Psychiatry Research: Neuroimaging 183 (2010) 225–229

Contents lists available at ScienceDirect

Psychiatry Research: Neuroimaging

journal homepage: www.elsevier.com/locate/psychresns

Autobiographical memories of anger in violent and non-violent individuals: A script-driven imagery study

Michele R. Spoont[a,*], Michael Kuskowski[b], Jose V. Pardo[c]

[a]Center for Chronic Disease Outcomes Research, 3E/111-0, VA Medical Center, One Veterans Drive, Minneapolis, MN 55417
[b]Geriatrics Research Education and Clinical Center, VA Medical Center, One Veterans Drive, Minneapolis, MN 55417
[c]Mental and Behavioral Health 116A, VA Medical Center, One Veterans Drive, Minneapolis, MN 55417

ARTICLE INFO

Article history:
Received 15 September 2009
Received in revised form 10 May 2010
Accepted 8 June 2010

Keywords:
PET
Mood-induction
Frontal lobes

ABSTRACT

Numerous studies have implicated frontal lobe dysfunction in anger-related impulsive violent behavior; however, few studies have looked at frontal activity during angry states in violent individuals. Using PET and a script-driven imagery paradigm, we report on autobiographical memories of angry vs. neutral memories in violent patients and psychiatric matched controls. Relative to recall of neutral memories, recall of anger-laden memories was associated with an activation of frontal regions among control subjects but not violent subjects. Violent subjects demonstrated relatively greater activations in the left amygdala, pontine, and cerebellar regions compared to control subjects.

Published by Elsevier Ireland Ltd.

1. Introduction

A growing convergence of evidence has implicated frontal lobe dysfunction in impulsive and violent behavior. For example, some murderers have decreased prefrontal glucose metabolism during an attentional task (Raine et al., 1998), and decreased left orbital frontal gray matter volume has been associated with aggressive behavior among psychiatric patients (Gansler et al., 2009). Impaired functioning of the ventromedial and medial frontal cortices may contribute to the expression of violent behavior by either poor modulation of amygdala-related negative affective states (Coccaro et al., 2007; New et al., 2007, 2009; Silbersweig et al., 2007), and/or by alteration in ability to perform moral judgments and to select alternate behavioral responses (Lawrence et al., 2009; Young et al., 2010).

Although several studies have found altered frontal functioning in individuals who engage in impulsive violent behavior, few studies have examined frontal regions in people who commit affectively-driven violent acts during an affectively charged state. Dougherty et al., 2004, found that depressed patients who have anger attacks have reduced activation of ventromedial and medial frontal regions relative to healthy controls during a script-driven anger induction task. More recently, individuals with impulsive violent behavior have been shown to have reduced activations in orbital frontal regions relative to healthy controls during an anger-provoking decision making task (New et al., 2009) and when processing angry faces (Coccaro et al., 2007). In this study, we examined neural correlates of an angry mood by using a script-driven imagery paradigm of autobiographical memories of intense anger among subjects with histories of impulsive violent behavior compared to patients without violent histories who were matched with violent subjects on Axis I diagnoses and handedness. We were specifically interested in whether inferior frontal regions decreased in violent subjects during recall of anger-related autobiographical memories.

2. Methods

2.1. Subjects

Potential subjects were solicited by advertisement, screened by phone for appropriateness and given a description of the study. Those who were eligible and interested were seen for in-person administration of the Structured Clinical Interview for DSM-IV (parts I and II; First et al., 1996, 1997) and the Brown -Goodwin Life History of Aggression Scale (Brown et al., 1979). To be eligible, violent subjects had to have at least one physically violent act within the prior year, a history of repetitive violent behavior that began by early adolescence, experienced legal consequences due to violent behavior, and met criteria for either a Cluster B personality disorder or Intermittent Explosive Disorder (APA, 1994). Control subjects had no history of violent behavior and matched violent subjects on Axis I disorders, age, handedness and use of psychotropic medications. All subjects were males between 18-55 years old. Excluded were those with psychotic

* Corresponding author. Tel.: +1 612 467 1428; fax: +1 612 727 5699.
E-mail address: michele.spoont@va.gov (M.R. Spoont).

0925-4927/$ – see front matter. Published by Elsevier Ireland Ltd.
doi:10.1016/j.pscychresns.2010.06.004

Table 1
Neutral autobiographical memory -Rest Condition comparisons between violent subjects and non-violent psychiatric controls.

Violent > Control Subjects						Control > Violent Subjects					
	X	Y	Z	Z-score (voxels)	BA		X	Y	Z	Z-score (voxels)	BA
M. Frontal G.	-45	34	34	4.72 (308)	**9/46/8**						
Inf. Frontal G.	-58	22	22	3.45 (29)	**45**	**Inf. Frontal G.**	-25	9	-14	4.81 (267)	**47**
	54	18	22	3.35 (98)	**9**						
Precentral G.	47	-14	43	3.71 (279)	**4/6**						
Cingulate G. /M. Frontal G.	7	-7	27	3.14 (235)	**24/6**	**Anterior Cingulate**	-2	16	-7	4.15 (226)	**25**
Insula	-36	4	11	3.90 (210)	**13**						
						M. Temporal G.	58	-25	-14	3.17 (80)	**21**
						Parahippocampal G. / Hippocampus	-25	-27	-4	3.67 (58)	**27**
Precuneus	-18	-68	27	3.34 (139)	**7**						
Cuneus	0	-81	32	3.05 (134)	**19**						
						Pons	-2	-18	-22	3.47 (147)	
						Cerebellum	20	-86	-27	3.73 (103)	
							43	-70	-29	3.24 (79)	

Notes:. Coordinates and Z-scores represent the maximum voxel values within the activated region.
X<0 is left of the midsagittal plane.

disorders, active substance abuse/dependence, seizure disorders, gross traumatic brain injury, or violent behavior only while intoxicated. The study was approved by the Minneapolis VAMC institutional review board and the Radioactive Drug Research Committee of the FDA. All subjects provided informed consent. A Certificate of Confidentiality (U.S. Department of Health and Human Services; MH-97-90) was acquired to ensure subjects' confidentiality about criminal activity.

2.2. Autobiographical Memories

In a separate appointment prior to imaging, subjects were interviewed about memories associated with intense anger and those that were affectively neutral. For each subject, two angry and two neutral memories were developed into brief scripts that were used in the mood-induction protocol using script-driven imagery. For violent subjects, angry memories were characterized by violent behavior (i.e., assault).

2.3. PET imaging

Subjects were scanned in a rest condition with their eyes closed, followed by presentation of previously prepared autobiographical scripts (i.e., script-driven memory conditions). Scripts of autobiographic memories associated with either intense anger (ANGER) or neutral affect (NEUTRAL) were read to the subject while in the scanner immediately before initiating measurement of brain blood flow. There were two scans of each condition per subject administered in a counterbalanced order. Imaging used a Siemens ECAT 953B camera in 3D mode. Image reconstruction used a filtered back-projection (0.5 cycles/pixel Hanning filter). A slow bolus infusion of $H_2^{15}O$ (average initial dose of 0.25 mCi/kg) was used, and rCBF was estimated from normalized tissue activity (measured attenuation corrected) and integrated over 90 s. Images were smoothed with a Gaussian filter, and final image resolution was 9 mm FWHM. Software by Minoshima et al., 1994, permitted image registration, ACPC determination, and nonlinear warping to Talairach space.

2.4. Analyses

Regional CBF was calculated by voxelwise subtraction of inter-subject averaged images from the contrasts between anger and neutral autobiographical memory conditions (ANGER - NEUTRAL). Violent subjects and controls were compared to ascertain significant group differences in the rest condition (RC), and for the ANGER - NEUTRAL subtraction. Foci were considered significant if the cluster contained at least 25 contiguous voxels, and the z-score of the peak voxel was at least 3.0 (p-value of ≤ 0.001) to account for multiple comparisons. The Talairach Daemon client (Lancaster et al., 1997, 2000) was used to identify anatomical labels (i.e., gyri and Brodmann areas) associated with coordinates of maximum and submaximum peak voxels.

3. Results

There were 8 subjects in each group. There were no significant differences between groups in age (violent = 44.25 ± 9.3 yo; control = 42.4 ± 9.2 yo), education (violent = 13.3 ± 1.5 yrs; control = 15 ± 1.8 yrs), or history of chemical dependency (all p's > 0.05). Three subjects from each group were on antidepressants. Two of the violent subjects on antidepressants were also prescribed mood stabilizers. Six of each group were right handed. Two violent subjects were left handed, and one control subject was left handed and one ambidextrous. Of the violent subjects, six met criteria for antisocial personality disorder, one for borderline personality disorder and one for intermittent explosive disorder. Of the four violent subjects who had major depression, two also had posttraumatic stress disorder. Among controls subjects, four had major depression, one had posttraumatic stress disorder, and one who had major depression also met criteria for anxiety disorder NOS with posttraumatic features.

Table 2
Anger-related autobiographical memory – Rest Condition comparisons between violent subjects and non-violent matched psychiatric controls.

Violent Subjects > Controls						Controls > Violent Subjects					
	X	Y	Z	Z-score (voxels)	BA		X	Y	Z	Z-score (voxels)	BA
						Inf. Frontal G. / M. Frontal G.	-36	20	-18	3.44 (405)	**47**
						Cingulate G.	25	-43	36	3.41 (39)	**31**
						S. Temporal G.	-63	-16	0	3.21 (36)	**22**
Inf. Parietal L.	61	-36	45	3.20 (49)	**40**	**Fusiform G.**	47	-20	-20	3.52 (125)	**20**

Notes:. Coordinates and Z-scores represent the maximum voxel values within the activated region.
X<0 is left of the midsagittal plane.

There were no significant differences between violent and control subjects during the RC (all z-scores<3.0). In the group comparison (double subtraction) of NEUTRAL- RC, violent subjects activated more medial frontal regions than control subjects (see Table 1; within subject contrasts are available in the online Supplementary Table 1). Additionally, whereas control subjects had greater activations of cerebellar and medial temporal lobe areas, violent subjects had greater activations in the left insula and areas associated with visual imagery.

Fewer significant differences emerged in the group comparisons of the affectively laden ANGER condition relative to RC (see Table 2; within subject contrasts are available in the online Supplementary Table 2). As predicted, control subjects had greater activations in medial and inferior frontal regions.

The primary comparison of interest, ANGER-NEUTRAL, can be seen in Tables 3 and 4 and Fig. 1. The within group comparisons in Table 3 are most notable for suppression of inferior frontal regions among violent subjects and activation of inferior frontal regions among controls, confirming our hypothesis. Additionally, violent subjects had greater activations in lentiform/caudate nuclei, midbrain and cerebellar regions during ANGER; whereas, in controls subjects these regions were relatively suppressed (midbrain, cerebellum), or the activation was much more circumscribed (lentiform). The group comparison (Table 4) further validates the greater frontal activations in control subjects, and reveals activations in violent subjects of the left amygdala, pontine and cerebellar areas.

4. Discussion

As predicted, and consistent with other studies, we found greater middle and inferior frontal cortex activations in controls relative to violent subjects during recall of ANGER autobiographical memories compared to affectively NEUTRAL memories. Of interest, across tasks, violent subjects had much less inferior frontal activations relative to controls. When violent subjects demonstrated some inferior frontal activation (e.g., during NEUTRAL – RC), the activation area was more lateralized and more circumscribed. These group differences in medial

Table 3
Within subject ANGER -. NEUTRAL autobiographical memory comparisons in violent subjects and non-violent matched psychiatric controls.

Violent Subjects						Controls Subjects					
	X	Y	Z	Z-score* (voxels)	BA		X	Y	Z	Z-score (voxels)	BA
S. Frontal G.	27	47	36	3.84 (51)	**9**	**S. Frontal G.**	11	38	50	3.26 (45)	**8**
							-34	56	32	3.20 (45)	**9/10**
Anterior Cingulate/ M. Frontal G.	16	43	-7	3.91 (41)	**32/10**	**M. Frontal G.**	-47	36	36	4.05 (65)	**9**
						Inf. Frontal G./S. Temporal G.	-54	20	-11	4.95 (936)	**47/38/22**
Subcollosal Cingulate G.	-2	11	-16	3.85 (142)	**25**						
Caudate	11	9	9	4.25 (111)							
Caudate/Lentiform N	-9	4	7	4.16 (222)		**Putamen/Lentiform**	-14	0	9	4.33 (64)	
Insula	38	9	9	3.64 (55)	**13**	**Claustrum/Insula**	-27	16	11	3.75 (46)	
							-40	-7	16	3.60 (37)	**13**
							27	-20	18	3.42 (39)	
Midbrain/ Pons	0	-27	-14	3.30 (120)							
Cerebellum	0	-58	-20	4.23 (106)							
	-18	-88	-27	3.72 (209)							
	38	-54	-22	3.48 (142)							
S. Frontal G.	-34	63	-2	-3.11 (36)	**10**	**S. Frontal G.**	18	18	43	-3.49 (186)	**8/9**
Medial Frontal G.	22	72	2	-4.20 (40)	**10**	**M. Frontal G.**	34	38	0	-3.86 (203)	**10**
	61	18	32	-4.01 (133)	**9**		-9	45	4	-3.58 (27)	**10/32**
	-27	65	20	-3.02 (32)	**10**		-14	40	-11	-3.43 (32)	**10**
							-27	7	47	-3.33 (109)	**6**
Inf. Frontal G.	-40	36	0	-4.19 (313)	**46**						
	52	27	9	-3.67 (318)	**45**						
	-54	29	11	-3.13 (59)	**46**						
Precentral G.	-36	2	29	-3.36 (60)	**6**	**Precentral G.**	-47	-7	7	-3.48 (62)	**6**
						Anterior Cingulate	-7	9	27	-3.98 (125)	**24/33**
						Corpus Collosum/Anterior Cingulate	0	27	7	-3.02 (86)	**24/32**
						Posterior Cingulate/ Lingual G.	-9	-52	7	-4.62 (267)	**30/31/18**
						Caudate	29	-34	11	-3.31 (79)	
Postcentral G./ Inf. Parietal L./ Insula	61	-22	32	-3.74 (345)	**2/40/13**	**Insula**	40	-18	4	-5.08 (120)	**13**
Inf. Parietal L./ Postcentral G.	-58	-25	29	-4.16 (336)	**40/2**						
						Uncus	27	-11	-29	-4.06 (47)	**28**
							-27	-7	-27	-3.65 (204)	**28**
						Parahippocampal G.	-40	-32	-22	-3.18 (118)	**36**
						M. Temporal G.	52	-7	-20	-3.42 (27)	**21/20**
						Inf. Temporal G./ Inf. Occipital G.	-45	-70	2	-4.22 (114)	**37/18**
Cerebellum/ Fusiform and PHG	-36	-34	-27	-3.93 (1077)	**36/35/37**	**Fusiform G.**	47	-32	-16	-3.40 (138)	**20**
						Inf. Parietal L.	-50	-43	47	-4.06 (90)	**40**
							-29	-34	38	3.73 (190)	**40**
							43	-43	34	-3.33 (319)	**40**
						S. Parietal L.	27	-63	50	-3.17 (30)	**7**
Precuneus/ Cuneus/											
M. Occiptal G.	-4	-65	22	-3.98 (1595)	**31/7/18**	**Precuneus/ Cuneus**	-20	-72	50	-4.75 (234)	**7/19**
							2	-58	38	-3.11 (102)	**7**
						M. Temporal G./ M. Occipital G.	50	-76	16	-3.17 (50)	**19**
							9	-92	32	-3.07 (211)	**19**
							7	-81	16	-3.02 (37)	**18**
M. Occipital G.	32	-74	14	-3.33 (339)	**19**	**Inf. Occipital G.**	29	-88	-11	-3.25 (124)	**19/18**
						Midbrain/Pons	0	-22	-18	-3.42 (32)	
						Cerebellum	0	-74	-14	-3.66 (475)	

Notes: *Positive values are activations and Negative values are deactivations. Coordinates and Z-scores represent the maximum voxel values within the activated region. X<0 is left of the midsagittal plane.

Table 4
ANGER - NEUTRAL autobiographical memory group comparisons between violent subjects and non-violent psychiatric controls.

Violent > Control Subjects						Control > Violent Subjects					
	X	Y	Z	Z-score (voxels)	BA		X	Y	Z	Z-score (voxels)	BA
						S. Frontal G.	-29	47	34	3.44 (46)	**9**
						Medial Frontal G.	-56	20	27	3.76 (38)	**46/9**
							-43	34	2	3.20 (124)	**47**
							-9	-18	47	3.02 (64)	**6/31**
						Paracentral Lobule	14	-36	52	3.04 (41)	**5/4/3**
						Corpus Collosum/ Thalamus	2	-2	18	3.22 (25)	
Caudate	29	-34	14	3.09 (42)							
Lentiform N.	-20	0	2	3.56 (41)		**Claustrum/Thalamus**	25	-14	20	3.66 (74)	
Insula	40	2	0	3.46 (62)	**13**	**Insula/Claustrum**	-36	-4	9	3.13 (86)	**13**
Uncus/ Amygdala	-27	-4	-25	3.24 (43)	**28/36**	**Parahippocampal G. /Hippocampus**	20	-14	-16	4.21 (64)	**28/34/35**
							34	-25	-14	3.99 (221)	**34/36**
						S. Temporal G.	-65	-22	9	4.06 (50)	**22/42**
Precuneus	-20	-70	50	3.59 (26)	**7**						
						Fusiform G.	-36	-45	-11	3.59 (58)	**37**
Midbrain/ Pons	0	-22	-18	3.12 (53)							
Cerebellum	40	-58	-25	3.88 (244)		**Cerebellum**	7	-56	2	3.02 (27)	
	2	-58	-22	3.83 (136)							
	-18	-86	-27	3.20 (76)							

Notes: Coordinates and Z-scores represent the maximum voxel values within the activated region.
X<0 is left of the midsagittal plane.

inferior frontal activations across tasks provide additional support for the theory that individuals who engage in impulsive violent behavior have trait-wise dysfunction in inferior frontal cortex (Coccaro et al., 2007; Dougherty et al, 2004; New et al., 2007, 2009; Raine et al., 1998; Silbersweig et al., 2007). Recent studies suggest that frontal dysfunction releases aggressive behavior through poor modulation of the amygdala (Coccaro et al., 2007; New et al., 2007, 2009). Our study provides indirect support for this theory since in addition to an absence of inferior frontal activation in the ANGER- NEUTRAL comparison, violent subjects had relatively greater activation in the left amygdala. Of interest, a recent meta-analysis of imaging studies on laterality of emotions implicated the left amygdala in negative mood states (Fusar-Poli et al., 2009). Unfortunately, we did not have behavioral data to associate with activations in the amygdala.

Although many of the activations and deactivations in the NEUTRAL-RC were similar between the groups, violent subjects extensively activated medial and superior frontal regions. The medial frontal activations among violent subjects could reflect greater difficulty with the task relative to control subjects. If so, recruitment of frontal control areas could be viewed as a compensatory process for dysfunction in regions that would normally be operational in memory retrieval tasks (e.g., temporal cortices). Consistent with this interpretation, recent evidence suggests that patients with similar kinds of affective instability (i.e., borderline personality disorder) may need to recruit more cortical areas to achieve comparable performance in retrieval of episodic memories relative to those without such instability (Mensebach et al., 2009). Within this context, greater difficulty with memory retrieval could adversely affect decision making when anger states limit frontal involvement. Perhaps this is why violent subjects activated only a limited portion of the orbital cortex, and no other frontal areas, in the ANGER-RC comparison. This activation pattern has been interpreted by others (New et al., 2009) as possibly reflecting diminished higher cognitive control of anger-related behavior.

In contrast to control subjects, in the ANGER- NEUTRAL double subtraction, violent subjects demonstrated relatively greater activations in pontine and cerebellar regions. While the significance of these activations is not yet clear, it could be that the extensive cerebellar activations reflect compensatory activity to engage executive functions (Stoodley and Schmahmann, 2009) when cortical areas associated with executive functions are compromised.

Taken together, these findings suggest that violent subjects demonstrate a functional organization that is qualitatively different from non-violent subjects, and which may predispose them to more limited executive control of behavior during states of intense anger. The interpretation relies on the assumption that retrieval of an affectively charged memory is in some ways similar to the affective experience at the time the event occurred. Given recent data on

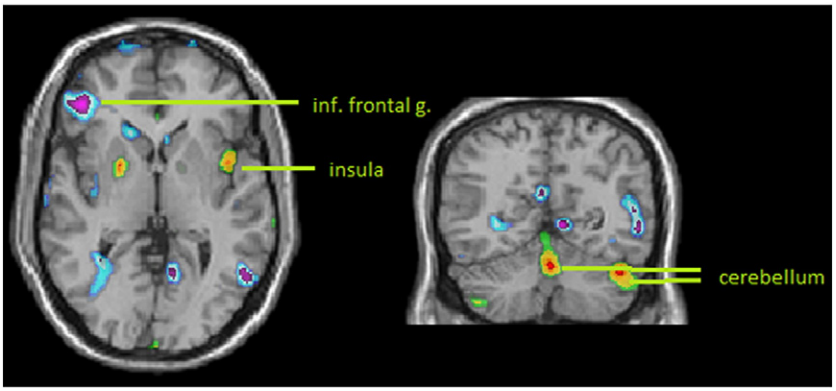

Fig 1. Violent subjects relative activations in insula vermis, and cerebellar hemispher with deactivation in inferior frontal cortex in the contrast between ANGER vs. NEUTRAL, script-driven, autobiographical imagery.

increased frontal activation during aggressive behavior in borderline patients (New et al., 2009), these findings are likely contextually specific and may not generalize to actual situations in which aggression is expressed.

Limitations of this study include small sample sizes, use of mood stabilizers in two violent subjects, lack of association of imaging with behavioral measures, and use of an exploratory approach to data analysis. Despite these limitations, this study adds to the growing evidence base for frontal dysfunction in violent behavior.

Acknowledgements

This study was supported by a grant from the Minnesota Medical Foundation awarded to the first author, and was supported with resources and the use of facilities at the Minneapolis VA Medical Center. The views expressed in this paper are those of the authors and do not necessarily reflect those of the Department of Veterans Affairs or the United States government.

Appendix A. Supplementary Data

Supplementary data associated with this article can be found, in the online version, at doi: 10.1016/j.pscychresns.2010.06.004.

References

American Psychiatric Association (APA), 1994. Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. APA, Washington, DC.

Brown, G.L., Goodwin, F.K., Ballenger, J.C., Goyer, P.F., Major, L.F., 1979. Aggression in humans correlates with cerebrospinal fluid amine metabolites. Psychiatry Research 1, 131–139.

Coccaro, E.F., McCloskey, M.S., Fitzgerald, D.A., Phan, K.L., 2007. Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. Biological Psychiatry 62, 168–178.

Dougherty, D.D., Rauch, S.L., Deckersbach, T., Marci, C., Loh, R., Shin, L.M., Alpert, N.M., Fischman, A.J., Fava, M., 2004. Ventromedial prefrontal cortex and amygdale dysfunction during an anger induction positron emission tomography study in patients with major depressive disorder with anger attacks. Archives of General Psychiatry 61, 795–804.

First, M.B., Spitzer, R.L., Gibbon, M., Williams, J.B.W., 1996. Structured Clinical Interview for DSM-IV Axis I Disorders, Clinician Version (SCID-CV). American Psychiatric Press Incorporated, Washington, D.C.

First, M.B., Gibbon, M., Spitzer, R.L., Williams, J.B.W., Benjamin, L.S., 1997. Structured Clinical Interview for DSM-IV Axis II Personality Disorders, (SCID-II). American Psychiatric Press Incorporated, Washington, D.C.

Fusar-Poli, P., Placentino, A., Carletti, F., Allen, P., Landi, P., Abbamonte, M., Barale, F., Perez, J., McGuire, P., Politi, P.L., 2009. Laterality effect on emotional faces processing: ALE meta-analysis of evidence. Neuroscience Letters 452, 262–267.

Gansler, D.A., McLaughlin, N.C., Iguchi, L., Jerram, M., Moore, D.W., Bhadelia, R., Fulwiler, C., 2009. A multivariate approach to aggression and the orbital frontal cortex in psychiatric patients. Psychiatry Research 171, 145–154.

Lancaster, J.L., Rainey, L.H., Summerlin, J.L., Freitas, C.S., Fox, P.T., Evans, A.C., Toga, A.W., Mazziotta, J.C., 1997. Automated labeling of the human brain: A preliminary report on the development and evaluation of a forward-transform method. Human Brain Mapping 5, 238–242.

Lancaster, J.L., Woldorff, M.G., Parsons, L.M., Liotti, M., Freitas, C.S., Rainey, L., Kochunov, P.V., Nickerson, D., Mikiten, S.A., Fox, P.T., 2000. Automated Talairach Atlas labels for functional brain mapping. Human Brain Mapping 10, 120–131.

Lawrence, N.S., Jollant, F., O'Daly, O., Zelaya, F., Phillips, M.L., 2009. Distinct roles of prefrontal cortex subregions in the Iowa gambling task. Cerebral Cortex 19, 1134–1143.

Mensebach, C., Beblo, T., Driessen, M., Wingenfeld, K., Mertens, M., Rulkoetter, N., Lange, W., Markowitsch, H.J., Ollech, I., Saveedra, A.S., Rau, H., Woermann, F.G., 2009. Neural correlates of episodic and semantic memory retrieval in borderline personality disorder: an fMRI study 171, 94–105.

Minoshima, S., Koeppe, R.A., Frey, K.A., Kuhl, D.E., 1994. Anatomic standardization: linear scaling and nonlinear warping of functional brain images. Journal of Nuclear Medicine 35, 1528–1537.

New, A.S., Hazlett, E.A., Buchsbaum, M.S., Goodman, M., Mitelman, S.A., Newmark, R., Trisdorfer, R., Haznedar, M.M., Koenigsberg, H.W., Flory, J., Siever, L.J., 2007. Amygdala-prefrontal disconnection in borderline personality disorder. Neuropsychopharmacology 32, 1629–1640.

New, A.S., Hazlett, E.A., Newmark, R.E., Zhang, J., Triebwasser, J., Meyerson, D., Lazarus, S., Trisdorfer, R., Goldstein, K.E., Goodman, M., Koenigsberg, H.W., Flory, J.D., Siever, L.J., Buchsbaum, M.S., 2009. Laboratory induced aggression: a positron emission tomography study of aggressive individuals with borderline personality disorder. Biological Psychiatry 66, 1107–1114.

Raine, A., Stoddard, J., Bihrle, M.A., Buchsbaum, M., 1998. Prefrontal glucose deficits in murderers lacking psychosocial deprivation. Neuropsychiatry, Neuropsychology, and Behavioral Neurology 11, 1–7.

Silbersweig, D., Clarkin, J.F., Goldstein, M., Kernberg, O.F., Tuescher, O., Levy, K.N., Brendel, G., Pan, H., Beutel, M., Pavony, M.T., Epstein, J., Lenzenwever, M.F., Thomas, K.M., Posner, M.I., Stern, E., 2007. Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. The American Journal of Psychiatry 164, 1832–1841.

Stoodley, C.J., Schmahmann, J.D., 2009. Functional topography in the human cerebellum: a meta-analysis of neuroimaging studies. Neuroimage 44, 489–501.

Young, L., Behara, A., Tranel, D., Damasio, H., Hauser, M., Damasio, A., 2010. Damage to ventromedial prefrontal cortex impairs judgment of harmful intent. Neuron 65, 845–851.