



Neuroanatomical Substrates for Sex Offenses

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Abstract

Sex offending involves an inability or unwillingness to conform one's sexual behavior to societal standards, acting without consent or against the wishes of others, and often resulting in considerable harm. This paper reviews clinical and forensic studies in order to elucidate the neuroanatomical basis of sexual behavior, and how dysfunction in these systems results in aberrant sexual behavior. Prefrontal-subcortical systems involving the striatum and thalamus are involved in the regulation of sexual behavior, mediating functions such as initiation, inhibition, choice, empathy, reward and punishment. Limbic structures such as the extended amygdala, septal nuclei, and hypothalamus mediate other aspects of sexuality such as sex drive, and likely mediate sexual orientation and gender identity. Dysfunction in these systems of various etiologies may lead to increased predisposition to commit sex offenses.

Keywords: Sex offender; prefrontal; limbic; pedophilia; hypersexuality

INTRODUCTION

Sex offenders create a considerable amount of suffering in their victims and have proven a difficult population to treat (Prentky, Lee, Knight, & Cerce, 1997). While there is some evidence for efficacy of treatment (e.g. Alexander, 1999), this population remains incompletely understood and the potential for more effective treatments exists. Sex offenders are a heterogeneous group differing along several dimensions, but by definition they share the common feature of being unable or unwilling to inhibit their socially inappropriate sexual behavior which leads to the harm of others. An understanding of this condition at a neurobiological level could further both pharmacological and psychotherapeutic treatment strategies.

Aberrant sexual behavior has long been associated with dysfunction of the nervous system. Kraft-Ebbing (1886) detailed numerous cases of paraphilias in individuals with identifiable neurological illnesses, including brain injury, dementia, and epilepsy (for which there was no effective pharmacological treatment at the time). For example, Ebbing's case #1 details a possible case of late-life behavioral changes manifesting as hypersexuality and disinhibition and reminiscent of frontotemporal dementia. Another case (#15) involved sexual homicide and zoophilia in an individual who had atrophy of the frontal, temporal, and occipital cortex.

Modern research in this vein has shown a relationship between neurological status and some

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forms of violent crime, including murder (Lewis, Pincus, Feldman, Jackson, & Bard, 1986; Lewis et al., 1988; Blake, Pincus, & Buckner, 1995). Raine and colleagues (1998) have shown that impulsive murderers have reduced prefrontal functioning compared to those who commit more premeditated, predatory murders. Neuroanatomical differences have been found in the prefrontal cortex of individuals with antisocial personality disorder (Raine et al., 1994; Raine, Lencz, Bihrlé, LaCasse, & Colletti, 2000). Thus it is possible that neurological deficits relate to sex offenses, possibly violent and nonviolent.

Several neuroanatomical structures have been implicated in sexual motivation and behavior (Pfaus, 1999; Meston & Frohlich, 2000). Animal and human research has implicated limbic and paralimbic structures, including prefrontal-striatal-thalamic circuits, extended amygdala, septal nuclei, hypothalamus, brainstem, and spinal cord. As the roles these structures play in sexual behavior become delineated, it becomes increasingly feasible to look for a neuroanatomical basis for sex offenses.

Studies of cognitive functioning in sex offenders are suggestive of cerebral impairments. Incest perpetrators tend to have lower IQ scores than non-sex offender controls, as measured by the Wechsler Adult Intelligence Scale—Revised (Langevin, Wortzman, & Dickey, 1988). Neuropsychological impairments were also evident on the Halstead-Reitan Battery in 30% of incest perpetrators. Male exhibitionists have shown disparities between verbal IQ and performance IQ (i.e. lower scores on VIQ) compared to controls (Langevin, Lang, Wortzman, Frenzel, & Wright, 1989). They also showed impairment on the Tactual Performance (total time), Trail Making Test A, and the Aphasia Screening Test. Pedophiles showed impairments on all WAIS-R subtests and several Halstead-Reitan subtests (i.e. Tactual Performance Test, Trail Making Test A and B, Categories, Aphasia Screening Test, and Impairment Index) relative to controls (Langevin, Wortzman, Wright & Handy, 1989). In contrast, there were no differences between offenders and controls on the Wechsler Memory Scale-Revised.

Early neuroimaging studies indicated greater nonspecific abnormalities on CT and cerebral blood flow in sex offenders (Hendricks et al., 1988; Graber, Hartmann, Coffman, Huey, & Golden, 1982). While these studies are suggestive of a neurological component to sex offending, more recent studies have addressed the issue with greater neuroanatomical detail.

This paper will review the several types of studies to corroborate a role for neuroanatomical structures in sex offenders. While space does not permit a comprehensive review of the role of neuroanatomical structures, a few illustrative studies from the experimental literature will be cited, such as lesion studies in animals and functional neuroimaging studies of normal human subjects. Studies or case reports of sex offenders will be examined where neuroimaging or neurobehavioral data are presented. Further, clinical studies or cases will also be examined where the onset of sex offenses was closely associated with the onset of demonstrated neurological illness.

PREFRONTAL-SUBCORTICAL SYSTEMS

Prefrontal cortex and associated subcortical structures, such as the striatum and mediodorsal thalamic nuclei, have been associated with processes of executive self-regulation, allowing for behavior that is more goal-oriented, autonomous, and flexible (Goldberg, 2001). Prefrontal systems have been associated with several aspects of sexual behavior. For example, two human neuroimaging studies in normal individuals convergently found activation of right prefrontal cortex during both sexual arousal and orgasm (Tiihonen et al., 1994; Stoléru et al., 1999). Similarly, an electrocorticogram study in rats showed prefrontal activation corresponds with copulation (Hernández-González et al., 1998).

Medial Prefrontal Cortex

Subregions of prefrontal cortex play differential roles in behavior. Medial prefrontal cortex, including the anterior cingulate gyrus, mediates the initiation of behaviors, and lesions of anterior cingulate diminish the initiation of sexual behavior in rats (Agmo, Villalpando, Picker, & Fernández, 1995; Yamanouchi & Arai, 1992; Devinsky, Morrell, & Vogt, 1995). Anterior cingulate activity increases in sexually receptive female sheep exposed to a male, but not in anestrous females (Ohkura et al., 1997). Human neuroimaging studies in males show an increase in blood flow in the anterior cingulate gyrus during sexual arousal (Deiber, Honda, Ibanez, Sadato, & Hallett, 1999; Stoléru et al., 1999; Rauch et al., 1999; Redoute et al., 2000). Cases of anterior cingulate epilepsy show varying interictal phenomena such as intermittent psychoses, antisocial behavior, or aggressive outbursts (Mazars, 1970; Devinsky et al., 1995).

Orbitofrontal Cortex

Orbitofrontal cortex (OFC) is of particular interest in sex offenders given its roles in regulating behavior and close association with limbic structures. Human neuroimaging studies show activation in OFC during sexual arousal (Stoléru et al., 1999; Redoute et al., 2000). Lesions of orbitofrontal cortex do not cause any deficits of sexual behavior in rats (de Bruin, van Oyen, & Van de Poll, 1983). Lesions of OFC in humans produce a syndrome of behavior disturbances including impulsivity, mood instability, a lack of empathy, behavioral disinhibition, and social inappropriateness (Malloy, Bihrlé, & Duffy, 1993). Orbitofrontal dysfunction has been associated with increased aggression, particularly of an impulsive nature (Brower & Price, 2001). Rolls (1996) emphasizes that this region of PFC mediates behavior based on learned reward and punishment associations. Accordingly, people with lesions of OFC, particularly the ventromedial region, show insensitivity to future consequences, exhibiting poor judgement and decision-making (Bechara, Damasio, Damasio, & Anderson, 1994). Ironically, they may make disadvantageous choices while being able to verbally express knowledge that better choices exist.

Not surprisingly, sexual promiscuity uncharacteristic of premorbid behavior may develop after a brain injury (Starkstein & Robinson, 1997; Malloy et al., 1993). Miller and colleagues (1986) have described cases of humans with orbitofrontal lesions with resultant changes in sexual behavior such as inappropriate advances and public masturbation. Traumatic brain injury has a predilection to affect orbitofrontal areas. In one sample of brain injury patients, a proportion (6.5%) committed sexual offenses, typically involving exhibitionism, frotteurism, toucherism, voyeurism, or overt sexual aggression (Simpson, Blaszczynski, & Hodgkinson, 1999). While this only occurs in a minority of brain injury patients, there was an absence of pre-injury history of sexual offenses in these cases, suggesting an etiological role of the brain injury. Hypersexuality has also been reported in numerous cases of frontotemporal dementia (Tang-Wai et al., 2002; Dell & Halford, 2002). One case described by Mendez and colleagues (2000), noted the manifestation of homosexual pedophilia.

A few cases of multiple sclerosis have been reported affecting prefrontal system function, either directly or indirectly. Some case studies have reported simple hypersexuality in cases of multiple sclerosis with frontal involvement (e.g. Gondim Fde, 2001). Other cases have involved more

extensive and severe pathological behavior. A case was reported by Ortega and colleagues (1993) involving a female who manifested exhibitionism, incest, scopophilia, and zoophilia. After multiple arrests, she died in jail and autopsy revealed severe demyelination in the frontal lobes, as well as the thalamus and mesencephalon. Another case of multiple sclerosis was reported in a young man, also involving frontal and periventricular structures. He manifested hypersexuality and fetishism, leading eventually to imprisonment (Huws, Shubsachs, & Taylor, 1991). A recent case has been reported of an individual with inappropriate sexual behaviors following the onset of multiple sclerosis (Frohman, Frohman, & Moreault, 2002). While his lesions were outside of the frontal lobes (involving the hypothalamus and septal nuclei), OFC was likely affected through diaschisis, as evidenced by anosmia, bimanual motor programming deficits, impulsivity, and poor judgement. While this individual had no premorbid history of sexual offenses, he sexually propositioned one minor and sexually assaulted another, as well as an adult female, for which he was eventually incarcerated.

OFC plays an important role in empathy, and sex offenders have been noted to be deficient in empathy (Marshall, Hamilton, & Fernandez, 2001; Eslinger, 1998; Spinella, 2002). Early developmental damage to prefrontal cortex can produce an "acquired sociopathy" syndrome with delayed onset and an insidious course (Eslinger, Grattan, Damasio, & Damasio, 1992). Accordingly, cognitive deficits consistent with orbitofrontal dysfunction have been reported in psychopathy (Lapierre, Braun, & Hodgins, 1995).

A sample of sex offenders found smaller left frontal lobe volume relative to normal controls (Wright, Nobrega, & Langevin, 1990). A functional neuroimaging (fMRI) study of a homosexual pedophile showed activation in right orbitofrontal cortex when exposed to pictures of young males (Dressing et al., 2001). In a mixed sample of sex offenders, Spinella, White, Frank and Schiraldi (in press) have shown significantly impaired performance neurobehavioral measures sensitive to orbitofrontal dysfunction (i.e. go/no-go, antisaccades, and delayed alternation).

Striatum and Thalamus

The striatum and thalamic nuclei work in close association with prefrontal cortex to regulate cognitive, emotional, and motor functions (Masterman & Cummings, 1997). The striatum, including the ventral striatum and nucleus accumbens, plays a role in reward processes and

sexual function, where lesions diminish sexual interest in animals (e.g. Schultz, Tremblay, & Hollerman, 1998; Robertson et al., 1991). Lesions of the accumbens have also been associated with increased impulsivity in rats (Cardinal, Pennicott, Sugathapala, Robbins, & Everitt, 2001). Human neuroimaging studies have reported activation in the head of the caudate and ventral pallidum during sexual arousal (Rauch et al., 1999; Redoute et al., 2000). The effects of accumbens lesions on human sexual behavior are still tentative: one recent case report of a male patient with a relatively selective lesion of the nucleus accumbens reported a loss of interest in pleasure and sex (Goldenberg, Schuri, Gromminger, & Arnold, 1999). However, hypersexuality was noted in an adult patient with bilateral pallidal lesions due to carbon monoxide poisoning (Starkstein, Berthier, & Leiguarda, 1989). Hypersexuality was also reported in an elderly male patient with a lacunar infarct of the subthalamic nucleus, which created hypometabolism in the basal forebrain, temporal lobes, medial prefrontal cortex, and striatum (Absher et al., 2000). Acute mania, chorea, and hypersexuality developed in a male after right thalamic infarction, disrupting frontal-striatal-thalamic circuits (Inzelberg, Nisipeanu, Joel, Sarkantus, & Carasso, 2001). Another case of hypersexuality after a thalamic infarct occurred in conjunction with mania (Benke, Kurtzthaler, Schmidauer, Moncayo, & Donnemiller, 2002). A third report of hypersexuality was involved a thalamic infarct manifesting a paramedian thalamic syndrome (i.e. hypersomnolence, confabulatory anterograde amnesia, and vertical gaze deficits) and dysexecutive syndrome (i.e. behavioral disinhibition, apathy, witzelsucht, motor regulation deficits, and environmental dependence) (Spinella, 2004). This elderly male individual manifested exhibitionism, public masturbation, and verbal sexual obscenities that were atypical of his premorbid behavior. Thus, alterations in sexual behavior have been reported with lesions at all points of prefrontal-striatal-thalamic circuits.

There do not appear to be any controlled studies to date that specifically examine the striatum or thalamic nuclei in sex offenders. On the other hand, disinhibition and altered sexual behavior have been noted in Huntington's disease, which involves degeneration of the striatum. In one study, 30 of 48 subjects showed some alteration of sexual behavior, including sexual jealousy, exhibitionism, homosexual aggression, sodomy, voyeurism, and promiscuity (Dewhurst, Oliver, & McKnight, 1970). A case has been reported of transvestic fetishism associated with selegiline treatment in a

man with Parkinson's disease (PD). His cross-dressing began after selegiline treatment was initiated, and ceased after it was discontinued. Another case was reported of a man with PD who developed zoophilia in close association with an increase in antiparkinsonian medications (Jimenez-Jimenez, Sayed, Garcia-Soldevilla, & Barcenilla, 2002). Hypersexuality has also been reported in PD patients treated with dopaminergic medications (Uitti et al., 1989). However, as dopaminergic therapy will affect multiple brain areas it cannot be determined whether the basal ganglia were the critical site for altering sexual behavior in these studies, or whether other sites (e.g. limbic) contributed. Another case of hypersexuality has been reported in association with Parkinson's disease, involving a right pallidotomy and left pallidal deep brain stimulator electrode (Roane, Yu, Feinberg, & Rogers, 2002).

TEMPOROLIMBIC STRUCTURES

Amygdala

The medial amygdala has been specifically implicated in sexual behaviors, since lesions impair copulation and stimulation facilitates sexual behavior in animals (Newman, 1999; Stark et al., 1998). Human neuroimaging studies show activation in the amygdala and the closely related anterior temporal cortex during sexual arousal (Rauch et al., 1999; Stoléru et al., 1999; Karama et al., 2002). Concordantly, electrophysiological activity in human males occurred in right temporal regions during maximum tumescence (Cohen, Rosen, & Goldstein, 1985).

Chronic temporal lobe seizures tend to create hyposexuality (Morrel et al., 1994), but seizure activity can activate discrete genital automatism in some cases of temporal lobe epilepsy (Leutmezer et al., 1999; Spencer, Spencer, Williamson, & Mattson, 1983). Epileptic auras sometimes present with orgasmic sensations (Tanuri, Thomaz, & Tanuri, 2000; Calleja, Carpizo, & Berciano, 1988). Lesions of the amygdala and anterior temporal lobes can produce a Klüver-Bucy syndrome, which includes hypersexuality (Klüver & Bucy, 1939). While uncommon, the Klüver-Bucy syndrome has been reported in humans (Hayman, Rexer, Pavol, Strite, & Meyers, 1998; Góscinski, Kwiatkowski, Polak, Orłowiejska, & Partyk, 1997; Trimble, Mendez, & Cummings, 1997). The hypersexuality in humans may manifest as overt sexual advances or may be limited to sexually inappropriate verbalizations.

A few neuroimaging studies have systematically examined temporolimbic structures in sex offenders. Sadistic sex offenders compared to non-sadist sex offenders and controls showed dilation of the right temporal horn (Hucker et al., 1988). A mixed sample of sex offenders (pedophiles, incest offenders, sexually aggressive offenders of adult females) found smaller left temporal lobe volume relative to normal controls (Wright et al., 1990). Two cases of homosexual pedophilia have been reported with onset later in life and in association with temporal lobe dysfunction (e.g. frontotemporal dementia and bilateral hippocampal sclerosis) (Mendez et al., 2000). In both cases, positron emission tomography showed prominent right temporal lobe hypometabolism. It is uncertain in these cases whether the lesions in these cases initiated the interest, or rather unmasked an existing predisposition. In accordance, an analysis of cases of frontotemporal dementia suggests that dysfunction of right anterior temporal cortex causes a decrease in empathy (Perry et al., 2001). Similarly, a neuropathology study revealed simple cell atrophy of pyramidal cells and reactive astrocytosis in the hippocampus of people diagnosed with various DSM-IV diagnoses of paraphilia (Casanova et al., 2002).

Bed Nucleus of the Stria Terminalis

The bed nucleus of the stria terminalis (BNST) is considered a part of the extended amygdala and has been implicated in sexual behaviors (de Olmos, 1990; Claro et al., 1995). The BNST has been implicated in gender identity, since it is larger in human males than females and male-to-female transsexuals (Zhou et al., 1995). However, it appears unrelated to sexual orientation or to hormonal treatments in transsexuals. Altered sexual behavior due to BNST lesions has not been reported in humans. However, the BNST and septal nuclei are small structures and in close proximity, so it is uncertain whether changes in behavior result from damage to one or both structures. In addition, sexual sadists often show a propensity for disturbance in gender identity (Langevin, Bain, & Wortzman, 1988).

SEPTAL NUCLEI

The septal nuclei have been implicated in sexual behavior by lesion studies in animals, producing the "septal syndrome," of hypersexuality, amnesia, and including sham rage (Cavazos et al. 1997). The septal nuclei mediate both male and female sexual

behaviors (Gogate et al., 1995; Imondi & Floody, 1998). Human studies corroborate the animal studies regarding a role in sexual behavior. Electrical or chemical stimulation (with acetylcholine) of the septal nuclei in conscious humans creates feelings of euphoria and orgasm (Heath & Fitzjarrell, 1984; Sem-Jacobson, 1968). Similarly, electrical recordings from the septal region in humans during sexual intercourse show spike-wave activity during orgasms (Heath, 1964). Lesions of the septal nuclei in humans have elicited hypersexuality in several documented cases (Gorman & Cummings, 1992). The changes in sexual behavior noted in such cases include inappropriate sexual advances, sexually explicit language, and public masturbation. The case of multiple sclerosis in a male with inappropriate sexual behavior reported by Frohman and colleagues (2002) also involved the septal region. During exacerbations, he masturbated excessively (i.e. between 10-12 times per day), and felt uncontrollable urges to grab women's breasts, even though he reported knowing that his behavior was inappropriate.

HYPOTHALAMUS

Several hypothalamic nuclei have been associated with sexual function, including the medial preoptic area (MPOA), ventromedial nucleus (VMN), and paraventricular nucleus (PVN) (Pfaus, 1999; Meston & Frohlich, 2000). The MPOA has been implicated in both sexual arousal and performance (Shimura et al., 1994), and while lesions may impair copulation in animals, male primates with MPOA lesions will masturbate when a female is nearby (Sлимп et al., 1978). The VMN has been associated with lordosis in female animals (Pfaff & Sakuma, 1979), and the PVN is implicated in penile erection (McKenna, 1998).

Neuroimaging has shown activation in the hypothalamus in human males (but not females) when sexually aroused by erotic material (Karama et al., 2002). The third interstitial nuclei of the anterior hypothalamus (INAH3) has been implicated in sexual orientation since it is larger in heterosexual men compared to women and homosexual men, although this association is under debate (LeVay, 1991). However, later research supported sexual dimorphism but not the relationship to sexual orientation (Byne et al., 2000). Further work is needed to clarify the neurobiology of sexual orientation (Swaa, Chun, Kruijver, Hofman, & Ishunina, 2002).

Clinical cases of pathologies of the hypothalamus produce alterations in sexual behavior, which is more typically a loss of libido (Martin & Riskind, 1992). As such, stereotactic hypothalamotomy has been used as a treatment for sexual aggressiveness, and has been shown to reduce sexual drive (Dieckmann et al., 1988). On the other hand, sex offenses may also be associated with dysfunction or lesions of the hypothalamus. Pedophiles show an exaggerated elevation in lutenizing hormone (LH) in response to lutenizing hormone releasing hormone (LHRH), suggesting dysfunction in the hypothalamic-pituitary-gonadal axis (Gaffney & Berlin, 1984). The case reported by Frohman and colleagues (2002) involving sexual impropriety during multiple sclerosis exacerbations also demonstrated hyperintensities hypothalamic area. Hypothalamic hamartoma has been implicated in a case of uncontrollable exhibitionism (Liebaltd, 1971).

CONCLUSIONS

The available data suggest that sexual behavior is regulated by a circuit of neuroanatomical structures, and that insults to these structures can produce drastic changes of sexual behavior. Further, there is preliminary evidence for abnormalities in some of these structures in criminal sex offenders. Clinical cases provide the most overt and salient examples of how sexual behavior can be radically altered by neurological insults. However, the offenses committed by sex offenders with no apparent diagnosable neurological illness may nonetheless result from more insidious and subtle forms of brain dysfunction.

This is not to say that all sex offenses result from overt neurological insult. Not all neurological insults, even those localized to the above-mentioned neuroanatomical regions, will inevitably lead to inappropriate sexual behavior. Sex offenses, like most other human behaviors involve biological predispositions and the acquired effects of experience, both of which would cause changes in brain structure and function. It has not been determined why some individuals with neurological insults experience altered sexual behavior while others do not, even when the same structures in the brain are affected. However, it is apparent from animal experimental studies that small-scale differences in damage to the microcircuitry of neural structures can create largely varying results. For example, while lesions of the medial amygdala impair sexual behavior, while lesions of the

basolateral amygdala do not (Kondo, 1992). Similarly, lesions of the lateral septal nuclei impair sexual behavior, while lesions of the medial septal nuclei facilitate it (Gogate et al., 1995). Conventional neuroimaging techniques may lack the resolution to detect such small scale differences.

The neuroanatomical structures that govern sexual behavior represent a widespread, distributed network of structures in the nervous system (Pfaus, 1999; Meston & Frohlich, 2000; Spinella, in review). The interconnections of these structures suggest a hierarchical organization of structures that control sexual behavior. Brainstem and spinal areas control the direct motor and autonomic aspects of sexual behavior, which are controlled by limbic areas (i.e. the hypothalamus, septal nuclei, and extended amygdala). Limbic structures mediate sexual motivation and urges, sexual preference, and gender identity. These, in turn, are regulated by the prefrontal-striatal-thalamic circuits, which play an executive role in sexual behavior, mediating the initiation, inhibition, and social judgement aspects (e.g. decision making, empathy).

Thus, it is possible to see differential manifestations of sex offenses based on the loci of neuroanatomical lesions. Dysfunction of prefrontal structures is more likely to involve reduced empathy for the victim, antisocial behavior, and generalized impulsivity (not limited to sexual behavior). Those with limbic dysfunction may experience uncontrollable sexual urges, which overwhelm the regulatory capacity of prefrontal structures (e.g. Frohman, 2002).

The aberrant sexual behaviors triggered by neurological illness are strikingly similar to those who become sex offenders with no overt or known neurological illness. In several cases, an illness was only discovered after criminal conviction. Further, the neurobehavioral and neuroimaging studies performed in sex offenders supports subtle or cryptogenic brain dysfunction in many of this forensic population. It is possible that these result from the extremes of normal variation in brain development, or they may constitute subtle insults acquired during early or adult development. On the other hand, sex offenders frequently have experienced sexual abuse themselves, particularly during childhood (Cohen et al., 2002). Abuse has been shown to alter the function, and likely the structure of the brain (Teicher et al., 1993; Ito et al., 1993; Ito et al., 1998). Sexual offenders were found to have a greater instance of non-right handedness than controls, suggesting subtle neurological differences (Bogaert, 2001). It has been suggested that sexual offenses are most likely to result when

one has a history of sexual abuse combined with a neurological insult (Blake et al., 1995; Cohen et al., 2002)

The putative neuroanatomical basis for sex offending has implications for treatment. Psychotherapeutic treatment programs should include a focus on impulse-control strategies. Fostering of self-control may create functional changes in the brain analogous to the way that behavioral changes normalize brain activation in obsessive-compulsive disorder treatment paradigms (Schwartz et al., 1996). Similarly, anti-androgen therapy and other forms of pharmacotherapy likely exert their effects on receptors located in the neuroanatomical structures discussed in this paper (Briken et al., 2001). However, a detailed study of the neurochemical anatomy of these structures may suggest novel pharmacological treatment strategies. Combined pharmacotherapy and cognitive-behavioral treatment, when applicable, would offer the most comprehensive neurobiological approach currently available to treat sex offenders.

As progress continues in the understanding of the neurological basis of sexual behavior, so will our understanding of variations in sexual behavior, including maladaptive and pathological forms. It is hoped that the integration of clinical and experimental research on the neurobiology of sex offenses over time will eventually lead to better strategies for prevention and treatment.

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