PTSD Symptoms Lead to Modification in the Memory of the Trauma:
A Prospective Study of Former Prisoners of War

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ABSTRACT

Objective: With the growing interest in the role of trauma memory in posttraumatic stress disorder (PTSD), this prospective study examined long-term changes in memory and the bidirectional relationship between symptoms of PTSD and trauma memory.

Method: A sample of Israeli former prisoners of the 1973 Yom Kippur War (N = 103) was assessed in 1991 and in 2008. Participants' PTSD symptom clusters, measured by the PTSD Inventory, and recollections of subjective and objective exposure during captivity, measured by a self-report questionnaire, were assessed at both times. Data on prewar and postwar negative life events and psychotherapy were also collected.

Results: Repeated-measures analysis revealed that participants' recollections were increasingly negative over time ($P < .001$). Applying an autoregressive cross-lagged modeling strategy showed that the PTSD symptoms of hyperarousal facilitated subsequent amplifications in their recollections ($P < .01$).

Conclusions: These findings challenge the accuracy of reports of traumatic experiences and show that PTSD symptoms, in part, lead to the formation of more negative recollections over time. The findings suggest that the original memory is repeatedly updated under the influence of the individual's emotional state. The findings are discussed in the context of the reconsolidation theory of memory.

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Posttraumatic stress disorder (PTSD) is a debilitating and potentially chronic psychiatric condition precipitated by exposure to a traumatic event.[1] The etiology, diagnosis, and treatment of PTSD rely on the survivor's memory of the event. A pressing matter of debate concerns the degree of consistency in the memory over time, which is generally assessed by comparing self-reports generated at different times. A number of recent studies document modifications in trauma recollections among individuals endorsing PTSD,[2–4] contrary to the long-held notion that the memory of trauma is fixed in time. While trauma memory and subsequent memory modifications are regarded as central to the development and persistence of PTSD,[5] the alternative notion that PTSD may lead to modifications in the memory has been overlooked. The present study intends to address this void in research.

Previous research on trauma memory and PTSD has relied heavily on cross-sectional, retrospective designs, which are inadequate for exploring sequential relationships and the possible dynamic nature of the memory. Longitudinal designs with repeated measures of both the trauma event report and PTSD level may allow for this kind of investigation and advance our knowledge.[6,7] A number of longitudinal studies using combat veteran samples show changes in the recollections of exposure to war-related stressors over time. Southwick and colleagues'[8] landmark study documented an association between veterans' current PTSD symptom severity 2 years after deployment and their tendency to report stressors that they did not recall at the initial assessment 1 month postdeployment. In support of this association, recent research suggests that reactivated fear-laden memories in humans are vulnerable to interference and distortion as they may undergo reconsolidation during each retrieval episode.[9]

Later studies utilizing military samples further document an association between changes in the trauma memory and the symptom clusters of PTSD. Veterans endorsing reexperiencing symptoms tended to report more trauma exposure than in an earlier assessment,[10] suggesting that when intrusive memories are more accessible, they may drive veterans' recollections. The findings accord with the well-documented effect of mood-dependent memory retrieval,[11] whereby individuals' recollections are congruent with their current emotional state.

The unclear directional relationship between PTSD symptoms and trauma memory exists as a limitation in these studies. The symptoms were measured at the second assessment rather than during the time of the initial recollection. Consequently, the possibility that PTSD symptoms may lead to the modification of the traumatic memory remains unexplored. If this possibility proves likely, it imposes a threat to the validation of PTSD, which is rooted in the premise that exposure leads to PTSD and not vice versa.

A related issue to consider is the subjective aspect of exposure to trauma and the emotional impact of the event. Little is known about possible changes in the recollections of subjective exposure,[7] although subjective exposure is more strongly associated with PTSD than objective measures of stressor severity.[12] Previous studies relied heavily on veterans' report of the factual and objective information of exposure (eg, witnessing dead bodies).[3,4,8] This accords with recent *DSM* revisions in PTSD criteria,[1] which ignore the A2 criterion (ie, individuals' emotional response at the time of the event).

Clinical Points

- The memory of the trauma is a hallmark in the development of PTSD symptoms, yet whether and how the symptoms affect the memory are unknown.
- Individuals endorsing PTSD symptoms tend to recall their traumatic experiences as increasingly negative over time, and the traumatic memories are modified and updated in part by their symptoms.
- Clinicians should be aware of the malleable nature of traumatic memories and the possible changes in recollections between assessments.

Finally, the issue of possible modifications in trauma memory is particularly pressing in individuals who have been exposed to extreme and prolonged trauma. These individuals were likely to have experienced high levels of fear, distress, and dissociation, which would then enhance encoding from an amygdala-based (rather than hippocampal) memory system.[13] The former entails a decontextualized, emotional, and fragmented memory, while the latter consists of a contextual, coherent, emotionally neutral memory.[14] Possibly, then, the amygdala-based memory of extreme trauma would be susceptible to the dynamic process of reconsolidation upon recall, resulting in a modified memory over time. To the best of our knowledge, previous military studies focused exclusively on reports of exposure to combat, and no study examined changes in recollections of war-related captivity.

The current study examined Israeli combat veterans who fought in the 1973 Yom Kippur War and were subsequently taken captive and subjected to prolonged torture and humiliation. We examined changes in their reports of subjective and objective captivity-related stressors 2 and 3 decades following the war. We used cross-lagged analysis of a longitudinal panel design, a methodology specifically targeted at elucidating the directionality of relationships. Our analysis focused on whether the recollections of captivity change over time and whether PTSD symptoms modify reports of exposure.

METHOD

This study uses data from a larger study on the psychological effects of war captivity.[15] A cohort of 103 Israeli male combat veterans who fell into captivity during the Yom Kippur War was assessed 18 (T1: 1991) and 35 (T2: 2008) years after the war. All participants provided data at both assessments. One hundred fifty veterans were included in the initial sample, constituting a 69% response rate.

The Yom Kippur War was fought by the coalition of Arab states led by Egypt and Syria against Israel from October 6 to 25, 1973. The war began when the Arab coalition launched a joint surprise attack on Israeli positions on Yom Kippur, the holiest day in Judaism. The war was part of the Arab-Israeli conflict, an ongoing dispute that included many battles and wars since 1948, when the state of Israel was formed.

The majority of the veterans (82%) were captured by the Egyptians, and some were imprisoned by the Syrians. Despite the difference in duration of captivity—8 months for those imprisoned in Syria and 6 weeks for those imprisoned in Egypt—POWs of both groups were subjected for the most part to the same captor protocol. Initially, they were exposed to prolonged isolation and harsh systematic torture coupled with interrogation, consisting of the infliction of severe physical pain and great mental pressure. For example, the POWs experienced extreme deprivation and interference with their personal hygiene. After a period of approximately 14 days, the interrogations stopped and in most cases so did the harsh torture.

Following approval from both the Israel Defense Forces (IDF) and Tel Aviv University review boards, lists of potential participants from the IDF computerized data bank were composed. The participants were contacted by telephone and invited to take part in the study. The questionnaire packet was administered in their homes or in another location of their choice. Informed consent was obtained from all participants.

The same questionnaires were completed in the 2 waves of measurement, and their order was randomized between participants to avoid any potential order effect. Participants' mean age during the war was 22 years (SD = 3.52; range, 18–35). The majority were born and raised in Israel, had completed high school education (70%), were single (75%) and secular (60%), and reported family household income above average (59%). Eighty percent of the participants were privates in the military during active duty, and there were no differences between the sample at the first and follow-up assessments on these background variables. Forty-two percent of the sample received psychological treatment following the war, and 22% were undergoing treatment at the time of the follow-up in 2008.

Captivity-related recollections were assessed using a set of standardized self-report measures. Owing to the lack of a valid and reliable standardized measure that assesses responses in captivity, the items were created by our group based on literature review and clinical interviews with the ex-POWs.[16] Following the *DSM-IV-TR*[17] criteria for a potentially traumatic event, 2 dimensions of exposure in captivity were measured. Subjective exposure questions asked participants about perceived psychological and physical suffering and humiliation during captivity using a 3-point Likert scale ranging from "not at all" to "very much." Objective exposure questions asked participants about injury in captivity and weight loss. Additionally, we assessed responses in captivity via 24 items that yield 3 main factors using a principal component analysis with Varimax rotation, including active coping, loss of emotional control, and detachment.[16]

PTSD symptoms were measured using the PTSD Inventory.[18] This self-report scale was initially based on *DSM-III-R* criteria,[19] which was the standard of practice at the beginning of the study. The inventory consists of items corresponding to the PTSD symptoms listed in *DSM-III-R*.

Table 1. Mean and Standard Error Values, Prevalence of Change, Test Statistics, Effect Sizes, and Significance Levels for Assessing Change in Ex-POWs' Recollections Between 1991 and 2008

	Mean[a]		SE		Ex-POWs With	Ex-POWs With	Ex-POWs With		
	1991	2008	1991	2008	Increased Level, %	Decreased Level, %	Same Level, %	$F_{1,54}$	η^2_p[b]
Physical suffering	3.88	4.46	0.16	0.11	42.2	6.7	51.1	3.79*	0.07
Psychological suffering	3.49	4.51	0.15	0.10	62.5	4.5	33.0	20.16***	0.28
Humiliation	4.04	4.69	0.15	0.10	48.9	4.5	46.6	20.72***	0.28
Active coping	2.50	2.23	0.08	0.10	12.4	59.6	28.1	5.45*	0.10
Loss of emotional control	2.24	2.76	0.10	0.12	70.0	16.7	13.3	9.15**	0.15
Detachment	2.65	3.14	0.09	0.11	59.6	20.2	20.2	6.95*	0.12
Kilograms of weight lost	12.47	15.60	0.99	1.14	58.3	23.6	18.1	10.14**	0.20

[a]All means are adjusted for the effects of therapy and negative life events before and after captivity.
[b]Partial η^2.
*$P < .05$.
**$P < .01$.
***$P < .001$.
Abbreviations: POW = prisoner of war, SE = standard error.

To conform to the updated definition of PTSD, both the 1991 and 2008 data were analyzed in accordance with the *DSM-IV-TR*[17] symptom clusters. Internal consistency was high (Cronbach α = 0.87 for 1991 and 0.95 for 2008). High convergent validity of the inventory was found when it was compared with diagnoses based on structured clinical interviews.[18] At T1 and T2, respectively, 8.7% and 34.7% of the sample endorsed PTSD in accord with *DSM* symptom criteria.

Negative events before and after the war were assessed in 1991. Participation in military operations before the war (6 items) was assessed by self-designed questionnaires for the larger project.[15] Postwar negative life events (eg, health, family stressors) were assessed by the Life Events Questionnaire[20] comprising 23 items (α was .91 for the total score).

Psychological treatment was assessed in 2008. Participants were asked whether they received psychotherapy following the war and whether they are currently in treatment.

RESULTS

Changes in Captivity Recollections Over Time

We examined changes in ex-POWs' captivity-related recollections between T1 and T2 while controlling for psychotherapy (yes, no) and negative life events before and after the war. To this end, we ran within-subject analyses of covariance (ie, repeated measures) in which time (T1, T2) served as the independent measure, and therapy and negative life events served as covariates (Table 1). The analyses revealed increasingly negative recollections across measurements. Significantly greater levels of physical and psychological suffering, more occurrences of humiliations, greater loss of emotional control, lower levels of active coping, and greater amount of weight loss were recalled by between 40% and 70% of ex-POWs at T2 compared with T1.

Additionally, to examine the differences in the recollections of the 2 dichotomous questions, we ran a series of McNemar correlations. The analysis revealed that at T1, 51 (49.5%) ex-POWs reported that they were not injured during their capture, whereas at T2, 28 (54.9%) of them reported that they were injured during their capture ($P < .001$). Only 6 (11.8%) of the ex-POWs who reported in T1 that they were injured reported in T2 that they were not injured. The only recollection to remain unchanged between T1 and T2 was the recollection of the injury during captivity (yes, no; $P = 1$).

Table 2. ARCL Model Fit Indexes

	χ^2_6	*P* Value	CFI	TLI	RMSEA	90% CI for RMSEA
Physical suffering	6.55	.36	1	0.99	0.01	0.00–0.06
Psychological suffering	6.56	.36	1	0.99	0.01	0.00–0.06
Humiliation	5.29	.51	1	1	0	0.00–0.05
Active coping	8.19	.22	1	0.97	0.03	0.00–0.07
Loss of emotional control	7.46	.28	1	0.98	0.02	0.00–0.06
Detachment	6.73	.35	1	0.99	0.02	0.00–0.06
Kilograms of weight lost	8.55	.20	1	0.97	0.03	0.00–0.07
Injury during their capture	5.88	.44	1	1	0	0.00–0.06
Injury during captivity	5.29	.51	1	1	0	0.00–0.05

Abbreviations: ARCL = autoregressive cross-lagged, CFI = comparative fit index, RMSEA = root-mean-square error of approximation, TLI = Tucker-Lewis index.

Bidirectional Relationship Between Captivity-Related Recollections and PTSD

To examine the bidirectional association between the captivity-related recollections and the severity of PTSD symptoms from T1 to T2, we employed autoregressive cross-lagged (ARCL) modeling strategy[20] (Tables 2 and 3). The ARCL allowed us to examine whether earlier measures of PTSD predicted a change in later measures of recollections and whether earlier measures of recollections predicted a change in later measures of PTSD. In these models, we also controlled for therapy (yes, no) and negative life events before and after the war.

We estimated the appropriateness of the model using MPlus 6.1 Structural Equation Models (SEM) software.[21] The model's fit was assessed by the comparative fit index (CFI), Tucker-Lewis index (TLI), and the root-mean-square error of approximation (RMSEA). A model is judged as reasonably fitting the data when CFI and TLI are higher than 0.95 and the RMSEA is lower than 0.05.[22] Missing data were handled with the case-wise maximum likelihood estimation.

Table 3. Cross-Lagged Associations (as β values) Between PTSD and the Recollections of the Trauma[a]

	Physical Suffering	Psychological Suffering	Humiliation	Active Coping	Loss of Emotional Control	Detachment	Kilograms Lost	Injury During Capture[b]	Injury in Captivity[b]
Recollection to reexperiencing symptoms	0.19*	0.27**	0.32***	−0.14	0.24**	0.06	0.01	−0.011	−0.12
Recollection to avoidance symptoms	0.16	0.28**	0.29**	−0.20*	0.29**	0.12	0.02	−0.017	0.02
Recollection to hyperarousal symptoms	0.17	0.31**	0.32***	−0.09	0.32**	0.19	0.04	−0.08	0.01
Reexperiencing symptoms to recollection	−0.03	−0.02	0.03	−0.14	0.10	0.04	0.01	0.21	0.20
Avoidance symptoms to recollection	−0.01	0.01	0.07	−0.04	−0.06	0.13	−0.03	−0.23	−0.01
Hyperarousal symptoms to recollection	0.20*	0.20**	0.08	0.010	0.04	−0.14	0.08	0.31**	−0.03

[a]All coefficients are adjusted for the effects of psychological treatment and negative life events before and after captivity.
[b]Injury during capture and injury while in captivity were coded such that 0 refers to "No" and 1 refers to "Yes."
*$P < .05$.
**$P < .01$.
***$P < .001$.
Abbreviation: PTSD = posttraumatic stress disorder.

Table 4. Standardized Regression Coefficients for Predicting a Change in PTSD Level From 1991 to 2008 by the Initial Recollections Level and Residual Measures of These Measures

	PTSD Reexperiencing Symptoms	PTSD Avoidance Symptoms	PTSD Hyperarousal Symptoms
Physical suffering initial level	0.26*	0.09	0.18
Physical suffering residual	0.38**	0.32**	0.38**
Psychological suffering initial level	0.27*	0.20	0.32**
Psychological suffering residual	0.38**	0.21	0.37**
Humiliation initial level	0.31**	0.15	0.31**
Humiliation residual	0.16	0.35**	0.08
Active coping initial level	−0.01	−0.07	0.07
Active coping residual	−0.13	0.04	−0.02
Loss of emotional control initial level	0.26*	0.16	0.25*
Loss of emotional control residual	0.33**	0.25*	0.31*
Detachment initial level	0.16	0.25*	0.21
Detachment residual	0.11	0.05	0.06
Weight loss initial level	0.11	0.14	0.07
Weight loss residual	0.17	0.23*	0.17
Injury during capture initial level	−0.15	−0.16	−0.11
Injury during capture residual	0.12	−0.04	0.17
Injury while in captivity initial level	0.03	0.20	0.13
Injury while in captivity residual	0.01	0.01	0.02
Psychological treatment (1 = yes)	0.28*	0.28*	0.11
Prewar negative life events	0.31*	0.39**	0.28*
Postwar negative life events	−0.01	0.05	−0.37**

*$P < .05$.
**$P < .01$.
***$P < .001$.
Abbreviation: PTSD = posttraumatic stress disorder

The analyses revealed that severity of hyperarousal symptoms in T1 predicted a change in recollection from T1 to T2 with respect to physical and psychological suffering and occurrence of an injury: the worse the symptoms, the higher the amplification in the exposure recollection.

The analyses also revealed that the recollections in T1 of psychological suffering, humiliation, and loss of emotional control predicted a change in PTSD level between T1 and T2: the more recollection of psychological suffering, more humiliations, and greater loss of emotional control, the higher the increase in severity of reexperiencing, avoidance, and hyperarousal symptoms. Recollection of physical suffering predicted greater increase in reexperiencing symptom severity; recollection of active coping predicted lower increase in avoidance symptoms severity. All other cross-lagged effects between PTSD and the recollection were not significant.

Does the Initial Level of the Recollections or the Amount of Change in the Recollections Over Time (or Both) Predict the Changes in PTSD?

We examined whether the initial level or the amplification of the captivity-related recollections over time predicted the change in PTSD level between T1 and T2 while controlling for therapy (yes, no) and negative life events before and after the war. To this end, we calculated for each participant residual measures tapping the change in the recollections of the trauma from T1 and T2 (for a total of 9 measures; we did not use difference scores because they are closely related to the initial level of the measures, whereas the residual measures are not). Next, we ran a series of hierarchical regressions predicting the level of PTSD in T2 (Table 4). In the first step of these regressions, we introduced ex-POWs' level of PTSD in T1 to control for the initial level of PTSD and thus to predict the change in the level of PTSD between T1 and T2. We also added the measures of therapy and negative life events as covariates. In the second step, we added the initial recollections level (separate analysis for each type of recollections) and the residual measures of these recollections.

For the increase in the number of reexperiencing symptoms over time, the analyses indicated that the initial recollections of physical and psychological suffering, and humiliation and loss of emotional control predicted a significant increase in the number of reexperiencing symptoms that an ex-POW endorsed between T1 and T2. Greater amplification in the recollections of physical and psychological suffering and loss of emotional control predicted a significant increase in the number of reexperiencing symptoms that an ex-POW endorsed between T1 and T2. Ex-POWs who underwent therapy and those who experienced more negative life events before captivity reported a significant increase in the number of reexperiencing symptoms that they endorsed between T1 and T2.

Table 5. Standardized Regression Coefficients for Predicting a Change in Level of Recollections From 1991 to 2008 by the Initial PTSD Cluster Levels and Residual Measures of These Clusters

	Physical Suffering	Psychological Suffering	Humiliation	Active Coping	Loss of Emotional Control	Detachment	Weight Loss	Injury During Capture	Injury in Captivity
Residual of reexperiencing symptoms	0.17	0.23	0.09	−0.21	0.18	0.24	0.05	−0.02	0.01
Residual of avoidance symptoms	0.11	−0.01	0.32*	0.12	0.06	0.01	0.13	−0.15	−0.01
Residual of hyperarousal symptoms	0.13	0.16	−0.18	0.14	0.13	−0.16	0.03	0.20	0.02
Initial level of reexperiencing symptoms	0.03	0.10	−0.01	−0.15	0.06	0.32*	0.23	−0.11	0.22
Initial level of avoidance symptoms	0.08	0.04	0.16	−0.05	−0.03	0.20	−0.07	−0.17	−0.04
Initial level of hyperarousal symptoms	0.22	0.31*	0.08	0.03	0.04	−0.21	0.11	0.37*	−0.21
Psychological treatment (1 = yes)	0.10	0.04	0.01	0.03	−0.04	−0.01	−0.10	0.18	0.11
Prewar negative life events	0.06	−0.05	−0.24*	−0.08	−0.01	0.06	0.03	0.01	0.21
Postwar negative life events	−0.01	0.01	−0.12	−0.01	0.12	−0.06	−0.17	0.18	0.10

*$P < .05$.
Abbreviation: PTSD = posttraumatic stress disorder.

For the increase in the number of avoidance symptoms over time, the analyses indicated that the initial recollections of detachment predicted a significant increase in the number of avoidance symptoms that an ex-POW endorsed between T1 and T2. Greater amplification in the recollections of physical suffering, humiliation, loss of emotional control, and weight loss predicted a significant increase in the number of avoidance symptoms that an ex-POW endorsed between T1 and T2. Ex-POWs who underwent therapy and those who experienced more negative life events before captivity reported a significant increase in the number of avoidance symptoms that they endorsed between T1 and T2.

For the increase in the number of hyperarousal symptoms over time, the analyses indicated that the initial recollections of psychological suffering, humiliation, and loss of emotional control predicted a significant increase in the number of hyperarousal symptoms that an ex-POW endorsed between T1 and T2. Greater amplification in the recollections of physical and psychological suffering and loss of emotional control predicted a significant increase in the number of hyperarousal symptoms that an ex-POW endorsed between T1 and T2. Ex-POWs who experienced more negative life events before captivity but fewer negative life events after captivity reported a significant increase in the number of hyperarousal symptoms that they endorsed between T1 and T2.

Does the Initial Level of PTSD or the Amount of Change in PTSD Symptoms Over Time (or Both) Predict the Change in the Recollections?

We examined whether the initial level of PTSD symptoms or the change in symptoms severity over time predict the change in the recollections of the trauma between T1 and T2 while controlling for therapy (yes, no), negative life events before the war, and negative life events after the war. To this end, we calculated for each participant residual measures tapping the change in PTSD reexperiencing, avoidance, and hyperarousal symptoms from T1 and T2. Next, we ran a series of hierarchical regressions predicting the level of recollections in T2 (Table 5). In the first step of these regressions, we introduced ex-POWs' level of recollections in T1 to control for their initial level and thus to predict the amount of change between T1 and T2 in the recollection. We also added the measures of therapy and negative life events as covariates. In the second step, we added the initial levels of the PTSD clusters and the residual measures of these clusters.

The analyses revealed that an increase in avoidance symptoms from T1 to T2 was associated with an amplification in the recollection of humiliation. The initial reexperiencing symptoms levels in T1 predicted an amplification of recalled detachment, whereas hyperarousal was linked with an amplification of recalled psychological suffering as well the occurrence of an injury while being captured. More negative life events before the war predicted a decrease of recalled humiliation. All other effects were not significant.

DISCUSSION

This study of former prisoners of war examined the directional relationship between PTSD and modifications in the long-term recollection of captivity. While recalled exposure predicted subsequent PTSD symptoms, at the same time, the recollections were modified by the symptoms of PTSD. Ex-POWs with initial elevated PTSD and those with symptoms that increased over time remembered their captivity experiences as worse than they had 17 years earlier. Altogether, the findings suggest a positive feedback loop whereby traumatic memory leads to PTSD symptoms and in turn the symptoms lead to an increasingly negative recollection of the traumatic event and so forth.

Modifications in ex-POWs' recollections of captivity might be expected over a 17-year period and accord with the previous studies[8,10] documenting inconsistency in recalled factual information of combat exposure over shorter periods. Extreme stress can impair encoding processes as the attention is narrowed onto the central details of the events and result in a fragmented trauma memory,[23] which is open to incorporate "new" elements into the memory trace. At the same time, we would expect that recollections of peritraumatic responses, like the experience of loss of emotional control, and central autobiographical facts, such as being injured in captivity, as we report here, would remain unchanged.

It is possible that the changes in captivity-related recollections, like memory of more neutral events, may be attributable to experiences that share features with and thus become associated with the target memory event.[24] The captivity recollections may have been also subject to normal processes of memory decay and the loss of retained information with the passage of time.[25] One could speculate, however, that the memory of an intense, prolonged, and extraordinary experience such as captivity would be neither distorted by other experiences nor lost over time. Our findings show that ex-POWs generated increasingly negative recollections over time, suggesting that they were not simply confused about what had happened 17 years ago.

Our findings point in the direction that modifications in the memory of the trauma are possibly accounted and updated under the influence of PTSD symptoms. In line with current research, memory for traumatic events—like the memory of ordinary life events—may undergo reconsolidation with each subsequent retrieval, and during the retrieval, it may be vulnerable to modifications just as it was when it was first encoded.[26,27] It is plausible that individuals' mental state when the memory is retrieved can affect the reconsolidation process, resulting in an altered memory upon the next voluntary recall.[28] It would be expected that a chronic state of distress would have a strong directional effect on how an experience is remembered over time.

Our findings show that the hyperarousal symptoms of PTSD lead to an increasingly negative recollection, suggesting that the ex-POWs may have not simply displayed motivational recall to justify their distress. Hyperarousal is seen as a marker of an elevated state of anxiety. Reminder of the trauma may trigger excessive activation of stress hormones such as norepinephrine release in the amygdala and initiate the reconsolidation of an increasingly negative-emotional laden memory, subsequently resulting in elevated hyperarousal symptoms. A number of pilot studies show that β-blockers (ie, adrenergic agonists), which reduce anxiety and symptoms of hyperarousal, may offer a novel treatment for PTSD by blocking the reconsolidation of the memory.[29,30]

Several study limitations should be noted: Although the follow-up sample did not differ from the initial one in main study variables, the inevitable attrition between assessments in longitudinal studies should be taken into account. Also, by using a self-report measure— although the PTSD Inventory has been validated against a clinical diagnosis—we may have assessed amplified reports in the symptoms attributed partly to cultural changes in the recognition of PTSD. A related issue is that the measures of the captivity recollection, which have good reliability, were designed by our group owing to the lack of relevant measures and the variability in captivity stressors. We did not include a detailed account of participants' individual experiences during captivity, which may have affected their trauma recollections. Finally, important changes in the trauma recollections may have occurred before our study commenced. Ideally, we would obtain more detailed data on ex-POWs' recollection of captivity using several assessment points from the immediate phase following the trauma to the long term, and we would include a clinical diagnosis evaluation of PTSD and an assessment of comorbid symptoms and cognitive impairments implicated in memory modifications.

Within the context of these limitations, the present study makes a significant contribution by documenting that survivors' PTSD symptoms lead to modification in their recollections of the trauma even 3 decades following exposure. These findings challenge the widely held notion of a unidirectional exposure-response effect and a diagnosis of PTSD based on retrospective reports of the trauma, and they call for the identification of alternative measures of assessment, such as biological markers of PTSD. Future longitudinal studies are warranted to clarify the mechanisms of trauma memory modifications linked with PTSD.

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