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Comparing Predatory Versus Affective Violence and Examining Early Life Stress as a Risk Factor

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In *The Boy Who Was Raised as a Dog* (Perry & Szalavitz, 2006), child psychiatrist Bruce Perry describes his encounter with a teenage boy referred to as Leon who, at age 16, killed two young teenage girls. It was three or four in the afternoon when Leon, who had been drinking, encountered the girls in the elevator of his apartment building and crudely propositioned them. After they rejected his proposal, he followed them into an apartment and attacked them with a kitchen knife, stabbing them to death and then proceeding to rape, kick, and stomp their bodies. Cherise was 12 and Lucy was 13 years old. Perry, hired to investigate the case and help the court decide between life without parole or capital punishment, visited Leon in a maximum-security prison and had discussions with Leon's parents and older brother. Perry would come to discover that Leon came from a relatively normal family, his father and brother both holding steady jobs. His mother, however, while kindhearted and polite, was mentally impaired such that she was completely ignorant of the crucial needs of an infant. Lacking the help that her extended family had provided in raising Leon's older brother, she found no fault in leaving Leon home alone in a dark apartment for most of the day from the time he was four weeks old. The extreme neglect and deprivation Leon's immature brain suffered had severe consequences for his responsiveness to punishment as a child and for his cognitive and emotional capabilities later in life (Perry & Szalavitz, 2006).

Leon's situation is an extreme example, not representative of most crimes, but the perpetration of violent crimes is undeniably a major social issue with substantial economic costs to society. In 2007, an estimated 23 million violent crimes were committed in the United States, which cost the nation $15 billion in economic losses and $179 billion in government spending on,

for example, police protection, judicial and legal activities, and corrections (McCollister, French, & Fang, 2012). Understanding the biological bases of criminal behavior and the factors that increase an individual's chance of acting violently can inform crime-prevention programs and thereby help to reduce the social and financial burdens associated with crime.

Although there is a high degree of variability between every incidence, violence can be classified as either predatory or affective in nature (Siever, 2008; Vallabhajosula, 2014). These two forms have distinct motivations, with predatory violence being premeditated and "cold blooded" and affective violence being impulsive and "hot blooded" (Declercq & Audenaert, 2011). The separation between predatory and affective offenders is supported by differences in intelligence and cognitive abilities as well as in various facets of their neurobiology (Hanlon, Brook, Stratton, Jensen, & Rubin, 2013; Raine et al., 1998). The nature of these neurobiological abnormalities suggests that alterations to the neurodevelopmental pathway play a role in their manifestation.

One of the main factors that have been identified to put an individual at risk for violent behavior is early life stress, with early life referring to the stages of development prior to adulthood (e.g., infancy, childhood, and adolescence) (Perry, n.d.). While most individuals who experience extreme stress during development do not end up committing crimes in adulthood, it has been found that most criminals endured adverse experiences in childhood, whether from maltreatment (e.g., abuse or neglect) or due to circumstance (e.g., low socioeconomic status). A host of neurophysiological changes occur in the body in response to a stressor and many of these changes are mediated by the hypothalamic-pituitary-adrenal (HPA) axis (Frodl & O'Keane, 2013). This pathway involves a series of chemical messengers that travel between the hypothalamus and pituitary in the brain and the adrenal glands on top of the kidneys. The

messengers work to prepare the body for adverse conditions thereby increasing one's chance of survival. By having both excitatory and inhibitory effects on various parts of the axis, these chemical signals regulate the activity of the HPA axis via a negative feedback loop. This feedback mechanism, however, can be impaired by excessive stress thereby disrupting the self-regulation of the axis and leading to abnormal stress responses (Frodl & O'Keane, 2013).

Despite an abundance of research on childhood trauma and studies of the biological correlates of antisocial behavior (e.g., aggression, violence, and psychopathy), the neurodevelopmental mechanisms by which psychological trauma can translate into the neurophysiological changes that increase one's risk of violent behavior require further elucidation. A host of significant differences exist between predatory and affective offenders (Hanlon et al., 2013) but previous research has mainly employed mixed groups of participants (Yang & Raine, 2009), which has limited the findings of these studies.

This paper argues for the consistent employment of the bimodal classification of violence into predatory and affective forms due to the extent to which they differ neurobiologically (e.g., neurotransmitter functionality and regional brain activity) and the potential applications of these differences (e.g., recidivism rates). To support this claim, this paper will first discuss predatory and affective forms of violence, addressing psychological (e.g., motivation), cognitive (e.g., task performance), and neurobiological (e.g., brain activity) differences. Then, the potential for stress caused by early life adversity to contribute to these differences will be evaluated. Although the body's stress response increases the chance of surviving through difficult conditions, excessive stress has detrimental effects on the body, to which the developing brain is particularly vulnerable. To demonstrate this, the normal neurodevelopmental pathway is reviewed and a number of alterations resulting from stress are discussed. It is then argued that more research

should be conducted on the relationship between the reward system and violence and that this research should separately study predatory and affective offenders.

Two Types of Violence

Violence, a highly heterogeneous phenomenon, must be differentiated from aggression because while violence is aggression, an aggressive person may not be violent (Perry, n.d.). Aggression can be defined as behavior that is forceful, hostile, injurious, or destructive (Vallabhajosula, 2014) and is characterized by verbal or physical attack (Perry, n.d.). Aggressive behavior can be destructive and violent, but it can also be appropriate and defensive (Perry, n.d.). On the other hand, violence is an unlawful type of aggression involving the unjustified infliction of injury (Vallabhajosula, 2014). Aggression, and therefore violence, can be classified along a continuum between predatory and affective (Siever, 2008; Vallabhajosula, 2014) and many perpetrators can be classified as committing predominantly predatory or affective violence (Vallabhajosula, 2014). This bimodal categorization has been validated in studies of both humans and non-human animals (Hanlon et al., 2013).

Predatory violence, called such in reference to its evolutionary basis in hunting (Meloy, 2012), is also described as premeditated, instrumental, proactive, and "cold blooded" (Declercq & Audenaert, 2011; Meloy, 2006; Vallabhajosula, 2014). It is purposeful and planned with clear goals in mind (Declercq & Audenaert, 2011; Fabian, 2010; Vallabhajosula, 2014) and involves relatively little emotion or physiological arousal (Hanlon et al., 2013; Miller, 2002; Scarpa & Raine, 2000). Affective violence, which is also called impulsive or reactive violence, involves hostility and retaliation as it often occurs without forethought in response to a perceived threat, provocation, or insult. Unlike predatory violence, affective violence is highly emotional with enhanced physiological arousal ("hot blooded") and is relatively uncontrolled (Declercq &

Audenaert, 2011; Hanlon et al., 2013; Miller, 2002; Scarpa & Raine, 2000). Although affective violence is much more common than the predatory subtype, this is not true for psychopathic individuals who are much more likely to commit predatory violence compared to other criminals (Meloy, 2012; Miller, 2002).

Evidence for a Distinction

The notion that impulsive and premeditated violence represent distinct psychological and neurological phenotypes has been supported in the literature (Fabian, 2010; Hanlon et al., 2013; Kockler, Stanford, Nelson, Meloy, & Sanford, 2006; Vallabhajosula, 2014). Hanlon et al. (2013) describe a host of other characteristics that distinguish perpetrators of predatory from those of affective violence. Predatory offenders tend to commit more severe physical violence and show more antisocial-narcissistic-aggressive personality traits, which is likely related to the increased rate of psychopathic traits. Although affective murderers are more likely to violate parole overall, predatory murderers were more likely to violate parole by committing a violent offence. Affective offenders, the more common type, tend to have a wider range of psychopathologies in addition to passive-aggressive and borderline personality traits. They usually have higher chronic anger as well as fearful attachment and are more likely to have had a close connection to their victim (Hanlon et al., 2013).

The relationship between these types of violence and neurotransmitter activity provides additional support for distinct neurotypologies. According to Meloy (2006), while cholinergic stimulation facilitates predatory aggression, dopaminergic neurotransmission facilitates affective aggression. Stimulating dopamine release in the brain has been shown to intensify affective aggression (McEllistrem, 2004). Additionally, administering a dopamine agonist, which activates dopamine receptors, decreases the threshold for attack while a dopamine antagonist, which

prevents dopamine from acting, inhibits affective attack. Enhancing noradrenergic neurotransmission increases the occurrence of affective aggression but decreases incidents of predatory aggression. The inhibitory neurotransmitter GABA has been implicated in affective aggression through the administration of benzodiazepines, which increase the binding of GABA to its receptors thereby potentiating its effects. It would be expected that increased GABA neurotransmission would inhibit affective aggression since GABA receptors are especially dense in the amygdala and in fact giving benzodiazepines to rats and other animals does decreases their expression of affective aggression.

Unlike other neurotransmitter systems, research (McEllistrem, 2004) suggests that changes to serotonergic neurotransmission have similar effects on the rates of predatory and affective aggression. When serotonergic neurotransmission is decreased via a reduction in dietary tryptophan (the precursor for serotonin) or the inhibition of the enzyme that synthesizes serotonin, both predatory and affective aggression occur more often. Additionally, lesions to neurons that release serotonin, located in the raphe nuclei of the brain stem, result in affective aggression and stimulation of these neurons inhibits predatory aggression.

Cognitive tests have revealed that affective offenders have significantly poorer performance on tests of intelligence, memory, attention, and executive functions compared to controls while predatory offenders have normal functioning in these areas. While affective offenders show impaired performance in impulse-control tasks regardless of motivational factors (e.g., reward opportunity while gambling), predatory offenders have normal, intact impulse control in the absence of motivational factors but show impaired impulse control when motivational factors are present (Levi, Nussbaum, & Rich, 2010).

A neuroimaging study by Raine et al. (1998) provided information regarding the neurological basis for these cognitive differences. They used positron emission tomography (PET) to compare the brain activity between predatory murderers, affective murderers, and control individuals. In order to distinguish affective and predatory murderers, each participant was rated on a scale from 1 (strongly predatory) to 4 (strongly affective) by two raters blind to the assessments of the other. Each rater was provided the following information: assessment reports from psychologists and psychiatrists, criminal transcript history, telephone interviews with attorneys, preliminary hearing transcripts, medical records, as well as national and local newspaper reports. If raters gave the same individual different scores, inconsistencies were resolved and consensus was reached on a single score, which defined the participant as either affective or predatory. While 41 murderers were rated, only 15 murderers were clearly predatory and nine were affective. Both predatory and affective murderers had elevated subcortical activity in the amygdala, midbrain, hippocampus, and thalamus compared to controls. Prefrontal activity, however, differed between the types of murderers such that affective murders had lower prefrontal activity than that of the controls while predatory murderers had similar prefrontal activity than controls (Raine et al., 1998).

These neurological findings support behavioral results since the prefrontal cortex governs executive functions such as planning, goal-directed behavior, as well as impulse control while subcortical systems are involved in generating aggressive and emotional responses (Andersen, 2003). While both types of murderers have increased activation of subcortical areas which produce aggressive impulses, predatory murders have the prefrontal capacity to regulate and control these impulses while affective murderers are less able to do so and thus act reactively (Fabian, 2010; Miller, 2002; Raine et al., 1998).

Reciprocal communication between the prefrontal cortex and the amygdala, a subcortical structure involved in fear and emotions, is crucial for assigning emotional significance to stimuli (Hoptman, 2003) and for suppressing negative emotion (Bufkin & Luttrell, 2005). Differences in prefrontal functioning between predatory and affective murderers suggest that affective murderers have a reduced capacity to regulate amygdala activity, and increased amygdala activation likely impairs emotion recognition (Hoptman, 2003). This may lead to incorrect fear responses, which in turn, may lead to the misinterpretation of social signals. Abnormal processing of fear and emotions, combined with decreased regulation, enhances an individual's risk of acting aggressively or violently in an affective, emotionally charged manner (Hoptman, 2003). Since the prefrontal cortex is important in the regulation of arousal (Hoptman, 2003) as well as the interpretation of environmental stimuli, the misinterpretation of situations as being potentially dangerous or threatening may contribute to the expression of affective violence (Bufkin & Luttrell, 2005).

Since the capacity of an individual to control impulses relies on the relative ratio of brain stem and subcortical activation and the moderating activity of the cortex, any factor that alters the ratio will affect an individual's capacity to act violently (Perry, n.d.). One factor that has been established to increase one's risk of violence is early life adversity, or, in other words, stress during development, which could come from abuse, neglect, maltreatment, trauma, malnutrition, or a host of other sources. The body's initial response to stressors such as these is adaptive in that it helps the individual cope to the change. Over time, however, repeated or prolonged stress begins to have negative effects on the body, effects that can alter developmental pathways.

Stress as an Adaptive Mechanism

The conditions in which we live are constantly fluctuating and thus, to survive through times of difficulty, we ourselves must be capable of change. To do so, the body employs the stress response, an adaptive mechanism allowing the body's functionality to shift in order to cope with a change in the environment but then ultimately return to a healthy state of homeostasis (Frodl & O'Keane, 2013; Tarullo & Gunnar, 2006). The major system involved in responding to a stressor is called the hypothalamic-pituitary-adrenal (HPA) axis (Frodl & O'Keane, 2013). While there are additional mechanisms involved in immediate responses to stress, discussion of the stress response in this paper refers to the activity of the HPA axis. When functioning properly, the job of this pathway is to mediate a range of bodily adjustments to activate energy stores and prepare the body for dealing with the environmental change. Normally, in response to a stressor (any environmental cue significant enough to warrant a stress reaction), the hypothalamus produces and releases more corticotropin releasing hormone (CRH) and arginine vasopressin (AVP) (Gunnar, 2007) into the pituitary gland. This prompts the pituitary to produce and release adrenocorticotropin hormone (ACTH) into the blood stream. Receptors for ACTH are located in the adrenal gland so upon ACTH binding, the adrenal gland produces and releases cortisol, a steroid hormone, into the blood.

Although cortisol will travel throughout the body via the blood stream, its principle target is the brain due to the abundance of cortisol receptors there (Tarullo & Gunnar, 2006). Cortisol levels can be easily measured via saliva samples and is thus commonly used to quantify the activation of the stress response. Its action on the brain helps to mobilize the body's energetic resources for use in times of stress but is also has long term effects and roles in the experience of fear, sensitivity to punishment (Glenn & Raine, 2008), and mediating a negative feedback loop. When cortisol acts on receptors in the hypothalamus and hippocampus, secretion of ACTH and

CRH is inhibited and, in this way, the HPA axis works to regulate its own activity and return to baseline levels (Frodl & O'Keane, 2013).

There are a few important notes to make regarding the stress response and the HPA axis. First, activation of the HPA axis is not an all-or-nothing response but rather its activity shifts continuously as an individual's circumstances change. Second, the severity of an individual's response to a certain stressor depends on many factors including individual differences and characteristics of the stressor. The same stimulus may elicit varying degrees of stress for different people and may be more or less stress-inducing at different times in an individual's life. Additionally, the nature, frequency, and intensity of the stressor will influence the degree to which the body's stress response is activated (Perry, 2001). Considering the function of the response, more severe stress reactions would be predicted for circumstances in which the body perceives a greater threat to survival. Severe changes in neurophysiology resulting from exposure to stressors have the potential to significantly alter the brain.

Negative Effects of Stress

Although the neurophysiological alterations mediated by the HPA axis aid with survival through adverse circumstances, the eventual return to homeostasis can be prevented by excessively frequent or severe stress responses (Frodl & O'Keane, 2013). Excessive levels of cortisol in the blood affect both the structure and functionality of the hippocampus, a subcortical structure crucial for learning and memory that possesses an abundance of receptors for cortisol (Sapolsky, 1996). Effects include a reduced ability for neurons to alter their connections, called synaptic plasticity, and impaired generation of new neurons, called neurogenesis. Dendritic atrophy can also occur and, although reversible if cortisol overexposure is short, can be permanent if exposure lasts long enough (neuronal death may even occur) (Sapolsky, 1996).

Since the hippocampus is important for the feedback loop of the HPA axis, hippocampal dysfunction results in an impaired inhibition of CRH secretion in response to elevated cortisol levels. The downstream consequence of uninhibited CRH secretion is increased cortisol secretion, which by worsening hippocampal function exacerbates the problem and prevents cortisol levels from normalizing. In other words, the HPA axis can become dysregulated by copious amounts of stress causing excessive cortisol secretion (Frodl & O'Keane, 2013).

Overactivation of the stress response leading to dysregulation, or the inability of the axis to regulate itself, can be precipitated by any number and combination of genetic and environmental factors. An individual may be, at birth, biologically predisposed to stress over-reactivity by the nature of their genetic composition. For instance, the expression level of a certain receptor may be abnormal or the subtype they express may have an abnormal degree of activity. On the other hand, an individual's genetic endowments may also predispose them to be relatively insensitive to stress. Their biological makeup may, for instance, lead them to not find a stimulus stressing when another individual would do so. Then of course the significance of these genetic abnormalities depends on the context, on the environment of the individual, which influences genetic expression.

The circumstances surrounding an individual may subject them to an increased number of, severity of, or duration of physiological stress reactions, which would increase the chance of dysregulation. Stress can be externally mediated, such as that caused by physical abuse, or internally mediated, such as stress brought on by the anticipation of being abused. It is worthwhile to emphasize the role of cognition, the psychological experience of stress, in the body's response to stress. An individual's response to a given stimuli depends on their perception of the stressor such that merely thinking about an event, whether in the past or future,

can elicit biological responses. For children, stressors can be related to the home, school, peers, and/or the community with examples being physical and emotional neglect or abuse, malnutrition, bullying, and familial conflict (Rosen, n.d.). Excessive stress may come from one or a combination of these sources and could be brought on by a single traumatic event or by repeated exposure to stressors. There are a number of reasons why children are more susceptible than adults to the adverse effects of stress.

Childhood Vulnerability to Stress

Infants, children, and adolescents are particularly vulnerable to the adverse effects of stress since the brain is still rapidly maturing during these times and developmental trajectories have great potential to be altered. As opposed to the mature adult brain that accommodates to changes in the environment, the very plastic immature brain incorporates external information permanently into its structure and function (Andersen, 2003). While the brain is continually influenced by information from the surrounding environment, the timing of stimuli affects the degree to which development is altered. During what are called critical periods of development, appropriate stimulation from the environment (e.g., motor, sensory, cognitive, and social experiences) is required for normal development: if a developmental milestone fails to be achieved during this time, then it will never be accomplished (Weber & Reynolds, 2004). An event occurring during a critical period has a significantly greater impact on development than the same event occurring later in life (Andersen, 2003). There are also sensitive periods in which information from unessential stimuli is incorporated into neuronal patters and may have a negative impact on development (Andersen, 2003). States become traits as the conditions in which the nervous system develops help determine how neural systems are organized and therefore the anatomy and functioning of the adult brain (Perry, Pollard, Blaicley, Baker, &

Vigilante, 1995). Alterations to the nervous system early in development affect how the brain's development later on and the adult brain will respond to stimuli. In order to evaluate the ways in which abnormalities during the brain's maturation can result in modifications that carry through to adulthood, it is necessary to review the typical neurodevelopmental timeline.

Normal Brain Development

As described by Andersen (2003), the process of brain development can be divided into seven stages: cell birth, cell migration, axonal/dendritic outgrowth, programmed cell death, synaptic production, myelination, and synaptic elimination/pruning. The beginnings of these phases are offset such the process of cell birth starts first, in the early embryo, and synaptic elimination/pruning starts after birth.

Once neuronal stem cells are born and differentiated into different types of brain cells, their patterning and placement is determined by glial cells, which are non-neuronal brain cells, and by molecular signals called neurotrophic factors. Some examples of these include brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), and glia-derived GF (GDNF). Even before neurons finish migrating, their axons extend between and connect many regions of the brain. Neurons reach their final destination around the 16^{th} fetal week and proceed to make connections, or synapses, with each other via dendritic outgrowth, a process also highly mediated by growth factors. Across the lifespan, growth factors will continually influence synaptic plasticity, or the ability of neuronal connections to change, which contributes to such processes as learning and memory. Immediately prior to birth, there is a time period characterized by extensive programmed cell death. About half of all neurons are eliminated, presumably as means of enhancing the efficiency of neural communication. This phase is followed by another round of excessive connectivity but the elimination phase does not occur until after birth.

The time surrounding birth, both before and after, is especially important for the development of the neurotransmitter systems involving monoamines (e.g., dopamine). This has been demonstrated in rat studies in which markers for these systems (e.g., quantity and firing rate of neurons, neurotransmitter levels, and quantity of receptors) are detectable in embryo and continue to become more numerous after birth. The second period of synaptic pruning, or the elimination of neuronal connections, begins about a year after birth. Between the ages of 7 and 14 years the density of synapses in the frontal cortex decreases by approximately 40%, a change that is mirrored in declining numbers of neural receptors, including those for dopamine and glutamate.

The timing of synaptic production and elimination is not uniform across all brain areas. These processes occur in subcortical regions prior to cortical areas and, within the cortex, they occur in posterior areas before anterior areas. The density of connections, for instance, peaks around 6 months of age for the primary visual cortex but around 2 years of age for the prefrontal cortex. As synaptic rearrangement occurs, alterations in receptor density for a variety of neurotransmitter systems occur in parallel, along with functional developments. For example, motor development, which is mediated by the striatum (a subcortical structure), precedes cognitive development, which is governed by the cortex. Less is known about the development of specific cognitive processes (e.g., abstract reasoning and emotional regulation) due to the difficulty of applying rodent models. Longitudinal fMRI studies, however, seem to suggest that the maturational timeline of cognition parallels that of the synaptic elimination phase.

The timing and localization of changes in neurotransmitter levels is crucial for normal neurodevelopment since these molecules serve as trophic factors, guiding the migration of neurons and the movement of dendrites. If neurotransmitter activity is inappropriate during a

critical period, perhaps as a result of stress, the developmental process is disrupted and abnormal maturation will occur. Serotonin, for example, mediates cell migration and synapse formation early in development while dopamine plays a role in both the promotion and inhibition of neuronal outgrowth. These processes are thought to be temporally mediated by the transient expression of some receptors, which are only expressed during select periods of early postnatal development and are absent in adulthood (Andresen, 2003).

Effects of Childhood Stress

As discussed, stress during development shifts from an adaptive compensatory response to being maladaptive and potentially detrimental if the associated neurobiological changes are of sufficient frequency, intensity, or duration to prevent return to homeostasis (Weber & Reynolds, 2004). Excessive developmental stress has the ability to impair, often permanently, the functioning of key neuroregulatory systems (Anda et al., 2005), such as the HPA axis, since these systems are immature at birth and are molded by experience (Tarullo & Gunnar, 2006).

Much research has focused on the adverse effects of stress on the hippocampus as these effects can be severe, but other areas, including the amygdala and frontal cortex, also contain high levels of cortisol receptors (Gunnar, 1998). While there is less known about how these areas are affected by excessive stress, they are likely influenced in a similar manner to the hippocampus (Gunnar, 1998). Any extreme stress responses resulting in modifications to the organization of the nervous system will result in effects that carry through to adulthood. Excessive stress can, for instance, lead to neural degeneration, neurochemical abnormalities, and cerebral dysfunction (Weber & Reynolds, 2004). By compromising the structural and functional integrity of the brain, these changes can cause profound and long-lasting neurobehavioral consequences (Anda et al., 2005). For instance, as they mature, cortical areas begin to modulate

the activity of lower, more reactive areas including the brain stem and subcortical structures (Perry, n.d.). If the maturation of the cortex is inappropriate, the activity of the lower regions will be improperly regulated thereby predisposing the individual to violence (Perry, n.d.).

As previously mentioned, excessive physiological stress responses have an impact on the development of the stress response itself such that adaptive responses early on in life can lead to maladaptive responses down the line. Adults who were maltreated as children, for instance, often exhibit lower resting levels of cortisol and higher ACTH responses to a stressor, indicative of a lower threshold for perceiving stress and an exaggerated response to a perceived stressor (Tarullo & Gunnar, 2006).

The brain's responsiveness to stress changes also changes within the developmental timeline. For example, a study in which a pharmacological stressor was administered to both immature and mature rats demonstrated the dopamine system to be particularly sensitive to early life stress. The increase in neuronal activation of the nucleus accumbens (the brain's so-called pleasure center, which receives dopaminergic input) caused by pre-pubertal stress was much greater than the stressor's effect during adulthood, when most of the activation occurred in the prefrontal cortex (Andersen, 2003). This experiment relating stress and dopamine is one of a relatively few number of studies examining the ways in which early life stress can influence the reward system and its development. Research in this area is most commonly done in rats since they can be subjected to various stressors and invasive procedures can be used to quantify dopamine levels in the brain (Abercrombie, Keefe, DiFrischia, & Zigmond, 1989; Gambarana et al., 1999; Rougé-Pont, Deroche, Le Moal, & Piazza, 1998). This does, however, limit the extent to which the results of the studies apply to humans and human behavior. Buckholtz et al. (2010)

found that individuals with psychopathic traits had a hypersensitive reward pathway, which lends support to the idea of reward system abnormalities contributing to antisocial behavior.

Future research should examine how the effects of early life stress on the reward system translate into which stimuli is considered rewarding and which behaviors are reinforced. This research should investigate differences between criminals and non-criminals as well as between predatory and affective criminals since a prior study identified the behavior of predatory offenders to be dependent on motivational cues (Levi et al., 2010). Learning more about variation in responsiveness to rewards and also to punishments can help to inform the development of interventions and therapies and has practical applications such as predicting recidivism rates (Hanlon et al., 2013). Most of the current neuroscientific research fails to differentiate between types of offenders, which by conflating findings relevant to one type and not the other, inhibits our understanding of violence as a heterogeneous phenomenon. Therefore, future work should take this classification system into account and compare results between predatory and affective violent offenders.

This paper reviewed the literature on types of violence, stress, and neurodevelopment to argue for the furthered use of the predatory vs. affective classification in research of aggression and violence. Predatory offenses are premeditated, involve clear goals, and are relatively unemotional while affective violence is usually provoked and highly emotional. Rates of parole violations differ as well as the influence of various neurotransmitter systems and studies have shown altered cognition and brain activity patterns in each type of violence. The differing ratios of cortical to subcortical activity suggest variation in developmental trajectories, possibly due to differential effects of early life stress. The maturing brain is extremely plastic, affected by the interaction between experience and genetic predispositions. The timing of, as well as quantitative

and qualitative differences in environmental stimuli, has the potential to significantly alter the maturation of the brain and consequently adult behavior. As we learn more about how violent criminals deviate from non-criminals we will better understand why some people commit violence and others do not. As we elucidate the differences between types of offenders we will have a fuller comprehension of the factors that lead to the perpetration of one type of violence compared to the other. Continued investigations into the neurobiological impact of developmental experiences, whether they be stress inducing or nurturing (Perry, n.d.), will provide knowledge relevant to the development and implementation of novel treatments aimed at redirecting the altered developmental trajectory to a more normal course rather than merely addressing the symptoms (Andresen, 2003).

Application of the Literature: A Proposal for Future Research

Studies on both rodents and adult humans have demonstrated the potential for dietary supplementation of omega-3 fatty acids to reduce the expression of aggressive behaviors (DeMar et al., 2006) with the research on humans being done both in real world and laboratory conditions (Gesch, Hammond, Hampson, Eves, & Crowder, 2002). Omega-3 fatty acids, which include eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are essential polyunsaturated fatty acids that humans must obtain from their diets, as they cannot synthesize omega-3s themselves. DHA has particular neurological relevancy as it is mostly localized to synaptic membranes and helps to mediate activity of receptors and associated signal transduction (Freeman et al., 2006). While dietary supplementation of omega-3s has beneficial consequences for a wide range of psychiatric disorders, the potential mechanisms of particular relevance to reducing aggression include increased serotonergic neurotransmission and regulation of CRH (Hibbeln, Ferguson, & Blasbalg, 2006). Animal studies have demonstrated an inverse

relationship between dietary omega-3 levels and concentrations of serotonin and 5-HIAA in certain regions of the brain (Hibbeln et al., 2006). Omega-3s have also been shown to increase dendritic branching, increase synapse formation, improve cerebral blood flow, and regulate gene expression (Freeman et al., 2006). With such global effects, the effects of omega-3s are likely to affect the expression of both predatory and affective aggression.

The therapeutic potential for omega-3s is extensive as evidenced by numerous experiments in both rodents and humans that have demonstrated a negative correlation between level of omega-3 fatty acids and aggressive behavior (DeMar et al., 2006; Fedorova & Salem, 2006; Hibbeln et al., 2006; Young & Conquer, 2005). A university study found that the aggression of students with clinically diagnosed aggression given omega-3 supplements did not change during exam period while the aggression of the control group showed a significant increase (Kidd, 2007). A prison study in which inmates were provided with omega-3 supplements found that, after two weeks, prisoners committed significantly fewer offenses while there was no change for those who received placebos (Gesch et al., 2002). Omega-3 supplementation has also shown to be efficacious in preventing stress-induced aggression and hostility and thus demonstrates promise as a therapeutic tool for reducing antisocial behavior (Vancassel et al., 2008). More research on these mechanisms could provide insight into the neurobiological foundations of aggressive and violent behavior and could further demonstrate the conditions in which omega-3 supplementation is an effective behavioral modifier.

To further examine the developmental interaction between omega-3s and stress as well as to assess the potential for omega-3s to reduce stress-induced aggression, a study could be performed on young rats separated from their mothers. In animals, early life adverse experience can be modeled through maternal separation (MS), which has been shown to elevate adulthood

aggression as measured by their behaviors in response to the presence of an intruder, called the resident-intruder paradigm (Veenema et al., 2006). Additionally, aggression is influenced by observing others behaving aggressively. Johnson, DeSisto, and Koenig (1972) showed that rats who had previously failed to attack frogs were more likely to become killers after observing another rat attacking a frog daily for one to two weeks compared to control rats.

The proposed study, adapted from Berg (2014), will use the introduction of an intruder to provoke aggression and thus will not compare impulsive versus premeditated aggression but will allow for developmental stress to be manipulated and for the execution of invasive molecular techniques. The study will also aim to elucidate how behavioral reinforcement via pleasure and reward contribute to the expression of aggression. Two types of early life experiences known to promote aggression in adulthood will be implemented in separate conditions as well as combined to test the strength of omega-3s' effects.

Experiment 1 will examine how lifelong omega-3 exposure, maternal separation, and witnessing aggression interact to influence levels of adult aggression. To test the potential for omega-3s to reduce adult aggression, rats will be exposed to omega-3s throughout their lifespan, both via their mother and through their own solid diet. They will experience maternal separation and/or witness aggression between other rats. With two diet conditions (enriched and control) and four stress conditions (maternal separation (MS), witness aggression, both, and control), a total of eight groups will be created.

Five pregnant females will be fed the omega-3 enriched diet (43% energy from fat) and five will receive the control diet (16% energy from fat). The enriched diet will be supplemented with fiber to ensure that the diets are isocaloric. After weaning, the pups will be fed the same diet as their mother. To obtain an average of two males, four offspring of undeterminable sex per

mother will be separated from their mother for three hours each day for 14 days starting the day after birth. Pup separation will entail handling every pup of the litter but the removal of only the focal pups, which will be transferred with some of the bedding to a small box with a heating pad. Pups will be weaned on Day 21 and housed in groups of 3-4 of the same gender and diet with the MS rats housed together. Only the cages of male rats (around 50 rats per diet condition) will be involved in the experiment.

For each diet condition, half of the cages with maternally separated (MS) rats (n = 10) and one third of the cages with non-MS rats (n = 10) will observe aggression between two other rats. The living space of these cages will be comparable to that of all the other cages but will have an additional section, separated by a wire screen, in which an adult rat that was already established to be aggressive will attack a smaller rat. The resident rats will observe one attack every day for one week beginning the day after moving into the cages (Johnson et al., 1972). For each diet condition, another third of the cages housing non-MS rats (n = 10) will undergo tests of aggression while the remaining third of the non-MS cages (n = 10) will serve as undisturbed controls.

Differences in care between mothers on different diets could lead to differences in offspring behavior independent from the effects of omega-3s. To check if and how omega-3 supplementation affects maternal care, mothers of each diet condition will be observed five times daily for the first week after giving birth.

Levels of 5-HIAA (metabolite of 5-HT) in the cerebrospinal fluid and blood cortisol will be quantified over the course of the experiment. To avoid risk of injury or trauma, collection of samples will begin the day after weaning and moving into the adult cages. Samples will be collected biweekly until postnatal Day 39 when the first RI test will be conducted. On the days

with RI tests, samples will be collected one hour before and 30 minutes before the beginning of the test and then 30 minutes after and one hour after the end of the test.

The resident-intruder (RI) test will be used to assess levels of aggression on postnatal Day 39 and 50. For the test, the focal rat will be housed in an observation cage with a female rat for ten days to stimulate territorial behavior. The female will then be removed from the cage 30 minutes prior to the test and is returned afterwards. An unfamiliar, lighter in weight (by 10%) male is placed in the cage for 10 minutes while the rats' behaviors are videotaped and scored in real-time. Various aggressive and non-aggressive behaviors will be recorded and calculated as a percentage of time. Attack latency and the number of attacks will also be measured (Beiderbeck et al., 2012).

Experiment 2 will investigate how the developmental timing of omega-3 exposure interacts with maternal separation and witnessing aggression to affect adult aggression. Given that omega-3s affect adult aggression (and that maternal omega-3 intake does not significantly alter care), how does the size of this effect change as omega-3 exposure occurs later in life? The timing of omega-3 exposure will emphasize weaning since brain changes due to omega-3 deficiency were shown to be potentially reversible by administering an adequate diet prior to, but not after, weaning (Vancassel et al., 2008). Experiment 2 will also involve an analysis of omega-3s' effect on the reward system by quantifying the mRNA expression of immediate early genes in the nucleus accumbens since there is some evidence to support the role of reward in the display of aggressive behaviors (Beiderbeck et al., 2012). Offspring will be initially exposed to increased omega-3s via their mother (either *in utero* or post-natal) or via their solid diet (either immediately after weaning or weeks after weaning). With five diet conditions and four stress conditions, a total of 20 groups will be created.

Fifty pregnant females will be randomly assigned to one of the five diet conditions. Ten mothers and their future offspring will consume the omega-3 enriched diet for the entirety of their lives. Ten mothers will begin consuming the enriched diet upon delivery of their offspring, who will also consume the enriched diet. Ten mothers will eat the control diet but their offspring will consume the enriched diet post-weaning. Ten pregnant females will be fed the control diet but their offspring will eat the enriched diet beginning three weeks after weaning. Ten mothers and their offspring will consume the control diet throughout their lifespan.

The procedures for maternal separation and witnessing aggression will mimic that of Experiment 1 and the controls will be identical. The RI test will be performed on rats of each condition as described in Experiment 1 except that only the first test on Day 39 will be conducted. Activation of the nucleus accumbens will be assessed using the procedure by Beiderbeck et al. (2012). Thirty minutes after the beginning of the RI test, each rat will be anaesthetized with carbon dioxide and rapidly decapitated. This is also done for the undisturbed control rats. Highly specific radiolabeled oligonucleotide probes and an imaging program will be used to quantify the mRNA expression of immediate early genes (Beiderbeck et al., 2012).

Rats fed an omega-3 enriched diet and who experience maternal separation or witness other rats' aggression are expected to show less aggression towards an intruder than those fed the non-enriched control diet. Although the brain continually relies on omega-3s and thus omega-3s are expected to have effects across the lifespan, the rats exposed to omega-3s earlier in life are expected to demonstrate less aggression than rats exposed later in life. Due to critical and sensitive periods, developing neural systems are more vulnerable to influence and therefore more subject to the benefits of omega-3s (Prescott & Dunstan, 2007). An earlier initial exposure also means an overall longer duration of supplementation. Since less aggression is expected among

rats fed the enriched diet, they are also expected to have reduced nucleus accumbens activation and reduced reward in response to an aggressive encounter.

If the results of Experiment 1 concur with the predictions, this would demonstrate promise of omega-3s to be implemented as a preventative measure against aggression in adulthood. If the results of Experiment 2 are as predicted, this would highlight the importance of mothers to supplement their diets with omega-3s as early as possible although further testing would be required in order to test for any detrimental effects of over-supplementation. Significant results would prompt further investigations that could focus on the severity of stressor or dose of omega-3s. Other future studies could examine the long-term consequences of increased omega-3 exposure over multiple generations or measure abnormal forms of aggression toward non-threatening intruders.

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