Research Article

MEDIA EXPOSURE AND SYMPATHETIC NERVOUS SYSTEM REACTIVITY PREDICT PTSD SYMPTOMS AFTER THE BOSTON MARATHON BOMBINGS

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Background: *Terrorist attacks have been shown to precipitate posttraumatic stress disorder (PTSD) symptomatology in children and adolescents, particularly among youths with high exposure to media coverage surrounding such events. Media exposure may be particularly likely to trigger PTSD symptoms in youths with high physiological reactivity to stress or with prior psychopathology or exposure to violence. We examined the interplay between media exposure, preattack psychopathology, autonomic nervous system (ANS) reactivity, and prior violence exposure in predicting PTSD symptom onset following the terrorist attack at the 2013 Boston Marathon.* **Methods:** *A community sample of 78 adolescents (mean age = 16.7 years, 65% female) completed a survey about the bombings, including media exposure to the event and PTSD symptoms. All respondents participated in a study assessing psychopathology prior to the attack, and sympathetic and parasympathetic reactivity to a laboratory-based stressor was assessed in a subset (N = 44) of this sample. We examined the associations of media exposure, ANS reactivity, preattack psychopathology, and prior violence exposure with onset of PTSD symptoms related to the bombings.* **Results:** *Media exposure, preattack psychopathology, and prior violence exposure were associated with PTSD symptoms. Moreover, media exposure interacted with sympathetic reactivity to predict PTSD symptom onset, such that adolescents with lower levels of sympathetic reactivity developed PTSD symptoms only following high exposure to media coverage of the attack.* **Conclusions:** *We provide novel evidence that physiological reactivity prior to exposure to an unpredictable traumatic stressor predicts PTSD symptom onset. These findings have implications for identifying youths most vulnerable to PTSD following wide-scale trauma. Depression and Anxiety 31:551–558, 2014.*

Key words: *media exposure; sympathetic nervous system; terrorism; posttraumatic stress disorder; stress*

INTRODUCTION

The terrorist attack at the 2013 Boston marathon resulted in the death of three spectators and injured

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hundreds of other bystanders. Four days later, residents of Boston and surrounding communities were confined to their homes during an unprecedented shelter-in-place request as police searched for the perpetrators. Schools and most workplaces were closed, and many residents watched the manhunt in real time on television. Considerable research demonstrates that exposure to terrorist attacks can exert a significant toll on the mental health of adults and children, including onset of posttraumatic stress disorder (PTSD) symptoms. Over 20% of New York City residents living in close proximity to the World Trade Center during the 9/11 attacks met criteria for PTSD two months later.[1] Similar epidemiological studies of the Oklahoma City bombings[2] and Israelis exposed to suicide bombings and terrorist attacks[3] indicate that PTSD symptoms develop in a substantial minority of individuals following terrorist attacks. However, the premorbid conditions that make some youths more susceptible to symptom onset following such events have yet to be fully identified.

The emergence of PTSD symptoms—including intrusive memories, avoidance, and physiological hyperarousal—has been linked to exposure to media coverage of terrorist attacks, particularly among children and adolescents.[4–8] Surveys conducted following 9/11 indicated that children watched an average of 3 hrs of media coverage on the day of the attacks, with 23 percent watching for 5 hrs or more.[9] Indirect exposure to these events likely elevates perceptions of personal risk and disrupts daily routines and activities,[8,10] which might increase risk for PTSD symptomatology. High levels of media exposure to terrorist attacks may be particularly detrimental for individuals who are already at risk for developing PTSD.

Although a variety of risk factors have been identified for child and adolescent PTSD, we focus here on three sets of factors consistently linked to PTSD vulnerability.[11,12] First, recent evidence from both prospective and cross-sectional population-based studies suggests that the vast majority of individuals who develop PTSD have a history of prior psychopathology, particularly anxiety, and mood disorders.[12,13] These findings suggest that a history of internalizing psychopathology is an important vulnerability marker for PTSD. Second, increasing evidence suggests that prior exposure to traumatic events or environmental adversities can increase risk for PTSD along with other mood and anxiety disorders following subsequent exposure to stressors, a mechanism commonly known as "stress sensitization."[14–17] Indeed, several prospective studies have demonstrated that pretrauma adversity exposure increases the odds of PTSD onset.[18,19] Finally, recent studies point to the role of autonomic nervous system (ANS) function as a risk factor for PTSD.[20–22] The ANS is a physiological index of stress sensitivity and reactivity,[23,24] and evidence suggests that physiologically reactive individuals are more susceptible to environmental stressors.[25] Studies with adults have reported differences in physiological reactivity between individuals with and without PTSD, including increased startle response.[26,27] However, these studies have predominately used cross-sectional designs, making it difficult to establish whether these differences represent antecedents or sequelae of PTSD.

The only prospective evidence for pretrauma, physiological risk factors for PTSD come from two studies of police and firefighter trainees.[28,29] Both studies reported positive associations between pretrauma physiological reactivity and severity of PTSD symptoms following duty-related traumas. However, to date, no study of children or adolescents has assessed exposure to traumatic events and physiological reactivity prior to a traumatic stressor.

In the current study, there was a unique opportunity to explore whether individual differences in stress physiology, internalizing symptoms, and trauma exposure, measured prior to a traumatic event, confer vulnerability to PTSD symptomatology in adolescents exposed to a terrorist attack. We hypothesized that adolescents with high levels of preattack internalizing symptoms and either high sympathetic or low parasympathetic reactivity would be particularly likely to develop PTSD symptoms following the attack. Consistent with the stress sensitization model, we hypothesized that previous violence exposure would predict PTSD symptom onset. We additionally examined whether associations between pretrauma risk factors and PTSD symptoms varied as a function of exposure to media coverage of the event. Several features of the terrorist attack in Boston (i.e. real-time media coverage, uncertainty about the identity or whereabouts of the perpetrators, shelter-in-place request, firefights, and car chases in residential neighborhoods), combined with previous research, led us to expect high levels of media exposure to these events and a positive association between such exposure and PTSD symptoms.

METHODS

PARTICIPANTS

A community-based sample of 78 adolescents aged 14–19 (65% female) living in the Boston metropolitan area at the time of the terrorist attacks was recruited for participation in this study. All participants had previously completed studies in our lab, including a subset ($N = 44$; 64% female) who had also completed a study assessing ANS reactivity to stress. Participants were recruited for two separate studies with different goals, leading to differences in sample composition. Accordingly, participants with ANS data had significantly lower preattack anxiety, depression, and violence exposure than those without ($Ps < .01$), but did not differ on age or gender. A total of 215 individuals (69 adolescents 18 years and older and 146 adolescents younger than 18 years) or their parents (i.e. for minors) were contacted and invited to participate in an online survey during a two-week period beginning one month following the terrorist attack. Of the total sample, 20 adolescents 18–19 years old (29%) and 58 adolescents 14–17 years old (40%) agreed to participate. Nonresponse was almost exclusively related to inability to reach the adolescent or parent/guardian by email/telephone during the two-week time period of data collection. Each participant who completed the survey was given a $5.00 gift card. Participants ranged from

14 to 19 years of age at the time of the survey ($M = 16.7$ years; 65% female). The sample was diverse with respect to race/ethnicity: 45.5% White, 18.2% Asian, 11.7% Black, 5.2% Latino, 18.2% multi-racial, and 1.3% from other racial/ethnic groups.

PROCEDURE

A subset of 44 participants completed the Trier Social Stress Test (TSST) approximately one year prior to the Boston marathon. The TSST is a standardized experimental protocol that has been shown to elicit a physiological response in clinical and nonclinical child and adolescent populations.[30,31] The TSST has several phases, each of which lasts 5 min: speech preparation, speech, and math. Throughout all phases of the TSST, continuous cardiac measures were measured noninvasively (see below). Upon arrival to the laboratory, baseline measures of ANS activity were measured while participants sat quietly in a chair (Baseline). Participants were then told they would be delivering a speech about "what makes a good friend" to two experimenters. It was emphasized that performance was important, that the experimenters were trained evaluators of public speaking, and that the speech would be video-taped and reviewed by other experts later. Adolescents were given 5 min to prepare their speech (Speech Preparation). Participants then delivered a 5 min speech to the experimenters (Speech). During this period, the experimenters provided only neutral or mildly negative feedback (e.g. appearing bored). No positive feedback was provided. Next, participants were asked to count backwards out loud from 758 in steps of 7 (Math). After the experiment was complete, adolescents were debriefed about the experimental protocol. Consistent with other literature using the TSST, reactivity was greatest to the Speech, which is the part of the task we examine here. A complete description of stress reactivity procedures in this prior study is available elsewhere.[32,33]

PHYSIOLOGICAL MEASURES

Electrocardiogram (ECG) recordings were acquired using a Biopac ECG amplifier (Goleta, CA), using a modified Lead II configuration (right clavicle, left lower torso, and right leg ground). Cardiac impedance recordings were obtained using a Bio-Impedance Technology model HIC-2500 impedance cardiograph (Chapel Hill, NC). One pair of Mylar electrodes was placed on the neck and another pair was placed on the torso. Biopac MP150 hardware and Acknowledge software were used to acquire the ECG and impedance cardiography data, both sampled at 1.0 kHz. ECG and impedance cardiography data were inspected by trained research assistants who were blind to participant identity, and scored using Mindware Heart Rate Variability (HRV) Software (Mindware Technologies, Gahanna, OH).

Respiratory sinus arrhythmia (RSA) was calculated from the interbeat interval time series using spectral analysis implemented in Mindware HRV Software. RSA was calculated for the frequency band 0.12–0.40 Hz. To ensure that RSA represents a measure of pure parasympathetic cardiac control,[34] respiration rate was derived from basal cardiac impedance signal and included in all analyses as a covariate. Preejection period (PEP) was calculated from impedance cardiography scoring. PEP represents the time interval beginning with ventricular depolarization and ending when blood is ejected from the left ventricle (electrical systole). Shorter intervals represent greater sympathetic activation.[35] As scoring of impedance cardiography data requires manual placement of the B point (the opening of the aortic valve),[36] these data were independently scored by two raters and differences of more than 5% were reviewed and adjudicated by the second author (McLaughlin).

PSYCHOPATHOLOGY

PTSD symptoms were assessed using the Impact of Events Scale-6 (IES-6), a six-item report of PTSD symptoms following exposure to a traumatic event.[37] The IES-6 is an abbreviated form of the Impact of Events Scale—Revised,[38] which is a widely used and well-validated screener for PTSD symptoms. Respondents rated the frequency of hyperarousal (e.g. "I had trouble concentrating"), intrusive thoughts (e.g. "I thought about it when I didn't want to") and avoidance (e.g. "I tried not to think about it") experienced since the week of the bombings, on a 5-point Likert scale ranging from 0 ("almost never") to 4 ("almost always"). Previous studies have shown that the IES-6 explains most of the variance of the IES-R,[37] and the measure demonstrated good internal consistency in our sample ($\alpha = .89$).

Premarathon depressive symptoms were assessed using the Childhood Depression Inventory (CDI).[39] The CDI is a 27-item self-report measure of depressive symptoms in children and adolescents aged between 8 and 17 years. For each item, the child selects one of three items that best describe them over the past two weeks (e.g. "I feel like crying everyday," "I feel like crying many days," "I feel like crying once in a while"). Each item was scored by assigning a number according to symptom severity (0 = mild; 1 = moderate; 2 = severe), and then summing across all items to generate a total score. The CDI's reliability and validity have been documented in several studies,[39,40] and demonstrated excellent internal consistency in our sample ($\alpha = .96$).

Premarathon anxiety symptoms were assessed using the Multidimensional Anxiety Scale for Children (MASC).[41] The MASC is a 38-item self-report measure that assesses the presence of anxiety symptoms in youth. Subscales include physical symptoms ("I feel restless and on edge"), social anxiety ("I get nervous if I have problems in public"), harm avoidance ("I stay away from things that upset me"), and separation anxiety ("I avoid going places without my family"), on a 4-point Likert scale from 1 ("never true") to 4 ("very true"). The MASC has been validated in a number of previous studies,[41,42] and demonstrated excellent internal consistency in our sample ($\alpha = .96$).

EXPOSURE MEASURES

Prior violence exposure was assessed using the Screen for Adolescent Violence Exposure (SAVE).[43] The SAVE is a 32-item measure of adolescents' exposure to direct and indirect violence in school, home, and neighborhood contexts. Items are rated on a 5-point Likert scale to indicate the frequency of indirect (e.g. "I hear gun shots") or direct (e.g. "someone has pulled a knife on me") exposures, on a scale from 1 ("never") to 4 ("almost always"). The SAVE has demonstrated good reliability and validity in samples of inner-city youth.[43] The SAVE demonstrated good reliability in our sample ($\alpha = .88$).

Media exposure to the marathon was measured by participants' response to two questions asking "approximately how much of the day on the day on Monday 15th/Friday 19th did you spend watching news coverage of the event on a TV, computer, iPad, or other electronic device?" These questions were on a Likert scale ranging from 1 ("None") to 5 ("All Day") and were summed to create an overall media exposure composite, with higher scores reflecting greater exposure.

DATA ANALYSIS

First, we examined the main effects of media exposure, pretrauma psychopathology, PEP, and RSA reactivity, and prior violence exposure on PTSD symptoms. Consistent with previous studies using the TSST,[44,45] we calculated sympathetic and parasympathetic reactivity to the TSST by subtracting RSA/PEP measured when participants first began the task (first minute of Baseline) from RSA/PEP measured during the speech portion of the test (first minute of Speech), as this was the period associated with the greatest physiological response. Next, in order to test our hypothesis that media exposure would moderate

TABLE 1. Descriptive statistics for RSA and PEP (baseline and speech; $N = 44$), violence exposure, psychopathology, and demographic variables ($N = 78$)

	Mean	SD	Range
Age	16.72	1.33	14–19
CDI	11.53	6.61	0–25
MASC	49.22	18.43	9–85
SAVE	46.95	9.90	32–85
RSA–Baseline	6.84	1.39	1.89–9.71
RSA–Speech	6.00	1.41	1.09–8.56
RSA–Math	6.20	1.51	.54–8.22
PEP–Baseline	102.78	12.83	62–189
PEP–Speech	84.27	19.16	50–123
PEP–Math	90.00	18.41	50–127
Media exposure	6.33	2.13	2–10
IES-6	5.75	5.31	0–22

CDI, childhood depression inventory; MASC, multidimensional anxiety scale for children; SAVE, screen for adolescent violence exposure; IES-6, impact of events scale–6 item.

the association between pretrauma risk factors and PTSD symptoms, we generated two-way interaction terms between the two physiological reactivity variables (parasympathetic and sympathetic), pretrauma anxiety and depression, and media exposure. For all significant interactions, simple slopes were evaluated using procedures described by Aiken and West.[46] Age and gender were included as covariates in all regression models, and respiration rate for all models involving RSA. All statistical analyses were conducted using Stata for Mac (StataCorp, College Station, TX).

RESULTS

DESCRIPTIVE STATISTICS AND BIVARIATE ASSOCIATIONS

Table 1 provides information on demographic variables, RSA and PEP at baseline and the speech portion of the TSST, violence exposure, and pretrauma psychopathology. Zero-order correlations between control variables (age and gender), predictors (prior anxiety and depression, prior violence exposure, and PEP/RSA reactivity), and the outcome (PTSD symptoms) are presented in Table 2. The full distribution of responses for the survey question assessing media exposure on both May 15th and May 19th is displayed in Fig. 1. Notably, PTSD symptoms after the attack were correlated with premorbid anxiety and depression symptoms and prior violence exposure, but not with PEP or RSA reactivity.

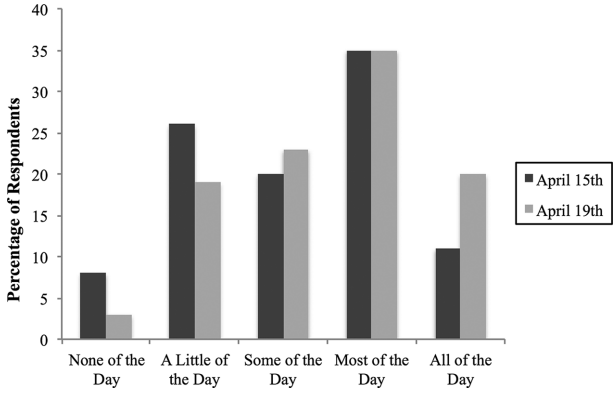

Figure 1. Exposure to media coverage on the day of the Boston Marathon bombing (April 15th) and the day of the manhunt and shelter in place request (April 19th).

MAIN EFFECTS OF MEDIA EXPOSURE, PRETRAUMA PSYCHOPATHOLOGY, ANS REACTIVITY, AND PRIOR VIOLENCE EXPOSURE

Media exposure, prior violence exposure, pretrauma depression, and pretrauma anxiety were significantly associated with PTSD symptoms. No significant main effects were found for PEP or RSA reactivity. Coefficients, standard errors, and *P*-values for these models are presented in Table 3.

TWO-WAY INTERACTIONS WITH MEDIA EXPOSURE

Next, we examined whether media exposure moderated the association between pretrauma risk factors and

TABLE 2. Correlations between RSA and PEP reactivity, violence exposure, psychopathology, and demographic variables

	1	2	3	4	5	6	7	8	9
1. Age	–								
2. Female	−.03	–							
3. CDI (depression symptoms)	.10	.19	–						
4. MASC (anxiety symptoms)	−.04	.26*	.68**	–					
5. SAVE (violence exposure)	.12	.26*	.40**	.38**	–				
6. RSA reactivity	.15	−.07	.11	.03	−.06	–			
7. PEP reactivity	.05	−.28	−.06	−.29	−.00	.43**	–		
8. Media exposure	.23	.09	.28*	.14	.20	.16	.05	–	
9. IES-6 (PTSD symptoms)	.03	.18	.41**	.29*	.35**	−.01	−.12	.26*	–

Note: *$P < .05$ **; $P < .01$.
CDI, childhood depression inventory; MASC, multidimensional anxiety scale for children; SAVE, screen for adolescent violence exposure; IES-6, impact of events scale 6.

TABLE 3. Results for regression models predicting PTSD symptoms

	β	(SE)	*P*-value
Main effects			
Media exposure	.64	.31	.042
Violence exposure	.22	.06	.005
Prior anxiety	.08	.03	.027
Prior depression	.32	.09	.001
RSA reactivity	.16	.29	.729
PEP reactivity	−.03	.06	.567
Interaction effects			
RSA reactivity × media	−.11	.22	.612
PEP reactivity × media	.08	.03	.021

Note: All models control for age and gender, and models with RSA control for respiration rate.

PTSD symptoms. PEP reactivity interacted significantly with media exposure ($\beta = .08$, $P = .021$). In order to further assess this finding, we plotted the association between media exposure and PTSD symptoms at +1 and –1 standard deviations of PEP reactivity (see Fig. 2). The relationship between media exposure and PTSD symptoms was significant for adolescents with low PEP reactivity ($\beta = 1.82$, $P = .002$), but not for adolescents with high PEP reactivity ($\beta = -.63$, $P = .40$). Further post-hoc analyses revealed that at low media exposure (–1 SD), adolescents with high PEP reactivity experienced significantly greater PTSD symptoms than adolescents with low PEP reactivity, $F(1,34) = 5.78$, $P = .02$. At high levels of media exposure (+1 SD), this difference was nonsignificant, $F(1,34) = 1.85$, $P = .18$. No significant interactions were found between media exposure and parasympathetic reactivity, nor with premorbid anxiety and depression.

As prior anxiety was marginally correlated with PEP, we ran a final model where we examined whether the interaction between PEP and media exposure predicted PTSD symptoms, controlling for prior anxiety. In this model, the interaction remained marginally significant ($\beta = .07$, $P = .061$).

DISCUSSION

Identifying predictors of child and adolescent mental health in the wake of mass trauma—including terrorist attacks—is critical for improving screening and detection efforts and the delivery of preventive interventions to youths most vulnerable to the negative sequelae of these events. Maladaptive patterns of ANS reactivity are putative risk factors underlying the development of psychopathology, including PTSD.[47–49] Sympathetic reactivity measured shortly after a trauma has been associated with an increased likelihood of developing PTSD symptoms, in both children[50,51] and adults.[27] However, we are unaware of a single prospective study of children or adolescents that has investigated physiological reactivity measured prior to a traumatic event as a risk factor for PTSD. In this study, we provide novel evidence that heightened sympathetic reactivity measured prior to a terrorist attack interacts with degree of media exposure to the attack to predict the onset of PTSD symptoms in adolescents. In contrast, we found no effect of parasympathetic reactivity on PTSD symptoms, nor an interaction between parasympathetic reactivity or pretrauma psychopathology with media exposure. Preattack violence exposure and anxiety and depressive symptoms also predicted PTSD, but did not vary according to media exposure. These findings support the hypothesis that sympathetic nervous system reactivity and pretrauma internalizing psychopathology are involved in the pathophysiology of PTSD.[20]

Our analyses show that adolescents with high sympathetic reactivity to a laboratory stressor exhibited elevated risk for PTSD symptoms as compared to adolescents with low sympathetic reactivity, in the context of low levels of exposure to media coverage of the attacks. These findings are consistent with a number of previous studies that have demonstrated associations between high sympathetic reactivity and internalizing problems in children.[25,52,53] We extend previous findings by demonstrating that sympathetic reactivity measured prior to a traumatic event predicts onset of PTSD symptoms. In contrast, we found no effect of

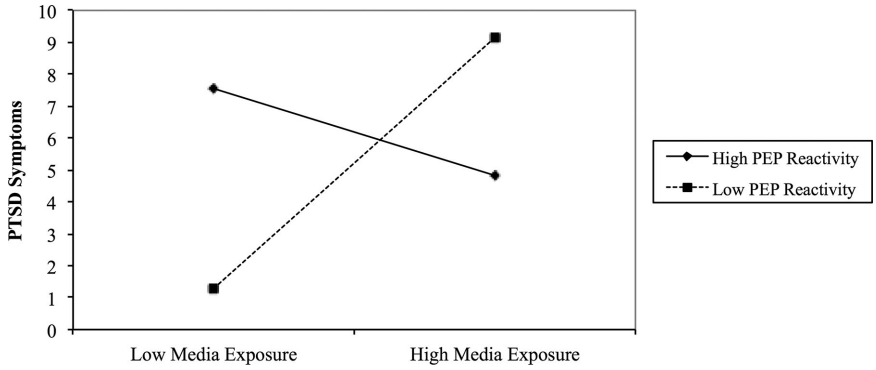

Figure 2. Interaction of media exposure and pretrauma sympathetic reactivity on PTSD symptoms. Fitted lines indicate predicted values for adolescents with high sympathetic reactivity (–1SD PEP) and low reactivity (+1SD PEP). Models control for age and gender.

parasympathetic reactivity on PTSD symptoms or significant interactions between parasympathetic reactivity and media exposure.

Our finding that media exposure was associated with greater onset of PTSD symptoms is consistent with prior studies.[6,8,54] In the context of high levels of media exposure, youth with high and low sympathetic reactivity exhibited equally high levels of PTSD symptoms. For those not directly exposed to these kinds of events, news reports are the primary vehicle through which individuals learn about and experience terrorist acts. Media exposure has previously been found to predict a wide range of functional outcomes in children following terrorist attacks, including not only PTSD symptoms but also sleep problems and behavioral withdrawal.[6,9,55] Parents are likely to have an important role to play in moderating their children's exposure to potentially distressing news coverage. However, as Comer et al.[56] acknowledge, parents and other caregivers may increase their children's media literacy (e.g. educating them about the dramatized coverage of events present in most news reports). Taken together, these findings suggest that parents should make efforts to limit youth's exposure to media coverage following terrorist attacks or other potentially traumatic events. Moreover, in spite of concerns about recent expansions in the definition of trauma,[57] our findings suggest that even lower intensity stressors may precipitate symptoms of distress and impairment. However, we have no evidence that these symptoms of distress would emerge as PTSD in the future as we only measure symptoms of PTSD dimensionally.

Finally, we observed an association between prior violence exposure and adolescent symptoms of PTSD following the terrorist attack. This is consistent with prior studies that have reported elevated risk for internalizing psychopathology in the wake of trauma exposure among individuals with a history of prior exposure to environmental adversity (i.e. stress sensitization effects), including for PTSD.[14–17] Prior experiences of trauma and environmental adversity may create a diathesis for internalizing psychopathology that is exacerbated by exposure to additional stressors. Previously, it has been hypothesized that a potential mechanism underlying this stress sensitization effect involves the impact of early experiences of adversity on the development of the ANS and patterns of ANS reactivity.[22] Although sympathetic reactivity was associated with postmarathon PTSD symptoms, prior violence exposure was not related to reactivity. The lack of association between violence exposure and sympathetic reactivity in our sample should be interpreted with caution given our small sample size. Prior violence exposure was associated with the onset of PTSD symptoms regardless of levels media exposure, capturing the central importance of early adversity in predicting response to future traumatic events.

Our findings should be viewed in light of several limitations. First, the response rate to our online survey was low, limiting the sample size, and making it likely that we identified a nonrandom sample of adolescents in the Boston area. The low response rate was likely a result of the short window of data collection following the bombings (i.e. 2 weeks), and the difficulty in reaching parents via email to provide consent. Second, we used a self-report questionnaire rather than a structured clinical interview to measure PTSD symptoms. Future research is needed to examine whether patterns of physiological reactivity, exposure to media coverage, and prior violence are related to onset of PTSD. Third, the study design precludes us from making a strong inference about the direction of causality between adolescents' media exposure and their degree of PTSD symptoms. For example, although it is plausible that exposure to media resulted in increased symptoms, it is also possible that the most distressed adolescents sought out more news coverage of the event. Fourth, most adolescents in our sample were exposed to the attacks primarily through the media. Based on the DSM-5 definition of traumatic events,[58] such exposures do not qualify as traumatic. We nevertheless observed PTSD symptoms related to the attacks in many youths in our sample. Finally, the present study only assessed PTSD symptoms one month after the attacks. As such, our assessment might have missed youths with delayed reactions to the attacks. This is unlikely, as a prior study found that PTSD symptoms after 9/11 were largely resolved within 6 months.[59] Nevertheless, further research is needed to chart the trajectory of symptoms over time following these types of mass trauma.

CONCLUSION

This represents one of the first studies to identify physiological risk factors for PTSD symptom onset in youths following a terrorist attack. Our findings demonstrate that sympathetic reactivity is a risk factor for the onset of PTSD symptoms. Moreover, we show that adolescents who were exposed to high levels of violence prior to the Boston Marathon bombings had increased PTSD symptoms following the attack, indicating that a history of exposure to childhood adversity places adolescents at risk for PTSD symptoms following future stressors. Future prospective research is needed to characterize the interplay of neurobiological and environmental factors in shaping vulnerability to PTSD in children and adolescents.

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