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Neural vulnerability and hurricane-related media are associated with post-traumatic stress in youth

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The human toll of disasters extends beyond death, injury and loss. Post-traumatic stress (PTS) can be common among directly exposed individuals, and children are particularly vulnerable. Even children far removed from harm's way report PTS, and media-based exposure may partially account for this phenomenon. In this study, we examine this issue using data from nearly 400 9- to 11-year-old children collected before and after Hurricane Irma, evaluating whether pre-existing neural patterns moderate associations between hurricane experiences and later PTS. The 'dose' of both self-reported objective exposure and media exposure predicted PTS, the latter even among children far from the hurricane. Furthermore, neural responses in brain regions associated with anxiety and stress conferred particular vulnerability. For example, heightened amygdala reactivity to fearful stimuli moderated the association between self-reported media exposure and PTS. Collectively, these findings show that for some youth with measurable vulnerability, consuming extensive disaster-related media may offer an alternative pathway to disaster exposure that transcends geography and objective risk.

In 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[1]. In particular, weather-related disasters, and their associated human and economic tolls, are on the rise[2,3]. In addition to their physical consequences, such disasters carry a broad and sustained mental health toll, with robust post-disaster evidence documenting elevated post-traumatic stress (PTS) responses among large subsets of individuals[4–7]. Children are among the most vulnerable, as they are still developing a stable sense of security and have relatively limited control over their environments[6].

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[8–10], with increasing evidence pointing to the important role that disaster-related media exposure may play in explaining PTS symptoms in distal individuals[11–14]. 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 often preceded by an extensive warning period and considerable pre-event threat-related media attention. Related research considering pre-event media exposure in adult samples[15] has predominantly focused 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 hurricane exposure 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 before 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[15], culminating in the largest human evacuation in American history (about seven million people[16]).

In this paper, we present the results of analyses on 454 well-characterized families from four sites of the Adolescent Brain Cognitive Development (ABCD) Study. The four participating study sites included three that were directly impacted by Hurricane Irma—that is, Florida International University (FIU) in Miami, Florida; the University of Florida (UF) in Gainesville, Florida; and the Medical University of South Carolina (MUSC) in Charleston, South Carolina—and one in a distal, non-impacted state with relatively comparable demographic characteristics—that is, the University of California, San Diego (UCSD) in San Diego, California (Table 1). In the year before 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 self-reports of children's objective hurricane exposure and Irma-related media exposure, as well as Irma-related PTS responses.

Results

Objective exposure is associated with PTS. We began our analysis by establishing the degree to which objective exposure to Hurricane

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

Site	Children	Parents
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%)

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

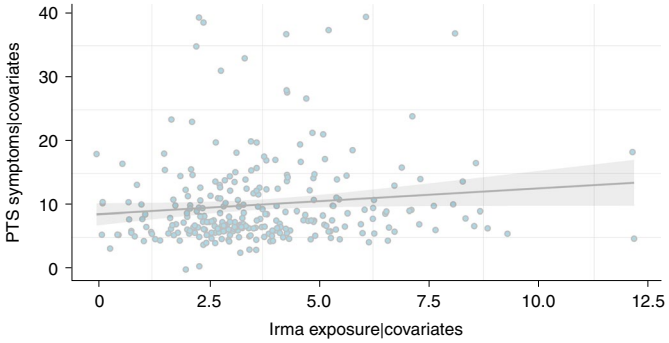

Fig. 1 | Irma exposure predicts post-Irma PTS symptoms among hurricane-exposed youth. The figure shows an added variable plot for data from all hurricane-impacted sites (controlling for covariates; Methods). The error shading represents the 95% CI. The data were rescaled to place the origin at (0,0).

Irma predicted PTS symptoms. We measured objective exposure using the Hurricane Related Traumatic Experiences-II (HURTE-II) survey, which assesses stressors such as 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 (that is, the FIU site in Miami; $t(109)=2.43$; $P=0.017$; standardized regression slope parameter estimate (β), 0.14; semipartial r (r_{sp}), 0.23; unstandardized regression slope parameter estimate (B), 0.43; 95% confidence interval (CI) for B, 0.08 to 0.78). In fact, the objective exposure 'dose' effect was not evenly distributed—it was stronger for the South Florida youth than for the UF and MUSC sites (mean, 3.57 versus 1.97; $t(297.85)=6.95$; $P<0.001$; Cohen's $d=0.81$; mean difference, 1.62; 95% CI, 1.16 to 2.07). However, we found the same pattern when all sites in states directly impacted by Irma (FIU, UF and MUSC) were collectively examined ($t(255)=2.21$; $P=0.028$; $\beta=0.09$; $r_{sp}=0.12$; $B=0.29$; 95% CI for B, 0.03 to 0.55; Fig. 1), although the effect size was notably smaller ($r_{sp}=0.23$ versus 0.12). Furthermore, the results were unchanged when children's baseline anxiety and exposure to prior trauma were entered as covariates ($t(107)=2.49$; $P=0.014$; $\beta=0.14$; $r_{sp}=0.23$; $B=0.48$; 95% CI for B, 0.09 to 0.80 for the South Florida FIU site; $t(253)=2.10$; $P=0.037$; $\beta=0.09$; $r_{sp}=0.11$; $B=0.28$; 95% CI for B, 0.02 to 0.54 for all affected sites). The results thus 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.

Media exposure is associated with PTS. Prior research shows that objective disaster exposure and threat are not always necessary to prompt PTS responses[11–14,17–19]. We therefore broadened our analysis to examine media-based effects. 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 self-reported 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%). Before 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 self-reported media exposure was associated with child PTS outcomes ($t(377)=4.84$; $P=0.000002$; $\beta=0.15$; $r_{sp}=0.23$; $B=0.41$; 95% CI for B, 0.24 to 0.57; even after controlling for prior anxiety and trauma, $t(375)=4.61$; $P=0.00003$; $\beta=0.15$; $r_{sp}=0.21$; $B=0.40$; 95% CI for B, 0.23 to 0.56). 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) or an unaffected state (UCSD youth in Southern California), this factor did not moderate the association between pre-storm self-reported media exposure and youth PTS ($t(376)=-0.30$; $P=0.72$; $\beta=-0.03$; $r_{sp}=-0.04$; $B=-0.09$; 95% CI for B, −0.64 to 0.47). Indeed, the effects of exposure to anticipatory self-reported media on child PTS were robust and uniform across youth, even among those who were over 4,500 kilometres from the storm's path (Fig. 2). Mental health effects associated with storm-related media exposure in the lead-up to Hurricane Irma thus seem to be wide-ranging, extending to youth far beyond the geographic boundaries of the storm's physical projected path.

Neural vulnerability moderates anticipatory media exposure. 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 effects. Here we examined neural biases in a priori defined brain regions associated with anxiety and stress[20–23] (that is, amygdala, hippocampus, orbitofrontal cortex (OFC), parahippocampal gyrus and anterior cingulate cortex (ACC); Fig. 3). Neural bias was measured as the difference in brain activity during the Emotional variant of the classic N-back working memory task (that is, 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 the chosen brain regions. Our reasoning was that this would indicate neural predisposition to processing plain faces as either fear-inducing (that is, essentially not different from overtly fear-inducing stimuli) or neutral (that is, very different from overtly fear-inducing stimuli). We predicted that the amygdala would respond more strongly to Fear versus Neutral conditions, and we predicted an interaction in amygdala such that high amygdala reactivity to fearful stimuli would confer specific vulnerability to self-reported objective and media exposure, as these variables relate to PTS[25]. In addition, because OFC is proposed to play a top-down regulatory role within an extended amygdala network[26], we expected the opposite pattern of response in this region—that is, lower reactivity (translating to lower top-down influence) would confer more risk for later PTS symptoms in response to self-reported objective or media exposure.

For the initial analysis, as expected, we found that fear-inducing stimuli elicit more activity in bilateral amygdala, consistent with the amygdala's important role in the processing of fear-related or threat-related stimuli (Fig. 3, top left panel)[23]. The response in other regions of this network, associated with the regulation of emotion and memory[27], was more variable (Fig. 3, other panels), and we examined whether the neural response biases in amygdala and these other regions moderated the reported associations of objective exposure and pre-storm self-reported media exposure with

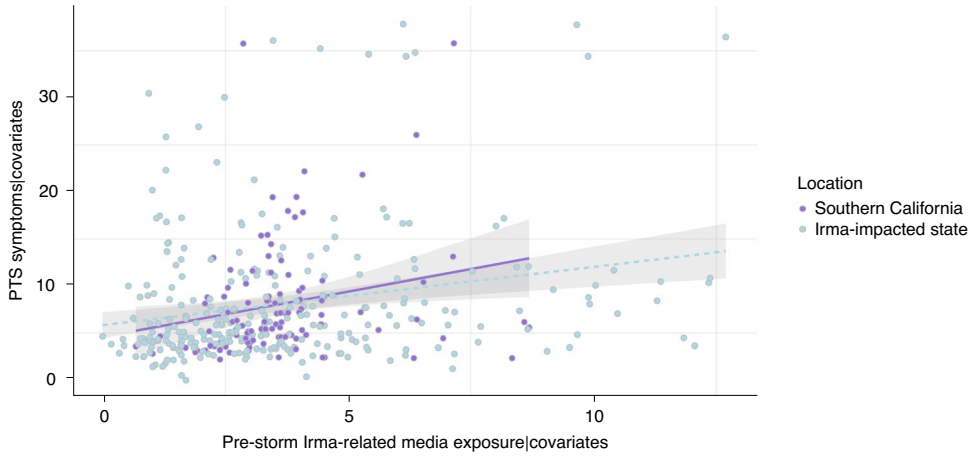

Fig. 2 | Pre-storm media exposure predicts PTS symptoms among children near and far. The figure shows an added variable plot (controlling for covariates; Methods) for self-reported media exposure before the hurricane predicting PTS symptoms in children, regardless of whether children were in a region directly impacted by the hurricane (Florida or coastal South Carolina) or were not (Southern California). The error shading represents the 95% CI. The data were rescaled to place the origin at (0,0).

Irma-related PTS (that is, the interaction of the EN-Back difference score by objective exposure and by pre-storm media exposure).

As a result of this moderation analysis and in line with our predictions, we found a moderating effect of the brain response on the association between anticipatory media exposure and PTS symptoms. First, at the whole-brain level, we found that right amygdala reactivity to fearful stimuli moderated the association between self-reported media exposure and PTS symptoms. The association was strongest for children who had a greater Fear versus Neutral activity difference (Fig. 4), with the interaction effect suggesting that self-reported media exposure affects children most prominently if they had heightened amygdala reactivity to fearful stimuli ($t(276)=3.90$; $P=0.0001$; $\beta=0.27$; $r_{sp}=0.21$; $B=0.82$; 95% CI for B, 0.41 to 1.24). Second, in bilateral OFC and parahippocampal gyrus, the effect is in the opposite direction—that is, the negative interaction slope reflects the fact that, in these regions, children who showed a weak response to the Fear face condition relative to the Neutral condition were especially susceptible to PTS as a result of self-reported media exposure (Fig. 5). These latter effects were also seen in the region of interest (ROI) analysis (Table 2). This suggests that, as amygdala reactivity to fearful stimuli is high, regions regulating that reactivity (such as OFC) fail to exert top-down control, leaving these children more susceptible to media exposure. This interpretation is explored in more detail below.

Contrary to our predictions, we did not find similar effects when we examined the moderating effect of the brain response on the association between objective exposure and PTS symptoms. At both the whole-brain and ROI levels, we found no statistically significant effects (after correction) in brain regions associated with the regulation of emotion and memory, nor in regions previously associated with PTS disorder (PTSD)[27] (Table 2). In the ROI analyses, we did find an effect in right ACC, but this finding did not survive correction for multiple comparisons (Table 2).

Discussion

To facilitate the interpretation of these results, we situate them within neural 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, OFC directly influences the response in amygdala in a top-down fashion[28] to modulate the threat or stress response[26]. Differences in OFC–amygdala interactions can thus partly account for individual differences in emotion regulation[29,30] and stress response[28].

In people with disorders of stress and anxiety, this modulation and the resulting reactivity of amygdala are atypical. For example, compared with people without PTSD, people with PTSD show greater amygdala activation when viewing negative emotional faces and scenes or other trauma-related stimuli[31,32]. More directly, surgical ablation of amygdala is associated with the remediation of PTS symptoms, suggesting its central role in the pathophysiology of the disorder[33].

Our finding of a strong association between media and PTS in children with elevated amygdala reactivity is consistent with the findings of prior work investigating how this extended amygdala system responds to and predicts the response to disaster or trauma exposure. For example, in a functional magnetic resonance imaging (fMRI) study, McLaughlin and colleagues[34] found that amygdala response to negative stimuli in 15 adolescents examined prospectively before a terrorist attack predicts PTS symptoms following the terrorist attack. Similarly, Stevens and colleagues[35] found that amygdala reactivity predicted PTS symptom maintenance after acute trauma (for example, after a car accident). Finally, Swartz and colleagues[25] found a similar effect in their prospective study of 340 young adults. In that fMRI study, there was a significant interaction between threat-related amygdala reactivity and life stress reported post-scanning in predicting the severity of symptoms of depression and anxiety. Consistent with what we found, individuals who had heightened amygdala reactivity at baseline and reported greater life stress also had more symptoms at follow-up, suggesting that amygdala reactivity as an indicator of vulnerability interacts with the experience of increased life stress. Our findings thus add to an existing literature suggesting that heightened amygdala reactivity to negative emotional information is associated with future onset of PTS symptoms or other psychological vulnerability, especially in cases where people experience additional life stressors[34].

The amygdala is only one node in an extended circuit supporting emotion processing and stress response. Neuroimaging research has also shown that brain regions functionally and structurally connected to amygdala are associated with PTSD. For example, PTSD is associated with underactivity and reduced functional connectivity among regions that regulate amygdala function, such as OFC[27,36–40].

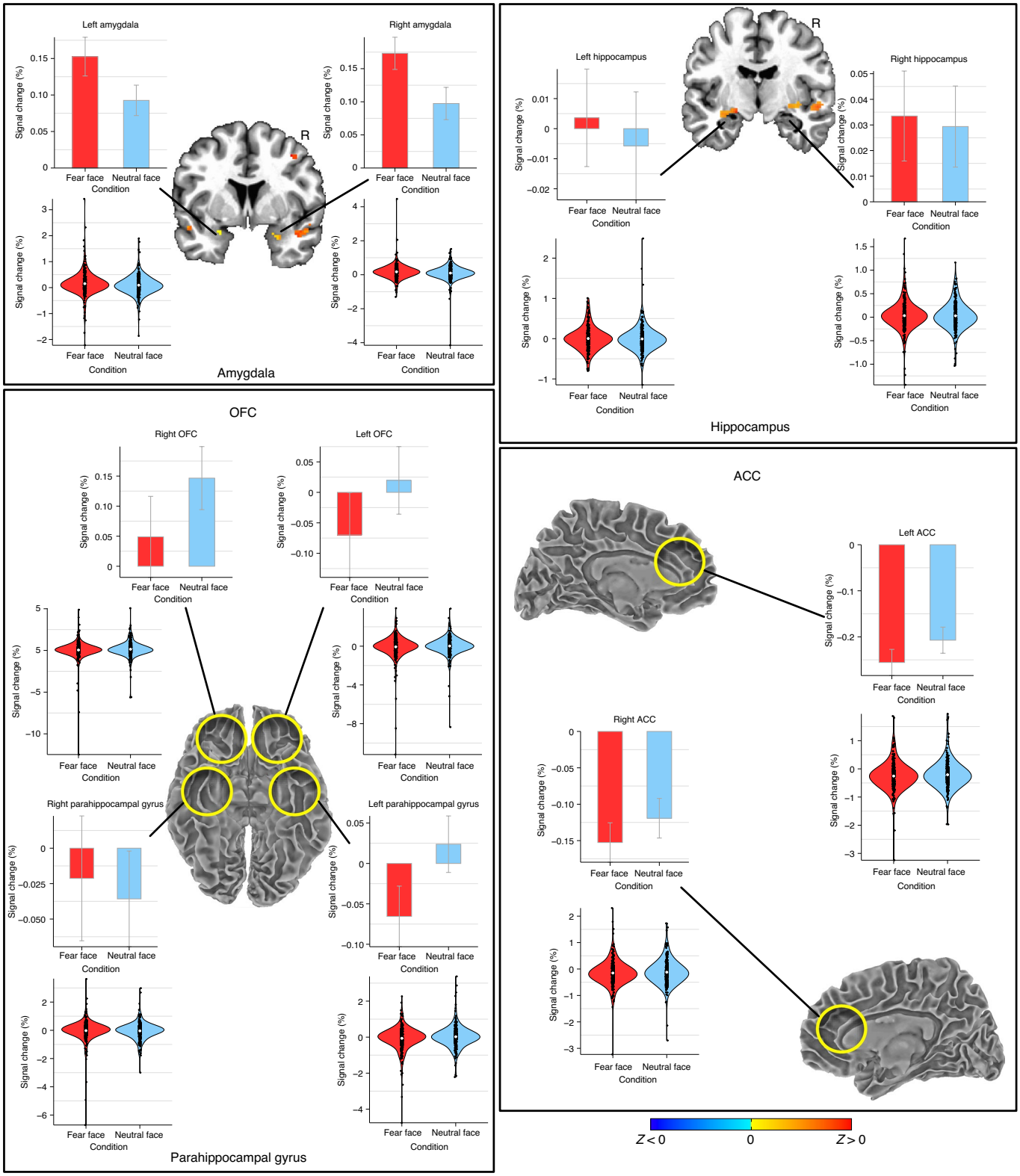

Fig. 3 | Activation in the EN-Back in cortical and subcortical ROIs. Five ROIs were defined with reference to an anatomical atlas for each hemisphere. These are (1) left and right amygdala, (2) left and right hippocampus, (3) left and right OFC (orbital H-shaped sulcus), (4) left and right parahippocampal gyrus (medial occipitotemporal parahippocampal gyrus), and (5) left and right ACC (anterior cingulate gyrus and sulcus). The whole-brain activation maps show the comparison of Fear versus Neutral ($P < 0.005$, corrected) and were apparent only for amygdala. The bar plots and violin plots (with mean and strip plots) show the summary activation profiles for each condition within each ROI. The error bars in the bar plots represent plus or minus one standard error.

This is thought to contribute to impairments in top-down emotion regulation and fear extinction in people with PTSD[27,29,37,41]. Indeed, OFC is differentially recruited in people with PTSD relative to non-trauma-exposed individuals[31] and in people with diagnosed anxiety disorders[32]. Furthermore, the attenuation of OFC activation is consistently associated with symptom severity in PTSD[28].

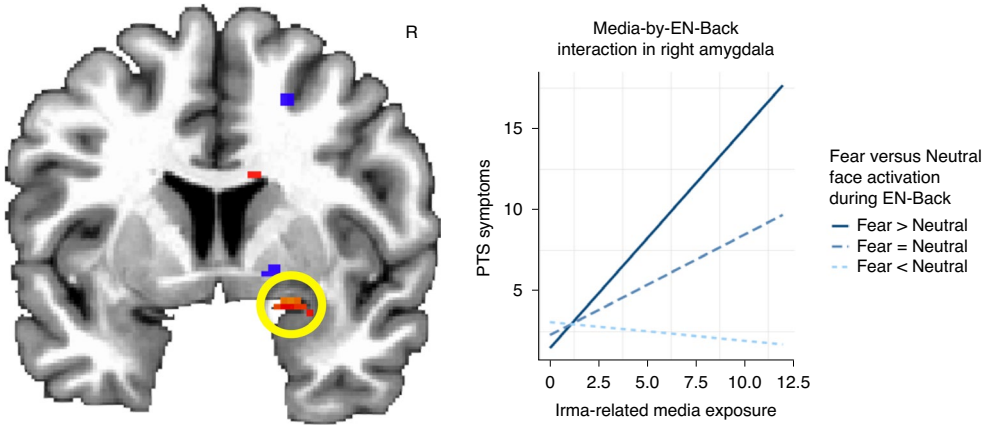

Fig. 4 | Prospective right amygdala reactivity moderates the relation between self-reported media exposure and PTS. Whole-brain analysis of the media-by-EN-Back interaction (left) reveals a significant interaction in right amygdala, denoted by the yellow circle ($P < 0.005$, corrected). The nature of the interaction in that cluster shows that the association between self-reported media exposure and PTS symptoms is the strongest for children who had heightened amygdala reactivity to Fear versus Neutral faces ($t(276) = 3.90$; $P = 0.0001$; $\beta = 0.27$; $r_{sp} = 0.21$; $B = 0.82$; 95% CI for B, 0.41 to 1.24). In the plot (right), the slope estimate is parsed at the mean (Fear = Neutral), one standard deviation below the mean (Fear < Neutral) and one standard deviation above the mean (Fear > Neutral).

The involvement of the parahippocampal gyrus is also a consistent finding in people with PTSD[27,42]. This region is more easily activated in response to traumatic imagery for people with PTSD than for non-trauma-exposed individuals[42], and like the OFC, its activity is positively associated with symptom severity[43]. Its role in this circuit is in contextual associative processing of autobiographical memories with high emotional valence[44], such as those related to threat or trauma[42]. Children who cannot emotionally regulate the response to anticipatory threat-related media might thus be at heightened risk of becoming overwhelmed by trauma-related memories.

There is an important caveat to speculation about mechanistic explanations: despite the prospective design, our data are inherently associative, and direct causal assertions are not warranted. We will thus only offer cautious speculation to inform future investigations. With that in mind, our data suggest that ineffective recruitment of downregulatory processes in response to fearful stimuli might confer a greater risk to increasing PTS from media exposure. Children who under-recruit OFC in response to fearful stimuli thus seem to be most at risk, possibly because this is associated with the degree of hyper-reactivity of amygdala. This circuit modulation is additionally reflected in parahippocampal gyrus, potentially contributing to the consolidation of traumatic memories, even when these arise from media exposure rather than from direct 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[45]. In children, previous research has shown that exposure to hurricane events alters neural reactivity (measured with electroencephalography) to negative stimuli in children who were tested before and after Hurricane Sandy[46,47]. 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 or dose. Indeed, negatively valenced arousal is known to increase attention to emotional stimuli and experiences[28]. Altered neural reactivity to negative emotional information may thus become exacerbated over development[48], or disaster exposure may confer particular vulnerability to later stressors in adulthood[46].

Findings from the present study indicate that specific pre-existing features of children's brain-based emotional reactivity may make them more or less susceptible to the negative influence of repeated exposure to disaster threat[48], even through media, elevating risk for the development of subsequent PTS. Trending effects were also seen for objective exposure. For example, although the objective-exposure-by-EN-Back interaction in right ACC did not survive correction for multiple comparisons, it is consistent with the fMRI study by Stevens and colleagues[35] showing that habituation in ACC (that is, a sharp decrease in ACC response to fearful stimuli) is associated with a slower course of recovery over the year after acute trauma. The more substantial effects for self-reported media exposure might reflect the fact that media exposure was widespread beyond the immediate disaster area, affecting all study sites. In contrast, as noted in the Results, the objective exposure dose effect was not evenly distributed—it was stronger for the South Florida youth than for the UF and MUSC sites. This may attenuate the sensitivity of the objective exposure measure in this particular study as it relates to predictive neural vulnerability effects.

When interpreting these results, it is also important to note that none of the children in the study showed a degree of PTS that reached the diagnostic threshold for PTSD. There was also no direct manipulation of exposure, and although face-valid self-report items are the standard strategy for assessing disaster-related media use (for example, see refs. [11,19]), the validity of this approach is somewhat limited[49]. In any case, the magnitudes of the effect sizes are modest (on the order of $r_{sp} = 0.12$ to 0.23 for effects of objective exposure, 0.21 for effects of self-reported media exposure and 0.13 to 0.17 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 or if they systematically accrue over time[50,51]. Small effect sizes are therefore meaningful when the degree of potential accumulation is substantial[52]. Our results point to effects of media exposure on future stress responses, regardless of proximity to the disaster event, and to a neural bias to processing threatening 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

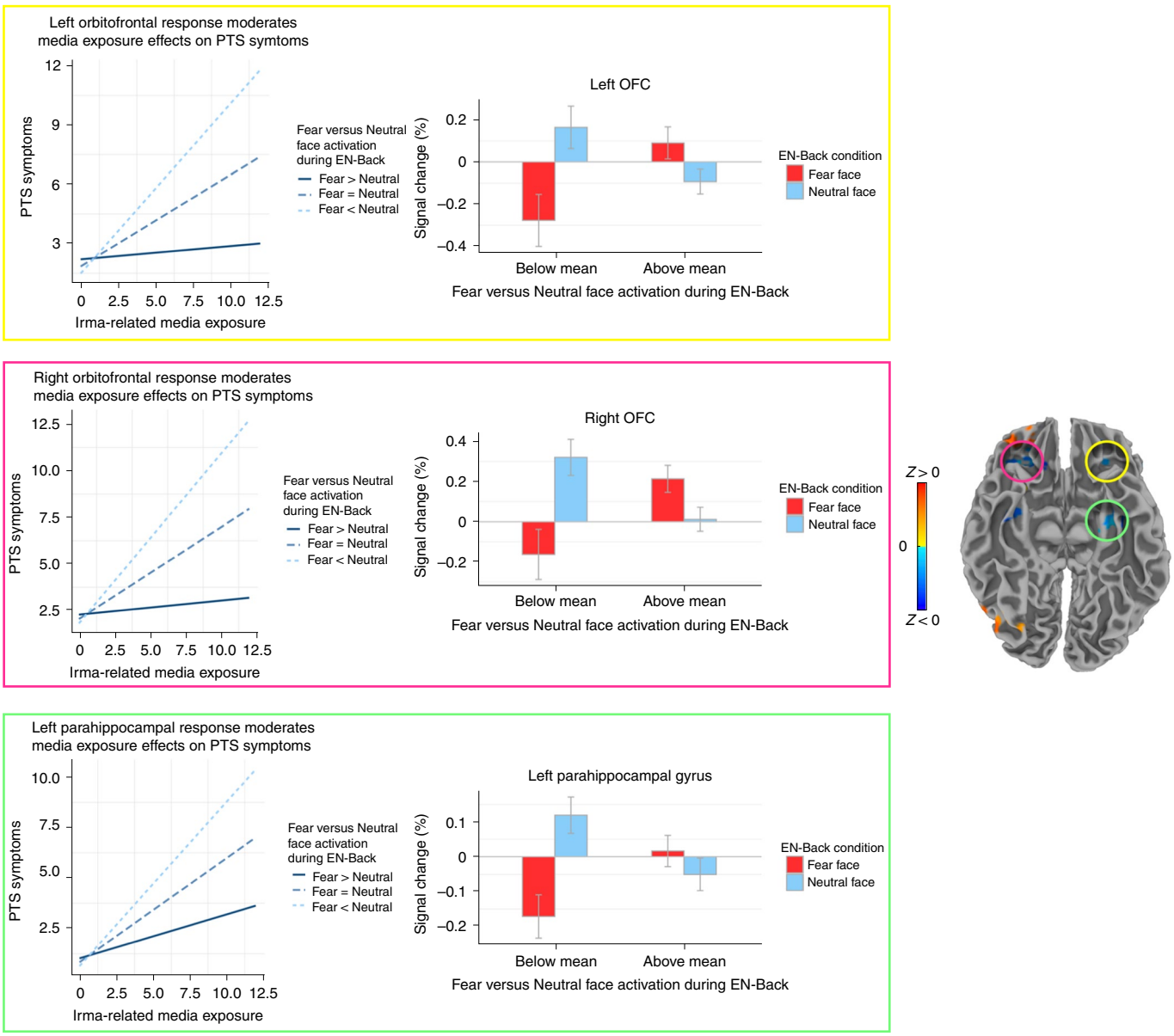

Fig. 5 | Prospective OFC and parahippocampal reactivity moderates the relation between self-reported media exposure and PTS. The plots on the left show slope estimates of the association between self-reported media exposure and PTS symptoms from the multiple regression controlling for eleven covariates: age, birth sex, race/ethnicity, highest degree of parental education, household income, parental marital status, Child Behavior Checklist (CBCL) Anxiety Problems, Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) Prior Trauma, degrees of freedom of the general linear model, performance on the EN-Back and 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), one standard deviation below the mean (Fear < Neutral) and one standard deviation above the mean (Fear > Neutral). In the plots on the right, 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. The error bars represent plus or minus one standard error.

traditional journalism; and rapidly advancing technologies that disrupt everyday experiences and 'push' news stories into our daily activities[53].

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 substantial. Thus, even when children do not reach the criteria for a disorder in the context of a single disaster, it is possible that 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[2,3]. 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. 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

Table 2 | Results of moderation analyses for each a priori defined ROI

Brain region	*B* (s.e.)	*β*	r_{sp}	*t*	*P*	Lower to upper 95% CI for *B*
Objective exposure by EN-Back (Fear versus Neutral) interaction						
Left hemisphere						
Amygdala	−0.21 (0.29)	0.02	−0.04	−0.73	0.463	−0.78 to 0.35
Hippocampus	−0.10 (0.41)	0.02	−0.01	−0.25	0.803	−0.92 to 0.71
OFC	−0.56 (0.28)	0.05	−0.16	−1.98	0.05	−1.11 to 0.01
Parahippocampal gyrus	−0.26 (0.28)	0.03	−0.06	−0.91	0.365	−0.81 to 0.30
ACC	−0.77 (0.39)	0.03	−0.11	−1.96	0.052	−1.53 to 0.00
Right hemisphere						
Amygdala	−0.56 (0.33)	0.02	−0.06	−1.71	0.088	−1.21 to 0.08
Hippocampus	−0.08 (0.52)	0.03	−0.04	−0.15	0.879	−1.11 to 0.95
OFC	0.03 (0.24)	0.04	−0.04	0.13	0.898	−0.45 to 0.51
Parahippocampal gyrus	0.06 (0.22)	0.03	0.01	0.25	0.799	−0.37 to 0.48
Anterior cingulate gyrus	−0.91 (0.37)	0.03	−0.14	−2.45	**0.015***	−1.64 to −0.18
Irma-related media by EN-Back (Fear versus Neutral) interaction						
Left hemisphere						
Amygdala	−0.05 (0.22)	−0.01	−0.05	−0.24	0.809	−0.49 to 0.38
Hippocampus	0.10 (0.15)	0.01	0.07	0.67	0.504	−0.19 to 0.39
OFC	−0.33 (0.10)	−0.05	−0.17	−3.16	**0.002**†**	−0.53 to −0.12
Parahippocampal gyrus	−0.44 (0.18)	−0.06	−0.15	−2.49	**0.013*†**	−0.78 to −0.09
ACC	0.02 (0.23)	0.00	−0.03	0.09	0.93	−0.42 to 0.46
Right hemisphere						
Amygdala	0.20 (0.13)	0.03	0.10	1.55	0.123	−0.05 to 0.45
Hippocampus	−0.11 (0.17)	−0.02	−0.05	−0.62	0.536	−0.44 to 0.23
OFC	−0.38 (0.10)	−0.05	−0.13	−3.84	**0.00015***†**	−0.57 to −0.18
Parahippocampal gyrus	−0.05 (0.09)	−0.01	−0.03	−0.55	0.583	−0.22 to 0.13
ACC	−0.41 (0.19)	−0.06	−0.17	−2.17	**0.031***	−0.79 to −0.04

Results of robust linear models for the interaction effect of exposure by EN-Back on PTS symptoms, for each ROI. The robust linear models controlled for the following covariates as fixed effects: age, birth sex, race/ethnicity, highest degree of parental education, household income, parental marital status, CBCL Anxiety Problems, K-SADS Prior Trauma, degrees of freedom of the general linear model, performance on the EN-Back and MRI scanner serial number. All *P* values are two-tailed. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$. *P* values marked with † indicate that these effects survived a false discovery rate (FDR) correction for multiple comparisons. For all objective exposure by brain regression models, d.f. = 188, $n = 211$. For all media by brain regression models, d.f. = 277, $n = 301$.

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.

Methods

Data analyses were conducted on the ABCD Fix Release 2.0.1. Comprehensive details about the ABCD Study and ethical considerations of the research are published elsewhere (*Developmental Cognitive Neuroscience* Special Issue 2018, vol. 32, pp. 1–164). Data from the substudy about Hurricane Irma were included in this curated annual release. The parent study and substudy were reviewed and approved by the UCSD Human Research Protections Program/Institutional Review Board. Informed consent was obtained from parents, and assent was obtained from children. In addition to compensation as part of the overall ABCD Study, those who participated in the Irma-focused substudy were compensated US$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. No statistical methods were used to pre-determine sample sizes, but our sample sizes are similar to or larger than those reported in previous publications[25,34,35].

Participants. The sample of participants comprised 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—FIU in Miami, Florida; UF in Gainesville, Florida; MUSC in Charleston, South Carolina; and UCSD in San Diego, California. The children and parents completed several measures as part of the original ABCD baseline visit and 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 (a large-scale survey of approximately 3.5 million households conducted annually by the US Census Bureau) and annual third- and fourth-grade school enrolment data maintained by the National Center for Education Statistics. The sampling strategy was additionally constrained by the requirement that study sites had available 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 American Community Survey demographic profiles[54], it is best described as a population-based, demographically diverse sample that is not necessarily representative of the US national population. Demographic assessments of the sample are summarized in Barch et al.[55]. The demographic profile of the present Irma substudy sample, separated by site, is presented in Supplementary Table 1.

Missing data. We focused on dealing with missing data for the demographic and covariate mental health measures, which were minimal to begin with (Supplementary Table 2). For the three missing demographic and covariate variables (highest household income, household marital status and K-SADS Pre-Hurricane trauma exposure), we proceeded to missing data imputation for demographic measures using the Multivariate Imputation via Chained Equations package in R (v.3.6)[56]. Missing data for other measures (for example, brain

measures and missing survey data) were dealt with using case-wise deletion and are 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 before Hurricane Irma. We also collected follow-up Hurricane Irma survey measures of direct hurricane exposure, anticipatory self-reported media exposure and self-reported 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 2018. A six- to eight-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 six months to develop PTS syndromes[6,57,58].

Pre-hurricane measures from the ABCD baseline visit. *Baseline anxiety.* Controlling for prior anxiety mitigates the possibility that media-related findings simply reflect the tendency of anxious youth to seek out more threat-related news. To control for pre-disaster anxiety, we used data from the CBCL[59] 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 subscales assessing anxiety, depression, somatic complaints, social problems, attention problems, rule-breaking behaviour 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[60] and is associated with PTS responses in disaster victims[61]. To control for pre-disaster exposure to trauma, we used the data from the Parent Diagnostic Interview for DSM-5 K-SADS, modified for ABCD[55]. 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, including learning about the unexpected death of a loved one, exposure to sexual or physical abuse, threats on the child's life, witnessing violence or mass destruction, involvement in a car accident or intensive medical treatment, or witnessing or being 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. The administration of the ABCD Emotional N-Back (EN-Back) is described in detail elsewhere[24]. 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[62], 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[63].

The task includes two runs of eight blocks each. On each trial, the participants are asked to respond as to whether the picture is a 'match' or 'no match'. The participants are told to make a response on every trial. In each run, four blocks are 2-back conditions, for which the 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 the 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 2.5 s cue indicates the task type ('2-back' or 'target=' and a photo of the target stimulus). A 500 ms coloured fixation precedes each block instruction to alert the child of a switch in the task condition. Each block consists of ten trials (2.5 s each) and four fixation blocks (15 s each). Each trial consists of a stimulus presented for 2 s, followed immediately by a 500 ms fixation cross. Of the ten trials in each block, two are targets, two or three are non-target lures and the remainder are non-lures (that is, stimuli presented only 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[64] and the racially diverse affective expression (RADIATE) stimulus set[65]. The place stimuli are drawn from previous visual perception studies[65].

Neuroimaging acquisition and analysis. The data are from the curated public release of the ABCD Study, which reports imaging activity profiles summarized in a priori defined ROIs. Data assessing the individual responses to Fear and Neutral EN-Back conditions were part of the 'fast-track' data release and are analysed at the whole-brain level. The acquisition parameters, image post-processing steps and selection of ROIs are described below.

Imaging parameters. Data were collected before the hurricane on 3T Siemens Prisma (FIU, MUSC and UF) and 3T GE 750 (UCSD) MRI scanners. These magnets employ the Harmonized Human Connectome Project Protocol optimized for ABCD[66]. This protocol uses state-of-the-art multiband imaging with prospective motion correction (PROMO/vNav) and EPI distortion correction. Real-time head motion monitoring (fMRI Integrated Real-time Motion Monitor[67]) was employed. The imaging data analysed as part of the present study include anatomical scans (used to define the ROIs) collected with a three-dimensional T1-weighted MPRAGE sequence with prospective motion correction (sagittal; $1\times1\times1\,mm^3$; matrix $=256\times256\,mm^2$) and fMRI scans collected with a three-dimensional T2*-weighted EPI sequence (axial; $2.4\times2.4\times2.4\,mm^3$; Field of View (FOV) $=216\times216\,mm^2$; Time Repetition/Time Echo (TR/TE) $=800/30$ ms; multiband acceleration, 6; 60 slices no gap).

Imaging analysis. 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 a priori defined ROIs associated with anxiety, emotion regulation and PTSD[20–22,68,69]. These ROIs, based on the Destrieux parcellation from Freesurfer[70], are (1) left and right amygdala, (2) left and right hippocampus, (3) left and right OFC (orbital H-shaped sulcus), (4) left and right parahippocampal gyrus (medial occipitotemporal parahippocampal gyrus), and (5) left and right ACC (anterior cingulate gyrus and sulcus). A second set of post-hoc ROIs was also examined: (1) left and right insula, (2) left and right inferior parietal cortex (supramarginal gyrus), (3) left and right mid-cingulate gyrus, and (4) left and right precuneus.

Notably, some children were fatigued by the length of the MRI scanner protocol, and due to this attrition, data on the EN-Back were available for only 74% of the sample. The results for analyses of the neuroimaging data are thus reported for this sample of children who completed the task, and the effective degrees of freedom after including covariates is d.f. $=279$ ($n=301$). The details on the post-processing steps are included in ref. [66], but briefly, the processing steps employed corrections for gradient nonlinearities 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 (that is, '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 versus the mean beta weight (activation over baseline) for the Neutral condition (that is, the difference score), collapsed across the 0-back and 2-back conditions. 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. In addition, quality control metrics are available in the public release. These detail (1) the degrees of freedom of the general linear model estimating the beta weights (activation for each conduction), which takes into account time points that did not meet the motion censoring threshold of framewise displacement (FD) >0.9 mm, and (2) whether children met the acceptable performance threshold of >60% on the EN-back task. Thirteen percent of children did not meet the 60% threshold. To control for performance and movement, both of these metrics were entered as covariates in all regressions that examined the brain data. For this sample, FD and the percentage of volumes exceeding the 0.9 FD threshold are as follows: FD, mean $=0.32$, s.d. $=0.32$; percent volumes exceeding 0.9 mm FD, mean $=0.19$, s.d. $=0.10$.

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. First, information about activity within each condition relative to resting baseline is 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. Second, 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 several comparisons: (1) Fear versus Neutral condition differences; (2) moderation of the association between objective exposure and PTS symptoms by the Fear versus Neutral activation difference (that is, the EN-Back-by-objective-exposure interaction); and (3) moderation of the association between self-reported media exposure and PTS symptoms by the Fear versus Neutral activation difference (that is, the EN-Back-by-self-reported-media-exposure interaction). The details of these comparisons are presented below. For each comparison, a per-voxel threshold of $P<0.005$ was applied. A family-wise error cluster correction ($P<0.05$) was applied by estimating the spatial smoothing from the residuals of the statistical model, iteratively generating a three-dimensional 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[71]).

Post-hurricane survey measures. From March to May 2018 (after 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 self-reported 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 (for example, UCSD participants did not answer certain questions related to direct exposure to the hurricane). In addition, the questions were translated into 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 HURTE-II, an updated iteration of the HURTE-R[72,73], which has been used extensively in hurricane research to assess hurricane exposure and post-disaster stressors. The HURTE-II assesses stressors before (for example, evacuation experiences), during (for example, perceived life threat, actual life threat and immediate loss or disruption) and after the storm (for example, ongoing stress, loss and disruption). A self-reported Irma-related media exposure questionnaire was also developed specifically for the context of Hurricane Irma[74].

For the present analysis, we focused on objective exposure and pre-storm self-reported media exposure. Objective exposure tallied the number of items that families endorsed reflecting direct Irma-related harm (for example, the child was hit by falling or flying objects during the hurricane), witnessing exposure (for example, the child saw someone badly hurt during the hurricane) or damage to property (for example, broken windows, flooding or water damage from the storm) during and after the hurricane. Data on an independent sample suggest that such exposures were substantial sources of stress for families involved in Hurricane Irma, both before and during the storm and surrounding evacuation[75]. 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 available sample size for this variable was thus $n = 324$.

For self-reported media exposure, we focused on pre-storm media exposure because 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 because 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. Thus, three child self-report items asked how often the child (1) viewed Irma-related television coverage before the storm (for example, news stations and weather channels), (2) checked for news and updates using the Internet (for example, news or National Oceanic and Atmospheric Administration websites), and (3) engaged in Irma-related social media activity (for example, Facebook, Twitter and Instagram). The items were rated on a scale of 0–4. The 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'). The anchors for the Internet and social media items included 0 ('Once per day or less'), 2 ('Almost every hour') and 4 ('Almost continuously'). Reliability analysis of the three before-hurricane items revealed good reliability: $\alpha = 0.76$; ω (hierarchical) $= 0.75$; ω (total) $= 0.77$. The ratings on the three items were thus 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 (refs. [76–78]). 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[6]. 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 (for example, 'When something reminds me of Hurricane Irma I get very upset, afraid or sad'). The responses were 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). The data for PTS Symptom Total and K-SADS Pre-Trauma exposure had very large outliers (data points more than seven standard deviations from the mean) and were Winsorized. The range, mean and standard deviation for these measures before and after Winsorization are as follows: for PTS Symptom Total before, range = (0, 78), mean = 4.88, s.d. = 8.22; and after, range = (0, 33.2), mean = 4.65, s.d. = 6.94; for K-SADS Pre-Trauma before, range = (0, 14), mean = 0.48, s.d. = 0.97; and after, range = (0, 2), mean = 0.42, s.d. = 0.63.

Robust multiple regression. Multiple regression was conducted using robust statistical and bootstrapping approaches. Specifically, we conducted robust regressions using a Huber loss function, which down-weights the influence of outliers but does not remove them. In cases where there are no outliers, robust regression provides similar or identical results to ordinary least-squares regression, but it performs better when there are outliers[79]. To conduct the bootstrap, we used a parametric bootstrap with 10,000 bootstrap replicates. The bootstrap standard errors were then used to define 95% CIs of the parameter estimates. The data distribution was assumed to be normal, but this was not formally tested. Instead, robust statistical models and bootstrapping were implemented to mitigate issues stemming from potential violations of these assumptions.

A small number of participants (21 families) had siblings in the substudy. Although modelling 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 modelled for the neuroimaging analysis to account for scanner differences.

In each regression, the following covariates were entered in the model as fixed effects: age, birth sex, race/ethnicity, highest degree of parental education, household income and parental marital status. 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; $t(378) = 1.58$; $P = 0.13$; $\beta = 0.05$; $r_{sp} = 0.05$; $B = 0.12$; 95% CI for B, −0.03 to 0.28), prior trauma exposure was associated with PTS symptoms (controlling for CBCL Anxiety and demographic covariates; $t(377) = 3.32$; $P = 0.0009$; $\beta = 0.10$; $r_{sp} = 0.18$; $B = 0.98$; 95% CI for B, 0.40 to 1.56). These two measures were also entered as covariates in all regressions.

For analyses investigating functional imaging predictors, the MRI scanner serial number was entered as a covariate to control for the use of four different scanners. Imaging quality control metrics are also available in the public release. These detail (1) the degrees of freedom of the general linear model estimating the beta weights (activation for each condition), which takes into account time points that did not meet the motion censoring threshold of FD > 0.9 mm, and (2) whether children met the acceptable performance threshold of >60% on the EN-back task. For the movement metric, the summary statistics were as follows: mean = 599.3; s.d. = 75.9; range, 196 to 656. For the performance metric, 13% of children did not meet the 60% threshold. To control for performance and movement, both of these metrics were entered as covariates in all regressions that examined the brain data. There was thus a total of 8 covariates for analyses of behavioural measures and 11 covariates for fMRI-related analyses.

Three main analyses were conducted. First, we established whether the brain measure (Fear versus Neutral EN-Back) was able to identify reliable differences in the expected regions (namely, amygdala). Second, we explored the association between objective Irma exposure and Irma-related PTS symptoms at the three sites that experienced the hurricane (MUSC, UF and FIU). We also examined the moderating effect of the activation difference between the Fear and the Neutral conditions of the EN-Back. Third, we explored the association between self-reported Irma-related media exposure and Irma-related PTS symptoms at all four sites. As in the second analysis, we also examined the moderating effect of the activation difference between the Fear and the Neutral conditions of the EN-Back. These brain-by-exposure analyses were conducted at both the whole-brain and ROI levels, for all a priori and post-hoc ROIs.

We first 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 and Neutral conditions in bilateral amygdala (Fig. 3; $P < 0.005$, corrected). The finding replicates a number of previous studies showing the amygdala's central role in processing fear-related stimuli[22], and the difference is in the expected direction (Fear > Neutral). Notably, this was the only significant effect at the whole-brain level that was evident in a priori defined ROIs (Fig. 3). 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 versus Neutral manipulation worked as expected, which establishes a framework on which to understand the results of our analyses of brain-by-exposure interactions.

For the second analysis, we examined the relation between objective Irma exposure and Irma-related PTS symptoms. We began with the South Florida youth sample most directly affected by Hurricane Irma (that is, the FIU site). There was a significant association between objective Irma exposure and PTS symptoms ($t(109) = 2.43$; $P = 0.017$; $\beta = 0.14$; $r_{sp} = 0.23$; $B = 0.43$; 95% CI for B, 0.08 to 0.78). When all sites affected by the hurricane were examined (that is, FIU, UF and MUSC), the effect was also significant ($t(255) = 2.21$; $P = 0.028$; $\beta = 0.09$; $r_{sp} = 0.12$; $B = 0.29$; 95% CI for B, 0.03 to 0.55; Fig. 1). The results were nearly identical when controlling for prior anxiety and trauma exposure ($t(107) = 2.49$; $P = 0.014$; $\beta = 0.14$; $r_{sp} = 0.23$; $B = 0.48$; 95% CI for B, 0.09 to .80 for the South Florida FIU site; $t(253) = 2.10$; $P = 0.037$; $\beta = 0.09$; $r_{sp} = 0.11$; $B = 0.28$; 95% CI for B, 0.02 to 0.54 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 third analysis, we examined the relation between self-reported media exposure before the hurricane and PTS symptoms, controlling for site (the Southern California/UCSD site versus Irma states—that is, FIU, UF and MUSC) and demographic covariates. The regression model revealed a significant association between self-reported media exposure and PTS symptoms

($t(377) = 4.84$; $P = 0.000002$; $\beta = 0.15$; $r_{sp} = 0.23$; $B = 0.41$; 95% CI for B, 0.24 to 0.57). The results were nearly identical when controlling for prior anxiety and trauma ($t(375) = 4.61$; $P = 0.00003$; $\beta = 0.15$; $r_{sp} = 0.21$; $B = 0.40$; 95% CI for B, 0.23 to 0.56). To determine whether those who experienced the direct effects of the hurricane were differentially influenced by self-reported media exposure, we added site as a moderator. The interaction between site and self-reported media exposure was not significant ($t(376) = -0.30$; $P = 0.72$; $\beta = -0.03$; $r_{sp} = -0.04$; $B = -0.09$; 95% CI for B, −0.64 to 0.47). This suggests that the effects of self-reported media exposure on PTS symptoms was uniform across youth in affected and non-affected regions (that is, children who were over 4,500 kilometres from the hurricane; Fig. 2).

Having established an association between objective exposure and Irma-related PTS symptoms, and an association between media exposure and Irma-related PTS symptoms, we entered into the regression models as a moderator the activation difference between the Fear and the Neutral conditions of the EN-Back. This analysis thus examines whether pre-existing neural vulnerability influences the strength of the relation between exposure and PTS symptoms. These analyses were conducted at both the whole-brain level and the ROI level, where ROIs were defined on the individual brain space of each subject. For all ROI analyses, the Benjamini–Hochberg FDR correction[80] was applied to control for a priori defined ROIs (ten comparisons).

For the whole-brain analysis of objective exposure, we found no statistically significant main effects of the EN-Back difference predicting PTS. We found only a few reliable objective-exposure-by-EN-Back interaction effects, which were evident in cerebellum, left angular gyrus, left cuneus and inferior occipital gyrus, left caudate nucleus, and right anterior superior temporal sulcus. However, none of these clusters were found in a priori defined ROIs or in post-hoc defined ROIs.

For the whole-brain analysis of self-reported media exposure (incorporating the UCSD site), we also found no statistically significant main effects of the EN-Back difference predicting PTS. We did find reliable media-exposure-by-EN-Back interaction effects, which were evident in bilateral orbital sulcus, parahippocampal gyrus, superior frontal gyrus, superior and middle occipital gyrus, right precentral gyrus, middle frontal sulcus, orbital gyrus, fusiform gyrus, and right amygdala. Three of these clusters were found in a priori defined ROIs (parahippocampal gyrus, OFC and amygdala), and we examined those further.

The nature of the finding in right amygdala is broadly consistent with effects reported in previous studies of exposure to disasters (Fig. 4)[25,34,35]. Here, inspection of the data in the cluster, in which the interaction slope is positive, indicates that the association between self-reported media exposure and PTS symptoms is the strongest for children who had heightened amygdala reactivity to Fear versus Neutral faces. In bilateral OFC and parahippocampal gyrus, the effect is in the opposite direction. The negative interaction slope reflects the fact that, in these regions, children who showed a weak or below-baseline response to the Fear face condition were especially susceptible to PTS as a result of self-reported media exposure (Fig. 5).

In our final analysis, we sought to determine the reliability, in individually a priori defined ROIs, of these whole-brain effects. As Table 2 shows, only three interaction effects (for the media-by-EN-Back interaction in bilateral OFC and left parahippocampal gyrus) had 95% CIs that did not cover zero and were statistically reliable after FDR correction. These regional effects were also found at the whole-brain level. The effect for right amygdala revealed at the whole-brain level for the media-by-EN-Back interaction was not observed at the ROI level ($P = 0.123$). This may be due to the fact that the significant cluster in the whole brain was confined to a circumscribed part of amygdala, and thus the effect may 'wash out' across the whole ROI. Regardless, caution in interpreting the strength of this effect is warranted.

Finally, Table 2 also shows that for one region there was a significant effect that was not observed at the whole-brain level—right anterior cingulate for both the media-by-EN-Back interaction and the objective-exposure-by-EN-Back interaction. However, this did not survive FDR correction in either case. Across both analyses, no other results were statistically reliable in either the a priori or post-hoc ROIs.

Power estimates and effect sizes. The effect sizes in this study, especially for the brain effects, were small. Thus, despite reasonable sample sizes (>300 for most analyses), the power to detect small effect sizes was low. For example, the power estimates for the brain measures predicting behaviour were low (power = 0.41 for effect sizes around $r = 0.10$ at $\alpha = 0.05$). There is thus the possibility of higher type II error for the brain effects. That said, the effect sizes for potentially missed effects were universally small (that is, approaching zero; Table 2). Analyses of the effects of Irma exposure and Irma media exposure had higher power, due to both larger sample size (that is, due to less missing data) and larger effects (for example, power = 0.99 for effect sizes around $r = 0.30$ at $\alpha = 0.05$). The power analyses were based on Cohen[81].

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

The ABCD data repository grows and changes over time. The ABCD data used in this report, including the substudy data collected outside of the baseline visits, came from RDS Fix Release 2.0.1 (https://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

All software used in the present analysis is open source. The R code (CRAN; v.3.6.0) to replicate the analysis is available at https://github.com/anthonystevendick/irmasubstudy_abcd.

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abcdstudy.org), held in the NIMH Data Archive. This is a multisite, longitudinal study designed to recruit more than 10,000 children ages 9–10 and follow them over 10 years into early adulthood. The ABCD Study is supported by the National Institutes of Health and additional federal partners under award numbers U01DA041048, U01DA050989, U01DA051016, U01DA041022, U01DA051018, U01DA051037, U01DA050987, U01DA041174, U01DA041106, U01DA041117, U01DA041028, U01DA041134, U01DA050988, U01DA051039, U01DA041156, U01DA041025, U01DA041120, U01DA051038, U01DA041148, U01DA041093, U01DA041089, U24DA041123 and U24DA041147 and National Science Foundation RAPID award number 1805645 to A.S.D. and J.S.C. A full list of supporters is available at https://abcdstudy.org/federal-partners.html. A list of participating sites and a complete list 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 the analysis or writing of this report. This manuscript reflects the views of the authors and may not reflect the opinions or views of the National Institutes of Health 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

All authors contributed to the conception of the study and/or the 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. K.S., R.G., M.T.S., A.R.L., W.K.T., S.F.T., L.M.S., K.M.G., S.J.N., L.B.C., A.M.L.G. and R.H.G. 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.

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Software and code

Policy information about availability of computer code

Data collection	RedCAP, NIH Toolbox, and Eprime software were used during data collection.
Data analysis	All software used in the present analysis is open source. The R code (CRAN; v. 3.6.0) to replicate the analysis is available at https://github.com/anthonystevendick/irmasubstudy_abcd.

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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).

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Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	We examined data from the Adolescent Brain and Cognitive Development study and measured survey responses about Hurricane Irma, as well as anxiety (from the CBCL), prior trauma (from the KSADS), and performance on an emotional n-back working memory task. It is a quantitative quasi-experimental design study.
Research sample	The sample is comprised of 454 9-11-year-old children and their families collected from 4 study sites in the Adolescent Brain Cognitive Development (ABCD) Study across the United States.
Sampling strategy	Demographically, the ABCD Study used a multi-stage probability sample of eligible children by probability sampling of schools within the catchment area of each site. The goal of this sampling strategy was to approximate 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 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. Despite this caveat, the ABCD Study sample was largely successful at matching the ACS survey demographic profiles. Demographic assessments of the ABCD sample are summarized here in Barch et al. The demographic profile of the substudy sample is presented in Extended Data Table 1 in the paper.
Data collection	Researchers were blind to the study hypotheses of the present paper. During data collection, the research assistant, the parent, and the children in the study were present (for baseline visits) or completed a survey online.
Timing	Data collection began in the Fall of 2016 (for Baseline visits). Sub-study data collection occurred in Spring 2018.
Data exclusions	Missing data analysis is described in the paper and was applied to demographic variables that were missing. All data that were available were analyzed.
Non-participation	Some participants declined participation, but this depended on the task/questionnaire. The number of participants who completed the questionnaire is available in Table 1. The sample size for each analysis is detailed in the Method.
Randomization	Random assignment was not used. Demographic and cognitive covariates were entered into the generalized linear model analyses. These covariates were age, sex, race/ethnicity, highest degree of education, household income, marital status, and (for the MRI analysis) MRI scanner model number.

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Methods

n/a	Involved in the study
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☒	☐ Flow cytometry
☐	☒ MRI-based neuroimaging

Human research participants

Policy information about studies involving human research participants

Population characteristics	See Above.
Recruitment	The sample is comprised of 454 9-11-year-old children and parents collected from 4 study sites across the United States. Demographically, the ABCD Study used a multi-stage probability 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 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. Despite this caveat, the ABCD Study sample was largely successful at approximating the ACS survey demographic profiles. That said, although it closely approximates the demographic profile of the ACS survey, because the sampling strategy heavily relied on schools in urban areas, it is more accurate to describe the sample as having a population-based, demographically diverse sample that is not necessarily representative of the U.S. national population.
Ethics oversight	University of California at San Diego Institutional Review Board

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Magnetic resonance imaging

Experimental design

Design type	The EN-Back task was conducted and analyzed as a block design.
Design specifications	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 2.5 s 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 (2.5 s each) and 4 fixation blocks (15 s each). Each trial consists of a stimulus presented for 2 s, followed immediately by a 500 ms 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).
Behavioral performance measures	Behavioral performance was not analyzed in this study.

Acquisition

Imaging type(s)	Functional and Structural images were collected.
Field strength	3 Tesla
Sequence & imaging parameters	Data were collected on 3T Siemens Prisma (FIU, MUSC, UF sites) and 3T GE 750 (UCSD site) MRI scanners. These magnets employ the Harmonized Human Connectome Project Protocol optimized for ABCD. 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) 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).
Area of acquisition	Whole-brain acquisition was used.

Diffusion MRI ☐ Used ☒ Not used

Preprocessing

Preprocessing software	AFNI and Freesurfer were used to process the data.
Normalization	For the region-of-interest analysis, data were not normalized. For the whole-brain analysis, data were normalized to the MNI template.
Normalization template	As noted, data were normalized to the MNI template.
Noise and artifact removal	The processing steps employed corrections for gradiant non-linearities and resampling to isotropic voxel resolution, and

Noise and artifact removal	additional motion correction and B0 distortion correction steps for the fMRI. Movement and drift terms were included in the General Linear Model.
Volume censoring	Volume censoring was not applied.

Statistical modeling & inference

Model type and settings	Mass univariate analysis was employed at the first level. Fixed-effects analysis was conducted at the second level.
Effect(s) tested	The main comparisons were 1) Fear Face and Neutral Face versus baseline; 2) Fear vs Neutral Face; 3) Fear vs. Neutral Face by Objective exposure predicting PTS outcomes. 4) Fear vs. Neutral Face by Media exposure predicting PTS outcomes.

Specify type of analysis: ☐ Whole brain ☐ ROI-based ☒ Both

Anatomical location(s)	ROIs, based on the Destrieux parcellation from Freesurfer, 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).
Statistic type for inference (See Eklund et al. 2016)	For each comparison, a per-voxel threshold of $p < .005$ 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).
Correction	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).

Models & analysis

n/a	Involved in the study
☒	☐ Functional and/or effective connectivity
☒	☐ Graph analysis
☒	☐ Multivariate modeling or predictive analysis