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Integrating the Somatic Marker and Social Cognition Theories to Explain Different Manifestations of Antisocial Personality Disorder

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Abstract ~ Despite an extensive body of research examining brain-behavior relationships underlying Antisocial Personality Disorder (APD), the findings have neither been consistent in terms of the strengths of these relationships nor the underlying mechanisms or processes being studied. This is because APD is comprised of a heterogeneous constellation of symptoms, and includes dimensions of implicit personality characteristics (lacking empathy and egocentricity) and explicit behaviors (impulsivity and poor behavioral control), which in turn are driven by cognitive (poor executive functioning and inhibition) and affective (lack of emotion) deficits. Dinn and Harris (2000) suggest that different

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manifestations of APD are best explained by deficits in different parts of an interactive network, as opposed to localized areas in the frontal lobe or amygdala. This paper argues that two theories in particular are useful for understanding this neuropathophysiology, and how dysfunction in different areas of the brain accounts for various manifestations of APD: 1) Damasio's (1996) Somatic Marker Hypothesis, and 2) Baron-Cohen's (1998) Social Cognition Model.

Introduction

Neuropsychological research examining the etiology of Antisocial Personality Disorder (APD, DSM-IV-TR, 2000) has focused almost exclusively on testing various localization hypotheses, the majority of which have centered on the frontal lobes and their role in executive function and inhibiting impulsive behavior (Dinn & Harris, 2000; Brower & Price, 2001). Although there is a robust body of research to support the association between specific regions of the frontal lobes (e.g., orbitofrontal, ventromedial and ventrolateral regions of the frontal cortex) and the propensity for aggression and APD behaviors, there has been some inconsistency with respect to the strength of this relationship.

Brower and Price (2001) recently conducted a meta-analysis of the literature, and concluded that while the association between frontal lobe dysfunction and APD behaviors/symptoms (e.g., aggression and violent behavior) was supported, the general strength of this relationship was unknown, as were the underlying processes and structures involved (cognitive versus affective, local versus diffuse). Damasio's (1996) *Somatic Marker Hypothesis* and Baron-Cohen's (1998) *Social Cognition Model* are particularly useful in understanding these various manifestations and dimensions of APD, and how they relate to different underlying neurological systems.

Localization Research

A number of studies implicate the frontal lobes in the neuropathophysiology of APD. According to Benson and Miller (1997), the frontal lobes are complicated neural networks that are responsible for the regulation of behavior. When damage is done to these frontal networks, greater irritability, aggressiveness, and violent behavior may result. In their study of frontal lobe functioning, Benson and Miller (1997) looked at two groups: a sample of people with APD known to have committed violent crimes, and a control group. The two groups were matched according to socioeconomic, psychological, and neurological status. The results of the study revealed frontal lobe injury (per review of neurological history) in a significantly higher percentage of the criminal group than the control, particularly in the orbitofrontal cortex. It was thus concluded that the frontal lobe plays a significant role in the regulation and inhibition of behavior associated with APD and damage to this cortex was a primary catalyst for antisocial behavior (Benson & Miller, 1997).

Another study conducted by Raine et al. (1994) assessed the rate of glucose metabolism in the frontal lobe region of the brain using Positron Emission Tomography (PET) scans. They examined a group of 22 subjects accused of having committed violent murder, and compared them to a control group of 22 subjects matched for age, socioeconomic status, and gender. Results showed that the experimental group (violent murderers) displayed significantly lower rates of glucose metabolism in the prefrontal cortex relative to the control group, supporting the contention that APD behaviors are strongly associated with deficits in the frontal lobe. Using the same methodology, Volkow et al. (1995) replicated this study, although found that for some of those with APD the region of deficit was the frontal lobe, while for others it was the temporal lobe.

Using electroencephalogram (EEG) analysis, Deckel,

Hesselbrock, and Bauer (1996) conducted a study at the University of Connecticut, examining whether brain electrical activity predicted APD and retrospective ratings of childhood problem behaviors characterized by conduct disorder. Regression analysis indicated frontal lobe activity was inversely related to the likelihood of being diagnosed with APD or with childhood problem behaviors. Similarly, Raine, Lencz, Bihrle, LaCasse, & Colletti (2000) measured gray and white matter volume in the prefrontal region of the brain across different groups using Magnetic Resonance Imaging (MRI), and found that APD subjects had 11% less gray and white matter and displayed significantly lower levels of autonomic arousal (stress response) when reading a self-critical speech than matched control groups.

Using a different approach, Dinn and Harris (2000) administered a battery of neuropsychological and cognitive tests sensitive to frontal lobe functioning to patients diagnosed with APD and matched control subjects. Participants' electrodermal stimulation when presented with emotionally charged stimuli was also measured. As hypothesized, APD participants displayed significantly greater deficits on measures of orbitofrontal functioning, and were "electrodermally hyporesponsive to aversive stimuli" (Dinn & Harris, 2000, p. 173) when compared to the control group. However, on classical tests of frontal lobe executive functioning, which implicate the dorsolateral prefrontal cortex, the APD group did not differ significantly from the control group. Additionally, on tests of reaction time and self-report measures of phobia, the APD and control groups did not differ significantly. Dinn and Harris explain this as both interesting and contradictory because APD groups have classically been thought of as having global deficits in executive functioning, fear response, and learning, while their approach seems to indicate more specific dysfunction of the orbitfrontal sub-region of the prefrontal cortex. They describe the frontal lobes as being a highly

complex system of multiple, discrete sub-systems driving behavior and functioning in different ways, which may in turn account for different manifestations of APD.

Using Theory to Conceptualize APD as a Heterogeneous Disorder

To better account for the research presented above, Dinn and Harris (2000) argue that APD is a heterogeneous set of disorders, involving a network of cortical-subcortical structures which influence both affective and cognitive propensities. APD dimensions include both implicit personality characteristics (e.g., lacking empathy and egocentricity) and explicit behaviors (e.g., impulsivity and poor behavioral control - Hart et al., 1995). According to the Diagnostic and Statistical Manual of Mental Disorders (DSM IV-TR, 2000), a person must satisfy three out of seven criteria in the first cluster, which includes dimensions of impulsivity (executive functioning and behavioral control component) and a lack of empathy (affective component) (American Psychiatric Association, 2000). As a consequence of these multiple symptom clusters and dimensions, it is possible for diagnoses of APD to be based on criteria from one dimension exclusively (e.g., impulsivity) without much consideration given to the others, resulting in different manifestations of APD.

These different manifestations have been broadly categorized as "Reactive" versus "Instrumental" APD (Blair, 1997), "Primary" versus "Secondary" APD (Wiebe, 2004), and "Predatory" versus "Impulsive" APD (Volvaka, 1999). Dinn and Harris (2000) suggest that different manifestations of APD are best explained by deficits in different parts of an interactive network, or circuit, as opposed to localized areas in the frontal lobes mentioned above. Two theories are particularly useful in detailing this cortical-subcortical network, specifically in terms of how damage to different areas accounts for different manifestations of APD: (1) Damasio's (1996) Somatic Marker Hypothesis, and (2) Baron-

Cohen's (1998) Social Cognition Model, also known as Theory of Mind (ToM).

Somatic Marker Hypothesis

The Somatic Marker Hypothesis assumes that there is a cortical-subcortical system (including the frontal lobes and limbic system) involved in decision-making, which is driven to a large extent by affect. According to Bechara, Damasio, and Damasio (2000), there are several assumptions underlying the model, which include that "cognitive operations depend on support processes such as attention, memory, and emotion… and that reasoning and decision making depend on the availability of knowledge about situations" (p. 296). This information is stored both cognitively and emotionally. The emotional component provides a dispositional, or visceral "marker" that guides all subsequent experience. Central to the Somatic Marker Hypothesis as it pertains to APD is the assumption that damage to specific frontal lobe areas (i.e., ventromedial prefrontal region and/or regions of the amygdala) results in an inability to "experience somatic states associated with both positive and negative affect" (Dinn & Harris, 2000, p. 185).

In terms of the underlying structures involved, Damasio and his colleagues argue that the prefrontal cortex is the primary region that integrates all sensory/perceptual, emotional, and affective information into a cognitive amalgam (Damasio, 1994; Damasio, 1996; Bechara, Damasio, & Damasio, 2000). When people experience new situations, which include elements that have already been organized and classified affectively, past dispositions (or markers) are activated to guide subsequent cognition and decision-making. For example, some experiences already marked as "bad" set off somatic states (e.g., a feeling of dread in the pit of the stomach) that act as a signal in guiding subsequent experience, alerting the person to the impending negative

outcome. According to Dinn and Harris (2000), "Without emotional coloring to guide action, decision-making, particularly in the social domain, becomes problematic" (p. 185).

To test this relationship, Bechara, Tranel, and Damasio (2000) used a variation of the "gambling task", originally developed by Bechara, Damasio, Damasio, and Anderson (1994), comparing ventromedial (VM) lesion patients to a control group. This gambling task was developed to mimic real-life circumstances, where behavior is guided by reward and punishment contingencies. Specifically, "advantageous" card decks were constructed to first yield immediate high punishment and then high reward over time, and a "disadvantageous" card deck was constructed to do the opposite (first yield immediate high reward, and then high punishment over time). The overall goal of the task is to make as much money as possible. Skin conductance was recorded each time a card was drawn as a measure of autonomic nervous activity, and an index of the extent to which somatic markers were guiding subsequent decision-making.

Results showed that although VM lesion patients preferred the disadvantageous card decks because of the immediate rewards, and their levels of skin conductance were not significantly different than the control group after they received an initial reward or punishment. The authors extrapolated from their neurological data to develop a pathophysiological model of APD. They argued their findings could provide evidence that people with APD characteristics are not concerned with future consequences, but rather only immediate punishment/reward. Additionally, as the control group became more experienced with the game, they began to generate anticipatory skin conductance responses prior to selecting the card indicating they were beginning to react to imagined scenarios. That is, they were looking into the future, and were reacting to potential contingencies rather than actual contingencies. The VM lesion patients did not

generate these anticipatory skin conductance responses, however. That is, both groups experienced the same levels of autonomic arousal with respect to the immediate rewards/punishment. However, only the VM group was unable to learn (guided by internal somatic states) and integrate this into a larger executive template capable of anticipating the future and altering behavior accordingly to achieve the most beneficial outcome over time (Bechara et al., 2000).

Van Honk, Hermans, Putnam, Montagne, and Schutter (2002) replicated this study with 32 college students, selected a priori from a larger database in order to provide a comparison between groups in terms of low and high APD behavioral characteristics. The researchers found that whereas low APD-symptom groups exhibited punishment learning, the high APD-symptom group did not. The investigators concluded that somatic markers in the low APD group were guiding subsequent decisions, but this process was impaired in the high APD group. As opposed to others (Damasio, 1996; Bechara et al., 2000; Damasio 2000), however, these researchers inferred that the dysfunction was localized to the orbitofrontal region of the frontal lobes specifically, in contrast to a complex network implicating many structures.

Damasio (2000) also discussed the study by Raine et al. (2000) (described above) in terms of providing support for the Somatic Marker Hypothesis. Results from this study showed that when asked to read a self-prepared paper about their flaws and failures to the researcher, the APD group exhibited significantly reduced skin conductance responses and heart rates than the control group. Damasio (2000) suggests that this social stressor did not induce the same sort of autonomic activity commonly seen in normal populations because there is a selective affective deficit with these individuals, limiting secondary emotions "such as guilt and embarrassment" (p. 128). The difference between this

and the study by Bechara, Tranel, and Damasio (2000) had to do with primary versus secondary emotions. Whereas both groups reacted the same to initial rewards/punishments (producing primary emotions such as joy), the APD group was unable to retain this in the form of somatic markers for use in future experience. When secondary emotions were then assessed (e.g., guilt), a lacking in somatic markers (a product of learning from social situations) resulted in socially abnormal behavior. That is, the VM lesion patients were unable to use previous experience to anticipate new experience. Bechara, Damasio, and Damasio (2000) discussed lack of anticipatory ability as related to defects in fear conditioning.

Given the amygdala is critical to emotional processing, Bechara, Damasio, and Damasio (1999) also examined the role of the amygdala in decision making, and sought to understand its role relative to the VMPFC (ventromedial prefrontal cortex). Two groups of participants were selected for study in relation to a control group: (1) those with bilateral amygdala damage and no damage to the VM, and (2) those with bilateral VM damage and no damage to the amygdala. Using the same "gambling task" described above (Bechara et al., 1994), the investigators found that both groups were significantly more impaired on the gambling task when compared to a control group, and did not generate anticipatory skin conductance responses (SCR's) when decision-making involved taking risks. However, whereas the damaged VM group was able to generate skin conductance responses as a result of receiving an initial reward/punishment, the damaged amygdala group was unable to do so. Additionally, whereas the damaged VM group did become conditioned to a loud sound (measured with SCR), the damaged amygdala group did not.

This study not only provided the first evidence for the association between the VM prefrontal cortex and amygdala in generating

somatic states, but it also provided some idea as to how the structures differ. According to the researchers, both groups are impaired in their abilities to make decisions. The differences, however, are rooted in the fact that the damaged amygdala group is completely unable to attach any sort of affective or emotional label to a stimulus. The damaged VM group, on the other hand, are able to do so, but are unable to integrate "effectively all of the somatic state information triggered by the amygdala…" (Bechara et al., 1999, p. 5478). Amygdala lesioned and VMPFC lesioned patients may provide models for primary and secondary emotion dysfunction, respectively.

Adolphs (2001) also discussed the role of the amygdala as being critical to the emotional and affective labeling of perceived stimuli. He says, "Its principal function appears to be the linking of perceptual representations to cognition and behavior on the basis of the emotional or social value of the stimuli" (Adolphs, 2001, p. 233). Specifically, Adolphs argues that the amygdala plays a critical role in assessing threat or danger, and cites the fact that people with amygdala damage are unable to recognize impending danger because they are unable to ascribe a particular emotional valence to the situation based on certain cues (e.g. facial expressions, intent gazing, etc.). In essence, those with amygdala damage lack the somatic markers for recognizing these stimuli, and are more prone to opening themselves to these sorts of dangers as a consequence.

Social Cognition Model

In contrast to Damasio's Somatic Marker Hypothesis, which assumes that the individual with APD has intact social cognition but deficits in attaching emotional/somatic labels to stimuli coloring social cognition, the Social Cognition Model assumes that people with APD have marked deficits in social cognition (Adolphs, 2001; Adolphs, Baron-Cohen, & Tranel, 2002; Stone,

Baron-Cohen, & Knight, 1999). Social cognition refers to the ability of people to infer the mental states of other people, including their implicit motivations, thoughts, beliefs, and desires in the world. Ultimately, over the course of development and based on a multitude of experiences, people develop comprehensive "theory of mind," which are general paradigms from which to understand the perspectives of other people (Stone et al., 1999; Pellicano & Rhodes, 2003). Simon-Cohen and colleagues originally developed the Social Cognition Model for purposes of studying Autistic Spectrum Disorders in children and adults, and found that people with these disorders were significantly impaired in terms of how well they were able to conceive of others' state of mind (Baron-Cohen et al., 1996; Charman et al., 1997; Baron-Cohen, Wheelright, Hill, Raste, & Plumb, 2001).

The "Eyes Test" has been used as a tool for assessing the abilities of people to infer the mental states of others, and thus the accuracy of their "theory of mind", or perspective-taking ability (Baron-Cohen et al., 2001). Specifically, participants are presented with a set of 25 pictures containing only the "eyes" region of the face, and are asked to choose between two words that best describe the thoughts and feelings of the person in the picture. According to Baron-Cohen et al., (2001), at a rapid and subconscious level, people then sort and match these eyes to faces stored in memory, and make judgments as to the mental states of these faces accordingly. These workers argue the ability to read others' faces is the implicit propensity for "mind reading, social intelligence, and overlaps with the term 'empathy'" (p. 241).

Usually, by the age of four, most children have an understanding that people have alternate experiences of the world, evidenced by their ability to infer the mindsets of others by looking at the eye region of the face (Pellicano & Rhodes, 2003). They also realize that these experiences or realities may be the same or different

from their own, providing a base for theories of mind to develop. Among children and adults with autism, however, these perspective-taking abilities are markedly impaired, including the capacity to have empathy for other people who appear in distress (Baron-Cohen et al., 1996; Charman et al., 1997). Interestingly, these results have been replicated using auditory modalities (listening to a dialogue between two people, and inferring the mindsets of those involved using vocal cues), indicating theory of mind is amodal (Rutherford, Baron-Cohen, & Wheelright, 2002).

The neurological processes underlying social cognition are complex, and involve a cortical-subcortical circuit, including the amygdala and orbitofrontal regions of the cortex (Adolphs, 2001; Adolphs et al., 2002; Dinn & Harris, 2000; Stone et al., 1999). Damage to these areas results in significant impairment in social cognition (a primary deficit in those with APD) and the ability to make inferences about other people, although the underlying processes are different. In one study, Adolphs et al., (2002) administered a series of cards with pictures of people's faces to subjects with unilateral amygdala damage (n = 30), bilateral amygdala damage (n = 2), and a control group with other types of brain damage (n = 47). Results showed that subjects with amygdala damage exhibited significantly more deficits in recognizing the complex social emotions (thoughtfulness, flirtatiousness, boredom) of others as compared to the control group, despite whether the entire-face or the eyes-region-only cards were administered. However, there were no differences between groups in recognizing basic emotions (e.g., happiness, sadness). The researchers concluded that the amygdala plays a critical role in recognizing social cues, and subsequently regulating social behaviors. Using the "Eyes Test", Stone, Baron-Cohen, Calder, Keane, and Young (2003) found similar deficits among adults who suffered bilateral amygdala damage in adulthood.

Stone et al. (1999) studied the frontal lobe in relation to develop-

ing a theory of mind. In their study, the performance of patients with unilateral and bilateral orbitofrontal lesions was compared to patients with Asperger's Syndrome on tasks involving social cognition and theory of mind. Results showed that bilateral lesion patients exhibited the same social cognition deficits as the Asperger's patients, performing well on simple emotion-detection tasks, but poorly on detecting more complex and subtle emotions. However, the unilateral lesion patients did not display these deficits, although they did have difficulty on tasks involving working memory. The researchers concluded that the prefrontal cortex is an essential structure involved in theory of mind, and serves as an interpreter of the "valence and significance of others' actions and intentions" (Stone et al., 1999, p. 649).

According to Adolphs (2001), the prefrontal regions (orbitofrontal and ventromedial) of the cortex are involved in social cognition, and participate in "response selection, decision-making, and volitional control of behavior" (p. 234), the result of integrating and synthesizing input from multiple structures such as the amygdala. When there is damage to these areas, severe impairment in social behavior results, including the ability to organize and plan (impulsivity), and the ability to respond to punishment (learning). Additionally, inappropriate social manners and interaction, as well as a lack of concern and empathy for other people (social cognition) manifests. Central to this theory, however, is the notion that multiple, interacting structures are at work in perceiving and understanding the social cues of others. Adolphs (2001) presents three hypotheses addressing the underlying function of the amygdala (and other limbic structures) in relation to higher cortical structures: (1) "They may directly modulate cognition by virtue of their extensive connectivity with high-level cortex; (2) they may modulate emotional state, which in turn can be used to modulate cognition; and (3) they may directly modulate perceptual processing via feedback"

(p. 236), which subsequently affects cognition and the recognition of social cues.

Interestingly, Stone et al. (1999) cite a study by Saver and Damasio (1991), which found that patients with ventromedial lesions displayed no deficits in "abstract social knowledge, that is, these patients could figure out solutions for interpersonal problems between other people quite well" (p. 650). However, when these patients had to make decisions regarding their own lives, deficits emerged, indicating that there was an important discrepancy between abstract reasoning in general, and reasoning affectively when the situation was personally/socially relevant. That is, the latter situation necessitated a certain amount of empathy and affect which informed and shaped subsequent social cognition, whereas the former situation was purely theoretical, existing in space. This difference illustrates the important link between affect and social cognition, and the difference between reasoning in general (which may be intact) and social reasoning that requires an element of perspective-taking.

Integrating the Somatic Marker andSocial Cognition Theories

Both the Somatic Marker and Social Cognition theories advocate for a neurological circuitry as opposed to a distinct, localized region of the cortex in describing how people interact with others socially, both successfully and unsuccessfully (as is evidenced by those with APD). Interestingly, both theories assert a cortical-subcortical network, involving limbic structures such as the amygdala and regions of the frontal lobes. However, these two theories diverge from one another in terms of how these circuits potentiate different dimensions of APD. As mentioned above, Damasio's Somatic Marker Hypothesis assumes that the individual with APD has intact social cognition, evidenced by the ability to reason abstractly about social situations that are not per-

sonally relevant, but severe impairments in attaching emotional and somatic coloring to personally relevant social situations. In essence, people with APD lack the inner, somatic, affective markers (learned from past experience) that help inform social cognition and guide subsequent behavior. The amygdala is critical in assigning these somatic markers, whereas the frontal lobes integrate all of this information into a master organizational template which guides subsequent action. Damage to any of these areas impacts the network, but in different ways, potentially explaining the different sub-types of APD (e.g., impulsive vs. premeditated, reactive vs. instrumental).

Amygdala dysfunction is thought to result in the inability to attach somatic markers (and lack emotional coloring), although those with these deficits still have intact executive functioning and behavioral control. On the other hand, those with damage to the orbitofrontal and/or ventromedial regions of the cortex have difficulties integrating these somatic markers into a coherent amalgam. Amygdala lesions may explain what many have described as a lack of feeling (but no problems with impulse control) among those with certain APD sub-types (premeditated/ secondary APD). On the other hand, prefrontal lesions may account for impulse dyscontrol and lack of social awareness of other APD subtypes (impulsive/primary APD). The amygdala may modulate the implicit affective (or lack thereof) component, whereas the prefrontal cortex may account for the explicit impulse and behavioral dyscontrol. Based on where damage is done to this network, different manifestations of APD may result. The amygdala hypothesis may also explain why not all people with APD display poor impulse control, or completely lack empathy (some are just impulsive, but subsequently feel bad).

In contrast to the Somatic Marker Hypothesis, the Social Cognition Model assumes that people with APD have marked deficits in cognitive abilities, fueled by damage to various areas

of the network. As Adolphs (2001) has argued, the amygdala may be directly affecting cognition in the cortex, or may be impacting other systems (such as affect & emotion, as well as sensory-perceptual processing) which in turn impact social cognition. Either way, deficits in social cognition and awareness are manifest, and are rooted in an inability to form a "theory of mind" about other people. Lacking perspective-taking ability and the propensity for empathy often results in APD behavior, which may or may not be impulsive depending on the location of the damage to the network. Using the Somatic Marker Hypothesis, defective affect systems misinform an intact system of cognition, whereas with the Social Cognition Model a defective affect system generates a defective social cognition system. In either model, the outcome is the same with respect to APD. Deficits in “theory of mind” may result in a socially 'obtuse' form of APD of the reactive type, in which individuals lacking social awareness or effectiveness act out their feelings of frustration. The differences, however, are rooted in the structures and systems that are affected, in turn affecting APD sub-type.

According to the DSM IV-TR (American Psychiatric Association, 2000) diagnostic criteria for APD, three out of seven symptoms in the first cluster need only be met for a diagnosis. However, these symptoms include both impulsivity and irritability/aggression, and a general lack of remorse and taking pleasure in conning or deceiving others. Qualitatively, these distinctions represent very different manifestations of APD, and may reflect very different underlying neuropathophysiology. Used simultaneously, the Somatic Marker and Social Cognition models are useful in differentiating among these distinctions, as they both stress an underlying, neurological network, while at the same time differentiate between affective and cognitive dimensions of this disorder that subsequently fuel the propensity for empathy and impulse control. Deficits in one may not necessarily imply deficits in the other, thus giving rise to

different sub-types of APD.

Conclusion

Summary

APD is a highly variable and heterogeneous disorder, reflecting deficits in both affective and cognitive domains (Dinn & Harris, 2000). Although the Somatic Marker and Social Cognition models both implicate cortical (prefrontal cortex) and limbic (amygdala) structures in APD, they diverge with respect to the specific, underlying processes involved. Whereas Damasio's Somatic Marker Hypothesis assumes that people with APD have intact social cognition (evidenced by their abilities to reason abstractly about social situations in socially acceptable ways), Baron-Cohen's Social Cognition Model asserts these people have deficits in their abilities to reason at all about social situations, specifically with respect to developing theories of mind about others. The former model assumes deficits in the ability to emotionally tag, or label social stimuli (e.g. people's eyes or faces), so people subsequently lack the internal cues to respond in socially acceptable ways. On the other hand, the latter model assumes people are able to tag this information, but are unable to organize it into higher-order cognition. Structures such as the amygdala appear to be involved in the emotional organization of stimuli, whereas higher structures such as the prefrontal cortex are involved in the integration and synthesis of all this information. Although both models implicate the same structures and different processes, both are appealing in terms of how they address different sub-types of APD.

For example, research has established that not all people diagnosed with APD display impulsive behavior, nor do they all completely lack emotion/affect. Rather, there appears to be some variability in these traits. One explanation for this variability may be in the underlying processes involved. Those with impulsive

sub-types of APD may be compromised in terms of their pre-frontal cortex's ability to modulate behavior (resulting in impulsivity), whereas those characterized by under-arousal and a general lack of social affect may be compromised in terms of the amygdala's ability to encode emotional information or the pre-frontal cortex ability to properly integrate this information. Conceptualizing APD as a heterogeneous disorder is helpful as it allows for multiple pathways and processes to explain various manifestations of APD (Dinn & Harris, 2000).

Additional Considerations in Conceptualizing APD

Although they do not fit neatly within the purview of the Somatic Marker Hypothesis and Social Cognition Model, other variables should also be considered when conceptualizing APD. These include neurotransmitter systems and genetic predisposition (Johnson, 1996; Brown & Linnoila, 1990; Maeyer, Seif, Cases, & Gaspar; 1997; Suomi, 1984). For example, research has shown a significant association between disturbances in serotonin levels in the brain, and impulsive homicide and arson (Brown & Linnoila, 1990). Serotonin levels and social behaviors have also been studied in Ververt monkeys, showing that when drugs were given to inhibit serotonin levels in the brain a higher frequency of antisocial behaviors resulted (Suomi, 1984). Conversely, when serotonin levels were stimulated, higher frequencies of grooming and proactive social behaviors resulted. Twin and adoption studies have also demonstrated strong genetic components both in personality and criminality (Gottesman, 1997). Gottesman (1997) argues that one-fifth to one-half of the differences within a population in such traits as aggression, criminality, and the diagnosis of APD is accounted for by genetic factors.

Needless to say, the propensity for APD is driven by many factors, involves a multitude of neurological structures and systems, and results in a heterogeneous set of APD sub-types. The Somatic Marker and Social Cognition models both provide com-

prehensive paradigms in which to conceptualize this variability, accounting differently for deficits in cognitive and affective processes, which in turn drive behavior. These models do not fully explain the discrete systems involved, however, such as "each fractionable and functionally distinct system" of the frontal lobe, which "may be differentially engaged in APD" (Dinn & Harris, 2000, p. 186). Thus, the association between clinical pathology and APD profile must be further researched to better understand the complex interplay between these discrete structures and systems.

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