



Exposure to violence reduces empathetic responses to other's pain

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ARTICLE INFO

Article history:

Accepted 13 April 2013

Keywords:

Exposure to violence

Empathy for pain

AI

aMCC

ABSTRACT

Past researches showed that empathy for pain not only triggers a resonance mechanism between other and self, but also is modulated by contextual factors. Using functional magnetic resonance imaging, the present study demonstrated that short-term media violence exposure reduced both pain ratings and also the activation of anterior insula and anterior mid-cingulate cortex to other's pain. Thus, violence exposure modulated empathic responses to other's pain based on a physiological desensitization.

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1. Introduction

Central to successful social interaction is the ability to understand other's emotion, pain and sensation. This capacity to represent and share the emotional and affective states of another person in relation to oneself is referred as empathy (Decety & Jackson, 2004; Guo et al., 2012; Hein & Singer, 2008; Lieberman, 2007). Studying the perception of pain in others provides an avenue for investigating the mechanism of human empathy.

In light of the analysis from social neuroscience, it has been proposed that perception of others' pain not only triggers a resonance mechanism between other and self (Cheng et al., 2007; Preston & de Waal, 2002), but also is modulated by contextual factors. In recent years, some fMRI studies have demonstrated that perception of others' pain activates similar regions of the pain matrix observed in the first-hand experience of pain (Derbyshire, 2000; Jackson, Brunet, Meltzoff, & Decety, 2006b), including both the areas for encoding the motivational-affective dimension of pain, such as bilateral anterior insula (AI), anterior cingulate cortex (ACC), and the anterior mid-cingulate cortex (aMCC) (e.g., Akitsuki & Decety, 2009; Botvinick et al., 2005; Danziger, Failenot, & Peyron, 2009; Gu & Han, 2007; Guo et al., 2012; Jackson, Brunet, Meltzoff, & Decety, 2006a; Jackson, Meltzoff, & Decety, 2005; Jackson, Rainville, & Decety, 2006b; Lamm, Batson, & Decety, 2007a; Lamm, Nusbaum, Meltzoff, & Decety, 2007b; Moriguchi et al., 2007; Morrison &

Downing, 2007; Morrison, Lloyd, di Pellegrino, & Roberts, 2004; Morrison, Peelen, & Downing, 2007; Saarela et al., 2007; Singer et al., 2004), and the areas for encoding the sensory dimension of pain, such as the somatosensory cortex (e.g., Akitsuki & Decety, 2009; Avenanti, Buetti, Galati, & Aglioti, 2005; Bufalari, Aprile, Avenanti, Di Russo, & Aglioti, 2007; Lamm et al., 2007b; Moriguchi et al., 2007; Valeriani et al., 2008). On the other hand, other studies revealed the modulation of contextual factors on empathy for other's pain, such as the affective link between individuals (Singer et al., 2006), the intentionality of the perceived agency who induced the pain (Akitsuki & Decety, 2009; Decety et al., 2008), the racial membership of the pain targets compared to the observer (Xu, Zuo, Wang, & Han, 2009), prior attitudes toward the pain targets based on their stigmatized status (Decety, Echols, & Correll, 2010), the facial expression of the pain targets (Han et al., 2009), and the monetary reward to the pain targets (Guo et al., 2012). The present study aimed at elucidating the effect of another contextual factor, i.e., exposure to violent video, on observers' empathic responses to other's pain. Direct empirical evidence for the role of such contextual factor in empathic perception and response to others allows key insights into the nature of the empathy system.

In considering how empathic responses to others' pain might be modulated by contextual factors, exposure to violent video may be important. Recent studies have demonstrated that exposure to violence and blood in the media lead to a reduced distressing emotional responses towards violence such as fear and anxiety—a process called desensitization (Bushman & Anderson, 2010; Carnagey,

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Anderson, & Bushman, 2007; Fanti, Vanman, Henrich, & Avraamides, 2009; Linz, Donnerstein, & Penrod, 1988; Mullin & Linz, 1995). One negative consequence of such physiological desensitization is that it may cause people to be numb to the pain and suffering of others, i.e. reduced empathy for other's pain (Anderson et al., 2010; Bartholow et al., 2005). A negative correlation was consistently found between long-term media violence exposure and empathy (Bartholow, Sestir, & Davis, 2005; Funk, Baldacci, Passold, & Baumgardner, 2004; Funk, Buchman, Jenks, & Bechtoldt, 2003; Krahé & Möller, 2010).

However, little is known about the effects of short-term media violence exposure. Will it, as long-term violence exposure, reduce the empathic responses to others' pain? Previous studies have already demonstrated that short-term media violence exposure could induce desensitization. Carnagey et al. (2007) measured heart rate and skin conductance of the violent and nonviolent group who played a violent or nonviolent video game for 20 min before game play, during game play, and after game play but while observing video of real life violence. They found participants' heart rate and skin conductance changed after playing video game. Also, Bushman and Anderson (2010) used the same procedure as Carnagey et al. (2007), except that after playing the assigned game, participants heard a fight that was staged outside the lab room door, while filling out a questionnaire. As a result, those who played the violent game showed less helping, were less likely to hear the fight, and rated it as being less severe. As a consequence of desensitization, we predicted that participants' empathic responses to others' pain might also be modulated by short-term media violence exposure.

The present study aims to investigate the cognitive and neural mechanism underlying the modulation of empathic responses to others' pain by short-term media violence exposure. Bearing in mind the effects of long-term media violence exposure, it is predicted that short-term media violence exposure can also lead to less empathy for other's pain and a reduced neural empathic response in pain-related areas accordingly. To test these hypotheses, forty participants were first exposed to 5-min violent or non-violent video clips and then were scanned using functional magnetic resonance imaging (fMRI) while viewing others in pain (Fig. 1A). We were interested in participant's responses to other's pain after violence exposure.

2. Methods

2.1. Participants

A total of forty right-handed participants (30 female, aged from 19 to 30, $M = 22.15$, $SD = 2.67$) were enrolled in this experiment and randomly assigned to two groups: violent group, in which the participants ($n = 20$, 15 female) were exposed to a violent video clip before viewing the pictures; non-violent group, in which the participants ($n = 20$, 15 female) were exposed to the non-violent video clip before viewing the pictures. All the participants were recruited from the university community and paid for their participation. None of them had a history of neurological or psychiatric disorders, alcohol or drug abuse. All the participants had normal or corrected-to-normal vision and were given informed consent before scanning. The entire study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Shanghai Psychological Society.

2.2. Materials

2.2.1. Video clips

We made up two independent 5-min video clips with several segments from two movies (The Ruins and Nim's Island) used by

Bushman and Anderson (2010). The violent video clip was composed of two segments (00:59:46–01:02:22; 01:11:30–01:13:54) from The Ruins (2008). The non-violent video clip was composed of one segment (00:00:00–00:05:00) from Nim's Island (2008). In a pilot study, 20 participants who were not adopted in the formal experiment were recruited to rate how violent the video clips were on a 9-point Likert-type scale ranging from 1 (non-violent) to 9 (extremely violent). Half of them viewed the violent video clip and the other half watched the non-violent video clip. Violence ratings were higher for the violent video (7.00 ± 2.00) than for the non-violent video (1.20 ± 0.42 , $t(9.798) = 8.97$, $p < .001$).

2.2.2. Pictures

The picture stimuli consisted of ninety-six pictures showing left index finger and right ear in painful and non-painful situations (48 each, used in our previous study, see Guo et al., 2012). Painful situations depicted four kinds of nociceptive stimulations: cut the finger or ear with a knife or a pair of scissors, and prick the finger or ear with a needle or an awl; Paired with each of eight painful situation, a non-painful situation in which the above-mentioned tools did not touch the finger or ear, but laid aside of body part, was also obtained. Six pictures were obtained for each situation (totally $8 * 2 * 6 = 96$) and all pictures were $300 * 400$ pixels in size.

2.3. Procedure

The participants were firstly scanned to acquire high-resolution structural images. Then, they were asked to watch the 5-min video clip attentively in the scanner. For each group, the corresponding video clip was presented. Immediately after that, participants were scanned while viewing painful or non-painful pictures. Ninety-six pictures were randomly displayed on a gray background, interspersed with null events. Each trial was presented for 3.5 s with jittered inter-stimulus intervals (ISI) from 0 to 1 s, during which a black fixation cross was presented against the gray background. During null events, the fixation cross remained on screen. The participants were instructed to watch the pictures attentively and try to experience the feelings of the owners of the body parts in the pictures.

After scanning, the participants were presented with the same stimuli as inside the scanner and were asked to rate how painful they thought the individuals in the pictures were experiencing by a 9-point Likert-type scale from no pain to extreme pain outside the scanner, where 1 indicated no pain, while 9 indicated extreme pain. They also filled out the emotional contagion scale (ECS) to assess the susceptibility to other's emotions (Doherty, 1997).

2.4. fMRI Image acquisition and analysis

Scanning was performed on a 3T Siemens Trio system (East China Normal University, Shanghai) with a standard head coil to obtain functional images using a gradient echo echo-planar imaging (EPI) sequence. Thirty-five transversal slices covering the whole brain were acquired sequentially with a 0.3 mm gap ($TR = 2200$ ms, $TE = 30$ ms, $FOV = 220$ mm, flip angle = 90° , matrix size = $64 * 64$, slice thickness = 3 mm, gap = 0.3 mm). There was one run of functional scanning which was about 9 min (245 EPI volumes). Before the functional run, a high-resolution structural image was acquired using a T1-weighted, multiplanar reconstruction sequence (MPR) ($TR = 1900$ ms, $TE = 3.42$ ms, 192 slices, slice thickness = 1 mm, $FOV = 256$ mm, flip angle = 9° , matrix size = $256 * 256$).

Data preprocessing was carried out with SPM5 (Statistical Parametric Mapping, Wellcome Department of Imaging Neuroscience, London, UK) implemented in MATLAB. The first five volumes were discarded to allow for T1 equilibration effects. During preprocessing, images were first realigned to the first volume to correct for

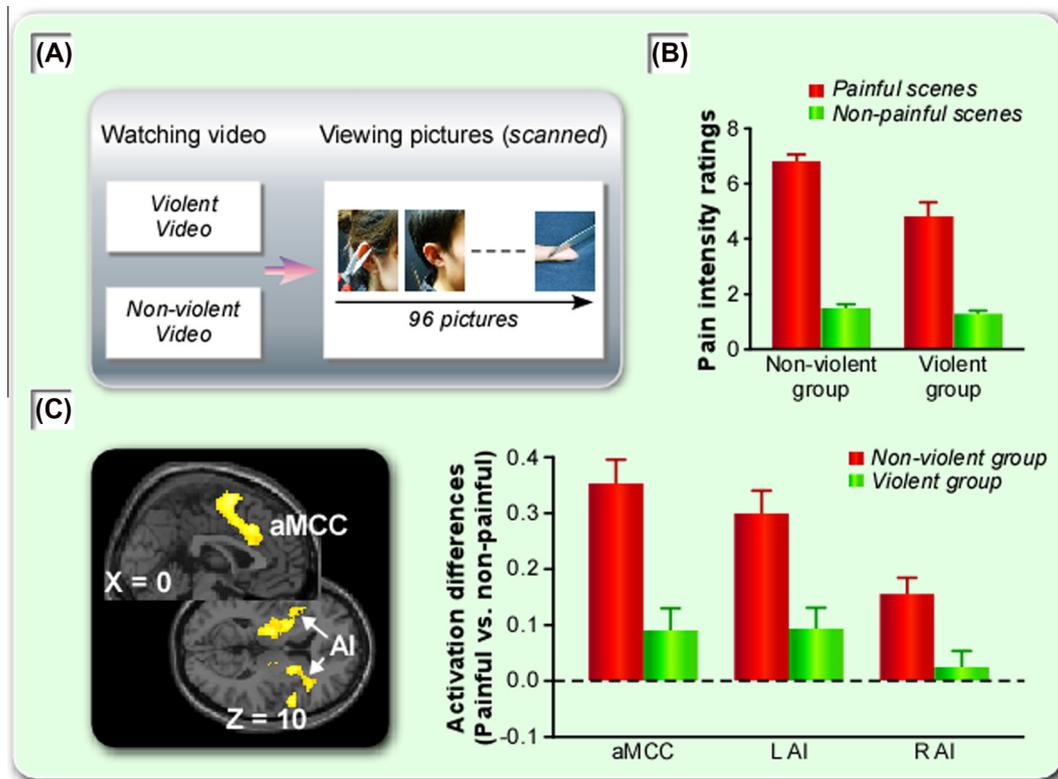


Fig. 1. Experimental procedure and results. (A) Experimental procedure. Participants were randomly assigned to watch either a violent or a non-violent video clip before scanning. All the participants were then scanned while viewing 96 painful or non-painful pictures. (B) Participants' self-ratings of pain intensity for painful and non-painful scenes. (C) aMCC and bilateral AI (aMCC: $-6\ 10\ 38$, L AI: $-34\ 6\ 2$, R AI: $36\ 20\ 14$) defined in the [(PN – NN)–(PV – NV)] contrast. L = left hemisphere; R = right hemisphere. The activation differences between painful and non-painful scenes in aMCC and bilateral AI were significantly smaller for the violent versus the non-violent group, indicating reduced empathic responses to other's pain after violent exposure.

inter-scan head movements, and then the mean EPI image of each subject was computed and spatially normalized to the MNI single subject template. The normalizing parameters were applied to the functional images, which were re-sampled to $2 \times 2 \times 2$ mm voxel size. The data were then smoothed with a Gaussian kernel of 8 mm full-width half-maximum to accommodate inter-subject anatomical variability.

We then analyzed neural responses to painful and non-painful scenes for violent group (PV and NV) and non-violent group (PN and NN). Statistical analyses were performed using the general linear model (GLM) implemented in SPM5. An event-related design was used at the first level analysis with two types of events (PV and NV for violent group, PN and NN for non-violent group). Events were convolved with a canonical hemodynamic response function (HRF) and its time derivatives. All the encoding trials were modeled as 3.5 s long events from the onset time of the picture. The models additionally included six movement parameters derived from realignment as covariates of no interest. High pass temporal filtering with a cutoff of 180 s was also applied in the models. For each subject at the first-level analysis, simple main effects for two types of events were computed by applying the '1 0' contrasts. The four first-level individual contrast images (PV, NV, PN and NN) were then analyzed at the second group level.

Brain activation related to perception of other's pain was defined using (PV + PN)–(NV + NN) contrast ($p < .001$, uncorrected, $k > 50$). Further, the (PN – NN)–(PV – NV) contrasts were tested to extract specific regions showing reduced empathic responses to other's pain for the violent compared with the non-violent group ($p < .001$, uncorrected, $k > 50$). The revised contrasts were tested to extract specific regions showing increased empathic responses to other's pain for violent compared with the non-violent group

($p < .001$, uncorrected, $k > 50$). Finally, the correlation analysis was performed to extract regions showing positive correlation between brain BOLD signal changes when viewing painful versus non-painful scenes and corresponding behavioral painful rating differences ($p < .005$, uncorrected, $k > 50$).

3. Results

3.1. Behavioral data

The ECS scores of two groups did not differ from each other ($t(38) = 0.14$, $p > .05$; violent group: 17.30 ± 4.40 ; non-violent group: 17.10 ± 4.83). And, the mean ratings of pain intensity for PV, NV, PN and NN were 4.85 ± 2.10 , 1.29 ± 0.50 , 6.84 ± 0.91 and 1.50 ± 0.60 (Fig. 1B). A 2 pain (painful vs. non-painful) \times 2 group (violent vs. non-violent) ANOVA revealed a significant main effect of pain ($F(1,38) = 334.41$, $p < .001$), a significant main effect of group ($F(1,38) = 14.01$, $p < .005$) and a significant interaction ($F(1,38) = 13.47$, $p < .005$). This result indicated that the difference of pain intensity ratings between painful and non-painful scenes for the non-violent group was significantly higher than that for the violent group.

3.2. fMRI results

Similar to previous studies (Akitsuki & Decety, 2009; Guo et al., 2012; Lamm et al., 2007a,b; Singer et al., 2004), experiencing the pain of others [(PV + PN)–(NV + NN)] not only activated regions implicated in encoding the emotional dimension of pain, such as AI and aMCC, but also regions for sensory encoding of pain, such as the postcentral gyrus (PCG, Table 1). Correlation analyses also

revealed positive linear correlation between painful rating differences (painful – non-painful) and the degree of activation in the aMCC and left (L) AI (aMCC: $r = 0.51$, LAI: $r = 0.45$, $ps < .01$) in the (PV – NV for violent group and PN – NN for non-violent group) contrast across participants (Table 2).

Further, the regions showing reduced empathic responses to other's pain for the violent compared with the non-violent group [(PN – NN)–(PV – NV)] were also the bilateral AI, aMCC and Supplementary motor area (SMA) (Fig. 1C and Table 3). To further test how exposure to violence affected brain responses to others' pain, specific activations identified in [(PN – NN)–(PV – NV)] contrast were used to compute regions of interest (ROIs). ROIs were defined as 6-mm spherical regions centered on the peak coordinate in the activated clusters and their parameter estimates were extracted to calculate pain-related signal changes for further statistics using the MarsBaR toolbox in SPM5. The activation differences between painful and non-painful scenes in these regions were significantly smaller for the violent as compared to the non-violent group (aMCC: $x = -6$, $y = 10$, $z = 38$; left AI: $x = -34$, $y = 6$, $z = 2$; right AI: $x = 36$, $y = 20$, $z = 14$; SMA: $x = 6$, $y = 2$, $z = 66$, $ts > 3.21$; $ps < .01$), which was in support of our hypothesis for reduced empathic neural responses after violence exposure. The reverse contrast revealed no significant activation in the pain matrix. Besides, the activation differences between painful and non-painful scenes in SMA were significantly correlated with those in other three regions ($rs > 0.60$; $ps = .000$).

4. Discussion

This study sought to investigate how violence exposure modulates empathic responses. At the behavioral level, the intensity of pain was modulated by violence exposure. That is, compared to participants who viewed non-violent video, participants who viewed violent video rated other's pain to be less painful. At the neural level, the activations of AI and aMCC were also modulated by violence exposure. Reduced activation in regions encoding emotional dimension of other's pain was observed after participants were exposed to a violent video. Furthermore, empathy-related activation was associated with painful ratings such that those individuals who gave higher painful ratings had higher AI and aMCC activation differences when viewing painful vs. non-painful scenes. These important findings highlight that even short-term violence exposure reduces empathy for pain and generally support the desensitization hypothesis.

Although the theory of desensitization suggests the relations between media violence exposure and empathy (Anderson et al., 2010), empirical studies only focused on the effects of long-term media violence exposure on empathy (Bartholow et al., 2005; Funk

Table 2

Regions showing positive correlation between self-rating score differences of pain intensity (painful – non-painful) and differential activations (painful – non-painful).

Region of activation	Lat.	Coordinates			T-score	k
		x	y	z		
Putamen	R	24	2	14	4.60	734
Supplementary motor area	L	-8	8	48	4.52	1121
Anterior Cingulate Cortex	R	6	22	24	4.36	
Middle Cingulate Cortex	R	8	20	30	3.91	
Rolandic Operculum	R	54	2	12	3.96	141
Putamen	L	-22	10	8	3.88	441
Insula	L	-42	10	-12	3.29	
Inferior Frontal Gyrus	R	52	26	-4	3.60	64

Note: coordinates (mm) are in MNI space. L = left hemisphere; R = right hemisphere. $p < .005$, uncorrected, $k > 50$.

Table 3

Regions showing reduced empathic responses to other's pain for violent compared with non-violent group.

Region of activation	Lat.	Coordinates			T-score	k
		x	y	z		
(PN – NN)–(PV – NV)						
Supplementary motor area	R	6	2	66	5.68	1791
Middle Cingulate Cortex	L	-6	10	38	5.22	
Anterior Cingulate Cortex	L	-8	30	24	3.95	
Middle Temporal Gyrus	L	-44	-2	-16	5.21	1386
Insula	L	-34	6	2	4.19	
Superior Temporal Gyrus	L	-54	-34	22	5.10	158
Thalamus	R	16	-10	0	4.96	147
Putamen	R	24	4	12	4.94	369
Insula	R	36	20	14	4.68	
Rolandic Operculum	R	54	4	14	4.53	266
Amygdala	R	20	0	-14	4.42	82
Precuneus	L	-6	-66	52	4.03	192
Superior Frontal Gyrus	L	-18	6	54	3.85	62
(PV – NV)–(PN – NN)						
Inferior Temporal Gyrus	L	-54	-8	-32	4.79	121
Middle Temporal Gyrus	R	56	-54	16	3.76	101

Note: coordinates (mm) are in MNI space. L = left hemisphere; R = right hemisphere. $p < .001$, uncorrected, $k > 50$.

et al., 2003; Funk et al., 2004; Krahé & Möller, 2010) and the modulations of short-term media violence exposure on other desensitization effects (Bushman & Anderson, 2010; Carnagey et al., 2007). The present findings extend previous studies by showing the modulation of empathic responses to others' pain by short-term media violence exposure.

It should be noted that desensitization not only includes emotional numb, but also is indicated by reduced physical arousal, such as decreased heart rate and galvanic skin response (Arriaga, Esteves, Carneiro, & Monteiro, 2006; Ballard, Hamby, Panee, & Nivens, 2006; Carnagey et al., 2007). However, our results revealed that only activations in AI and aMCC, but not PCG were in response to violence exposure, which might reflect that the effects of 5-min exposure to media violence limit to emotional dimension. We guess that such desensitization effects might extend to the sensory components of empathy after longer violence exposure. Furthermore, such 5-min exposure to media violence might just have short-lived effects on empathy for pain.

Acknowledgments

This research was supported by National Natural Science Foundation of China (31271090, 31100728 & 90924013), Projects Planning in Shanghai Philosophy and Social Sciences Research (2012JJY001), Innovation Program of Shanghai Municipal

Table 1

Activations associated with experiencing other's pain.

Region of activation	Lat.	Coordinates			T-score	k
		x	y	z		
Inferior Frontal Gyrus	L	-54	8	18	13.3	28,255
Insula	R	34	20	6	9.31	
Middle Cingulate Cortex	L	-6	10	40	8.97	
Insula	L	-34	20	6	8.27	
Anterior Cingulate Cortex	R	4	20	24	7.05	
SupraMarginal Gyrus	L	-58	-26	40	11.76	4277
Postcentral Gyrus	L	-36	-40	66	3.60	
SupraMarginal Gyrus	R	68	-22	32	10.82	2167
Inferior Occipital Gyrus	R	30	-92	-4	6.86	1304
Middle Occipital Gyrus	L	-30	-96	-8	6.16	2072

Note: coordinates (mm) are in MNI space. L = left hemisphere; R = right hemisphere; M = medial activation. $p < .001$, uncorrected, $k > 50$.

Education Commission (12ZS046), 985 Project of Fudan University (2011SHKXZD008).

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