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Secure attachment partners attenuate neural responses to social exclusion: An fMRI investigation

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ABSTRACT

Research has shown that social exclusion has devastating psychological, physiological, and behavioral consequences. However, little is known about possible ways to shield individuals from the detrimental effects of social exclusion. The present study, in which participants were excluded during a ball-tossing game, examined whether (reminders of) secure attachment relationships could attenuate neurophysiological pain- and stress-related responses to social exclusion. Social exclusion was associated with activation in brain areas implicated in the regulation and experience of social distress, including areas in the lateral and medial prefrontal cortex, ventral anterior cingulate cortex, and hypothalamus. However, less activation in these areas was found to the extent that participants felt more securely attached to their attachment figure. Moreover, the psychological presence (i.e., salience) of an attachment figure attenuated hypothalamus activation during episodes of social exclusion, thereby providing insight into the neural mechanisms by which attachment relationships may help in coping with social stress.

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1. Introduction

Social rejection can have devastating consequences, including depression and social anxiety (Williams, 2001). Indeed, social rejection hurts. Although often regarded as a metaphor, recent evidence suggests that the “pain” experienced upon social rejection or exclusion may actually be associated with the same neural mechanisms underlying physical pain experience (Eisenberger et al., 2003; MacDonald and Leary, 2005). Such findings are in line with the idea that because both physical pain and social pain are cues that signal situations that may threaten survival, physical pain and social pain rely on a shared system that helps detecting and preventing such situations.

If social exclusion and rejection have such profound effects, and lead to the actual experience of pain, a theoretically interesting – and societally important – question is whether the opposite might also be true (Panksepp, 2003). That is, can supportive and loving relationships reduce the literal sting of pain by rejection? Can supportive relationship partners help in coping with the social pain and stress experienced upon social exclusion? Based on principles of attachment theory, the central goal of the present study was to examine whether reminders of secure attachment relationships can diminish the activation of brain areas related to pain and stress during exclusion.

In his early accounts of attachment theory, Bowlby (1982) sought to understand, from an evolutionary perspective, how and why infants become emotionally attached to their primary caregivers. When infants experience distress (from hunger, noise, pain, illness, or other causes) the attachment system is activated, causing them to seek protection and comfort from the primary caregiver, which increases survival chances. The distress-reducing or “safe have” function of attachment has been widely established in infant and child research (Ainsworth et al., 1978). It is supposed that also adults rely on attachment figures – often a romantic partner, close friend, or family member – when coping with distressing or threatening situations (Hazan and Shaver, 1987). Indeed, various lines of research have demonstrated that in anxiety-provoking situations, adults seek support from an attachment figure (Collins and Feeney, 2004; Simpson et al., 1992). Furthermore, the psychological or actual presence of an attachment figure has been shown to diminish the harmful consequences of stressful situations, as indicated both by self-reports and physiological responses (Feeney and Kirkpatrick, 1996).

Can secure attachment bonds also serve as a buffer against the distress caused by social exclusion? There is some suggestive evidence relevant to this proposition. For example, a recent fMRI study by Eisenberger and colleagues revealed that individual differences in perceived social support – a central function of attachment bonds – were negatively correlated with stress-related brain activity (e.g., dorsal anterior cingulate and hypothalamus) during a social exclusion episode (Eisenberger et al., 2007). However, because this study was correlational, it was not possible to determine the direction of

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causality (for example, it is plausible that people that have weaker neural responses to social exclusion also perceive more social support in their daily lives). Other evidence relevant to our hypothesis comes from a recent experimental fMRI study, in which women were subjected to the threat of receiving an electric shock, while their hand was held either by their spouse (i.e., an attachment figure) or by a stranger (Coan et al., 2006). In the spouse hand-holding condition, participants displayed diminished activation in threat-related brain areas such as anterior insula, superior frontal gyrus, and hypothalamus.

Although the latter finding suggests that attachment bonds can alleviate the distress experienced upon (anticipated) physical pain, it is not yet clear whether attachment bonds can diminish neural activity related to social distress resulting from social exclusion. Such findings would provide further support for the idea that shared neural substrates are involved in both physical and social pain, and would provide insight into the neural mechanisms by which attachment may buffer stress-responses. To address these issues, we conducted a study that used a procedure similar to Eisenberger and colleagues (Eisenberger et al., 2003; Eisenberger et al., 2007). In these studies, participants played a ball tossing game with two alleged participants on a computer, ostensibly via an internet connection. At some point, participants did not receive the ball anymore and were thus excluded from the game. While being excluded, participants typically exhibit blood-flow changes in brain areas related to the experience and regulation of pain and distress. Although initial social exclusion research mainly focused on the dorsal anterior cingulate cortex (dACC), insula, and right ventral lateral prefrontal cortex (RVLPFC; Eisenberger et al., 2003), more recent studies found that other stress-related regions such as the hypothalamus and regions in the lateral and medial prefrontal cortex (in particular, BA8) were involved in responses to social exclusion as well (Eisenberger et al., 2007). In addition, other studies – using the same ball-tossing exclusion paradigm – have found activity in the ventral part of the anterior cingulate cortex (vACC; Burklund, Eisenberger, & Lieberman, 2007; Masten et al., 2009). This latter finding is consistent with results from other studies indicating that the ventral areas of the anterior cingulate cortex are associated with affective responses to social rejection (Bush et al., 2000; Somerville et al., 2006).

The major purpose of the present research was to experimentally examine whether attachment bonds can attenuate neural activity related to pain and distress upon social exclusion. We examined whether this proposed attenuation effect could be obtained by the mere psychological (rather than physical) presence of the attachment figure, as previous research has suggested that the mere exposure of an attachment figure's name is sufficient to activate the attachment system (Mikulincer and Shaver, 2001). Therefore, while being socially excluded during the cyberball game, participants were either exposed to the name of their most important attachment figure, or to the name of a non-attachment control figure. By doing so, we examined whether regions activated upon social exclusion are attenuated by the mere psychological presence of an attachment figure (i.e., the *attachment buffer hypothesis*).

2. Method

2.1. Participants

Fifteen (10 females, 5 males) participants mean 22 years, range 19–33 years) were paid 15 Euros for participation.

2.2. Stimuli and task

Participants were invited to the lab to participate in a neuro-imaging study on 'visual imagination.' They were told they were going to play a virtual ball-tossing game ('Cyberball') with two other people

while their brain activation would be scanned. Before the game, participants were asked to provide the name of two persons – an attachment figure and a non-attachment control figure. The *attachment figure* was defined as “the person that first comes to mind that you would go to when you need help, for instance in times when things are not going very well, when you are experiencing any kind of problem, or when you simply do not feel very well.” The *non-attachment figure* was defined as “a person that you would not go to when you need help in such situations – however, it is not that you dislike this person, in fact you think of this person as a nice person, but you simply would not seek out help from him or her.” This operationalization aimed to distinguish between feelings of mere liking versus feelings of attachment, including the providing of a “safe haven” to rely on when in need for support or help (Bowlby, 1982; Collins and Feeney, 2004). Next, participants played Cyberball while in the scanner. Cyberball is ostensibly played via a network, with three players – two preprogrammed stooges and the participant – throwing a ball at each other on the computer screen. The participant can throw the ball at one of the two players by pressing one of two keys on a button box.

Participants played six rounds of Cyberball, three rounds in which the attachment figure's name was made salient, and three rounds in which the non-attachment figure's name was made salient. Each round consisted of 60 throws. To make it realistic, the “other” players waited for 0.5–1.5 seconds before making a throw. In the first round, the *control condition*, instructions stated that “on the screen, you will see the two other players throw a ball at each other. However, you are not connected, and will thus not play along in this round. In the lower right corner of the screen, the name [name of the attachment figure as provided by the participant, e.g., Barbara] will appear. Please imagine that Barbara stands a little behind you, while you are watching the two other players throwing the ball at each other.” In the second round, the *inclusion condition*, participants were told they were now connected and would play the game with the two other players. Again, in the bottom right corner the name of the attachment figure appeared, and the participant was instructed to imagine that this person was standing a little behind him or her during the game. In the third round, the *exclusion condition*, participants were given the same instructions as in the second round. However, after about 12 throws, the participant did not get the ball anymore from the two other players, and was thus excluded from the game.

The three (control, inclusion, exclusion) conditions were repeated, except that now the non-attachment figure's name appeared on the screen, and participants were instructed to imagine that this person was standing a little behind them while playing. Which name appeared in the first three rounds was counterbalanced between participants.

After participants left the fMRI scanner, they completed a questionnaire assessing feelings of attachment to the two persons named (3 items for both the attachment and non-attachment figure, e.g. “This person can comfort me when I feel tensed”, $\alpha = .72$ for the attachment figure; $\alpha = .66$ for the non-attachment figure). Participants also indicated on three items how much distress they experienced when they did not receive the ball anymore. They completed this measure twice, both regarding when the attachment person was “present,” and when the non-attachment person was “present” (e.g., “When Barbara was “present” at the game, it felt unpleasant when I did not receive the ball anymore,” “When Barbara was “present” at the game, I felt invisible,” and “When Barbara was “present” at the game, I felt distressed”; $\alpha = .77$ for the attachment condition; $\alpha = .93$ for the non-attachment condition). We averaged the three items as an indicator of experienced distress, separately for the attachment and the non-attachment condition. It is important to note that, as we did not want to raise suspicion about the goals of our study by giving the distress measure while in the scanner and during the game, completing the measurement of distress after the experiment has other limitations,

Table 1
Regions of Interest, pairwise differences between exclusion conditions, and interaction effects with attachment.

Region	BA	x	y	z	Volume (ml)	Control vs. Inclusion	Control vs. Exclusion	Inclusion vs. Exclusion	Condition x Attachment
<i>Cortical regions</i>									
Medial frontal gyrus (M)	6	−6	−8	71	0.299	ns	**	**	ns
Medial frontal gyrus (M)	6/8	−6	21	54	3.639	ns	**	*	ns
Medial frontal gyrus (M)	9	−3	50	31	5.774	ns	**	**	ns
Medial frontal gyrus (M)	10	−6	50	3	0.879	ns	**	**	ns
Anterior Cingulate (M)	24/33	2	29	1	0.632	ns	**	**	ns
Precuneus (M)	7/31	−5	−58	35	0.602	ns	*	**	ns
Middle frontal gyrus (L)	6	−38	5	42	1.643	ns	**	*	ns
Inferior frontal gyrus (L)	47	−48	30	−4	3.553	ns	**	**	ns
Superior temporal gyrus (L)	39	−45	−58	26	6.426	ns	**	**	ns
Middle temporal gyrus (L)	21	−47	−32	−2	1.522	ns	**	ns	ns
Superior frontal gyrus (R)	6	13	7	65	0.206	ns	*	*	ns
Superior frontal gyrus (R)	9	14	44	26	0.242	ns	**	ns	ns
Middle frontal gyrus (R)	9/44	37	9	34	0.453	ns	*	ns	ns
Superior temporal gyrus (R)	22	53	−7	−8	0.266	ns	**	**	ns
Superior temporal gyrus (R)	38	50	15	−20	1.105	ns	**	**	ns
Supramarginal gyrus (R)	40	52	−53	19	1.638	ns	**	*	ns
Lingual gyrus (R)	18	13	−76	5	0.271	ns	**	†	ns
<i>Subcortical regions</i>									
Hypothalamus		−1	−1	−12	0.177	ns	ns	ns	**

** $p < .01$, * $p < .05$, † $p < .10$. BA = Brodmann area.

such as recall bias (e.g., “it was not so bad after all when I was excluded”). Hence, from the outset, we should note that correlations of the distress measures with brain activation should be interpreted with care. We return to this issue in the Discussion.

2.3. MRI procedures and analyses

Brain imaging was performed on a 1.5 T Siemens Sonata MRI scanner (Siemens Medical Systems, Erlangen, Germany). An echo-planar imaging (EPI) sequence was used for functional imaging. EPI volumes consisted of 36 near-axial slices covering the whole brain. Scanning parameters were: repetition time: 3 s, echo time: 53 ms, flip angle: 90°, slice thickness: 2.8 mm, slice gap: 0.56 mm, acquisition matrix: 64×64 voxels, in-plane resolution: 3×3 mm. Series of 52 volumes were acquired in each of the six runs; volumes were online motion-corrected. After the functional runs, a structural scan using a T1-weighted MP-RAGE sequence was acquired for each participant. Scanning parameters were: repetition time: 2.73 s, inversion time: 1 s, echo time: 3.43 ms, flip angle: 7°, sagittal slice thickness: 1 mm, acquisition matrix: 224×256 voxels, in-plane resolution: 1×1 mm.

Preprocessing and statistical analyses of the MRI data were performed using BrainVoyager software (Brain Innovation, Maastricht, Netherlands). The first 2 volumes of each run were discarded in order to avoid differences in T1 saturation. Voxel time-series of the remaining volumes were high-pass filtered (0.01 Hz), temporally smoothed (4 s FWHM Gaussian kernel), corrected for slice acquisition times and 3D spatially smoothed (6 mm FWHM Gaussian kernel). Each functional run was coregistered to the individual structural scan, transformed into Talairach space and resampled (3×3×3 mm). For each run, a GLM design matrix was created in order to predict the relevant brain responses to each condition. The responses were predicted in the middle of the run (volume 18–34), coinciding with the moment of exclusion in the exclusion condition. Predictors were convolved with a standard hemodynamic response function. Constant term predictors (signal level confounds) were added to account for baseline differences between runs, and voxel time-series were z-transformed in order to stabilize their variance.

The statistical analyses were performed in two steps. The first step was to identify all brain regions that responded in any way to social exclusion. We used a voxel-wise whole-brain random-effects two-way GLM, with the factors Condition and Attachment. The factor

Condition contrasted the exclusion (third) with the control (first) condition. These conditions differ only in terms of experienced social exclusion and not on other factors such as planning or motor activity (i.e., participants were watching and not actively playing in both conditions). The factor Attachment contrasted the attachment with the non-attachment name. The purpose of this first analysis was to define regions of interest (ROIs) that would respond in any way to social exclusion. We were particularly interested in regions responding to exclusion independent of attachment (i.e., the main effect of exclusion), as well as regions responding to exclusion dependent on attachment (i.e., the interaction exclusion×attachment). The statistical threshold for these whole-brain analyses was $p=0.05$ after correction for multiple comparisons (minimal cluster size 0.14 ml). They revealed activation in a number of brain regions that are given in Table 1 and Fig. 1. More specifically, activation was found in areas in the lateral and medial prefrontal cortex, ventral ACC, lateral and anterior temporal lobe, and hypothalamus.

The second step involved testing our hypotheses more specifically within these regions of interest. That is, we pooled the voxels in each ROI and examined in post-hoc random-effects analyses, for each ROI, 1) the contrasts between the control, inclusion, and exclusion conditions, 2) whether there were interaction effects between Condition (control, inclusion, exclusion) and Attachment (attachment versus non-attachment), and 3) whether the effects occurred irrespective of order. Finally, we analyzed correlations between brain activity, self-reported distress, and self-reported feelings of secure attachment (regarding both the attachment and non-attachment figure).

3. Results

3.1. Feelings of secure attachment

Participants reported stronger feelings of secure attachment for the attachment figure, $M=6.56$, $SD=0.39$, than for the non-attachment figure, $M=3.49$, $SD=1.13$, $t(12)=11.18$, $p<.0001$.¹

¹ Due to technical failure, data of the self-report attachment and distress measures were not saved for two participants.

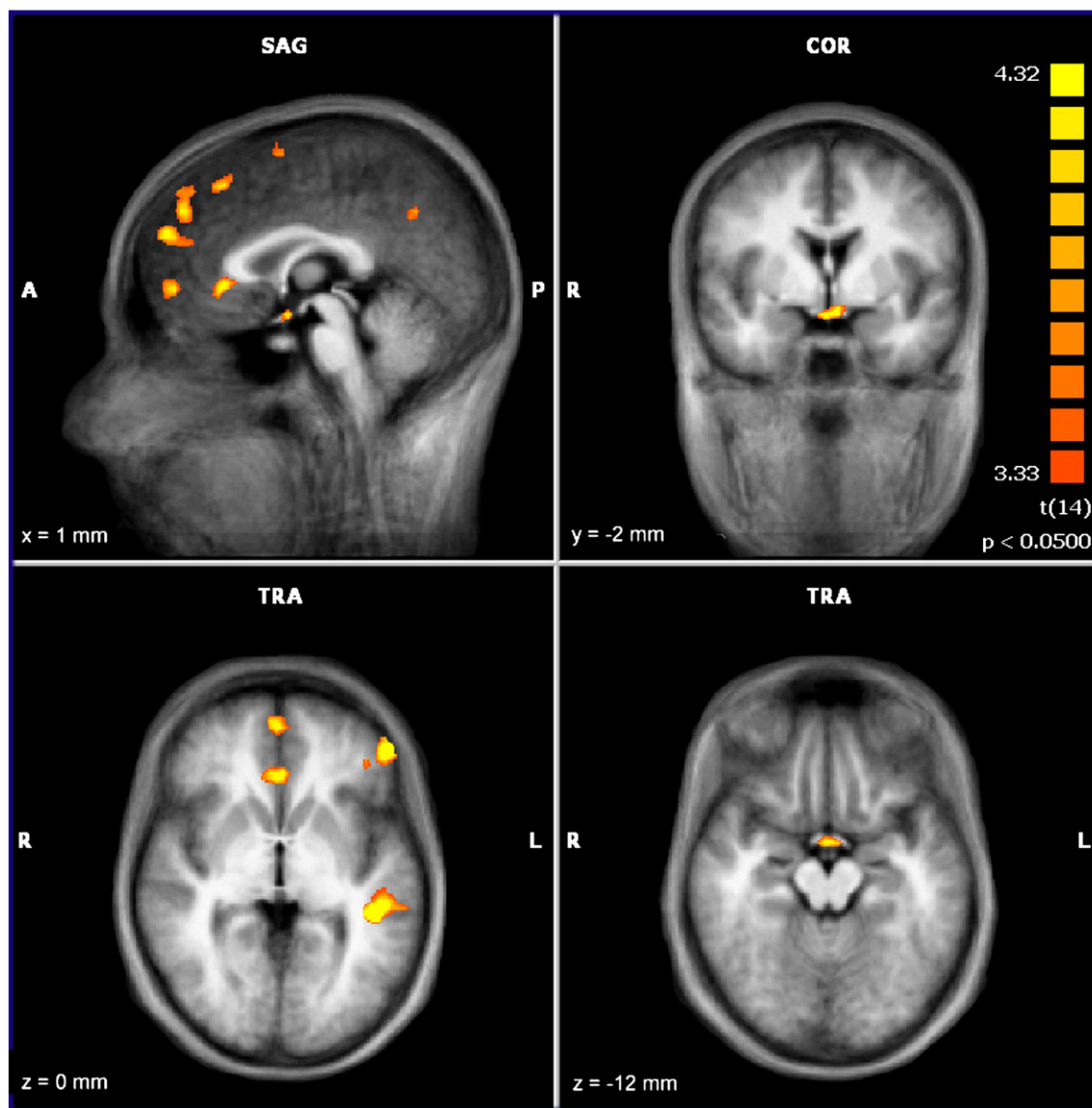


Fig. 1. Cortical and subcortical brain regions affected by social exclusion, superimposed on the averaged anatomical scan ($n = 15$) in Talairach space. Views at $x = 1$ mm, $y = -2$ mm, and $z = 0$ and -12 mm, respectively (bottom row). Threshold is $p = 0.05$ after correction for multiple comparisons. For further details, see text.

3.2. Self-reported distress

Confirming the attachment-buffer hypothesis, participants reported less distress upon exclusion when the attachment figure was present, $M = 3.23$, $SD = 1.42$, than when the non-attachment figure was present, $M = 4.54$, $SD = 1.70$, $F(1, 12) = 8.69$, $p < .02$, $\eta^2 = .44$. Importantly, there was no order effect, $F(1, 12) < 1$, indicating that participants did not report different levels of distress the second time they were excluded. Also, the order of presenting the attachment and non-attachment figures did not interact with presence of the attachment versus non-attachment figure on reported distress, $F(1, 12) < 1$. Finally, although not significant, correlations between secure attachment and self-reported distress were in the expected direction (and would likely reach significance with a larger sample); for the attachment figure $r = -.17$, $p = .29$; for the non-attachment figure $r = -.30$, $p = .15$.

3.3. Neural responses to social exclusion

In Table 1 the ROIs are given, with Brodmann area (BA), Talairach xyz-coordinates and cluster size. For each ROI, we conducted post-hoc

ANOVAs with Condition (control, inclusion, exclusion) and Attachment (attachment, non-attachment) as within-participant variables, and Order (attachment first, non-attachment first) as between-participants variable. Except for the hypothalamus and right middle gyrus (BA9/44), these analyses revealed significant main effects of Condition, all $F_s(2, 14) \geq 4.61$, p 's $\leq .05$, $\eta_p^2 \geq 0.44$. The main effect of Condition for left BA9/44 was marginal, $F(2, 14) = 3.61$, $p = .059$. Except for one marginal effect of order for right middle temporal gyrus, $F(2, 14) = 3.04$, $p = .086$, condition did not interact with order for all other areas, all $F_s \leq 1.56$, ns , indicating that effects of exclusion did not significantly differ between the first and second time participants were excluded.

To examine the main effects of condition more closely, pairwise comparisons were examined. The results are given in Table 1. There were no significant differences between the control and the inclusion condition in any of these areas. However, activation in all areas was significantly or marginally significantly stronger in the exclusion condition than in the control condition (except hypothalamus). Except for right BA9, right BA9/44, left BA21, and hypothalamus, all areas also revealed significantly stronger activation in the exclusion condition than in the inclusion condition.

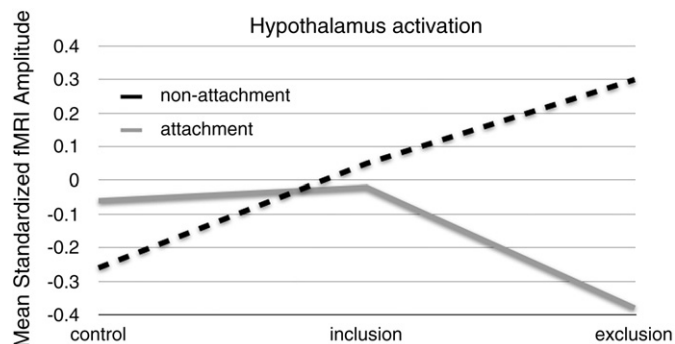


Fig. 2. Interaction between Condition × Attachment for the hypothalamus activation.

In support of the attachment buffer hypothesis, the analysis revealed a significant interaction between Condition and Attachment in the hypothalamus, $F(2, 14) = 11.53, p < .01, \eta_p^2 = 0.66$ (see Fig. 2; we did not find this interaction in other regions). Pairwise comparisons revealed no significant effect of Attachment in the control condition, $t(14) = 0.79, ns$, or in the inclusion condition, $t(14) = -.52, ns$. However, in the exclusion condition there was a significantly stronger activation of the hypothalamus when the non-attachment figure was present compared to when the attachment figure was present, $t(14) = -5.54, p < .001$ (see Fig. 2). Furthermore, when the non-attachment figure was present, hypothalamus activation was significantly stronger in the exclusion condition as compared to the control and marginally stronger as compared to the inclusion condition, respectively $t(14) = -3.78, p < .01$, and $t(14) = -1.70, p = .10$. In contrast, when the attachment figure was salient, hypothalamus activation was marginally weaker in the exclusion condition compared to the control condition, $t(14) = 1.90, p < .09$, and significantly weaker compared to the inclusion condition, $t(14) = 2.95, p < .01$. Thus, providing support for the attachment buffer hypothesis, whereas exclusion was related to increased hypothalamus activity when the non-attachment figure was present, exclusion was related to attenuated hypothalamus activation when the attachment figure was present.

Table 2
Correlations between self-reported distress and self-reported attachment security and brain activation in the exclusion condition.

Region	BA	r with self-reported distress ^a		r with self-reported attachment security ^b	
		NonAtt	Att	NonAtt	Att
<i>Cortical regions</i>					
Medial frontal gyrus (M)	6	.44†	-.51*	-.29	-.22
Medial frontal gyrus (M)	6/8	.60*	.33	-.29	-.56*
Medial frontal gyrus (M)	9	.44†	.36	.07	-.56*
Medial frontal gyrus (M)	10	.37	.55*	.09	-.13
Anterior Cingulate (M)	24/33	.27	.10	-.08	.11
Precuneus (M)	7/31	.46*	.03	-.16	-.55*
Middle frontal gyrus (L)	6	.43†	.21	-.10	-.55*
Inferior frontal gyrus (L)	47	.47*	.10	-.37	-.72**
Superior temporal gyrus (L)	39	.23	.24	-.03	-.65**
Middle temporal gyrus (L)	21	.46*	.21	-.29	-.43†
Superior frontal gyrus (R)	6	.23	-.31	-.07	-.42†
Superior frontal gyrus (R)	9	.47*	.06	-.28	-.03
Middle frontal gyrus (R)	9/44	.46†	.07	.14	-.27
Superior temporal gyrus (R)	22	-.07	-.24	.15	-.21
Superior temporal gyrus (R)	38	-.07	-.10	.17	-.70**
Supramarginal gyrus (R)	40	-.07	.10	.05	-.29
Lingual gyrus (R)	18	.46*	-.22	.10	-.55*
<i>Subcortical regions</i>					
Hypothalamus		.31	.12	-.38	.29

** $p < .01$, * $p < .05$, † $p < .10$. NonAtt = Non-attachment figure, Att = Attachment figure.

^a Correlations represent the association between distress participants reported regarding the non-attachment/exclusion condition and the attachment/exclusion condition and brain activation in these conditions respectively. ^b Correlations represent the association between self-reported attachment security regarding the non-attachment figure and the attachment figure, and brain activation in the non-attachment/exclusion and attachment/exclusion condition respectively.

3.4. Self-reported distress and neural responses to social exclusion

Correlations were calculated between the amount of distress experienced and brain activation in the exclusion condition relative to control condition 1) when the attachment figure was present, and 2) when the non-attachment figure was present (see Table 2). These analyses revealed significant or marginally significant correlations between distress and activation in many regions of interest (BA9, 9/44, 18, 7/31, 6/8, 6, 21). Although not all correlations reached significance, in general the correlations were positive, with the exception of left superior frontal gyrus (BA6). Specifically, stronger activation of left BA6 was related to less experienced distress when the attachment figure was present.

3.5. Feelings of attachment and neural responses to social exclusion

Feelings of secure attachment toward the attachment figure correlated negatively with brain activity in the exclusion condition (relative to control) for most regions of interest (see Table 2), except right medial frontal, left medial ventral frontal, and ACC. Interestingly, feelings of secure attachment toward the non-attachment figure were not significantly correlated with brain activation in any of these regions.

4. Discussion

Rooted in an attachment-theoretical framework, the main goal of the present research was to examine whether the psychological presence of an attachment figure could modulate neural responses to social exclusion. Before discussing the major findings relating to this central research question, we briefly summarize the results regarding social exclusion.

In the present study, social exclusion was associated with activation in areas in the lateral and medial PFC (including BA8 and BA47, which corresponds with the previous studies by Eisenberger); ventral ACC; and lateral and anterior temporal lobe (Fig. 1). As noted in the introduction, several areas, including lateral and medial PFC and vACC have been found in previous research on social exclusion (e.g., Burkund, Eisenberger, & Lieberman, 2007; Eisenberger et al., 2003;

Masten et al., 2009). Both the lateral and medial PFC have been implicated in the cognitive regulation of negative emotional responses (Ochsner and Gross, 2005), while vACC have been implicated in the affective processing of social rejection (Bush et al., 2000; Sommerville et al., 2006). Moreover, social exclusion was also associated with activation of the hypothalamus (Eisenberger et al., 2007), in particular when the attachment figure was absent. As part of the HPA-axis, the hypothalamus is an important regulator of immune functions, and repeated activity or hyperactivity of the hypothalamus is often found in major depression, susceptibility to infectious diseases, and cardiovascular problems (Dickerson and Kemeny, 2004; Kemeny, 2003).

The present study did not find activation in dACC and insula, as found in previous work (e.g., Eisenberger et al., 2003; but see Masten et al., 2009, who also did not find dACC activation). As the main goal of our study was to compare the effects of social exclusion with an attachment versus non-attachment figure available, in both exclusion conditions 'someone else' (i.e., either the attachment or non-attachment figure) was present during exclusion. In previous research there never was an imagined person watching the ball-tossing game while being excluded. Although we did replicate some of the previous exclusion effects despite this difference, at the same time this difference may have accounted for the fact that we did not replicate all previous findings.

Thus, we did replicate previous evidence that social exclusion activated regions in the brain that have been implicated in the regulation of emotions (MPFC), affective processing of pain and other negative experiences (vACC), and stress (hypothalamus). The critical question is: Are these neural effects modulated by the – even just psychological – presence of an attachment figure? In support of the attachment buffer hypothesis, the psychological presence of an attachment figure attenuated hypothalamus activation upon social exclusion. Specifically, whereas there was a significant increase in hypothalamus activation upon social exclusion when the non-attachment figure was psychologically present, in the attachment condition exclusion did not increase hypothalamus activation – if anything, hypothalamus activation slightly decreased. (It should be noted, however, that self-reported attachment did not significantly correlate with hypothalamus activation). As responses of the HPA axis have been regarded as the quintessential response to stress (Kemeny, 2003), the attenuation of the hypothalamus activation in the present study strongly implies that attachment relationships indeed buffer stress-related brain responses after social exclusion. The self-report measures of experienced distress corroborated these neurophysiological findings, as participants reported lower levels of distress upon social exclusion when the attachment figure was psychologically present than when the non-attachment figure was present.

These findings are in line with Coan et al. (2006). In their study, women who experienced a threat of receiving an electric shock, displayed diminished hypothalamus activation when their spouse was holding their hands (compared to a stranger hand-holding and no hand-holding). This attenuation was even stronger when the relationship quality was higher. Moreover, the present findings show strong similarities to results from animal studies. For example, experimentally induced stressors cause increased cortisol levels in Siberian hamsters, but only in socially isolated animals and not in socially housed ones (Detillion et al., 2004). In addition, maternal touching reduces HPA stress responses in rats (Wang et al., 1996). The present results are in line with such findings, and may imply that the beneficial effects of attachment relationships in buffering social stress may at least partly be driven by hypothalamus attenuation (Diamond, 2001).

The salience of an attachment figure did not attenuate activity in other brain regions. At first glance, these findings seem not to support the attachment buffer hypothesis. However, for many of these areas, feelings of secure attachment towards the attachment figure were negatively associated with brain activity induced by social exclusion.

Specifically, during social exclusion, activity in frontal areas that have been implicated in emotion- and stress-regulation was less pronounced to the extent that participants felt more secure toward the attachment figure. Do these findings support the attachment buffer hypothesis? On the one hand, it could be argued that the attachment buffer hypothesis actually would predict positive correlations with emotion-regulation areas, as the attachment figure would help participants to effectively deal with social threat by facilitating emotion regulation. However, in contrast to this reasoning, our findings may suggest that feelings of secure attachment buffer an individual against the immediate negative effects of social exclusion, which in turn requires less emotion regulation (and thus less activation in these areas to the extent that an individual feels more secure to the attachment figure). Notably, the fact that feelings of secure attachment toward the non-attachment figure were not significantly associated with regulation-areas may suggest that feelings of attachment toward someone have to exceed a certain threshold for that person to function as an attachment figure.

Before closing, we should acknowledge some limitations of the present research. First, the self-reported distress measure included the names of the attachment- and non-attachment figures (i.e., "When Barbara was present, ...etc"), and was assessed only in the end of the experiment, not immediately following the exclusion experience. As participants were excluded twice we did not want to assess distress during the experiment as it may have raised suspicion. However, the limitation of our procedure is that several processes may have contaminated the distress measure (e.g., recall bias, or participants' answers may have been biased by the names of the attachment/non-attachment figures). As we already noted, correlations with the brain imaging results should be interpreted with care. Second, future research should examine the generalizability of our findings. Although the present findings are in line with previous findings suggesting that attachment figures may help people to cope with negative events (for an overview, see Cassidy and Shaver, 2008), the findings were based on a relatively small sample size.

To conclude, the present findings provided some initial evidence that attachment relationships can buffer the effects of social exclusion, even though these effects are known to be very pervasive, thereby underscoring the power of attachment relationships in coping with stress.

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