Evidence of Orbitofrontal Dysfunction in Sex Offenders

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Abstract

Sex offenders demonstrate a lack of executive regulation of behaviour that is suggestive of prefrontal system dysfunction. Prefrontal systems connect reciprocally with limbic and other subcortical structures that regulate sexual behaviour. Prior research demonstrates prefrontal dysfunction in other criminal populations and several neuropsychological studies and case reports suggest prefrontal, particularly orbitofrontal, dysfunction in association with sex offenses. In this study, sex offenders performed worse than controls on measures sensitive to orbitofrontal dysfunction: go/no-go, antisaccades, and delayed alternation. There was a trend toward differences on smell identification which did not reach significance but were in the predicted direction. Overall, the sex offenders showed difficulty on tasks involving response modulation according to learned rules.

Keywords: Sex offender; prefrontal; executive; impulsivity; orbitofrontal

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INTRODUCTION

Sex offenders are individuals who have acted upon inappropriate sexual impulses, failing to inhibit behavior that is conditioned against by societal rules. In many cases, they are aware of the impropriety of their actions, but either unable or unwilling to inhibit their behavior. A neurobiological basis for these behaviors remains a matter of speculation. Failure to inhibit inappropriate impulses and disadvantageous decision-making represent a lack of executive functioning and are suggestive of prefrontal system dysfunction. Prior research has demonstrated dysfunction in prefrontal systems in criminal populations. Prefrontal, particularly orbitofrontal, dysfunction has been associated with increased aggression, particularly of an impulsive nature (Brower and Price, 2001; Raine et al., 1998). Accordingly, cognitive deficits consistent with orbitofrontal dysfunction have been reported in psychopathy, which involves a lack of concern about harm towards others (Lapierre et al., 1995; Roussy and Toupin, 2000).

A limited number of studies have been directed at the neurobehavioral basis of sex offenses. Subtle forms of cognitive impairment have been shown in sex offenders on the Luria-Nebraska and Halstead-Reitan Test Batteries, and the Wechsler Adult Intelligence Scale (Hucker et al., 1988; Langevin et al., 1989). However, sex offenders do not necessarily show impairments on all neuropsychological or executive function measures. For example, one study did not show any

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impairment in sex offenders relative to controls on the Wisconsin Card Sort Test (Dolan et al., 2002).

Several neuroanatomical structures have been implicated in sexual motivation and behavior (Pfaus, 1999; Meston and Frohlich, 2000). Animal and human research has implicated limbic and paralimbic structures, including the hypothalamus, amygdala, bed nucleus of the stria terminalis, septal nuclei, claustrum, brainstem, and spinal cord. Insults to these structures have resulted in alterations of sexual behavior. For example, cases of late-life homosexual pedophilia have been reported in cases with anterior temporal lobe disturbances, particularly in the right hemisphere (Mendez et al., 2000). Hypersexuality and inappropriate sexual behaviors have been reported as sequelae to septal injury in humans (Gorman and Cummings, 1992).

Prefrontal-subcortical systems (including the prefrontal cortex, striatum, and thalamic nuclei) have been implicated in the execution and regulation of sexual behaviors. Since prefrontal systems are also necessary for inhibiting inappropriate behaviors and choosing appropriate alternatives, they likely play a role in the social aspect of sexual behavior. For example, brain injuries often result in behavioral disinhibition and altered sexual behavior, and occasionally result in sexual offenses (Starkstein and Robinson, 1997; Malloy et al., 1993; Miller et al., 1986; Simpson et al., 1999).

Orbitofrontal cortex (OFC) is of particular interest in this context because of its close association with limbic structures (Bechara et al., 2000; Shoenbaum et al., 1998). OFC mediates the formation of reward and punishment associations, and the control of behavior based on reinforcement contingencies (Rolls, 1996; O'Doherty et al., 2001; Tremblay and Schultz, 1999). The behavioral syndrome associated with orbitofrontal damage characteristically involves alterations in personality and disinhibition of social behaviors (Zald and Kim, 1996; Starkstein and Robinson, 1997). People with lesions of OFC, particularly the ventromedial portion, make poor choices stemming from an insensitivity to future consequences (Bechara et al., 2000). Child molesters also show deficits of empathy, which is associated with orbitofrontal dysfunction (Marshall et al., 2001; Eslinger, 1998; Spinella, 2002).

OFC has been directly associated with sexual functions. Human neuroimaging studies show activation in OFC during sexual arousal (e.g. Stoléru et al., 1999; Redoute et al., 2000). Human cases involving orbitofrontal lesions have been described with resultant changes in sexual behavior,

such as inappropriate advances and public masturbation (Miller et al., 1986). An individual with a right orbitofrontal tumor developed pedophilia, agraphia, and constructional apraxia, which resolved after tumor resection (Burns and Swerdlow, 2003). This individual was unable to inhibit his sexual urges despite knowledge that his actions were morally inappropriate. Further, a case has been reported of a homosexual pedophilic sex offender who showed right orbitofrontal activation on functional neuroimaging when exposed to erotic material (Dressing et al., 2001).

Many traditional neuropsychological tests have proven insensitive to orbitofrontal function. However, there are a few that have been validated by physiological studies. Go/no-go tests involve inhibition of automatic motor responses. Both lesion and electrophysiology studies in humans implicate OFC in go/no-go tasks (Drewe, 1975; Ikeda et al., 1996; Malloy et al., 1989). A functional neuroimaging studies concordantly implicate OFC during go/no-go (Liddle et al., 2001; Tamm et al. 2002). Accordingly, go/no-go deficits are seen in populations with impulsivity and behavioral inhibition problems, such as attention-deficit/hyperactivity disorder (Castellanos et al., 2000). Similarly, antisaccades are an oculomotor equivalent of go/no-go tests, requiring making a saccade in the direction opposite a visual stimulus. Lesion studies indicate that OFC is necessary for the performance of antisaccades (e.g. Hodgson et al., 2002).

Alternation tests involve a subject learning to make alternating responses, each opposite to a previously rewarded response. Impairments are seen in animals and humans with OFC lesions (Freedman et al., 1998). Alternation performance is also impaired in individuals with obsessive-compulsive disorder, which is associated with orbitofrontal dysfunction (Zohar et al., 1999; Gross-Isseroff et al., 1996). Several functional neuroimaging studies have demonstrated alternation tasks to activate orbitofrontal cortex (Gold et al., 1996; Curtis et al., 2000; Zald et al., 2002).

Smell identification tests are sensitive to orbitofrontal function, due to olfactory projections to this region (Scalia and Winans, 1975). OFC is activated during smell identification tasks, and smell tests often show anosmia in individuals with orbitofrontal insults (Savic et al., 1997). Functional neuroimaging studies show that anosmia following brain injury is closely associated with hypometabolism in orbitofrontal cortex (Varney et al., 2001). Olfactory tests have also been shown to relate to inhibition of automatic responses as

measured by the Stroop Color-Word Test in people with schizophrenia (Purdon, 1998).

Given the theoretical rationale for orbitofrontal dysfunction in sex offenders, this study examined this population using the above-mentioned measures putatively sensitive to orbitofrontal function.

METHOD

Participants

The study was approved by an institutional review board and all subjects read and signed an appropriate informed consent in accordance with the Declaration of Helsinki. Participation for all subjects was entirely voluntary. They did not receive any financial compensation for their participation, nor were there any adverse consequences for declining participation. None of the sex offender or control subjects declined to participate.

The sex offender subjects were 21 males, enrolled in an outpatient treatment program. They ranged in age from 19 to 69 years ($M = 37.96$, $SD = 15.3$), and had 10 to 16 years of formal education ($M = 11.9$, $SD = 1.4$). They had been enrolled in treatment for 1 to 42 months ($M = 13.5$, $SD = 11.7$). The control subjects were a convenience sample recruited by word-of-mouth. Thirty-one subjects participated (20 female, 11 male), ranging in age from 19 to 89 years of age ($M = 30.6$, $SD = 16.4$), and completed between 8 and 16 years of formal education ($M = 14.2$, $SD = 1.6$).

Procedures

Go/No-Go Tapping. An imitation tapping set (20 trials) was performed where the subject imitated a standardized sequence of taps (either 1 or 2 taps) performed by the examiner. A conflict tapping set (GNGc) involved the subject performing the opposite of the examiner (one tap for two, and vice versa), and an inhibition set (GNGi) involved suppressing a response (subject imitates the examiner for one tap, but does not tap when the examiner does two). Conflict and Inhibition sets involved standardized sets of 30 trials each. All taps by the examiner are done at 1 second intervals. An incorrect response or a response delayed by more than 1 second is counted as an error. More errors indicate greater dysfunction.

Antisaccades. The procedure and norms were defined by Currie and colleagues (1991). A prosaccade set (10 trials) required the subject to make saccades left or right toward a visual target, presented in randomized order. An antisaccade set (AS, 25 trials), required the subject to look in the direction opposite the visual target, presented in randomized order. More errors indicate greater dysfunction.

Delayed Spatial Alternation. A delayed spatial alternation test was used, whose procedure was adapted from Gross-Isseroff an colleagues (1996). The subject is shown two opaque cups and told a penny is underneath one of them. A movable screen prevented the subject from seeing the placement of the penny, and a felt board underneath minimized extraneous auditory cues. Both cups were baited with pennies for the first trial, and on every successive trial, the cup opposite the subject's last choice was baited. The task involves 25 trials, with 24 possible alternations. Thus, subject must correctly remember the placement of the penny on the last trial and alternate his response on each successive trial. Two indices were recorded: the number of alternations made, (DAL), and trials-to-criterion, which was the first set of five successive alternations (DTC). Both fewer alternations and more trials to reach criterion indicates greater dysfunction.

Alberta Smell Test. The Alberta Smell Test is a measure of olfactory identification (Green and Iverson, 2001). It employs 8 scented markers as stimuli (Raspberry, Grape, Mint, Licorice, Orange, Lemon, Cinnamon, and Melon), presented monorhinally and blindly to the subject. After each stimulus item, they are asked to open their eyes and choose from a list of the 8 possible scents. Both left-sided smell (SL) and right-sided smell (SR) were recorded. The markers are capped in between uses and retain their scent well across multiple uses. However, to prevent a diminishing in the potency of smell, the markers were changed after 10 uses for all subjects.

RESULTS

Several procedures were used to control for demographic differences between the sex offenders and controls. There were no significant differences in age between the sex offenders and controls, $t(51) = 1.54$, $p = .129$. Since the sex offenders in this sample were uniformly male and the controls were comprised of both males and females, a one-way ANOVA was conducted to compare male versus female controls on all seven measures used (GNGc, GNGi, AS, DAL, DTC, SL, and SR). None of the differences were significant, so both female and

male controls were retained for the comparison against the sex offenders.

Secondly, univariate ANOVAs were performed to determine whether education (years completed) influences performance on any of the measures. Results were non-significant for all measures except SR, $F(1,51) = 5.07, p = .029$.

Since age differences were non-significant and education did not contribute to task performance in this sample (except on SR) one-way ANOVAs were performed to compare sex offenders versus

controls for all measures. A comparison of performance on SR was analyzed with an ANCOVA to control for the influence of education. Sex offenders performed significantly worse on all measures except SL (Table 1 and Figure 1). However, comparing differences on SR with an ANCOVA controlling for education was not significant, $F(1, 59) = .42, p = .518$. Cohen's d was calculated to determine effect size, yielding medium to large effects (see Table 1).

Table 1

Comparison of sex offenders and controls on go/no-go (conflict GNGc, and inhibition GNGi), antisaccades (AS), Delayed Alternation (alternations DAL, and trials-to-criterion DTC), and smell identification (left SL and right SR; SR^a = ANOVA, SR^b = ANCOVA controlling for education).

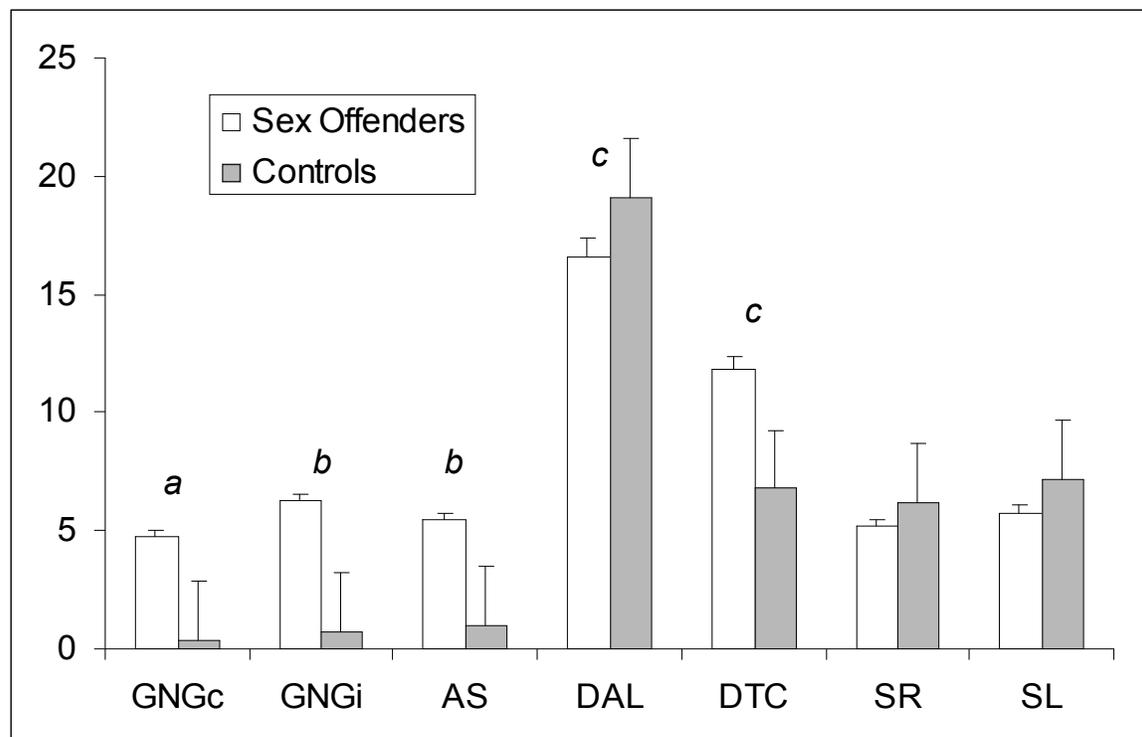|	F	p	d
GNGc,	14.87	< .001	.96
GNGi,	10.52	.002	.81
AS	10.97	.002	.83
DAL	5.82	.019	.69
DTC	6.41	.014	.71
SL	2.42	0.126	N/A
SR^a	5.99	.018	.50
SR^b	.42	.518	N/A

Figure 1. Mean scores for sex offenders and controls. (a. $p < .001$, b. $p < .01$, c. $p < .05$.)

DISCUSSION

Sex offenders performed worse than controls on all of the measures given with the exception of left-sided smell identification and right-sided smell identification after adjusting for education. While none of these measures selectively activates OFC, they all have been demonstrated through lesion, electrophysiological, and neuroimaging studies to require involve cortex. The consistent impairment of sex offenders, as a group, on multiple OFC-sensitive measures convergently suggests an impairment of OFC function relative to normal controls.

The measures on which they differed are measures that involve response modulation. More specifically, these measures involve modulating one's responses in the context of acquiring a rule (DAL, DTC) or responding in the context of changing rules (GNGc, GNGi, and AS). Thus, in keeping with a fundamental role of orbitofrontal cortex, the sex offenders were impaired on tasks that require altering one's behavior in accordance with learned rules.

One limitation of this study is that it analyzes sex offenders as a single group. Certainly, sex offenders constitute a heterogeneous group. While they exhibit more impulsivity as a group, not all offenses committed by sex offenders are impulsive. Rather, some of them involve careful planning and forethought. In future research, it may be fruitful to analyze sex offenders according to the degree of impulsivity or lack of forethought in their crimes. Nonetheless, despite the heterogeneous nature of this sample, the group differences still pointed to greater OFC dysfunction in the offenders. Particular subregions of OFC may be more relevant to the control of sexual impulses.

This study does not address the origin of OFC dysfunction in sex offenders. While it may result from anatomical lesions occurring during development or acquired later in life, it may also result from functional changes in cortical function as a consequence of environmental circumstances and learning. Indeed, this study does not explain why all people with prefrontal injuries do not proceed to become sex offenders.

It must be emphasized that the executive regulation of sexual behavior not only involves prefrontal systems, but also their interaction with limbic structures. Thus, individuals who commit sex offenses may have some form of dysfunction in these prefrontal-subcortical systems. This could involve excessive or aberrant sexual impulses and associations formed by the limbic system that overwhelm inhibitory capacity. On the other hand, it could involve an impaired ability of inhibitory structures to dampen inappropriate drives and behaviors based on social conditioning. These two possibilities are not mutually exclusive, and may exist to differing degrees simultaneously. Indeed, the population of sex offenders may be a heterogeneous group with dysfunction at various levels of the neuraxis.

Another limitation of this study is the fact that the sex offenders are both a selected and self-selected group. These were individuals who were caught and convicted, and may potentially represent a different population from those are able to avoid apprehension. Further, while their attendance in the treatment group has been court-mandated, to a large degree, remaining in the group requires voluntary cooperation. Several members were non-compliant and removed from the group before being tested in this study. These subjects represent those who showed at least adequate motivation to change. Thus, the differences between a sample all sex offenders (apprehended and non-apprehended, compliant and non-compliant) might yield different results.

This study provides neuropsychological evidence that agrees with behavioral indications of orbitofrontal dysfunction in sex offenders. Further work with other measures sensitive to orbitofrontal dysfunction (e.g. reversal learning, Iowa Gambling Task), would corroborate these findings. Further, structural and functional neuroimaging would provide more direct evidence of cerebral dysfunction.

These results have implications for the treatment of sex offenders--a population with a high rate of recidivism. Treatment programs would benefit from focusing on teaching techniques for impulse control and response modulation, as do many programs at present. However, techniques from cognitive remediation could well be adapted into treatment paradigms for sex offenders.

REFERENCES

Bechara, A., Damasio, H., Damasio, A. R. (2000). Emotion, decision making and the orbitofrontal cortex. *Cerebral Cortex*, *10*, 295-307.

Brower, M. C., Price, B. H. (2001). Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: a critical review. *Journal of Neurology, Neurosurgery, & Psychiatry*, *71*(6), 720-6.

Burns J. M. & Swerdlow R. H. (2003). Right orbitofrontal tumor with pedophilia symptom and constructional apraxia sign. *Archives of Neurology*, *60*, 437-440.

Castellanos, F. X., Marvasti, F. F., Ducharme, J. L., Walter, J. M., Israel, M. E., Krain, A., Pavlovsky, C., & Hommer, D. W. (2000). Executive function oculomotor tasks in girls with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*. *39*(5), 644-50.

Currie, J., Ramsden, B., McArthur, C., Maruff, P. (1991). Validation of a clinical antisaccadic eye movement test in the assessment of dementia. *Archives of Neurology*, *48*(6), 644-8.

Curtis, C. E., Zald, D. H., Lee, J. T., & Pardo, J. V. (2000). Object and spatial alternation tasks with minimal delays activate the right anterior hippocampus proper in humans. *Neuroreport*, *11*(10), 2203-7.

Dolan, M., Millington, J., & Park, I. (2002). Personality and neuropsychological function in violent, sexual and arson offenders. *Medical Science and the Law*, *42*(1), 34-43.

Dressing, H., Obergriesser, T., Tost, H., Kaumeier, S., Ruf, M., Braus, D.F. (2001). [Homosexual pedophilia and functional networks - An fMRI case report and literature review] *Fortschritte der Neurologie-Psychiatrie*, *69*(11), 539-44.

Drewe, E. A. (1975). An experimental investigation of Luria's theory on the effects of frontal lobe lesions in man. *Neuropsychologia*, *13*(4), 421-9.

Eslinger, P. J. (1998). Neurological and neuropsychological bases of empathy. *European Neurology*, *39*(4), 193-9.

Freedman, M., Black, S., Ebert, P., & Binns, M. (1998). Orbitofrontal function, object alternation and perseveration. *Cerebral Cortex*, *8*(1), 18-27.

Gold, J. M., Berman, K. F., & Randolph, C. (1996). PET validation of a novel prefrontal task: Delayed response alteration. *Neuropsychology*, 10(1), 3-10.

Gorman, D. G. & Cummings, J. L. (1992). Hypersexuality following septal injury. *Archives of Neurology*, *49*(3), 308-10.

Green, P. & Iverson, G. L. (2001). Effects of injury severity and cognitive exaggeration on olfactory deficits in head injury compensation claims. *NeuroRehabilitation*, *16*(4), 237-43.

Gross-Isseroff, R., Sasson, Y., Voet, H., Hendler, T., Luca-Haimovici, K., Kandel-Sussman, H., & Zohar, J. (1996). Alternation learning in obsessive-compulsive disorder. *Biological Psychiatry*, *39*(8), 733-8.

Hodgson, T., Mort, D., Chamberlain, M., Hutton, S., O'Neill, K., & Kennard, C. (2002). Orbitofrontal cortex mediates inhibition of return. *Neuropsychologia*, *40*(12), 1891.

Hucker, S., Langevin, R., Dickey, R., Handy, L. (1988). Cerebral damage and dysfunction in sexually aggressive men. *Annals of Sex Research*, *1*(1), 33-47.

Ikeda, A., Luders, H.O., Collura, T.F., Burgess, R.C., Morris, H.H., Hamano, T., & Shibasaki, H. (1996). Subdural potentials at orbitofrontal and mesial prefrontal areas accompanying anticipation and decision making in humans: a comparison with Bereitschaftspotential. *Electroencephalography and Clinical Neurophysiology*, *98*(3), 206-12.

Langevin, R., Wortzman, G., Wright, P., & Lorraine, H. (1989). Studies of brain damage and dysfunction in sex offenders. *Annals of Sex Research*, 2(2), 163-179.

Lapierre D., Braun Claude M. J., & Hodgins, S. Ventral frontal deficits in psychopathy: Neuropsychological test findings. *Neuropsychologia*, *33*(2), 139-151.

Liddle, P. F., Kiehl, K. A., & Smith, A. M. (2001). Event-related fMRI study of response inhibition. *Human Brain Mapping*, *12*(2), 100-9.

Malloy, P., Rasmussen, S., Braden, W., & Haier, R. J. Topographic evoked potential mapping in obsessive-compulsive disorder: evidence of frontal lobe dysfunction. *Psychiatry Research*, *28*(1), 63-71.

Malloy, P., Bihrle, A., & Duffy, J. (1993). The orbitomedial frontal syndrome. *Archives of Clinical Neuropsychology*, *8*(3), 185-201.

Marshall, W. L., Hamilton, K., & Fernandez, Y. (2001). Empathy deficits and cognitive distortions in child molesters. *Sex Abuse*, *13*:123-30.

Mendez, M.F., Chow, T., Ringman, J., Twitchell, G., & Hinkin, C.H. (2000). Pedophilia and temporal lobe disturbances. *Journal of Neuropsychiatry and Clinical Neurosciences*, *2*(1), 71-6.

Meston, C.M. & Frohlich, P.F. (2000). The neurobiology of sexual function. *Archives of General Psychiatry*, *57*(11), 1012-30.

Miller, B.L., Cummings, J.L., McIntyre, H., Ebers, G., & Grode, M. (1986). Hypersexuality or altered sexual preference following brain injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *49*(8), 867-73.

O'Doherty, J., Kringelbach, M.L., Rolls, E.T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, *4*(1), 95-102.

Pfaus, J.G. (1999). Neurobiology of sexual behavior. *Current Opinions in Neurobiology*. *9*(6), 751-8.

Purdon, S.E. (1998). Olfactory identification and Stroop interference converge in schizophrenia. *Journal of Psychiatry and Neuroscience*, *23*(3), 163-71.

Raine, A., Meloy, J.R., Bihrle, S., Stoddard, J., LaCasse, L., (1998). Buchsbaum, M.S. Reduced prefrontal and increased subcortical brain functioning assessed using positron emission tomography in predatory and affective murderers. *Behavioral Science and the Law*. *16*(3), 319-32.

Redoute, J., Stoleru, S., Gregoire, M.C., Costes, N., Cinotti, L., Lavenne, F., Le Bars, D., Forest, M.G., & Pujol, J.F. (2000). Brain processing of visual sexual stimuli in human males. *Human Brain Mapping*, *11*(3), 162-77.

Rolls, E.T. (1996). The orbitofrontal cortex. *Philosophical transactions of the Royal Society of London. Series B, Biological sciences, 351*, 1433-43.

Rosse, R.B., Collins, J.P. Jr., Fay-McCarthy, M., Alim T.N., Wyatt, R.J., and Deutsch, S.I., (1994). Phenomenologic comparison of the idiopathic psychosis of schizophrenia and drug-induced cocaine and phencyclidine psychoses: a retrospective study. *Clinical Neuropharmacology,* 1994 Aug; *17*(4), 359-69.

Roussy, S. & Toupin J. (2000). Behavioral inhibition deficits in juvenile psychopaths. *Aggressive Behavior*, *26*(6), 413-424.

Savic, I., Bookheimer, S.Y., Fried, I., Engel, J. Jr. (1997). Olfactory bedside test. A simple approach to identify temporo-orbitofrontal dysfunction. *Archives of Neurology*, *54*(2), 162-8.

Scalia, F., & Winans, S.S. (1975). The differential projections of the olfactory bulb and accessory olfactory bulb in mammals. *Journal of Comparative Neurology*, *161*(1), 31-55.

Schoenbaum, G., Chiba, A.A., & Gallagher, M. (1998). Orbitofrontal cortex and basolateral amygdala encode expected outcomes during learning. Nature Neuroscience, *1*, 155-9.

Simpson, G., Blaszczynski, A., Hodgkinson, A. (1999). Sex offending as a psychosocial sequela of traumatic brain injury. *Journal of Head Trauma Rehabilitation,*.*14*(6), 567-80.

Spinella, M. (2002). A relationship between smell identification and empathy. *International Journal of Neuroscience*, *112*, 605-612.

Starkstein, S. E., Robinson, R. G. (1997). Mechanism of disinhibition after brain lesions. *Journal of Nervous and Mental Disease*, *185*(2), 108-14.

Stoleru, S., Gregoire, M. C., Gerard, D., Decety, J., Lafarge, E., Cinotti, L., Lavenne, F., Le Bars, D., Vernet-Maury, E., Rada, H., Collet, C., Mazoyer, B., Forest, M. G., Magnin, F., Spira, A., & Comar, D. (1999). Neuroanatomical correlates of visually evoked sexual arousal in human males. *Archives of Sexual Behavior*, *28*(1), 1-21.

Tamm, L., Menon, V., Reiss, A. L. (2002). Maturation of brain function associated with response inhibition. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*(10), 1231-8.

Tammany, J.M., Evans, R.G., and Barnett, R.W. (1990). Personality and intellectual characteristics of adult male felons as a function of offense category. *Journal of Clinical Psychology. 46*(6), 906-911

Toomey, R., Lyons, M.J., Eisen, S.A., Xian, H., Chantarujikapong, S., Seidman, et al (2003). A twin study of the neuropsychological consequences of stimulant abuse. *Archives of General Psychiatry*, Mar; *60*(3), 303-10.

Tremblay, L., Schultz, W. (2000). Reward-related neuronal activity during go-nogo task performance in primate orbitofrontal cortex. *Journal of Neurophysiology*, *83*(4), 1864-76.

Varney, N. R., Pinkston, J. B., Wu, J. C. (2001). Quantitative pet findings in patients with posttraumatic anosmia. *Journal of Head Trauma Rehabilitation*, *16*(3), 253-9.

Zald, D. H. & Kim, S. W. (1996). Anatomy and function of the orbital frontal cortex, II: Function and relevance to obsessive-compulsive disorder. *Journal of Neuropsychiatry and Clinical Neurosciences*, *8*(3), 249-61.

Zald, D. H., Curtis, C., Folley, B. S., Pardo, J. V. (2002). Prefrontal contributions to delayed spatial and object alternation: a positron emission tomography study. *Neuropsychology*, *16*(2), 182-9.

Zohar, J., Hermesh, H., Weizman, A., Voet, H., & Gross-Isseroff R. (1999). Orbitofrontal cortex dysfunction in obsessive-compulsive disorder? I. Alternation learning in obsessive-compulsive disorder: male-female comparisons. *European Neuropsychopharmacology*, *9*(5), 407-13.