doi:10.1093/scan/nst104 SCAN (2014) 9, 1223–1231

Neural bases of antisocial behavior: a voxel-based meta-analysis

Yuta Aoki,[1,2,3] Ryota Inokuchi,[3,4] Tomohiro Nakao,[5] and Hidenori Yamasue[1,6]

[1]Department of Neuropsychiatry, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo 113-8655, Japan, [2]Department of Psychiatry, Tokyo Metropolitan Health and Medical Treatment Corporation, Ebara Hospital, Ota-ku, Tokyo 145-0065, Japan, [3]Department of Emergency and Critical Care Medicine, [4]Department of Emergency and Critical Care Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo 113-8655, Japan, [5]Department of Neuropsychiatry, Graduate School of Medical Sciences, Kyushu University, Higashi-ku, Fukuoka 812-8582, Japan, and [6]Japan Science and Technology Agency, CREST, 5 Sanbancho, Chiyoda-ku, Tokyo 102-0075, Japan

Individuals with antisocial behavior place a great physical and economic burden on society. Deficits in emotional processing have been recognized as a fundamental cause of antisocial behavior. Emerging evidence also highlights a significant contribution of attention allocation deficits to such behavior. A comprehensive literature search identified 12 studies that were eligible for inclusion in the meta-analysis, which compared 291 individuals with antisocial problems and 247 controls. Signed Differential Mapping revealed that compared with controls, gray matter volume (GMV) in subjects with antisocial behavior was reduced in the right lentiform nucleus ($P < 0.0001$), left insula ($P = 0.0002$) and left frontopolar cortex (FPC) ($P = 0.0006$), and was increased in the right fusiform gyrus ($P < 0.0001$), right inferior parietal lobule ($P = 0.0003$), right superior parietal lobule ($P = 0.0004$), right cingulate gyrus ($P = 0.0004$) and the right postcentral gyrus ($P = 0.0004$). Given the well-known contributions of limbic and paralimbic areas to emotional processing, the observed reductions in GMV in these regions might represent neural correlates of disturbance in emotional processing underlying antisocial behavior. Previous studies have suggested an FPC role in attention allocation during emotional processing. Therefore, GMV deviations in this area may constitute a neural basis of deficits in attention allocation linked with antisocial behavior.

Keywords: antisocial personality disorder; Brodmann area (BA) 9; conduct disorder; frontal pole; psychopathy

INTRODUCTION

Individuals with antisocial behavior, such as conduct disorder (CD), antisocial personality disorder (ASPD) and callous-unemotional traits or psychopathy, place a great physical and economic burden on society (Moffitt, 1993; Kratzer and Hodgins, 1997; Loeber and Stouthamer-Loeber, 1998). People with such disorders have symptoms of emotional detachment and a propensity for disinhibited, impulsive behavior combined with a general callousness and lack of insight for the impact that such behavior has on others (Cleckley, 1941; Anderson and Kiehl, 2012). Though not all children with CD have life-course persistent symptoms, genetic studies have suggested that continuous antisocial behavior is heritable (Moffitt, 2005) and children with CD or callous-unemotional traits frequently develop an ASPD or psychopathy in adulthood (Frick and Viding, 2009). Neurodevelopmental theories also suggest that brain abnormalities in early life are associated with lifelong antisocial behavior (Frick and Viding, 2009; Gao *et al.*, 2009), indicating that individuals with CD, callous-unemotional traits, ASPD and psychopathy share a common neural basis.

Whether antisocial behavior is characterized by fundamental deficits in attention or emotion is a long-standing debate (Sadeh and Verona, 2012). Although numerous studies have confirmed that deficits in emotional processing are involved in the pathophysiology of antisocial behavior (Blair, 2007; Sadeh and Verona, 2012), individuals with antisocial behavior also perform abnormally on emotionally neutral tasks (Jutai and Hare, 1983; Blair *et al.*, 2006; Vitale *et al.*, 2007; Zeier *et al.*, 2009; Sadeh *et al.*, 2013). Thus, the pathophysiology model of abnormal emotional processing cannot fully account for such deficits in emotionally neutral information processing. Based on psychological experiments in which appropriate emotional responses were reported when attention was focused on emotional stimuli (Glass and Newman, 2006; Newman *et al.*, 2010; Baskin-Sommers *et al.*, 2011), it is hypothesized that it is not only emotional processing deficits but also attention deficits, especially attention allocation and maintenance during emotional processing, that contributes to the pathophysiology of antisocial behavior (Sadeh and Verona, 2012).

Neuroimaging studies have investigated the neural bases of the pathophysiology of antisocial behavior. The amygdala, one of the centers of emotional processing (Phelps and LeDoux, 2005), is the region most commonly implicated in functional and structural abnormalities of the brain in individuals who display antisocial behavior (Blair *et al.*, 2006; Yang *et al.*, 2009). In addition to the amygdala, the paralimbic system is also recognized as a center of emotional processing and has often been investigated in antisocial behavior research (Blair et al., 2006; Blair, 2007; Raine *et al.*, 2010; Finger *et al.*, 2012; Ly *et al.*, 2012). Among the paralimbic regions, the orbitofrontal cortex (OFC) and ventromedial prefrontal cortex (vmPFC) are those most commonly studied in the field of antisocial behavior, and a number of structural magnetic resonance imaging (MRI) studies have reported abnormalities in these regions in people with antisocial behavior (Boccardi *et al.*, 2011; Hyatt *et al.*, 2012). Functional MRI (fMRI) studies have consistently revealed abnormal activity in the OFC/vmPFC in individuals with antisocial problems during tasks related to emotional processing in value-oriented or social situation, such as making judgments about legal actions (Marsh *et al.*, 2011), social cooperation tasks (Rilling *et al.*, 2002, 2007) and gambling tasks (Mitchell *et al.*, 2002; Blair, 2003). Thus, dysfunction of the amygdala and paralimbic regions has been proposed as fundamental to the pathophysiology of abnormal emotional processing in people with antisocial behavior (Koenigs, 2012).

Received 23 January 2013; Revised 20 May 2013; Accepted 16 July 2013
Advance Access publication 6 August 2013
Part of this study was a funded by the Grants-in-Aid for Scientific Research (22689034 to H.Y.).
Correspondence should be addressed to Yuta Aoki, Department of Neuropsychiatry, Graduate School of Medicine, The University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan. E-mail: yuaoki-tky@umin.ac.jp

Neuroimaging studies with healthy volunteers indicate that the FPC is associated with allocating and maintaining attention on emotional stimuli (Koechlin *et al.*, 1999; Burgess *et al.*, 2007; Tsujimoto *et al.*, 2011). Given that the FPC is a potential neural correlate of attention allocation deficits during emotional processing in individuals with antisocial behavior, we hypothesized that structural abnormalities would be observed in the FPC of such individuals.

A number of whole-brain voxel-based morphometry (VBM) studies of people with antisocial problems have reported various regional gray matter volume (GMV) abnormalities. Some have reported abnormalities in the FPC but results are inconsistent (Tiihonen *et al.*, 2008; Gregory *et al.*, 2012). A contributing factor to the inconsistency of results may be each study's insufficient sample size. Therefore, integration of these results with a statistically conservative threshold would address our hypothesis. To clarify whether VBM studies demonstrate abnormality in the areas related to attention allocation deficit, we conducted a systematic review and meta-analysis of unbiased VBM studies.

METHOD

Study selection

A comprehensive literature search of VBM studies published in peer-reviewed journals between 2001 (the date of the first VBM study in subjects with antisocial behavior) and April 2013 that compared individuals with ASPD, CD, callous-unemotional trait, disruptive behavior disorder and psychopathy with healthy subjects was conducted using the MEDLINE, Embase and Web of Knowledge databases. The search keywords were 'antisocial', 'conduct', 'disruptive', 'oppositional defiant', 'callous-unemotional', 'psychopathy' or 'psychopath', plus 'morphometry', 'voxel-based', 'VBM' or 'voxel-wise'. The titles and abstracts of the studies were examined to determine whether or not they should be included. The reference lists of the included articles were also examined to search for additional relevant studies to be included. We defined the individuals with ASPD, CD, disruptive behavior disorder, callous-unemotional trait and psychopathy as individuals with antisocial behavior.

Selection of studies

Studies were included in our database if (i) they reported a voxel-wise comparison between patients with antisocial behavior and controls for GMV; and similar to previous studies (Radua and Mataix-Cols, 2009; Radua *et al.*, 2010), (ii) they reported whole-brain results in stereotactic coordinates and used thresholds for significance corrected for multiple comparisons, or uncorrected with spatial extent thresholds. The literature search was performed without language restriction. If the study did not provide sufficient data, we emailed the corresponding author to obtain more data. In cases where the author did not respond, we excluded the study. Two of the authors (Y.A. and R.I.) independently screened the studies.

Comparison of regional GMVs

For coordinate-based meta-analysis, we used Signed Differential Mapping (SDM) software (www.sdmproject.com/software/) (Radua and Mataix-Cols, 2009; Radua *et al.*, 2010, 2011; Bora *et al.*, 2011; Nakao *et al.*, 2011) to analyze GM abnormalities in patients with antisocial behavior. Briefly, a map of GMV differences, comprising the reported stereotactic coordinates for each significant group difference, was generated for each study. In SDM, unlike in other coordinate-based, meta-analytic methods, both positive and negative differences are reconstructed in the same map, which prevents a particular voxel from appearing significant in opposite directions. Importantly, when using SDM, the effects of negative studies are also included in the meta-analysis. Meta-analytic statistical maps were subsequently obtained by calculating the corresponding statistics from the study maps, weighted by the square root of the sample size of each study, to enable studies with large sample sizes to contribute proportionally more. A random effect model is applied to integrate the effect sizes of the studies (Radua *et al.*, 2012). The statistical significance of each voxel was determined using randomization tests ($P < 0.001$) as in previous studies (Radua and Mataix-Cols, 2009; Radua *et al.*, 2010; Bora *et al.*, 2011).

Data extraction

We extracted the number of participants of both groups, and the coordinates and effect sizes of peak voxels. When different types of statistical values were reported, such as *z*-values, they were converted to *t*-values, accounting for the number of participants in both groups and the number of covariates. In addition, we extracted the mean age of participants.

In one study, which reported peak coordinates and threshold without statistical values of the coordinates, we determined the threshold value as the effect size of the coordinates (Fahim *et al.*, 2011). The uncorrected statistical thresholds were set at $P < 0.001$ based on previous literature (Radua and Mataix-Cols, 2009; Radua *et al.*, 2010; Bora *et al.*, 2011).

RESULTS

Study selection for database

The literature search produced 23 potential candidates for the meta-analysis. Three studies were excluded because they did not involve healthy control comparisons (Ermer *et al.*, 2012, 2013; Cope *et al.*, 2012). Four studies were discarded because they did not adopt voxel-wise comparison or did not report peak coordinate (Yang *et al.*, 2005; McAlonan *et al.*, 2007; Schiffer *et al.*, 2011; Sato *et al.*, 2011). One study was discarded because it did not ensure any diagnosis of antisocial behavior we defined above in all the participants (Dalwani *et al.*, 2011). One study was excluded because it did not directly compare individuals with antisocial behavior and healthy individuals (Sasayama *et al.*, 2010). One study was discarded because it focused on only white matter (Wu *et al.*, 2011). One study was not included because it was a review article with unpublished data (Vloet *et al.*, 2008) (Figure 1).

Demographic characteristics

A comprehensive literature search identified 12 independent studies which were eligible for inclusion in the meta-analysis (Sterzer *et al.*, 2007; de Oliveira-Souza *et al.*, 2008; Huebner *et al.*, 2008; Müller *et al.*, 2008; Tiihonen *et al.*, 2008; De Brito *et al.*, 2009; Fahim *et al.*, 2011; Fairchild *et al.*, 2011, 2013; Gregory *et al.*, 2012; Stevens and Haney-Caron, 2012; Bertsch *et al.*, in press) (Table 1). In total, these 12 studies compared 291 individuals with antisocial problems and 247 control subjects. Nine studies included only male subjects (Sterzer *et al.*, 2007; Huebner *et al.*, 2008; Müller et al., 2008; Tiihonen *et al.*, 2008; De Brito *et al.*, 2009; Fahim *et al.*, 2011; Fairchild *et al.*, 2011; Gregory *et al.*, 2012; Bertsch *et al.*, in press), while one study recruited only female subjects (Fairchild *et al.*, 2013). Seven reports involved children with antisocial problems; among these seven, five studied children with CD (Sterzer *et al.*, 2007; Huebner *et al.*, 2008; Fairchild *et al.*, 2011, 2013; Stevens and Haney-Caron, 2012), one studied conduct problems (De Brito *et al.*, 2009) and one studied disruptive behavioral disorder (Fahim *et al.*, 2011). Five studies recruited adults, two included individuals with ASPD with psychopathy (Tiihonen *et al.*, 2008; Gregory *et al.*, 2012), one involved psychopathy (de Oliveira-Souza *et al.*, 2008) and two recruited individuals with ASPD (Müller *et al.*, 2008; Bertsch

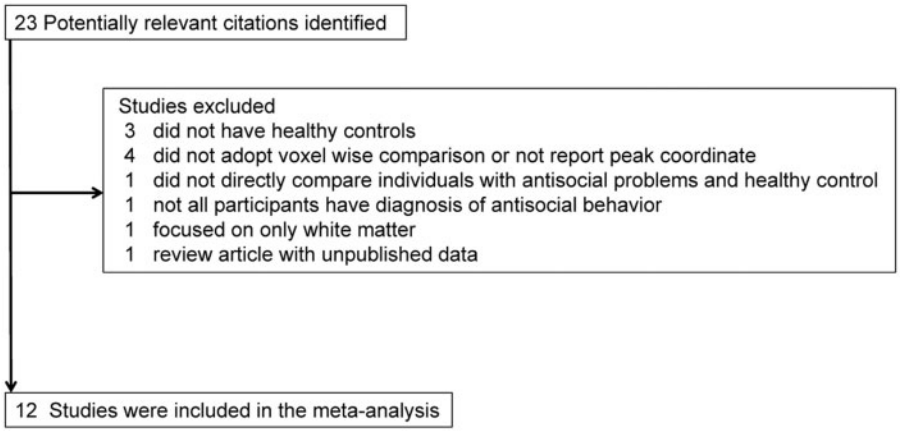

Fig. 1 Process of study selection.

et al., in press). All the studies in the meta-analysis had excluded individuals with mental retardation.

Regional differences in GMV

A meta-analysis revealed that individuals with antisocial behavior had significantly smaller-than-normal GMV in the left superior frontal gyrus in its frontopolar portion (Talairach coordinates: $x=-10$, $y=62$, $z=26$; SDM value $=-2.261$, $P=0.0006$; 16 voxels) (Table 2 and Figure 2a), in the left anterior insula (Talairach coordinates: $x=-40$, $y=8$, $z=10$; SDM value $=-2.389$, $P=0.0002$; 53 voxels) (Table 2 and Figure 2b) and in the right lentiform nucleus (Talairach coordinates: $x=18$, $y=6$, $z=-4$; SDM value $=-2.541$, $P<0.0001$; 110 voxels) (Table 2 and Figure 2c). Although the meta-analysis demonstrated a tendency of smaller-than-normal GMV in the amygdala in individuals with antisocial behavior, it did not reach statistical significance (Talairach coordinates: $x=-28$, $y=2$, $z=-16$; SDM value $=-1.957$, $P=0.0049$).

Furthermore, the analysis also showed a significant increase in GMV in the right fusiform gyrus (Talairach coordinates: $x=46$, $y=-22$, $z=-24$; SDM value $=1.385$, $P<0.0001$; 60 voxels) (Table 2 and Figure 2d), in the right inferior parietal lobule (Talairach coordinates: $x=38$, $y=-30$, $z=42$; SDM value $=1.040$, $P=0.0003$; 39 voxels) (Table 2 and Figure 3a), in the left superior parietal lobule (Talairach coordinates: $x=-36$, $y=-68$, $z=44$; SDM value $=1.002$, $P=0.0004$; 41 voxels) (Table 2 and Figure 3b), in the right cingulate gyrus (Talairach coordinates: $x=12$, $y=8$, $z=44$; SDM value $=1.001$, $P=0.0004$; 41 voxels) (Table 2 and Figure 3c) and right postcentral gyrus (Talairach coordinates: $x=58$, $y=-20$, $z=30$; SDM value $=1.001$, $P=0.0004$; 43 voxels) (Table 2 and Figure 3d) in subjects with antisocial behavior, compared with control subjects. The statistical conclusions for differences in regional brain volumes were preserved after controlling for the effect of age.

DISCUSSION

To the best of our knowledge, this is the first meta-analysis of studies integrating VBM in individuals with antisocial behavior. The analysis identified a significant regional GMV reduction in the left FPC as well as in the paralimbic region, such as the anterior insula, in individuals with antisocial behaviors compared with healthy controls, supporting our hypothesis. The current analysis also found significantly increased GMV in the right fusiform gyrus and the right inferior parietal lobule.

Although the function of the FPC is yet to be elucidated (Tsujimoto *et al.*, 2011), it is thought to be responsible for holding in mind a goal while exploring and processing secondary goals, a process generally required in planning and reasoning, which integrates working memory and attentional resource allocation (Koechlin *et al.*, 1999; Burgess *et al.*, 2007). The FPC is also recognized to be responsible for cognitive branching—the maintenance of pending information related to a previous behavioral episode during an ongoing behavioral episode for future use (Koechlin and Hyafil, 2007; Charron and Koechlin, 2010). Recently, Boorman *et al.* (2009, 2011) showed that the FPC not only represents pending information or intentions for future use but also encodes the reward-based evidence favoring the best counterfactual option for future decisions. These results indicate that the FPC is not simply involved in attention allocation but also plays an important role in complex social decision based on its fundamental role. This notion is supported by results of a number of fMRI studies that have reported that the FPC is responsible for guiding complex social decisions such as moral judgment (Greene *et al.*, 2001; Moll *et al.*, 2002) or charitable donation (Moll *et al.*, 2006). In addition, one study with transcranial direct current stimulation (tDCS) showed that inhibiting the excitability of the FPC with cathodal tDCS did not lead to impairment, but rather to a significant within-subject improvement of deceptive behavior (Karim *et al.*, 2007). These previous studies have strongly indicated the possibility that abnormality in the FPC results in antisocial behavior.

Previous studies in individuals with antisocial behavior have identified an association between the impulsive trait and working memory deficit (Carlson *et al.*, 2009; Venables *et al.*, 2011). It is also thought that the FPC is associated with keeping information in working memory (Koechlin *et al.*, 1999; Charron and Koechlin 2010). Therefore, a reduction in GMV in the FPC may relate to impulsivity. Interestingly, one study describing two case reports of individuals who sustained injury to the FPC reported that damage in this region resulted in impulsive antisocial behavior (Anderson *et al.*, 1999).

The current analysis showed GMV reduction in the anterior insula that consists of the paralimbic regions. The anterior insula has a strong connection with the amygdala (Naqvi and Bechara, 2009; Meyer-Lindenberg and Tost, 2012; Sescousse *et al.*, 2013) and is involved in emotional processing and empathy (Vogt, 2005; de Vignemont and Singer, 2006; Fan *et al.*, 2011; Morita *et al.*, in press; Ponz *et al.*, in press). The previous studies demonstrated abnormal activation of the anterior insula during empathy or emotional processing tasks in individuals with antisocial behavior (Herpertz *et al.*, 2008; Sadeh *et al.*, 2013). In addition, the previous study reported a thinner-than-normal cortex in the anterior insula in individuals with psychopathy (Ly *et al.*,

Table 1 Summary of studies included in the meta-analysis

Study	Diagnosis	No. of ASB (male)	Age of ASB	FIQ	No. of CTRL (male)	Age of CTRL	FIQ	Handedness	Substance abuse	Comorbidity	Strength of magnetic field	FWHM (mm)	Significance	Covariates
Bertsch *et al.*, in press	ASPD	13 (13)	28.9	99.6	14 (14)	26.1	104	NA	54%	BPD	1.5	8	$P < 0.005$, uncorrected	Total intracranial volume
		12 (12)	27.3	97.6					58%	No				
De Brito *et al.* (2009)	Callous–unemotional traits	25 (25)	11.75	107	23 (23)	11.6	95.4	NA	No	NA	3	8	$P < 0.001$, uncorrected	GMV, FIQ, hyperactivity–inattention symptoms
de Oliveira-Souza *et al.* (2008)	Psychopathy	15 (8)	32	NA	15 (8)	32	NA	NA	NA	No	1.5	8	$P < 0.005$, uncorrected	ICV
Fahim *et al.* (2011)	Disruptive behavior	22 (22)	8.39	NA	25 (25)	8.36	NA	All right	NA	No	1.5	10	$P < 0.05$, corrected, FDR	NA
Fairchild *et al.*, 2013	CD	22 (0)	17.23	99.8	20 (0)	17.6	106	One left handed	NA	ADHD, MDD	3	8	$P < 0.001$, uncorrected	GMV
Fairchild *et al.* (2011)	CD	63 (63)	17.78	99.1	27 (27)	18.5	101	NA	9%	Substance abuse	3	8	$P < 0.001$, uncorrected	GMV
Gregory *et al.* (2012)	ASPD with psychopathy	17 (17)	38.9	89.9	22 (22)	32.4	99.4	NA	yes	No	1.5	8	$P < 0.05$, corrected FDR	Age, IQ
Huebner *et al.* (2008)	CD	23 (23)	14.5	96.7	23 (23)	14.2	98.9	NA	No	No	1.5	10	$P < 0.05$ corrected for cluster level $P < 0.001$ uncorrected for voxel level	ICV
Muller *et al.* (2008)	ASPD	17 (17)	33	NA	17 (17)	30.6	NA	NA	53%	Substance dependence	1.5	10	$P < 0.05$, corrected	Time of drug intake
Tiihonen *et al.* (2008)	ASPD and psychopathy	26 (26)	32.5	NA	25 (25)	34.6	NA	NA	77%	Substance abuse	1	8	$P < 0.05$, corrected	Age, ICV
Sterzer *et al.* (2007)	CD	12 (12)	12.75	101	12 (12)	12.5	107	NA	NA	ADHD	1.5	8	$P < 0.05$, corrected	Global grey matter sign, Age, IQ
Stevens and Haney-Caron (2012)	CD	24 (16)	16	NA	24 (16)	16	NA	NA	4%	Substance abuse	3	8	$P < 0.05$, uncorrected	No

ASB, antisocial behavior; FIQ, full-scale intelligence quotient; FWHM, full-width at half-maximum; FDR, false discovery rate; CD, conduct disorder; ASPD, antisocial personality disorder; ADHD, attention deficit hyperactivity disorder; MDD, major depressive disorder; NA, not available; GMV, gray matter volume; ICV, intracranial volume.

2012). Although we have predicted abnormality in the amygdala as a potential neural correlate of abnormal emotional processing in individuals with antisocial behavior, the current meta-analysis suggested that abnormality in the anterior insula may also be responsible for abnormal emotional processing among them.

The analysis identified significantly smaller-than-normal GMV in the lentiform nucleus, mainly the putamen. Neuroimaging studies have repeatedly reported that the putamen is involved in reward-based learning (O'Doherty *et al.*, 2004, 2006; Samejima *et al.*, 2005; Liu *et al.*, 2011, Wunderlich *et al.*, 2012). Furthermore, it was also demonstrated that the putamen, along with the insula, is involved in judgment of distribution of justice (Hsu *et al.*, 2008). As reward-based learning is disturbed in individuals with antisocial behavior (Finger *et al.*, 2011), abnormality in these structures is suggested to be a potential pathophysiology of antisocial behavior (Glenn and Yang, 2012). However, the current finding should be interpreted with caution, because of comorbid attention deficit personality disorder (ADHD). ADHD is a frequent comorbidity in individuals with antisocial behavior clinically and sub-clinically, and some of the included studies in the current meta-analysis recruited individuals with comorbid diagnosis of ADHD (Sterzer *et al.*, 2007; Fairchild *et al.*, 2013). As it has been shown that there is a smaller-than-normal GMV in the right lentiform nucleus in individuals with ADHD (Nakao *et al.*, 2011), comorbid ADHD may have influenced the results.

The meta-analysis further identified a GMV increase in the fusiform gyrus as a potential neural basis of antisocial behavior. Although we could not statistically test relationship between symptoms of antisocial behavior and abnormality of GMV in the fusiform gyrus, previous fMRI studies suggest how abnormal GMV in the fusiform gyrus attributes to antisocial behavior. The fusiform gyrus is thought to be directly involved in the process of social categorization via top-down modulation of social and face perception (Sabatinelli *et al.*, 2011; Schwarz *et al.*, 2013; Shkurko, in press) and emotions of guilt and shame (Takahashi *et al.*, 2004; Michl *et al.*, in press). It is also thought that the right fusiform gyrus is a center of rapid learning regarding the moral status of others (Singer *et al.*, 2004). In addition, individuals with Klinefelter syndrome, a chromosomal condition (XXY) whose phenotype is high risk for antisocial behavior, displayed less activation of the fusiform gyrus during judgment of faces with regard to trustworthiness (van Rijn *et al.*, 2012). These evidence suggest that abnormal GMV in the fusiform gyrus is related to deviated face recognition (Dolan and Fullam, 2006) and sense of guilt and shame (Tangney *et al.*, 2011) in individuals with antisocial behavior.

The analysis also identified an increase in GMV in the inferior parietal lobule as a potential neural correlate of antisocial behavior. This area has been suggested to contain mirror neurons (Molenberghs *et al.*, 2012), indicating that disturbance in this region results in various social dysfunctions. For example, the inferior parietal lobule is involved in gaze processing (Pelphrey *et al.*, 2003, 2004), action perception in understanding intentions (Gallese *et al.*, 2004), comprehending impressions of others (Mende-Siedlecki *et al.*, in press), predicting the actions of from their gaze (Ramsey *et al.*, 2012) and risk-taking action (Tamura *et al.*, 2012). Based on previous fMRI studies, increased GMV in the inferior parietal lobule may reflect inappropriate eye gazing of individuals with antisocial behavior (Dadds *et al.*, 2008). The analysis also demonstrated larger-than-normal GMV in the left superior parietal lobule. The superior parietal lobule, which is often activated together with the inferior parietal lobule (Culham *et al.*, 1998), is involved in spatial attention (Molenberghs *et al.*, 2007) and is reported to be abnormally activated for fearful congruent in individuals with antisocial behavior (White *et al.*, 2012). The current analysis also demonstrated larger-than-normal GMV in the postcentral gyrus of subjects with antisocial behavior. Recent studies suggested

Table 2 Results of meta-analysis of VBM studies comparing individuals with antisocial behavior and controls

Anatomical location	Maximum			Cluster		
	Talairach coordinate	SDM value	*P*-value (uncorrected)	Total cluster size	Breakdown	Sub-cluster size
Smaller gray matter volume (individuals with antisocial behavior < controls)						
Right lentiform nucleus	18, 6, −4	−2.541	<0.0001	110	rt. putamen	69
					rt. lateral globus pallidus	21
					rt. caudate head	18
					Other sub-lobar region	2
Left insula	−40, 8, 10	−2.389	0.0002	53	lt. insula	46
					lt. precentral gyrus	7
Left superior frontal gyrus	−10, 62, 26	−2.261	0.0006	16	lt. superior frontal gyrus	16
Larger gray matter volume (individuals with antisocial behavior > controls)						
Right fusiform gyrus	46, −22, −24	1.385	<0.0001	60	rt. fusiform gyrus	33
					rt. inferior temporal gyrus	27
Right inferior parietal lobule	38, −30, 42	1.040	0.0003	39	rt. inferior parietal lobule	37
					rt. postcentral gyrus	2
Left superior parietal lobule	−36, −68, 44	1.002	0.0004	41	lt. superior parietal lobule	35
					lt. inferior parietal lobule	5
					lt. precuneus	1
Right cingulate gyrus	12, 8, 44	1.001	0.0004	63	rt. cingulate gyrus	15
					rt. medial frontal gyrus	40
					rt. superior frontal gyrus	8
Right postcentral gyrus	58, −20, 30	1.001	0.0004	43	rt. postcentral gyrus	38
					rt. inferior parietal lobule	5

SDM, signed differential mapping.

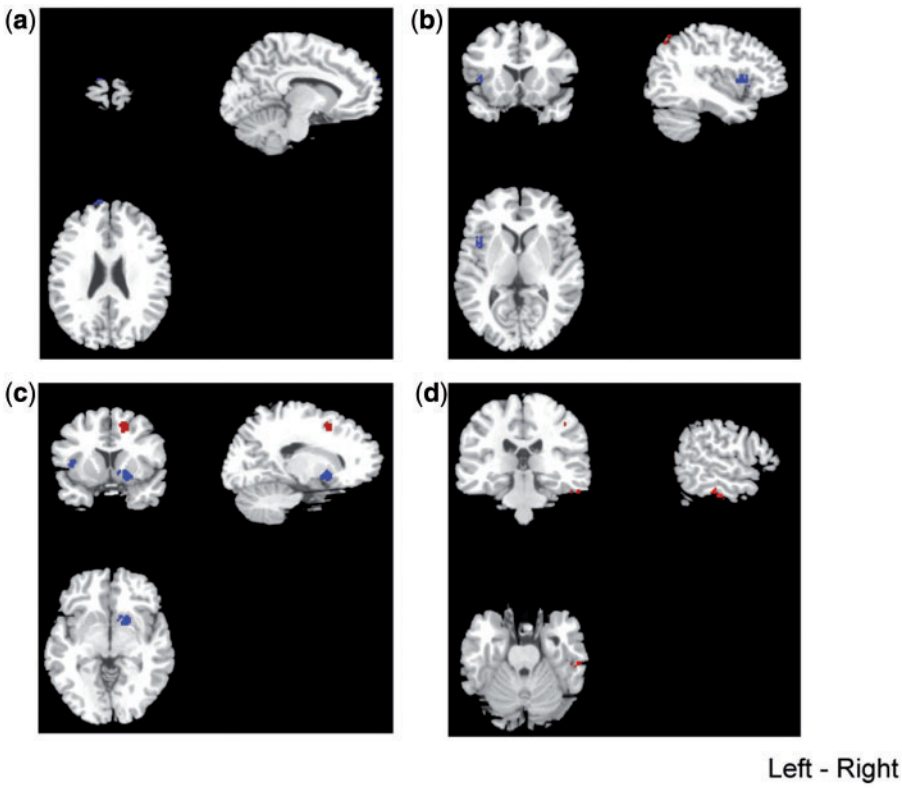

Fig. 2 Regions of decreases (blue) or increases (red) in regional gray matter volume in individuals with antisocial behavior, compared with controls voxel threshold $P < 0.001$. (**a**) Left frontopolar cortex; (**b**) left insula; (c) lentiform nucleus; (d) right fusiform.

that the right postcentral gyrus was associated with emotional processing and empathy (Bernhardt and Singer, 2012; Morelli *et al.*, in press; Sarkheil *et al.*, in press). Thus, this abnormality may relate to a disturbance of emotional processing and empathy in individuals with antisocial behavior. The cingulate gyrus is also demonstrated to be larger-than-normal in individuals with antisocial behavior. As a number of previous fMRI studies reported abnormal activation of the BOLD signal during moral- or shame-related tasks (Raine and

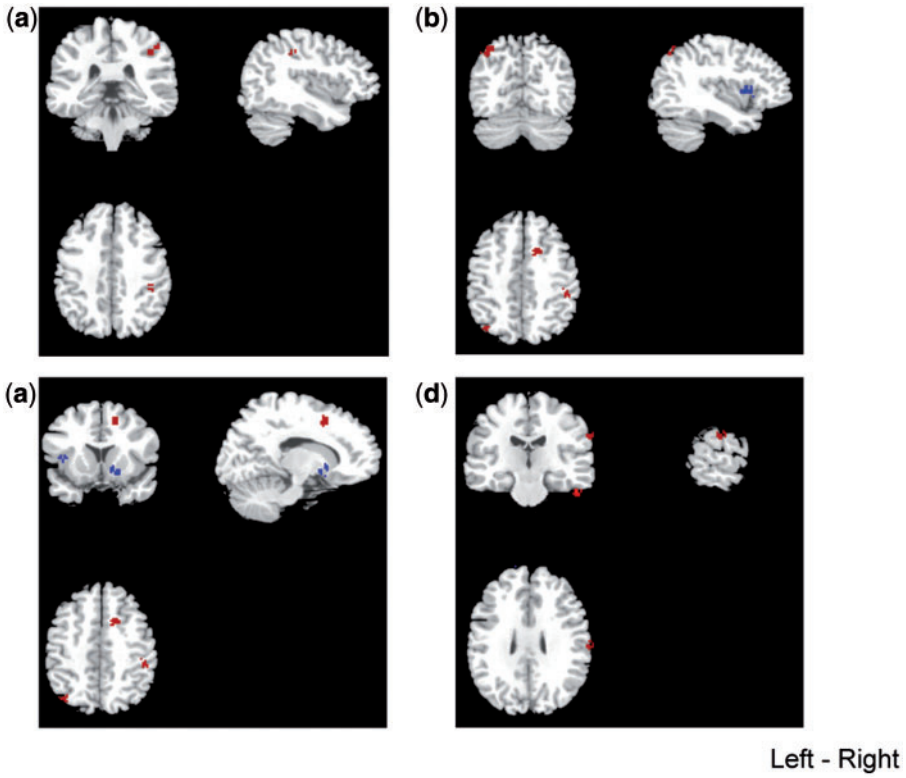

Fig. 3 Regions of decreases (blue) or increases (red) in regional gray matter volume in individuals with antisocial behavior, compared with controls voxel threshold $P < 0.001$. (**a**) Right inferior parietal lobule; (b) left superior parietal lobule; (c) right cingulate gyurs; (d) right postcentral gyrus.

Yang, 2006; Christensen *et al.*, in press; Michl *et al.*, in press), the structural abnormality may contribute to these dysfunctions.

As a number of functional neuroimaging studies of individuals with antisocial behavior have repeatedly shown functional abnormality in the amygdala and OFC/vmPFC (Phelps and LeDoux, 2005; Blair, 2007; Yang *et al.*, 2009; Hyatt *et al.*, 2012), we have predicted GMV reduction in these regions. But contrary to the prediction, the analysis did not show significant GMV reduction in the amygdala and OFC/vmPFC. This dissociation between functional and structural alteration is surprising. A possible explanation for this negative result is the heterogeneity of the participants. We integrated people with several different disorders into the analysis, because all were at higher risk of antisocial behavior. Further, it is thought that people with these disorders share a common neural basis of antisocial behavior. However, some participants with CD and ASPD had a dual diagnosis of psychopathy. The diagnosis of CD and ASPD has also been criticized for over-emphasizing behavioral outcomes (such as criminality) and neglecting core psychological features (Blair, 2007). With this in mind, it is possible that we have integrated individuals with similar behavioral phenotypes but with partially different neural correlates. Another explanation is that functional abnormality derives from abnormality in the white matter instead of the gray matter. As abnormal connectivity between the amygdala and the OFC/vmPFC has been reported in individuals with antisocial behavior (Passamonti *et al.*, 2012), white matter abnormality without GMV reduction may contribute to their well-established functional abnormality.

Limitations

There are some methodological considerations in the reported meta-analysis. First, although we have integrated only whole-brain VBM studies in individuals with antisocial behavior, there is considerable heterogeneity between studies, in terms of participants and methodologies. For example, as we discussed above, we may have included studies with individuals with similar phenotypes but different neural or psychological bases for their symptoms. In addition, there is significant diversity in the methodology of imaging between the studies we included, such as smoothing function used and strength of magnetic field. Further, the studies adopted different statistical analyses. Thus, although we used a conservative threshold in our analysis to minimize study heterogeneity, the results should nevertheless be treated with caution. Second, the majority of participants within the integrated studies had psychiatric comorbidity, such as substance abuse or subclinical features of other psychiatric disorders, including depression, anxiety disorder, autism, and attention deficit hyperactivity disorder. It is known that these comorbid conditions have an impact on structure of the frontotemporal cortex (Yamasue *et al.*, 2003, 2004; Aoki et al, 2012a,b,c; Lucantonio *et al.*, 2012). Therefore, it is possible that the abnormal GMV was an artifact of the comorbid psychiatric disorders. In addition, differences in the subjects' behavioral and emotional traits may also affect GMV (Takeuchi *et al.*, 2011; Morishima *et al.*, 2012; Takeuchi *et al.*, in press). We could not conduct sensitivity analysis or meta-regression due to an insufficient number of studies, although our conservative meta-analysis of unbiased studies demonstrated significant abnormalities in GMV. Thus, although we robustly found GMV abnormalities, the functions of which may relate to a psychological trait of individuals with antisocial behavior, we could not directly address the relationship between abnormalities in brain structure and behavior. Third, as non-significant data have a higher possibility of not being published, there exists strong publication bias. In addition, although SDM reconstructs both positive and negative differences in the same map (signed map) (Radua and Mataix-Cols, 2009; Radua

et al., 2010), peak-based meta-analyses are based on highly significant data (i.e. $P < 0.001$ uncorrected) rather than raw statistical brain maps, and this approach may result in less accurate results.

CONCLUSION

In conclusion, the meta-analysis of unbiased whole-brain VBM studies of individuals with antisocial behavior demonstrated significantly abnormal GMV reductions in the FPC and parahippocampal gyrus, including the amygdala, and GMV increases in the right fusiform gyrus and the right inferior parietal lobule. These abnormalities may correspond to deficits in keeping information in the working memory during allocation of attention, emotional processing and inappropriate face information processing in social context. The current analysis emphasized that attention deficit is also an important factor in the pathophysiology of individuals with antisocial behavior.

Conflict of Interest

None declared.

REFERENCES

Anderson, N.E., Kiehl, K.A. (2012). The psychopath magnetized: insights from brain imaging. *Trends in Cognitive Sciences*, *16*, 52–60.

Anderson, S.W., Bechara, A., Damasio, H., Tranel, D., Damasio, A.R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*, *2*, 1032–7.

Aoki, Y., Abe, O., Yahata, N., et al. (2012a). Absence of age-related prefrontal NAA change in adults with autism spectrum disorders. *Translational Psychiatry*, *2*, e178.

Aoki, Y., Aoki, A., Suwa, H. (2012b). Reduction of N-acetylaspartate in the medial prefrontal cortex correlated with symptom severity in obsessive-compulsive disorder: meta-analyses of 1H-MRS studies. *Translational Psychiatry*, *2*, e153.

Aoki, Y., Kasai, K., Yamasue, H. (2012c). Age-related change in brain metabolite abnormalities in autism: a meta-analysis of proton magnetic resonance spectroscopy studies. *Translational Psychiatry*, *2*, e69.

Baskin-Sommers, A.R., Curtin, J.J., Newman, J.P. (2011). Specifying the attentional selection that moderates the fearlessness of psychopathic offenders. *Psychological Science*, *22*, 226–34.

Bernhardt, B.C., Singer, T. (2012). The neural basis of empathy. *Annual Reviews Neuroscience*, *35*, 1–23.

Bertsch, K., Grothe, M., Prehn, K., et al. (in press). Brain volumes differ between diagnostic groups of violent criminal offenders. *European Archives of Psychiatry Clinical Neuroscience*.

Blair, K.S., Newman, C., Mitchell, D.G.V., et al. (2006). Differentiating among prefrontal substrates in psychopathy: neuropsychological test findings. *Neuropsychology*, *20*, 153–65.

Blair, R.J.R. (2003). Neurobiological basis of psychopathy. *The British Journal of Psychiatry*, *182*, 5–7.

Blair, R.J.R. (2007). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends in Cognitive Sciences*, *11*, 387–92.

Blair, R.J.R., Peschardt, K.S., Budhani, S., Mitchell, D.G.V., Pine, D.S. (2006). The development of psychopathy. *Journal of Child Psychology and Psychiatry*, *47*, 262–76.

Boccardi, M., Frisoni, G.B., Hare, R.D., et al. (2011). Cortex and amygdala morphology in psychopathy. *Psychiatry Research*, *193*, 85–92.

Bora, E., Fornito, A., Yücel, M., Pantelis, C. (2011). The effects of gender on grey matter abnormalities in major psychoses: a comparative voxelwise meta-analysis of schizophrenia and bipolar disorder. *Psychological Medicine*, 1–13.

Boorman, E.D., Behrens, T.E., Woolrich, M.W., Rushworth, M.F. (2009). How green is the grass on the other side? Frontopolar cortex and the evidence in favor of alternative courses of action. *Neuron*, *62*, 733–43.

Boorman, E.D., Behrens, T.E., Rushworth, M.F. (2011). Counterfactual choice and learning in a neural network centered on human lateral frontopolar cortex. *PLoS Biology*, *9*, e1001093.

Burgess, P.W., Dumontheil, I., Gilbert, S.J. (2007). The gateway hypothesis of rostral prefrontal cortex (area 10) function. *Trends in Cognitive Sciences*, *11*, 290–8.

Carlson, S.R., Thái, S., McLarnon, M.E. (2009). Visual P3 amplitude and self-reported psychopathic personality traits: frontal reduction is associated with self-centered impulsivity. *Psychophysiology*, *46*, 100–13.

Charron, S., Koechlin, E. (2010). Divided representation of concurrent goals in the human frontal lobes. *Science*, *328*, 360–3.

Christensen, J.F., Flexas, A., de Miguel, P., Cela-Conde, C.J., Munar, E. (in press). Roman Catholic beliefs produce characteristic neural responses to moral dilemmas. *Social Cognitive and Affective Neuroscience*.

Cleckley, H.M. (1941). *The Mask of Sanity, An Attempt to Reinterpret the So-called Psychopathic Personality*. St Louis, MO: Mosby.

Cope, L.M., Shane, M.S., Segall, J.M., et al. (2012). Examining the effect of psychopathic traits on gray matter volume in a community substance abuse sample. *Psychiatry Research*, *204*, 91–100.

Culham, J.C., Brandt, S.A., Cavanagh, P., Kanwisher, N.G., Dale, A.M., Tootell, R.B. (1998). Cortical fMRI activation produced by attentive tracking of moving targets. *Journal of Neurophysiology*, *80*, 2657–70.

Dadds, M.R., Masry, El.Y., Wimalaweera, S., Guastella, A.J. (2008). Reduced eye gaze explains "fear blindness" in childhood psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 455–63.

Dalwani, M., Sakai, J.T., Mikulich-Gilbertson, S.K., et al. (2011). Reduced cortical gray matter volume in male adolescents with substance and conduct problems. *Drug and Alcohol Dependence*, *118*, 295–305.

De Brito, S.A., Mechelli, A., Wilke, M., Laurens, K.R., Jones, A.P., Barker, G.J., et al. (2009). Size matters: increased grey matter in boys with conduct problems and callous-unemotional traits. *Brain*, *132*, 843–52.

de Oliveira-Souza, R., Hare, R.D., Bramati, I.E., et al. (2008). Psychopathy as a disorder of the moral brain: fronto-temporo-limbic grey matter reductions demonstrated by voxel-based morphometry. *Neuroimage*, *40*, 1202–13.

de Vignemont, F., Singer, T. (2006). The empathic brain: how, when and why? *Trends in Cognitive Science*, *10*, 435–41.

Dolan, M., Fullam, R. (2006). Face affect recognition deficits in personality-disordered offenders: association with psychopathy. *Psychological Medicine*, *36*, 1563–9.

Ermer, E., Cope, L.M., Nyalakanti, P.K., Calhoun, V.D., Kiehl, K.A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of Abnormal Psychology*, *121*, 649–58.

Ermer, E., Cope, L.M., Nyalakanti, P.K., Calhoun, V.D., Kiehl, K.A. (2013). Aberrant paralimbic gray matter in incarcerated male adolescents with psychopathic traits. *Journal of American Academy of Child and Adolescent Psychiatry*, *52*, 94–103.

Fahim, C., He, Y., Yoon, U., Chen, J., Evans, A., Pérusse, D. (2011). Neuroanatomy of childhood disruptive behavior disorders. *Aggressive Behavior*, *37*, 326–37.

Fairchild, G., Hagan, C.C., Walsh, N.D., Passamonti, L., Calder, A.J., Goodyer, I.M. (2013). Brain structure abnormalities in adolescent girls with conduct disorder. *Journal of Child Psychology and Psychiatry*, *54*, 86–95.

Fairchild, G., Passamonti, L., Hurford, G., et al. (2011). Brain structure abnormalities in early-onset and adolescent-onset conduct disorder. *The American Journal of Psychiatry*, *168*, 624–33.

Fan, Y., Duncan, N.W., de Greck, M., Northoff, G. (2011). Is there a core neural network in empathy? An fMRI based quantitative meta-analysis. *Neuroscience and Biobehavioral Reviews*, *35*, 903–11.

Finger, E.C., Marsh, A., Blair, K.S., Majestic, C., Evangelou, I., Gupta, K., et al. (2012). Impaired functional but preserved structural connectivity in limbic white matter tracts in youth with conduct disorder or oppositional defiant disorder plus psychopathic traits. *Psychiatry Research*, *202*, 239–44.

Finger, E.C., Marsh, A.A., Blair, K.S., et al. (2011). Disrupted reinforcement signaling in the orbitofrontal cortex and caudate in youths with conduct disorder or oppositional defiant disorder and a high level of psychopathic traits. *American Journal of Psychiatry*, *168*, 152–62.

Frick, P.J., Viding, E. (2009). Antisocial behavior from a developmental psychopathology perspective. *Development and Psychopathology*, *21*, 1111–31.

Gallese, V., Keysers, C., Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Sciences*, *8*, 396–403.

Gao, Y., Glenn, A.L., Schug, R.A., Yang, Y., Raine, A. (2009). The neurobiology of psychopathy: a neurodevelopmental perspective. *Canadian Journal of Psychiatry*, *54*, 813–23.

Glass, S.J., Newman, J.P. (2006). Recognition of facial affect in psychopathic offenders. *Journal of Abnormal Psychology*, *115*, 815–20.

Glenn, A.L., Yang, Y. (2012). The potential role of the striatum in antisocial behavior and psychopathy. *Biological Psychiatry*, *72*, 817–22.

Greene, J.D., Sommerville, R.B., Nystrom, L.E., Darley, J.M., Cohen, J.D. (2001). An fMRI investigation of emotional engagement in moral judgment. *Science*, *293*, 2105–8.

Gregory, S., Ffytche, D., Simmons, A., et al. (2012). The antisocial brain: psychopathy matters: a structural MRI investigation of antisocial male violent offenders. *Archives of General Psychiatry*, *69*, 962–72.

Herpertz, S.C., Huebner, T., Marx, I., et al. (2008). Emotional processing in male acolescents with childhood-onset conduct disorder. *Journal of Child Psychology and Psychiatry*, *49*, 781–91.

Hsu, M., Anen, C., Quartz, S.R. (2008). The right and the good: distributive justice and neural encoding of equity and efficiency. *Science*, *320*, 1092–5.

Huebner, T., Vloet, T.D., Marx, I., et al. (2008). Morphometric brain abnormalities in boys with conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 540–7.

Hyatt, C.J., Haney-Caron, E., Stevens, M.C. (2012). Cortical thickness and folding deficits in conduct-disordered adolescents. *Biological Psychiatry*, *72*, 207–14.

Jutai, J.W., Hare, R.D. (1983). Psychopathy and selective attention during performance of a complex perceptual-motor task. *Psychophysiology*, *20*, 146–51.

Karim, A.A., Schneider, M., Lotze, M., Veit, R., Sauseng, P., Braun, C., Birbaumer, N. (2010). The truth about lying: inhibition of the anterior prefrontal cortex improves deceptive behavior. *Cereb Cortex*, *20*, 205–13.

Koechlin, E., Basso, G., Pietrini, P., Panzer, S., Grafman, J. (1999). The role of the anterior prefrontal cortex in human cognition. *Nature*, *399*, 148–51.

Koechlin, E., Hyafil, A. (2007). Anterior prefrontal function and the limits of human decision-making. *Science*, *318*, 594–8.

Koenigs, M. (2012). The role of prefrontal cortex in psychopathy. *Reviews in the Neurosciences*, *23*, 253–62.

Kratzer, L., Hodgins, S. (1997). Adult outcomes of child conduct problems: a cohort study. *Journal of Abnormal Child Psychology*, *25*, 65–81.

Liu, X., Hairston, J., Schrier, M., Fan, J. (2011). Common and distinct networks underlying reward valence and processing stages: a meta-analysis of functional neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, *35*, 1219–36.

Loeber, R., Stouthamer-Loeber, M. (1998). Development of juvenile aggression and violence. Some common misconceptions and controversies. *American Psychologist*, *53*, 242–59.

Lucantonio, F., Stalnaker, T.A., Shaham, Y., Niv, Y., Schoenbaum, G. (2012). The impact of orbitofrontal dysfunction on cocaine addiction. *Nature Neuroscience*, *15*, 358–66.

Ly, M., Motzkin, J.C., Philippi, C.L., et al. (2012). Cortical thinning in psychopathy. *The American Journal of Psychiatry*, *169*, 743–9.

Marsh, A.A., Finger, E.C., Fowler, K.A., et al. (2011). Reduced amygdala-orbitofrontal connectivity during moral judgments in youths with disruptive behavior disorders and psychopathic traits. *Psychiatry Research*, *194*, 279–86.

McAlonan, G.M., Cheung, V., Cheung, C., et al. (2007). Mapping brain structure in attention deficit-hyperactivity disorder: a voxel-based MRI study of regional grery and white matter volume. *Psychiatry Research Neuroimaging*, *154*, 171–80.

Mende-Siedlecki, P., Cai, Y., Todorov, A. (in press). The neural dynamics of updating person impressions. *Social Cognitive and Affective Neuroscience*.

Meyer-Lindenberg, A., Tost, H. (2012). Neural mechanisms of social risk for psychiatric disorders. *Nature Neuroscience*, *15*, 663–8.

Michl, P., Meindl, T., Meister, F., et al. (in press). Neurobiological underpinnings of shame and guilt: a pilot fMRI study. *Social Cognitive and Affective Neuroscience*.

Mitchell, D.G.V., Colledge, E., Leonard, A., Blair, R.J.R. (2002). Risky decisions and response reversal: is there evidence of orbitofrontal cortex dysfunction in psychopathic individuals? *Neuropsychologia*, *40*, 2013–22.

Moffitt, T.E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychological Reviews*, *100*, 674–701.

Moffitt, T.E. (2005). Genetic and environmental influences on antisocial behaviors: evidence from behavioral-genetic research. *Advances in Genetics*, *55*, 41–104.

Molenberghs, P., Cunnington, R., Mattingley, J.B. (2012). Brain regions with mirror properties: a meta-analysis of 125 human fMRI studies. *Neuroscience and Biobehavioral Reviews*, *36*, 341–9.

Molenberghs, P., Mesulam, M.M., Peeters, R., Vandenberghe, R.R. (2007). Remapping attentional priorities: differential contribution of superior parietal lobule and intraparietal sulcus. *Cerebral Cortex*, *17*, 2703–12.

Moll, J., de Oliveira-Souza, R., Eslinger, P.J., et al. (2002). The neural correlates of moral sensitivity: a functional magnetic resonance imaging investigation of basic and moral emotions. *Journal of Neuroscience*, *22*, 2730–6.

Moll, J., Krueger, F., Zahn, R., Pardini, M., de Oliveira-Souza, R., Grafman, J. (2006). Human fronto-mesolimbic networks guide decisions about charitable donation. *Proceedings of the National Academy of Sciences of the United States of America*, *103*, 15623–8.

Morelli, S.A., Rameson, L.T., Lieberman, M.D. (in press). The neural components of empathy: predicting daily prosocial behavior. *Social Cognitive and Affective Neuroscience*.

Morishima, Y., Schunk, D., Bruhin, A., Ruff, C.C., Fehr, E. (2012). Linking brain structure and activation in temporoparietal junction to explain the neurobiology of human altruism. *Neuron*, *75*, 73–9.

Morita, T., Tanabe, H.C., Sasaki, A.T., Shimada, K., Kakigi, R., Sadato, N. (in press). The anterior insular and anterior cingulate cortices in emotional processing for self-face recognition. *Social Cognitive and Affective Neuroscience*.

Müller, J.L., Gänßbauer, S., Sommer, M., et al. (2008). Gray matter changes in right superior temporal gyrus in criminal psychopaths. Evidence from voxel-based morphometry. *Psychiatry Research*, *163*, 213–22.

Nakao, T., Radua, J., Rubia, K., Mataix-Cols, D. (2011). Gray matter volume abnormalities in ADHD: voxel-based meta-analysis exploring the effects of age and stimulant medication. *The American Journal of Psychiatry*, *168*, 1154–63.

Naqvi, N.H., Bechara, A. (2009). The hidden island of addiction: the insula. *Trends in Neuroscience*, *32*, 56–67.

Newman, J.P., Curtin, J.J., Bertsch, J.D., Baskin-Sommers, A.R. (2010). Attention moderates the fearlessness of psychopathic offenders. *Biological Psychiatry*, *67*, 66–70.

O'Doherty, J., Buchanan, T.W., Seymour, B., Dolan, R.J. (2006). Predictive neural coding of reward preference involves dissociable responses in human ventral midbrain and ventral striatum. *Neuron*, *49*, 157–66.

O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., Dolan, R.J. (2004). Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science*, *304*, 452–4.

Passamonti, L., Fairchild, G., Fornito, A., et al. (2012). Abnormal anatomical connectivity between the amygdala and orbitofrontal cortex in conduct disorder. *PLoS One*, *7*, e48789.

Pelphrey, K.A., Singerman, J.D., Allison, T., McCarthy, G. (2003). Brain activation evoked by perception of gaze shifts: the influence of context. *Neuropsychologia*, *41*, 156–70.

Pelphrey, K.A., Viola, R.J., McCarthy, G. (2004). When strangers pass: processing of mutual and averted social gaze in the superior temporal sulcus. *Psychological Science*, *15*, 598–603.

Phelps, E.A., LeDoux, J.E. (2005). Contributions of the amygdala to emotion processing: from animal models to human behavior. *Neuron*, *48*, 175–87.

Ponz, A., Montant, M., Liegeois-Chauvel, C., et al. (in press). Emotion processing in words: a test of the neural re-use hypothesis using surface and intracranial EEG. *Social Cognitive and Affective Neuroscience*.

Radua, J., Mataix-Cols, D. (2009). Voxel-wise meta-analysis of grey matter changes in obsessive-compulsive disorder. *The British Journal of Psychiatry*, *195*, 393–402.

Radua, J., Mataix-Cols, D., Philips, M.L., et al. (2012). A new meta-analytic method for neuroimaging studies that combines reported peak coordinates and statistical parametric maps. *European Psychiatry*, *27*, 605–11.

Radua, J., van den Heuvel, O.A., Surguladze, S., Mataix-Cols, D. (2010). Meta-analytical comparison of voxel-based morphometry studies in obsessive-compulsive disorder vs other anxiety disorders. *Archives of General Psychiatry*, *67*, 701–11.

Radua, J., Via, E., Catani, M., Mataix-Cols, D. (2011). Voxel-based meta-analysis of regional white-matter volume differences in autism spectrum disorder versus healthy controls. *Psychological Medicine*, *41*, 1539–50.

Raine, A., Lee, L., Yang, Y., Colletti, P. (2010). Neurodevelopmental marker for limbic maldevelopment in antisocial personality disorder and psychopathy. *The British Journal of Psychiatry*, *197*, 186–92.

Raine, A., Yang, Y. (2006). Neural foundations to moral reasoning and antisocial behavior. *Social Cognitive and Affective Neuroscience*, *1*, 203–13.

Ramsey, R., Cross, E.S., de C. Hamilton, A.F. (2012). Predicting others' actions via grasp and gaze: evidence for distinct brain networks. *Psychological Research*, *76*, 494–502.

Rilling, J.K., Glenn, A.L., Jairam, M.R., Pagnoni, G., Goldsmith, D.R., Elfenbein, H.A. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological Psychiatry*, *61*, 1260–71.

Rilling, J., Gutman, D., Zeh, T., Pagnoni, G., Berns, G., Kilts, C. (2002). A neural basis for social cooperation. *Neuron*, *35*, 395–405.

Sabatinelli, D., Fortune, E.E., Li, Q., et al. (2011). Emotional perception: meta-analyses of face and natural scene processing. *Neuroimage*, *54*, 2524–33.

Sadeh, N., Spielberg, J.M., Heller, W., et al. (2013). Emotion disrupts neural activity during selective attention in psychopathy. *Social Cognitive and Affective Neuroscience*, *8*, 235–46.

Sadeh, N., Verona, E. (2012). Visual complexity attenuates emotional processing in psychopathy: implications for fear-potentiated startle deficits. *Cognitive Affective and Behavioral Neuroscience*, *12*, 346–60.

Samejima, K., Ueda, Y., Doy, K., Kimura, M. (2005). Representation of action-specific reward values in the striatum. *Science*, *310*, 1337–40.

Sarkheil, P., Goebel, R., Schneider, F., Mathiak, K. (in press). Emotion unfolded by motion: a role for parietal lobe in decoding dynamic facial expressions. *Social Cognitive and Affective Neuroscience*.

Sasayama, D., Hayashida, A., Yamasue, H., et al. (2010). Neuroanatomical correlates of attention-deficit-hyperactivity disorder accounting for comorbid oppositional defiant disorder and conduct disorder. *Psychiatry and Clinical Neuroscience*, *64*, 394–402.

Sato, J.R., de Oliveira-Souza, R., Thomaz, C.E., et al. (2011). Identification of psychopathic individuals using pattern classification of MRI images. *Social Neuroscience*, *6*, 627–39.

Schiffer, B., Müller, B.W., Scherbaum, N., et al. (2011). Disentangling structural brain alterations associated with violent behavior from those associated with substance use disorders. *Archives of General Psychiatry*, *68*, 1039–1049.

Schwarz, K.A., Wieser, M.J., Gerdes, A.B.M., Mühlberger, A., Pauli, P. (2013). Why are you looking like that? How the context influences evaluation and processing of human faces. *Social Cognitive and Affective Neuroscience*, *8*, 438–45.

Sescousse, G., Caldu, X., Segura, B., Dreher, J.C. (2013). Processing of primary and secondary rewards: a quantitative meta-analysis and review of human functional neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, *37*, 681–96.

Shkurko, A.V. (in press). Is social categorization based on relational ingroup/outgroup opposition? A meta-analysis. *Social Cognitive and Affective Neuroscience*.

Singer, T., Kiebel, S.J., Winston, J.S., Dolan, R.J., Frith, C.D. (2004). Brain responses to the acquired moral status of faces. *Neuron*, *41*, 653–62.

Starcke, K., Brand, M. (2012). Decision making under stress: a selective review. *Neuroscience and Biobehavioral Reviews*, *36*, 1228–48.

Sterzer, P., Christina Stadler, C., Poustka, F., Kleinschmidta, A. (2007). A structural neural deficit in adolescents with conduct disorder and its association with lack of empathy. *Neuroimage*, *37*, 335–42.

Stevens, S., Haney-Caron, E. (2012). Comparison of brain volume abnormalities between ADHD and conduct disorder in adolescence. *Journal of Psychiatry Neuroscience*, *37*, 389–98.

Takahashi, H., Yahata, N., Koeda, M., Matsuda, T., Asai, K., Okubo, Y. (2004). Brain activation associated with evaluative processes of guilt and embarrassment: an fMRI study. *Neuroimage*, *23*, 967–74.

Takeuchi, H., Taki, Y., Nouchi, R., et al. (in press). Regional gray matter density is associated with achievement motivation: evidence from voxel-based morphometry. *Brain Structure and Function*.

Takeuchi, H., Taki, Y., Sassa, Y., et al. (2011). Regional gray matter density associated with emotional intelligence: evidence from voxel-based morphometry. *Human Brain Mapping*, *32*, 1497–510.

Tamura, M., Moriguchi, Y., Higuchi, S., et al. (2012). Neural network development in late adolescents during observation of risk-taking action. *PLoS One*, *7*, e39527.

Tangney, J.P., Stuewig, J., Hafez, L. (2011). Shame, guilt and remorse: implications for offender populations. *Journal of Forensic Psychiatry and Psychology*, *22*, 706–23.

Tiihonen, J., Rossi, R., Laakso, M.P., et al. (2008). Brain anatomy of persistent violent offenders: more rather than less. *Psychiatry Research*, *163*, 201–12.

Tsujimoto, S., Genovesio, A., Wise, S.P. (2011). Frontal pole cortex: encoding ends at the end of the endbrain. *Trends in Cognitive Sciences*, *15*, 169–76.

van Rijn, S., Swaab, H., Baas, D., de Haan, E., Kahn, R.S., Aleman, A. (2012). Neural systems for social cognition in Klinefelter syndrome (47,XXY): evidence from fMRI. *Social Cognitive and Affective Neuroscience*, *7*, 689–97.

Venables, N.C., Patrick, C.J., Hall, J.R., Bernat, E.M. (2011). Clarifying relations between dispositional aggression and brain potential response: overlapping and distinct contributions of impulsivity and stress reactivity. *Biological Psychology*, *86*, 279–88.

Vitale, J.E., Brinkley, C.A., Hiatt, K.D., Newman, J.P. (2007). Abnormal selective attention in psychopathic female offenders. *Neuropsychology*, *21*, 301–12.

Vloet, T.D., Konrad, K., Huebner, T., Herpertz, S., Herpertz-Dahlmann, B. (2008). Structural and functional MRI- findings in children and adolescents with antisocial behavior. *Behav Sci Law*, *26*, 99–111.

Vogt, B.A. (2005). Pain and emotion interactions in subregions of the cingulate gyrus. *Nature Reviews Neuroscience*, *6*, 533–44.

White, S.F., Williams, W.C., Brislin, S.J., et al. (2012). Reduced activity within the dorsal endogenous orienting of attention network to fearful expressions in youth with disruptive behavior disorders and psychopathic traits. *Development and Psychopathology*, *24*, 1105–16.

Wu, D., Zhao, Y., Liao, J., Yin, H., Wang, W. (2011). White matter abnormalities in young males with antisocial personality disorder—evidence from voxel-based morphometry-diffeomorphic anatomical registration using exponentiated lie algebra analysis. *Neural Regeneration Research*, *6*, 1965–70.

Wunderlich, L., Dayan, P., Dolan, R.J. (2012). Mapping value based planning and extensive trained choice in the human brain. *Nature Neuroscience*, *15*, 786–93.

Yamasue, H., Iwanami, A., Hirayasu, Y., et al. (2004). Localized volume reduction in prefrontal, temporolimbic, and paralimbic regions in schizophrenia: an MRI parcellation study. *Psychiatry Research*, *131*, 195–207.

Yamasue, H., Kasai, K., Iwanami, A., et al. (2003). Voxel-based analysis of MRI reveals anterior cingulate gray-matter volume reduction in posttraumatic stress disorder due to terrorism. *Proceedings of the National Academy of Sciences of the United States of America*, *100*, 9039–43.

Yang, Y., Raine, A., Narr, K.L., Colletti, P., Toga, A.W. (2009). Localization of deformations within the amygdala in individuals with psychopathy. *Archives of General Psychiatry*, *66*, 986–94.

Yang, Y., Raine, A., Lencz, T., Bihrle, S., LaCasse, L., Colletti, P. (2005). Volume reduction in prefrontal gray matter in unsuccessful criminal psychopaths. *Biological Psychiatry*, *57*, 1103–8.

Zeier, J.D., Maxwell, J.S., Newman, J.P. (2009). Attention moderates the processing of inhibitory information in primary psychopathy. *Journal of Abnormal Psychology*, *118*, 554–63.