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Reduced Prefrontal and Increased Subcortical Brain Functioning Assessed Using Positron Emission Tomography in Predatory and Affective Murderers

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There appear to be no brain imaging studies investigating which brain mechanisms subserve affective, impulsive violence versus planned, predatory violence. It was hypothesized that affectively violent offenders would have lower prefrontal activity, higher subcortical activity, and reduced prefrontal/subcortical ratios relative to controls, while predatory violent offenders would show relatively normal brain functioning. Glucose metabolism was assessed using positron emission tomography in 41 comparisons, 15 predatory murderers, and nine affective murderers in left and right hemisphere prefrontal (medial and lateral) and subcortical (amygdala, midbrain, hippocampus, and thalamus) regions. Affective murderers relative to comparisons had lower left and right prefrontal functioning, higher right hemisphere subcortical functioning, and lower right hemisphere prefrontal/subcortical ratios. In contrast, predatory murderers had prefrontal functioning that was more equivalent to comparisons, while also having excessively high right subcortical activity. Results support the hypothesis that emotional, unplanned impulsive murderers are less able to regulate and control aggressive impulses generated from subcortical structures due to deficient prefrontal regulation. It is hypothesized that excessive subcortical activity predisposes to aggressive behaviour, but that while

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predatory murderers have sufficiently good prefrontal functioning to regulate these aggressive impulses, the affective murderers lack such prefrontal control over emotion regulation.

Clinical and forensic research has frequently referred to "affective", "reactive", "defensive", "impulsive", or "hot blooded" aggression (a response to physical or verbal aggression initiated by others with violence that is relatively uncontrolled and emotionally charged), as opposed to "predatory", "instrumental", "proactive", "attack", or "cold blooded" aggression (controlled, purposeful aggression lacking in emotion that is used to achieve a desired goal) (Dodge, 1991; Meloy, 1988). In this context, Meloy (1988; 1997) has developed forensic criteria that take a categorical approach which views human violence as either predominantly affective or predatory. Similarly, Dodge (1991) categorizes childhood aggression as either proactive or reactive, while acknowledging that very few aggressive acts are purely reactive or proactive in nature.

There is increasing construct validity for the notion that impulsive, affectively aggressive individuals differ from predatory aggressives. Extensive animal research on cats has demonstrated separate neurophysiological pathways mediating predatory versus affective aggression (Mirsky and Siegel, 1994; Siegel and Pott, 1988). In children, reactive aggressives relative to proactive aggressives are much more likely to have social information processing deficits and to have been physically abused (Dodge, Lochman, Harnish, & Bates, 1997). In adults, psychopathic criminals are much more likely to engage in predatory violence, while non-psychopathic violent criminals are more likely to engage in affective violent acts (Meloy, 1988; Serin, 1991; Williamson, Hare, & Wong, 1987; Meloy, 1995). Impulsive violent offenders differ from controls on event-related potential and neuropsychological measures of information processing deficits (Barratt, Stanford, Kent, & Felhous, 1997), low serotonin (Coccaro, Kawoussi, Sheline, & Lish, 1996; Linnoila, Virkkunen, Scheinin, Nuutila, Rimon, & Goodwin, 1983; Virkunnen & Linnoila, 1993), and monoamine oxidase A deficiency (Brunner, Nelen, Breakefield, & Ropers, 1993).

Despite this increasing support for the predatory–affective distinction, surprisingly little is known about the neurophysiological and neuroanatomical factors that characterize predatory versus affective aggression in humans. Indications can nevertheless be gleaned from the literature on the cortical and subcortical mechanisms thought to be involved in aggression and violence *per se*. At a cortical level, the prefrontal cortex has been viewed as critically important in the modulation of aggression, and this notion has been supported in the past by neurological studies of patients with damage to the prefrontal cortex (Damasio, Tranel, & Damasio, 1990; Weiger, & Bear, 1988). Recent brain imaging studies are now beginning to confirm the role of the prefrontal cortex in modulating and controlling violence in humans (Goyer, Andreason, Semple, Clayton, King, Compton-Toth, Schulz, & Cohen, 1994; Volkow, Tancredi, Grant, & Gillespie, 1995; Raine & Buchsbaum, 1996; Raine, Buchsbaum, Stanley, Lottenberg, Abel, & Stoddard, 1994; Raine, Buchsbaum, & LaCasse, 1997).

Subcortically, four structures which are viewed as important with respect to aggression consist of the amygdala, hippocampus, midbrain area, and thalamus. Experimental animal research together with neurological studies of patients have implicated the amygdala and hippocampus in modulating aggression (Bear, 1991; Elliott, 1992; Mirsky & Siegel, 1994; Watson, Troiano, Poulakos, Weiner, Block, & Siegel, 1983), while the thalamus also provides an important afferent source of the hypothalamic-induced attack in cats (Mirsky & Siegel, 1994). Similarly, stimulation and lesion studies of the midbrain central gray has been repeatedly linked to the expression of affective aggression (Shaikh, Barrett, & Siegel, 1987; Mirsky & Siegel, 1994). Nevertheless, such research on animals and humans who have suffered brain insults, while of key importance, is one step removed from the question of whether severely violent offenders have brain dysfunction localized to specific subcortical brain areas.

Traditionally, subcortical and limbic regions of the brain have been viewed as involved in the generation of aggressive feelings and behavior, while the prefrontal cortex is viewed as inhibiting and modulating these basic emotions (Weiger & Bear, 1988). Consequently, it could be argued that what may be critically important in predisposing to violence is the *relative balance* of activity between the prefrontal and subcortical brain regions. If prefrontal functioning is reduced relative to subcortical activity (or if the subcortex is unusually active relative to the prefrontal cortex), the individual may be more prone to violence in general, and perhaps affective violence in particular. Surprisingly, there appears to have been no proposition or test of such a relatively straightforward hypothesis.

To our knowledge, there has been no previous brain imaging research that has explicitly addressed the issue of predatory versus affective aggression, and furthermore no imaging research on subcortical functioning in violent offenders outside of our recent work (Raine *et al.*, 1997). The current study set out to assess differences between affective and predatory murderers in cortical and subcortical brain functioning. Specifically, we tested the hypotheses that an affective subgroup of violent offenders in particular are characterized by (i) low prefrontal functioning, (ii) high subcortical functioning, and (iii) lower prefrontal-to-subcortical functioning, whereas predatory violent offenders would show relatively normal brain functioning relative to comparisons.

METHOD

Subjects

Full subject details are reported by Raine *et al.* (1997). Briefly, subjects consisted of 41 individuals who had been tried for murder or attempted murder in the state of California and 41 age and sex matched normal controls. All murderers pleaded either not guilty by reason of insanity (NGRI) or incompetent to stand trial (IST). Offenders were not receiving regulated psychoactive medication at the time of PET scanning, and were instructed to be completely medication free for the 2 week period preceding brain scanning. Urine screens for drug use at the time of PET scanning were negative for every murderer. Normal comparisons had been screened for health by physical exam, medical history, and psychiatric interview.

No comparison subject was taking any medication, had a history of psychiatric illness in self or first-degree relatives, or had significant medical illness. Comparison subjects with a history of seizure disorder, head trauma, or substance abuse were excluded. Subjects participated under protocols and consent forms approved by the Human Subjects Committee of the University of California, Irvine.

Rating of Affective Versus Predatory Murders

Two raters blind to both glucose values and each other's assessments rated each of the 41 murderers on a four-point scale of predatory–affective violence. Sources used to determine ratings included the following: (i) assessment reports from psychologists and psychiatrists; (ii) criminal transcript history; (iii) telephone interviews with prosecuting or defending attorneys; (iv) preliminary hearing transcripts; (v) medical records; (vi) national and local newspaper reports. Interrater reliability (r) for this four-point scale on the total sample was .72 ($p < .0001$).

As expected, it was difficult to rate some participants as either clearly affective or clearly predatory in their violent acts. Therefore, to improve classification, reliability was recomputed after retaining only those individuals scoring 1 (strongly predatory) or 4 (strongly affective). Increase in agreement between raters was confirmed by a coefficient kappa on this reduced sample calculated as .86. Remaining inconsistencies between raters were resolved through consensus and a single rating was used to define participants as either affective or predatory. Consequently, in the analyses below, 15 participants formed the Predatory group of murderers and nine formed the affective group, with 41 normal comparisons.

PET Task Procedure

Full details of general PET scanning procedures and quantification may be found in Buchsbaum, Nuechterlein, and Haier (1990). Briefly, the flurodeoxyglucose (FDG) tracer was injected into the subject in the test room and taken up by the brain as a tracer of brain metabolic rate for a 32 min period during which the subject completed the continuous performance task (CPT; Nuechterlein, Parasuraman, & Jiang, 1983). A degraded stimulus version of the CPT was employed as the frontal challenge task because it has been shown to produce increases in relative glucose metabolic rates in the frontal lobes in normal controls, in addition to increases in right temporal and parietal lobes (Buchsbaum *et al.*, 1990). The key signal detection performance measure of d' reflects target recognition accuracy across the 32 min period (Davies and Parasuraman, 1982; Nuechterlein, 1991). Split-half reliability for the task is high ($r = .843$, $p < .001$). Full procedural details are reported by Buchsbaum *et al.* (1990).

Ten minutes before the FDG injection, subjects were given practice trials on the CPT. Thirty seconds before injection, the task was started so that initial task novelty would not be FDG labeled. After 32 min of FDG uptake, the subject was transferred to the adjacent PET scanner room. An individually molded, thermosetting plastic head holder was used to hold the head still during the scan. Ten slices at 10 mm intervals parallel to the canthomeatal line were obtained. Scans

started at the level of 80% of head height above the canthomeatal line (vertex to canthomeatal line, usually 12–14 cm) and step downward at 10 mm intervals.

Brain regions were identified using two techniques as follows:

(i) *Cortical peel technique (lateral areas).* Surface cortical regions of interest were measured using a modification of the original cortical peel technique (Buchsbaum, DeLisi, & Holcomb, 1984) with the four lobes and four anatomical subdivisions of each identified stereotactically (Buchsbaum, Gillin, & Wu, 1989). This technique has been used by at least nine different PET groups, and a review of its advantages for facilitating intrasubject and intersubject differences may be found in by Harris, Links, Pearlson, & Camaigo (1991). Absolute glucose values for each region of interest were expressed as a measure relative to all other regions contained in that slice. Relative rather than absolute metabolic rates were used because relative rates are more widely reported, have the advantages of removing whole brain metabolic rate, are more likely to be related to function in specific neuroanatomical systems (Fox and Mintum, 1989), and show greater reliability within subjects over time (Bartlett *et al.*, 1991). The following three prefrontal values (averaged across slices) for each hemisphere were extracted: superior frontal gyrus, middle frontal gyrus, and inferior frontal gyrus.

(ii) *Box technique (medial areas).* Medial cortical and subcortical regions of interest were located on PET slices by reference to stereotaxic coordinates as detailed by Buchsbaum *et al.* (1989). A 3 × 3 pixel region-of-interest box was placed on cortical and subcortical structures at each level, according to a standard list (see Raine *et al.*, 1994, for further details). As each pixel measured 2 mm × 2 mm, the size of the region-of-interest box was approximately one full-width at half-maximum. Prefrontal measures extracted from each slice level (given as a percentage of the distance from the external auditory meatus to the top of the head) according to a brain atlas (Matsui and Hirano, 1978) were as follows: superior frontal gyrus (average of 80%, 74%, 68%, 61% slice levels—see Raine *et al.*, 1994, for further details), anterior medial frontal gyrus (68% level), medial frontal gyrus (average of 61%, 54%, 47% levels), and orbital gyrus (21% level). Subcortical areas were also assessed as follows: medial temporal lobe including the hippocampus (average of 34%, 28%, 21% levels), amygdala (21%), thalamus (41%), and midbrain (21%).

To assess stereotaxic error due to individual differences in structure location within the plane, we evaluated the stereotaxic frame based on the brain outline. Stereotaxic error could place boxes in the caudate into the ventricle, thereby diluting metabolic rates with cerebrospinal zero rates, but confidence limits based on application of the current system to magnetic resonance images confirm 2 SD limits within the caudate (Buchsbaum, Potkin, Marshall, & Lottenberg, 1992).

Overall Brain Functioning Variables

For both cortical and sub-cortical analyses, values were averaged across slices for each of the two hemispheres separately to produce for each hemisphere one

measure of each of the following: (i) lateral prefrontal; (ii) medial prefrontal; (iii) subcortical; (iv) lateral prefrontal/subcortical ratio; (v) medial prefrontal/subcortical ratio.

RESULTS

Data were analyzed using a 3 (controls, predatory, affective) × 2 (left and right hemispheres) repeated measures multivariate analysis of variance using the MANOVA approach in SPSS. Separate analyses were conducted on (i) prefrontal, (ii) subcortical, and (iii) prefrontal/subcortical ratios. Interactions were broken down using univariate F tests and two-tailed ($p < .05$) t-test comparisons.

Prefrontal Functioning

Lateral

There was a main effect for group, $F(2, 62) = 6.5$, $p < .003$, a main effect for hemisphere $F(1, 62) = 21.8$, $p < .0001$, but no group × hemisphere interaction ($p > .62$)—see Figure 1. A breakdown of the main effect for group (averaging across hemispheres) indicated that affective murderers had significantly lower lateral prefrontal functioning than comparisons ($t = 3.7$, $\mathrm{df} = 48$, $p < .001$). Predatory murderers did not differ significantly from comparisons ($p > .17$) or affective murderers ($p > .07$).

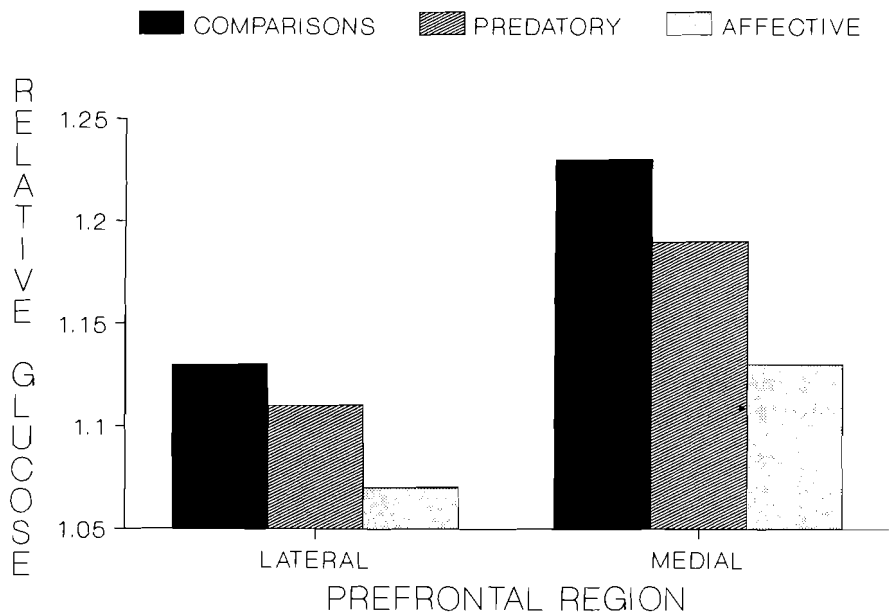

Figure 1. Lower lateral and medial prefrontal glucose metabolism in affective murderers

Medial

There was a main effect for group, $F(2, 62) = 6.3$, $p < .003$, a main effect for hemisphere, $F(1, 62) = 4.6$, $p < .04$, but no group × hemisphere interaction ($p > .76$)—see Figure 1. A breakdown of the main effect of group (averaging across hemispheres) indicated that affective murderers had significantly lower medial prefrontal functioning compared to comparisons ($t = 3.2$, $df = 48$, $p < .002$). Predatory murderers did not differ significantly from affective murderers ($p > .17$) but did differ from comparisons ($t = 2.1$, $df = 54$, $p < .04$).

Subcortical Functioning

The main effect for group was non-significant ($p < .18$), as was the main effect for hemisphere ($p < .19$). There was, however, a significant group × hemisphere interaction, $F(2, 62) = 8.1$, $p < .001$—see Figure 2. A breakdown in this interaction indicated no group effect for the left hemisphere ($p > .76$), but a group effect for the right hemisphere, $F(2, 62) = 3.4$, $p < .04$. As indicated in Figure 2, the affective murderers had levels of right subcortical glucose metabolism ($p > .46$) that were significantly higher than comparisons ($t = 2.9$, $df = 48$, $p < .005$). Predatory murderers had right subcortical glucose levels which did not differ to those of affective murderers ($p > .46$), and which were significantly higher than comparisons ($t = 2.4$, $df = 54$, $p < .02$). In contrast, predatory murderers had non-significantly lower left hemisphere functioning than comparisons.

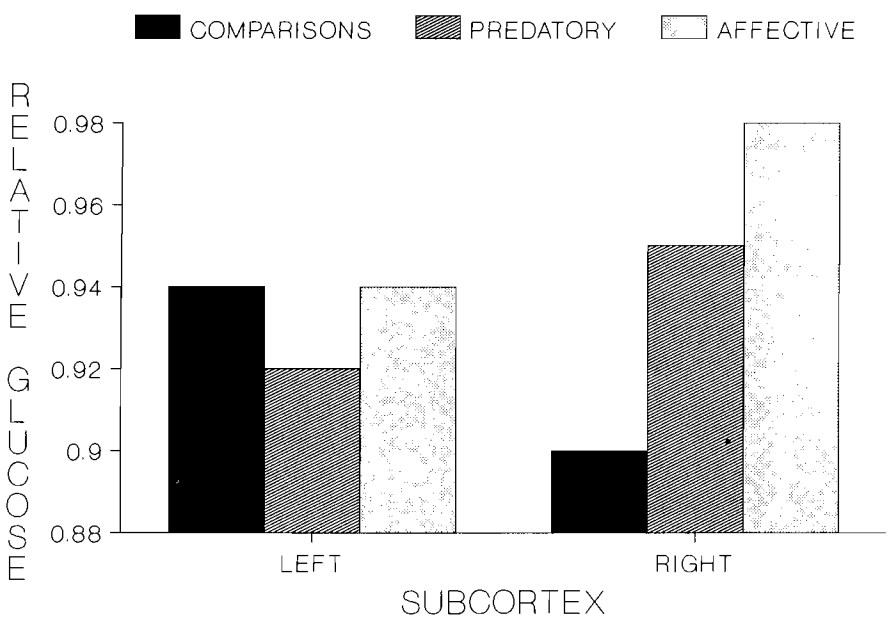

Figure 2. Increased right hemisphere subcortical glucose metabolism in both affective and predatory murderers

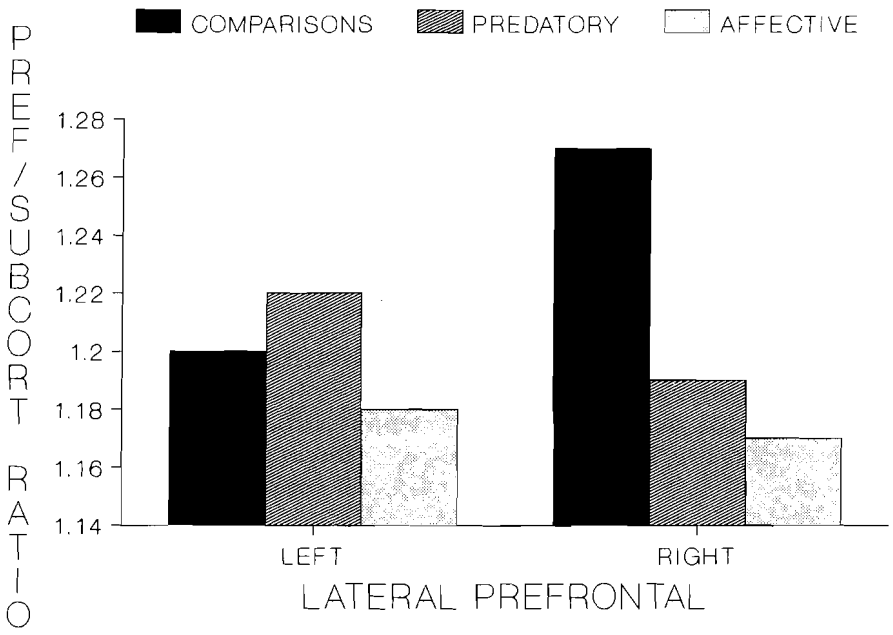

Figure 3. Lower right lateral prefrontal/subcortical glucose metabolic ratios in affective murderers

Prefrontal/Subcortical Ratios

Lateral Prefrontal/Subcortical Ratio

There was a significant main effect of group, $F(2, 62) = 4.1$, $p < .02$, no effect for hemisphere ($p > .47$), but a significant group x hemisphere interaction, $F(2, 62) = 6.2$, $p < .003$—see Figure 3. A breakdown of this interaction indicated no group differences for the left hemisphere ($p > .68$), but a group effect for the right hemisphere, $F(2, 62) = 3.8$, $p < .03$. As indicated in Figure 3, affective murderers had significantly lower right prefrontal/subcortical ratios than comparisons ($t = 3.4$, $df = 48$, $p < .002$). Predatory murderers showed a lower right prefrontal/subcortical ratio relative to comparisons ($t = 2.3$, $df = 54$, $p < .03$), with no difference between predatory and affective murderers ($p > .26$).

Medial Prefrontal/Subcortical Ratio

There was a significant main effect of group, $F(2, 62) = 4.7$, $p < .02$, a main effect for hemisphere, $F(1, 62) = 5.3$, $p < .03$, and a significant group x hemisphere interaction, $F(2, 62) = 4.2$, $p < .02$—see Figure 4. A breakdown of this interaction indicated no group differences for the left hemisphere ($p > .80$), but a group effect for the right hemisphere, $F(2, 62) = 4.1$, $p < .02$. As indicated in Figure 4, affective murderers had significantly lower right prefrontal/subcortical ratios than comparisons ($t = 3.1$, $df = 48$, $p < .003$). Predatory murderers also had significantly lower right prefrontal/subcortical ratios than comparisons ($t = 2.6$, $df = 54$, $p < .02$), with no difference between affective and predatory murderers ($p > .30$).

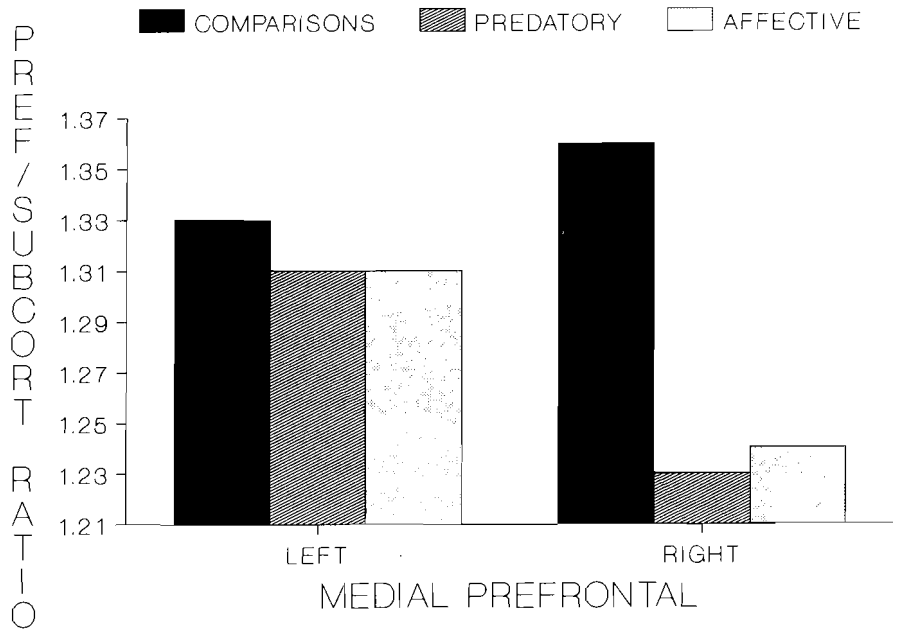

Figure 4. Lower right medial prefrontal/subcortical glucose metabolic ratios in both affective and predatory murderers

Behavioral Performance on the Continuous Performance Task

We have previously shown that murderers did not differ from controls with respect to behavioral performance on the CPT. It is possible, however, that subgroups of murderers differ on CPT performance and that such performance differences account for difference in brain functioning as indicated by PET. Means and SDs for d' for murderer subgroups were as follows: predatory murderers, $M = 3.51$, $SD = .72$; affective murderers, $M = 3.14$, $SD = .77$. There was no significant difference between the groups ($t = .3$, $df = 18$, $p > .75$).

Effect Sizes

Averaged effect sizes (d; Cohen, 1988) for differences between affective murderers and comparisons were 1.27 for the prefrontal cortex, 1.07 for the right subcortex, and 1.20 for right prefrontal/subcortical ratios. Effect sizes for differences between predatory murderers and comparisons were .72 for the right subcortex and .74 for right prefrontal/subcortical ratios.

DISCUSSION

The key findings in this study are: (i) Affective murderers have lower prefrontal activity and higher subcortical activity than comparisons. (ii) Predatory murderers have prefrontal activity levels similar to comparisons, but had excessive subcortical

activity. (iii) The excessive subcortical activity in both affective and predatory murderers was restricted to the right hemisphere. Effect sizes were large, ranging from 1.07 to 1.27 for differences between affective murderers and comparisons (Cohen, 1988). Prefrontal functioning was reduced by an average of 7.1% in affective murderers compared to comparisons, while subcortical functioning was increased in affective murderers by 8.7%. Group differences were not a function of behavioral performance differences on the cognitive challenge task. These findings appear to be the first brain imaging data of any type on affective versus predatory violent offenders. We propose a neurophysiological theory based on a cortical/subcortical gradient of activation to explain these findings.

With respect to prefrontal deficits in affective murderers, this finding is consistent with the notion that damage to this brain region can result in impulsivity, loss of self-control, immaturity, altered emotionality, and the inability to modify behavior, all of which can in turn facilitate impulsive aggressive acts (Damasio, 1985, Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Moffitt and Henry, 1991; Stuss and Benson, 1986; Weiger and Bear, 1988). In addition to its modulatory role, the prefrontal cortex is involved in the interpretation of sensory stimuli and the potential for danger (Kalin, 1993). Consequently, poor prefrontal functioning may result in affective aggressives misinterpreting environmental and situational stimuli as dangerous and threatening, which in turn results in seemingly inappropriate, unreasoned violent behavior that is in effect a pre-emptive strike against a perceived threat.

In contrast, the predatory group were less likely to suffer from these prefrontal deficits. One implication of this finding is that it may help to resolve conflicting findings on the neuropsychological basis of violence. For example, there is considerable debate as to whether violent psychopathic criminals suffer from prefrontal dysfunction measured through neuropsychological tests (Hare, 1984; LaPeirre, Braum, and Hodgins, 1995; Gorenstein, 1982). Because the majority of psychopaths show predominantly predatory as opposed to affective aggression (Meloy, 1988; Williamson, Hare, and Wong, 1987; Serin, 1991), the current findings which indicate relatively normal prefrontal metabolism in predatory murderers would predict that psychopaths as a whole would not show prefrontal dysfunction, but that a minority of psychopaths showing affective violence would more clearly show these deficits. Similarly, future neuropsychological research on violent offenders should differentiate between affective and predatory forms of violence in order to test the prediction that affective violent offenders are the specific group with prefrontal dysfunction.

The affective and predatory murderers both showed subcortical deficits. Subcortical abnormalities in the amygdala, hippocampus, thalamus, and midbrain have been less discussed in the human literature than cortical deficits because subcortical structures are not measured by neuropsychological and psychophysiological techniques which have been used in most previous physiological research on violence in intact humans. Nevertheless, subcortical abnormalities in these areas are theoretically consistent with previous neurological and animal research. The amygdala has been repeatedly associated with aggressive behavior in both animals and humans (Bear, 1989; Mirsky and Siegel, 1994; Weiger and Bear, 1988). In particular, stimulation of the medial (Shaikh, Steinberg, & Siegel, 1993), lateral basomedial (Adamec, 1990), and basal regions of the amygdala have been variously

implicated in facilitation of both predatory and affective attacks in cats, while stimulation of the ventral hippocampus facilitates predatory attack (Adamec, 1991; Adamec and Stark, 1983). The amygdala, hippocampus and prefrontal cortex make up part of the limbic system governing the expression of emotion, while the thalamus relays inputs from subcortical limbic structures to the prefrontal cortex (Fuster, 1989; Mirsky and Siegel, 1994). The hippocampal formation is thought to modulate aggression in cats through its action on the lateral hypothalamus via the lateral septal area (Mirsky and Siegel, 1994; Siegel and Flynn, 1968); together with the septal area and prefrontal cortex, it forms the neurobiological basis of the behavioral inhibition system of Gray (1982), which is theorized to be dysfunctional in violent and psychopathic individuals (Gorenstein and Newman, 1980). The amygdala is believed to act on the medial hypothalamus through at least two pathways in the modulation of aggression in animals (Watson *et al.*, 1983). The hippocampus, amygdala, and thalamus are also of critical importance to learning, memory, and attention; stimulation and lesion studies in animals indicate that they relate to deficits in forming conditioned emotional responses (LeDoux, 1994) and the failure to learn from experience displayed by criminal and violent offenders (Raine, 1993).

Murderers only showed subcortical deficits in the right hemisphere. In humans, activation of the right hemisphere has been implicated in the generation of negative affect (Davidson and Fox, 1989). In animals, rats who are stressed early in life are right hemisphere dominant for mice-killing (Garbanati, Sherman, Rosen, Hofmann, Yutzey, & Denenberg, 1983). It is possible therefore that, depending on presence of other social triggers and early stressful environmental circumstances, increased right hemisphere subcortical activity could predispose the individual to experience negative affect which fosters aggressive feelings and which in turn act as a general predisposition to violent behavior.

The predatory murderers were more equivalent to comparisons than affective murderers in terms of prefrontal functioning. This is consistent with the view that such offenders have relatively intact ability to plan and regulate their aggressive behavior in order to achieve desired goals. This leaves unanswered the question of why such individuals are overly violent in the first place. Part of the answer may be that predatory murderers had higher right subcortical activity than comparisons, and also had lower right medial prefrontal/subcortical ratios than comparisons. Excessive subcortical activity could be a contributing factor towards a more aggressive temperament which is common to both types of violent offender, particularly since increased activity of the ventral hippocampus and lateral aspect of the basomedial amygdala have been associated with increased predatory attack in cats (Adamec and Stark, 1983; Adamec, 1991). However, we hypothesize that affective and predatory offenders differ in terms of the regulatory cortical control they exert over such impulses. While predatory violent offenders have sufficient left prefrontal functioning to modulate such aggressive behavior in a way to bully and manipulate others to achieve desired goals, affectively violent offenders lack this prefrontal modulatory control over their impulses, resulting in more unbridled, dysregulated, aggressive outbursts.

It is important to clarify the strengths and limitations of this study so that findings can be accurately viewed. One limitation of the study is the relatively small *N* sizes for sub-groups with the potential for type II error. On the other hand,

findings were statistically significant with large effect sizes being obtained in the expected direction, while additionally there are no other brain imaging studies on reactive versus predatory aggression. Second, findings cannot currently be generalized at the present date from NGRI murder cases to other types of violent offenders in the community. Nevertheless, this is a group of severely violent offenders who are of particular importance in forensic psychiatry, and findings provide both theoretical directions and a crucial empirical base upon which future brain imaging may build. Third, knowledge on brain functioning in humans is crude relative to finer neurophysiological research in animals due to limitations in spatial resolution in PET. Consequently, the neurophysiological interpretation of findings is by necessity rudimentary. In particular, little is known about asymmetries in subcortical glucose metabolism in humans and prefrontal/subcortical ratio measures. It is hoped, however, that the theoretical directions provided here may provide a starting point for more sophisticated brain imaging research in larger samples of violent offenders.

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