Dahl (2004) has described the adolescent brain as "a natural tinderbox," which helps to make adolescence a period of both vulnerabilities and opportunities. His description is in reference to a new body of research, stemming primarily from neuroscience literature, that has evidenced continuing development and plasticity in the prefrontal cortex (PFC) area of the brain in adolescence and early adulthood. Because the PFC is the primary locus of executive functioning in the brain, its continual development during adolescence has important implications for the understanding of many behavioral phenomena that occur at this time. One serious, costly, and often controversial type of behavior in adolescence is juvenile delinquency. Commonly conceptualized as a social (or antisocial) phenomena, I will argue that delinquency may have biological and developmental roots.

The aim of this paper is two-fold. First, I will review the recent research on development and plasticity of the PFC in adolescence. Second, I will present an argument for a relationship between immature or impaired development of the prefrontal cortex and juvenile delinquency. I will make this argument in two phases: first, that normative immaturity of the PFC creates normative risk for delinquency and second, that exposure to a set of additional risk factors greatly increases the probability that an adolescent will commit a delinquent act.

Development of the Prefrontal Cortex in Adolescence

Evidence for continuing development of the PFC into and beyond adolescence is very recent, being published only in the past 5-10 years. The newness of this research is due primarily to the recent advent of safe technology for conducting it. The development of functional magnetic resonance imaging (fMRI) technology in the early 1990's for the

first time permitted ethical, non-harmful imaging to be performed on the brain. Its predecessors, X-ray and CT scan, use potentially dangerous radiation to produce their images, and are thus only used when necessary on ill or injured patients. The non-radiation based fMRI allows scans to be performed without the undesirable risks and side effects associated with earlier scanning technology. Thus, the ability to ethically image the brains of non-ill adolescents is a relatively new one in the field of neuroimaging. fMRI measures level of activation of brain regions via the amount of blood flow to the region (Buonocore & Hecht, 1995). It is often used to pinpoint areas of the brain that are activated when a subject is asked to attend to a prompt meant to stimulate an emotion, or to perform a task that tests a certain cognitive ability. fMRI is often used in combination with MRI (magnetic resonance imaging), which captures structural aspects of the brain, including maturity and volume of neural structures.

Using MRI and fMRI technologies, researchers have begun to accumulate evidence that neural plasticity, until recently thought to exist primarily in childhood, is significantly present during adolescence as well. Neural plasticity represents the brain's openness to environmental influence and its method of adapting to and becoming specialized for its environment. Because human DNA are not complex enough to code for a neural response to every potential stimulus that may be encountered in development, plasticity is an evolutionary solution that allows each individual's brain to "wire" itself most efficiently for the environment it is expected to function in. The process begins when environmental stimuli activate neurological responses in the brain and lay the foundation for neuronal pathways of response. With repeated stimulation, the pathways become increasingly strong, meaning that those that are most often stimulated eventually

become the default responses (Huntenlocher, 2002). This phenomenon is understood to be the basis of most early learning and was traditionally believed to occur primarily during childhood. However, neuroimaging studies have revealed that the two primary neurological processes that operationalize neural plasticity, synaptic pruning and myelination, are still occurring rapidly in the PFC during adolescence and even into early adulthood.

Synaptic pruning is the process by which neurons are selectively eliminated based on the degree of stimulation they receive. In early adolescence, there is an overproduction of neurons, with the volume of gray matter, brain tissue composed of unmyelinated neurons, peaking between ages ten and eleven in normally developing children (Casey, Giedd & Thomas, 2000). Following this peak in gray matter volume, synaptic pruning takes place. Many neurons are selectively eliminated, and the remaining neurons and connections between them become stronger and more specialized. Elimination of neurons is commensurate with the amount of stimulation and activation they experience during development, meaning that the basic structure of the brain is affected by the sum of the adolescent's experiences with his or her environment. The process of synaptic pruning occurs at least through late adolescence. Sowell, Thompson, Holmes, Jernigan, & Toga (1999) studied adolescents ages 11-18 and found that the oldest participants, the 18-year-olds, still showed evidence of significant synaptic pruning. More research with older participants is needed to determine how far into development this process continues.

The second neurological process contributing to adolescent PFC development and plasticity is myelination, in which myelin, a type of fatty tissue, surrounds the axon of the

neuron, acting as insulation. Myelinated axons transmit signals up to 100 times faster than those that are unmyelinated (Thompson, 2000). Thus, the results of myelination are solidified neural pathways that are able to process responses in a faster, more accurate, and more efficient manner. Myelination occurs rapidly throughout adolescence and continues more gradually through the twenties (Giedd, 2004; Goldberg, 2001; Sowell, Petersen, Thompson, Welcome, Henkenius, & Toga, 2003). Unmyelinated neural pathways are able to fire and form connections, but their functioning is slower and less precise than those pathways that have been solidified via myelination. Until these processes are complete, the efficiency and accuracy of adolescents' PFC functioning are much lower than that of adults.

One of the primary sets of functions governed by the PFC are the executive functions. Executive functions are high-level cognitive processes that control the "when" or "whether" aspects of behavior as opposed to the nonexecutive "what" and "how" functions (Barkley, 2000). Deciding not to do something or not to do it right now requires accurate usage of executive functioning ability, one which is not yet fully developed in adolescence. Examples of executive functions are motivation, inhibition, logical decision making, problem solving, planning, emotional regulation, anticipation of consequences, and prioritization (Goldberg, 2001). Immaturity of or disturbance in executive functioning has been linked to a number of behavioral disorders in childhood, when the PFC has long been assumed to be immature. These include Attention-Deficit Hyperactivity Disorder (Barkley, 2003) and early-onset antisocial behavior (Moffit & Lynam, 1994).

An important feature of the PFC is that its functioning is very vulnerable to stress, especially in the still-developing adolescent brain (Arnsten & Shansky, 2004). The PFC is linked with the brain's limbic system, which processes emotions and emotional stimuli, thus its component structures are necessarily responsive to stress. There is a significant relationship between high social anxiety levels and low reasoning ability, indicating that teens are less able to use their already limited logical reasoning during times of stress (Rosso, Young, Femia, & Yurgelun-Todd, 2004). This decreased functioning is predicted by the adolescent's perceived level of stress (Spear, 2000), a key distinction, as most would agree that adolescence is nothing if not subjectively stressful. Immature executive functions are further compromised in situations of perceived stress or social anxiety.

Some neuroimaging research suggests that adolescents' decision making in emotional situations is often accomplished by the amygdala, the area associated with instinctive "gut" reactions, to compensate for immature executive functioning (Baird, Gruber, Fein, Maas, Steingard, & Renshaw, et al., 2003). The amygdala, like the PFC, is a part of the limbic system, but it completes its development earlier in the lifespan than the PFC. It seems to perform some functions that are later relegated to the PFC when the PFC has not yet fully developed. However, due to its orientation toward emotional stimuli and its tendency to react impulsively, the amygdala does not constitute an equivalent substitute for the self-regulatory ability of the PFC's fully developed executive functions.

A challenge in neurological research is linking brain development and functional processes to actual behavior. One method of doing so is to present a research subject

with a task or visual stimulus and then to measure the neurological processes that occur in response. Tamm, Menon, & Reiss (2002) found the level of PFC development to be associated with an adolescent's inhibitory ability on a computerized task. Specifically, they found that younger subjects showed more inefficient activation of the prefrontal cortex, showing activation of many areas in their attempt to inhibit their response. In contrast, older adolescents showed specific, focal activation of the prefrontal cortex and were able to inhibit their responses more quickly than their younger counterparts. Rosso et al. (2004) found that response inhibition and abstract reasoning showed continued development and significant age effects through age 18. These findings represent evidence that adolescents show immature ability to exercise executive functions in their behavior, even in relatively non-emotional laboratory settings.

A second method of linking executive functioning to behavior does not involve neuroimaging. Rather, researchers present the subject with a paper-and-pencil or computerized test of executive functioning and then study whether the measured executive functioning ability is related to hypothesized behaviors. In one such experiment, Ellis, Rothbart, and Posner (2004) found that level of executive attention, as measured by a computerized test, was negatively associated with adolescents' emotional reactivity and involvement in antisocial behavior. Henry and Moffit (1997) found lower performance on neuropsychological tests of executive function to predict more episodes and greater severity of adolescent antisocial behavior.

At this point it is important to stress that continued PFC development and immature executive functioning are normative aspects of adolescent development. Subjects in the cited studies are chosen because they are developing normally; they are

screened for many variables that could affect the results of the neuroimaging, particularly past or current mental illness, exposure to toxins, head injury, malnutrition, and serious physical illnesses. In addition, they are usually Caucasian and from middle- to upper-class background. Considering their characteristics, these subjects are normal, if not supernormal, in their development. Do the results of these studies present, then, a best case scenario of adolescent PFC development? If healthy, screened subjects show evidence of immature PFC and executive functioning, what about the individuals who have been exposed to risk factors for which the experimental populations have been screened?

In the following section, I will discuss the implications of the literature reviewed above for the understanding of juvenile delinquency. Juveniles who commit delinquency are at risk not only due to normative processes of continuing PFC and executive function development, but also due to their higher likelihood of exposure to many risk factors that may further inhibit the development of their PFC and their ability to exercise executive functions.

Juvenile Delinquency

In this section I will discuss executive functions and PFC development in relationship to adolescent-onset delinquency, which occurs when an adolescent without a history of antisocial behavior in his or her childhood commits a delinquent act (Moffit, 1993). According to both arrest (FBI, 2004) and self-report (Hawkins, Smith, & Hill, et al., 2003; Huizinga, Weiher, Espiritu, & Esbensen, 2003) data, delinquent behavior peaks in adolescence. Noted delinquency scholar Laurence Steinberg has concluded that adolescent delinquent behavior often involves a failure of inhibition, a lack of judgment,

or a spontaneous decision in an often emotional or high-stress situation (Steinberg, 2004). The failures he describes clearly involve executive functions, and the stress of the situation that eventually leads to delinquency serves to overwhelm the already limited ability of the adolescent brain to exercise the executive functions.

As discussed earlier, immature or problematic executive functioning has been named as a potential major factor in several childhood behavioral disorders. This hypothesis has been fairly palatable given the general acceptance of incomplete neurological development in childhood. If immaturity of executive functioning is now found to be probable into the adolescent years, is it not plausible that it is associated with behavioral disturbance during that period of development as well? In adolescence, behavioral disturbances receive a new label, delinquency, and they carry much more serious consequences. But why is there such a drastic increase in antisocial behavior during adolescence?

Raine (2002) hypothesizes that the psychosocial demands of adolescence are much higher in proportion to the ability of the executive functions to handle them than at any other point in the lifespan. Within the normative adolescent experience, there are demands to juggle peer group relations, academic and familial expectations, and emerging sexuality in an environment that is much less supervised and scaffolded than it had been earlier in development. Sometimes, the adolescent's ability to self-regulate, selectively attend, and/or inhibit action or reaction may not be commensurate with the demands of his or her environment, and a failure of executive function can result from this imbalance.

As adolescents, delinquent juveniles experience the normative continuing development and plasticity that has been shown to occur in the PFC during this period of development. However, delinquent juveniles are significantly more likely than non-delinquents to have experienced poverty, history of abuse, mental illness, malnutrition, exposure to toxins, and chronic stress (Kapp, 2000; United States Surgeon General, 2001). In the Rochester Youth Development Study, Smith and Thornberry (1995) found that, even after controlling for effects gender, race, SES, family structure, and mobility, delinquent youth were significantly more likely to have been abused than their non-delinquent youth counterparts.

Risk factors such as head injury, malnutrition, exposure to toxins, and history of abuse or chronic stress exposure all present serious threats to neurological development and function. Glaser (2000) reviewed the documented structural and functional effects of child abuse on the brain and highlighted their severity and the seeming universality of their impact on response and behavior. Weber and Reynolds (2004) make a similar argument, presenting evidence that trauma and abuse can result in "neural degeneration, neurochemical abnormalities, cerebral dysfunction, and neuroanatomical disconnection." Abuse has been shown to have both structural and functional impacts on the developing brain, which can permanently alter children's patterns of response and make them highly reactive, neurologically and behaviorally, to perceived stresses (Nemeroff, 2004; Nutt & Malizia, 2004). As the PFC is naturally responsive to stress, an individual who is neurologically predisposed to be even more reactive to stress is at great risk of acting improperly in an intense situation. Because the PFC continues its development so late into the lifespan, it has a prolonged period of vulnerability, so that environmental effects

through the adolescent years can impact its development. Finally, the number of opportunities for delinquency is higher in impoverished neighborhoods, where these risk factors, as well as a high crime rate and low parental supervision, are more likely to be present, representing a double dose of risk for children who live there (Morenoff, Sampson, & Raudenbusch, 2001).

At this point I would like to present a real life case example of an act of delinquency that, although a severe example, I believe illustrates the spontaneity, lack of judgment, and failure of self-inhibition believed by Steinberg and others to be key contributors to juvenile delinquency.

> *Chris (all names have been changed) was a quiet, shy sixteen-year-old high school junior who had no criminal record and who had recently moved with his father from inner-city Detroit to a middle-class suburb in order to leave the violence of the city and to receive a better education. He made friends at his new school and also began dating. One night, his girlfriend reported that her house had been broken into and a television had been stolen. She claimed she knew who had taken the television and asked Chris to go with her to ask the thief, a young man named Dave who they knew from school, to give it back. They drove in Chris's father's car to an empty parking lot where Dave was waiting. Feeling nervous, Chris took the baseball bat his father kept in the car (to ward off dogs in his job as a newspaper deliveryman) and walked out to meet Dave. The two spoke, and Dave denied stealing the television. Dave swung a fist at Chris, and Chris reacted by hitting Dave with the baseball bat. When Dave fell to the ground, Chris hit him several more times, and then, panicking, he left the scene. Dave survived but suffered severe injuries that resulted in permanent physical and mental disabilities. Chris was tried and convicted as an adult for Assault with Intent to Murder and was sentenced to 15-50 years in prison.*

While the act committed by this individual was harmful and wrong, the circumstances surrounding it lend themselves to the argument for a failure of executive functioning in acts of delinquency. His susceptibility to peer influence (allowing his girlfriend to persuade him to accompany her to an unknown location), failure to anticipate possible consequences (that by bringing the bat he may use it as a weapon, and that by using it as a weapon he may seriously hurt someone), inability to inhibit his actions (continuing to hit the other boy after knocking him to the ground), and his illogical reaction after the event (fleeing in panic rather than seeking help) to me clearly demonstrate immature executive functioning. Chris was adjudicated as an adult in the criminal court and sentenced to spend at least the next 15 years (essentially the length of his life thus far) in adult prison. Due to the nature and severity of his crime, he was deemed to be equivalent to an adult offender and was treated as one.

The developmental and neurological research presented here offer evidence that adolescents are not equivalent to adults, at least in terms of their neurological development and their ability to exercise important self-regulatory behaviors. In addition, the average delinquent juvenile is likely to be much more at risk for even lower executive functioning ability than this research indicates. This evidence has important implications for both the legal and social conceptualizations of juvenile delinquency and how it should be handled.

Two important legal concepts involved in the understanding of juvenile delinquency are the competence and the culpability of juveniles. Competence refers to the juvenile's ability to stand trial and involves the ability to participate meaningfully in his or her defense and to make strategic decisions throughout the court proceedings.

Meaningful participation and strategic decision making require the ability to anticipate and weigh potential consequences and to think logically about complex and highly emotional topics. Juveniles are traditionally considered able to perform these tasks unless they are extremely mentally ill or developmentally disabled. Culpability refers to the extent to which a juvenile is held responsible for his or her actions. Similar to competence, juveniles are traditionally considered to be fully culpable for the actions they engage in unless they are severely handicapped by a mental illness or developmental disability. However, word of immaturity of executive functions has begun to reach the legal community and has resulted in a call by some (Cauffman and Steinberg, 2000) to reexamine traditional conceptualizations of juveniles' competence and culpability in light of this new scientific evidence.

When a potential biological explanation or association is discovered for any negative human affliction, there are always major social implications. In the past delinquency was considered a social and behavioral phenomenon, and society placed much blame on the offenders themselves. The popular reaction to possible biological associations of delinquent behavior could go one of two ways. First, there is always the risk of a deterministic interpretation of any biological risk, whereby those who are at risk are assumed destined to embody that for which they are at risk. Because delinquent juveniles tend to overrepresent populations that are already somewhat marginalized in our society, including racial minorities and people of lower socioeconomic background, this is an especially dangerous possibility. It is the responsibility of researchers and activists who present this evidence to do so responsibly in order to avoid sparking such deterministic ideologies.

A second, more promising reaction would be to come to be understand delinquency as an unfortunate, but at least partially preventable, consequence of normal development. Knowledge regarding the developmental and neurological contributions to delinquent behavior has the potential to inform and direct prevention and treatment programs. Prevention efforts can be directed at decreasing children's exposure to risk factors that further problematize the development of executive functions and at teaching the conscience utilization of the self-regulatory skills they already possess. Treatment for juvenile offenders can incorporate education and training on how to use executive function and self-regulation more effectively.

It is clear from the evidence reviewed here that the adolescent brain, particularly the PFC, is still very much in a state of development. Until the PFC finalizes its development, adolescents are unable to fully exercise executive functions. Given situational opportunity, this inability sometimes manifests itself as an act of delinquency. Adolescence is a time of normative risk for delinquency, and some adolescents are at even greater risk due to environmental factors to which they have been exposed. Collectively, this evidence necessitates a reconceptualization of both the legal and social understandings of delinquency and a renewed commitment to evidence-based prevention and treatment programs.

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