

Brain Processing of Fearful Facial Expression in Mentally Disordered Offenders

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Abstract

Emotional facial expressions are important cues for interaction between people. The aim of the present study was to investigate brain function when processing fearful facial expressions in offenders with two psychiatric disorders which include impaired emotional facial perception; autism spectrum disorder (ASD) and psychopathy (PSY). Fourteen offenders undergoing forensic psychiatric assessment (7 with ASD, and 7 psychopathic offenders) and 12 healthy controls (HC) viewed fearful and neutral faces while undergoing functional magnetic resonance imaging (fMRI). Brain activity (fearful versus neutral faces) was compared both between HC and offenders and between the two offender groups (PSY and ASD). Functional co-activation was also investigated. The offenders had increased activity bilaterally in amygdala and medial cingulate cortex as well as the left hippocampus during processing fearful facial expressions compared to HC. The two sub-groups of offenders differed in five regions compared with each other. Results from functional co-activation analysis suggested a strong correlation between the amygdala and anterior cingulate cortex (ACC) in the left hemisphere only in the PSY group. These findings suggest enhanced neural processing of fearful faces in the amygdala as well as in other facial processing brain areas in offenders compared to HC. Moreover, the co-activation between amygdala and ACC in the PSY but not the ASD group suggested qualitative differences in amygdala activity in the two groups. Since the sample size is small the study should be regarded as a pilot study.

Keywords: Psychopathy, Autism Spectrum Disorder, Offenders, fMRI, Emotional Facial Processing

1. Introduction

Emotional facial expressions are unique cues, crucial for social interaction [1]. They convey information about how another individual feels, as well as the motivations and intentions of others. This increases our ability to predict the behavior of other persons and also possible threats, thus facilitating adjustment of our own behavior [2]. Impairment in perceiving emotional facial expressions can lead to strange and inappropriate behavior and may also lead to antisocial behavior. A meta analysis of antisocial populations and emotional facial recognition suggested a robust link between antisocial behavior and specific

deficits in recognizing fearful expressions [3].

Psychopathy, as defined by Hare [4], and autism spectrum disorder (ASD) [5] are two developmental disorders with impairments in emotional facial expression processing [6-9]. Psychopathy has been associated with increased risk of offending behavior [10] and ASD has been suggested to be overrepresented in forensic psychiatric settings [11,12].

ASD includes impairments in mentalizing, emotional reciprocity, communication, and social interaction [5]. Also impairment in perceiving emotional facial expressions has been suggested. Brain imaging studies have shown that subjects described as high-functional autism

(HFA) have showed parallels to subjects with amygdala damage concerning facial perception [9,13] and in other studies it have been showed that autistic subjects use alternative pathways in the brain when processing emotional facial expressions [14,15]. In comparison to healthy individuals who commonly focus on the eye-region when looking at human faces, these individuals seem to focus on other parts of the face, for example on the mouth [16]. Psychopathic subjects often present a good, though superficial, ability to understand social signals [17]. Despite this, it has been shown that these subjects often have specific impairments in recognizing sad and fearful facial expressions [18,19], as well as more global impairment in identifying facial expressions [6]. Regardless of the fact that both groups (ASD and PSY) demonstrate impairment in recognizing emotional facial expressions, the different clinical symptoms shown in these two groups could possible correspond to recruitment of different brain networks when processing various facial expressions. Blair has discussed Asperger syndrome and psychopathy in this respect and suggests that the amygdala could be the key structure here [20, 21].

A specific neural network for processing human faces has been suggested [22]. This network consists of brain regions processing static information, such as the shape and size of the face (including inferior occipital gyrus, lateral fusiform gyrus, and superior temporal sulcus), and areas specific for other aspects of facial perception. In this network, the amygdala has a specific role in processing emotional facial expression. It is not clarified whether there is differential neural processing of fearful faces in psychopathic offenders as compared to autistic offenders.

In the present functional magnetic resonance imaging (fMRI) study, we contrasted fearful and neutral faces [23] in a blocked design to study differences in cerebral activation between offenders with ASD or psychopathy and a healthy control group. In many previous studies on criminal offenders the inclusion criteria for subjects studied have varied substantially [24], so in the present study, we addressed this specific problem and were very restrictive in the inclusion criteria, in order to select well defined subjects, resulting in small but fairly homogeneous study groups. To the best of our knowledge, this is the first fMRI-study to compare offenders with ASD with psychopathic offenders in the forensic psychiatric setting, and the present study should be regarded as a pilot study.

We hypothesized that the offender group would exhibit a different pattern of neural activity in the network processing fearful facial expressions, compared to controls. We also hypothesized that the two subgroups of

offenders would differ from each other in this network, especially in amygdala activation, and in functional coactivation between the amygdala and other regions within the network.

2. Material and Methods

2.1. Subjects

The sample consisted of 26 right-handed male subjects; 14 offenders (7 with ASD and 7 psychopathic offenders) undergoing forensic psychiatric assessment, and 12 healthy non-criminal controls. There were no age differences between the groups (**Table 1**).

Healthy controls (HC) were recruited from posters advertised at a nearby hospital. Offenders were recruited from the Department of Forensic Psychiatry in Stockholm. In Sweden major forensic psychiatric assessments are performed after the court has found evidence for the crime but before conviction. In this unit, approximately 280 forensic psychiatric assessments of inmates in custody are performed each year, 10% of inmates are female, and of the remaining 90%, approximately 5% have marked psychopathic traits (*i.e.* Psychopathy Check List Revised, PCL-R [4] score > 30) and a further 10% are diagnosed with autism spectrum disorder. The study subjects were recruited consecutively during, in all, 24 months.

Inclusion criteria for the offenders were either a main diagnosis of autism spectrum disorder (ASD) or psychopathy (PSY), as defined by Hare with PCL-R scores > 30 [4]. Exclusion criteria were axis I diagnoses, difficulties in understanding Swedish or acute psychosis at the time of assessment.

All subjects underwent an interview including the Structured Clinical Interview for DSM-IV (SCID I) Screening Interview [25,26], Asperger Syndrome Diagnostic Interview, (ASDI) [27], and Psychopathy Check List Screening Version (PCL-SV) [28]. Age, intelligence quotient (IQ) according to the Wechsler Adult Intelligence Scale-Revised (WAIS-R) [29] and medication were collected from the forensic psychiatric reports (**Table 1**). After the MR-scans, all participants performed State Trait Anxiety Inventory State (STAI-S) [30] in order to measure how stressful the situation was perceived. One subject in the ASD group was HIV-positive without clinical symptoms, structural brain damage, and neuropsychological dysfunction. All analyses including the ASD group were performed both with and without this subject, and the results remained stable.

The study was approved and conducted in accordance with ethical guidelines established by the Regional Ethical Committee at the Karolinska Institutet in Stockholm.

Table 1. Demographic and clinical characteristics. Mean value, SD in parenthesis. Mann-Whitney U-test was used in comparisons between groups.

	HC	Offenders	ASD	PSY
Age	28.1 (8.2)	28.6 (6.9)	29.9 (4.9)	27.3 (4.9)
IQ	-	98.1 (17.7)	100 (22.3)	94.8 (4.3)
PCL-SV*	0.5 (0.5)	15.1 (5.5)	10.6 (3.5)	19.7 (2.1)
STAI-S	32.6 (11.6)	42.4 (16.4)	41.5 (19.1)	43.1 (15.4)

*Significant differences in HC vs Offenders: $p < 0.0001$, and PSY vs ASD: $p < 0.0001$; HC = healthy controls; ASD = offenders with autism spectrum disorder; PSY = psychopathic offenders; IQ = intelligence quotient; PCL-SV = Psychopathy Checklist—Screening Version; STAI-S = State Trait Anxiety Inventory-State.

After description of the study, written informed consent was obtained.

2.2. Statistical Analysis of Demographic Variables

Age, IQ, PCL-SV scores, and STAI-S scores are presented as means (SD) and tested with Mann Whitney U-test. The confidence interval was set at 95%, and the level of statistical significance of differences was $p < 0.05$.

2.3. PCL-SV Scoring

The PCL-SV was performed in all study subjects in order to make sure that no subjects in the ASD and the HC group had a high amount of psychopathic traits. The PCL-SV was used as the HC group consisted of non criminals. It has high correlation with the PCL-R [31,32] but is preferred for use in non-criminal settings. The PCL-SV rating was assessed by two independent raters and the cut off score for psychopathy in the PCL-SV was 18.

Inter-rater reliability was computed using the intra-class correlation coefficient (ICC) [33], which was calculated using a two-way mixed effects model. The single measure ICC was 0.98 (95% CI = 0.95 – 0.99, $n = 25$).

Two-tailed t -tests showed significant differences between the HC group and the offenders ($t = 9.93$, $df = 13.3$, $p < 0.0001$). The same analysis was performed between the two subgroups of offenders (PSY vs AUT) ($t = 5.95$, $df = 9.7$, $p < 0.0001$). For mean scores see **Table 1**.

2.4. Stimuli

The stimuli comprised photographs of human facial expressions from the standardized Ekman and Friesen face set [23]. A blocked design was used which consisted of alternating blocks of fearful and neutral faces. Each block

consisted of 15 different faces, presented for 2 seconds each, followed by a fixation cross for 400 ms.

The 36 sec ‘face-blocks’ were interspersed with 18 sec ‘rest-blocks’ with a white fixation cross on a black screen. The subjects were asked to identify the sex (male or female) of the face by pressing buttons with the right index finger. The pictures were presented with a projector on a screen and the subjects viewed the pictures through a mirror on the head coil.

2.5. Behavioral Data

Mean reaction time and accuracy according to the sex discrimination task were used as a proxy for attention to the faces. Data are presented as means (SD) and comparisons between groups were performed using two-tailed t -test. Behavioral data was missing for one subject in the ASD group due to technical problems.

2.6. MRI Acquisition

The MR-scans were acquired with Siemens Avanto 1.5 T whole body MRI system, with a 12-channel matrix head coil. The subjects were placed supine in the scanner wearing headphones to reduce noise from the machine, and their heads were fixated with a vacuum pillow.

Functional imaging was performed using a T2*-weighted gradient echo planar imaging sequence (EPI)-mosaic sequence (TR = 3000 ms, TE = 50 ms, slice thickness = 5 mm, gap between slices = 0.5 mm, FOV = 220 mm, matrix size = 64 × 64, voxel dimension = 3.4 mm × 3.4 mm × 5 mm, 30 coronal slices, covering the whole brain), 114 volumes were collected. For structural data we used a 3D magnetization-prepared rapid-acquisition gradient echo (MP-RAGE) sequence, 176 sagittal slices were acquired with the following parameters: repetition time (TR) = 2300 ms, inversion time (TI) = 1100 ms, echo time (TE) = 3.93 ms, slice thickness = 1 mm, field of view (FOV) = 256 mm × 256 mm, Matrix =

256 × 256, isotropic voxel size = 1 mm³.

2.7. fMRI-Analyses

The first three volumes in each run were discarded to allow for T1 equilibration effects. Image time-series analysis was performed using Brain voyager, BVQX 1.9. Preprocessing comprised a correction of slice scan time, 3D motion correction (Trilinear/sinc interpolation) and temporal filtering removing linear trend. The images of each subject were co registered to the 3D anatomical volume, normalized into Talairach space, and hereby re-sample into 3 mm isotropic voxels. The resulting volume-time course files (VTC) were then spatially smoothed with a Gaussian filter of 8 mm full-width at half maximum for the group analyzes.

We chose the following anatomical region of interests (ROIs), from the model for facial perception by Haxby *et al.* [22]; inferior occipital gyrus, superior temporal sulcus, lateral fusiformis gyrus, insula, hippocampus, parahippocampus, cingulate gyrus and amygdala. Within these predefined areas, we used a threshold level of $p < 0.001$ (uncorrected) in line with many other studies in the field. Because the amygdala has been shown in many studies to be of major importance for fearful facial processing [34], we used a threshold of $p < 0.05$ (uncorrected) in this specific ROI. We also performed a whole brain analysis, in which we used a threshold level of $p < 0.05$ (FDR-corrected).

Individual analysis; we investigated blood oxygen level dependent (BOLD) activation associated specifically with the processing of the emotional cues in fearful faces (*i.e.* fearful faces > neutral faces). This was done by applying a fixed effects model [35], with condition-specific stimulus boxcar functions, convolved with a gamma-kernel to model the hemodynamic response behaviour. Three predictors were entered into the design matrix: fear faces, neutral faces, and baseline (looking at a white fixation cross on a black screen). The outputs of the model are beta values for the different conditions.

Between-group analyses were performed between (1) the HC and the offenders and (2) between the two subgroups of offenders (ASD vs PSY). Random effects models were used for both within and between group analyses. The between group analyses were calculated in the following steps:

first all individual VTCs were created for the contrast fearful faces > neutral faces, then a two-tailed *t*-test was performed between the HC group and the offender group and finally the same procedure was performed between the ASD and the PSY groups.

2.8. Functional Co-Activation

The functional co-activation analysis was performed in the following steps: first functional ROIs in the right and the left amygdala in the offender group were defined based on the group contrast fearful faces > neutral faces, consisting of 8 voxles located around the peak activation voxel in each of the two regions (mean TAL: X = 21, Y = -11, Z = -11; X = -19, Y = -7, Z = -13).

Individual beta values from these specific ROIs were collected. The difference between the beta values for the fear condition minus the beta value for the neutral condition for each subject was used as a measurement of amygdala reactivity to fearful expressions. From the between group analyses (ASD vs PSY), we found five regions (**Table 3**) where the two offender groups differed from each other within the network for the processing facial expressions [22]. In all these five regions we defined functional ROIs, as described above for the amygdala, and extracted beta values from these ROIs as well. We then correlated amygdala reactivity with activity in these specific ROIs, using Pearsons correlation, on each side separately (right and left), within each of the two groups (ASD and PSY). Grubb's test was used to test for outliers [36].

3. Results

3.1. Behavioral Responses

There were no significant differences between groups in any of the behavioral responses (**Table 2**).

3.2. fMRI BOLD-Responses

3.2.1. Between-Group Comparisons of Fearful Versus Neutral Faces Contrast

The offender group, collapsed across diagnoses, compared to HC had higher activation in the amygdala bilat-

Table 2. Behavioral responses. Mean values and SD in parenthesis.

	HC	Offenders	ASD	PSY
Accuracy (%)	96.9 (2.9)	96.3 (3.4)	95.7 (3.7)	96.9 (3.3)
Reaction time, neutral (ms)	668.2 (89.7)	725.6 (97.9)	759.2 (130.6)	696.8 (53.5)
Reaction time, fear (ms)	671.7 (111.1)	686.5 (74.9)	703.7 (98.8)	671.8 (50.3)

HC = healthy controls; ASD = offenders with autism spectrum disorder; PSY = psychopathic offenders.

Table 3. Between-group comparisons. The contrast fear > neutral, $p < 0.001$ (uncorrected).

Region	Brodmann's Area	Hemisphere	X	Y	Z	Numbers of voxels	t-value
Offenders > HC							
Amygdala	Amygdala	L	-19	-7	-13	118	2.22
Amygdala	Amygdala	R	21	-11	-11	126	2.18
Cingulum	24	R	21	0	34	7	3.85
Cingulate gyrus	23	L	-23	23	27	44	3.93
Parahippocampus-Hippocampus	Hippocampus	L	-43	-33	-5	22	3.89
HC > Offenders							
No areas							
PSY > ASD							
Insula	13	L	-39	-7	-5	24	4.36
Anterior cingulate cortex	24	L	-4	27	17	19	4.40
ASD > PSY							
Insula	13	R	34	-25	0	258	4.61
Lingual gyrus/fusiform gyrus	18	L	-7	-91	-14	26	4.37
Cingulate gyrus	23	L	-15	-13	31	7	4.45

HC = healthy controls; PSY = psychopathic offenders; ASD = offenders with autism spectrum disorder.

erally, the left hippocampus as well as in the medial cingulate cortex bilaterally (**Table 3**).

In a comparison between the two subgroups of offenders (ASD vs PSY), there were two regions where the PSY group had significantly higher activation than the ASD group; anterior cingulate cortex (ACC), and insula on the left side (**Table 3, Figure 1**). The ASD group, on the other hand, had higher activation in the right insula and left cingulate cortex, and left fusiform gyrus (**Table 3**). In the whole brain analysis we did not find any significant differences in any of the between group analyses.

3.2.2. Functional Co-Activation

In the PSY group there was a correlation between the amygdala and the ACC on the left side ($r^{xy} = 0.97$, $p < 0.001$), which was not found in the ASD group ($r^{xy} = 0.327$, $p = 0.475$) (**Figure 2**). No other correlations with amygdala reactivity were significant in any of the two groups.

4. Discussion

The present study investigated the neural underpinnings of emotional facial perception in offenders with ASD or psychopathy. The whole offender group had increased BOLD activity, compared to the HC group, in specific

nodes in the neural network involved in perceiving and processing facial information [34] namely bilateral amygdala, cingulate gyrus, and left hippocampus. In studies applying brain imaging techniques to other groups of antisocial subjects, functional impairments in the frontal lobes [37] and the limbic system [38,39] have been demonstrated, and a suggested impairment in the balance between these two systems has been described [40-42]. These studies have reported both hypoactivity [38,43] and hyperactivity [40,42] in the amygdala and the limbic system compared to healthy controls.

Hyper activation in amygdala in the current offender group could reflect an imbalance between the limbic system and more frontal systems located outside of the network processing facial expressions; it could also be related to alteration in the way fearful facial expressions are processed in these specific groups. Since both of these groups (PSY and ASD) have shown impairments in recognizing fearful facial expressions before [3,6,9,44], the enhanced amygdala activity may be related to ambiguity in the processing of the emotional face signal [45]. If so, this ambiguity could be related to enhance cognitive elaborative processing associated with exposure to fearful faces in the offender group, which could explain the activations also in more "cognitive regions", such as the medial cingulate gyrus and the hippocampus.

In the contrast between the two offender groups (ASD

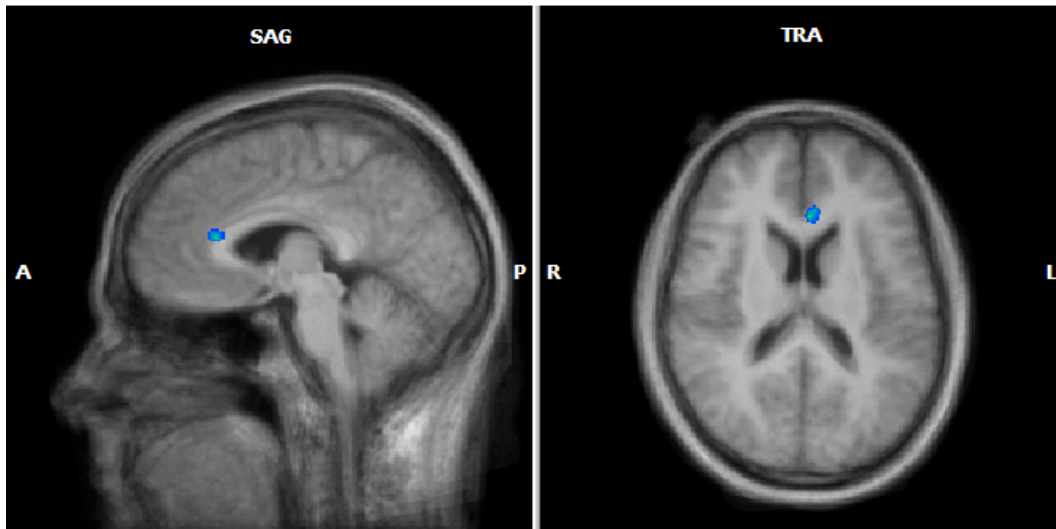


Figure 1. Between-group analyses. Blue dot illustrates PSY vs ASD in the contrast fearful > neutral faces; PSY > ASD in ACC (mean TAL-coordinates: $-4, 27, 17$), p -value < 0.01 (corrected). This region was correlated with amygdala in the PSY-group. PSY = psychopathic offenders; ASD = offenders with autism spectrum disorder syndrome; ACC = anterior cingulate cortex.

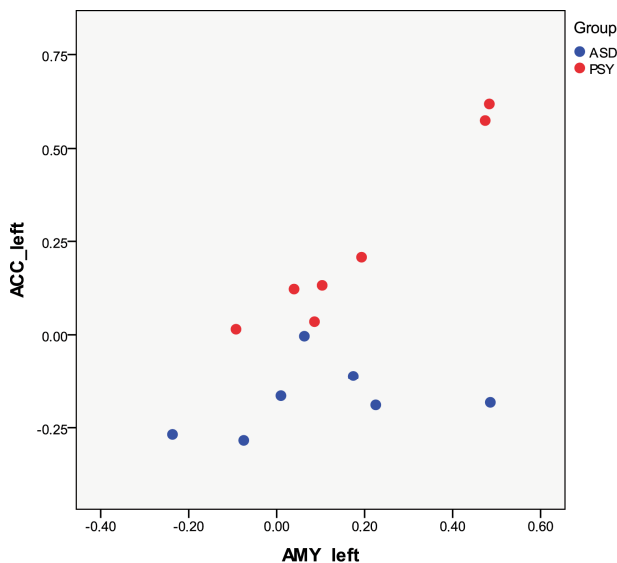


Figure 2. Functional co-activation. Correlation between reactivity in the amygdala (AMY) and in the anterior cingulate cortex (ACC), on the left side in the group of psychopathic offenders, PSY, ($r^{xy} = 0.97$, $p < 0.001$), but no significant correlation in the group of offenders with autism spectrum disorder, ASD, ($r^{xy} = 0.327$, $p = 0.475$).

vs PSY) we observed differential activations in both the insular cortex (left vs right) and the cingulate gyrus. Hence, ASD and PSY seem to have differential activation patterns within these two cortical regions during perception of fearful faces. The insula cortex has been connected to visceral representation of autonomic arousal [46,47]. Because the ASD-group activated the right insula more than the PSY-group this could reflect

that exposure to fearful facial expressions is associated with more bodily arousal in the ASD-group compared to the PSY-group. This reasoning is in line with earlier studies showing that psychopathic subjects have lower autonomic responses during exposure to emotionally relevant information [43,48-52]. The PSY-group had higher activation in the ACC compared to the ASD-group which could be connected to the possible inhibition of amygdala reactivity. The ASD-group, on the other hand, had higher activity in the posterior cingulate gyrus, Brodmann area 23. The posterior cingulate cortex has been connected to both emotional processing and memory-related functions [53-55]. Accordingly, the higher posterior cingulate activation in the ASD-group could reflect altered emotional or memory related processing of fearful facial information compared to the PSY-group.

Our third main result is a strong correlation between amygdala reactivity and ACC activity in the PSY group on the left side, which was not found in the ASD group. This suggests that the amygdala activation in the two offender groups may differ qualitatively, even though there were no quantitative differences. The ACC influences emotional processing [56] and is suggested a role in modulating the amygdala [57]. A possible explanation in the current study is that the ACC in the PSY-group inhibits the amygdala reaction, resulting in impaired emotional information processing. Hence, the amygdala-ACC correlation could reflect a marked ACC influence on fearful emotional facial processing in the amygdala resulting in the disturbed fearful facial processing pattern often seen in psychopathic subjects [6,7]. An important note is that it is not possible to tell anything about the

direction of the co-activation with the current analysis.

Some limitations have to be mentioned. Firstly, the sample size in the subgroups was small, thus increasing the risk of type II error, and the present study should be regarded as a pilot study. Secondly, five of the offenders and one HC were on medication. It was not possible to withdraw all medication, as the study was conducted in a clinical context. Some offenders had a history of drug and alcohol misuse prior to arrest; as they were remanded in custody, all of the offenders had been free from drugs and alcohol for at least 6 - 10 weeks prior inclusion. Finally, the use of HC as a comparison group to the offenders involves some problems; HC consisted of non criminals who probably have different socioeconomic status than the offenders, they all had completed high school education and were not tested with WAIS-R.

In summary, our findings indicate altered neural processing of fearful facial expressions in the offender group compared to HC within the neural network involved in processing facial information [22]. Moreover, the two subgroups of offenders differed from each other in direct or indirect functional communication between the amygdala and ACC, both located within the face processing network. The behavioral relevance of these differences is however unclear. Facial processing most certainly interacts with our decision making in various situations and is one important factor in the development of empathy [58]. Lack of empathy is a common trait in offenders [58]. Both psychopathy [4] and ASD [5] have been associated with reduced empathic ability. Empathy is a construct consisting of many aspects; for example emotional and cognitive empathy. While cognitive empathy or theory of mind requires mentalizing, emotional empathy includes the ability to interpret emotional facial expressions. In ASD the cognitive empathy is affected, which have not yet been found in psychopathy. Blair discussed these two disorders and their differences regarding the empathic ability. He points out their different dysfunctions, both regarding empathy deficit and differences in amygdala, and suggests that a “fine cut” between autism and psychopathy can be made in both amygdala and in the different aspects of empathy, emotional and cognitive empathy [20,21]. In future studies, brain activation patterns to facial expressions in subjects with autistic and psychopathic traits, with and without offending behavior, ought to be compared and related to measures of empathy and its different aspects.

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