies with clinical samples precludes understanding of the associations between changes in IU and PTSD severity over the course of treatment. As few studies have investigated IU in trauma-exposed samples, generalization of findings to people in varying roles that involve high levels of exposure to trauma events such as police, paramedics, and fire fighters may be limited. Given that only a single study has investigated IU as a two-factor construct with regard to people seeking treatment for PTSD (Raines et al., 2019), further studies are needed to clarify how each domain of IU might best be conceptualized in relation to PTSD. So far as PTSD symptoms are concerned, previous research has reported inconsistent relationships between IU domains and PTSD symptom clusters, underlining the

Key Practitioner Messages

- IU appears to be a transdiagnostic factor across psychopathologies including PTSD.
- Psychoeducation and skills building interventions that include cognitive and behavioural strategies may be of benefit in decreasing levels of IU and, subsequently, PTSD, at the overall and symptom cluster levels.
- Our intervention did not specifically address IU, leaving scope for further improvements in PTSD symptoms with the inclusion of IU-specific strategies.
- Group interventions that incorporate psychoeducation and skills building may be of benefit in providing cost-effective, accessible support following trauma exposure for individuals who are waitlisted for individual treatment.

importance of examining the relationship between IU and individual PTSD symptom clusters. As such, we conducted a repeated-measures study in a diverse sample that included first responders, defence personnel, and occupationally injured participants over the course of an inpatient group trauma-informed psychoeducation and skills (TIPS) intervention. IU was analysed as single and two-factor construct in relation to PTSD total score and symptom cluster scores.

Numerous psychological therapies have the objective of improving a person's capacity to respond to threat and uncertainty (Carleton, 2012; Clark & Beck, 2010). We therefore anticipated that the TIPS intervention would produce reductions in IU based on its components of psychoeducation for trauma and strategies for recovery consistent with cognitive behaviour therapy principles. These strategies included management of negative self-talk using cognitive challenging, generation of an exposure hierarchy for avoided situations that are not necessarily directly associated with the trauma events, and implementation of adaptive coping strategies.

Extending from previous cross-sectional studies, we hypothesized that decreases in total IU would be associated with decreases in overall PTSD severity, and the avoidance, and arousal symptom clusters that comprise PTSD. An a priori prediction was not made with regard to IU and the re-experiencing cluster given mixed previous findings. In regard to the negative mood and cognitions cluster, no a priori hypothesis was made noting that few past studies have utilized a measure that included this subscale.

Although analyses by Hong and Lee (2015) have demonstrated support for IU as a two-factor construct, assessment of IU in an Italian sample indicated only a unidimensional factor structure (Bottesi et al., 2019). However, Bottesi and colleagues (2019) cited cross-cultural considerations that may have impacted on respondent's endorsement levels for some of the IU items. We therefore assessed IU as a single- and two-factor construct over the course of the intervention to extend on previous research (e.g., Raines et al., 2019) in the PTSD arena and to assist with ascertaining the appropriate clinical utility of this measure in the development of interventions follow trauma exposure. Due to the limited literature regarding prospective and inhibitory IU, a priori hypotheses were not specified.

2 | METHOD

2.1 | Participants

Participants were 123 clinical inpatients undertaking a routine TIPS intervention for PTSD at a private mental health hospital over a 3-week period. Participants were from first responder, military, and community backgrounds and required to be (i) aged 18-years or over, (ii) have experienced the index trauma in adulthood, and (iii) be English speakers. Participants were required to have a diagnosis of PTSD as per the referral from their treating psychiatrist. Participants did not receive any compensation for participation in the study.

Exclusion criteria were consistent with those stipulated by the hospital for patients attending the group PTSD intervention and included current psychosis, chronic psychotic illness, active self-harm, severe dissociative episodes, or substance detox, instability, or misuse (including alcohol, opioids, buprenorphine/naloxone, and methadone).

Ethical approval was obtained from the Human Research Ethics Committee at the University of Technology Sydney (reference number: ETH17-1665) and the Departments of Defence and Veterans' Affairs Human Research Ethics Committee (reference number: 036-18).

2.2 | Design

After providing informed consent, participants completed measures as part of routine care on Days 1 and 20 of the TIPS intervention, with additional study-specific measures also administered at this time (demographic information, intolerance of uncertainty, rumination, and symptom perception).

2.3 | Self-report measures

2.3.1 | Demographics

A questionnaire was used to obtain participant demographics of age, gender, main occupation, first language, medication use, and physical health ailments on Day 1 only.

2.3.2 | Posttraumatic stress disorder checklist for DSM-5 (PCL-5)

The PCL-5 (Weathers et al., 2013) is a 20-item questionnaire that provides an overall severity rating and also measures criterion cluster severity using a five-point Likert scale (0 = *Not at all* to 4 = *Extremely*). Bovin et al. (2015) previously reported strong reliability for this scale (Cronbach's $\alpha = .93$). In the current study, strong internal consistency using Cronbach's α was indicated for the PCL-5 scores overall (pre = .84, post = .88).

2.3.3 | Intolerance of uncertainty—Short form (IUS-12)

The 12-item IUS-12 (Carleton et al., 2007) measures the predisposition toward distress in response to ambiguous stimuli. The subscales are reflective of approach-oriented (prospective) and avoidance (inhibitory) behaviours (Birrell et al., 2011). The IUS-12 is strongly correlated ($r = .96$) with the original 27-item scale (Freeston et al., 1994) but demonstrated greater divergent validity from the construct of worry than the original scale (Carleton et al., 2007).

Jacoby et al. (2013) have shown confirmation of the two-factor structure for IU, and their study also indicated high reliability scores for the overall and dimensional factors (IUS-12, $\alpha = .93$; IUS-Prospective [IUS-P], $\alpha = .90$; IUS-Inhibitory [IUS-I], $\alpha = .90$). Confirmatory factor

analyses have demonstrated superiority for the two-factor model based on the 12-item IU scale as compared to the 18- and 27-item scales (Hong & Lee, 2015). Participants rated each item on a 5-point Likert scale (1 = *not at all characteristic of me* to 5 = *entirely characteristic of me*). The current study also yielded high levels of internal consistency using Cronbach's α for total IU (pre = .88, post = .90), prospective IU (pre = .81, post = .83), and inhibitory IU (pre and post = .85).

2.3.4 | Hospital anxiety and depression scale (HADS)

The 14-item HADS (Zigmond & Snaith, 1983) was developed in a hospital setting to measure anxiety and depression. Participants rated each item on a 4-point scale (0 to 3). In a review of 747 studies, mean Cronbach α subscale scores for the HADS ranged from .68 to .93 (*m* = .83) for anxiety and .67 to .90 (*m* = .82) for depression (Bjelland et al., 2002). In the current study, Cronbach's α scores were similar for anxiety (pre = .78, post = .86) and depression (pre = .67, post = .75).

2.4 | Statistical analysis

Data were analysed using the IBM Statistical Package for Social Sciences (SPSS) Version 27. A non-directional alpha level of .05 was used for all statistical tests. Independent samples *t* tests were used to compare pre- and post-treatment, and differences scores by gender. Differences in IU, PTSD, anxiety, and depression severity over the course of treatment were calculated by subtracting the pre-treatment scores from the post-treatment scores. A regression model applying the Method of First Differences approach (Liker et al., 1985) was then used to determine whether changes in IU were associated with changes in PTSD, at the overall and subscale levels, with anxiety and depression difference scores, along with gender accounted for in each model. Effects sizes were calculated using the standard deviation of the mean difference and are reported as Cohen's *d*. Outcomes for rumination and symptom perception were also collected but are beyond the scope of this paper and will be reported elsewhere.

3 | RESULTS

3.1 | Sample and demographic information

Initial data screening showed that 17 participants did not complete the pre- or post-PCL, IU, or HADS measures. As these were the main variables under investigation, data for these participants were not included in further analyses. The final sample consisted of 106 participants: 57 males aged 27–69 years (M_{age} = 47 years, 3 months, *SD* = 9 years, 4 months) and 49 females aged 19–70 years (M_{age} = 42 years, 6 months, *SD* = 12 years, 0 months).

Participant backgrounds were police (22.2%), defence (17.6%), legal/health/teaching, (14.8%), home duties/retired/unemployed (12.0%), administration/business (9.3%), fire fighter/paramedic (7.4%), other (10.2%), and unspecified (6.5%). Almost all participants (*n* = 104) reported English as their first language. The majority of participants reported medication use for PTSD, depression, and/or anxiety (97%). Detailed medication use data, which were available for 85 participants, indicated that few participants had commenced medication in the month preceding, or at the time of starting, the intervention (6 months +, *n* = 50; 3–6 months, *n* = 6; 1–3 months, *n* = 21; <1 month, *n* = 8).

3.2 | Descriptive and preliminary analysis

Decreases were reported over the course of treatment for all IUS-12 and PCL-5 totals, subscales and clusters. All correlations at pre-treatment were significant between IUS-12, IUS-P, and IUS-I difference scores and

TABLE 1 Difference scores pre- to post-treatment: Zero order correlations, means, and standard deviations

		1	2	3	4	5	6	7	8	9	10
1	PCL-5 Total	1									
2	IU Total	.37**	1								
3	PCL_Re-experiencing	.76**	.34**	1							
4	PCL_Avoidance	.66**	.24*	.43**	1						
5	PCL_Neg Mood & Cognitions	.89**	.25*	.56**	.54**	1					
6	PCL_Arousal	.84**	.35**	.44**	.46**	.64**	1				
7	IU_Prospective	.28**	.85**	.26**	.20*	0.18	.26**	1			
8	IU_Inhibitory	.35**	.88**	.32**	.22*	.24*	.34**	.51**	1		
9	HADS_Anxiety	.64**	.25**	.49**	.49**	.57**	.52**	.22*	.22*	1	
10	HADS_Depression	.61**	.32**	.47**	.50**	.52**	.49**	.21*	.34**	.61**	1
	Mean	−8.41	−2.25	−1.33	−1.12	−2.48	−3.47	−1.02	−1.23	−1.43	−2.05
	SD	12.72	5.96	3.85	1.79	5.23	4.73	3.24	3.62	3.77	3.53

Note: *n* = 106.
*p < .05.
**p < 0.01.

TABLE 2 Regression outcomes by independent variable

	B	SE	β	*t*	CI 95%		*B*	SE	β	*t*	CI 95%		*B*	SE	β	*t*	CI 95%
Models 1, 2, 3: PCL-5 total score (DV)																	
IUS-total						IUS-prospective						IUS-inhibitory					
IUS-12*	0.35	0.15	0.16	2.26	0.04, .65	IUS-P	0.43	0.28	0.11	1.52	−0.13, 0.99	IUS-I*	0.6	0.26	0.17	2.35	0,09, 1.10
HADS-A***	1.34	0.29	0.4	4.58	0.76, 1.92	HADS-A***	1.34	0.3	0.4	4.47	0.74, 1.93	HADS-A***	1.38	0.29	0.41	4.74	0.81, 1.96
HADS-D**	1	0.32	0.28	3.13	0.36, 1.63	HADS-D**	1.11	0.32	0.31	3.51	0.48, 1.74	HADS-D**	0.94	0.32	0.26	2.92	0.30, 1.58
Gender*	−4.3	1.82	−0.17	−2.37	−7.90, −6.9	Gender*	−4.24	1.84	−0.17	−2.31	−7.9, −0.59	Gender*	−4.41	1.81	−0.17	−2.43	−8.00, −0.81
Models 4, 5, 6: PCL-5 re-experiencing score (DV)																	
IUS-12*	0.12	0.06	0.19	2.19	0.01, 0.23	IUS-P	0.16	0.1	0.13	1.57	−0.04, 0.36	IUS-I*	0.2	0.09	0.19	2.17	0.02, 0.38
HADS-A*	0.27	0.1	0.26	2.58	0.06, 0.48	HADS-A*	0.27	0.11	0.26	2.51	0.06, 0.48	HADS-A**	0.28	0.1	0.28	2.78	0.08, 0.49
HADS-D*	0.23	0.11	0.21	2.02	0.00, 0.45	HADS-D*	0.27	0.11	0.24	2.38	0.04, 0.49	HADS-D	0.21	0.12	0.2	1.86	−0.02, 0.44
Gender**	−1.76	0.65	−0.23	−2.73	−3.04, −.48	Gender**	−1.74	0.65	−0.28	−2.67	−3.04, −0.45	Gender**	−1.8	0.65	−0.23	−2.79	−3.08, −0.52
Models 7, 8, 9: PCL-5 avoidance score (DV)																	
IUS-12	0.02	0.03	0.07	0.83	−0.03, 0.07	IUS-P	0.04	0.05	0.07	0.79	−0.06, 0.13	IUS-I	0.03	0.04	0.06	0.65	−0.06, 0.11
HADS-A*	0.13	0.05	0.27	2.59	0.03, 0.23	HADS-A*	0.13	0.05	0.27	2.54	0.03, 0.23	HADS-A*	0.13	0.05	0.28	2.64	0.03, 0.23
HADS-D*	0.14	0.05	0.28	2.64	0.04, 0.25	HADS-D**	0.15	0.05	0.29	2.8	0.04, 0.25	HADS-D*	0.14	0.06	0.28	2.59	0.03, 0.25
Gender	−0.53	0.31	−0.15	−1.71	−1.14, 0.09	Gender	−0.52	0.31	−0.15	−1.68	−1.13, 0.09	Gender	−0.53	0.31	−0.15	−1.72	−1.14, 0.08
Models 10, 11, 12: PCL-5 negative mood and cognitions score (DV)																	
IUS-12	0.05	0.07	0.06	0.73	−0.09, .20	IUS-P	0.04	0.13	0.02	0.3	−0.22. 0.30	IUS-I	0.11	0.12	0.08	0.94	−0.12, 0.35
HADS-A***	0.53	0.14	0.38	3.88	0.26, 0.80	HADS-A*	0.53	0.14	0.39	3.87	0.26, 0.81	HADS-A***	0.54	0.14	0.39	3.94	0.27, 0.81
HADS-D*	0.34	0.15	0.23	2.29	0.05, 0.63	HADS-D**	0.36	0.15	0.24	2.46	0.07, 0.65	HADS-D*	0.32	0.15	0.22	2.15	0.03, 0.62
Gender	−1.63	0.85	−0.16	−1.92	−3.31, .06	Gender	−1.62	0.85	−1.6	−1.91	−3.31, 0.06	Gender	−1.64	0.85	−0.16	−1.94	−3.32, 0.03
Models 13, 14, 15: PCL-5 arousal score (DV)																	
IUS-12*	0.15	0.07	0.19	2.28	0.02, 0.29	IUS-P	0.2	0.12	0.13	1.57	−0.05, .44	IUS-I*	0.26	0.11	0.2	2.32	0.04, 0.48
HADS-A**	0.42	0.13	0.33	3.21	0.16, 0.67	HADS-A*	0.41	0.13	0.33	3.13	0.15, 0.67	HADS-A***	0.43	0.13	0.35	3.37	0.18, 0.69
HADS-D*	0.29	0.14	0.21	2.04	0.00, 0.56	HADS-D**	0.34	0.14	0.25	2.41	0.06, 0.61	HADS-D	0.26	0.14	0.2	1.85	−0.02, 0.55
Gender	−0.38	0.8	−0.04	−0.48	−1.97, 1.21	Gender	−0.36	0.81	−0.04	−0.44	−1.97, 1.25	Gender	−0.43	0.8	−0.05	−0.54	−2.02, 1.16

Note. $n = 106$.Gender: 0 = male, 1 = female.

Abbreviations: CI, confidence interval; DV, dependent variable; HADS-A, anxiety total score; HADS-D, depression total score; IUS-12, IUS total score; IUS-I, IUS inhibitory dimension; IUS-P, IUS prospective dimension.

*$p < .05$.

**$p < 0.01$.

***$p < .001$.

the PCL-5 total and subscale scores, except for the correlation between the IUS-P and PCL-5 negative mood and cognitions cluster. Bivariate correlations, means, and standard deviations are shown in Table 1.

It was noted that females scored greater on the majority of self-report measures at pre-treatment and for pre- to post-treatment changes (see Table S1 for summary). No between-gender differences were reported post-treatment.

3.3 | Primary analysis

Detailed outcomes of the regressions are shown in Table 2.

3.3.1 | PTSD total scores

The first regression examined the association between total IU and overall PTSD severity. The model was significant (F [5, 100] = 23.26, p < .001), with 53.8% of the variance in PTSD severity accounted for by IU, gender, anxiety, and depression. Consistent with our hypothesis, decreases in overall IU from pre- to post-treatment were associated with decreases in PTSD scores over the course of the TIPS intervention ($\beta = .16$, $t = 2.26$, 95% CI [.04, .65]).

The second regression examined the association between prospective IU and PTSD severity overall. The model was significant (F [5, 100] = 22.10, p < .001), with 52.5% of the variance in PTSD severity accounted for by gender, anxiety, and depression, but not by prospective IU (p > .05).

The third regression examined the association between inhibitory IU and PTSD severity overall. The model was significant (F [5, 100] = 23.42, p < .001), with 53.9% of the variance in PTSD severity accounted for by inhibitory IU, gender, anxiety, and depression. Decreases in inhibitory IU from pre-to post-treatment were associated with decreases in PTSD scores over the course of the intervention ($\beta = .17$, $t = 2.35$, 95% CI [.09, 1.10]).

3.3.2 | PTSD re-experiencing symptoms

The fourth regression examined the association between total IU and PTSD re-experiencing symptoms. The model was significant (F [5, 100] = 11.48, p < .001), with 36.5% of the variance in PTSD severity accounted for by IU, gender, anxiety, and depression. Decreases in overall IU from pre- to post-treatment were associated with decreases in PTSD re-experiencing scores over the course of the intervention ($\beta = .19$, $t = 2.19$, 95% CI [.01, .23]).

The fifth regression examined the association between prospective IU and PTSD re-experiencing symptoms. The model was significant (F [5, 100] = 10.79 p < .001), with 35% of the variance in PTSD severity accounted for by gender, anxiety, and depression, but not by prospective IU (p > .05).

The sixth regression examined the association between inhibitory IU and PTSD re-experiencing symptoms. The model was significant (F [5, 100] = 11.46, p < .001), with 36.4% of the variance in PTSD severity accounted for by inhibitory IU, gender, and anxiety. Decreases in inhibitory IU from pre- to post-treatment were associated with decreases in PTSD scores over the course of the intervention ($\beta = .19$, $t = 2.17$, 95% CI [.02, .38]).

3.3.3 | PTSD avoidance symptoms

The seventh regression examined the association between total IU and PTSD avoidance symptoms. The model was significant (F [5, 100] = 9.77, p < .001), with 32.8% of the variance in PTSD severity accounted for by anxiety and depression. Decreases in overall IU were not associated with decreases in PTSD avoidance scores over the course of the intervention (p > .05), which was inconsistent with our hypothesis.

The eighth regression examined the association between prospective IU and PTSD avoidance symptoms. The model was significant (F [5, 100] = 9.78 p < .001), with 32.8% of the variance in PTSD severity accounted for by anxiety and depression, but not by prospective IU (p > .05).

The ninth regression examined the association between inhibitory IU and PTSD avoidance symptoms. The model was significant (F [5, 100] = 9.69, p < .001), with 32.6% of the variance in PTSD severity accounted for by anxiety and depression, but not by inhibitory IU (p > .05).

3.3.4 | PTSD negative mood and cognition symptoms

The tenth regression examined the association between total IU and PTSD negative mood and cognition symptoms. The model was significant (F [5, 100] = 13.46, p < .001), with 40.2% of the variance in PTSD severity accounted for by anxiety and depression. Decreases in overall IU were not associated with decreases in PTSD negative mood and cognition scores over the course of the intervention ($p = .47$), which was inconsistent with our hypothesis.

The eleventh regression examined the association between prospective IU and PTSD negative mood and cognition symptoms. The model was significant (F [5, 100] = 13.31 p < .001), with 40% of the variance in PTSD severity accounted for by anxiety and depression, but not by prospective IU (p > .05).

The twelfth regression examined the association between inhibitory IU and PTSD negative mood and cognition symptoms. The model was significant (F [5, 100] = 13.58, p < .001), with 40.4% of the variance in PTSD severity accounted for by anxiety and depression, but not by inhibitory IU (p > .05).

3.3.5 | PTSD arousal symptoms

The thirteenth regression examined the association between total IU and PTSD arousal symptoms. The model was significant (F [5, 100]

= 10.78, $p < .001$), with 35% of the variance in PTSD severity accounted for by total IU, anxiety, and depression. Consistent with our hypothesis, decreases in overall IU were associated with decreases in PTSD arousal scores over the course of the intervention ($\beta = 19$, $t = 2.28$, 95% CI [.02, .29]).

The fourteenth regression examined the association between prospective IU and PTSD arousal symptoms. The model was significant (F [5, 100] = 9.98 $p < .001$), with 33.3% of the variance in PTSD severity accounted for by anxiety and depression, but not by prospective IU ($p > .05$).

The fifteenth regression examined the association between inhibitory IU and PTSD arousal symptoms. The model was significant (F [5, 100] = 10.84, $p < .001$), with 35.1% of the variance in PTSD severity accounted for by inhibitory IU and anxiety. Decreases in inhibitory IU from pre- to post-treatment were associated with decreases in PTSD scores over the course of the intervention ($\beta = .20$, $t = 2.32$, 95% CI [.04, .48]).

4 | DISCUSSION

Decreased propensity to tolerate uncertainty may play an important role in management of PTSD symptoms. The present study investigated the associations between IU and PTSD symptoms over the course of an inpatient TIPS intervention. In particular, this study examined the relationship between changes in IU as a single and two-factor construct, with changes in PTSD symptom severity overall and at symptom cluster levels, from pre- to post-treatment for a trauma-exposed sample that included first responders, military personnel, and community members presenting with heterogeneous trauma histories.

Consistent with our prediction, lower IU was associated with higher post-treatment PTSD symptom severity change. Changes in prospective and inhibitory IU were also shown to predict post-treatment PTSD levels. These findings are broadly consistent with past findings from IU research. For example, Raines et al. (2019) reported an association between IU and PTSD severity; however, their study indicated that prospective IU, but not inhibitory IU, uniquely predicted PTSD severity. Oglesby et al. (2017) reported associations between IU and symptoms of posttraumatic stress in a trauma-exposed sample, although the study did not include assessment of IU as a two-factor construct. Our findings contribute to the burgeoning body of literature demonstrating the role of IU in PTSD maintenance and the broader literature indicating the transdiagnostic nature of IU across various psychopathologies (Angehrn et al., 2020; Carleton, 2016a).

At the PTSD re-experiencing cluster level, changes in total and inhibitory IU were associated with post-treatment re-experiencing cluster severity, but prospective IU was not. A priori hypotheses had not been specified given the mixed findings in previous studies. In the present study, the association between changes in total IU and the post-treatment re-experiencing cluster was in line with Oglesby et al.'s (2016) non-clinical prospective study; however, these authors reported associations between IU prior to exposure to a trauma event and the subsequent re-experiencing cluster. Our sample varied in that participants already had a DSM-5 (APA, 2013) diagnosis of PTSD when completing IU measures, making direct comparisons to Oglesby et al.'s study difficult.

In studies that controlled for anxiety sensitivity and negative affect (Oglesby et al., 2017; Raines et al., 2019), IU did not predict the re-experiencing cluster of symptoms. Although our findings in regard to inhibitory IU are consistent with these studies in that they did not predict re-experiencing, we reported differences in relation to the re-experiencing cluster and both total and prospective IU. Given the cross-sectional design of these earlier studies, and our focus on changes in IU across treatment, direct comparison is again difficult. Intrusive symptoms involving perceived threat and danger tend to characterize PTSD, with prospective studies showing that decreases in intrusive symptoms predict decreases in overall symptom severity (Solberg et al., 2016), particularly those that arise in the early period after exposure to a trauma event (Bryant et al., 2017). As such, affecting change in IU many confer important changes to re-experiencing symptoms and therefore influence overall symptom severity.

Our hypothesis that changes in overall IU would be associated with changes in the avoidance cluster of PTSD symptoms was not supported. Our findings are contrary to previous research showing positive associations between overall IU and the avoidance cluster of PTSD symptoms (Fetzner et al., 2013; Oglesby et al., 2017; Raines et al., 2019). However, we note that these studies were of a cross-sectional or non-clinical nature rather than assessing for decreases over time. Although decreases in prospective and inhibitory IU were reported from pre- to post-treatment in the current study, associations with differences in the avoidance cluster were not significant and are in line with previous research (Raines et al., 2019). Changes in the avoidance cluster were partially accounted for by anxiety and depression, lending further support to the premise of IU and anxiety as divergent constructs (Carleton et al., 2007).

With regard to the negative mood and cognitions cluster, neither overall, prospective nor inhibitory IU was associated with changes in this PTSD cluster. Our findings are consistent with those reported in a veteran sample (Raines et al., 2019). Although previous studies have shown associations between IU and numbing scores (Fetzner et al., 2013) and IU and negative affect in relation to traumatic stress symptoms (Oglesby et al., 2017), it is possible that the measures used in previous studies focused more on the affect aspect rather than a combination of mood and cognitions as per the PCL-5 measure used in this study. Further, the profile of the participants in the current study varied from studies utilizing community samples, as participants were more likely to have been exposed to occupational trauma events on multiple occasions.

Findings that changes in overall IU were associated with differences in the arousal cluster of PTSD symptoms were consistent with our prediction and previous research (Fetzner et al., 2013; Oglesby et al., 2017; Raines et al., 2019). Decreases in inhibitory, but not prospective, IU were also associated with changes in PTSD arousal cluster scores. Distress related to uncertainty may render an individual more likely to engage in hypervigilance, startle patterns, and sleeplessness as means of pre-empting future stressors. Given the mental, emotional, and physical toll that a constant heightened states takes on

individuals, targeting IU to assist with reducing arousal and reactivity would likely be of benefit in equipping people exposed to trauma to manage in their daily environment.

4.1 | Limitations

Limitations of the current study should be considered when interpreting findings and in future investigations. First, participants presenting for treatment were from a range of backgrounds, making comparisons with previous studies difficult. Although use of a heterogeneous sample this may assist with generalizability of the findings, future studies may also seek to assess changes in IU over the course of treatment in homogeneous samples to assist with elucidating whether IU profiles differ between civilian and non-civilian trauma-exposed groups, given findings by Kelley et al. (2009) showing different profile patterns in civilian trauma.

Second, our study was limited by the reliance on self-report measures. Given the nature of IU and PTSD symptoms, there is dependence on participants to provide accurate information. Although validated measures were used to assess IU (Carleton et al., 2007) and PTSD (Weathers et al., 2013) symptoms, multi-method assessment may be beneficial in future studies. For instance, assessments that capture information from family members about the person's IU tendencies would allow the convergent validity of IU self-report measures to be determined. For PTSD symptoms, additional indices may include behavioural observation and measurement of biological stress markers in the presence of novel stimuli.

Further limitations present in relation to two of the measures used. Despite the widespread use of the HADS (Zigmond & Snaith, 1983) as a measure for depression, the questions focus on the depressive symptoms consistent with anhedonia. As such, future studies may seek to utilize a measure that more broadly captures the varying facets that comprise depression. In relation to the IUS-12 (Carleton et al., 2007), the literature indicates mixed findings as to whether IU should be measured as a single (Bottesi et al., 2019) or two-factor (Hong & Lee, 2015) construct. From a transdiagnostic perspective, general IU appears to have greater clinical utility (Shihata et al., 2018). Although we have reported on IU at the overall and subscale levels, we recommend that the results with regard to the prospective and inhibitory subscales be interpreted with caution, particularly given the limited literature available for clinical PTSD samples.

Lastly, we note that the majority of participants were taking medication for PTSD, depression, and/or anxiety while concurrently engaged in the group intervention. Although it is likely that people attending on an inpatient basis for PTSD treatment will be utilizing medication given the degree of distress they are presenting with, inferences about changes in PTSD symptoms arising due to the group intervention should be interpreted with caution. However, we note that few participants in the study had been on psychotropic medication for less than a month prior to commencing the intervention, suggesting that the effects of medication were likely to have stabilized for the majority of participants.

4.2 | Conclusion

Our study adds to a growing body of literature focused on the relationship between IU and PTSD. In particular, the assessment of changes in IU in relation to PTSD severity across treatment demonstrates the moderate association between IU and PTSD clinical outcomes. Although the globally recommended treatment for PTSD is trauma-focused therapy including exposure work (National Institute for Health and Care Excellence, 2018), this approach often gives rise to anxiety with up to 60% of individuals with PTSD refusing trauma-focused therapy (Kehle-Forbes et al., 2016) and 20–34% not completing a full course of treatment (Forbes et al., 2012; Tuerk et al., 2011). Therefore, provision of a TIPS intervention with relation to IU may enhance adherence and tolerance of treatment. Our findings support this premise given that, although exposure therapy did not form part this program, individuals reported associations between decreases in IU levels and PTSD symptom severity over the course of a group intervention that focused on psychoeducation, mood and cognitive management strategies, and skills development.

AUTHOR CONTRIBUTIONS

AB and DB contributed to the development of the study concept. AB developed the study design under the supervision of, and in consultation with, DB. Testing, data collection, data analysis, and data interpretation were performed by AB. MH and CM assisted with study implementation. AB and DB drafted the paper. ZS, MH, and CM provided critical revisions. All authors approved the final version of the paper for submission.

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CONFLICT OF INTEREST

The authors declared no conflict of interest with respect to the authorship or the publication of this article.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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