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Cumulative Exposure to Prior Collective Trauma and Acute Stress Responses to the Boston Marathon Bombings

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Abstract

The role of repeated exposure to collective trauma in explaining response to subsequent community-wide trauma is poorly understood. We examined the relationship between acute stress response to the 2013 Boston Marathon bombings and prior direct and indirect media-based exposure to three collective traumatic events: the September 11, 2001 (9/11) terrorist attacks, Superstorm Sandy, and the Sandy Hook Elementary School shooting. Representative samples of residents of metropolitan Boston (n = 846) and New York City (n = 941) completed Internet-based surveys shortly after the Boston Marathon bombings. Cumulative direct exposure and indirect exposure to prior community trauma and acute stress symptoms were assessed. Acute stress levels did not differ between Boston and New York metropolitan residents. Cumulative direct and indirect, live-media-based exposure to 9/11, Superstorm Sandy, and the Sandy Hook shooting were positively associated with acute stress responses in the covariate-adjusted model. People who experience multiple community-based traumas may be sensitized to the negative impact of subsequent events, especially in communities previously exposed to similar disasters.

Keywords

collective trauma, terrorism, acute stress

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On April 15, 2013, two pressure-cooker bombs exploded near the Boston Marathon finish line. Three people died, 264 were injured, and neighborhoods were locked down during the hunt for the perpetrators; events were widely televised, so many Americans were exposed to potentially disturbing images of chaos, injury, and death. Collectively experienced traumas like this can have widespread effects on mental and physical health for people who were exposed either directly (i.e., they themselves or close others were present at the event; Brackbill et al., 2009; Nair et al., 2012) or indirectly through live media (Goodwin, Palgi, Hamama-Raz, & Ben-Ezra, 2013; Holman, Garfin, & Silver, 2014; Silver et al., 2013). Repeated exposure to community disasters may take a cumulative toll on well-being, especially given pervasive dissemination of disaster-related information and images through traditional and social media.

The probability of being exposed indirectly to events like the Boston Marathon bombings (BMB) is also rising: Reports indicate that Americans increasingly obtain news in real time through online news, mobile-phone applications, and social-networking sites (Pew Center for Research, 2013). Thus, people have more opportunities to view collective traumas as they occur and to accumulate exposures over time. Moreover, these exposures may influence people's responses to future events: Sensitization and habituation are two possible response patterns documented in prior studies of response to individual trauma (see Franklin, Saab, & Mansuy, 2012; Rutter, 2012; Seery, Holman, & Silver, 2010).

Sensitization models of trauma exposure (e.g., Hammen, Henry, & Daley, 2000; Post, 1992) posit that

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prior adversity may heighten sensitivity to negative outcomes following subsequent stress. Such effects were seen in a sample of workers exposed to an airline crash (Dougall, Herberman, Delahanty, Inslicht, & Baum, 2000); epidemiological research has also demonstrated that prior trauma exposure may increase the risk of developing posttraumatic stress disorder after subsequent trauma (Breslau, Chilcoat, Kessler, & Davis, 1999; McLaughlin, Conron, Koenen, & Gilman, 2010). Alternatively, prior exposure to trauma may protect against adverse responses through habituation processes. Laboratory (Wüst, Federenko, van Rossum, Koper, & Hellhammer, 2005) and field (Andersen, Silver, Stewart, Koperwas, & Kirschbaum, 2013; Resnick, Yehuda, Pitman, & Foy, 1995) studies of stress-related physiology demonstrate that stress-hormone responses can become less intense after repeated exposure to similar stressors. Epidemiological studies of repeated exposure to flooding in Kentucky (Norris & Murrell, 1988) and to terrorism in Israel (Bleich, Gelkopf, & Solomon, 2003) also document minimal psychopathology despite high trauma exposure. A third alternative is suggested by research demonstrating a quadratic relationship between exposure to negative life events and psychological outcomes: People who reported some exposure to negative life events fared better than people who reported either no prior exposure or many prior exposures to such events (Seery et al., 2010). This suggests that people may develop psychological "toughness" or resilience after experiencing a moderate amount of adversity (Dienstbier, 1989). To our knowledge, no research has examined these three alternatives in the context of repeated direct exposure and indirect, media-based exposure to collective traumas.

Acute stress responses may occur following exposure to a traumatic event and are composed of a cluster of psychological symptoms (dissociation, reexperiencing, avoidance, arousal) that occur within the first month (see the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders*, or *DSM–5*; American Psychiatric Association, 2013). Note that media-based trauma exposure does not officially qualify as a potential trigger for serious mental-health responses (e.g., acute and posttraumatic stress disorder) unless related to professional duties (American Psychiatric Association, 2013). Nonetheless, indirect exposure to collective stressors through the media has been associated with acute stress symptoms comparable to (or stronger than) those linked to direct exposure (Holman et al., 2014). Moreover, the presence of acute stress symptoms following indirect, media-based exposure to collective traumas has been prospectively associated with negative physical- (Holman et al., 2008) and mental-health (Brewin, Andrews, Rose, & Kirk, 1999; Silver et al., 2013) outcomes. This suggests that acute responses may help identify individuals at risk for trauma-related mental and physical disorders over time, making them an important risk marker to assess in the early aftermath of trauma. However, little is known about the role of cumulative direct and indirect, media-based exposure to community trauma in shaping early response to subsequent trauma, because logistical challenges (e.g., securing funding and gaining ethics-board approval) often preclude immediate assessment of acute responses after collective traumas. Exploring the association between cumulative exposure to prior collective trauma and acute stress response to subsequent collective trauma could also answer important theoretical questions about the role of cumulative exposure in sensitizing individuals to or inoculating them against negative responses to subsequent trauma.

Using representative samples of residents from metropolitan Boston and New York City—the site of America's previous large-scale terrorist attack—we examined the relationship between BMB-related acute stress symptoms and cumulative counts of prior direct and indirect exposure to three high-profile collective traumas that occurred on the East Coast of the United States: the September 11, 2001 (9/11) terrorist attacks, Superstorm Sandy, and the Sandy Hook Elementary School shooting. We tested whether cumulative exposure to prior collective trauma would (a) sensitize individuals to react more negatively to the BMB, (b) result in habituation processes minimizing acute stress symptoms, or (c) exhibit a quadratic effect on responses.

Method

Design, sample, and data collection

Between April 29 and May 13, 2013, we conducted an Internet-based survey with representative samples of residents from metropolitan Boston and New York City, drawn from the GfK KnowledgePanel. GfK uses address-based sampling methods to randomly sample and recruit potential panelists. To ensure panel representativeness, GfK provides compensation or free Internet service (with computer if needed) as an incentive for participation in their Web-based surveys. Our survey was fielded to representative subsamples of Boston (n = 1,021) and New York (n = 1,231) metropolitan residents from the KnowledgePanel. To encourage participation, GfK used e-mail and telephone reminders. We received 1,787 completed surveys (Boston: n = 846, 82.9% participation rate; New York: n = 941, 76.4% participation rate), for an overall participation rate of 79.4%. Seventy-four percent of respondents completed the survey by May 7, 2013, within 2.5 weeks of the end of the lockdown of Boston neighborhoods and the manhunt for the perpetrators. (A national sample was also surveyed, but their data are not

relevant to this report. Full details of the sampling design are described by Holman et al., 2014.) All procedures for this study were approved by the institutional review board of the University of California, Irvine.

The panel-selection methods provide statistical control of the representativeness of GfK panel samples and ensure samples' comparability to the general population (for detailed demographic comparisons between data from the GfK panel and data from the U.S. Census Bureau, see Holman et al., 2014). Panel design weights are calculated to reflect unequal selection probabilities (according to demographic categories) for different sampled members of the KnowledgePanel. Subsequently, design weights are poststratified to the benchmarks from the most recent U.S. government statistics to compensate for any differential nonresponse to the survey. Consequently, the weighted composition of our sample closely matched that of the target population as defined by the benchmarks from the American Community Survey of the U.S. Department of Commerce (U.S. Census Bureau, 2012).

The final weights were computed using the method of iterative proportional fitting (commonly referred to as raking) along the following dimensions: age (18–29, 30–44, 45–59, 60+), gender, race-ethnicity (Hispanic, non-Hispanic White, non-Hispanic Black, non-Hispanic other, non-Hispanic multiracial), education (less than high school, some college, bachelor's degree or higher), annual income ($0–$24,999, $25,000–$49,999, $50,000–$74,999, $75,000+), and Internet access (yes, no). When sample sizes permitted, variables were crossed (e.g., age and gender) so that joint distributions could be used to adjust weights. As needed, categories of weighting variables were collapsed to increase samples of available respondents and avoid the creation of extreme weights.

Measures

Acute stress response to the BMB and its aftermath was assessed with the Stanford Acute Stress Reaction Questionnaire (Cardeña, Koopman, Classen, Waelde, & Spiegel, 2000), a well-validated and reliable measure of acute stress symptoms. Respondents used a 6-point scale from 1 (*not experienced*) to 6 (*very often experienced*) to describe how often they had experienced 30 possible reactions "since the Boston Marathon bombings and their aftermath" (e.g., "I try to avoid thoughts about the Boston Marathon bombings and their aftermath," "I feel hypervigilant or 'on edge'"). Responses were summed (range: 30–180) to create a continuous score for acute-stress symptoms (which is standard practice in postdisaster research; Cardeña et al., 2000) and to capture maximum variability in potential responses (cf. MacCallum, Zhang, Preacher, & Rucker, 2002). Respondents were not assumed to have acute stress disorder because many did not meet *DSM–5* Acute Stress Disorder criterion A for direct exposure to a traumatic event (for full diagnostic criteria, see http://www.ptsd.va.gov/professional/treatment/early/acute-stress-disorder.asp).

Direct exposure to the BMB was assessed using a measure modified from prior research on disaster exposure (Holman & Silver, 1998; Koopman, Classen, & Spiegel, 1994). It included 15 items assessing respondents' (or their close others') proximity to the bombing site and subsequent lockdown (e.g., "I was a spectator at the Boston Marathon at the time of the bombings"). Responses were coded dichotomously according to whether they indicated (a) direct or no direct exposure to the BMB and (b) direct or no direct exposure to the subsequent Boston-area lockdown, consistent with the *DSM–5* criterion for direct exposure to a traumatic event (American Psychiatric Association, 2013): 0 = no direct exposure, 1 = self or close other exposed.

Cumulative direct exposure to collective trauma was defined according to the *DSM–5* Acute Stress Disorder criterion A (i.e., self or close other physically present; American Psychiatric Association, 2013). Direct exposure to 9/11, Superstorm Sandy, and the Sandy Hook Elementary School shooting was assessed dichotomously: 0 = no direct exposure, 1 = self or close other directly exposed. A count of cumulative direct exposure to these prior collective traumas (range: 0–3) was generated by summing the three dichotomous scores.

Cumulative indirect exposure to collective trauma was assessed by using participants' reports of having witnessed 9/11, Superstorm Sandy, or the Sandy Hook School shootings as they occurred in real time via (a) live television coverage or (b) live radio or online streaming: 0 = no live-media-based indirect exposure, 1 = live-media-based indirect exposure. Given the possibility of simultaneous indirect exposures via multiple sources (e.g., watching live television while streaming live coverage on another device), participants could report up to two possible indirect exposures for each of the three events; a count (range: 0–6) of total prior indirect exposure was calculated.

Demographics and mental-health history were collected by profile surveys administered to KnowledgePanelists before the BMB. Survey items regarding lifetime history of physician-diagnosed depression or anxiety disorders were modified from the Centers for Disease Control's National Center for Health Statistics annual National Health Interview Survey (U.S. Department of Health and Human Services, National Center for Health Statistics, 2000). Respondents were asked, "Has a medical doctor ever diagnosed you as suffering from any of the following ailments?" with

prompts for depression and anxiety. Just over 75% of respondents provided mental-health histories before the BMB. To retain sample representativeness, we imputed missing values for depression and anxiety using sequential hot-deck imputation (Andridge & Little, 2010; and see Holman et al., 2014). This method identifies the best predictors of each disorder and uses them to match appropriate donors from the data set with respondents who are missing the mental-health data. Hot-deck simulations are less sensitive to imputation-model misspecification than other methods and generate relatively low bias (Andridge & Little, 2010). For the analyses, prior mental health was coded as follows: 0 = no disorders, 1 = depression or anxiety, or 2 = depression and anxiety.

Analytic strategy

Statistical analyses were conducted using Stata (Version 11.1; Stata Corp, College Station, TX), a program well suited to handle complex, weighted survey data. We constructed a multivariate ordinary least squares regression model using a hierarchical variable-entry strategy to analyze correlates of continuous acute stress scores. Variables were entered in theoretically relevant blocks: (a) pre-BMB mental-health history and cumulative direct and indirect media-based exposure to prior collective traumas (9/11, Superstorm Sandy, and Sandy Hook shootings; Model 1), (b) metropolitan area of residence (New York or Boston) and direct exposure of self or close other to the BMB and to the Boston lockdown (Model 2), and (c) demographics (Model 3). Demographic variables were age, gender, ethnicity, education, income, and marital status (single; married; or divorced, widowed, or separated). Substituting employment status for income produced similar results. Demographic indicators were screened for statistical significance; for the sake of parsimony, those indicators with p values greater than .05 were not included in Model 3. Although some of the Boston panelists (n = 124) lived in the same households, their nonindependence was not associated with acute stress scores. Quadratic terms for both cumulative direct and cumulative indirect exposure to prior collective trauma were also calculated and tested for statistical significance.

All analyses were conducted with and without the final poststratification weights; the pattern of results remained the same. Analyses conducted with and without imputed data yielded comparable effect sizes. To retain sample size and demographic representativeness, we report all statistics using imputed values and poststratification weights.

Results

Table 1 presents regional rates of exposure to the BMB and other prior collective traumas. Figure 1 depicts mean acute stress scores by cumulative direct exposure to prior community trauma; Figure 2 presents mean acute stress scores by cumulative indirect exposure to prior community trauma. These figures illustrate that BMB-related acute stress responses increased with greater cumulative direct and cumulative indirect, media-based exposure to prior community trauma. More specifically, Table 2 presents standardized regression coefficients for predictors of BMB-related acute-stress-symptom scores for Models 1, 2, and 3. Metropolitan area of residence was not associated with BMB-related acute stress responses: Stanford Acute Stress Reaction Questionnaire scores were not significantly different for Boston residents (M = 44.70, SD = 18.67) than for New York residents (M = 45.14, SD = 21.46) in bivariate (β = −0.02, 95% confidence interval, or CI = [−0.12, 0.16]) or adjusted models. Personally being in or being close to someone who was in the Boston lockdown area was also not associated with acute stress responses in bivariate (β = 0.17, 95% CI = [−0.01, 0.35]) or adjusted models. Prior mental-health diagnoses and both direct and indirect exposure to prior collective traumas were correlated with higher acute stress scores in Model 1, after adjusting for BMB exposure (Model 2), and after adjusting for statistically significant demographic covariates (Model 3). Quadratic terms were not significantly associated with acute stress responses; a curvilinear relationship between cumulative trauma exposure and acute stress was not supported.

Discussion

We demonstrated that prior direct and indirect, live-media-based exposure to three recent community traumas in the northeastern United States appeared to sensitize residents of the Boston and New York areas to react more negatively to a subsequent community trauma—the BMB. Cumulative counts of both direct and indirect exposure to these prior collective traumas were positively associated with the frequency of BMB-related acute stress responses. Boston residents did not exhibit more acute stress symptoms than New York City residents after the bombings; proximity to the bombings was not associated with acute stress scores.

New York residents were directly exposed to more of the prior collective traumas we examined—in particular, 9/11 and Superstorm Sandy. They also reported BMB-related acute stress responses comparable to those reported by the Boston respondents, which highlights the potentially harmful consequences of cumulative exposure to both human-made and natural disasters. The 9/11 attacks were a highly publicized, high-impact collective trauma with long-lasting repercussions for individuals' health, psychological well-being, and behaviors; therefore, 9/11 exposure may have been especially potent. Because the BMB and 9/11 were both acts of

Table 1. Percentages of Respondents Reporting Prior Exposure to Collective Trauma

Variable	Boston (n = 846)	New York (n = 941)
Direct exposure to Boston Marathon bombings		
Self	9.22 (78)	0.39 (4)
Close other	30.43 (257)	7.68 (72)
Any (self or close other)	34.65 (292)	7.80 (73)
Direct exposure to September 11, 2001, attacks		
Self	3.68 (31)	25.03 (233)
Close other	13.73 (115)	37.02 (344)
Any (self or close other)	15.30 (128)	47.13 (438)
Direct exposure to Superstorm Sandy		
Self	8.34 (70)	48.03 (447)
Close other	9.79 (82)	29.15 (271)
Any (self or close other)	14.37 (120)	55.00 (512)
Direct exposure to Sandy Hook School shootings		
Self	1.27 (11)	1.91 (18)
Close other	1.84 (15)	2.25 (21)
Any (self or close other)	2.34 (20)	3.67 (35)
Indirect exposure to September 11, 2001, attacks		
Live television	73.01 (612)	69.73 (649)
Live radio or Internet streaming	24.08 (202)	28.40 (264)
Either category of indirect exposure	76.73 (643)	73.98 (688)
Indirect exposure to Superstorm Sandy		
Live television	59.29 (497)	59.62 (554)
Live radio or Internet streaming	23.84 (200)	33.49 (311)
Either category of indirect exposure	65.48 (549)	68.66 (639)
Indirect exposure to Sandy Hook School shootings		
Live television	40.67 (341)	44.82 (417)
Live radio or Internet streaming	18.98 (159)	25.92 (241)
Either category of indirect exposure	48.70 (408)	54.16 (504)

Note: The table reports weighted percentages, with weighted ns in parentheses. For each event, respondents could report more than one category of exposure.

terrorism, exposure to 9/11 may have particularly sensitized people to react negatively to the BMB. The weak relationship between physical location and acute stress response echoes findings reported following 9/11; after researchers accounted for the most severe exposure, responses to 9/11 were associated with factors other than geographic proximity (Galea et al., 2002; Holman et al., 2008; Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002). Finally, almost half of New York City residents were also directly exposed to Superstorm Sandy. Together,

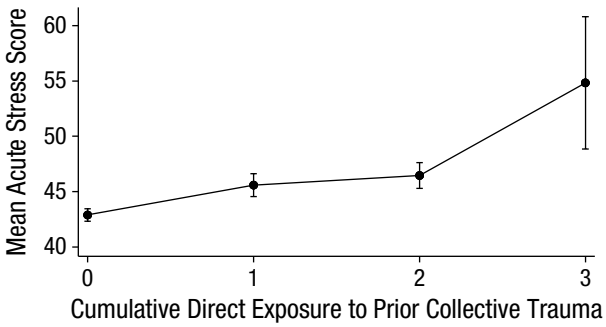

Fig. 1. Mean acute stress score as a function of cumulative direct exposure to prior collective trauma. Error bars represent ±1 *SE*.

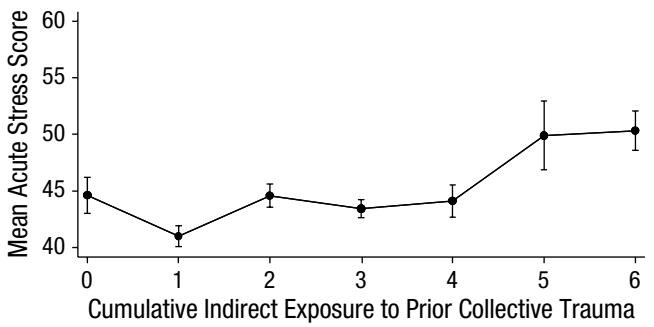

Fig. 2. Mean acute stress score as a function of cumulative indirect exposure to prior collective trauma. Error bars represent ±1 *SE*.

Table 2. Predictors of Acute Stress Responses After the Boston Marathon Bombings (Weighted $N = 1{,}768$)

	Model 1			Model 2			Model 3		
Variable	β	*SE*	*t*	β	*SE*	*t*	β	*SE*	*t*
Prior mental health and collective trauma exposure									
Pre-BMB mental-health ailments	0.11 [0.04, 0.18]**	0.04	2.92	0.11 [0.04, 0.19]**	0.04	2.97	0.10 [0.03, 0.17]**	0.04	2.64
Prior direct exposure	0.09 [0.02, 0.16]*	0.04	2.45	0.09 [0.00, 0.18]*	0.04	2.05	0.11 [0.03, 0.19]**	0.04	2.72
Prior indirect exposure	0.09 [0.01, 0.16]*	0.04	2.18	0.09 [0.01, 0.16]*	0.04	2.20	0.09 [0.02, 0.16]*	0.04	2.55
Exposure to BMB									
Boston resident				–0.01 [–0.18, 0.17]	0.09	–0.10	0.02 [–0.14, 0.18]	0.08	0.28
Direct BMB exposure				0.27 [0.05, 0.49]*	0.11	2.39	0.33 [0.14, 0.53]**	0.10	3.32
Direct lockdown exposure				0.06 [–0.15, 0.28]	0.11	0.55	0.09 [–0.10, 0.28]	0.10	0.92
Demographics									
Income							–0.15 [–0.22, –0.08]***	0.03	–4.36
Female gender							0.18 [0.04, 0.31]**	0.07	2.61
Race									
African American							–0.12 [–0.36, 0.11]	0.12	–1.03
Other, mixed race, non-Hispanic							0.62 [0.19, 1.05]**	0.22	2.81
Hispanic							0.03 [–0.16, 0.21]	0.09	0.27
Education									
High school							–0.34 [–0.82, 0.13]	0.24	–1.41
Some college							–0.51 [–0.99, –0.03]*	0.24	–2.09
Bachelor's degree or higher							–0.48 [–0.96, 0.00]	0.24	–1.95
Constant	0.03 [–0.04, 0.10]	0.04	0.76	–0.04 [–0.15, 0.08]	0.06	–0.63	0.21 [–0.25, 0.68]	0.24	0.89

Note: The weighted *N* differs from the total sample size (1,787) because of missing data. Robust standard errors are provided. Values in brackets are 95% confidence intervals. Prior mental health was coded as follows: 0 = no anxiety or depression, 1 = depression or anxiety, 2 = both depression and anxiety. We counted cumulative direct exposure (range: 0–3) and indirect exposure (range: 0–6) to prior collective trauma. Direct exposure to the Boston Marathon bombings (BMB) or Boston-area lockdown was coded as follows: 0 = no direct exposure, 1 = self or close other directly exposed. The reference group for Boston residents was New York residents. The reference groups for gender, race, and education were males, Whites, and "less than high school," respectively. All models accounted for a significant amount of variance—Model 1: $F(3, 1764) = 6.13$, $p < .001$, $R^2 = .03$; Model 2: $F(6, 1761) = 5.34$, $p < .001$, $R^2 = .04$; Model 3: $F(14, 1753) = 7.30$, $p < .001$, $R^2 = .12$.
*$p < .05$. **$p < .01$. ***$p < .001$.

these findings indicate that exposures to both natural disasters and violent events may have compound effects (Garfin, Silver, Ugalde, Linn, & Inostroza, 2014; Goenjian et al., 1994), with important implications for responses to subsequent events.

The association between cumulative indirect exposure to prior collective trauma and response to the BMB offers another explanation for the null relationship between location of residence and acute stress. Given that community-based traumas and related media coverage are ubiquitous in modern society, understanding how direct and indirect, media-based exposure to these events may influence response to subsequent trauma has important theoretical and public-health implications. Several studies have now shown that indirect media exposure to collective trauma is negatively associated with physical and mental health (Bernstein et al., 2007; Holman et al., 2014; Silver et al., 2013; see also Bourne, Mackay, & Holmes, 2013). Our findings extend this work by demonstrating that the accumulation of prior indirect exposures may sensitize individuals to subsequent trauma regardless of event proximity.

Overall, our results support a sensitization model of prior trauma exposure in which more exposure—direct or indirect—to collective trauma may predispose people to experience greater acute stress after subsequent collective trauma. It is noteworthy that cumulative effects were identified for indirect, media-based exposures. Several mechanisms may underlie how media-based exposures are encoded. Functional MRI studies have demonstrated that vicarious exposure to traumatic images elicits activation of brain regions linked to subsequent intrusive flashbacks, a hallmark of acute and posttraumatic stress responses (Bourne et al., 2013). Recent research in neurobiology suggests that sensitization may operate through cellular changes in neural circuitry after stressful events; these changes promote the increased efficiency of similar cognitive, physiological, and emotional processes in response to future stimuli (Ursin, 2014). Identifying the specific mechanisms underlying our findings is an important next step that would move researchers closer to developing early secondary interventions to prevent trauma-related disorders.

Our findings do not support a habituation effect, such as has been observed in studies of Israelis exposed to terrorism (e.g., Bleich et al., 2003). Perhaps the repetitious ongoing nature of terrorism in Israel generates emotional and biological processes that are different from those promoted by the accumulation of unexpected, single community traumas (e.g., the BMB, 9/11, a natural disaster). Alternatively, the compounding effect of prior exposures may depend on the type of traumatic events examined (e.g., individual negative events, ongoing terrorism; Andersen et al., 2013; Baum, 1987). Finally, repeated exposures to collective stressors might not produce a linear increase in stress response; individuals may eventually reach a plateau such that each repeated exposure has less impact over time. This hypothesis was suggested by Seery et al. (2010), who demonstrated that the negative psychological impact of recent adverse events depended on the individual's overall lifetime exposure to adversity: Individuals with a history of lifetime adversity appeared less negatively affected by recent adverse events than other individuals. Future research should examine these possibilities more closely.

We acknowledge several limitations of our study. We assessed BMB-related exposure and acute stress response 2 to 4 weeks after the event (within 2.5 weeks of the BMB for the vast majority of respondents), which is a strength of our study; however, all reports of cumulative direct and indirect exposure to prior community traumas were collected retrospectively and may have been susceptible to recall bias. We did not have an equivalent measure of BMB-related indirect exposure (e.g., live media exposure at the time of the event) and thus could not include it in our analyses. We assessed exposure to only three prior collective traumas, but residents may have experienced many more; this may have contributed to our null quadratic effects. Our findings illustrate the likely incremental effects of cumulative exposure to several traumatic events over time, although our effect sizes were relatively small. Our cross-sectional, correlational study cannot demonstrate causality. Approximately 25% of the prior mental-health data were imputed; however, we implemented sophisticated and valid imputation techniques, and results were comparable in analyses conducted with and without imputed data. Finally, our conclusions may not generalize to all traumas or types of response (e.g., psychological, physiological): A study of rape victims found evidence for sensitization processes for posttraumatic stress disorder and habituation processes for physiological responses (Resnick et al., 1995); a similar mixed pattern was seen in college students after a series of deaths among their peers (Andersen et al., 2013).

Conclusions

Cumulative exposure to prior collective trauma may have helped sensitize people to the BMB. Our findings extend those of other studies documenting trauma-related physical- (Felitti & Anda, 2010) and mental-health (Seery et al., 2010; Turner & Lloyd, 1995) problems after multiple exposures to individual-level traumas. Screening for prior exposure to collective traumas may help researchers and practitioners identify individuals at greatest risk for acute stress response and perhaps longer-term health problems (Holman et al., 2008) after subsequent trauma. Indeed, both direct and indirect, media-based cumulative exposure

to community trauma should be considered risk factors that can sensitize people to experience more serious impacts of large-scale negative events. Thus, they warrant the attention of first responders and other groups seeking to help victims of these disasters.

Author Contributions

R. C. Silver and E. A. Holman designed the research. D. R. Garfin, E. A. Holman, and R. C. Silver performed the research. D. R. Garfin analyzed the data. D. R. Garfin, E. A. Holman, and R. C. Silver wrote the manuscript. All authors approved the final version of the manuscript for submission.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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