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Early Pain and Other Somatic Symptoms Predict Posttraumatic Stress Reactions in Survivors of Terrorist Attacks: The Longitudinal Utøya Cohort Study

Synne Øien Stensland,[1,2] Siri Thoresen,[1] Tine Jensen,[1,3] Tore Wentzel-Larsen,[1,4] and Grete Dyb[1,5]

[1]Norwegian Centre for Violence and Traumatic Stress Studies, Oslo, Norway
[2]Research and Communication Unit for Musculoskeletal Health (FORMI), Division of Clinical Neuroscience, Oslo University Hospital, Oslo, Norway
[3]Department of Psychology, Faculty of Social Sciences, University of Oslo, Norway
[4]Centre for Child and Adolescent Mental Health, Eastern and Southern Norway, Oslo, Norway
[5]Institute of Clinical Medicine, University of Oslo, Oslo, Norway

Survivors of traumatic events commonly suffer from long-term pain and related somatic symptomatology. To test the predominant hypothesis that survivors' pain comprises sequela of persistent posttraumatic stress symptoms (PTSS), we assessed the sequential order of symptom development among young survivors of a terrorist attack. All 490 survivors of the 2011 Utøya (Norway) attacks were invited to the longitudinal Utøya cohort study; 355 (72.4%) participated. The mean survivor age was 19.3 years ($SD = 4.6$) and 169 were female (47.6%). Somatic symptoms, including headache, other pain and fatigue, and PTSS, were measured 4–5 months (T1), 14–16 months (T2), and 32–33 months (T3) after the attack. Longitudinal associations between somatic symptoms and PTSS were assessed in cross-lagged structural equation model (SEM) analyses, which were adjusted for known confounders. Higher pain levels and other somatic symptoms at T1 consistently predicted PTSS at T2 in SEM analyses, $r = .473$, $p < .001$. Beyond this early-to-intermediate posttraumatic phase, somatic symptoms did not significantly predict PTSS: T2–T3, $r = .024$, $p = .831$; T1–T3, $r = -.074$, $p = .586$. PTSS did not significantly predict later somatic symptomatology at T1–T2, $r = .093$, $p = .455$; T2–T3, $r = .272$, $p = .234$; or T1–T3, $r = -.279$, $p = .077$. The findings indicate that survivors' early pain and related somatic symptoms strongly and consistently predict later psychopathology. After severe psychological trauma, early interventions may need to address individuals' pain to hinder chronification.

Mental and somatic disorders commonly impair the long-term health and functioning of individuals exposed to armed conflicts, terrorist attacks, other violence, and disasters (Lanius et al., 2010; Shalev et al., 2017; Steinert et al., 2015). A heightened risk of adverse somatic health outcomes, such as chronic pain (George et al., 2016; Noel et al., 2016; Suri et al., 2017), somatic symptom disorder (Andreski et al., 1998; Kizilhan & Noll-Hussong, 2018), and gastrointestinal (Gradus et al., 2017) or cardiometabolic disease (Edmondson & von Kanel, 2017) has been observed among survivors with higher levels of posttraumatic stress symptoms (PTSS) or posttraumatic stress disorder (PTSD) compared to those with lower levels (Pacella et al., 2013; Seng et al., 2005; Steinert et al., 2015).

Survivors' adverse somatic health outcomes have traditionally been understood as long-term sequelae of persistent PTSS (Andreski et al., 1998; McLaughlin et al., 2016; Pacella et al.,

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Correspondence concerning this article should be addressed to Synne Øien Stensland, Norwegian Centre for Violence and Traumatic Stress Studies, P.O box 181 Nydalen, 0409 Oslo, Norway. E-mail: synne.stensland@nkvts.no

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2013) and the related dysregulation of physiological stress responses (Schnurr & Jankowski, 1999). This understanding builds on a chronic stress model (McEwen, 1998), whereby the adverse physiological impact of persistent fear, negative alterations in cognition and mood, and related cognitive behavioral correlates, such as avoidant coping, poor sleep, or substance abuse, synergize to increase the risk of onset or exacerbation of a range of adverse somatic health outcomes over time (Koenen, Sumner, et al., 2017; Schnurr & Jankowski, 1999).

Yet knowledge of the actual sequential order of symptom development is scarce and, in part, contradictory (Koenen, Sumner, et al., 2017; Pacella et al., 2013). Alternatively, somatic symptoms could precede PTSS. Findings from disaster studies lend some support to this suggestion, indicating that shortly after trauma exposure, survivors commonly experience severely adverse somatic health outcomes ranging from frequent migraines (Stensland et al., 2018) to stomachaches, low back pain, sleep problems, and fatigue (Hensley & Varela, 2008; Zhang et al., 2015) to acute myocardial infarction (Goldberg et al., 2005).

A recent study of disaster survivors found that somatic symptoms assessed 3 months posttrauma predicted higher levels of PTSS at 6 months posttrauma but also that PTSS predicted later somatic symptoms (Zhang et al., 2015). Similarly, such mutual influence was found between pain and PTSS in a study of 824 injured patients followed for 1 year from the time of acute hospitalization (Liedl et al., 2010) as well as in a 1-year follow-up study of 209 young military personnel following combat-related blast exposure (Stratton et al., 2014). Thus, the development and role of somatic symptoms and PTSS remain entwined (Koenen, Sumner, et al., 2017; Pacella et al., 2013; Yehuda et al., 2015). A better understanding of the development of PTSS and somatic symptoms following trauma exposure could help professionals identify survivors in need of early intervention efforts and improve the timeliness and efficiency of services.

The aim of the present study was to determine the sequential order of PTSS and somatic symptom development following trauma exposure. We hypothesized that somatic symptoms would predict PTSS from one assessment point to the next and vice versa, with mutual maintenance among symptoms over time (Asmundson & Katz, 2009). We investigated this hypothesis in a cohort of 355 young survivors, who had been highly and relatively homogeneously exposed to a terrorist attack, across three posttrauma time points. Previous research has revealed that trauma-related factors, such as physical injuries and the loss of someone close (Bugge et al., 2015; Dyb et al., 2014; Liu et al., 2014), as well as background factors, such as age, sex, ethnicity, financial resources, and prior trauma exposure, may impact posttraumatic health development (Alisic et al., 2014; George et al., 2016; Lewis et al., 2019; Pacella et al., 2013; Shalev et al., 2017; Trickey et al., 2012; Yehuda et al., 2015). Consequently, we intended to account for these factors as much as possible in our analyses.

Method

Participants

On July 22nd, 2011, 564 people were present on the Utøya island for the yearly Norwegian Labour Party youth summer camp, when a right-wing extremist opened fire, killing 69 people and severely wounding 33 (Figure 1). All survivors were highly exposed to the atrocities, many risked hypothermia and drowning while trying to escape, and most lost friends or with whom they were close (i.e., a family member or partner; Dyb et al., 2014). The severely injured were treated in trauma-care units (Jorgensen et al., 2016). Participants were mainly adolescents and young adults who ranged in age from 13 to 57 years ($M = 19.3$ years; $SD = 4.6$; Table 1) and were sociodemographically comparable to the Norwegian population within that age group (Stensland et al., 2018).

Procedure

The full cohort of adolescent and adult survivors settled in Norway ($N = 490$) was invited to participate in the longitudinal Utøya Study in the early posttraumatic phase (i.e., 4–5 months after the attack; Time 1 [T1]) and intermediate posttraumatic phase (i.e., 14–15 months; Time 2 [T2]) after the massacre. Anyone participating at T1 or T2 was invited to participate at Time 3 (T3), 31–32 months posttrauma, which was termed the long-term posttraumatic phase. Altogether, 355 (72.4%) survivors participated in at least one wave of the study, 206 (42.0%) of whom participated at all three assessment points. Two survivors with traumatic brain injuries were excluded, leaving a study sample of 353 (72.0%) participants ($n = 169$ female; 47.9%). Interviews were conducted face to face by trained personnel at all three time points. Interviews were semistructured and included scale-based measures of somatic symptoms and PTSS.

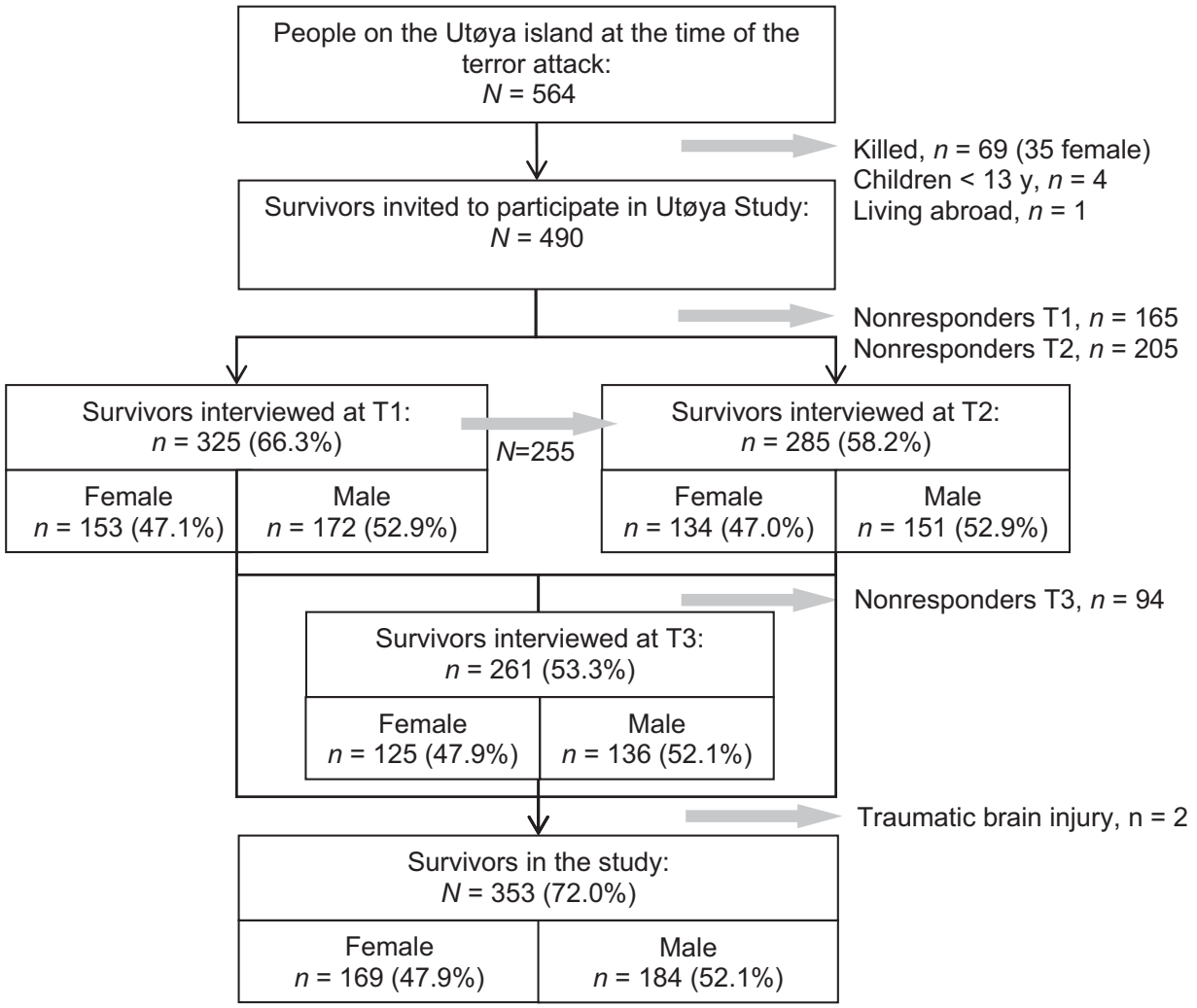
The Utøya Study was based on written, informed consent from participants 16 years of age or older or the parents of individuals under 16 years of age. At the end of each interview, survivors' current needs for health services were assessed, and the interviewers provided help in contacting the appropriate resources if needed. The study was approved by the Norwegian Regional Committee for Medical and Health Research Ethics in Norway (document 551224, 2011/1625; 2014/246).

Measures

Somatic Symptoms

Participants' level of somatic symptoms (Pacella et al., 2013) was measured at each time point using the eight-item Children's Somatic Symptoms Inventory (CSSI-8; Walker et al., 2016). These items were derived from the validated 24-item Children's Somatization Inventory (CSI-24; L. S. Walker et al., 2009) in collaboration with the author of the original instrument. Participants were asked to rate how much they had been bothered by stomachaches, headaches, lumbar pain, pain in the arms or legs, faintness or dizziness,

Figure 1
Flow Chart for the Study Sample From the Longitudinal Utøya Study of Survivors.



Note. N = 353. Interviews were conducted 4–5 months (T1), 14–16 months (T2), and 32–33 months (T3) after the attack.

palpitations, nausea or upset stomach, and weakness during the past 2 weeks. Responses were scored using a four-point Likert scale ranging from 1 (*not at all*) to 4 (*a whole lot*). For descriptive purposes, mean scores were computed for all participants at each time point, with bothersome symptoms defined as single items with a score of 3 or higher. In the present sample, Cronbach's alpha values were .77 at T1, .78 at T2, and .76 at T3, without substantial deviation between the sexes.

Posttraumatic Stress Symptoms

Symptoms of posttraumatic stress were measured using a 27-item extended version of the validated UCLA PTSD Symptom Index for Children and Adolescents (Steinberg et al., 2004). To cover the 20 diagnostic criteria to be described in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013), the index was customized in collaboration with the authors of the original instrument in preparation for the first data collection of the Utøya study in 2011. The measure comprises subscales to assess symptoms of reexperiencing (five items), avoidance (two items), negative alterations in cognition and mood (seven items), and arousal and reactivity (six items). Participants were asked to rate how much they had been bothered by each symptom over the past month using a 5-point Likert scale ranging from 0 (*never*) to 4 (*almost all of the time*). Maximum scores were used to estimate values for the three diagnostic criteria measured by multiple items. For descriptive purposes, mean scores of PTSS were computed for all participants at each time point. In the present sample, Cronbach's alpha values were .90 at T1, .90 at T2, and .92 at T3, and were comparable for both sexes.

Demographic Characteristics and Trauma-Related Variables

"Severe injury" was defined as hospitalization as a consequence of terror-inflicted injuries (Bugge et al., 2017). "Loss

Table 1
Sociodemographic Characteristics, Prior Exposure to Interpersonal Violence, Severe Injury, and Loss of Someone Close During the Terror Attack, by Sex

		Female ($n = 169$)			Male ($n = 184$)		
Variable	Total *n*	*M*	*SD*	*n*	*M*	*SD*	*p*
Sociodemographic characteristic							
Age at the terror attack (years)	169	18.91	4.00	184	19.71	5.06	.105[a]
		n	%		*n*	%	
Non-Norwegian origin	164	13	7.9	182	29	15.9	.023[b]
Low financial status	160	35	21.9	172	38	22.1	.962[b]
Prior interpersonal violence exposure							
Physical violence	143	22	15.4	162	33	2.1	.258[b]
Sexual abuse	141	13	9.2	166	5	3.0	.02[b]
Neglect	130	9	6.9	148	11	7.4	.870[b]
Mass-shooting injury and loss							
Severe injury	169	16	9.5	184	8	4.3	.056[b]
Loss of someone close (family/partner)	167	14	8.4	182	10	5.5	.287[b]

Note. [a]t test. [b]Pearson's chi-square test.

of someone close" was based on the self-report that a family member or partner had been killed during the terrorist attack. Information on age and sex at the time of the attack was based on data from the Norwegian National Population Registry. Non-Norwegian ethnicity was defined as both parents having been born abroad. Participants were asked to report if their financial status was above average, average, or below average; we later dichotomized this variable into "low financial status" (i.e., below average) versus the other two groups collapsed. Prior exposure to interpersonal violence (*yes* or *no*) included self-reported exposure to physical violence, sexual abuse, and neglect prior to the attack. Sexual abuse and physical violence were measured at T2 and T3, whereas neglect was measured at T2 only.

Data Analysis

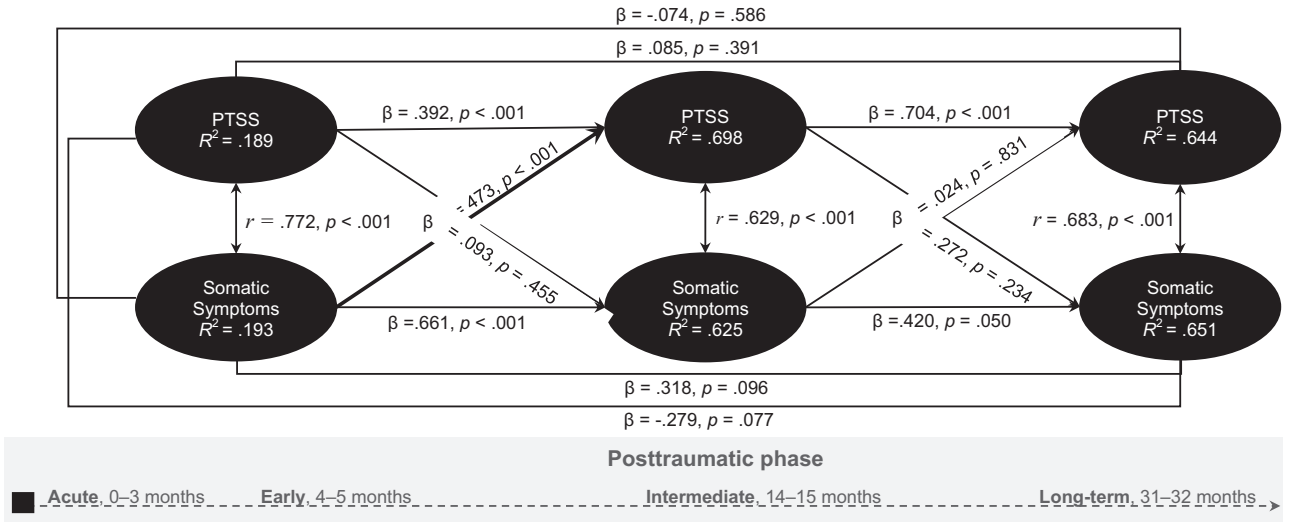
In the presentation of the descriptive data, the calculation of mean scale scores was handled using the half-rule, meaning that scale measures were coded as missing if over half the scale items were missing. Analyses of variance (ANOVAs) and exact Pearson's chi-square tests were conducted to assess sex differences in symptom levels. To judge causal dominance, longitudinal, cross-lagged association between the modeled latent factors for somatic symptoms and PTSS were estimated using structural equation modeling (SEM) following the weighted least squares mean and variance adjusted (WLSMV) procedure to account for ordinal indicators. Missing data were handled using procedures comparable to full information maximum likelihood (FIML). The same indicators were allowed to covary over time. The two-factor indicators for gastrointestinal symptoms (i.e., stomachache and nausea or upset stomach) were modeled as related, based on the high conceptual overlap, and supported by results from the preparatory confirmatory factor analyses (CFAs). Results of difference tests, as part of the CFAs, indicated invariance in the somatic symptoms and PTSS latent factor loadings on their respective factor indicators over time (i.e., T1–T3), $\chi^2(14, N = 112 = 13.70, p = .472$ for somatic symptoms; $\chi^2(38, N = 324) = 49.98, p = .092$ for PTSS.

To determine model fit, we assessed the chi-square distribution, root mean square error of approximation (RMSEA), comparative fit index (CFI; Browne & Cudeck, 1992; Hu & Bentler, 1999), and Tucker–Lewis index (TLI). For the RMSEA, values between .10 and .08 were considered to indicate a mediocre model fit, .08 to .05 an acceptable model fit, and below .05 a good model fit. For the CFI, values between .90 and .94 were considered to indicate an acceptable model fit, and values above .95 were considered to indicate a well-fitting model.

The CFA models assuming invariance in factor loadings over time showed satisfactory model fit for both latent factors, $\chi^2(236, N = 353) = 298.364, p = .004$, RMSEA = .027, CFI = .981, TLI = .977 for somatic symptoms; $\chi^2(1{,}685, N = 353) = 2274.352, p < .001$, RMSEA = .031, CFI = .958, TLI = .956 for PTSS. Main cross-lagged panels were therefore modeled with assumed invariance of factor loadings over time in the SEM.

The main SEM analysis, adjusted for age, sex, economy, ethnicity, severe injury, and loss of someone close, was run in the full cohort of 353 participants (Figure 2). Due to exclusion of missing data on modeled adjustment variables, the main model included 322 participants. As data on prior exposure to interpersonal violence was not collected until T2 due to ethical

Figure 2
Early Somatic Symptoms as Predictors of Posttraumatic Stress Symptoms (PTSS) in 322 Survivors of the Utøya Terror Attacks.



Note. Standardized correlations (r) and regression coefficients (β), as estimates of effect sizes in longitudinal associations, are adjusted for age, sex, ethnicity, financial status, severe injury, and loss of someone close.

reasoning at the time of the terrorist attack, we did not adjust for this variable in the main model to avoid high levels of missing data.

Sensitivity analyses were run among the 206 individuals who participated at all three assessment points and among the 188 individuals with data on prior exposure to physical violence, sexual abuse, and neglect. Additionally, an unplanned sensitivity SEM analysis was performed to assess the temporal cross-lagged association between PTSS and somatic symptoms in the later posttraumatic phase (i.e., T2–T3) without including measures from T1 ($n = 297$). Coefficients for cross-lagged relationships were standardized.

Simple analyses were performed using IBM SPSS statistics (Version 22) in combination with R (Version 3.1.2); SEM models were analyzed using *M*plus (Version 7.31).

Results

Survivors' sociodemographic backgrounds and the prevalence of inflicted injuries and loss are presented, by sex, in Table 1. Tables 2–4 present survivors' levels of somatic symptoms and PTSS, by sex and time since the terrorist attack. Headaches, fatigue, and lumbar pain were the most frequently reported early somatic symptoms among survivors regardless of sex, with 44.1% of female and 16.9% of male participants reporting considerable headaches, 29.6% of female and 18.7% of male participants reporting fatigue, and 26.3% of female and 19.2% of male participants reporting lumbar pain (Table 2). Headaches and lumbar pain remained the most commonly reported somatic symptoms regardless of sex in the intermediate and long-term phases (Tables 3 and 4). One-third (33.8%) of the women and girls in the sample reported three or more bothersome somatic symptoms in the early posttraumatic phase, with rates of 26.0% in the intermediate phase and 18.9% in the long-term phase; the corresponding figures for men and boys were 17.5%, 11.5%, and 6.6%, respectively (Tables 2–4). The mean rating of PTSS was significantly higher in female compared to male participants at each assessment point. For both sexes, the PTSS symptoms of hyperarousal and reactivity had the highest mean levels throughout the observation period.

The results from preparatory SEM analyses indicated fair stability of the somatic symptoms and PTSS constructs over time. Results from the main SEM analysis suggested a good model fit, RMSEA = .023, CFI = .963, TLI = .961. Results from planned and unplanned sensitivity SEM analyses indicated acceptable-to-good fit of all models tested.

In the main SEM analysis, higher levels of somatic symptoms in the early posttraumatic phase consistently predicted more PTSS in the intermediate phase (Figure 2). In contrast, higher levels of early PTSS did not significantly increase the risk of later somatic symptoms. No significant crossover effects between somatic symptoms and PTSS were observed beyond 14–15 months posttrauma. The *r* effect size of the observed predictive value of early somatic symptoms on later PTSS was estimated to be .473, $p < .001$, in the main SEM analysis. The results from all planned and unplanned sensitivity analyses were consistent with the main findings.

As expected, somatic and PTSS clusters were cross-sectionally correlated at all three time points. Somatic symptoms consistently predicted later somatic symptoms. Additionally, PTSS in the intermediate posttraumatic phase consistently predicted long-term PTSS.

Regarding covariates, female sex and non-Norwegian ethnicity significantly predicted PTSS, $r_{\text{female}} = .277$, $r_{\text{ethnicity}} = .297$;

Table 2
Somatic Symptoms and Posttraumatic Stress Symptoms (PTSS) Among Participants in the Early Posttraumatic Phase (4–5 Months Posttrauma), by Sex and Time Since Trauma

	Female				Male				
Variable	*n*	%[a]	*M*	*SD*	*n*	%[a]	*M*	*SD*	*p*
Somatic symptoms[b]	152		1.88	0.54	172		1.58	0.50	< .001[d]
Stomach aches	27	17.8			15	8.7			.016[e]
Headaches	67	44.1			29	16.9			< .001[e]
Lumbar pain	40	26.3			33	19.2			.125[e]
Pain arms/legs	25	16.4			22	12.8			.351[e]
Faintness	19	12.6			10	5.8			.034[e]
Palpitations	25	16.4			23	13.4			.437[e]
Nausea	34	22.4			19	11.0			.006[e]
Weakness	45	29.6			32	18.7			.022[e]
Number of symptoms	151				171				< .001[e]
No symptoms	46	3.5			93	54.4			
1	29	19.2			31	18.1			
2	25	16.6			17	9.9			
3	24	15.9			19	11.1			
≥ 4	27	17.9			11	6.4			
PTSS subscale[c]	152		1.66	0.66	172		1.29	0.67	< .001[d]
Reexperiencing			1.71	0.91			1.24	0.90	< .001[d]
Avoidance			1.25	0.97			.94	0.92	.004[d]
Cognition/mood			1.53	0.74			1.22	0.72	< .001[d]
Arousal/reactivity			1.92	0.68			1.51	0.75	< .001[d]

Note. $N = 353$.
[a]Due to rounding, percentages may not total 100%. [b]Measured using the Children's Somatic Symptoms Inventory (eight items; score range: 1–4); prevalence of single items self-rated with a score of 3 or higher (*quite a lot bothered* to *very much bothered*) is presented. [c]Measured using the UCLA Symptom Index for children and adolescents (20 items; score range: 0–4). The mean of the four subscale scores (between two and seven items each) is presented. [d]*t* test. [e]Pearson's chi-square test.

and somatic symptoms, $r_{female} = .352$, $r_{ethnicity} = .274$, at T1 in the main SEM analysis, $ps < .001$. Additionally, younger age, $r = -.118$, $p = .012$, predicted PTSS at T1; injury predicted somatic symptoms at T2, $r = .119$, $p = .016$; and female sex predicted PTSS at T3, $r = .105$, $p = .041$. In the sensitivity SEM analysis with additional adjustment for prior exposure to interpersonal violence, physical violence also predicted higher level of PTSS, $r = .137$, $p = .040$; and somatic symptoms, $r = .183$, $p = .012$, at T1.

Discussion

The present findings demonstrated that survivors with high levels of early somatic symptoms were at elevated risk of later PTSS, whereas PTSS did not significantly increase the risk of later somatic symptoms. This finding is at odds with current theory and practice that assumes symptoms of posttraumatic stress precede the development of adverse somatic health outcomes after trauma (Pacella et al., 2013). Rather, the present findings suggest that somatic symptoms constitute early predictors of later psychopathology.

In agreement with findings from prior disaster studies (Hensley & Varela, 2008; Zhang et al., 2015), headaches, lumbar pain, and fatigue comprised the most frequently reported somatic symptoms among survivors in the early posttrauma months. More specifically, about one-third of female participants and 1 in 5 male participants reported early fatigue and/or three or more bothersome somatic symptoms. In a prior study of the Utøya survivors, one-third of the adolescent female survivors and about 1 in 6 boys experienced recurrent migraines at 4–5 months posttrauma (Stensland et al., 2018). The likelihood of survivors experiencing frequent headaches in the early posttrauma months was increased 3- to 4-fold as compared to age-matched, unexposed controls, after adjusting for known risk factors, including posttraumatic psychological distress (Shalev et al., 2019; Stensland et al., 2018). Frequent pain and fatigue commonly cause functional impairment (Vos et al., 2015), and it seems likely that a higher burden of such severe somatic symptoms may have adversely impacted survivors' chances of early recovery.

Although evidence is scarce (Koenen, Sumner, et al., 2017; Yehuda et al., 2015), the results of a few prior studies that have examined the association between pain and PTSS point in the

Table 3

Somatic Symptoms and Posttraumatic Stress Symptoms (PTSS) Among Participants in the Intermediate Posttraumatic Phase (14–15 Months Posttrauma), by Sex and Time Since Trauma

	Female				Male				
Variable	*n*	%[a]	*M*	*SD*	*n*	%[a]	*M*	*SD*	*p*
Somatic symptoms[b]	132		1.78	.54	151		1.52	0.46	< .001[d]
Stomach aches	22	16.7			7	4.6			.001[e]
Headaches	45	34.1			27	17.9			.002[e]
Lumbar pain	30	22.7			31	2.5			.654[e]
Pain arms/legs	26	19.7			16	1.6			.032[e]
Faintness	16	12.1			8	5.3			.040[e]
Palpitations	20	15.3			17	11.4			.342[e]
Nausea	26	19.7			12	7.9			.004[e]
Weakness	24	18.2			11	7.3			.006[e]
Number of symptoms	131				148				.002[e]
No symptoms	49	37.4			87	58.8			
1	30	22.9			25	16.9			
2	18	13.7			19	12.8			
3	13	9.9			10	6.8			
≥ 4	21	16.0			7	4.7			
PTSS subscale[c]	132		1.33	0.63	*151*		1.04	0.63	< .001[d]
Reexperiencing			1.15	0.79			0.84	0.72	.001[d]
Avoidance			1.17	0.98			0.91	0.95	.023[d]
Cognition/mood			1.34	0.71			1.09	0.77	.005[d]
Arousal/reactivity			1.53	0.67			1.19	0.69	< .001[d]

Note. $N = 353$.

[a]Due to rounding, percentages may not total 100%. [b]Measured using the Children's Somatic Symptoms Inventory (eight items; score range: 1–4); prevalence of single items self-rated with a score of 3 or higher (*quite a lot bothered* to *very much bothered*) is presented. [c]Measured using the UCLA Symptom Index for children and adolescents (20 items; score range: 0–4). The mean of the four subscale scores (between two and seven items each) is presented. [d]*t* test. [e]Pearson's chi-square test.

same direction as the results of the present study. In a study of middle-aged, socioeconomically deprived, inner-city, primary care patients with PTSD, pain at baseline predicted higher levels of PTSS within the following year, whereas PTSS did not significantly influence changes in pain (Vaughan et al., 2016). Similarly, in the previously mentioned study of military service members exposed to combat, 55% of whom had possible or probable traumatic brain injury (TBI), pain severity baseline predicted later PTSS more strongly than early PTSS predicted later pain (Stratton et al., 2014). As mechanisms underlying the relations among PTSS, pain, and other somatic symptoms may vary with the presence and degree of brain injury (Nampiaparampil, 2008), individuals with TBI were excluded from the present study. Moderate and severe pain has been found to predict mood and anxiety disorders in general population–based studies (de Heer et al., 2018), although the potential role of trauma exposure and PTSS remain largely unaccounted for in these studies.

There are several plausible, partially overlapping mechanisms that may help explain how somatic symptoms could hinder early recovery from PTSS. Mechanisms could relate to somatic symptoms (a) leading to excessive functional impairment, (b) functioning as potent reminders of persistent threat, (c) reflect more extensive underlying neurobiological adaptive or maladaptive responses to trauma exposure, or (d) impact access to or efficiency of early interventions. Early recovery from posttraumatic stress relates to survivors' and their families' capability to regain a sense of safety, calmness, the experience of self- and community efficacy, social connectedness, and hope (Hobfoll et al., 2007). For survivors and families, this involves continuous effort and engagement in tasks to build, or rebuild, daily routines and nurture supportive social relationships. Accomplishing these tasks requires energy, motivation, and the ability to shift focus from traumatic experiences to everyday tasks. This may be particularly challenging in the face of high levels of somatic symptoms and mental turmoil related to what the pain and fatigue may signal. Thus, somatic symptoms, such as frequent and severe pain or fatigue, may distort survivors' ability to engage in tasks necessary for recovery. For example, severe pain often adversely affects one's sleep, appetite, and the ability to rest and engage in physical activity and positive social relationships (Dyb et al., 2015; Roth-Isigkeit et al., 2005). Adverse family functioning (Palermo & Holley, 2013), social withdrawal, and work absenteeism or drop-out from

Table 4

Somatic Symptoms and Posttraumatic Stress Symptoms (PTSS) Among Participants in the Long-Term Posttraumatic Phase (31–32 Months Posttrauma), by Sex and Time Since Trauma

	Female				Male				
Variable	*n*	%[a]	*M*	*SD*	*n*	%[a]	*M*	*SD*	*p*
Somatic symptoms[b]	123		1.67	0.50	*136*		1.42	0.41	< .001[d]
Stomach aches	13	10.6			9	6.6			.255[e]
Headaches	39	31.7			20	14.7			.001[e]
Lumbar pain	27	22.1			20	14.7			.123[e]
Pain arms/legs	10	8.1			12	8.8			.842[e]
Faintness	12	9.8			2	1.5			.003[e]
Palpitations	15	12.2			11	8.1			.272[e]
Nausea	23	18.7			7	5.1			.001[e]
Weakness	14	11.4			12	8.8			.494[e]
Number of symptoms	122				136				.015[e]
No symptoms	57	46.7			85	62.5			
1	26	21.3			31	22.8			
2	16	13.1			11	8.1			
3	9	7.4			3	2.2			
≥ 4	14	11.5			6	4.4			
PTSS subscale[c]	123		1.26	0.68	*136*		0.90	0.64	< .001[d]
Reexperiencing			1.03	0.78			0.70	0.76	.001[d]
Avoidance			1.02	0.92			0.78	0.94	.033[d]
Cognition/mood			1.32	0.78			0.92	0.71	< .001[d]
Arousal/reactivity			1.47	0.76			1.07	0.68	< .001[d]

Note. $N = 353$.

[a]Due to rounding, percentages may not total 100%. [b]Measured using the Children's Somatic Symptoms Inventory (eight items; score range: 1–4); prevalence of single items self-rated with a score of 3 or higher (*quite a lot bothered* to *very much bothered*) is presented. [c]Measured using the UCLA Symptom Index for children and adolescents (20 items; score range: 0–4). The mean of the four subscale scores (between two and seven items each) is presented. [d]*t* test. [e]Pearson's chi-square test.

school often follow. The resulting experience of disability could fuel a disturbing, negative self-perception that one is helpless and damaged in a world unable to alleviate the pain. Under these circumstances, it may be particularly hard for survivors and their families to build or rebuild a sense of safety, trust, connectedness, agency, and hope for recovery.

Further, some somatic symptoms, such as headache or palpitations, could act as internal reminders of persistent threat and contribute to PTSS over time (Asmundson & Katz, 2009; Glad et al., 2017). The hidden, yet intrusive, unpredictable, inescapable, and painful nature of somatic reminders could be particularly baffling, frightening, or shameful, thereby holding specifically high pathogenicity. Persistent pain is known to affect attention toward the detection of potentially salient stimuli (Bushnell et al., 2013). An increased tendency to perceive internal and external signals as threatening could further fuel somatic symptomatology, fear, and development of PTSS.

There is some indication that stress-activated neurobiological alterations may lead to systemic dysregulation of bodily responses that precede and predict, rather than result from, PTSD (Koenen, Sumner, et al., 2017; McFarlane, 2010). Exposure to extreme threat triggers alterations within the overlapping salience network (Shalev et al., 2017; Yehuda et al., 2015) and pain matrix (Peirs & Seal, 2016), which are jointly responsible for detection and processing of and response to potentially threatening stimuli, including pain. Trauma-induced alterations within these and neighboring neural networks affect the release of neurotransmitters and trophic factors, such as norepinephrine, cortisol, neuropeptide Y, and endogenous opioids, which may fuel the development of central sensitization and the dysregulation of autonomic, endocrine, immunological, and metabolic systemic responses, resulting in onset and persistence of headaches and other pain, fatigue, and a range of other somatic symptoms (Reichmann & Holzer, 2016; Yehuda et al., 2015). Thus, a higher level of early somatic symptoms could reflect more extensive underlying neurobiological alterations (McFarlane, 2010; Peirs & Seal, 2016) and, thereby, an increased risk of later posttrauma psychopathology (McFarlane, 2010).

There is a possibility that a clinical picture dominated by severe migraine, other headaches (Stensland et al., 2018) and pain, fatigue, and related somatic symptoms could confuse or hinder efficient help-seeking behavior or access to effective interventions or the ability to make use of them. Unnrecommended

prescription and overuse of analgesics, relaxants, anxiolytic, or sedative medication could result and potentially contribute to the aggravation of symptoms (Bilevicius et al., 2018; Bisson et al., 2018; May & Schulte, 2016).

It is worth mentioning that beyond 14–15 months posttrauma we found no evidence for cross-lagged associations between somatic symptoms and PTSS over time. This finding could lend support to prior research results that have indicated that PTSS may be particularly malleable in the early posttrauma phase, whereas long-term symptoms tend to follow more rigid, chronic trajectories (Steinert et al., 2015).

The present results should be considered in light of its strengths and limitations. Strengths of this study include the longitudinal design; high participation rates; high, relatively homogeneous exposure among the survivors; and the use of continuous measures of PTSS and somatic symptoms (Koenen, Sumner, et al., 2017; Pacella et al., 2013). Moreover, the survivors seem to constitute a fairly representative sample of the same-age Norwegian population (Stensland et al., 2018). In addition, the fact that the Utøya terrorist attack did not coincide with exposure to toxicants, radiation, other contaminants (Lucchini et al., 2017), or severely adverse living conditions, which often precede or follow a traumatic event (Koenen, Sumner, et al., 2017; Williams et al., 2009), increases the validity of the findings. Further, we were able to mitigate the risk of confounding by adjusting for known risk factors (Shalev et al., 2019), including injury (Bugge et al., 2017).

A major study limitation relates to the fact that we could not study the mechanisms at play prior to the first assessment at 4–5 months posttrauma. Deviation from prior findings could relate to sample-specific factors in prior studies of population subgroups, such as the injured, veterans, or treatment-seeking individuals. The types of trauma exposure and time since trauma, as well as differences in study methodology, such as variation in measures of PTSS and somatic health and statistical models, could have affected the results (Koenen, Ratanatharathorn, et al., 2017). For example, the measure of physical health problems (CSSI) used in the present study was primarily developed for use in children and adolescents (Walker et al., 2016). The scale was included in this study because the majority of participants were adolescents at T1. This measure was used for all participants across all time points to enable longitudinal analyses, although some participants were older at T1, and others could no longer be considered to be adolescents at later assessments. Although the CSSI is highly similar to the Somatic Symptoms subscale of the Hopkins Symptom Checklist (Derogatis et al., 1974), we cannot rule out that our results may have been affected by our choice of measures. Further, the young participants in this study reported relatively low levels of PTSS (Dyb et al., 2014). However, their levels of PTSS and somatic symptoms, such as headaches, as reported 4–5 months posttrauma (i.e., Wave 1) was 4–6 times higher than expected among unexposed peers (Stensland et al., 2018; Thoresen et al., 2012). In a previous paper, we demonstrated that participants with a high symptom level were less likely to participate in Wave 1 (Stene & Dyb, 2016), indicating that nonparticipating survivors may have been too ill to take part or were concerned about the potential strain involved in participating in the interview study. Hence, replication is needed in other settings and clinical samples. Although it is well known that female girls and women often display higher posttraumatic levels of health problems than their male counterparts, we had no particular hypothesis about gender differences in the sequential relation between mental and physical health. In addition, our sample size did not allow for subgroup analyses. Hence, potential gender differences might have gone undetected in our study. Further, we could not identify or control for any potential effect of interventions or treatment. In conclusion, early somatic symptoms predict later psychopathology following trauma exposure. Early identification of survivors' somatic needs and the provision of adequate services may represent untapped potential, increasing the efficiency of intervention efforts in the aftermath of a traumatic event.

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