Neural vulnerability and hurricane-related media predict posttraumatic stress in youth

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ABSTRACT

As natural disasters increase in frequency and severity[1,2], mounting evidence reveals that their human toll extends beyond death, injury, and loss. Posttraumatic stress (PTS) can be common among exposed individuals, and children are particularly vulnerable[3,4]. Curiously, PTS can even be found among youth far removed from harm's way, and media-based exposure may partially account for this phenomenon[5–8]. Unfortunately, susceptibility to media effects has been difficult to characterize because most research is initiated post-event, precluding examination of pre-disaster factors. In this study, we mitigate this issue with data from nearly 400 9- to 11-year-old children collected prior to and after Hurricane Irma. We evaluate whether preexisting neural patterns predict degree of media effects on later Irma-related PTS. We show that "dose" of Irma-related *media* exposure predicted Irma-related PTS– even among children dwelling thousands of kilometers away from the hurricane. Furthermore, we show, using pre-hurricane functional magnetic resonance imaging data, that neural responses in brain regions associated with anxiety and stress confer particular vulnerability to media effects and PTS among certain children. Specifically, right amygdala predicted Irma-related PTS, and bilateral orbitofrontal cortex and left parahippocampal gyrus moderated the association between Irma-related media exposure and PTS. Collectively, these findings run counter to outdated "bullseye" models of disaster exposure that assume negative effects are narrowly circumscribed around a disaster's geographic epicenter[9]. In contrast, for some youth with measurable preexisting vulnerability, consumption of extensive disaster-related media appears to offer an alternative pathway to disaster exposure that transcends geography and objective risk. This preventable exposure should be considered in disaster-related mental health efforts.

Across the past decade, natural disasters have killed over 700,000 people and left over two billion others injured, homeless, or in need of emergency assistance for survival[10]. In particular, weather-related disasters, and their associated human and economic tolls, are on the rise[1,2]. In addition to their physical consequences, such disasters carry a broad and sustained mental health toll, with robust post-disaster evidence documenting elevated posttraumatic stress (PTS) responses among large subsets of individuals[4,11,12]. Children are among the most vulnerable, as they are still developing a stable sense of security and have relatively limited control over their environments[4].

The mental health burdens of disasters are not confined to proximally exposed youth. Individuals near *and* far show elevated PTS responses in the aftermath of disasters[13–15],

with increasing evidence pointing to the important role that disaster-related media exposure may play in explaining PTS symptoms in distal individuals[6–8]. That said, research on this front has predominantly focused on manmade disasters with malicious intent, such as terrorism and mass shootings. Related work has not considered youth media effects in the context of increasingly common weather-related disasters, which are typically preceded by an extensive warning period and considerable pre-event threat-related media attention. Related research considering pre-event media exposure in *adult* samples[16] has focused exclusively on regionally affected individuals, and does not speak to media effects in youth, given cognitive developmental differences in risk assessment, threat perception, and media literacy. In addition, studies considering the effects of disaster-related media exposure have typically focused on exposure to coverage

during and after the event. Little is known about mental health consequences following exposure to pre-disaster media coverage of impending disaster. Large-scale research has also failed to consider potential neural vulnerabilities that may forecast which youth are most susceptible to PTS responses related to anticipatory disaster-related media exposure.

To overcome these limitations, in a multi-state sample of youth, we examined interactions between prospective neural vulnerability and reports of pre-disaster anticipatory media exposure in the context of Hurricane Irma—one of the most powerful Atlantic hurricanes on record. In the week prior to Irma's landfall, internet-based and nationally televised media coverage provided sensationalized, around-the-clock forecasting of the impending "catastrophic" storm and its threatened "unprecedented" destruction of "epic proportions" to the Southeastern United States[16], culminating in the largest human evacuation in American history (~7 million people[17]).

In this paper, we present results of analyses on 454 well-characterized families from four sites of the Adolescent Brain and Cognitive Development (ABCD) Study. The four participating study sites included three that were directly impacted by Hurricane Irma—i.e., Florida International University (FIU) in Miami, Florida; University of Florida (UF) in Gainesville, Florida; Medical University of South Carolina (MUSC) in Charleston, South Carolina—and one in a distal, non-impacted state with relatively comparable demographic characteristics—-i.e., University of California, San Diego (UCSD) in San Diego, California (Table 1). In the year prior to Hurricane Irma's United States landfall on September 10, 2017, these four sites collected demographic, mental health, and neuroimaging measures during the standard ABCD Baseline Visit. After the storm, these four ABCD sites collected a post-Irma follow-up survey that assessed reports of children's objective hurricane exposure and Irma-related media exposure, as well as Irma-related PTS responses.

Table 1. Breakdown of parents and children who completed the post-hurricane surveys at each site.

Site	Child	Parent
FIU	128 (44%)	154 (53%)
MUSC	89 (78%)	92 (81%)
UF	69 (56%)	82 (67%)
UCSD	110 (36%)	126 (41%)
Total	396 (48%)	454 (56%)

Note. The completion percentage for eligible families, by site, is provided in parentheses.

Objective Exposure to Hurricane Irma is Associated with Posttraumatic Stress

We began our analysis by establishing the degree to which objective exposure to Hurricane Irma predicted PTS symptoms. We measured objective exposure using the Hurricane Related Traumatic Experiences–II (HURTE-II) survey, which assesses stressors like life threat, injury, loss, evacuation experiences, and property damage. As expected, objective exposure was associated with PTS in the South Florida youth sample most directly affected by Hurricane Irma (i.e., the FIU site in Miami; $B = 0.43$, $t(109) = 2.43$, $p = 0.017$, 95% Confidence Interval (CI) = 0.08 to .78, $\beta = 0.14$). We found essentially the same result when all sites in states directly impacted by Irma (FIU, UF, and MUSC) were collectively examined ($B = 0.29$, $t(255) = 2.21$, $p = 0.028$, 95% CI = 0.03 to 0.55, $\beta = 0.09$; Figure 1). Furthermore, the results were unchanged when children's baseline anxiety and exposure to prior trauma were entered as covariates ($B = 0.48$, $t(107) = 2.49$, $p = 0.014$, 95% CI = 0.09 to .80, $\beta = 0.14$ for the South Florida FIU site; ($B = 0.28$, $t(253) = 2.10$, $p = 0.037$, 95% CI = 0.02 to 0.54, $\beta = 0.09$ for all affected sites). Thus, the results showed that objective exposure to the hurricane was associated with increased PTS symptoms in youth from these three sites in Irma-affected states, and this was not explained by prior trauma or pre-existing anxiety.

Figure 1. Irma exposure predicts post-Irma PTS symptoms among hurricane exposed youth. Figure shows added variable plot for data from all hurricane-impacted sites (controlling for covariates, see Method). Error shading represents the 95% Confidence Interval. Data were rescaled to place the origin at (0,0).

Irma-Related Media Exposure Prior to Hurricane is Associated with Posttraumatic Stress, Regardless of Distance from Hurricane

With prior research showing that objective disaster exposure and threat is not always necessary to prompt PTS responses[5–8,18,19], we broadened our analysis to examine media-based effects. Indeed, in the lead-up to Irma's arrival in Florida, national news coverage was saturated with sensationalized, around-the-clock forecasting, and children were watching. Roughly one-third of the sample reported

Figure 2. Pre-storm media exposure predicts PTS symptoms among children near and far.. Figure shows added variable plot (controlling for covariates, see Method) for Irma-related media exposure before the hurricane predicting PTS symptoms in children, regardless of whether children were in a region directly impacted by the hurricane (e.g., Florida or coastal South Carolina), or were not (Southern California). Error shading represents the 95% Confidence Interval. Data were rescaled to place the origin at (0,0).

that in the lead-up to the storm they consumed at least an hour of daily Irma-related television coverage (31.1%) and checked online coverage almost every hour (32.2%). Prior to landfall, 19.1% also engaged with Irma-related social media at least several times per day. Across the full sample, we found that the degree of media exposure was associated with child PTS outcomes ($B = 0.41$, $t(377) = 4.84$, $p = 0.000002$, 95% CI = 0.24 to 0.57, $\beta = 0.15$; even after controlling for prior anxiety and trauma, $B = 0.40$, $t(375) = 4.61$, $p = 0.00003$, 95% CI = 0.23 to 0.56, $\beta = 0.15$). Interestingly, there was no evidence that being safely out of the storm's physical path mitigated the impact of storm-related news exposure on youth. When we dichotomously classified youth as dwelling in either an Irma-affected state (FIU, UF, and MUSC youth) versus an unaffected state (i.e., UCSD youth in Southern California), this factor did not moderate the association between pre-storm Irma-related media exposure and youth PTS ($B = -0.09$, $t(376) = -0.30$, $p = 0.72$, 95% CI = -0.64 to 0.47; $\beta = -0.03$). Indeed, the effects of exposure to anticipatory Irma-related media on child PTS were robust and uniform across youth, even among those who were over 4500 kilometers from the storm's path (Figure 2). Thus, mental health effects associated with storm-related media exposure in the lead-up to Hurricane Irma appear to be wide-ranging, extending to youth far beyond geographic boundaries of the storm's physical projected path.

Neural Vulnerability Moderates Media Effects on Posttraumatic Stress

Because baseline mental health and neural measures were collected in the two years before the hurricane, we also had a unique opportunity to examine potential vulnerabilities to these storm-related media effects. Here we examined

Figure 3. **Activation in bilateral amygdala is higher in Fear vs. Neutral conditions during EN-Back**. Activation in the remaining *a priori*-defined ROIs are shown in Extended Data Figure 1. Whole-brain activation maps show the comparison of Fear vs. Neutral ($p < .01$, uncorrected). Bar plots show the summary activation profiles for each condition. Error bars = +/- standard error.

neural biases in brain regions associated with anxiety and stress[20–23] (see Extended Data Figure 1). Neural bias was measured as the difference in brain activity within regions of interest (ROIs) during an Emotional variant of the classic N-back working memory task (i.e., the ABCD EN-Back[24]). In the EN-back, blocks of trials consist of happy, fearful, and neutral facial expressions as well as places. We focused on the child's neural responses to fearful versus neutral facial expressions within chosen brain regions. Our reasoning was that this would indicate neural predisposition to processing ambiguous faces as either fear-inducing (i.e., essentially not different from overtly fear-inducing stimuli), or neutral (i.e., very different from overtly fear-inducing stimuli). As expected, we found that, across the sample, fear-inducing stimuli elicit more activity in bilateral amygdala, consistent with the amygdala's important role in the processing of fear- or threat-related stimuli (Figure 3)[23]. The response in other regions of this network, associated with the regulation of emotion and memory, was more variable (Extended Data Figure 1), and we examined whether these neural response biases either directly predict Irma-related PTS (i.e., the main effect of the EN-Back difference score), or moderate the association between pre-storm media exposure and Irma-related PTS (i.e., the interaction of the EN-Back difference score and pre-storm media exposure). In this analysis, one main-effect and three interaction effects were

statistically significant (Table 2 and Figure 4).

First, we found that the main effect in pre-Irma right amygdala for fearful versus neutral faces predicted post-Irma PTS. This is consistent with a number of neuroimaging studies showing that the amygdala is a core region implicated in anxiety and adaptive stress response[22,25], and in more extreme stress responses that meet criteria for PTSD[25]. For example, compared to people without PTSD, people with PTSD show greater amygdala activation when viewing negative emotional faces and scenes or other trauma-related stimuli[26,27]. Furthermore, surgical ablation of amygdala is associated with remediation of PTS symptoms, suggesting its central role in the pathophysiology of the disorder[28]. In our study, children who readily differentiate fearful from neutral stimuli and typically show greater amygdala activation in response to fearful faces were at reduced risk for storm-related PTS (B = -1.6, $t(280)$ = -3.25, p = 0.001, 95% CI = -2.57 to -0.64, β = -0.14). Our findings suggest that individual differences in right amygdala response to potentially fear-inducing stimuli may confer differential risk to subsequent negative outcomes following disaster-related media exposure.

More novel findings pertain to a set of neural moderation effects we identified (i.e., media by EN-Back interactions; Figure 4 and Table 2). These were evident in bilateral OFC and left parahippocampal gyrus, brain regions associated with emotion regulation and with autobiographical memory, respectively. In all three regions, children who showed a strong positive response to the Neutral condition, coupled with a weak or below-baseline response to the Fear condition, were especially susceptible to PTS when exposed to anticipatory media coverage of Hurricane Irma. Those who showed the opposite pattern were less susceptible to PTS when exposed to such Irma-related media exposure.

To facilitate interpretation of these results, we situate them within models that propose that disorders of anxiety and stress are in part characterized by pre-conscious response biases in neural circuits designed to process and respond to threat and stress in everyday situations. These neural circuits include regions interacting with amygdala in the context of threatening or stressful situations, including OFC and parahippocampal gyrus. In this characterization, the OFC directly interacts with amygdala to modulate the threat or stress response[29], down-regulating amygdala in a top-down fashion[30]. Thus, differences in OFC-amygdala interactions can, in part, account for individual differences in emotion regulation[31] and stress response[30]. In people with disorders of stress and anxiety, this modulation is atypical. Indeed, neuroimaging research has shown that the OFC is differentially recruited in people with PTSD relative to non-trauma exposed individuals[26], and in people with diagnosed anxiety disorders[27]. Disaster exposure is also

shown to be sufficient to modify this circuit. Thus, attenuation of OFC activation in response to visual presentation of disaster events (e.g., earthquakes) occurs in people with PTSD relative to controls[32], and this attenuation is consistently associated with symptom severity[30]. Hyperactivity of the parahippocampal gyrus is also a consistent finding in people with PTSD[33]. This region is more easily activated in response to traumatic imagery for people with PTSD relative to non-trauma exposed individuals[32], and like the OFC, its activity is positively associated with symptom severity[34]. Its role within this circuit is in contextual associative processing of autobiographical memories with high emotional valence[35], such as those related to threat or trauma[32]. Thus, children who cannot emotionally regulate the response to anticipatory threat-related media might be at heightened risk for becoming overwhelmed by trauma-related memories.

In our data, ineffective recruitment of downregulatory processes in response to fearful stimuli, or simultaneously over-reaction to neutral/non-threatening stimuli, seems to confer a greater risk to increasing PTS from media exposure. Thus, children who under-recruit OFC in response to fearful stimuli, or over-recruit OFC in response to non-threatening stimuli, seem to be most at risk. The effect is mirrored in parahippocampal gyrus, potentially contributing to the consolidation of traumatic memories, even when these arise from media exposure rather than from actual exposure. Repeated stress exposure through media could have long-term effects on interactions among brain regions of this extended circuit, although this remains to be established. However, in animal models, stress exposure changes the way that OFC interacts functionally with amygdala, altering the way in which fear-related memories are processed[36]. In children, previous research has shown that exposure to hurricane events alters neural reactivity to negative stimuli in children who were tested before and after Hurricane Sandy[37]. In that research, conducted with children who were the same age as those studied here, there was an effect of "dose," such that children who experienced high exposure were most susceptible to changes in neural reactivity. This shows that disaster-related stress has a persistent impact on brain functioning, and further suggests that these effects may snowball with increasing exposure "dose." Indeed, negatively valenced arousal is known to increase attention to emotional stimuli and experiences[30], and altered neural reactivity to negative emotional information following disasters may confer particular vulnerability to later stressors in adulthood[37]. Our results indicate that specific pre-existing features of a child's brain-based emotional reactivity may make them more or less susceptible to the negative influence of repeated exposure to disaster threat, even through media, elevating risk for the development of subsequent PTS.

When interpreting these results, it is important to note that none of the children in the study showed a degree of

Figure 4. Prospective brain moderators of the relation between Irma-related media exposure and posttraumatic stress (PTS). Results are reported for effects that were reliable when anatomical ROIs were examined at the individual level. Left panel: Results of the whole-brain analysis (ventral surface view; $p < .01$, corrected). Middle panel: Slope estimates of the association between Irma-related media exposure and PTS symptoms from the multiple regression controlling for covariates (1) age, (2) birth sex, (3) race/ethnicity, (4) highest degree of parental education, (5) household income, (6) parental marital status, and (7) MRI scanner serial number. This slope estimate is parsed along the residualized EN-Back activation difference to illustrate the interaction effect. It is parsed at the mean (Fear = Neutral), and -1 (Fear < Neutral) and +1 (Fear > Neutral) standard deviations above the mean. Right panel: Summary measures of the condition differences illustrate the nature of the interaction, and are plotted for subjects below the mean (Fear < Neutral), and above the mean (Fear > Neutral), of the EN-Back activation difference.

PTS that reached diagnostic threshold for PTSD. There was also no direct manipulation of media exposure, and in any event the magnitude of effect sizes are modest (on the order of semipartial $r = .23$ for effects of objective exposure; .22 for effects of media exposure; and .03 to .06 for interaction effects of media by brain). However, this does not mean that these effects are trivial. Small effects, interpreted in the correct context, are important when they impact large populations and/or if they systematically accrue over time[38]. Thus, small effect sizes are meaningful when the degree of potential accumulation is substantial[39]. Our results point to effects of media exposure on future stress

Table 2. Results of Media by EN-Back (Fear vs Neutral) interaction predicting PTS symptoms in each *apriori* defined region of interest.

Brain Region	B (SE)	β	t	p	Lower to Upper 95% CI
Left Hemisphere					
Amygdala	0.00 (0.21)	0.00	0.00	0.997	-0.41 to 0.41
Hippocampus	0.06 (0.14)	0.00	0.41	0.684	-0.22 to 0.34
Orbitofrontal Cortex	-0.31 (0.10)	-0.05	-3.06	**0.002 ** †**	-0.50 to -0.11
Parahippocampal Gyrus	-0.43 (0.18)	-0.04	-2.44	**0.015 * †**	-0.78 to -0.09
Anterior Cingulate Cortex	0.07 (0.23)	0.00	0.32	0.751	-0.37 to 0.52
Right Hemisphere					
Amygdala	0.17 (0.12)	0.02	1.39	0.167	-0.07 to 0.41
Hippocampus	-0.13 (0.16)	-0.01	-0.79	0.43	-0.45 to 0.19
Orbitofrontal Cortex	-0.38 (0.10)	-0.06	-3.95	**0.00004 *** †**	-0.57 to -0.19
Parahippocampal Gyrus	-0.08 (0.09)	-0.01	-0.95	0.34	-0.25 to 0.09
Anterior Cingulate Cortex	-0.34 (0.19)	-0.03	-1.78	0.077	-0.71 to 0.04

Note. The robust linear models controlled for the following covariates as fixed effects: (1) age, (2) birth sex, (3) race/ethnicity, (4) highest degree of parental education, (5) household income, (6) parental marital status, and (7) MRI scanner serial number. B = Unstandardized regression slope parameter estimate. SE = Standard error of the regression slope parameter estimate. β = Standardized regression slope parameter estimate. CI = 95% Confidence Interval. All *p*-values are two-tailed. ** $p < .01$. *** $p < .001$. *p*-values marked with † indicate that these effects survived a False Discovery Rate (FDR) correction for multiple comparisons. Degrees of Freedom for all regression models were $df = 281$.

responses, regardless of proximity to the disaster event, and to a neural bias to processing both threatening and ambiguous stimuli that may confer vulnerability to PTS. The modern mass media landscape now includes 24-hour news networks and a continuous news cycle, decreasing objectivity in news presentations, online and social media that are not governed by the same standards, ethics, and sensibilities as traditional journalism, increased prominence of largely unregulated citizen/crowd-sourced journalism, and rapidly advancing technologies that disrupt everyday experiences and "push" news stories into our daily activities[40].

Against this backdrop, coupled with the unprecedented penetration of mass media into the daily lives of youth, there is cause for concern that negative (albeit small) media effects can accumulate with repeated exposure to threat-related media presentations across development. In the context of impending, but remote, disasters, the propensity for nonetheless encountering anxiety- or fear-inducing events and stimuli via the media is significant. Thus, even when children do not reach criteria for a disorder in the context of a single disaster, it is possible sub-threshold variability within the constellation of stress symptoms can accumulate to incur increased susceptibility to disorder in future situations. This is all the more concerning in light of the increasing frequency with which natural disasters are now occurring[1,2]. Indeed, the oldest children in the ABCD study in South Florida have been exposed to 200 named storms, 95 of which turned into hurricanes, and 43 of which were major hurricanes. It is possible

that such repeated "micro-exposures" to threat-related media may accumulate and influence the processing of traumatic experiences in neural systems designed to respond to threat and stress in everyday situations, putting some children at increased risk for media-related PTS. Coupled with the increasingly dramatic and sensationalized nature of modern media coverage, children's exposure to disaster-related media constitutes a serious public health concern.

References

1. Cai, W. *et al.* Increasing frequency of extreme el niño events due to greenhouse warming. *Nat. Clim. Chang.* **4**, 111–116 (2014).

2. Kessler, R. C. *et al.* Trends in mental illness and suicidality after hurricane katrina. *Mol Psychiatry* **13**, 374–84, DOI: 10.1038/sj.mp.4002119 (2008).

3. Goldmann, E. & Galea, S. Mental health consequences of disasters. *Annu. Rev Public Heal.* **35**, 169–83, DOI: 10.1146/annurev-publhealth-032013-182435 (2014).

4. Furr, J. M., Comer, J. S., Edmunds, J. M. & Kendall, P. C. Disasters and youth: a meta-analytic examination of posttraumatic stress. *J Consult. Clin Psychol* **78**, 765–80, DOI: 10.1037/a0021482 (2010).

5. Comer, J. S. *et al.* Adjustment among area youth after the boston marathon bombing and subsequent manhunt. *Pediatrics* **134**, 7–14, DOI: 10.1542/peds.2013-4115 (2014).

6. Holman, E. A., Garfin, D. R. & Silver, R. C. Media's role in broadcasting acute stress following the boston marathon bombings. *Proc Natl Acad Sci U S A* **111**, 93–8, DOI: 10.1073/pnas.1316265110 (2014).

7. Thompson, R. R., Jones, N. M., Holman, E. A. & Silver, R. C. Media exposure to mass violence events can fuel a cycle of distress. *Sci Adv* **5**, eaav3502, DOI: 10.1126/sciadv.aav3502 (2019).

8. Vasterman, P., Yzermans, C. J. & Dirkzwager, A. J. The role of the media and media hypes in the aftermath of disasters. *Epidemiol Rev* **27**, 107–14, DOI: 10.1093/epirev/mxi002 (2005).

9. Marshall, R. D. *et al.* The psychology of ongoing threat: relative risk appraisal, the september 11 attacks, and terrorism-related fears. *Am Psychol* **62**, 304–16, DOI: 10.1037/0003-066X.62.4.304 (2007).

10. Societies, I. F. o. R. C. & Crescent, R. World disasters report. Report (2018).

11. North, C. S. & Pfefferbaum, B. Mental health response to community disasters: a systematic review. *JAMA* **310**, 507–18, DOI: 10.1001/jama.2013.107799 (2013).

12. Orengo-Aguayo, R., Stewart, R. W., de Arellano, M. A., Suarez-Kindy, J. L. & Young, J. Disaster exposure and mental health among puerto rican youths after hurricane maria. *JAMA Netw Open* **2**, e192619, DOI: 10.1001/jamanetworkopen.2019.2619 (2019).

13. Pfefferbaum, B. *et al.* Posttraumatic stress two years after the oklahoma city bombing in youths geographically distant from the explosion. *Psychiatry* **63**, 358–70, DOI: 10.1080/00332747.2000.11024929 (2000).

14. Schuster, M. A. *et al.* A national survey of stress reactions after the september 11, 2001, terrorist attacks. *N Engl J Med* **345**, 1507–12, DOI: 10.1056/NEJM200111153452024 (2001).

15. Suvak, M., Maguen, S., Litz, B. T., Silver, R. C. & Holman, E. A. Indirect exposure to the september 11 terrorist attacks: does symptom structure resemble ptsd? *J Trauma Stress.* **21**, 30–9, DOI: 10.1002/jts.20289 (2008).

16. Thompson, R. R., Holman, E. A. & Silver, R. C. Media coverage, forecasted posttraumatic stress symptoms, and psychological responses before and after an approaching hurricane. *JAMA Netw Open* **2**, e186228, DOI: 10.1001/jamanetworkopen.2018.6228 (2019).

17. Wong, S. D., Pel, A. J., Shaheen, S. A. & Chorus, C. G. Fleeing from hurricane irma: Empirical analysis of evacuation behavior using discrete choice theory. *Transp. Res.* **79**, 102227 (2020).

18. Comer, J. S. & Kendall, P. C. Terrorism: The psychological impact on youth. *Clin. Psychol. Sci. Pract.* **14**, 179–212 (2007).

19. Comer, J. S., D'eSerisy, M. & Green, J. G. Caregiver-reports of internet exposure and posttraumatic stress among boston-area youth following the 2013 marathon bombing. *Evidence-Based Pract. Child Adolesc. Mental Heal.* **1**, 86–102 (2016).

20. Robinson, O. J., Pike, A. C., Cornwell, B. & Grillon, C. The translational neural circuitry of anxiety. *J Neurol Neurosurg Psychiatry* DOI: 10.1136/jnnp-2019-321400 (2019).

21. LeDoux, J. E. & Pine, D. S. Using neuroscience to help understand fear and anxiety: A two-system framework. *Am J Psychiatry* **173**, 1083–1093, DOI: 10.1176/appi.ajp.2016.16030353 (2016).

22. Shin, L. M. & Liberzon, I. The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology* **35**, 169–91, DOI: 10.1038/npp.2009.83 (2010).

23. Tovote, P., Fadok, J. P. & Luthi, A. Neuronal circuits for fear and anxiety. *Nat Rev Neurosci* **16**, 317–31, DOI: 10.1038/nrn3945 (2015).

24. Casey, B. J. *et al.* The adolescent brain cognitive development (abcd) study: Imaging acquisition across 21 sites. *Dev Cogn Neurosci* **32**, 43–54, DOI: 10.1016/j.dcn.2018.03.001 (2018).

25. Ressler, K. J. Translating across circuits and genetics toward progress in fear- and anxiety-related disorders. *Am J Psychiatry* **177**, 214–222, DOI: 10.1176/appi.ajp.2020.20010055 (2020).

26. Bryant, R. A. *et al.* Enhanced amygdala and medial prefrontal activation during nonconscious processing of fear in posttraumatic stress disorder: an fmri study. *Hum Brain Mapp* **29**, 517–23, DOI: 10.1002/hbm.20415 (2008).

27. Killgore, W. D. *et al.* Cortico-limbic responses to masked affective faces across ptsd, panic disorder, and specific phobia. *Depress. Anxiety* **31**, 150–9, DOI: 10.1002/da.22156 (2014).

28. Bijanki, K. R. *et al.* Case series: Unilateral amygdala ablation ameliorates post-traumatic stress disorder symptoms and biomarkers. *Neurosurgery* DOI: 10.1093/neuros/nyaa051 (2020).

29. Hiser, J. & Koenigs, M. The multifaceted role of the ventromedial prefrontal cortex in emotion, decision making, social cognition, and psychopathology. *Biol. psychiatry* **83**, 638–647 (2018).

30. Thome, J., Terpou, B. A., McKinnon, M. C. & Lanius, R. A. The neural correlates of trauma-related autobiographical memory in posttraumatic stress disorder: A meta-analysis. *Depress. Anxiety* **37**, 321–345, DOI: 10.1002/da.22977 (2020).

31. Stevens, J. S. *et al.* Disrupted amygdala-prefrontal functional connectivity in civilian women with posttraumatic stress disorder. *J Psychiatr Res* **47**, 1469–78, DOI: 10.1016/j.jpsychires.2013.05.031 (2013).

32. Sakamoto, H. *et al.* Parahippocampal activation evoked by masked traumatic images in posttraumatic stress disorder: a functional mri study. *Neuroimage* **26**, 813–21, DOI: 10.1016/j.neuroimage.2005.02.032 (2005).

33. Etkin, A. & Wager, T. D. Functional neuroimaging of anxiety: a meta-analysis of emotional processing in ptsd, social anxiety disorder, and specific phobia. *Am J Psychiatry* **164**, 1476–88, DOI: 10.1176/appi.ajp.2007.07030504 (2007).

34. Shin, L. M. *et al.* Hippocampal function in posttraumatic stress disorder. *Hippocampus* **14**, 292–300, DOI: 10.1002/hipo.10183 (2004).

35. Aminoff, E. M., Kveraga, K. & Bar, M. The role of the parahippocampal cortex in cognition. *Trends Cogn Sci* **17**, 379–90, DOI: 10.1016/j.tics.2013.06.009 (2013).

36. Maroun, M. Stress reverses plasticity in the pathway projecting from the ventromedial prefrontal cortex to the basolateral amygdala. *Eur J Neurosci* **24**, 2917–22, DOI: 10.1111/j.1460-9568.2006.05169.x (2006).

37. Kessel, E. M. *et al.* Hurricane sandy exposure alters the development of neural reactivity to negative stimuli in children. *Child Dev* **89**, 339–348, DOI: 10.1111/cdev.12691 (2018).

38. Funder, D. C. & Ozer, D. J. Evaluating effect size in psychological research: Sense and nonsense. *Adv. Methods Pract. Psychol. Sci.* **2**, 156–168 (2019).

39. Abelson, R. P. A variance explanation paradox: When a little is a lot. *Psychol. Bull.* **97**, 129–133 (1985).

40. Kavanagh, J. *et al. News in a Digital Age: Comparing the Presentation of News Information over Time and Across Media Platforms* (RAND Corporation, Santa Monica, CA, 2019).

Methods

Data analyses were conducted on the ABCD Fix Release 2.0.1. Comprehensive details about the ABCD Study are published elsewhere (see Developmental Cognitive Neuroscience Special Issue 2018, v32, pp. 1-164). Data from the sub-study about Hurricane Irma were included in this curated annual release. The study was approved by the University of California at San Diego Institutional Review Board. In addition to compensation as part of the parent ABCD study, participants who participated in the Irma-focused sub-study were compensated $20 for each survey completed. Parents who had more than one child enrolled in the study completed a parent survey for each child. Each child completed a child survey for themselves.

Participants

The sample of participants was comprised of those children and families who enrolled in the ABCD study and were tested at the baseline visit before September 7, 2017, at one of four study sites—Florida International University (FIU) in Miami, Florida; University of Florida (UF) in Gainesville, Florida; Medical University of South Carolina (MUSC) in Charleston, SC; and University of California at San Diego (UCSD) in San Diego, CA. Children and parents completed several measures as part of the original ABCD baseline visit, and also completed additional online questionnaires (via REDCap) about their experiences during Hurricane Irma (described below). All youth were subdiagnostic for PTSD. Table 1 provides a breakdown of the number of children and parents who filled out the surveys. The average response rate was 48% for children, and 56% for parents.

Demographically, the ABCD Study used a multi-stage sample of eligible children by probability sampling of schools within the catchment area of each site. The goal of this sampling strategy was to match the demographic profile of two national surveys, the American Community Survey (ACS; a large-scale survey of approximately 3.5 million households conducted annually by the U.S. Census Bureau) and annual 3rd and 4th grade school enrollment data maintained by the National Center for Education Statistics. The sampling strategy was additionally constrained by the requirement that study sites had available magnetic resonance imaging (MRI) scanners. Because these are typically available at research universities in urban areas, the sampling tends to oversample urban as opposed to rural students and families. Thus, although the ABCD Study sample was largely successful at matching the ACS survey demographic profiles[1], it is best described as a population-based, demographically diverse sample that is not necessarily representative of the U.S. national population. Demographic assessments of the sample are summarized here in Barch et al.[2]. The demographic profile of the present Irma sub-study sample, separated by site, is presented in Extended Data Table 1.

Missing Data

We focused on dealing with missing data for the demographic and covariate mental health measures, which was minimal to begin with (see Extended Data Table 2). For the three missing demographic and covariate variables (highest household income, household marital status, K-SADS Pre-Hurricane trauma exposure), we proceeded to missing data multiple imputation for demographic measures using the Multivariate Imputation via Chained Equations (MICE) package in R (v. 3.6). Missing data for other measures (e.g., brain measures, missing survey data) was dealt with using case-wise deletion, and is detailed in the relevant section describing each measure.

Measures

In the present study, we used demographic, mental health, and neuroimaging measures from the ABCD Baseline Visit, all of which were collected prior to Hurricane Irma. We also collected follow-up Hurricane Irma Survey measures on direct hurricane exposure, anticipatory Irma-related media exposure, and Irma-related PTS from participants at the four study sites: FIU, UF, MUSC, and UCSD. Hurricane Irma occurred in September, 2017, and these follow-up data were collected in March-May of 2018. A 6-8 month post-Irma follow-up interval was selected for the supplemental survey to detect PTS responses that could be distinguished from more transitory acute stress responses, and to account for the number of children who take up to 6 months to develop PTS syndromes[3–6].

Pre-Hurricane Measures from ABCD Baseline Visit

Baseline Anxiety. Controlling for prior anxiety mitigates the possibility that media-related findings simply reflect the possibility that anxious youth seek out more threat-related news. To control for pre-disaster anxiety, we used data from the Child Behavior Checklist (CBCL[7]) collected as part of the baseline visit. The CBCL is a well-supported, standardized parent-report assessing internalizing and externalizing youth psychopathology. Empirically based scales, normed for age and gender, are generated, including Internalizing, Externalizing, and Total Problems, as well sub-scales assessing anxiety, depression, somatic complaints, social problems, attention problems, rule-breaking behavior, and aggression. Our analysis focused on the Anxiety Problems subscale.

Prior Trauma Exposure. Controlling for prior trauma is important because exposure to past traumatic experience is a predictor of future PTSD[8] and is associated with PTS responses in disaster victims[9]. To control for pre-disaster exposure to trauma, we used the data from the Parent Diagnostic Interview for DSM-5 Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS), modified for ABCD[2]. This was collected as part of the ABCD baseline visit. The K-SADS is a semi-structured interview that asks about the child's history of general trauma exposure,

Extended Data Table 1. Demographics of the Irma Sub-Study of the Adolescent Brain and Cognitive Development (ABCD)

Demographic	Site			
	FIU	MUSC	UF	UCSD
Age in Months (M (SD))	118.7 (7.1)	119.8 (6.9)	120.4 (7.2)	119.2 (7.3)
Gender %				
Male	52.9	53.5	51.1	53.5
Female	47.1	46.4	48.8	46.5
Household Married %				
No	42.7	15.2	34.5	31.0
Yes	57.3	84.8	65.5	69.0
Race/Ethnicity %				
Hispanic	79.0	3.0	6.0	47.9
White	7.6	78.8	60.7	35.2
Black	10.2	9.1	19.0	11.3
Asian	1.9	1.0	2.4	2.1
Other	1.3	8.0	11.9	8.5
Household Income %				
<50K	48.4	13.1	35.7	40.1
>=50K and <=100K	34.4	29.3	38.1	23.9
>=100K	17.2	57.6	26.1	35.2
Household Highest Education %				
< High School	9.6	0.0	8.3	9.1
High School Diploma/GED	5.1	3.0	7.1	15.5
Some College	35.7	17.1	26.2	31.1
Bachelor's Degree	28.7	28.3	11.9	21.8
Post-graduate Degree	21.0	51.5	46.4	22.5

including learning about unexpected death of a loved one, exposure to sexual or physical abuse, threats on the child's life, witness to violence or mass destruction, involvement in a car accident or intensive medical treatment, or witness to or present during an act of terrorism or natural disaster. Parents either endorse or do not endorse each question about their child, for a total of 17 questions.

Functional MRI: EN-Back Task. Administration of the ABCD Emotional N-Back (EN-Back) is described in detail elsewhere[10]. Briefly, the EN-Back is designed to engage emotion regulation and working memory processes. The memory component of the EN-back activates core brain networks relevant for working memory[11], while the emotional valence of the stimuli of the task (happy, fearful, and neutral faces) is designed to elicit responses from fronto-limbic circuitry implicated in emotional reactivity and regulation[12].

The task includes two runs of eight blocks each. On each trial, participants are asked to respond as to whether the picture is a "Match" or "No Match." Participants are told to make a response on every trial. In each run, four blocks are 2-back conditions for which participants are instructed to respond "match" when the current stimulus is the same as the one shown two trials back. There are also four blocks of the 0-back condition for which participants are instructed to respond "match" when the current stimulus is the same as the target presented at the beginning of the block. At the start of each block, a 2.5s cue indicates the task type ("2-back" or "target=" and a photo of the target stimulus). A 500 ms colored fixation precedes each block instruction, to alert the child of a switch in the task condition. Each block consists

of 10 trials (2.5s each) and 4 fixation blocks (15s each). Each trial consists of a stimulus presented for 2s, followed immediately by a 500ms fixation cross. Of the 10 trials in each block, 2 are targets, 2–3 are non-target lures, and the remainder are non-lures (i.e., stimuli only presented once). There are 160 trials total with 96 unique stimuli of 4 different stimulus types (24 unique stimuli per type).

In the Emotional variant of the task, blocks of trials consist of happy, fearful, and neutral facial expressions as well as places. The facial stimuli are drawn from the NimStim emotional stimulus set[13] and the Racially Diverse Affective Expressions (RADIATE) stimulus set[14]. The place stimuli are drawn from previous visual perception studies[14].

Extended Data Table 2. Missing data by demographic or covariate measure

Measure	Missing Data Points (%)
CBCL Anxiety Index	0 (0.0)
K-SADS Trauma Exposure	63 (12.9)
Age	0 (0.0)
Biological Sex	0 (0.0)
Highest Household Income	51 (11.0)
Highest Household Education	0 (0.0)
Household Marital Status	4 (0.8)
Race/Ethnicity	0 (0.0)
ABCD Site	0 (0.0)
ABCD Family ID Number	0 (0.0)

Neuroimaging Acquisition and Analysis

Data are from the the curated public release of the ABCD study, which reports imaging activity profiles summarized in *apriori* defined regions of interest (ROIs). Data assessing the individual response to Fear and Neutral EN-Back conditions were part of the "fast-track" data release, and are analyzed at the whole-brain level. The acquisition parameters, image post-processing steps, and selection of ROIs are described below.

Imaging Parameters. Data were collected prior to the hurricane on 3T Siemens Prisma (FIU, MUSC, UF) and 3T GE 750 (UCSD) MRI scanners. These magnets employ the Harmonized Human Connectome Project Protocol optimized for ABCD[15]. This protocol makes use of state of the art multiband imaging with prospective motion correction (PROMO/vNav), and EPI distortion correction (EPIC). Real-time head motion monitoring (fMRI Integrated Real-time Motion Monitor, FIRMM[16]) was employed. The imaging data analyzed as part of the present study are (1) Anatomical scans (used to define ROIs) collected with a 3D T1-weighted MPRAGE sequence with prospective motion correction (sagittal; 1 x 1 x 1 mm; matrix = 256 x 256mm), (2) fMRI scans collected with a 3D T2*-weighted EPI sequence (axial; 2.4 x 2.4 x 2.4 mm; FOV = 216 x 216 mm; TR/TE =

800/30 ms; multiband acceleration = 6; 60 slices no gap).

Our analysis focused on the comparison between the Fear and Neutral face conditions of the EN-Back task. We conducted a whole-brain analysis, and an analysis of *apriori* defined regions of interest (ROIs) associated with anxiety, emotion regulation, and PTSD[17–21]. These ROIs, based on the Destrieux parcellation from Freesurfer[22], are: 1) left and right **amygdala**; 2) left and right **hippocampus**; 3) left and right **orbitofrontal cortex** (orbital H-shaped sulcus); 4) left and right **parahippocampal gyrus** (medial occipototemporal parahippocampal gyrus); 5) left and right **anterior cingulate cortex** (anterior cingulate gyrus and sulcus).

Notably, some children were fatigued by the length of the MRI scanner protocol, and due to this attrition data on the EN-back were only available for 74% of the sample. Thus, the results for analyses of neuroimaging data are reported for this sample of children who completed the task, and the effective degrees of freedom after including covariates is $df = 281$. Details on the post-processing steps are included here[15], but briefly the processing steps employed corrections for gradiant non-linearities and resampling to isotropic voxel resolution, and additional motion correction and B0 distortion correction steps for the fMRI. For the ROI analysis, estimates of activation strength were computed at the individual subject level (i.e., "original space") using the general linear model, and averaged across the two runs (weighted by degrees of freedom). We examined the contrast of the mean beta weight (activation over baseline) for the Fear condition vs the mean beta weight (activation over baseline) for the Neutral condition (i.e., the difference score). The average of the difference score for these conditions was summarized for each ROI. These ROI data are available through the National Data Archive as part of the tabulated data release.

Simultaneous to this examination, we conducted a whole-brain analysis on the minimally post-processed brain images not available as part of the tabulated data release. This was done for two reasons: 1) information about activity within each condition relative to resting baseline are not available as part of the tabulated release, and examining activation within each condition above baseline is necessary for understanding the nature of activation differences from the ROI analysis; 2) it is possible that results would be revealed in regions outside those we focused on in the ROI analysis, and these would be identified by the whole-brain analysis.

For the whole-brain analysis, the post-processing steps were identical, except that each brain was warped to the MNI template to facilitate group-level voxel-wise analysis. Using AFNI (v.20.1.14), we explored two comparisons: 1) Fear vs. Neutral condition differences and the association of these activation differences and PTS symptoms (i.e., the EN-Back main effect); and 2) moderation of the association between

media exposure and PTS symptoms by the Fear vs. Neutral activation difference (i.e., the EN-Back by Media interaction). Details of these comparisons are presented below. For each comparison, a per-voxel threshold of $p < .01$ was applied. A family-wise error cluster correction was applied by estimating the spatial smoothing from the residuals of the statistical model, iteratively generating a 3D grid of independent and identically distributed random deviates, smoothing them to the level estimated from the residuals, and finally generating a distribution of cluster sizes at the established per-voxel threshold (AFNI 3dClustSim[23]).

Post-Hurricane Survey Measures

From March-May 2018 (following Hurricane Irma in September 2017), children and parents each completed an online survey of their experiences before, during, and after hurricane Irma, relating to both objective and subjective experiences about the hurricane and media exposure surrounding the hurricane. The survey was presented online using the REDCap software, which incorporated skip logic for questions that did not apply to certain participants (e.g., San Diego participants did not answer certain questions related to direct exposure to the hurricane). In addition, questions were translated to Spanish by certified translators at FIU, and thus were available in either English or Spanish. The online questionnaire was distributed at each of the four study sites via email, which linked to the survey.

Hurricane Exposure. Children and parents completed the Hurricane Related Traumatic Experiences–II (HURTE-II), an updated iteration of the HURTE-R[24, 25] which has been used extensively in hurricane research to assess hurricane exposure and post-disaster stressors. The HURTE-II assesses stressors before (e.g., evacuation experiences), during (e.g., perceived life threat, actual life threat, immediate loss/disruption), and ongoing stress, loss, and disruption after the storm. An Irma-related media exposure questionnaire was also developed specifically for the context of Hurricane Irma[26].

For the present analysis, we focused on Objective Exposure and pre-storm Irma-related Media Exposure. Objective Exposure tallied the number of items families endorsed reflecting direct Irma-related harm (e.g., child hit by falling or flying objects during hurricane?), witnessing exposure (e.g., child saw someone badly hurt during hurricane), or damage to property (e.g., broken windows, flooding, or water damage from storm) during and after the hurricane. Data on an independent sample suggest that such exposures were significant sources of stress for families involved in Hurricane Irma, both before and during the storm and surrounding evacuation[27]. The Objective Exposure variable is determined by parent report. Because California was not in the storm's path, participants at the UCSD site did not answer these questions, and some parents at other sites did not provide

answers. The effective sample size for this variable was thus $n = 324$.

For Irma-related media exposure, we focused on *pre*-storm media exposure because (a) most research on mental health consequences of disaster-related media exposure has focused on coverage during and after the event, neglecting potentially important effects of threat-related anticipatory coverage; and (b) storm-related power outages restricted media access in hurricane-affected areas, which would differentially affect some children in the study but not others. Focusing on *pre*-storm coverage allowed us to compare and integrate data from children across affected and non-affected regions, who all had comparable opportunity for media exposure. Three child self-report items asked how often the child (1) viewed Irma-related television coverage before the storm (e.g., news stations, weather channel etc); (2) checked for news and updates using the Internet (e.g., news or NOAA websites); and (3) engaged in Irma-related social media activity (e.g., Facebook, twitter, Instagram). Items were rated on a scale of 0-4. Anchors for the television item included 0 ("Not at all"), 2 ("Somewhat, about an hour per day"), and 4 ("A whole lot, more than 2 hours per day"). Anchors for the Internet and social media items included 0 ("Once per day or less"), 2 ("Almost every hour"), and 4 ("Almost continuously"). Ratings on the three items were summed to yield a total score, and 396 scores were available for analysis.

Irma-related PTS. To assess Irma-related PTS symptoms, children completed the well-validated UCLA Reaction Index for DSM-5[28–30]. The UCLA Reaction Index is a child self-report, and it is the most commonly used measure of child PTS used in research conducted in the aftermath of disasters[4]. The measure maps onto DSM-5 PTS symptoms, and measures how often children experienced each symptom in the past month (ranging from 0-"Never." to 4-"Almost every day."). For all items, PTS symptoms were worded to specifically pertain to Hurricane Irma (e.g., "When something reminds me of Hurricane Irma I get very upset, afraid or sad"). Responses are summed to obtain a "PTS Symptom Total". There were 393 scores available for analysis.

Outlier Detection and Correction

We did not remove outliers but down-weighted their influence using a conservative 97.5% Winsorization procedure, and robust statistical procedures (see below). Data for PTS Symptom Total and K-SADS Pre-Trauma exposure had very large outliers (data points greater than 7 standard deviations from the mean) and were Winsorized.

Robust Multiple Regression

Multiple regression was conducted using robust statistical procedures and bootstrapping approaches. Specifically, we conducted robust regressions using a Huber loss function, which down-weights the influence of, but does not remove, outliers. In cases where there are no outliers, robust regression

provides similar or identical results to ordinary least-squares regression, but performs better when there are outliers[31]. To conduct the bootstrap we used a parametric bootstrap with 10,000 bootstrap replicates. The bootstrap standard errors were then used to define 95% Confidence Intervals of the parameter estimates.

A small number of participants (21 families) had siblings in the sub-study. Although modeling family-related effects is recommended for the full ABCD sample, the number of families was too small to do so here. As detailed below, site effects were investigated for questions related to objective and media exposure, and specifically modeled for the neuroimaging analysis to account for scanner differences.

In each regression, the following covariates were entered in the model as fixed effects: (1) age, (2) birth sex, (3) race/ethnicity, (4) highest degree of parental education, (5) household income, (6) parental marital status. For analyses investigating functional imaging predictors, the MRI scanner serial number was entered as a 7th covariate, to control for the use of four different scanners. In follow-up analyses, CBCL Anxiety Problems and K-SADS Prior Trauma were also examined to establish whether hurricane-related measures were predictive of PTS outcomes over and above what might be predicted by baseline anxiety and prior trauma exposure. Although CBCL Anxiety was not associated with Irma-related PTS (controlling for demographic covariates; $B = 0.12$, $t(378) = 1.58$, $p = 0.13$, 95% Confidence Interval = -0.03 to .28, $\beta = 0.05$), prior trauma exposure was strongly associated with PTS symptoms (controlling for CBCL Anxiety and demographic covariates; $B = 0.98$, $t(377) = 3.32$, $p = .0009$, 95% Confidence Interval = 0.40 to 1.56, $\beta = 0.10$).

For the first analysis, we examined the relation between objective Irma exposure and Irma-related PTS symptoms within the South Florida youth sample most directly affected by Hurricane Irma (i.e., the Florida International University site). There was a significant association between objective Irma exposure and PTS symptoms, $B = 0.43$, $t(109) = 2.43$, $p = 0.017$, 95% Confidence Interval = 0.08 to .78, $\beta = 0.14$. When all sites affected by the Hurricane were examined (i.e., FIU, UF, and MUSC), the effect was also significant ($B = 0.29$, $t(255) = 2.21$, $p = 0.028$, 95% Confidence Interval = 0.03 to 0.55, $\beta = 0.09$, see Figure 1). The results were nearly identical when controlling for prior anxiety and trauma exposure ($B = 0.48$, $t(107) = 2.49$, $p = 0.014$, 95% Confidence Interval = 0.09 to .80, $\beta = 0.14$ for the South Florida FIU site; ($B = 0.28$, $t(253) = 2.10$, $p = 0.037$, 95% Confidence Interval = 0.02 to 0.54, $\beta = 0.09$ for all affected sites). This analysis shows that objective exposure to the hurricane at Irma-affected sites predicted PTS symptoms, even after controlling for baseline anxiety and trauma.

For the second analysis, we examined the relation between

Irma-related media exposure before the hurricane and PTS symptoms, controlling for site (Southern California/UCSD site versus Irma State, i.e., FIU, UF, and MUSC) and demographic covariates. The regression model revealed a significant association between Irma-related media exposure and PTS symptoms, $B = 0.41$, $t(377) = 4.84$, $p = 0.000002$, 95% Confidence Interval = 0.24 to 0.57, $\beta = 0.15$. The results were nearly identical when controlling for prior anxiety and trauma, $B = 0.40$, $t(375) = 4.61$, $p = 0.00003$, 95% Confidence Interval = 0.23 to 0.56, $\beta = 0.15$. To determine if those who experienced the direct effects of the hurricane were deferentially influenced by media exposure, we added site as a moderator. The interaction between site and Irma-related media exposure was not significant, $B = -0.09$, $t(376) = -0.30$, $p = 0.72$, 95% Confidence Interval = -0.64 to 0.47; $\beta = -0.03$. This suggests that the effects of Irma-related media exposure on PTS symptoms was uniform across youth in affected and non-affected regoins (i.e., children who were over 4500 kilometers from the hurricane; Figure 2).

For the third analysis, we examined the relation between Irma-related media exposure before the hurricane and PTS symptoms, with the activation difference between the Fear and the Neutral conditions of the EN-Back entered as a moderator. This analysis thus examines whether 1) the activation difference in any brain region predicts PTS symptoms (i.e., the main effect of EN-Back Fear vs. Neutral), and 2) whether pre-existing neural vulnerability influences the strength of the relation between media exposure and PTS symptoms, which was established in the prior analysis (i.e., the EN-Back by Media interaction). These analyses were conducted at both the whole-brain level and at the ROI level, where ROIs were defined on the individual brain space of each subject.

First we report the results of the whole-brain analysis of activation differences between Fear and Neutral conditions. At the whole-brain level, for the main comparison between conditions, we found a reliable difference between the Fear vs. Neutral conditions in bilateral amygdala (see Figure 3; $p < .01$, uncorrected). This did not survive the cluster correction, although this may be due to the small area of amygdala, and thus some caution in dismissing these results is warranted. Furthermore, the finding replicates a number of previous studies showing the amygdala's central role in processing fear-related stimuli[20], and the difference is in the expected direction (Fear > Neutral). Notably, though, this was the only significant effect at the whole brain that was evident in *a priori* defined ROIs (see null effects, Extended Figure 1). Other regions outside our *a priori* defined ROIs showed a reliable difference between conditions, but are not examined further here. The purpose of this analysis is simply to understand whether the Fear vs. Neutral manipulation worked as expected, which establishes a framework on which to understand the results of our main analysis, that of the

Extended Data Figure 1. **Activation in EN-Back in cortical and subcortical regions of interest (ROIs).** Five ROIs were defined with reference to an anatomical atlas for each hemisphere. These are 1) left and right **amygdala**; 2) left and right **hippocampus**; 3) left and right **orbitofrontal cortex** (orbital H-shaped sulcus); 4) left and right **parahippocampal gyrus** (medial occipototemporal parahippocampal gyrus); 5) left and right **anterior cingulate cortex** (anterior cingulate gyrus and sulcus). Whole-brain activation maps show the comparison of Fear vs. Neutral ($p <$ .01, uncorrected). Bar plots show the summary activation profiles for each condition within each ROI.

association between brain response and PTS symptoms, and the brain response by media interaction predicting PTS symptoms.

Next, we report the results of the main effect of EN-Back predicting PTS symptoms. At the whole-brain level, there were no significant clusters indicating an association between the EN-Back difference and PTS symptoms. Despite no significant effects at the whole-brain level, to be comprehensive

we did examine whether there were reliable associations in our ten *a priori* defined ROIs. There was a reliable association in right amygdala ($B = $ -1.65, $t(281) = $ -3.27, $p = $ 0.001, 95% Confidence Interval = -2.63 to -0.66, $\beta = $ -0.14), but no reliable main effects were revealed in the remaining regions of interest. Thus, there was some modest evidence that reactivity in amygdala pre-hurricane predicted PTS symptoms post-hurricane, but it was only evident in the ROI analysis.

Finally we statistically explored our primary question of interest and investigated whether any regions showed an EN-Back by Media interaction. We found several reliable effects at both the whole-brain and ROI levels of analysis. At the whole-brain level, significant interaction effects (p < .01, corrected) were evident in bilateral orbital sulcus, parahippocampal gyrus, superior frontal gyrus, superior and middle occipital gyrus, and right middle frontal sulcus, orbital gyrus, and fusiform gyrus (Figure 4). With these whole brain results in hand, we moved to examine the reliability of these clusters at the ROI level.

Some of the regions revealed at the whole-brain analysis level were unexpected (i.e., frontal pole/orbital gyrus, superior frontal gyrus, superior and middle occipital gyrus, and right middle frontal sulcus, and fusiform gyrus). None of those results were reliable at the subject-specific anatomical space (smallest $p = $ 0.11). There were, however, regions where reliable results were found at both the whole-brain and in our *a priori* defined ROIs (shown in Table 2; the Benjamini-Hochberg False Discovery Rate (FDR) correction[32] was applied across the 10 *a priori* defined ROIs). The three findings that were statistically reliable in both analyses were **left and right orbital sulcus**, and **left parahippocampal gyrus**. In these regions the EN-Back activation difference measured pre-hurricane moderated the relation between pre-hurricane media exposure and post-hurricane PTS symptoms, and the nature of the interaction was the same in all three regions (Figure 4). That is, in these regions children who showed a strong positive response to the Neutral Face condition, coupled with a weak or below-baseline response to the Fear Face condition, were especially susceptible to PTS as a result of Irma-related media exposure. At the same time, those who showed a strong positive response to the Fear Face condition, coupled with a weak or below-baseline response to the Neutral Face condition, were less susceptible to PTS as a result of Irma-related media exposure. Because the results for these three regions were reliable at both the whole-brain and individual-subject levels, we focus on them in our discussion.

Power Estimates and Effect Sizes
Effect sizes in this study, especially for the brain effects, were small. Thus, despite reasonable sample sizes (> 300 for most analyses), power to detect small effect sizes was

low. For example, power estimates for the brain measures predicting behavior were low (power = 0.41 for effect sizes around r = .10, at α = .05). There is thus the possibility of higher Type II error for the brain effects. That said, effect sizes for potentially missed effects were universally small (i.e., approaching zero), and inspection of the confidence intervals for these effects suggests that the failure to reject the null hypothesis in these cases is likely to be due to a true absence of effect (see Table 2). Analyses of the effects of Irma exposure and Irma media exposure had higher power, due to both larger sample size (i.e., due to less missing data) and larger effects (e.g., power = 0.99 for effect sizes around r = .30 at α = .05). Power analyses were based on Cohen[33].

Data Availability

The ABCD data repository grows and changes over time. The ABCD data used in this report came from RDS Fix Release 2.0.1 http://dx.doi.org/10.15154/1504431 and from the minimally processed imaging data available through abcd-sync. The data are available by request from the NIMH Data Archive (https://data-archive.nimh.nih.gov/abcd).

Code Availability

Acknowledgements

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A listing of participating sites and a complete listing of the study investigators can be found at https://abcdstudy.org/consortium_members/. ABCD consortium investigators designed and implemented the study and/or provided data but did not necessarily participate in analysis or writing of this report. This manuscript reflects the views of the authors and may not reflect the opinions or views of the NIH or ABCD consortium investigators. The funders had no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

Author contributions statement

All authors contributed to the conception of the study and/or collection and curation of the data. A.S.D. and W.K.T analysed the data. A.S.D. and J.S.C. wrote the draft manuscript. All authors reviewed and commented on the draft for the final write-up of the study. All authors reviewed and approved the manuscript.

Competing Interests

The authors declare no competing interests.

References

1. Garavan, H. *et al.* Recruiting the abcd sample: Design considerations and procedures. *Dev Cogn Neurosci* DOI: 10.1016/j.dcn.2018.04.004 (2018).

2. Barch, D. M. *et al.* Demographic, physical and mental health assessments in the adolescent brain and cognitive development study: Rationale and description. *Dev Cogn Neurosci* DOI: 10.1016/j.dcn.2017.10.010 (2017).

3. Association, A. P. *Diagnostic and statistical manual of mental disorders* (Author, Washington, DC, 2013), 5th edn.

4. Furr, J. M., Comer, J. S., Edmunds, J. M. & Kendall, P. C. Disasters and youth: a meta-analytic examination of posttraumatic stress. *J Consult. Clin Psychol* **78**, 765–80, DOI: 10.1037/a0021482 (2010).

5. Lai, B. S., Lewis, R., Livings, M. S., La Greca, A. M. & Esnard, A. M. Posttraumatic stress symptom trajectories among children after disaster exposure: A review. *J Trauma Stress.* **30**, 571–582, DOI: 10.1002/jts.22242 (2017).

6. Green, B. L. & Lindy, J. D. Post-traumatic stress disorder in victims of disasters. *Psychiatr Clin North Am* **17**, 301–9 (1994).

7. Achenbach, T. M. & Rescorla, L. *Manual for the ASEBA school-age forms profiles : an integrated system of multi-informant assessment* (ASEBA, Burlington, VT, 2001).

8. Delahanty, D. L. & Nugent, N. R. Predicting ptsd prospectively based on prior trauma history and immediate biological responses. *Ann N Y Acad Sci* **1071**, 27–40, DOI: 10.1196/annals.1364.003 (2006).

9. Bonanno, G. A., Brewin, C. R., Kaniasty, K. & Greca, A. M. Weighing the costs of disaster: Consequences, risks, and resilience in individuals, families, and communities. *Psychol Sci Public Interest* **11**, 1–49, DOI: 10.1177/1529100610387086 (2010).

10. Casey, B. J. *et al.* The adolescent brain cognitive development (abcd) study: Imaging acquisition across 21 sites. *Dev Cogn Neurosci* **32**, 43–54, DOI: 10.1016/j.dcn.2018.03.001 (2018).

11. Barch, D. M. *et al.* Function in the human connectome: task-fmri and individual differences in behavior. *Neuroimage* **80**, 169–89, DOI: 10.1016/j.neuroimage.2013.05.033 (2013).

12. Hare, T. A. *et al.* Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biol Psychiatry* **63**, 927–34, DOI: 10.1016/j.biopsych.2008.03.015 (2008).

13. Tottenham, N. *et al.* The nimstim set of facial expressions: judgments from untrained research participants. *Psychiatry Res* **168**, 242–9, DOI: 10.1016/j.psychres.2008.05.006 (2009).

14. Conley, M. I. *et al.* The racially diverse affective expression (radiate) face stimulus set. *Psychiatry Res* **270**, 1059–1067, DOI: 10.1016/j.psychres.2018.04.066 (2018).

15. Hagler, J., D. J. *et al.* Image processing and analysis methods for the adolescent brain cognitive development study. *Neuroimage* **202**, 116091, DOI: 10.1016/j.neuroimage.2019.116091 (2019).

16. Dosenbach, N. U. F. *et al.* Real-time motion analytics during brain mri improve data quality and reduce costs. *Neuroimage* **161**, 80–93, DOI: 10.1016/j.neuroimage.2017.08.025 (2017).

17. Robinson, O. J., Pike, A. C., Cornwell, B. & Grillon, C. The translational neural circuitry of anxiety. *J Neurol Neurosurg Psychiatry* DOI: 10.1136/jnnp-2019-321400 (2019).

18. Robinson, O. J. *et al.* Towards a mechanistic understanding of pathological anxiety: the dorsal medial prefrontal-amygdala 'aversive amplification' circuit in unmedicated generalized and social anxiety disorders. *Lancet Psychiatry* **1**, 294–302, DOI: 10.1016/S2215-0366(14)70305-0 (2014).

19. Heller, A. S. *Functional brain imaging and PTSD*, 315–330 (Oxford University Press, New York, 2018).

20. Shin, L. M. & Liberzon, I. The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology* **35**, 169–91, DOI: 10.1038/npp.2009.83 (2010).

21. LeDoux, J. E. & Pine, D. S. Using neuroscience to help understand fear and anxiety: A two-system framework. *Am J Psychiatry* **173**, 1083–1093, DOI: 10.1176/appi.ajp.2016.16030353 (2016).

22. Destrieux, C., Fischl, B., Dale, A. & Halgren, E. Automatic parcellation of human cortical gyri and sulci using standard anatomical nomenclature. *Neuroimage* **53**, 1–15, DOI: 10.1016/j.neuroimage.2010.06.010 (2010).

23. Cox, R. W., Chen, G., Glen, D. R., Reynolds, R. C. & Taylor, P. A. Fmri clustering in afni: False-positive rates redux. *Brain Connect.* **7**, 152–171, DOI: 10.1089/brain.2016.0475 (2017).

24. La Greca, A. M., Silverman, W. K., Lai, B. & Jaccard, J. Hurricane-related exposure experiences and stressors, other life events, and social support: concurrent and prospective impact on children's persistent posttraumatic stress symptoms. *J Consult. Clin Psychol* **78**, 794–805, DOI: 10.1037/a0020775 (2010).

25. Vernberg, E. M., Silverman, W. K., La Greca, A. M. & Prinstein, M. J. Prediction of posttraumatic stress symptoms in children after hurricane andrew. *J Abnorm Psychol* **105**, 237–48 (1996).

26. Carpenter, A. L. *et al.* Event-related household discussions following the boston marathon bombing and associated posttraumatic stress among area youth. *J Clin Child Adolesc Psychol* **46**, 331–342, DOI: 10.1080/15374416.2015.1063432 (2017).

27. La Greca, A. M. *et al.* Before the storm: Stressors associated with the hurricane irma evacuation process for families. *Disaster Med Public Heal. Prep* **13**, 63–73, DOI: 10.1017/dmp.2019.9 (2019).

28. Steinberg, A. M., Brymer, M. J., Decker, K. B. & Pynoos, R. S. The university of california at los angeles posttraumatic stress disorder reaction index. *Curr Psychiatry Rep* **6**, 96–100 (2004).

29. Steinberg, A. M. *et al.* Psychometric properties of the ucla ptsd reaction index: part i. *J Trauma Stress.* **26**, 1–9, DOI: 10.1002/jts.21780 (2013).

30. Elhai, J. D. *et al.* Psychometric properties of the ucla ptsd reaction index. part ii: investigating factor structure findings in a national clinic-referred youth sample. *J Trauma Stress.* **26**, 10–8, DOI: 10.1002/jts.21755 (2013).

31. Wilcox, R. R. *Introduction to robust estimation and hypothesis testing.* (Academic Press, Waltham, MA, 2012), 3rd edn.

32. Benjamini, Y. & Hochberg, Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J. royal statistical society. Ser. B (Methodological)* 289–300 (1995).

33. Cohen, J. *Statistical power analysis for the behavioral sciences* (Erlbaum, Mahwah, NJ, 1988), 2nd edn.