THE RELATIONSHIP BETWEEN AGGRESSION AND DEPRESSION: TESTING THE MODERATING EFFECTS OF AGE AND GENDER

by

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DEDICATION

To my mother: I would not have made it without you. To my family: thank you all for supporting me in my academic pursuits. I hope this thesis serves as an example that focus, drive, and passion can lead one to create much, and open doorways into knowledge, truth, and enlightenment. To Marriah: depression swept you from the world, and much of this thesis was written with you in mind. I will never forget the light you brought to my life.

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ABSTRACT

The purpose of this thesis is to a) explore the research related to aggression and depression and b) test hypotheses that focus on the relationship between aggression and depression in relation to age and gender over time. Aggression and depression are discussed extensively by examining a broad range of literature that points to environmental, social, individual, psychological, and biophysiological influences. First, aggression and depression are discussed separately in terms of their underlying theory, biosocial perspectives, typologies, behavioral and personality abnormalities, brain structures related to each, and neurochemical/neurological influences. The final sections of the literature review discuss how aggression and depression intersect as well as the research regarding age of onset for both behaviors. These subsections are meant to provide the reader with different lenses through which to frame a deeper understanding of aggression and depression and to expose the reality that aggression and depression are highly complex behavioral outputs for which there are a multiplicity of causes. A methodological outline including hypotheses, research design, sample characteristics, measurements of aggression and depression and covariates, and an analytical strategy frame this study. Lastly, results from the analyses are reported and discussed, contextualizing the findings drawn in this paper in light of extant research.

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LIST OF ABBREVIATIONS

Ach	Acetylcholine
ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactivity Disorder
A-H-PAG	Amygdala-Hypothalamus-Periaqueductal Grey
ANS	Autonomic Nervous System
ASB	Antisocial Behavior
ASPD	Antisocial Personality Disorder
BIS	Behavior Inhibition System
CD	Conduct Disorder
GABA	Gamma-Aminobutyric Acid
HPA	Hypothalamic-Pituitary-Adrenal
LD	Learning Disorder
MDD	Major Depressive Disorder
MAO	Monoamine Oxidase
NIMH	National Institute of Mental Health
ODD	Oppositional Defiance Disorder
PFC	Prefrontal Cortex
PIP	Planned, Instrumental, Predatory
RADI	Reactive, Affective, Defensive, Impulsive

CHAPTER ONE: INTRODUCTION

Aggression is inherently intertwined in the nature of human beings and is, in some cases, a celebrated pastime. It is not uncommon to see a myriad of television shows, news, movies, documentaries, sporting events, and many others depicting human beings fighting and shedding blood for the sake of victory, honor, and reputation. UFC fighters battle against each other in the octagon for a shot at a title belt. Television shows like *Dexter* glorify a psychopathic killer who only kills the bad people. Overzealous fans attend sporting events and erupt with cheers and screams when fights break out between the players. Movies like those recently released by the Marvel Universe depict stories of the tragic hero (or group of tragic heroes) who engage in explosive violence and bloodshed for the sake of humanity. These conduits of aggression are of course nothing new to humanity and are eerily reminiscent of the gladiatorial games in the Roman coliseum, put on by emperors to appease the plebian masses. As Erich Fromm (1973) wisely noted, "scenes of bloodshed and cruelty . . . are the staple diet fed to the public by press, radio, and television. People eagerly respond to such reports because they are the quickest way to produce excitement" (p. 278). While common knowledge intuitively understands what aggression looks and feels like, there is likely less awareness of the deeper anatomy of aggression: what its causal mechanisms are, how and where these causal mechanisms exist within human beings, and why aggression has roots much deeper that what is socially perceived.

Depression, in contrast, is certainly not a celebrated past time as it actually represents the bane and tragedy of many people's existence. Depression poses a series of physiological, mental, and emotional health problems that afflict people of every age, race, status, and walk of life. The National Institute of Mental Health (NIMH) (2018) estimated that in the year 2016, approximately 16.2 million (6.7% of the population) adults in the United States experienced a major depressive episode. Prevalence and risk for depression was highest among females, and the age bracket with the highest reported number of depressive episodes was 18 to 25 years of age. Of the 16.2 million individuals who experienced depression, approximately 10.3 million adults (4.3% of the population) experienced a major depressive episode with severe impairment (NIMH, 2018). These figures are quite staggering and suggest that depression is a serious issue affecting people in the U.S. Similar to perceptions and understandings of aggression, people in general may have some idea of what depression is and what it feels like to experience it. However, the deeper underpinnings of depression and its symptoms are likely less understood.

As such, the purpose of this thesis is to a) explore the research related to aggression and depression and b) test hypotheses that focus on the relationship between aggression and depression in relation to age and gender over time. Aggression and depression are discussed extensively by examining a broad range of literature that points to environmental, social, individual, psychological, and biophysiological influences. In sequential order, this thesis first discusses each behavior separately in terms of their underlying theory, biosocial perspectives, typologies, behavioral and personality abnormalities, brain structures related to each, and neurochemical/neurological

influences. The final sections of the literature review discuss how aggression and depression intersect as well as the research regarding age of onset for both behaviors. These subsections are meant to provide the reader with different lenses through which to frame a deeper understanding of aggression and depression and to expose the reality that aggression and depression are highly complex behavioral outputs for which there are a multiplicity of causes. A methodological outline including proposed hypotheses, research design, sample characteristics, measurements of aggression and depression and covariates, and an analytical strategy frame this study. Lastly, results from the analyses are reported and discussed, contextualizing the findings drawn in this paper in light of extant research.

CHAPTER TWO: LITERATURE REVIEW

Criminology has historically been akin to a pendulum, such that theoretical propositions emerge as valid explanations, but then subside as other theories take precedence as better, more useful explanations. Many researchers have developed theories that attempt to explain why crime occurs and perhaps indirectly why criminals are aggressive before, during, and after their criminal actions. Criminology has predominantly explained crime and criminality from social and environmental perspectives. The purpose of this section is to give a broad overview of criminological theories that can be used to explain aggressive propensities in humans.

Theories Explaining Aggression

In their theory of differential association, Sutherland and Cressey (1960) contend that individuals learn to be criminals through interactions with other criminals. Individuals differentially associate with other individuals and groups, and the values, beliefs, motivations, norms, and culture of these groups are learned by and transmitted to the individual, thereby reinforcing either favorable or unfavorable definitions that facilitate or impede criminal behavior. Favorable definitions of criminality become living definitions in that the individual imbued with them begins to actualize them during social interaction. If an individual learns that aggressive responsivity is at the core of the normative behavioral framework of the group, and also learns that aggressive behavior best accomplishes these norms and beliefs, then aggressive propensities will persist

(Sutherland & Cressey, 1960).[1] This theoretical viewpoint was further refined by Akers (1994) who expanded on differential association, definitions favorable to crime, differential reinforcement, and imitation with the aim of describing the deeper mechanisms of the learning process. Akers (1994) defined differential association in more detail, identifying an interactional dimension where individuals have both direct and indirect interactions and associations with other individuals and a normative dimension in which patterns of normative behavior are learned through the process of association. Further, Akers (1994) added the concept of differential reinforcement, where individuals' behavior generates perceived and real rewards and punishments resulting from the behavior through operant conditioning. If an individual not only perceives but actually experiences some form of external or internal reward for engaging in aggressive behavior, then aggression would likely become a common behavior within the individual.

Another branch of criminological theory which provides possible insight into aggressive behavior involves labeling, stigmatization, and how individuals respond to threats against the labels that have become intertwined with personal identity. Lemert's (1952) labeling theory focused broadly on two categories of deviance: primary and secondary. Primary deviance is generally a result of sociopsychological factors. Individuals engaging in primary deviance are not necessarily committed to a criminal identity, and are influenced by cultural, social, and psychological factors. Secondary deviance refers to the attachment of criminal labels that reinforce a "master status" (Lemert, 1952). A master status refers to the assimilation of a strong, entrenched criminal

[1] See Eron (1987), who argued aggression is learned through a four-staged process: 1) frustration is instigated, 2) aggression is reinforced, 3) the individual identifies, internalizes, and models their behavior around aggression, and 4) it becomes normative/acceptable behavior to the individual. Further, drives, cues, responses, and rewards are integral in the process of learning aggression.

identity as a result of being perpetually caught, labeled, and stigmatized. Consequences of this labeling process may involve individuals deliberately behaving in alignment with the negative label because there are assumed rewards and penalties of doing so (Lemert, 1952). A good example of this process can be seen in individuals who consistently enter and exit prison, or who have been in prison so long that the culture, values, and lifestyle behind bars permeates the identity of the individual. This process is well known in criminological literature as prisonization. In these circumstances, aggression is a kind of currency in the prison atmosphere, and individuals often face circumstances where there is no choice but to be violent. In other cases, a prisoner or criminal has earned a reputation for being overtly violent and violent reputations are equated with respect.

Prisonization closely aligns with two other theoretical positions in criminology, including Anderson's (1994) *Code of the Streets* and Messerschmidt's (1993) *Masculinities and Crime*. Anderson (1994) observed the culture among what he called street families and decent families in the urban ghetto which promoted the use of violence and aggression as a means to achieve street credibility or respect. The code by which both types of families lived by was based on respect, and when disrespect occurred, individuals were encouraged to use violence in defense of their personal image and self-worth, as well as to regain respect which was perceived to have been lost. Respect, according to Anderson (1994), was not only attributed to the ideological components and characteristics of the individual but was also attributed to physical objects owned by the individual, such as cars, clothing, and generally symbols of personal wealth. The individuals most likely to adopt this code of respect or "juice" were most likely poor, poorly supervised, harshly disciplined, unsuccessful, male, and who had experienced

some form of discrimination. Messerschmidt (1993) had similar views, arguing that crime is a means used to achieve masculine status. Young men engage in a variety of criminal activities because they perceive authority and disrespect as a source of emasculation. Crime, in this sense, is the process of doing gender where any means deemed necessary are taken by the individual to distance themselves and their identities from anything feminine (Messerschmidt, 1993). Much of the violence that occurs in response to disrespect or feeling emasculated is a response to deep feelings of shame and humiliation. Gilligan (1996) stated "the purpose of violence is to diminish the intensity of shame and replace it as far as possible with its opposite, pride, thus preventing the individual from being overwhelmed by the feeling of shame" (p. 111).[2] Aggression from a sociological perspective, then, can be conceptualized as a response to an encroachment or diminution of personal appraisals of self-identity, impact, and self-worth.

Control theorists offer a different perspective, often highlighting the nature and impact of social bonds between people, familial dynamics, and parenting practices on bad behavior (Gottfredson & Hirschi, 1990). For example, Gottfredson and Hirschi (1990) stipulated that bad behavior can be attributed to poor parenting practices within the family. When children are young, impulsive, and are otherwise showing low self-control, behaviors and actions deemed to be bad will most certainly happen, and the failure of parents to correct these behaviors when they do happen will lead to the flourishing of detrimental, impulsive, insensitive, aggressive, risky, shortsighted, and nonverbal

[2] "The emotion of shame is the primary or ultimate cause of all violence, whether toward others or toward the self. Shame is a necessary but not a sufficient cause of violence, just as the tubercle bacillus is necessary but not sufficient for the development of tuberculosis. Several preconditions have to be met before shame can lead to the full pathogenesis of violent behavior. The pathogenic, or violence-inducing, effects of shame can be stimulated, inhibited, or redirected, both by the presence or absence of other feelings, such as guilt or innocence, and by the specific social and psychological circumstances in which shame is experienced" (Gilligan, 1996, pp. 110-111).

behavior. Thus, these theorists argue that greater control and monitoring of behavior during childhood is key for the development of self-control (Gottfredson & Hirschi, 1990).

Other theoretical positions that closely align with these perspectives fall within the branches of developmental/life-course theory and integrative theories. One idea in life-course theory centers on social institutions which provide both formal and informal social controls which generate prosocial bonds and stakes in conformity, ultimately leading to the desistance of criminal behavior (Laub & Sampson, 2003). In the most basic sense, changes in situational and structural life circumstances from childhood to adolescence to adulthood (i.e., good marriage, stable job, positive individual behavior)[3] serve as turning points in an individual's life, resulting in criminal desistance. Of course, the absence of stability, prosocial bonds, and stakes in conformity are thought to increase criminal activity, leading to a much longer persistence of criminal behavior across the individual's life course (Laub & Sampson, 2003). In *Why Criminals Offend: A General Theory of Crime and Delinquency*, Agnew (2005) sought to explain the dynamic relationships between five clustered variables (i.e., the self, family, schools, peers, and work). It is argued that each of these variables occupies a separate domain. As constraints are reduced in each domain, motivation for crime is increased. Each domain is intertwined and interactive with the others, in varying degrees, and the relationships between them are nonlinear and contemporaneous (Agnew, 2005; see also Thornberry, 1987). Using developmental/life-course and integrative theoretical positions, aggressive

[3] Marriage as a stake in conformity does not always produce prosocial outcomes. For example, a marriage between two aggressive spouses may reciprocally reinforce criminal behavior, may result in the reproduction of genetically aggressive offspring, and may result in a larger quantity of aggressive children since research has shown aggressive parents tend create larger families (Wright, Tibbets, & Daigle, 2015).

behavior is a result of the absence of prosocial bonds or conflicting relational dynamics between the different domains of individual life. Without pillars of conformity optimally functioning in the dynamics of life, aggressive behavior is not tempered.

A pivotal development in developmental/life-course theory emerged when Moffitt (1993) unpacked the age-crime curve which depicts different trajectories of criminal behavior over the life-course among two distinct groups: adolescent-limited offenders and life-course persistent offenders. Among the adolescent-limited offenders, Moffitt (1993) argued that a maturity gap exists between biological maturity and social maturity. The maturity gap between individuals' social and biological development, then, encourages youth to engage in different forms of antisocial behavior, generally from ages seven to 17, followed by a steady decline in antisocial behavior as social and biological maturation converge. Moffitt (1993) contends this effect can be considered normative and part of the standard adjustment process of young individuals as they move through the life-course. In contrast, Moffitt (1993) suggests life-course persistent offenders are afflicted by neurophysiological deficits which interact with criminogenic environments. This interaction process facilitates the culmination of a pathological personality, leading to persistent offending and criminal behavior across the life-course. According to Moffitt (1993), the continuity of offending and life-course persistence of criminal behavior occurs among a small percentage of individuals, amounting to approximately five or six percent of offenders. Through Moffitt's (1993) theoretical lens, perhaps some forms of aggressive behavior are part of the normative developmental processes individuals experience as they age through biological and social maturation. Contrariwise, more severe forms of persistent aggression may partly be a function of the neurophysiological

deficits outlined by Moffitt (1993). Combined with other social and environmental factors, it is possible that some individuals who are extremely aggressive exhibit continuity in aggressive behavior as they age over the life-course.

Biosocial Perspectives of Aggression

In the early 1950s, Glueck and Glueck (1950), contrary to dominant sociological views of criminality, argued that no one theory can adequately explain criminal behavior, and an integrative approach that combines social, environmental, psychological, and biological elements would have higher explanatory power in identifying criminal traits. As such, this argument was one of the first to promote a biosocial perspective that recognized the power of integrating theory in the pursuit of explaining criminal and violent behavior. In their analysis of delinquent youth, these early theorists noted that many delinquents tended to have a mesomorphic body type (i.e., muscular and fit), and their temperaments were often marked by extroversion, impulsivity, aggression, and low self-control. Further, Glueck and Glueck (1950) alluded to the impact of heredity on delinquents, stating that, among the parents of delinquents, "There was a greater incidence of emotional disturbances, mental retardation, alcoholism, and criminalism" (p. 55). Criminality, according to this view, is the result of convergence between physical characteristics, temperamental traits, intelligence, and social influences (Glueck & Glueck 1950; see also Englander, 2006, p. 98).

More advanced theoretical propositions and research-based findings emanating from the biosocial perspective have pointed to the relationships between positive and negative emotionality, genetic and neurological implications of criminal behavior, and evolutionary characteristics that influence aggression and other criminal behavior (Caspi,

Moffitt, Silva, Stouthamer-Loeber, Krueger & Schmutte, 1994; Ellis & Walsh, 1997; Peskin, Gao, Glenn, Rudo-Hutt, Yang & Raine, 2013). For example, Ellis and Walsh (1997) suggest that to some extent, criminality is inherited. Genes likely contribute to the development of individual traits such as "pushiness" or "deception" because reproductive success may, in part, depend on their use. Further, genes influencing aggression may also explain why some males reproduce offspring but invest minimal to no support in raising their offspring (Cads). Because females may be disinclined to mate with a male who they sense will not invest time and energy, Ellis and Walsh (1997) theorize that "cad males will use just about any tactic that works to coax, trick, and/or force numerous females to copulate . . ." (p. 69). From this perspective, aggression can be viewed as an evolutionarily and genetically adaptive behavior designed to ensure that males successfully spread their genes by prolifically mating with as many females as possible, by whatever means are necessary, up to and including violence.

Biosocial perspectives in criminology have further advanced theoretical understanding by probing a variety of physiological mechanisms thought to be responsible for aggression, such as impulsivity, stress, self-control, intelligence, brain abnormalities, neurological deficits, and many more (Peskin et al., 2013). Peskin, Gao, Glenn, Rudo-Hutt, Yang, and Raine (2013) revealed that numerous studies involving health risks, genetics, neuroimaging, neuropsychology, psychophysiology, and endocrinology have identified multiple biological and physiological correlates in aggression and violent behavior. Furthermore, with the advancement in neuro-imaging techniques,[4] structural deficits in the lobes of the brain, including the frontal and temporal

[4] Neuro-imaging techniques use advanced medical technology that scan and measure different elements like glucose metabolism and blood flow within the human brain, which can be accomplished

lobes, have been found among individuals who exhibit antisocial symptomology and aggression.

Biosocial research on aggression (and similarly related behaviors) has also been advanced via neuropsychological studies which have shown that delinquent and criminal individuals, as well as those with serious aggressive behaviors, have executive cognitive function deficits, spatial and verbal deficits, and decreased intellectual function (Cohen, Brumm, Zawacki, Paul, Sweet, & Rosenbaum, 2003; Séguin & Zelazo, 2005). It has also been discovered that some aggressive individuals and criminals exhibit discrepancies in their autonomic nervous system (ANS) with evidence pointing to low arousal issues and hyporesponsivity or a diminished responsivity to stimuli (Peskin et al., 2013). Many different hormones, neurochemicals, and physiological abnormalities are considered culprits in aggressive behavior, including cortisol and glucocorticoids (which are particularly important in anxiogenesis), afferent dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, and abnormal levels of serotonin and testosterone (Montoya, Terburg, Bos, & Van Honk, 2012; Peskin et al., 2013). With a base understanding of the biosocial perspective, along with the general findings above, a more detailed discussion can be had regarding the nuanced and intricate aspects of aggression.

Aggression Typologies and Definitions

Generally, aggression falls into two main categories: aggression that is instrumental in purpose, and aggression that is hostile or reactive in purpose (Englander, 2006; Hochstetler, Copes, & Williams, 2010). Instrumental aggression is goal oriented

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using Computerized Axial Tomography (CAT), Magnetic Resonance Imaging (MRI), functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), and Diffusion Tensor Imaging (DTI). For a brief description of the nuances of each scanning technique, see Jorgensen, Anderson, and Barnes, (2016).

and is common among both human beings and all other living species. It typically emerges in several different forms, including predatory aggression, territorial aggression, intraspecies aggression, and parental aggression (Moyer, 1968; Fromm, 1973; see also Levi, Nussbaum, & Rich, 2010). In contrast, hostile aggression has at its core a desire to harm another, either physically, verbally, or otherwise in reaction to some stimuli (Englander, 2006). Hostile aggression is also sometimes considered to be hedonistic and as a means in and of itself; in this sense, it is violence for the sake of violence.[5]

Recent research in biosocial criminology and neurology has explored aggression as either reactive or proactive, often with the objective of identifying the many different biological aspects of each type of aggression. Studies have shown that reactive aggression is often marked by emotional instability or poor emotional control, whereas proactive aggression appears as calloused, typically akin to the types of behavior emitted by psychopaths (Lickley & Sebastian, 2018; see also Bezdjian, Tuvblad, Raine, & Baker, 2011). Researchers have further explored reactive and proactive aggression according to each's subtypes. Subtypes include "reactive, affective, defensive, impulsive (RADI) or 'emotionally hot' [aggression] and planned, instrumental, predatory (PIP) or 'emotionally cold'" (Steiner, Silverman, Karnik, Huemer, Plattner, Clark, & Haapanen, 2011, p. 21). Further, through rigorous scientific study, it was discovered that these two categories of aggression occur via separate circuit/structural systems in the brain (Steiner et al., 2011). For example, RADI or emotionally hot forms of aggression are linked to discrepancies within the circuitry connecting the medial nucleus of the amygdala to the medial

[5] "The wish to destroy for the sake of destruction is different. Only man seems to take pleasure in destroying life without any reason or purpose other than that of destroying. To put it more generally, only man appears to be destructive beyond the aim of defense or of attaining what he needs" (Fromm, 1973, p. 211).

hypothalamus and subsequently to the dorsal periaquaeductal gray (Steiner et al., 2011; see also Blair, 2010). This particular circuit is intertwined with the threat response systems in the brain, and research has shown that depending on the intensity of the stimuli experienced in the circuit, responsivity to the threat can involve simply freezing, engaging in flight or evasion, and lastly fighting to neutralize the stimulus (Steiner et al., 2011).[6] PIP or emotionally cold aggression types appear to incorporate a wider variety of brain structures in their etiology, such as the amygdala, ventro and medial prefrontal cortex (Steiner et al., 2011). A simple example provided by the authors is deceitful behavior, which may involve more complex processing schemes to accomplish the goal aside from simply using aggression. This would suggest that the behavior of deceit forces a complex interplay between multiple brain structures dealing with planning, reason, and emotional stability at the same time (Steiner et al., 2011).

Research has indicated hyper-responsivity in the amygdalae, insular cortices, and limbic systems in test subjects are indicative of higher reactive aggression levels (Lickley & Sebastian, 2018). It has been argued that, because the limbic system in adolescents develops much quicker than other parts of the brain (such as the neocortex and prefrontal cortex), emotional dysregulation, hypersensitivity to threats, and reactive aggression in adolescents is a result of brain development being non-concurrent (Lickley & Sebastian, 2018). This suggests that brain maturation is key in the development of higher regulatory systems and discordance between the growth rates of primitive and complex brain

[6] Activation of threat circuitry, including freezing, flight from the situation, or reactive aggression depends on the amount of stimulation being processed by the threat circuitry system. The more the threat circuity is stimulated, the higher the likelihood reactive aggression will result (Blair, 2010, p. 287)

structures may have disparate impacts on behavior, including aggression (Lickley & Sebastian, 2018).

In a study assessing aggressive and emotional responsivity, as well as fear potentiation in response to threats and provocative opponents, Beyer, Münte, Erdmann, and Krämer (2013) found that participant responses were neurologically modulated in particular areas of the brain, including the temporal-parietal junction, the medial prefrontal cortex, the precuneus, and the temporal poles. Interestingly, these structures have previously been found to directly mediate mentalizing processes, including empathy, predicting the actions of another person, and identifying emotional instability coming from external stimuli. The results of this study showed that individuals with low levels of fear potentiation simultaneously activated mentalizing networks, whereas mentalizing networks in high fear potentiation individuals were shut down or diminished. The inference here is that those individuals whose mentalizing networks were working avoided aggressive responses, and instead focused on empathizing or understanding the provocateur (Beyer, Münte, Erdmann, & Krämer, 2013).

There is an extensive body of biosocial research that has explored psychopaths and reactive versus proactive aggression. Some studies have focused on genetic and environmental characteristics influencing reactive and proactive aggressive propensities in psychopaths (Bezdjian et al., 2011), whereas others have analyzed reactive and proactive aggression through the lens of emotional or psychological frustration and its relation to brain structures in the psychopathic brain (Blair, 2010; Harenski & Kiehl, 2010). For example, it has been suggested that dysregulation in the amygdala-hypothalamus-periaqueductal grey (A-H-PAG) facilitates the ontogenesis of abnormal

behavior in psychopaths, including distorted aggressive reactions, unplanned aggressive acts, and enraged aggressive acts (Blair, 2010). There are several components of the brain that are responsible for regulating the A-H-PAG, including the orbital, medial, and ventrolateral frontal cortex. Blair (2010) posited that high susceptibility to frustration correspondingly activates these areas of the brain, which are also commonly malfunctioning structures in psychopathic brains (see also Harenski & Kiehl, 2010). The research linking biology with the different forms of human aggression is quite extensive and has empirically implicated the influence of multiple biological systems in the development of particularized typologies of aggressive behavior.

Behavioral/Personality Abnormalities

Behavioral and personality disorders and the overall temperament of individuals have strong correlations with biology. For example, according to one study, some 71% to 80% of the variance in externalizing behaviors like aggression, defiance, hyperactivity, and inattention in toddlers was genetically influenced (Van Hulle, Lemery-Chalfant, & Goldsmith, 2007 as cited in Wright, Tibbets, & Daigle, 2015). Furthermore, based on a meta-analysis of 103 studies of aggression, one researcher found that approximately 65% of the variance in aggressive behavior was genetically heritable (Burt, 2009 as cited in Wright, Tibbets, & Daigle, 2015). Other researchers have pointed out that genetically inherited aggressive temperaments in children may be environmentally exacerbated, especially in cases where parents engage in parenting practices that are aggressive as well, ultimately amplifying the temperamental abnormalities (Englander, 2006).[7]

[7] "Because the temperament of a child is influenced by the genes of the parents, it is likely that a child with a difficult, impulsive, or aggressive temperament also has parents with the same characteristics. Unable to effectively manage their problem child, parents may use hostility, criticism, and indifference as parenting tools. This child, much like the one experiencing peer rejection, will find his or her already

Compounding the issue of aggressive responsivity are findings suggesting that individuals who exhibit overly aggressive temperaments may find threats in emotional stimuli where the actual validity of threats are minimal or non-existent (Wilkowski & Robinson, 2010). This would seem to suggest that genetic traits, along with other factors, are sensitizing individuals in their perceptions of feeling threatened. This poses unique challenges in that correcting temperamental aggressive responses must include uncovering the intricacies of personal, albeit inaccurate appraisals of what constitutes threatening stimuli.

The biological roots of aggression also appear to have strong links with severe behavioral, personality, and cognitive disorders, including attention deficit hyperactivity disorder (ADHD), attention deficit disorder (ADD), oppositional defiance disorder (ODD), antisocial personality disorder (ASPD), antisocial behavior (ASB), conduct disorders (CD), learning disorders (LD), negative emotionality, lack of constraint, impulsive behavior coupled with low self-control, and other cognitive developmental impairments (Caspi et al., 1994; Englander, 2006; Levi, Nussbaum, & Rich, 2010; Wright et al., 2015; Carlotta, Borroni, Maffei, & Fossati, 2011). There is also evidence to suggest that ADHD, ODD, and other CDs can co-occur within a single individual and share a causal relationship with one another (Pringsheim, Hirsch, Gardner, & Gorman, 2015). There are likely a number of mediating factors that influence both the strength and predictability of these relationships. One particular study assessed the mediating role of impulsivity, sensation seeking, and reactive and proactive aggression on ADHD and

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unpleasant and ineffectual interaction style reinforced by the authoritarian style of parenting. In these families, an aura of distrust and a pattern of aggressiveness are clearly visible" (Wright, Tibbets, & Daigle, 2015, p. 192).

ASPD (Carlotta et al., 2011). In the sample of 729 high school students, the researchers discovered that males reported significantly higher proactive aggression scores than females. Furthermore, regression analysis found that all measures employed in the study were statistically significant. The findings suggested that early childhood ADHD symptomology has persistent impacts on other behavioral discrepancies across the life course, including impulsivity, aggression, sensation seeking, and ASB (see also Nevels, Dehon, Alexander, & Gontkovsky, 2010). While there are numerous biological, physiological, and environmental influences on behavior, personality, and individual temperament abnormalities, research does indicate that aggression seems to appear as a reoccurring behavior in different personality disorders.

Brain Structures and Aggression

Numerous physical structures that make up the human brain are implicated in the etiology of aggressive behavior. Many studies have focused on smaller structures within the limbic system (especially the amygdala and hippocampus) as possible culprits in the generation of aggressive propensities and a wide range of abnormal behavior (Blair, 2016: Lane, Kjome, & Moeller, 2011). According to Jorgensen, Anderson, and Barnes (2016), the amygdala (a small almond shaped structure within the limbic system) is directly involved in threat detection and anxiety. Interestingly, there appears to be evidence supporting the notion that aggressive behavior may share some relationship to the volume or size of the amygdala. For example, one study found in a comparison of aggressive and non-aggressive groups, the individuals in the aggressive group showed between a 16% to 18% reduction in the volume of the amygdala (Matthies, Rüsch, Weber, Lieb, Philipsen, Tuescher, & van Elst, 2012). Furthermore, smaller amygdala

volume was associated with a lifetime history of aggressive behavior. However, some research indicates that, instead of the whole amygdala volume having an effect on aggression and impulsivity, it may actually be particular parts of the amygdala that are responsible (Gopal, Clark, Allgair, D'Amato, Furman, Gansler, & Fulwiler, 2013). Instead of looking at the entire volume of the amygdala, recent research has parcellated the structure by observing smaller aspects of it, including the ventral and dorsal areas of the amygdala. When looking at the dorsal and ventral areas of the amygdala, research shows that these subsections are highly correlated with aggression and impulsivity. Moreover, there appears to be a vast array of studies focusing on amygdala volume and parcellation, some of which have indicated medial orbital frontal cortex-amygdala connectivity discrepancies, as well as more nuanced influences of neurochemicals on amygdala functionality (Gopal et al., 2013; see also Blair, 2016).

Because physical structures like the amygdala and hippocampus have previously been found to be associated with fearlessness and disinhibition, researchers have explored and targeted the compositional aspects of these structures as possibly indicative of aggressive behavior. Walters and Kiehl (2015) assessed grey matter volume in both the amygdala and hippocampus to see if reduced grey matter volume shared any relationship to increased fearlessness and disinhibition. Their findings revealed that reduced grey matter volume in the amygdala was associated with increased fearlessness and reduced grey matter volume in the hippocampus was associated with increased disinhibition. Furthermore, both brain structures negatively correlated with fearlessness, which suggests both structures play some part in the regulation of fear-based behavior and in the behavioral inhibition system (BIS) (Walters & Kiehl, 2015). Prior findings also indicate

that a poorly functioning amygdala may generate a kind of afferent dysregulation where social cues that would normally instigate fear, emotionality, or empathy are replaced by callous, unemotional, and aggressive responses (Lozier, Cardinale, VanMeter, & Marsh, 2014). However, research also supports the notion that increased activity in the right amygdala, especially in youth with severe conduct disorders, over-sensitizes individuals to threatening stimuli, resulting in higher levels of external aggression (Lozier et al., 2014).

Another brain structure found to be important in the understanding of aggression is the corpus callosum. The corpus callosum is the largest tract of white matter within the human brain. It is situated medially in the brain, connecting the left and right hemispheres, allowing them to properly communicate with each other (Schutter & Harmon-Jones, 2013; see also Jorgensen et al., 2016; Wright et al., 2015). Typically, research has found that females have a much larger corpus callosum than males have (Wright et al., 2015). This brain structure is necessary for message transmission and communication in the brain and has been shown to be pivotal in the cognitive well-being and functioning of human beings. While the corpus callosum and its connection to anger and aggressiveness is apparently up for interpretation, some research has shown that various forms of interhemispheric interference are linked with violent individuals and those with ASB (Schutter & Harmon-Jones, 2013). There are three kinds of interhemispheric issues thought to be associated with aggression, all of which are integral in the “callosal dysfunction theory of aggression” (Schutter & Harmon-Jones, 2013). These are suppression, isolation, and interference. Interhemispheric suppression occurs when one hemisphere prohibits the other hemisphere from processing information.

Interhemispheric isolation occurs when both hemispheres are actively processing information but are not transmitting or communicating with each other.[8] Interhemispheric interference occurs where one hemisphere sends conflictual information to the other which inhibits processing capability. These kinds of discrepancies, as well as abnormal structural aspects of the corpus callosum, have appeared in highly aggressive individuals (Schutter & Harmon-Jones, 2013; see also Wright et al., 2015).

Several studies of brain structures and aggression have pointed to elements such as the anterior cingulate cortex and different areas of the frontal cortex. For example, Visser, Ohan, Whittle, Yücel, Simmons, and Allen (2013) found asymmetric variations in these structures (as well as asymmetric variations in the amygdala and hippocampus) were inextricably linked with aggression and clinical disorders in psychiatric patients. In addition, their research revealed these asymmetries varied according to biological sex, such that increased hippocampal volume was found to increase aggression among females in the sample. Among males, asymmetric abnormalities were found in the anterior cingulate cortex which the researchers suggested may predispose males to engage in overt aggression more often than in socially accepted behavior. Findings also indicated that the anterior cingulate cortex is generally activated in the face of painful life events and has been directly linked with the creation of depression and anxiety when individuals face feelings of rejection (Wright et al., 2015). Analysis of the prefrontal cortex and the lateral prefrontal cortex has revealed 13% of the variance in impulsive behaviors and 10% of the variance in aggressive behaviors are explained by these structures alone (Gansler, Lee, Emerton, D'Amato, Bhadelia, Jerram, & Fulwiler, 2011).

[8] For example, split brain experiments have assessed how the two hemispheres of the brain process information after the corpus callosum was severed.

Another study of same sex twins focused on grey matter volume in the medial prefrontal cortex and lateral prefrontal cortex which were found to be associated with aggressive propensities (Coccaro, Cremers, Fanning, Nosal, Lee, Keedy, & Jacobson, 2018). Beyond these brain structures, there is a large amount of evidence pointing to other external influences impacting the optimal functioning of these brain structures. For example, brain lesions, head injuries, drug abuse, physical abuse, exposure to environmental toxins (e.g., lead), nutritional deficits and the like have all been shown to negatively impact both the anatomy and physiology of the brain (Bannon, Salis, & O'Leary, 2015; Englander, 2006; Jorgensen et al., 2016; Wright et al., 2015; Lane, Kjome, & Moeller, 2011).

Neurochemicals and Aggression

Perhaps the most fascinating research surrounding the etiology of aggression involves studies focusing on the influence of neurochemicals and hormones. Multiple neurochemicals and hormones are implicated in the etiology of aggressive behavior including Gamma-aminobutyric acid (GABA), acetylcholine (Ach), serotonin, dopamine, norepinephrine, monoamine oxidase (MAO), testosterone, and cortisol (Bronsard & Bartolomei, 2013; Englander, 2006; Mehta & Beer, 2010; Montoya, Terburg, Bos, & Van Honk, 2012; Shiina, 2015; Siegel & Douard, 2011; Takahashi, Quadros, de Almeida, & Miczek, 2011; Van Honk, Harmon-Jones, Morgan, & Schutter, 2010; Wright et al., 2015). There is substantial evidence to suggest that MAO is strongly linked with aggression. Firstly, MAO is a metabolizing neurochemical, designed to break down excess neurochemicals (like serotonin, norepinephrine, and dopamine) that linger in the synaptic cleft during message transmission (Wright et al., 2015). Further, excessive levels of serotonin point to increased aggressive behavior and impulsivity (Englander, 2006).

One possibility may be that MAO is not doing its job of cleaning up excess serotonin, dopamine, and norepinephrine, and thus these neurochemicals flood the brain, thereby clogging up the synaptic cleft which slows neurotransmission. Research has also indicated that high levels of norepinephrine and dopamine are linked with aggressive behavior (Fishbein, 2001 as cited in Wright et al., 2015). Another possibility occurs when MOA is doing too good of a job by cleaning up too much, thereby leaving the body quenching for appropriate levels of necessary neurochemicals. Both low and high levels of MAO are directly linked with aggression and criminal behavior, and it is not surprising that males typically have lower amounts of MAO than do females (Wright et al., 2015).

Serotonin is widely distributed throughout the human body, interacting with multiple systems, including blood circulation, muscle movement, mood, and behavior (Wright et al., 2015). In particular, the *s* allele of the 5-HTTLPR (a serotonin transporter gene) has been linked with depression, anxiety, hostile behavior, and aggression (Takahashi et al., 2011). Specifically, Takahashi, Quadros, de Almeida, and Miczek, (2011) identified "agonists at 5-HT1A and 5-HT1B receptors in the medial prefrontal cortex or septal area can *increase* aggressive behavior under specific conditions" (p. 1). One study of serotonin levels and aggression had participants reduce the amount of tryptophan (an essential amino acid in the formation of serotonin) in their diet and found that reduced levels of tryptophan increased the levels of aggression in test subjects (Moeller et al., 1996 as cited in Siegel & Douard, 2011). Still other studies have suggested that low levels of cerebrospinal fluid 5-hydroxyindoleacetate acid (CSF 5-HIAA) appear to increase levels of aggression, hostility, violence, and suicide (Wright et al., 2015). CSF 5-HIAA is a metabolite of 5 HT, meaning that it is a by-product of

serotonin being metabolized in the body (Moore, Scarpa, & Raine, 2002), and thus it is useful to track serotonin and its physiological transformation as it relates to aggression. Discrepancies in the underlying mechanisms responsible for creating, transmitting, and up-taking serotonin ultimately impact how the body and brain process serotonin neurochemicals. The consequences of these kinds of imbalances tend to appear as maladaptive behavior, such is impulsivity, selfishness, risk-taking behaviors, and aggression.

Research surrounding the nature of hormones, especially testosterone and the stress hormone cortisol has provided good insight into the nature of hostile behavior. Hormones, in comparison to neurochemicals, are much slower moving because these biochemicals are typically sent long distances throughout the body (Wright et al., 2015). Further, hormones are secreted by glands (such as the pituitary gland) into the blood stream and they interact with multiple systems in the body. It is well understood that the hormone testosterone plays a large role in transitioning the body from one state to another by dramatically increasing during the process of puberty (Rowe, Maughan, Worthman, Costello, & Angold, 2004) which is a time of rapid physiological change in young individuals' lives. Testosterone has also been suggested as an important hormone in the etiology of aggression, especially among males, and its study has received attention from researchers for several decades. For example, in an older study comparing violent versus non-violent criminals, it was found that testosterone levels were significantly higher among prison inmates who committed the most violent crimes (Dabbs, Frady, Carr, & Besch, 1987 as cited in Giammanco, Tabacchi, Giammanco, Majo, La Guardia, 2005). Another study by Booth and Osgood (1993) found a significant moderate relationship

between testosterone and adult deviance in a sample of men in the military. Some research on testosterone and aggression has produced mixed results, suggesting that the influence of testosterone on aggressive behavior is questionable (Giammanco et al., 2005). Rowe, Maughan, Worthman, Costello, and Angold (2004) noted that in their sample of younger boys, testosterone was related to nonaggressive behaviors among boys with deviant peers versus boys without deviant peers. This study also found that testosterone levels did not predict aggressive symptoms in conduct disorders (CD) (Rowe et al., 2004). In contrast, recent research suggests testosterone and aggression share a strong link with the orbital frontal cortex, with findings from one study reporting that reduced activity in the medial orbital frontal cortex mediated testosterone levels and aggression in test subjects (Mehta & Beer, 2010). Other hormones, such as the female estrogen and progesterone hormones, have also been linked with aggressive tendencies. One study found that women tended to commit more crimes during their premenstrual cycle, during which time estrogen and progesterone decrease while testosterone increases (Brown et al, 2006 as cited in Wright et al., 2015).

The stress hormone cortisol has also been studied by researchers in relation to its potential influences on aggressive behavior. Several studies have shown cortisol does share a relationship with aggression in certain individuals (McBurnett, Lahey, Rathouz, & Loeber, 2000; Van Bokhoven, Van Goozen, Van Engeland, Schaal, Arseneault, Séguin, & Tremblay, 2005; Vogel & Schwabe, 2019). McBurnett, Lahey, Rathouz, and Loeber's (2000) study of 38 young boys (ages 7 to 12) found that decreased amounts of cortisol in subjects were associated with early onset aggression. Furthermore, boys with low levels of cortisol showed three times the number of aggressive symptoms (i.e.,

threatening, fighting, using weapons, cruelty towards people and animals, and forceful sexual behavior) and were three times as likely to be considered aggressive by their peer groups (McBurnett et al., 2000). In a longitudinal study of young males aging from prepubescence through adolescence, researchers discovered that individuals who had the most aggressive symptoms of conduct disorders had the highest levels of cortisol as compared to controls (Van Bokhoven et al., 2005). It was also discovered that individuals with a history of antisocial behavior had correspondingly higher levels of cortisol, and that higher levels of cortisol shared a relationship with the group of individuals who had the highest rates of reactive aggression (Van Bokhoven et al., 2005). A recent study by Vogel and Schwabe (2019) also found links between cortisol levels and aggression, specifically among respondents in their sample who had high trait aggression scores. Although the study indicated that high cortisol levels were associated with more avoidant behaviors in response to threats, individuals who were predisposed to aggressive behaviors, coupled with high cortisol levels, engaged in more approaching, rather than avoiding behaviors (Vogel & Schwabe, 2019).

There are several studies that focus on testosterone and cortisol as a duo, typically finding less than positive results in terms of prosocial behavior. Van Honk, Harmon-Jones, Morgan, and Schutter (2010) state the Triple Imbalance Hypothesis of reactive aggression holds that "reactive aggression is essentially subcortically motivated by an imbalance in the levels of the steroid hormones cortisol and testosterone" (p. 67). The major threat is that not only are individuals with dysregulated testosterone and cortisol levels more likely to be predisposed to violence, but cortical-subcortical communication in the brain is greatly inhibited, which happens to be integral in the regulation of socially

aggressive behaviors (Van Honk, Harmon-Jones, Morgan, & Schutter, 2010; see also Bronsard & Bartolomei, 2013). Unfortunately, high levels of testosterone and low levels of cortisol appear to negatively influence regulatory conduct among humans. More recent inquiries suggest that testosterone and cortisol are highly correlated with aggressive propensities, such that individuals who show high aggression with high levels of testosterone also show low levels of cortisol at the same time (Montoya, Terburg, Bos, & Van Honk, 2012). Further, aggression resulting from sleep deprivation has been directly linked with both testosterone and cortisol, with one study showing that testosterone and cortisol have some impact on sleep cycles and circadian rhythm (Bronsard & Bartolomei, 2013). The research in this area of human biology is quite extensive, and it is likely that the more researchers focus in on the nature and influence of hormones, the more findings are surely to arise.

Theories Explaining Depression

Criminological theory is noticeably scarce in terms of explaining the relationship between depression and criminal behavior. Although there is no canonical theory of depression and crime, criminologists have researched the topic in different ways. However, much of the theory exploring the etiology and persistence of depressive behavior has its roots in completely separate academic disciplines, mainly psychology and psychiatry. While a discussion of the entire theoretical base of depression is not accomplished in this thesis, a few key theoretical underpinnings are explored with the intention of providing a basis through which depression can be understood, and how depression relates to aggression and criminal behavior.

There are several different theoretical explanations of depression. Some consider cognitive mechanisms of depression while others point to purely biological causes of depression. The behaviorism approach was founded by J. B. Watson (1914), holding at its core that the behaviors and activities of individuals were the most important items to be measured since they could be directly observed, versus studying subjective and internal qualities that could not be directly observed (as cited in Fromm, 1973). Years later, B. F. Skinner (1953) took the foundations of Watson's literature and argued for an operant conditioning model. This model suggests individuals' behavior is positively reinforced (behavior is repetitiously rewarded), ultimately creating conditioned responses (the individual learns to expect a reward in return for engaging in the behavior). The main assumption of operant conditioning is that previous goal or value attainment and reinforcement of those same goals or values condition specific behavioral responses (as cited in Fromm, 1973). Lewinsohn (1974) argued that low positive reinforcement could actually lead to the onset of depressive symptoms in certain individuals. In their study of the impact of depression on positive and negative social skills, Libet and Lewinsohn (1973) contended that depressed individuals were more likely to have lower social skills than non-depressed individuals. Social skill, according to their definition, is "the complex ability both to emit behaviors which are positively or negatively reinforced and not to emit behaviors which are punished or extinguished by others" (p. 304). The authors hypothesized that depressed individuals, in a social setting, would emit fewer interpersonal behaviors and actions than their non-depressed counterparts. Results indicated that, indeed, depressed individuals showed lower rates of behavior emission compared to non-depressed individuals (Libet & Lewinsohn, 1973). Fundamentally,

depression and its common symptoms are considered as a secondary consequence of the causal influence of low positive reinforcing stimuli emanating from both social and environmental conditions.

Another major theoretical approach in the exploration of depression concerns the Cognitive Theory of Depression. Originally developed by Aaron Beck (1967), the Cognitive Theory of Depression holds that self-appraisals of self-worth and value, negative personal beliefs, and persistent negative interpretations of one's self form the basis of what is called cognitive depression (as cited in McLeod, 2015). Generally, three cognitive mechanisms are considered as the root causes in the development of depression. These three mechanisms are the cognitive triad, negative self-schemas, and errors in logic. The cognitive triad refers to negative views about one's self, negative views about the world, and negative views about the future. A central feeling operating and creating substance within these three views is the feeling of absolute helplessness about who one is, the state of the world as it relates to the individual, and one's ability to alter future trajectories (McLeod, 2015). Beck and Alford (2009) would later note that certain kinds of thought content are foundational in depression as precipitated by helplessness, including "ideas of personal deficiency, impossible environmental demands and obstacles, and nihilistic expectations" (p. 240). As the three elements of the cognitive triad intersect, individual cognitive stability is disrupted, thereby facilitating conditions conducive to depressive behavior. After depression has settled in, the result is often the creation of negative self-schemas (McLeod, 2015). A negative self-schema is a cognitive framework marked by negative and pessimistic belief structures pertaining to the internal and external components of an individual's life. McLeod (2015) suggested that specific

experiences likely contribute to these types of schemas, including loss of a parent, bullying, being abused, receiving criticism, and rejection among other things. The third component of the triad, logical errors, tend to be the result of an entrenched negative schema. Logical errors are self-defeating thought patterns predicated on a series of flawed, illogical, or otherwise misguided justifications. Beck's (1967) original cognitive theory identified five particular logical errors which include the following: (a) making arbitrary or negative inferences in the absence of evidence; (b) selectively or only abstracting the most negative elements of a circumstance; (c) magnifying the scope and size of particular problems, and minimizing the solutions to those problems; (d) personalizing or continually perceiving one's self as responsible; and (e) dichotomous thinking where it is always either A or B, but never both (Beck, 1967; see also McLeod, 2015).

A theory that overlaps with the Cognitive Theory of Depression is Martin Seligman's (1974) Theory of Learned Helplessness. According to Peterson and Seligman (1984), learned helplessness results when individuals personally experience events that are entirely out of their control, and this lack of control facilitates cognitive maladaptive responses, including emotional and motivational impediments. The original foundation of this theoretical construct was based on animal studies, particularly involving canines. In the original study, dogs were set in a cage divided by a small partition. One side of the cage floor administered a light shock, and the other side did not. As a result of being shocked, the dogs would move to the other side to avoid discomfort. However, when the dog's mobility was restricted, forcing them to receive the shock with no escape, they learned to simply sit on the side that administered shocks, even when the mobility

restriction had been removed (Peterson & Seligman, 1984; see also Alloy, Abramson, Metalsky & Hartlage, 1988; McLeod, 2015; Seligman, 1974). By simulating a state of absolute powerlessness to effect any change in position or circumstance, the research showed that, even when options to escape some unpleasant experience existed, the prior knowledge and experience of absolute helplessness overpowered the ability to potentially escape. This insight would later be applied as a theoretical justification for human depression, such that individual circumstances can be so overwhelming that, even if options are available to reconcile the situation, individuals have learned that the circumstances are simply hopeless, and thus the depression cycle perpetuates itself. After a series of criticisms of Seligman's learned helplessness theory, Abramson, Seligman, and Teasdale (1978) reformulated the theory to include multilevel causes that individuals would subsequently attribute their learned helplessness and depression to. According to the reformulation, individuals may assign causality as stable or unstable, global or specific, or internal or external. Each of these views are thought to be related to the persistence, desistance, and longevity of depressive symptomology. Peterson and Seligman (1984) state that internal explanations refer to individuals who believe their learned helplessness is caused by some individual characteristic within themselves, versus an external explanation that places blame on different aspects of circumstances or situations. Global causality stipulates that helplessness will affect a variable number of outcomes, whereas specific causality will impact a single event or single concern of the individual. Lastly, causes of helplessness and resulting depression can either be stable (remaining persistent over time) or unstable (changing and dynamic over time). Ultimately, it is believed that the prevalence of both internal explanations and stable

circumstances undermine individual self-esteem and further degrade personal well-being, resulting in persistent feelings of helplessness and depression (Peterson & Seligman, 1984).

Another theoretical perspective of depression focuses on the power of ruminative cogitation on negative thoughts, experiences, or circumstances. According to this view, individuals engage in repetitious thought patterns that focus a large amount of mental energy on negative emotions, feelings, and states of distress which then influence depression (Nolan-Hoeksema, 2000). Included in this etiology is the intersectionality and compounding of depressive symptomology with anxiety symptomology. For example, Nolan-Hoeksema (2000) noted that many ruminative thought patterns are marked by thoughts of uncertainty about situations and this uncertainty generates some level of anxiety within individuals. Further, the combination of depression and anxiety may share a relationship with states of learned helplessness, negative views of one's self and negative cognitive schemas, as well as the view that solutions to problems are worthless or are unlikely to succeed (Nolen-Hoeksema, 2000). Thus, individuals who engage in ruminative thoughts about the causes and consequences of their depression or anxiety struggle to take action in remedying the situation, often resorting to thinking deeply and repetitiously about the dismal characteristics of their experiences (Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Often, the result of distorted and repetitive thinking, especially concerning depression, generates and sustains the underlying causes of depression, and sustains more superficial depressive symptoms. There is also evidence suggesting that ruminative thoughts can contribute to depression in women more than men. For example, Nolen-Hoeksema, Larson, and Grayson (1999) sampled 1,100 adults

ranging in age from 25 to 75, and found that chronic strain levels, low mastery of one's life, and ruminative thought patterns significantly contributed to depression in women more than in men. Theory on this subject matter suggests that women have less power and control over their environments than do men, and this loss of power and control directly impacts self-esteem levels, ruminative thought, stress, and life strain. As a consequence, feedback loops are established which enhance the persistence of ruminative thought patterns, especially in women (Nolen-Hoeksema, Larson, and Grayson, 1999).

The theories and perspectives of depression and its etiology have served and continue to serve as a foundation for researchers in the study of depression. While most of these perspectives have focused on environmental, social, and psychological aspects of depression, there is research suggesting that biology (and biology in tandem with social factors) plays a role in the development and sustainability of depressive symptomology.

Biosocial Perspectives of Depression

Biosocial perspectives of depression generally recognize that depression can develop as a consequence of the dynamic interplay between psychological, social, environmental, and biological factors (Iloh, Orji, Chukwuonye, & Ifedigbo, 2018). Some individuals have biological or physiological predispositions for depressive behavior which can be exacerbated by experiencing negative or stressful social and environmental stimuli. For example, some studies on environmental and social generators of depression have focused on family disruption as a possible explanation. Iloh, Orji, Chukwuonye, and Ifedigbo (2018) found depression shared a significant relationship with negative family functioning, low familial support, a lack of cohesiveness within families, and familial conflict. The authors suggest that positive family environments are likely to exhibit

strong kinship bonds that provide support and reinforcement in a myriad of ways, including food, shelter, resources, and emotional support. When the ecological and social dynamics of families are broken or lack cohesion, the probability of experiencing and promoting the persistence of individual adversity and depressive mood increases (Iloh et al., 2018). Other studies on family disruption indicate that familial conflict, divorced parents, parental conflict, and low socioeconomic status increase the probability of experiencing depression (Gilman, Kawachi, Fitzmaurice, & Buka, 2003). When degraded familial conditions intersect with biological factors (such as heritability of traits), the outcomes can be negative.

Family studies of depression have also explored the heritability of depressive symptomology. An early study by Kallmann (1952) generated expected heritability percentages of manic depressive disorders among blood relatives of patients with manic depression. For parents, half siblings, full siblings, non-identical twins, and identical twins, the expected heritability percentages were 23.5, 16.7, 23, 26.3, and 100 percent, respectively (as cited in Beck & Alford, 2009), although these heritability estimates are debatable. Lesch (2004) noted that considerable evidence exists for the complex interplay between genetic and environmental influences of depression, especially when considering the findings from twin and family-based research. For example, the heritability of depressive symptoms apparently ranges from 40% to 70%, and relatives of individuals experiencing depression are three times more likely to experience depression (Lesch, 2004; ; see generally Jang, Livesley, Taylor, Stein, & Moon, 2004). Heritability of depression has also been analyzed in a number of twin studies. Kallman (1952) found that in 22 separate cases of monozygotic twins, both co-twins were classified as manic

depressive, often showing the same kinds of depressive symptoms (as cited in Beck & Alford, 2009). A much more recent study by McGuffin, Rijsdijk, Andrew, Sham, Katz, and Cardno (2003) found very high heritability of bipolar affective disorder. The study tested 67 twin pairs (30 monozygotic and 37 dizygotic) and found that the heritability estimates of bipolar affective disorder among the sample was approximately 85% (McGuffin, Rijsdijk, Andrew, Sham, Katz, & Cardno, 2003). Twin studies have also assessed depression in monozygotic twins who have been reared and raised in separate environments. Shields (1962) analyzed 44 pairs of monozygotic twins raised in separate environments and compared them with a sample of 44 monozygotic twin pairs who were raised together. Findings from this study indicated that both separated and non-separated twin pairs shared numerous behavioral similarities, including overall mannerisms and temperament (as cited in Beck & Alford, 2009). These findings would suggest that biological and physiological underpinnings related to depression among twins are heritable to a certain degree.

There is a wealth of other biological and physiological related research into depression, its underpinnings, and its persistence. The notion that neurological components influence depressive states was considered by theorists many years ago, including Kraines (1965) who suspected that certain depressive types, such as postpartum depression, premenstrual depression, and manic episodes were ultimately caused by hormonal imbalances in the human body (as cited in Beck & Alford, 2009). Studies have focused on biochemical abnormalities in blood glucose levels, physiologic tolerance to glucose, alkalinity and acidity levels in the blood, calcium and phosphorus levels in the blood, and a host of other physiologic elements that seem to influence depression to one

degree or another (Beck & Alford, 2009). Other studies have exposed potential influences on depression stemming from endocrine system functions, with some evidence indicating that rate of steroid metabolism shares a relationship with different levels of depression. Still other research has focused on other pertinent physiological systems and functions in the development of depression, such as the autonomic nervous system, blood pressure and salivation, neurochemical influences, structural neurophysiological components, and in some cases, anatomical pathologies within specific brain structures. All of these components appear to share some relationship with depressive symptoms (Beck & Alford, 2009).

Evidence suggests that individual biological factors coupled with social adversity and other environmental drawbacks collectively precipitate depression, and this view is becoming an accepted approach that acknowledges bioenvironmental influences on depression (Lesch, 2004). However, among the numerous studies focusing on depression, very few solidified results have persisted due to methodological errors or subsequent studies finding alternative results. Beck and Alford (2009) note that some findings in the biological literature of depression are reliable, including the relationship between depression and excessive steroid levels in the body, sodium retention issues, and sleep disturbances. While these findings are not exhaustive, it is important to keep in mind that much of the biosocial literature has produced tentative, rather than concrete, results.

Depression typologies and Definitions

Depression types can broadly be categorized as endogenous and exogenous. As Beck and Alford (2009) point out, two schools of thought have opposing views on the inclusion of these terms and as to the nature and types of depression. Gradualists view

depression as a unitary concept that simply manifests itself in a number of ways. Gradualist proponents do not subscribe to multiple definitions of depression. Separatists, on the other hand, view the etiology of depression as either endogenous or exogenous. Endogenous depression refers to depression that is caused by biological abnormalities or underpinnings within individuals. Examples of endogenous depression causality include such things as neurochemical imbalances, hormonal imbalances, and other types of physiologic dysregulation. Exogenous depression (also called reactive depression) is thought to be caused by external factors which individuals react to and that cause some form of depressive behavior. Examples of external influences can include financial strain, external loss, and a host of other social and environmental influences (Beck & Alford, 2009).

The list of depression types and their symptoms are extensive and well documented. According to the National Institute of Mental Health (NIMH) (2018) some well-known examples of depression types include persistent depressive disorder, postpartum depression, psychotic depression, seasonal affective disorder, bipolar disorder, and disruptive mood dysregulation disorder. Along with the several types of depression comes a very long list of notable symptoms that are psychological, emotional, and physical. Some examples of common depressive symptomologies outlined by the NIMH include persistent sadness, anxiety, hopelessness and helplessness; pessimism, irritability, and guilt; feelings of worthlessness; lack of motivation, interest, or pleasure; lower energy levels and fatigue; restlessness and disrupted sleep cycles; talking and moving slowly; impeded concentration, memory, and indecisiveness; appetite and weight fluctuations; suicidal ideation, suicide attempts and a few others (NIMH, 2018).

Aside from the litany of symptoms, each depression type is marked by different characteristics. For example, the NIMH (2018) states that persistent depressive disorder, previously dysthymia, is marked by fluctuations in depressive behavior from less to more severe depressive symptoms and persists for no less than two years. Post partem depression may affect women during pregnancy and after birth. Typically, women suffering from post partem depression experience major depression with symptoms of exhaustion, sadness, and anxiety both during and immediately following their pregnancy. Psychotic depression involves a combination of major depression and different forms of psychosis, including the existence of delusions and hallucinations of personal guilt or adversity. Seasonal affective disorder is a form of depression that waxes and wanes according to the time of year or season. Typically, the winter months or wet season increase depressive feelings, but they reduce in severity during the summer months. Disruptive mood dysregulation disorder was more recently added to the DSM-5 and is unique in that it pertains to children. In this type, there are severe bouts of aggression and irritability, as well as a high occurrence in the frequency of temperamental or violent outbursts. The symptoms, as they occur, also impair individual focus and social abilities, such as retaining positive relationships with family or peers (NIMH, 2018). Bipolar disorder (which can be manic or hypomanic) is characterized by severe mood swings which oscillate between major bouts of depression and upswings during manic episodes. The pendulum-like nature of this disorder facilitates changes in mood, energy, appetite, and activity. According to the NIMH (2018), symptoms on the depression side include sadness, feeling down, lack of energy, low activity, loss of sleep, and several other symptoms. Interestingly, manic episodes are often marked by feelings of elation and

gratification, high energy or activity levels, insomnia, feeling “wired” or upbeat, as well as an increase in risk-taking behaviors. Beck and Alford (2009) specify notable differences between Bipolar-I and Bipolar-II disorder types. Bipolar-I differs from Bipolar-II in that it involves at least one prior manic episode or mixed depressive episode, whereas Bipolar II has no prior manic episodes but is marked by prior hypomanic episodes. Hypomanic episodes are a less severe manic episode, but generally involve the same symptoms as regular manic episodes and have the potential of evolving into a full blown manic state. Beck and Alford (2009) outline several common behaviors and symptoms that are likely to occur during a manic phase which can include some or all of the following: (a) emotional manifestations in the form of elation, increased gratification, self-love, increased attachment to people/activities, and increased mirth responses (aka happiness); (b) cognitive manifestations involving increases in positive self-image, positive expectations, assignment of blame, denial, arbitrariness, and delusions; (c) motivational manifestations including impulsivity, orientation toward action, drive for independence, and focus on self-enhancement; and (d) physical/vegetative manifestations including hyperactivity, aversiveness to fatigue, increases and decreases in appetite, increased libido, and insomnia (Beck & Alford, 2009, pp. 95-100). Many of these behaviors and symptoms, at face value, may appear quite positive. However, it is the pendulum changes between the above listed behaviors and symptoms and their complete opposites which serve as the hallmark of bipolar disorder or manic depression (Beck & Alford, 2009).

Behavioral/Personality Abnormalities

There is reason to believe that depression commonly shares a relationship with other personality disorders and behavioral abnormalities within individuals. A personality disorder is generally defined as a routinized, enduring pattern of behavior that deviates substantially from an individual's expected normative behavior (NIMH, 2018). Other researchers have stated personality disorders are a manifestation of maladaptive personality traits which are rigid and persistent, and typically emerge in adolescent or early adult years (Shea, Widiger, & Klein, 1992). Based on the National Comorbidity Study Replication (NCS-R) conducted between 2001 and 2003, the prevalence of personality disorders in U.S. adults 18 years of age and older was approximately 9.1%. Shockingly, 84.5% of those with a personality disorder were also found to have co-occurring mental disorders (NIMH, 2018). Research has indicated that in clinical samples of both inpatient and outpatient depressed individuals, "high rates of personality disorders is the typical finding. Reported estimates range from 23% to as high as 87%, with most reporting at least 30% to 40%" (Shea et al., 1992, p. 4). Several researchers have explored the comorbidity of depression and personality disorders and have found relationships between the two. For example, Hirschfeld (1999) noted that depression and personality disorders share a unique relationship in three distinct ways. First, individuals with personality disorders can subsequently develop some form of depression which suggests personality disorders precede the onset of depression and/or serve as a predisposition toward depression vulnerability. Second, depression can precede a personality disorder, indicating that depression may be facilitating the development of a particular personality disorder. Third, depression and personality disorders interface and

co-exist simultaneously, and this has subsequently been referred to as depressive personality disorder (Hirschfeld, 1999). In one study of 294 individuals with major chronic depression, approximately 48% showed the existence of any personality disorder, and more than half of the sample was found to have a co-occurring personality disorder, including avoidant (21%), dependent (9%), obsessive compulsive (13%), self-defeating (15%), paranoid (6%), and borderline personality (8%) disorders (Keller et al., 1998, as cited in Hirschfeld, 1999). Research has suggested that the nature of some personality disorders (both in terms of affiliated behavioral discrepancies and specific personality traits) come with predispositions that may enhance the probability of developing and sustaining depression. Personality traits or behaviors that predispose toward depressive outcomes can include increased sensitivity to perceived slights, quickness toward frustration or angst, high levels of interpersonal dependency, the need for assurance, and excessive need for support (Hirschfeld, 1999).

As previously mentioned, numerous conduct disorders, such as ADHD, CD, LD, ADD, ODD, ASPD, anxiety disorder, and several other forms of psychopathological discrepancies share a relationship with aggressive tendencies. Interestingly, scholars analyzing depression have found systematic links between these same behavioral pathologies and depression. For example, Angold and Costello (1993) conducted meta-analysis on several studies that focused on the comorbidity of depression and particular conduct related disorders in children and adolescents. Based on their findings, every study analyzed in their analysis (excepting two pertaining to ADHD) showed a comorbid relationship existed between depression and behavioral disorders. Regarding the studies done on CD/ODD and depression, every study reported statistically significant comorbid

relationships with depression (Angold & Costello, 1993). Further, CD and ODD rates were approximately 3.6 to 9.5 times more prevalent in individuals with depression than those with no depression (Angold & Costello, 1993). Similarly, every study focusing on anxiety disorders and depression indicated individuals with depression were between 2 and 26 times more likely to have an anxiety disorder versus individuals with no depression (Angold & Costello, 1993). With respect to studies on the comorbid relationship between depression and ADHD, five of the seven studies in the meta-analysis found significant relationships between ADHD and depression in the samples observed (Angold & Costello, 1993). The authors ultimately concluded that "conduct or oppositional disorder and anxiety disorders are definitely, and attention deficit disorder probably, more common in depressed than non-depressed children and adolescents" (Angold & Costello, 1993, p. 1783; see also Wozniak, Spencer, Biederman, Kwon, Monuteaux, Rettew, & Lail, 2004).

Cole and Carpentieri (1990) conducted a similar study of comorbidity between CD and depression in a sample of 1,464 4th graders. Due to concerns about similar measures being used to examine both depression and CD, each construct was measured via dissimilar methods and potential similarities were controlled for to avoid arbitrary inflation of comorbidity. The results of this study found CD and depression shared a comorbid relationship, even after controlling for potential similarities. The authors noted one of their models found the expected probability threshold for a child having both depression and a conduct disorder was exceeded by two and a half times, indicating that the occurrence of dual pathologies was beyond random chance. Another model indicated a strong correlation value between the dimensional components of both CD and

depression, which substantiates potential comorbidity (Cole & Carpentieri, 1990). Other research findings indicate a similar relationship between ODD and depression. One study of females between five and eight years old indicated that one of the core dimensional qualities of ODD (i.e., negative affective behavior) shared a direct relationship with depression (Burke, Hipwell, & Loeber, 2010). Interestingly, two other dimensional qualities of ODD (oppositional behavior and antagonistic behavior) were more closely associated with CD, yet the results of the study did not find that CD predicted later depression (Burke, Hipwell, & Loeber, 2010). Still other studies have sought to address the interrelatedness and co-development of depression with ODD and anxiety. In this regard, Leadbeater, Thompson, and Gruppuso (2012) measured the relationship between these three behavioral pathologies using four waves of data on individuals ranging in age from 12 to 26 from the Victoria Healthy Youth Survey (HYS). Several important findings were yielded in this study. Depressive symptoms were found to be more common among females than for males. For every one-year increment in age, anxiety symptomology increased for both males and females. ODD symptomology was more prevalent among younger sample members, appearing to level off as age increased. The data consistently showed that high initial levels of one measured construct showed high corresponding levels in the other two. Further, moderate correlations were found between depression and anxiety, and depression and ODD. Perhaps most importantly, depression, anxiety, and ODD appeared to co-occur at every time point, and individuals who had increasing levels of depression simultaneously had increasing levels of both anxiety and ODD (Leadbeater, Thompson, & Gruppuso, 2012; see also Wolff & Ollendick 2006).

Recognizing that the literature extensively documents the comorbid relationships between depression and behavioral disorders is important. Another important consideration involves recognizing that many of the symptoms that comprise depression are also evident in other behavioral disorders, irrespective of whether depression is apparent or not. This represents a potential problem since many of the variables used to measure depression exist within other disorders, potentially causing false collinearity or arbitrary significance in shared relationships. As one researcher noted, while the DSM manuals have consistently kept depression as its own construct despite its strong relationship with other disorders, such as conduct disorder, other manuals such as the International Classification of Diseases (ICD-10) has combined depression with other disorders into a single pathological indicator (Burke, Hipwell, & Loeber, 2010). In response to the research on comorbidity, Wolff and Ollendick (2006) sought out to address why research typically finds such a strong link between the depression and other disorders and why the rates appear to surpass simple random chance. They propose four possible reasons. First, methodological errors may arise due to pitfalls in informational or referral biases. Second, many of the definitional components of depression and behavioral disorders overlap, thus causing an over-inflation of comorbidity rates, and perhaps other methodological/statistical issues like multicollinearity. Third, as was mentioned earlier, one outcome may be predicated on the other, such that certain disorders cause depression, or depression causes subsequent disorders via a pathological vulnerability. Lastly, depression and a particular disorder may be comorbid because the same risk factors that undergird one simultaneously undergird the other (Wolff and Ollendick 2006). Despite these potential influences, it is very likely that both researchers

are well aware of the interconnectedness between depression and behavioral disorders, and thus are likely to control for these influences in their research to avoid potential biases in the results. Moreover, based on the above-mentioned research, it is likely that depression shares a unique causal relationship with a myriad of behavioral disorders and pathologies.

Brain Structures and Depression

Several studies have identified particular brain structures and areas of the brain associated with depression and its common symptoms. Key structures that regulate emotion and stress, like the thalamus, hypothalamus, pituitary gland, and the hypothalamic-pituitary-adrenal (HPA) axis are pivotal in studies of depression. Other areas of interest associated with depression include the cingulate gyrus, caudate nucleus, and cerebellum, each of which has been associated with depression, especially when coupled with brain atrophy (i.e., decreases in brain tissue and/or loss of neurons) (Videbech & Ravnkilde, 2004). Common brain structures and areas associated with depression typically studied by researchers include the hippocampus (Lee, Ogle, & Sapolsky, 2002; Posener, Wang, Price, Gado, Province, & Miller, 2003; Sheline, Mittlera, & Mintun, 2002; Videbech & Ravnkilde, 2004), amygdala (Hastings, Parsey, Oquendo, Arango, & Mann, 2004; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007; Whalen, Shin, Somerville, McLean, & Kim, 2002; Zavorotnyy, Zöllner, Schulte-Güstenberg, Wulff, Schöning, & Dannlowski, 2018), and particular areas of the ventral, medial, dorsal, and lateral prefrontal cortex (Drevets, Price, & Furey, 2008; Koenigs & Grafman, 2009; McEwen, 2005).

The hippocampus resides in the lower limbic system and is integral in the formation of long term memory, information integration, differentiation and associational tasks, and forming cognitive maps (Wright et al., 2015). Functional deficits of the structure, including "episodic, declarative, contextual, and spatial learning and memory deficits" are thought to be related to depression (Videbech & Ravnkilde, 2004, p. 1957). The hippocampus is fundamental in the etiology of depression, especially when persistent stressors are applied to it which can result in structural damage, including atrophy and neuronal death (Lee, Ogle, & Sapolsky, 2002; Wright et al., 2015). Meta-analyses and other studies on hippocampal volume and depression linked reduced volume of the hippocampus in individuals with unipolar depression and major depressive disorder (Sheline, Mittlera, & Mintun, 2002; Videbech & Ravnkilde, 2004). Changes in terms of volume, water content, density, size, shape, as well as volumetric changes in both the right and left hemispheres of the hippocampus have been associated with depression in certain instances. However, not all studies find similar results. A meta-analysis of 12 studies assessing hippocampal volume discovered heterogenous results in terms of volumetric changes, but simultaneously found positive associations between depression and hippocampal volume, particularly in the right hemisphere. (Videbech & Ravnkilde, 2004). In contrast, one study of 27 individuals with major depression found hippocampal volume was not significantly different from the healthy control group in the study. However, results did indicate the shape of depressed individuals' hippocampi were significantly different from healthy subjects, indicating size as a potential structural deficit common to depression (Posener et al., 2003). Another study highlighted that stress (with emphasis on glucocorticoids and functional receptors) has detrimental impacts of

hippocampal functioning by inducing neuron death, dendritic regression, and inhibiting neurogenesis which may play a part in the etiology of depression (Lee, Ogle, & Sapolsky, 2002).

The amygdala is also an important structure in the brain that has previously been implicated in depression. The amygdala is integral in emotional processing, memory, threat and fear detection, and integrating sensory inputs (such as integrating visual and auditory stimuli) (Wright et al., 2015). Generally, neural plasticity in the amygdala (and other brain structures) is thought to be influenced by major depressive disorder and other types of depression in the form of atrophy and apoptosis. Similar to research on the hippocampus, volumetric changes in the amygdala have also been associated with depression in individuals (Zavorotnyy et al., 2018; see also Hastings et al., 2004). Specifically, grey matter volume in the amygdala was found to be reduced in individuals with prolonged unipolar depression as compared to individuals with late onset depression. Results of this study suggest that grey matter volume (i.e., neuronal density) shares a negative relationship with prolonged depression, indicating retraction of neuron density, and perhaps neuronal death (Zavorotnyy et al., 2018).

Amygdala responsivity to negative stimuli has also been studied in relation to depression. It has been previously noted that depressive emotional outputs can be measured in the amygdala since this particular structure shows electrical stimulation when subjects are exposed to negative stimuli. In depressed individuals, the amygdala appears to generate higher electrical activity in response to negative stimuli as compared to non-depressed individuals (Drevets, 1999 as cited in Hastings et al., 2004). For example, studies have observed greater sustained bilateral and left amygdala stimulation

in depressed individuals when confronted with negative stimuli, such as reactivity to negative words, as compared to healthy controls (Seigle et al., 2007). Ultimately, sustained over-stimulation and exaggerated amygdala responsivity "could be associated with increased emotional reactivity, as well as decreased function in brain regions subserving executive control and potentially initiating emotion regulation" (Seigle et al., 2007, p. 206; see also Deldin, Keller, Gergen, & Miller, 2000; Foland-Ross, Hamilton, Joormann, Berman, Jonides, & Gotlib, 2013). These results are consistent with other studies which implicate hyperstimulation of the amygdala in individuals with bipolar disorder (Whalen et al., 2002). Yet, other studies have indicated that while hyper-stimulatory responses to negative stimuli were common in the left amygdala of individuals with anxiety, depressed individuals showed inhibited or blunted responses corresponding to the right amygdala (Thomas et al., 2001, as cited in Whalen et al., 2002). While results do appear to be mixed, there is ample evidence suggesting structural and physiological components of the amygdala are pertinent in depression etiology.

Depression has also been associated with the prefrontal cortex (PFC) and its particularized subsections. Located in the anterior portion of the frontal lobe, this section of the brain is responsible for regulating and enacting executive cognitive functions, some of which pertain to reasoning and intellectual processing, planning and execution, and behavioral regulation and self-control (Jorgensen, Anderson, & Barnes, 2016). The PFC is further subdivided based on functional activity and specialized processing, typically being separated between the ventromedial (vmPFC) portion (inferior locale) and the dorsolateral (dlPFC) portions (superior locale) (Koenigs & Grafman, 2009). In particular, the dlPFC is responsible for both cognitive and executive processing, whereas

the vmPFC is implicated in emotional processing and affective behavior. Koenigs and Grafman (2009) note that imaging studies related to these particular areas and depression tend to share a relationship. For example, resting activity of the vmPFC is associated with negative affective behavior (which is pertinent in depression), and damage in the form of lesions has been associated with other depressive symptomology, including guilt, sadness, shame, regretful feelings, and other emotional substrates related to ruminative processing. In contrast, the dlPFC in its "maintenance and manipulation of items working memory, intention formation, goal-directed action, abstract reasoning, and attentional control" also share a relationship with emotional processing by regulating "negative emotion through reappraisal/suppression strategies" (p. 242). The implications of research on these processes generally indicate that disruption or impedance in reappraisal/suppression processing of the dlPFC shares a strong association with depressive behavior, simply because the suppression functions serve as a protective mechanism against depression (Koenigs & Grafman, 2009). Other research has pointed to many of the same results, with some suggesting that since the medial prefrontal cortex (MPFC) is central to emotional processing, discrepancies in this area (either in the form of lesions or other neurological and structural deficits) are a probable culprit in the etiology of serious depressive behavior (Drevets, Price, & Furey, 2008). As was the case for both the hippocampus and amygdala, volume attrition in the various areas of the PFC share some relation to depression. According to Drevets, Price, and Furey (2008), gray matter volume in the orbital and ventrolateral areas of the PFC is indicative of major depressive and bipolar disorders in certain individuals. Further, in individuals with bipolar disorder, gray matter reductions have been observed in the lateral orbital cortex, a

region that is closely associated with the MPFC (Drevets, Price, & Furey, 2008). Similar findings from other studies indicate general volumetric reductions in the PFC among depressed males but not in depressed females, and that certain areas of the PFC are generally reduced in size as compared with healthy controls (Hastings et al., 2004). Other researchers report that stress has negative impacts on the PFC, specifically by causing neuron death (apoptosis) and dendritic regression (McEwen, 2005). The research on specific brain structures and depression is quite extensive and highly complex. While not all brain structures that share a relationship with depression were discussed, readers should be cognizant that many other potentially influential structures likely share a role in depression.

Neurochemicals and Depression

Researchers have identified many different neurochemicals that share a relationship with depression. For example, consider the following statement regarding neurochemical influences in the etiology of depression:

> Of the neurochemical systems that modulate neural transmission within the visceromotor network, mood disorders have been associated with abnormalities of serotonergic, dopaminergic, noradrenergic, cholinergic, glutamatergic, GABA-ergic, glucocorticoid and peptidergic [e.g., corticotrophin releasing factor (CRF)] function. (Drevets, Price, & Furey, 2008, p. 102)

Of all the neurochemicals and biochemical systems studied, the three most common that are associated with depression are norepinephrine, dopamine, and serotonin (Blier, 2001). Although not mentioned above, monoamine oxidase (MAO) has been studied for its influence on depression. In fact, it was once theorized that MAO might share a relation with depressive behavior specifically through the regulation of norepinephrine. For example, Beck and Alford (2009) describe the catecholamine

hypothesis which postulates that MAO inhibits the free flow of norepinephrine neurochemicals (an important monoamine in synaptic activity), and that reduction influences depressive outcomes. However, subsequent findings detracted from this postulation, leading to mixed reviews of the hypothesis as well as the relationship directly between MAO and depression (Dubovsky & Buzan, 1999, as cited in Beck & Alford, 2009; see also Goddard, Ball, Martinez, Robinson, Yang, Russell, & Shekhar, 2010). Despite mixed opinions, the topic has more recently been assessed, and some findings point to influences of the MAOA promoter gene on depression. One study supporting this view found that major depressive disorder shared a significant relationship with a particular polymorphism of the MAOA promoter gene (i.e., MAOA-uVNTR) (Younger, Tsai, Hong, Chen, Chen, & Yang, 2005). It is more likely that MAO is indirectly related to depression via its regulatory functions of other neurochemicals (like norephinephrine) that do directly influence depression. Regardless, the research in this area is mixed but perhaps future research on the topic will generate more conclusive results.

Norepinephrine is a derivative of dopamine, which is why the two share similar chemical characteristics (Wright et al., 2015). This excitatory neurochemical functions to catalogue information into long term memory by aiding in the construction of new synapses. Norepinephrine is also central to the fight/flight response and is often activated when the sympathetic nervous system is engaged (Wright et al., 2015). Part of norepinephrine's functionality includes regulating vigilant behavior, as well as facilitating adaptive responses to internal and external stressors (Goddard et al., 2010). Because depression shares a relationship with anxiety and stress, and because stress and anxiety are greatly influenced by norepinephrine, anxiety-driven depression and

norepinephrine imbalances are considered to be correlated (Goddard et al., 2010). Other evidence leads to the conclusion that depressive pathology shares a relationship with norepinephrine anomalies. For example, Moret and Briley (2011) note that norepinephrine is supplied in the limbic system which is directly related to emotional regulation. Emotional dysregulation is a principle facet of depression. Further, differing levels of norepinephrine have consistently been documented in comparisons of healthy versus depressed individuals (Moret & Briley, 2011). Recall the structural deficits mentioned earlier that involve particular brain structures, such as the amygdala and hippocampus. These structures exist within the limbic system, one of the primary areas in which norepinephrine interacts with. As such, studies indicate abnormal norepinephrine activity within these structures as a causal mechanism in the pathological determinants of depression (Moret & Briley, 2011; see also Goddard et al., 2010).

Depression has also been consistently linked with abnormalities concerning dopamine. Dopamine is highly influential in mood regulation, and it is particularly associated with reward/punishment circuitry in the brain (Wright et al., 2015). Dopamine can be considered the pleasure chemical because as the dopaminergic system releases it, an elevation of mood, arousal, and pleasure often results. Dopamine also serves a functional purpose in multiple states of arousal, and has implications in the elevation of mood, problem solving, attention span and focus, and overall mental performance (Wright et al., 2015). Fluctuations in dopamine and other anomalous occurrences in the dopaminergic system share a relationship with depression. For example, studies have indicated abnormally low striated dopamine release among depressed patients (a striatum regulates uptake and release) (Ebert et al., 1996, as cited in Laasonen-Balk, Kuikka,

Viinamäki, Husso-Saastamoinen, Lehtonen, & Tiihonen, 1999). These results were reaffirmed by Nutt (2006) who found decreased striated dopamine release in particular dopaminergic receptor cites in individuals with major depressive disorder (as cited in Drevets et al., 2008). In lay terms, low dopamine levels share a relationship with depression. Furthermore, in a study of 15 depressed patients and 18 healthy controls, results indicated a significant reduction in dopamine transmission among depressed individuals compared to non-depressed individuals (Laasonen-Balk et al., 1999). The dopaminergic system has strong implications in the regulation of reward, arousal, and pleasure, and research indicates that dysregulation in dopaminergic pathways is often discovered among individuals with depression (Rampello, Nicoletti, & Nicoletti, 2000). As research has generally shown, depressive symptomology generally includes decreases in pleasure and arousal, inability to experience rewards, low positive affective behavior, poor emotional regulation, low motivation, and feelings described as feeling bland, empty, or blunted (Finan & Smith, 2013). Given that dopamine is directly involved in the regulation of these kinds of moods and feelings, it should come as no surprise that dopamine transmission is important in depressive etiology. In response to these kinds of findings, pharmacological measures are taken to develop antidepressants which directly target this pathway with the aim of diminishing depressive behavior (see Rampello et al., 2000 for further discussion of pharmacological developments).

The neurochemical serotonin has also been linked with depression, especially since it plays a functional role in mood regulation and anxiety levels. Low serotonin levels in the body are associated with a wide range of clinical disorders, depression included (Wright et al., 2015). Further, serotonergic pathways in the body and particular

promotor genes that code for serotonin have also been implicated in depressive symptomology. Recall from earlier that the *s* allele of 5-HTTLPR has been linked with depression and anxiety (Takahashi et al., 2011). In further support of this connection, a study by Caspi, Sugden, Moffitt, Taylor, Craig, & Harrington (2003) found that a particular polymorphism of the 5-HTT serotonin gene moderated stressful life events and depression. In particular, their results indicated that "childhood stress predicted adult depression only among individuals carrying an s allele" of that particular gene (Caspi, Sugden, Moffitt, Taylor, Craig, & Harrington, 2003, p. 388). Aside from genes, several brain structures in which serotonin receptor cites operate show evidence of being inhibited or decreased among individuals with major depressive disorder and other depressive pathologies, like bipolar depression (Drevets et al., 2008). Despite these discoveries, there appears to be a lack of consensus about whether serotonin or depression comes first; that is, does depression cause dysregulation in serotonin transmission, or does serotonin transmission cause depression, or is it both? (Drevets et al., 2008). Some researchers suggest that it is more likely that depressive outcomes are predicated on abnormalities within the specific genes that code for serotonin (Caspi et al., 2003). However, not all researchers agree. Arguments have been made that particular gene determinants related to serotonin remain largely unknown since no one has deciphered the exact mechanisms influencing hypersensitivity to serotonin in the pivotal brain structures linked with depression (Beck, 2008). While the three above mentioned neurochemicals appear to interact with depression, there are many other potential neurochemical pathways in depression. Although not discussed here, some of the other neurochemical substrates and systems associated with depression include Glutamatergic,

GABA-ergic, Glucocorticoid, Catecholaminergic, and Cholinergic systems (Drevets et al., 2008; see also Disner, Beevers, Haigh, & Beck, 2011).

Intersectionality of Aggression and Depression

Aggression and depression, in certain respects, seem to emanate from similar biological underpinnings and physiological processes. Many of the same neurochemicals and specific neurocircuitry systems in the human body regulate both aggression and depression in certain respects (Laasonen-Balk et al., 1999; Montoya et al., 2012). Further, it should be noted that many of the same structural components and areas of the human brain appear to stimulate, regulate, and control both aggression and depression (Blair, 2016: Lane et al., 2011; Lee et al., 2002; Zavorotnyy et al., 2018). Recall as well that many of the behavioral and personality disorders found to be associated with aggressive propensities are similarly associated with depressive propensities (Angold & Costello, 1993; Caspi et al., 1994; Englander, 2006; Levi et al., 2010; NIMH, 2018). Of course, the characteristics of each behavior, at least on their face, might also intuitively suggest that they are starkly opposed, with depression showing a multitude of internalizing destructive symptoms (NIMH, 2018; Quiggle, Garber, Panak, & Dodge, 1992) and aggression showing highly destructive external behaviors (Caspi et al., 1994; Englander, 2006; Steiner et al., 2011). Prior research has analyzed the relationship between them, often discovering intersectionality between these seemingly different behaviors. This is evidenced by the fact that aggression and depression co-occur in humans, especially among younger individuals (Messer & Gross, 1994; Muris, van der Pennen, Sigmond, & Mayer, 2008; Quiggle et al., 1992). Furthermore, there are serious impacts when aggression and depression are comorbid, such as the chronic persistence of more

depressive symptoms, social ineptness and impairment, interpersonal rejection, sociopathological dysfunction, suicidality, and increased substance abuse issues (Messer & Gross, 1994).

Beyond the reality that both aggression and depression can co-exist at the same time, aggressive individuals who also have depression are unique in that the distinctive patterns and informational processing schemes of each behavior not only occur at the same time but emerge in varying degrees. Moreover, the degree, level, and strength of the co-occurring patterns may be a function of which behavior came first. For example, Quiggle, Garber, Panak, and Dodge (1992) assessed aggression and depression levels in 220 individuals in 3rd through 6th grade. Three distinct groups were assessed: an aggressive group, a depressive group, and an aggressive-depressive group. Based on peer nominations and teacher ratings, results indicated that aggressive-only individuals were more likely to engage in further violent behavior without much obstruction. Depressed-only individuals also showed evidence of hostile attributional biases, yet the act of asserting hostility was more commonly internalized or directed towards very specific causes. Interestingly, individuals identified as comorbid showed the existence of both of these patterns of behavior simultaneously. The comorbid group was further split between individuals who showed slightly more aggressive propensities than depressive ones, and vice versa. The authors speculated the variation in comorbid aggression-depression levels are likely a function of temporal influence; that is, if aggressive behaviors were established and were then followed by depressive symptomology, then informational processing of specific events may show a slight tendency toward aggression mixed with

depression. The same concept applied to depression being the initial behavior, followed by aggression (Quiggle et al., 1992).

The harder questions for research to answer involves determining whether the relationship between aggression and depression are recursive (unidirectional) or non-recursive (bidirectional). It has been argued that low self-esteem, a common marker of depressive behavior, serves as a link in the causal chain leading to aggression (Baumeister, Bushman, & Campbell, 2000). However, evidence suggests that this may not be the case, considering that many aggressive actions, such as murder or rape, are often engaged in by individuals who actually hold themselves in high regards, or by people who have a strong sense of self or personal superiority. Another example can be found in the aggressive propensities of manic depressives. Baumeister, Bushman, and Campbell (2000) note that aggression among this group of individuals often occurs in the manic phase. This is counter-intuitive, considering that the manic phase encompasses high levels of self-esteem, positive sense of self, high energy, and positive outlook. As far as the old yet dominant view that low self-esteem can cause aggression, the validity of this claim seems to be contested among researchers (Baumeister, Bushman, & Campbell, 2000). Despite the uncertainty of low self-esteem causing aggression, some evidence does suggest that individuals suffering from Major Depressive Disorder (MDD) may be more prone to severe anger attacks. Winkler, Pjrek, and Kasper (2005) assessed 217 depressed subjects (roughly even between males and females) to determine whether anger attacks occurred subsequent to a major depressive episode. Results from the study found that men, more than women, showed significantly higher levels of impulsivity, irritability, overreaction, and expressive anger following their depressive episodes.

Further, men in the study engaged in higher rates of symptomatic substance abuse and hyperactive behaviors (Winkler, Pjrek, & Kasper, 2005). Other studies have found that certain depressive symptoms do precede and influence aggression at a later time, including "isolation, lost social support, increased alcohol use, angry rumination, and impulsivity" (Dutton & Karakanta, 2013, p. 310). It would appear that certain symptoms inherent in depression share some causal link with aggressive propensities, whereas other common depressive symptoms do not.

Research has also addressed the possibility of aggressive propensities stimulating the development of depression. Van Praag (2001) suggests that anxiety-driven aggression disrupts the proper regulation of mood by lowering stability which results in mood and behavioral dysregulation in the form of depressive symptoms. As was previously mentioned, aggression is a common characteristic of different mood and personality disorders, and thus it is not uncommon to see aggressive behavior intermingling with other dysfunctional behaviors, like depression, which are also components of mood and personality disorders (Angold & Costello, 1993; Caspi et al., 1994; Englander, 2006; Levi et al., 2010; NIMH, 2018). According to one researcher, specific types of aggressive dysregulation, including irritability, impulsivity, and angry outbursts may pace the development and persistence of subsequent depressive episodes (Van Praag, 2001). Impulsivity is well known to be a function of low self-control (Walters, 2018), and research has established both in the etiological development of aggression (Lickley & Sebastian, 2018; Steiner et al., 2011). Similar to low self-control, Muris, van der Pennen, Sigmond, and Mayer (2008) discussed the nature of effortful control, which is the ability to regulate or control one's behavior given particular experiences. Low effortful control

is indicative of dysregulated responsivity in the face of trying circumstances, and the responses may manifest themselves as highly aggressive or depressive depending on circumstance and individual ability to control behavioral responses. Other factors thought to generate the occurrence of aggression before depression point toward social influences, such as negative peer relationships, social rejection, school failure, independent social stressors, and familial disruption (Little & Garber, 2004; Messer & Gross, 1994; Sigfusdottir, Farkas, & Silver, 2004; Sijtsema, Oldehinkel, Veenstra, Verhulst, & Ormel, 2014). However, it should not be assumed that these mechanisms always cause aggression before depression since they also seem to increase levels of depression and aggression absent the existence of the other.

Important findings in the development and persistence of depression and aggression have strong ties with personal sense of achievement, rejection, and peer networks. Centering on perceived achievement, Little and Garber (2004) studied 129 subjects transitioning into the 9th grade (high school entry) to determine the impact of peer relations and achievements on aggressive and depressive outcomes. The authors outlined the personality-event congruence hypothesis, which holds that individuals who direct a high level of investment toward a particular set of goals or goal are particularly susceptible to depression and aggression following the failure to achieve desired ends. By measuring individual's achievement orientation and the influence of academic stressors, the authors discovered that among girls with high achievement orientations, academic stressors dually increased aggression and depression levels. Boys, regardless of achievement orientation, showed higher rates of aggressive propensities in response to academic stressors (Little & Garber, 2004). These findings suggest that a normative

response to failure likely includes some level of frustration over not achieving one's goals. However, the degree of frustration seems to revolve around the interpretation and processing of personal expectations which suffer in the face of failure to achieve desired objectives. For example, emotional flooding may occur in a similar situation where individual affective and cognitive appraisals that normally mediate certain experiences, such as personal "encodings, expectancies, values, and competencies," are overcome by low goal attainment (Dutton & Karakanta, 2013, p. 315). This phenomenon impinges on the cognitive framework of achievement orientation, influencing the development of aggressive and depressive responses in turn.

Another pivotal component where aggression and depression intersect concerns rejection. Human beings are by their nature highly gregarious, enjoying and benefiting much from sociality and networking with others. The well-being of individuals is often predicated on what is garnered from their social group, ranging anywhere from the accrual of tangible resources, personal and social stability, survival, cognitive development, and to a large extent, an individual's evaluation of their own personal sense of self-worth and identity. Thus, research has identified connections between rejection and depressive/aggressive outcomes, especially among youth. In a study of 521 3rd, 4th, and 5th graders, Panak and Garber (1992) discovered that aggression levels increased as depression increased, and that both of these outcomes were partially mediated by peer rejection. The authors note that perception of self, especially among children, is in part constructed by an individual's perception of what other people think about them. If high valuation is placed on the opinions of those apart from the individual and the others subsequently reject the individual, this is devastating to identity and personal sense of

worth. As such, depressive and aggressive responses among children are thought to be common outcomes when a situation like this occurs (Panak & Garber, 1992). This unfortunate predicament appears to degrade interpersonal relationships, such that the resulting aggression and depression inhibits the subsequent development of healthy peer relations. Brendgen, Vitaro, Turgeon, and Poulin (2002) found that among 4th, 5th, and 6th graders, the co-occurrence of depression and aggression negatively impacted peer relationships, regardless of peer perceptions of the individual. The implication lurking here is that a highly detrimental feedback loop seems to be created wherein a) personal valuation of self-worth is damaged by peer rejection which b) generates depressive/aggressive outcomes which c) then impedes further development of peer networks that would otherwise provide some level of cognitive and social support for identity and self-worth (Brendgen, Vitaro, Turgeon, & Poulin, 2002).

A multitude of cognitive elements and social circumstances could conceivably instigate the formulation and persistence of both aggression and depression. Researchers have suggested that they share a unique relationship with each other, often arising from a series of similar configurations both internal and external to the individual. Another pertinent factor imbued in in the etiology of these maladaptive behaviors revolves around individual physiological age.

Aggression, Depression, and Age

The age of onset of aggression, the stability of aggression across the life-course and across generations, heritability, and a multitude of predictors of aggression across age have all been extensively researched (Hart, Nelson, Robinson, Olsen, & McNeilly-Choque, 1998; Huesmann, Eron, Lefkowitz, & Walder, 1984; Eron, 1987; Tremblay,

Japel, Perusse, McDuff, Boivin, & Zoccolillo, 1999). In terms of age of onset of aggression, researchers suggest there are differences between specific types of aggression, including verbal, physical, relational, and other forms of aggression (Tremblay et al., 1999). According to Tremblay and colleagues (1999), physical aggression onset appeared at roughly a year old. Their study assessed interview information gathered from mothers of 511 children (approximately even between boys and girls). Based on these reports, results suggested that aggression increased rapidly in children between the ages of 12 to 17 months. Specifically, the most common aggressive behaviors at 17 months old were taking items away from another individual (70.4%) and pushing or shoving to get something one wants (46%). These rates were greater among children with siblings. Further, percentages of aggressive behaviors were slightly higher among boys than for girls, but differences were so slight as to be negligent. Other common aggressive behaviors, including biting, kicking, fighting, threatening to strike, physically attacking, and a few others mostly showed low percentage occurrences roughly between 24% and 5% (Tremblay et al., 1999).

Another study assessing different levels of physically aggressive behavior (ranging from low to high and whether aggression was persistent or decreasing) analyzed a sample of children between 24 months and 9 years old from the NICHD Study of Early Child Care and Youth Development. Results from this study indicated that individuals with the highest level of aggressive propensities had serious social and conduct problems by the age of 12 (Campbell, Spieker, Burchinal, Poe, & NICHD Early Child Care Research Network, 2006). Findings showed that in cases where children had initially low levels of aggression, but the low aggression remained persistent, this predicted later

conduct and performance issues at a later age. In contrast, it was discovered that individuals who had initially moderate levels aggression but who showed decreases and eventual dissipation of aggression over time were more likely to exhibit proper social and conduct adjustment by the age of 12 (Campbell et al., 2006).

Some studies suggest that there is an early childhood aggression curve. For instance, Alink, Mesman, Van Zeijl, Stolk, Juffer, and Koot (2006) analyzed a sample of 2,253 children between 10 and 50 months old from the Dutch SCRIPT study. Aggressiveness in children was measured via reports on the children given by their mothers and fathers. Overall, findings indicated aggressive behavior was present in individuals who were a year old, but it was noted that aggression dramatically increased at two and three years of age. Further, aggression at one year old remained moderately stable, whereas aggression at two and three years old showed relatively high stability. Not surprisingly, aggression was found to be higher among males (Alink, Mesman, Van Zeijl, Stolk, Juffer, & Koot, 2006). Collectively, these studies appear to suggest relatively early onset and development of initial aggressive behavior among infants between one to two years old (see also Tremblay, Nagin, Séguin, Zoccolillo, Zelazo, & Boivin, 2004, for further studies on infant and early childhood onset of aggression).

Other studies have assessed aggression in association with temporal stability and generational persistence. In a longitudinal study of 600 individuals spanning 22 years, Huesmann, Eron, Lefkowitz, and Walder (1984) found stability of aggression in participants over time, and stability strongly predicted later behavioral issues and criminal offending. Other findings indicated a higher prevalence of aggressive behavior among males. The authors also discovered that stability of aggression across the life-

course shared a strong relationship with mental acumen and intelligence (Huesmann, Eron, Lefkowitz, & Walder, 1984; see also Loeber, Menting, Lynam, Moffitt, Stouthamer-Loeber, Stallings, & Pardini, 2012). Whatever the underlying causes are, the conclusion drawn by these researchers was that aggression, especially when it begins at an early age, is a highly persistent yet variable trait among certain individuals and strongly predicts subsequent socially maladaptive behavior across the life-course (Huesmann et al., 1984).

Several other studies focusing on aggression have analyzed a myriad of different predictors of aggressive behavior according to age. For example, low SES and social disadvantage have been linked with aggressive propensities among adolescents between the ages of 12 and 25 years of age (Fabio, Tu, Loeber, & Cohen, 2011). Results from this study found curvilinear differences between serious violent behavior and four different levels of social disadvantage among adolescents and young adults. Specifically, individuals with the highest levels of social disadvantage who also lived in public housing showed the highest aggression levels, followed by those with high disadvantage but who did not live in public housing. The two lower curvilinear projections of the age-crime curve were among those with average levels of social disadvantage and those were considered advantaged. Each trend among the four groups showed a visible increase between the ages of 12 and 19, and all generally declined thereafter (Fabio et al., 2011, fig. 1). Other predictors of aggression as it pertains to age include parenting, maternal care, and marital status (Hart et al., 1998; Nagin & Tremblay, 2001), cognitive impulsivity and intelligence levels (Loeber et al., 2012), gender in relation to adolescent conflict and withdrawal behavior (Lindeman, Harakka, & Keltikangas-Järvinen, 1997),

absence of prosocial behavior (Eron & Huesmann, 1984), and even low resting heart rate as a predictor of persistent aggression across the life-course (Raine, Venables, & Mednick, 1997).

Age of onset for depression is much more erratic than aggression, with plenty of evidence suggesting high variability in terms of age and type of depression. Some research has indicated discrepancies in reporting measures used to gauge age of onset, resulting in different results depending on the particular measure used (Knäuper, Cannell, Schwarz, Bruce, & Kessler, 1999). Furthermore, some kinds of depression are exclusively within the domain of adult-aged individuals, such as postpartum depression which typically affects women after childbirth (NIMH, 2018). Researchers have also focused on depression onset among varying age groups. Some researchers focus on individuals starting at age 18 and above (Husain, Rush, Sackeim, Wisniewski, McClintock, & Craven, 2005), others have focused on birth cohorts that span several decades (Kessler, Birnbaum, Bromet, Hwang, Sampson, & Shahly, 2010), and still others have focused solely on children and adolescents (Luby, Belden, Sullivan, Hayen, McCadney, & Spitznagel, 2009). Onset of depression, then, can be assumed to be a function not confined to any particular age as is evidenced by the fact that it occurs in just about every age group and manifests in different ways depending on age.

Some researchers note that age is quite complex because different age-related factors, such as “maturity, decline, life-cycle, survival, and historical trend” independently influence depressive outcomes among different age groups (Mirowsky & Ross, 1992, p. 187). For example, research that analyzed five distinct age cohorts (ages ranging from 18 to 75) found distinct differences in depressive outcomes between cohorts

(Husain et al., 2005). Results showed that the 51-65 age group had the highest number of recorded major depressive episodes, followed closely by the 36-50 age group. Interestingly, the youngest age bracket (18-25) reported the youngest onset age for major depressive episodes (15.9 years old) whereas the oldest age bracket (66-75) reported much older age onsets (44.3 years old) which is indicative of late onset depression (Husain et al., 2005; see also Kessler et al., 2010). Other studies on the manifest differences between early onset depression and late onset depression have also found intriguing insights. According to one study, early onset depression, as compared to late onset depression, significantly correlated with "longer duration of symptoms, a personal history of depressive episodes, a serious suicide attempt, childhood events, a family history of depression, and high neuroticism" (Korten, Comijs, Lamers, & Penninx, 2012, p. 259). Further, feelings of sadness, reduced concentration, and suicidal ideation were more common in individuals with early onset depression. In contrast, late onset depression was found to be significantly correlated with weight loss, decreased appetite, less suicide ideation, and less issues with concentration (Korten et al., 2012). Aside from these findings, researchers have also explored the possibility of early onset of depression in much younger children.

Luby, Belden, Sullivan, Hayen, McCadney, and Spitznagel (2009) explored the prevalence of shame and guilt pertaining to depressive behavior among preschool aged individuals. The authors noted that prior studies had indicated shame and guilt were present in individuals around that age, and that major depressive disorder can potentially begin around three years old. However, they suggest studies have not clearly assessed complex emotions among children, including guilt, shame, and embarrassment and their

potential relationships to or influences on childhood depression. The authors' study consisted of 305 preschool children, with ages approximately between 3 and 5 years old. Four distinct groups of children were created based on their different diagnoses (MDD, disruptive, anxiety, and control). Results indicated that older age and higher depression levels independently affect shame variance. Interestingly, findings also showed that female preschoolers were more apt to use guilt themes than were male preschoolers, and they used shame themes at a higher rate than did males. The themes were a product of testing based on the MacArthur Story Stem Battery (MSSB) which is a story line started by the researcher and then children complete the story using props as they choose. Furthermore, it was discovered that the use of shame themes increased as age increased. Lastly, children diagnosed with major depressive disorder had significantly higher guilt scores than any other group (Luby et al., 2009). These findings are quite informative regarding the onset of depression in children, especially since guilt and shame have been well documented in other depressed populations.

Longitudinal cohort studies have also been conducted on childhood depression. For example, Nolen-Hoeksema, Girgus, and Seligman (1992) discovered that early negative life events significantly influence the onset of childhood depression. Their study focused on a 5-year longitudinal study of 508 third grade children, which ultimately indicated negative life events significantly predicted depressive outcomes among the younger children. However, negative life outcomes impacted depression differently as the child aged. Specifically, it was more common for older children to develop pessimistic explanatory styles which strongly predicted depressive behavior in combination with negative life events. Further, continued significance of negative life events was found in

older children experiencing depression (Nolen-Hoeksema, Girgus, & Seligman, 1992). Subsequent to this study, Twenge and Nolen-Hoeksema (2002) conducted a meta-analysis on 310 different sample populations of depressed children and adolescents ranging from 8 to 16 years old. Findings indicated Child Depression Inventory (CDI) scores among females were generally higher, more stable, and more persistent than males as age increased. Exceptions to these findings included female depression scores being lower than males prior to age 13, but then surpassed males after this age. Male depression scores were also found to be stable, with slight spikes in CDI scores around age 12 (Twenge and Nolen-Hoeksema, 2002). The finding that females typically have higher rates of depression has been consistently documented in other research which indicates clear gender differences in depressive etiology (see generally Brodaty, Cullen, Thompson, Mitchell, Parker, & Wilhelm, 2005; Nolen-Hoeksema, Larson, & Grayson, 1999).

Furthermore, Moffitt, Harrington, Caspi, Kim-Cohen, Goldberg, and Gregory (2007) followed 1,037 individuals from birth to age 32 to assess anxiety and depression development. Participants were routinely checked in on every few years and measures were taken regarding potential abnormal behaviors, disorders, depression, anxiety, among other things. Results of the analysis showed that females had consistently higher occurrences of MDD than men, specifically between the ages of 15 and 32. It was noted that occurrences of MDD were relatively low in both male and female participants in their early adolescent years, but occurrences increased sharply as the age of participants entered early adulthood. Further, by age 32, two-thirds of women had recurrent cases of MDD, versus half of the male participants (Moffitt, Harrington, Caspi, Kim-Cohen,

Goldberg, & Gregory, 2007). Overall, research on age of onset and depression is highly variable, with research finding depressive behavioral onset in very young children, prepubescent individuals, young adolescents and teens, young adults, and individuals anywhere between 30 and 70 years old.

While prior research has indicated a co-occurrence of and shared relationship between aggression and depression according to neurochemicals (Laasonen-Balk et al., 1999; Montoya et al., 2012), brain structures (Blair, 2016: Lane et al., 2011; Lee et al., 2002; Zavorotnyy et al., 2018), and differing personality and behavior disorders (Angold & Costello, 1993; Caspi et al., 1994; Englander, 2006; Levi et al., 2010; NIMH, 2018), research focusing on the relationship between aggression and depression and how it changes across the individual life-course seems less clear. Particularly, longitudinal studies that directly focus on the relationship between aggression and depression and how this relationship behaves over time are apparently few in number. Although longitudinal studies have addressed the characteristics of depression over time (Moffitt et al., 2007) and aggression over time (Huesmann et al., 1984), there is a gap in the research that combines them to assess changes over time in the relationship between aggression and depression. As such, it is currently unknown whether there is a stable or dynamic relationship between aggression and depression over time. Based on the literature discussed above, this thesis generally sought to investigate the relationship between aggression and depression over time and the potential moderating effects of age and biological sex.

CHAPTER THREE: METHODOLOGY

It is apparent from the literature on both aggression and depression that different frequencies pertaining to aggression levels and depression levels vary according to age, and that these frequencies fluctuate both up and down as age increases. Based on this, the following hypothesis is central to this study:

H_1: The relationship between aggression and depression will change over time.

Literature on aggression and depression also suggests that differences in the occurrences of aggression and depression vary according to biological sex. Specifically, most research indicates males exhibit much higher frequencies of aggressive behavior than do females. Conversely, some literature on depression suggests that females exhibit higher frequencies of depressive behavior than do males. Based on these general stipulations, the following hypothesis will also be tested:

H_2: Biological sex will moderate the relationship between aggression and depression.

Research Design

This study is a longitudinal cohort study using data gathered from The National Longitudinal Study of Adolescent to Adult Health (Add Health) which is a nationally representative study collecting data across multiple waves. The four waves of data analyzed here are specific to aggression and depression measures, as well as other demographic measures. As such, the unit of analysis for this study is the individual. While Add Health includes a multiplicity of variables and measures, only variables that

pertained to aggression, depression, and demographics were assessed in this study. Further, only the aggression and depression survey items that were present and consistently measured across all waves were included. The reason for excluding other arguably pertinent measures of aggression and depression hinges on the fact that some measures occurred in three of the four waves or within single waves only. Because this study is longitudinal, pertinent survey items had to be present in all waves for the purposes of consistency in the analysis.

Sample

The sample consists of four waves of data from The National Longitudinal Study of Adolescent to Adult Health (Add Health). Wave I (N = 6,504) represents data collected from individuals between 7th and 12th grade between the years 1994 and 1995. Wave II (N = 4,834) represents interview data collected from individuals between the months of April and August in the year 1996. Wave III (N = 4,882) consists of interview data collected on individuals between 18 and 26 years of age in the months between August 2001 and April 2002. Wave IV (N = 5,114) contains data collected from individuals when they were between the ages of 24 and 32 in the year 2008. Demographic variables on sample participants were collected for all waves, including age, gender, and race. Specific measures of aggression and depression were included in this analysis with the caveat that only those survey items that were present and consistently measured in all four waves were included. Several possible, and likely pertinent, measures of aggression and depression were excluded from this analysis due to inconsistent measurement across the four waves. For more information on the specifics of

Add Health's research design and sampling procedures, please visit the following web address; https://www.cpc.unc.edu/projects/addhealth/design/wave.

Demographic Measures

Demographic variables include age, gender, and the reported race/ethnicity of respondents which represent both ratio and nominal levels of measurement. To determine the age of respondents at each wave, respondents' birth years were subtracted from the year(s) in which the interview took place at each wave. For example, this calculation produced respondent ages ranging from 12 to 21 in Wave I. Because ages 12, 20, and 21 were outliers, those observations in Wave I were dropped from further analysis leaving respondents aged 13 through 19 in the sample (N = 6,431). By dropping outliers and truncating Wave I ages, this automatically truncated the age ranges in Waves II, III, and IV. Wave II respondent ages ranged from 14 to 20 (N = 4,807). Wave III respondent ages ranged from 19 to 26 (N = 4,833), and Wave IV respondent ages ranged from 25 to 33 (N = 5,067). Race/ethnicity included response options of White (N = 4,265), Black/African American (N = 1,595), Hispanic (733), American Indian/Native American (N = 234), Asian/Pacific Islander (N = 263), and other (N = 416). All race/ethnicity variables were dichotomized (1 = Yes, 0 = No) which produced the number of respondents who identified as a particular race in each race variable. Respondent gender concerned reported biological sex as male or female and was dichotomized (1 = Male, 2 = Female). At Wave I, 51.75% of respondents were female, 48.25% were male. At Wave II, 52.13% of respondents were female, 47.87% were male. At Wave III, 53.9% of respondents were female, 46.1% were male. At Wave IV, 54.1% of respondents were female, 45.9% were male. Descriptive statistics of each wave are provided in Table 3.1.

Table 3.1 Descriptive Statistics of Respondents' Race/Ethnicity and Gender per Wave

Wave	N	Age	Male	Female	White	Black	Hisp.	AI/ NA	A/PI	Other
I	6,431	13-19	48.2%	51.7%	66.3%	24.8%	11.3%	3.6%	4%	6.4%
II	4,807	14-20	47.8%	52.1%	67.6%	23.7%	11.6%	3.8%	3.9%	6.2%
III	4,833	19-26	46%	53.9%	66.8%	24.5%	10.4%	3.8%	4.2%	6%
IV	5,067	25-33	45.9%	54%	67.7%	24.3%	10.3%	3.5%	3.5%	6%

Note: Hisp. = Hispanic; AI/NA = American Indian/Native American; A/PI = Asian/Pacific Islander.

Measures of Aggression

Measures of aggression consisted of five distinct questions regarding respondents' reported actions that fell in the range of aggressive/violent behavior. Questions included the following: "How often in the past 12 months did you use or threaten to use a weapon to get something from someone?"; "Take part in a fight where a group of your friends was against another group?"; "Deliberately damage property that didn't belong to you?". These first three aggression measures were measured ordinally and coded in the same direction (0 = never, 1 = one to two times, 2 = three to four times, and 3 = five or more times). Two other aggression measures asked respondents: "How often in the past 12 months did you pull a knife or gun on someone?" and "Shoot or stab someone?". These two aggression measures were dichotomized (0 = no, 1 = yes). Although both of these questions were originally scaled and coded with more response options, Waves III and IV in Add Health deviated from the original scaling method and changed them into dichotomized questions. Thus, these last two questions were dichotomized for measurement consistency across all four waves. Further, wording of these questions were altered to match the new dichotomized format by removing "How often" from the

beginning of the questions. In total, these five aggression measures were used to create an aggression latent factor. Appendix A contains the individual aggression measures and their respective response options as used in this thesis.

Measures of Depression

Measures of depression consisted of 10 distinct questions regarding respondents' reported actions that fell in the range of depressive behavior. Questions included the following: "How often in the past week did you feel sad?"; "Feel that people disliked you?"; "Feel that you could not stop feeling blue, even with the help of family and friends?"; "Feel too tired to do anything?"; "Have trouble focusing your mind on what you were doing?"; "Feel bothered by things which normally are not bothersome to you?"; and "Feel depressed?" These first seven depression measures were all coded and scaled in the same direction (0 = never/rarely, 1 = sometimes, 2 = a lot of the time, and 3 = most/all the time). Two more depression measures asked respondents "How often in the past week did you feel that you were just as good as others?" and "How often in the past week did you enjoy life?". These two measures were reverse coded to match the scaled direction of the previous seven measures (0 = most/all the time, 1 = a lot of the time, 2 = sometimes, and 3 = never/rarely). The last depression measure asked respondents the following: "In the past 12 months did you ever seriously contemplate suicide?". This measure was dichotomized (0 = No, 1 = Yes). Similar to the aggression measures, the 10 depression items were used to create a depression latent factor. Each question measuring depression used an ordinal level of measurement, except for the question asking respondents about suicide contemplation which is a nominal level of measurement.

Appendix A contains the individual depression measures and their respective response options as used in this thesis.

Analytical Strategy

First, the data were separated into nine distinct groups by stratifying the data according to three separate age intervals, as well as by gender. Specifically, the first group incorporated all respondents both male and female ages 13 through 19. This group contains all observations in the data after dropping outlying observations and was then stratified by gender, resulting in a group for 13 through 19 year old females, and a group for 13 through 19 year old males. The same process was applied to stratify the data by age group. Respondents with ages 13 through 15, and 16 through 19 were split into respective groups and then these groups were each stratified by gender as well. To test the hypotheses in this thesis, factor analysis, correlation analysis, equality of coefficients tests, and multiplicative interaction regression models were employed. Factor analysis is a data reduction tool that is generally designed to discern overall patterns expressed by a multitude of variables. This method is clearly superior to conducting a series of single correlations to determine the same patterns (see generally Babbie, 2016, p. 474), and thus represents an appropriate statistical technique to see if the relationship between aggression and depression changes over time. In factor analysis, several survey items are loaded onto an archetypal construct to determine whether those measures can adequately define the construct. In this case, the 10 measures of depression and the five measures of aggression were loaded on to their respective archetypal constructs to determine whether they were reflective of depression and aggression. Next, post-estimation diagnostics revealed which factor loadings should be accepted or rejected. All factor loadings, both

for the five aggression measures and the 10 depression measures, successfully loaded onto their respective constructs with factor loadings above .3, and thus none were rejected. Furthermore, all Eigenvalues were found to be acceptable and above 1. Eigenvalues for all aggression and depression latent constructs can be found in Tables 3.2, 3.3, and 3.4. Latent constructs for both the aggression and depression measures were created using factor scores for each construct.

Subsequent to this process, correlation coefficients between the aggression and depression factors were assessed consecutively across the four waves of data in question. Equality of coefficients tests were employed to test for any statistically significant changes in the correlations across waves. Proportional changes in correlations between each wave and the subsequent wave following it were then calculated. For example, the correlation between aggression and depression latent constructs for wave I was compared with the correlation of the latent constructs from Wave II and so on. These calculations ultimately determined whether any significant proportional changes in the correlation statistics between the aggression factor variable and depression factor variable were present across all waves for all groups. Results of the correlation analysis, equality of coefficients tests, and proportional changes in correlations can be found in Tables 4.1, 4.2, and 4.3.

The results from the correlational analysis prompted further exploration of the moderating effects of age and gender, and thus multiplicative interaction terms were used to determine whether age and/or gender was moderating the relationship between aggression and depression. Interaction terms are useful in determining the moderating effect of particular independent variables on dependent variables. In this case, both age

and biological sex were multiplied with the depression and aggression latent constructs (when tested as independent variables) independently to create the interaction terms. OLS regression was used to assess the interaction effects. The justification for using interaction terms in this case resulted from what was observed in the correlation analysis. The OLS model also serves as a robustness check of those findings. Essentially, the first part of the analysis indicated significant proportional changes in the relationship between aggression and depression over time for females only. To further understand this relationship, multiplicative interaction models using OLS regression were an appropriate statistical approach to address whether age, biological sex, or both were moderating the relationship between aggression and depression. Results from the multiplicative interaction models are reported in Tables 4.4, 4.5, 4.6, and 4.7.

Reliability and Validity

While both depression and aggression measures chosen for this analysis are theoretically assumed to be reliable and valid measures of aggressive and depressive behavioral constructs, statistical analyses assessing the reliability and validity of these measures were required. To test the scale reliability of the aggression and depression measures, Cronbach's alpha was used prior to conducting factor analysis and regression of the latent depression and aggression constructs. Generally, alpha scores for the depression measures fell between good and acceptable ranges of scale reliability. In contrast, alpha scores for the aggression measures ranged from questionable reliability to poor reliability to unacceptable reliability. For example, several of the alpha scores for the aggression measures were below .5 which is considered unacceptable. Most of the alpha scores were between .6 and .5, indicating poor scale reliability. Only a few were

between .7 and .6, indicating questionable scale reliability. Alpha scores of scale reliability for all aggression and depression latent constructs can be found in Tables 3.2, 3.3, and 3.4. After the depression and aggression measures were factored and loaded onto their respective constructs, Kaiser-Meyer-Olkin's (KMO) test of sampling adequacy was used to assess each depression and aggression measure Overall, KMO statistics for the depression latent constructs were meritorious (i.e., between .80 and .89). In contrast, KMO statistics for the aggression latent constructs ranged from unacceptable (i.e., between .00 and .49) to middling (i.e., between .70 and .79). KMO statistics for all aggression and depression latent constructs can be found in Tables 3.2, 3.3, and 3.4.

Table 3.2 Assessments of Reliability and Validity Ages 13-19

Wave	All Respondents 13-19					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=6,363)	.581	.790	.751	.882	2.262	3.704
WII (N=4,759)	.595	.800	.743	.890	2.270	3.838
WIII (N=4,679)	.463	.803	.623	.876	1.832	3.844
WIV (N=5,020)	.548	.807	.574	.885	1.916	3.895

Wave	Males Ages 13-19					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=3,062)	.609	.742	.755	.859	2.332	3.320
WII (N=2,269)	.609	.748	.749	.861	2.307	3.445
WIII (N=2,145)	.456	.756	.605	.845	1.836	3.415
WIV (N=2,288)	.555	.780	.595	.870	1.951	3.618

Wave	Females Ages 13-19					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=3,301)	.478	.812	.713	.889	1.991	3.917
WII (N=2,490)	.539	.822	.704	.898	2.106	4.019
WIII (N=2,534)	.424	.827	.625	.888	1.730	4.115
WIV (N=2,732)	.496	.822	.496	.887	1.770	4.065

Note: KMO = Kaiser-Meyer-Olkin test for sampling adequacy.

Table 3.3 Assessments of Reliability and Validity Ages 13-15

Wave	All Respondents 13-15					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=2,534)	.571	.787	.741	.879	2.271	3.687
WII (N=2,247)	.560	.801	.731	.895	2.173	3.898
WIII (N=1,982)	.445	.799	.607	.869	1.790	3.769
WIV (N=2,052)	.524	.807	.573	.881	1.945	3.883

Wave	Males Ages 13-15					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=1,166)	.616	.715	.751	.835	2.380	3.064
WII (N=1,024)	.564	.739	.730	.869	2.192	3.478
WIII (N=858)	.421	.726	.588	.800	1.798	3.151
WIV (N=876)	.526	.776	.592	.869	1.983	3.604

Wave	Females Ages 13-15					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=1,368)	.473	.815	.701	.888	2.046	3.987
WII (N=1,223)	.542	.821	.707	.897	2.114	4.037
WIII (N=1,124)	.439	.829	.587	.889	1.713	4.108
WIV (N=1,176)	.484	.822	.508	.880	1.805	4.035

Note: KMO = Kaiser-Meyer-Olkin test for sampling adequacy.

Table 3.4 Assessments of Reliability and Validity Ages 16-19

Wave	All Respondents 16-19					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=3,829)	.588	.788	.753	.880	2.266	3.686
WII (N=2,512)	.629	.799	.752	.881	2.365	3.779
WIII (N=2,697)	.480	.806	.625	.879	1.879	3.901
WIV (N=2,968)	.569	.807	.575	.883	1.906	3.904

Wave	Males Ages 16-19					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=1,896)	.604	.751	.750	.858	2.320	3.407
WII (N=1,245)	.644	.751	.759	.844	2.398	3.392
WIII (N=1,287)	.485	.773	.614	.864	1.894	3.604
WIV (N=1,412)	.579	.783	.594	.865	1.947	3.632

Wave	Females Ages 16-19					
	Cronbach's α		KMO		Eigen Value	
	Aggression	Depression	Aggression	Depression	Aggression	Depression
WI (N=1,933)	.481	.809	.720	.885	1.956	3.854
WII (N=1,267)	.529	.823	.709	.896	2.141	4.005
WIII (N=1,410)	.405	.825	.634	.877	1.813	4.117
WIV (N=1,556)	.507	.822	.496	.888	1.739	4.091

Note: KMO = Kaiser-Meyer-Olkin test for sampling adequacy.

It was also discovered from summary statistics and histograms of the aggression constructs that they were positively skewed. Normality of a distribution is an assumption of the GLM, and the distributions of the aggression latent constructs were skewed and violated that assumption. Based on the skewed nature of the distributions, low alpha

scores, and low KMO statistics, several other measurement models were considered in an attempt to normalize the distributions, increase scale reliability, and increase sampling adequacy, especially among the aggression latent constructs. The “gladder” command in STATA was used to check for different transformations of the data with the objective of normalizing the skewed distributions. STATA initially recommended taking the natural log, but this transformative approach did not normalize the distributions. Factor solutions were also rotated both orthogonally and obliquely. This approach, too, had no impact on measurement statistics. Next, of the five aggression measures, one was eliminated at a time and the remaining four were subjected to alpha tests, factor analysis, and KMO statistics. Every permutation produced lower alpha and KMO statistics. Last, additive scales were created to determine if more normalized latent constructs would emerge. Unfortunately, the distributions of the additive scales were not normalized. Ultimately, the measurement strategy used throughout this paper had shown to be the best possible measurement approach given the nature of the data.

CHAPTER FOUR: RESULTS

Results from the correlation analysis of the aggression and depression latent constructs for all nine groups of data generally indicated weak or negligible correlations within waves. For example, in Table 4.1, the correlation value between the aggression and depression latent constructs in Wave I for the entire sample was .167, indicating a weak relationship. The strongest correlation between the aggression and depression latent constructs occurred for females in Wave I, but still represented a weak correlation (r = .219). From the correlation values listed in Table 4.1, it can be seen that there is a weak relationship between aggression and depression per wave. Table 4.1 also reports that the correlation coefficients for all respondents steadily decreased across waves. This indicates a weakening of the relationship between aggression and depression over time. The same result was found for males' ages 13 to 19, with correlations showing a steady decline across all four waves, and thus a weakening relationship between aggression and depression over time. For females ages 13 to 19, declines in correlation coefficients were observed between Waves I and II, as well as between Waves III and IV. However, an increase in correlation was observed between Waves II and III. Equality of coefficients tests were used to determine if the changes in correlation between waves were statistically significant. Results showed that for all respondents combined and males only, the differences in correlations were not significant between waves successively. However, for females ages 13-19, z-statistics suggested the observed differences in correlation between Waves I and II, as well as between Waves III and IV were

statistically significant. Additionally, the proportional change in those correlations suggested that they are substantively significant as well. These results are reported in Table 4.1. Figure 4.1 and 4.2 provide a visual depiction of the changes in the correlation between aggression and depression over time for these groups.

Table 4.1 Correlations, Z statistics, and Proportional Change in the Relationship Between Aggression and Depression over Time Ages 13-19

	All Respondents 13-19		
Wave	r	*z*	Δ
WI (N=6,346)	.167	-	-
WII (N=4,741)	.136	1.630	-
WIII (N=4,640)	.133	.131	-
WIV (N=4,567)	.113	.980	-
Wave	**Males Ages 13-19**		
	r	*z*	Δ
WI (N=3,053)	.206	-	-
WII (N=2,255)	.205	.051	-
WIII (N=2,128)	.164	1.383	-
WIV (N=2,124)	.151	.423	-
Wave	**Females Ages 13-19**		
	r	*z*	Δ
WI (N=3,293)	.219	-	-
WII (N=2,486)	.151	2.624***	30.8%
WIII (N=2,512)	.190	1.409	-
WIV (N=2,443)	.099	3.239***	47.7%

Note: Delta (Δ) values only calculated for significant proportional changes. The correlation coefficients for males and females ages 13-19 were compared to test the equality of the coefficients across all four waves, and none were significantly different. p<.001***

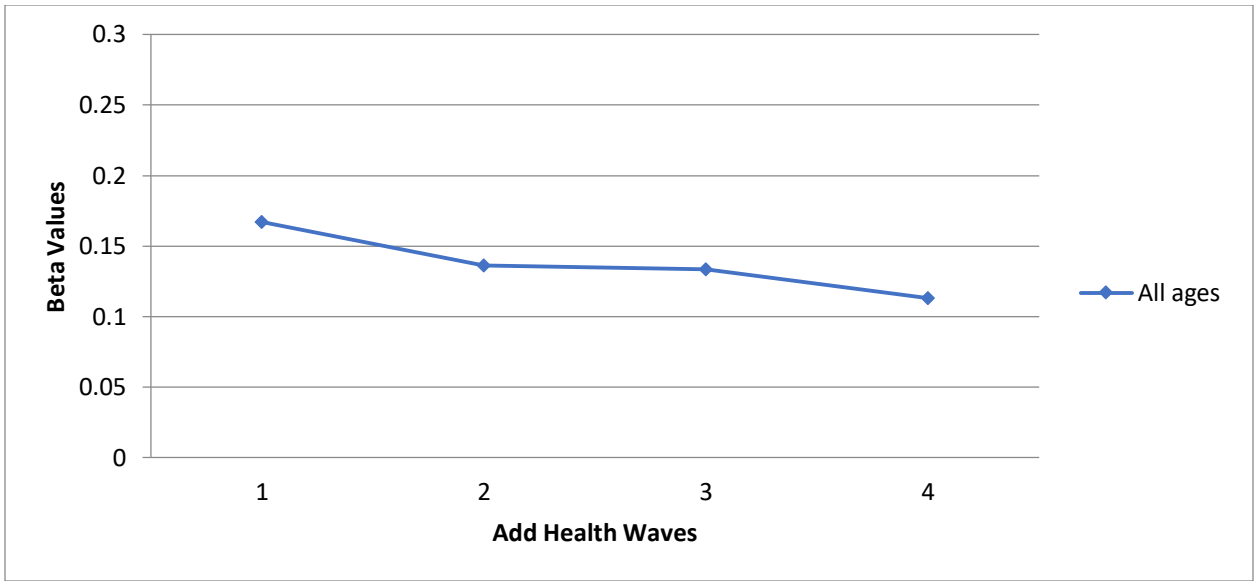

Figure 4.1 Proportional Change in the Relationship Between Aggression and Depression over Time for All Respondents Ages 13-19

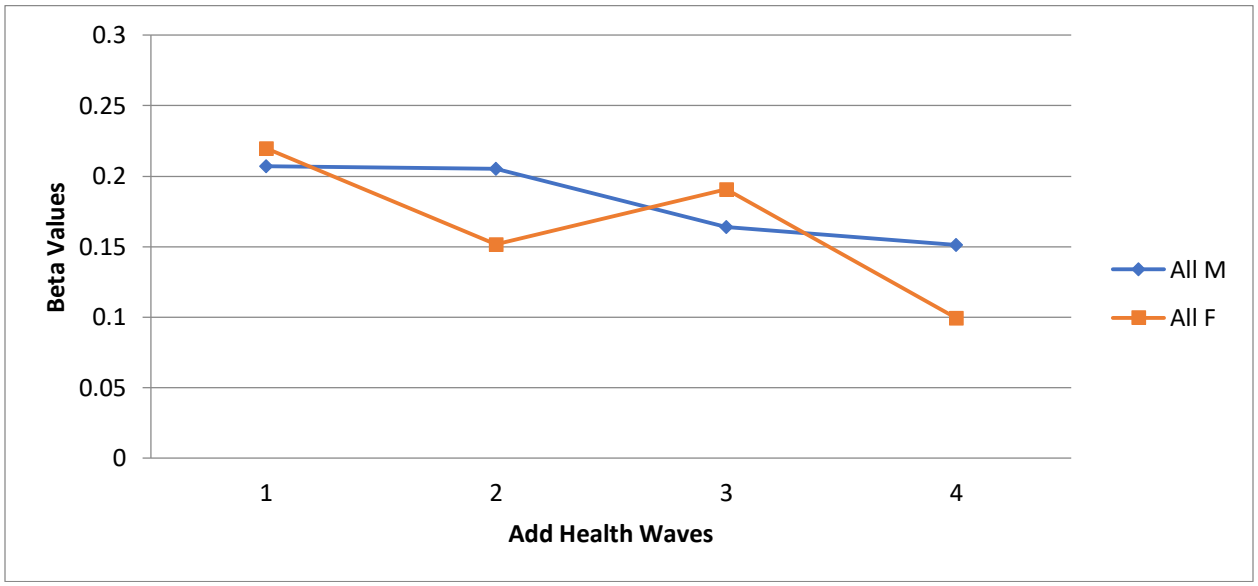

Figure 4.2 Proportional Change in the Relationship Between Aggression and Depression over Time for Males and Females Separated Ages 13-19

Table 4.2 reports the correlation coefficients, z-statistics, and proportional changes for all respondents, males only, and females only in the 13 through 15 age groups. Correlations for all respondents in all waves for this group were weak or borderline negligent. This suggests a weak relationship between aggression and

depression per wave and over time for this age group. Further, equality of coefficients tests revealed no significant proportional changes between waves, although a steady decline in correlation coefficients can be seen. In contrast, both males and females in the 13 through 15 age group showed decreases in correlations between Waves I and II, as well as Waves III and IV, but increases between Waves II and III. These results suggest a weakening relationship between aggression and depression between Waves I and II, and III and IV, but a slight increase in the strength of the relationship between aggression and depression between Waves II and III. Similar to the other age categories, the correlations for males, females, and all respondents in the 13 through 15 age models combined were weak, suggesting a weak relationship between aggression and depression over time. Equality of coefficients tests revealed no significant proportional changes between waves for males but did reveal a significant proportional change in correlation coefficients for females between Waves III and IV. Overall, the relationship between aggression and depression over time for this age category was weak. Figure 4.3 shows the changes in correlations for all respondents in the 13 through 15 age group, depicting a decreasing relationship between aggression and depression over time. Figure 4.4 shows the correlations for males and females in the 13 through 15 age group, indicating decreases and slight increases in the strength of the relationship between aggression and depression over time.

Table 4.2 Correlations, Z-Statistics, and Proportional Change in the Relationship Between Aggression and Depression over Time Ages 13-15

	All Respondents 13-15		
Wave	r	*z*	Δ
WI (N=2,525)	.157	-	-
WII (N=2,240)	.136	.755	-
WIII (N=1,963)	.133	.081	-
WIV (N=1,878)	.137	.109	-
	Males Ages 13-15		
Wave	r	*z*	Δ
WI (N=1,160)	.145	-	-
WII (N=1,019)	.178	.777	-
WIII (N=852)	.147	.650	-
WIV (N=816)	.179	.645	-
	Females Ages 13-15		
Wave	r	*z*	Δ
WI (N=1,365)	.238	-	-
WII (N=1,221)	.182	1.477	-
WIII (N=1,111)	.245	1.562	-
WIV (N=1,062)	.132	2.661***	45.8%

Note: Delta (Δ) values only calculated for significant proportional changes. The correlation coefficients for males and females ages 13-15 were compared to test the equality of the coefficients across all four waves, and waves 1 and 3 were significantly different. p<.001***

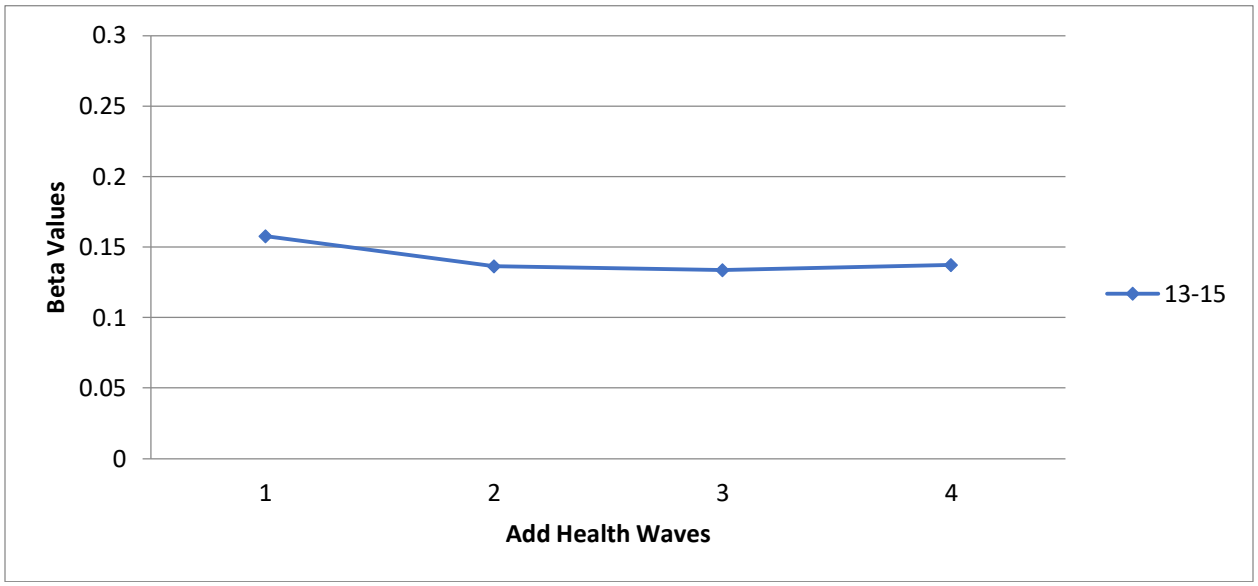

Figure 4.3 Proportional Change in the Relationship Between Aggression and Depression over Time for All Respondents Ages 13-15

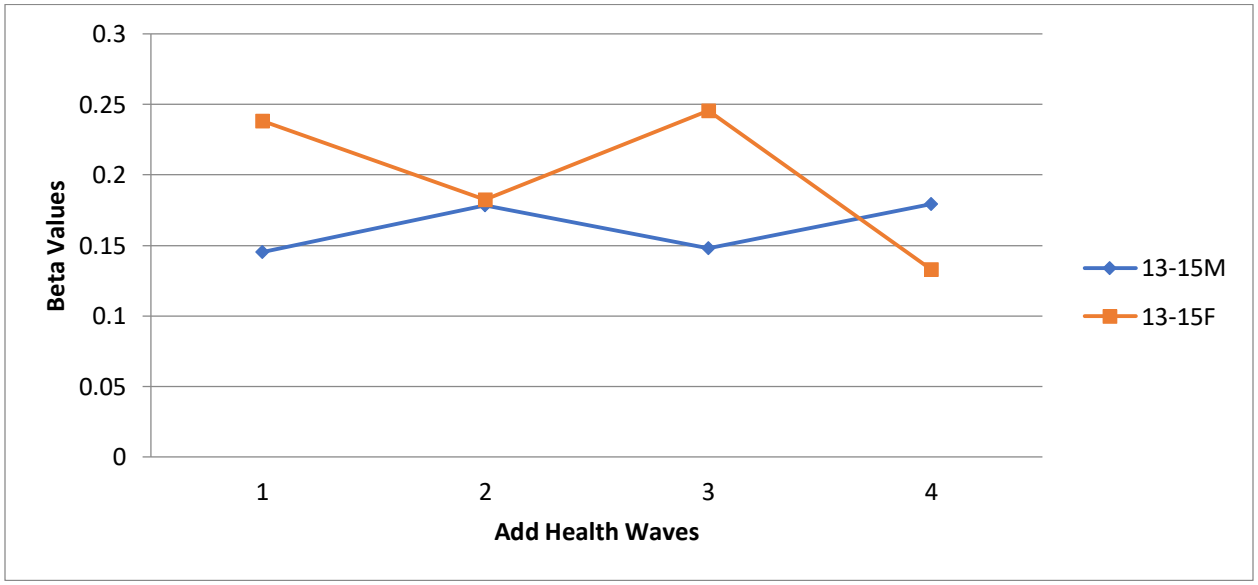

Figure 4.4 Proportional Change in the Relationship Between Aggression and Depression over Time for Males and Females Separated Ages 13-15

Table 4.3 reports the correlation coefficients, z-statistics, and proportional changes for all respondents, males only, and females only in the 16 through 19 age groups. Generally, correlations for all waves in all three models were weak, except two correlations that were negligent. Specifically, the correlation for all respondents in the 16 through 19 age group in Wave IV was negligent (r = .090). The correlation for females in the 16 through 19 age group in Wave IV was also negligent (r= .070). For all respondents in the 16 through 19 age group, correlation coefficients showed a steady decrease across all four waves, indicating the relationship between aggression and depression over time is weak, ultimately becoming negligent by Wave IV. Equality of coefficients tests for all respondents revealed no significant proportional changes in correlation values between waves. Similar results were found for males in the 16 through 19 age group. As can be seen in Table 4.3, the correlations for males steadily decreases across all four waves, indicating that the relationship between aggression and depression over time is weak and also approaches negligence by Wave IV. Equality of coefficients tests for males in this age group also showed no statistically significant proportional changes across time. For females in the 16 through 19 age group, a significant decrease can be seen in the correlation coefficients between Waves I and II. A slight increase occurs in the correlations between Waves II and III, but then declines between Waves III and IV. As was the case in all other models tested, the relationship between aggression and depression over time for females in the 16 through 19 age model is weak, ultimately becoming negligent by Wave IV. Equality of coefficients tests showed that a significant proportional change in correlation values occurred for females in this age category between Waves I and II. As can be seen in Table 4.3, the initial correlation value at Wave

I is .214, but then drops by nearly half by Wave II (r = .118). Figure 4.5 depicts the proportional changes in correlation coefficients for all respondents in the 16 through 19 age group. The graph clearly shows a steady decline in the relationship between aggression and depression over time. Figure 4.6 shows the proportional changes in correlation values for both males and females in the 16 through 19 age group, respectively. A slight increase in the correlation values can be seen for females at Wave III, but a steady decline across all waves for males.

Table 4.3 Correlations, Z-Statistics, and Proportional Change in the Relationship Between Aggression and Depression over Time Ages 16-19

	All Respondents 16-19		
Wave	r	*z*	Δ
WI (N=3,821)	.170	-	-
WII (N=2,501)	.134	1.418	-
WIII (N=2,677)	.133	.031	-
WIV (N=2,689)	.090	1.571	-
Wave	For Males Ages 16-19		
	r	*z*	Δ
WI (N=1,893)	.224	-	-
WII (N=1,236)	.222	.060	-
WIII (N=1,276)	.179	1.114	-
WIV (N=1,308)	.128	1.307	-
Wave	Females Ages 16-19		
	r	*z*	Δ
WI (N=1,928)	.214	-	-
WII (N=1,265)	.118	2.683***	44.7%
WIII (N=1,401)	.136	.487	-
WIV (N=1,381)	.070	1.752	-

Note: Delta (Δ) values only calculated for significant proportional changes. The correlation coefficients for males and females ages 16-19 were compared to test the equality of the coefficients across all four waves, and only wave 2 was significantly different. p<.001***

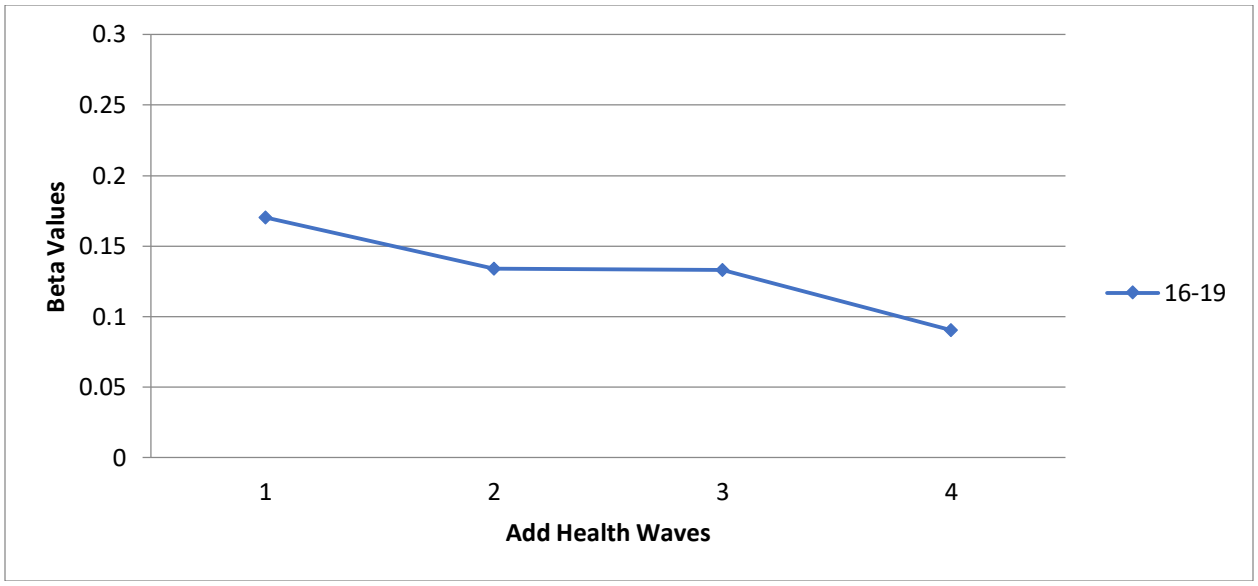

Figure 4.5 Proportional Change in the Relationship Between Aggression and Depression over Time for All Respondents Ages 16-19

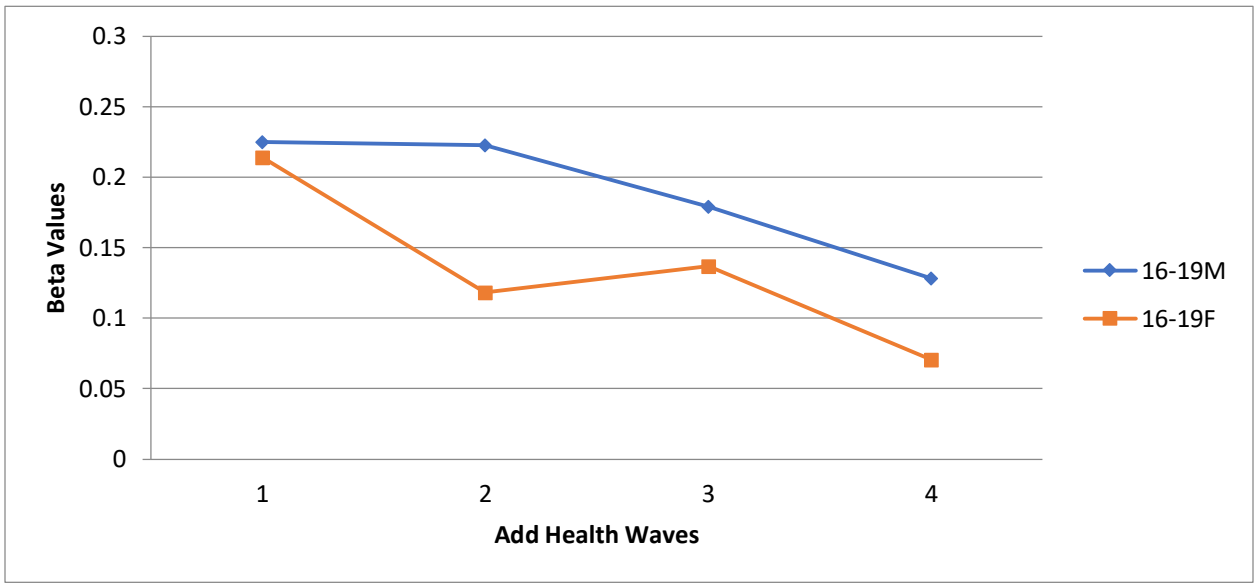

Figure 4.6 Proportional Change in the Relationship Between Aggression and Depression over Time for Males and Females Separated Ages 16-19

Overall, based on the results of the correlational analysis, significant proportional changes in the strength of the relationship between aggression and depression were observed predominantly among females as compared to males. By proxy, age (or aging)

is a weak moderator of the relationship between aggression and depression across the life-course according to these results. As can be seen in Tables 4.1, 4.2, and 4.3, significant proportional changes were only observed within those models that specifically assessed female respondents over time. This recurring pattern was somewhat perplexing, although it is likely that these results may indicate a two-way interaction between age and biological sex and their moderating effects on the relationship between aggression and depression. To further explore whether age or biological sex was a stronger moderator, interaction terms were created between age and the depression latent constructs, biological sex and the depression latent constructs, age and the aggression latent constructs, and biological sex and the aggression latent constructs. Each variable was first mean centered by converting the raw distribution of scores into z-scores then multiplied appropriately to create the interaction terms. In the first two models depression interaction terms were modeled as the independent variables and aggression the dependent variable. The following two models used the aggression interaction terms as the independent variables and depression as the dependent variable.

Table 4.4 shows the output of OLS regression model estimating the moderating effect of biological sex on the relationship between aggression and depression. Results showed that for Wave I, the relationship between aggression and depression was weaker for females than for males ($\beta = -.253$). The moderating effect of biological sex further weakened the association between aggression and depression for females by Wave II ($\beta = -.327$) but strengthened slightly by Waves III ($\beta = -.233$) and IV ($\beta = -.224$). These results collectively indicate that the moderating effect of biological sex on the relationship between aggression and depression weakens that relationship for females as compared to

males. It also appears that biological sex is moderating the relationship between aggression and depression marginally over time.

Table 4.4 Regression Models Estimating the Multiplicative Effect of Biological Sex on the Relationship Between Aggression and Depression

Wave 1	Variable	Coefficient	p-value	β
Aggression				
	Depression	.439	.000	.441
	Sex	-.391	.000	-.196
	Age	-.019	.005	-.034
	White	-.124	.000	-.058
	Sex X Depression	-.149	.000	-.253
$R^2 = .0737$				
N = 6,332				
Wave 2				
Aggression				
	Depression	.487	.000	.488
	Sex	-.388	.000	-.194
	Age	-.028	.002	-.044
	White	-.128	.000	-.060
	Sex X Depression	-.192	.000	-.327
$R^2 = .0653$				
N = 4,731				
Wave 3				
Aggression				
	Depression	.371	.000	.372
	Sex	-.384	.000	-.193
	Age	-.036	.000	-.066
	White	-.106	.000	-.050
	Sex X Depression	-.135	.000	-.233
$R^2 = .0629$				
N = 4,628				
Wave 4				
Aggression				
	Depression	.338	.000	.337
	Sex	-.277	.000	-.139
	Age	-.019	.020	-.033
	White	-.088	.005	-.041
	Sex X Depression	-.133	.000	-.224
$R^2 = .0377$				
N = 4,556				

Note: Negative Beta (β) values indicate a weaker association among females.

Table 4.5 shows the output of an OLS regression model estimating the moderating effect of age on the relationship between aggression and depression. All of the coefficients for the ageXdepression interaction terms were negative, suggesting that the relationship between aggression and depression is weaker for the older respondents in the sample. These coefficients were also small suggesting that the moderating effect of age on the relationship between aggression and depression is negligible. Although the interaction terms were found to be statistically significant in both Waves III and IV, the coefficients suggest that the moderating effect is not substantively significant in any of the waves in the model. However, this is likely due the continuous measurement of age. Overall, the results from this interaction model collectively indicate that the moderating effect of age on the relationship between aggression and depression is weak and negligible.

Table 4.5 Regression Models Estimating the Multiplicative Effect of Age on the Relationship Between Aggression and Depression

Wave 1	Variable	Coefficient	p-value	β
Aggression				
	Depression	.198	.000	.199
	Sex	-.384	.000	-.193
	Age	-.018	.007	-.032
	White	-.124	.000	-.059
	Age X Depression	-.014	.236	-.014
R^2 = .0687				
N = 6,332				
Wave 2				
Aggression				
	Depression	.171	.000	.172
	Sex	-.376	.000	-.188
	Age	-.025	.005	-.039
	White	-.132	.000	-.062
	Age X Depression	-.013	.367	-.012
R^2 = .0573				
N = 4,731				
Wave 3				
Aggression				
	Depression	.146	.000	.147
	Sex	-.378	.000	-.190
	Age	-.036	.000	-.067
	White	-.109	.000	-.051
	Age X Depression	-.033	.020	-.033
R^2 = .0598				
N = 4,628				
Wave 4				
Aggression				
	Depression	.120	.000	.120
	Sex	-.272	.000	-.136
	Age	-.019	.020	-.033
	White	-.088	.004	-.041
	Age X Depression	-.034	.017	-.034
R^2 = .0347				
N = 4,556				

Note: Negative Beta (β) values indicate weaker associations.

Table 4.6 shows the output of an OLS regression model estimating the moderating effect of biological sex on the relationship between depression and aggression. All of the coefficients for the sexXaggression interaction term in this model

were positive, suggesting that the relationship between depression and aggression is stronger for females than for males in the sample. The results specifically show that at Wave I, biological sex is having a weak put positive effect for females ($\beta = .291$). By Wave II, the moderating effect of biological sex dropped by more than half ($\beta = .132$) but strengthened by Wave III ($\beta = .271$). At Wave IV, the moderating effect of biological sex was again reduced by more than half ($\beta = .111$), suggesting a weakening but positive moderating effect of biological sex. Collectively, these results suggest that the moderating effect of biological sex on the relationship between depression and aggression is stronger for females than for males in the sample.

Table 4.6 Regression Models Estimating the Multiplicative Effect of Biological Sex on the Relationship Between Depression and Aggression

Wave 1	Variable	Coefficient	p-value	β
Depression				
	Aggression	-.079	.033	-.079
	Sex	.408	.000	.204
	Age	.074	.000	.128
	White	-.122	.000	-.057
	Sex X Aggression	.220	.000	.291
R^2 = .0918				
N = 6,332				
Wave 2				
Depression				
	Aggression	.042	.316	.042
	Sex	.449	.000	.224
	Age	.062	.000	.097
	White	-.151	.000	-.070
	Sex X Aggression	.097	.002	.132
R^2 = .0819				
N = 4,731				
Wave 3				
Depression				
	Aggression	-.104	.022	-.104
	Sex	.293	.000	.146
	Age	-.013	.093	-.024
	White	-.127	.000	-.059
	Sex X Aggression	.211	.000	.271
R^2 = .0488				
N = 4,628				
Wave 4				
Depression				
	Aggression	.018	.689	.018
	Sex	.223	.000	.112
	Age	-.007	.375	-.012
	White	-.146	.000	-.068
	Sex X Aggression	.085	.015	.111
R^2 = .0319				
N = 4,556				

Note: Positive Beta (β) values indicate a stronger association among females.

Table 4.7 shows the output of an OLS regression model estimating the moderating effect of age on the relationship between depression and aggression. In Wave I the moderating effect of age was found to be positive (β = .016) but negligible. This

result suggests that the moderating effect of age is slightly stronger for older participants in the sample, but statistically insignificant. Waves II, III, and IV had negative correlation values that were also found to be negligible, suggesting that the moderating effect of age is slightly weaker for older sample participants. When assessing the ageXaggression interaction term, these results generally suggest the moderating effect of age on the relationship between depression and aggression is negligible and not statistically significant. Overall, these results collectively indicate that the moderating effect of age on the relationship between depression and aggression is consistently negligible as individuals' age across the life-course.

Table 4.7 Regression Models Estimating the Multiplicative Effect of Age on the Relationship Between Aggression and Depression

Wave 1	Variable	Coefficient	p-value	β
Depression				
	Aggression	.196	.000	.195
	Sex	.393	.000	.196
	Age	.072	.000	.124
	White	-.125	.000	-.058
	Age X Aggression	.017	.184	.016
$R^2 = .0833$				
N = 6,332				
Wave 2				
Depression				
	Aggression	.168	.000	.167
	Sex	.443	.000	.221
	Age	.061	.000	.095
	White	-.153	.000	-.071
	Age X Aggression	-.008	.524	-.008
$R^2 = .0801$				
N = 4,731				
Wave 3				
Depression				
	Aggression	.148	.000	.148
	Sex	.274	.000	.137
	Age	-.013	.089	-.024
	White	-.132	.000	-.062
	Age X Aggression	-.013	.292	-.015
$R^2 = .0417$				
N = 4,628				
Wave 4				
Depression				
	Aggression	.118	.000	.118
	Sex	.218	.000	.109
	Age	-.007	.369	-.013
	White	-.150	.000	-.070
	Age X Aggression	-.022	.133	-.022
$R^2 = .0311$				
N = 4,556				

Note: Negative Beta (β) values indicate weaker associations.

CHAPTER FIVE: DISCUSSION

Several important findings emerged from the results of this thesis. First, based on the results of the factor analysis, correlational tests, and equality of coefficients tests, it can be said with a high level of confidence that age, by proxy of aging over time across waves, is a weak moderator of the relationship between aggression and depression as individuals age across the life-course. Despite several theorists (Agnew, 2005; Moffitt, 1993; Laub & Sampson, 2003) suggesting that aging and different life-course interactions influence criminal offending, and perhaps indirectly, aggression and depression over time, the results of this study did not find direct support of these theories. This is partially due to the simplicity of this study, and partially due to the limitations of the data which are addressed below. However, several changes over time were observed in the relationship between aggression and depression, which may provide partial support for the notion that aging, physiological change, and social change are apparent across the life-course, as Moffitt's (1993) theoretical view suggests. Although significant proportional changes were observed among female participants in three separate age groups, the correlation coefficients were still very low, with none surpassing a weak level of association. Overall, these results were somewhat unexpected as there was an assumption made that the correlations between the depression and aggression constructs would be much higher because prior research has elucidated that such a relationship is possible (Messer & Gross, 1994; Muris et al., 2008; Quiggle et al., 1992). However, they did not, and were in fact negligible in certain respects. Perhaps it is likely that a stronger

correlation between aggression and depression would be found in a sample of individuals with well documented behavioral disorders or pathological issues rather than, presumably, an otherwise normal sample of adolescents and young adults.

Second, based on the correlational tests and equality of coefficients tests, inter-wave changes in the relationship between aggression and depression were not significantly different from each other, suggesting that the incremental changes in participants' ages did not affect the relationship to any significant degree. However, when observing the figures in the results section, a clear and steady decline in the relationship between aggression and depression can be seen, especially when observing the starting correlation values at Wave I in relation to the correlation values at wave IV in most of the different age groups. Thus, the first hypothesis in this study was partially supported in that the relationship between aggression and depression did in fact change over time. This may be due to aggression decreasing over time as evidenced by the age-crime curve while depression prevalence was found to be more erratic. Nevertheless, the differences between each wave were mostly negligible, suggesting that (a) age, by proxy of aging, is a weak moderator, and (b) the changes observed in the relationship over these shorter periods of time are not significant from wave to wave.

The analysis from the multiplicative interaction models also yielded some interesting results. As was mentioned earlier, the results from the first half of the analysis revealed a recurring pattern among the different age groups of females in the sample. Although only nine models were presented in this thesis, the author went to great lengths by assessing 21 different age groups of females, ultimately finding that 90.4% of those female only models showed significant proportional differences. This strange pattern

implicated a possible two-way interaction between age and biological sex, which is why multiplicative interaction models were employed to serve as a robustness check of the current findings. Based on the results of the multiplicative interaction models, it was discovered that, when using the sexXdepression interaction term as the independent variable, the moderating effect of biological sex was clearly present, albeit marginal, and that the relationship between aggression and depression was weaker for females than for males. Generally, this provides support for the second hypothesis in this thesis. While this does imply that the moderating effect of biological sex does exist, the strength of the effect seems to be of low magnitude.

Interestingly, when the interaction term was flipped (sexXaggression as the independent variable), biological sex moderated the relationship between aggression and depression more strongly for females than for males. This finding also provides support for the second hypothesis in this thesis. While it may seem perplexing that one model indicated the moderating effect of biological sex was weaker for females but stronger in the other model for females, it is likely the case that one or more variables in the multiplicative models are dominating the variance in the dependent construct being measured. Nevertheless, based on the results provided in Table 4.6, the moderating effect of biological sex on the relationship between depression and aggression was found to be stronger for females than for males in the sample, yet the moderating effect was quite weak.

In contrast, both multiplicative interaction models that assessed age as a moderator indicated negligible results. The use of the ageXaggression term as the independent variable collectively indicated no statistically significant results of the

moderating effect, and further that the beta values were so low that they could almost be considered zero. When observing the effect of the ageXdepression interaction term, it should be noted that Waves I and II were not statistically significant, yet Waves III (p = .020) and IV (p =.017) were statistically significant. However, this is likely the product of having large sample sizes per wave, indicating that some effect could be found significant, irrespective of its substantive significance. Overall, the combined results from the factor analysis, correlational tests, equality of coefficients tests, proportional assessments, and the multiplicative interaction models suggest that age, both directly and by proxy, is a weak moderator of the relationship between aggression and depression. This ultimate finding is both true when looking at age as a continuous variable, and aging over time.

Limitations

While this study filled a gap in the literature, there are limitations that need to be addressed. As was alluded to earlier, the depression measures used to generate the latent depression construct were all viable, with alpha and KMO statistics indicating strong reliability and sampling adequacy. Furthermore, the distributions for the depression constructs were normally distributed, meeting the general assumptions of the GLM. However, several statistical concerns emerged with the aggression measures. First, the alpha scores indicated very poor scale reliability, and the KMO statistics ranged from unacceptable to middling scores. Even more alarming was the extreme positive skew associated with the aggression constructs which clearly violated the assumptions of the GLM. While several transformative and measurement techniques were used in an attempt to remedy these issues, none proved successful. These statistical warnings were foregone

on two bases. First, the Eigenvalues for the aggression factors were slightly above 1 and thus were considered acceptable. This was a clear mistake as Eigenvalues above 1 do not confirm scale reliability or sampling adequacy. In deciding whether to keep an Eigenvalue slightly above 1, one author noted, “Oftentimes, the answer is no, suggesting that the eigenvalue rule may be too generous a basis for retaining factors” (DeVellis, 2017, p. 167). Second, with no other data available, the decision was made to simply move forward while simultaneously accepting the reality that these errors may, in the end, prove detrimental in the statistical analyses.

The second major limitation concerns the types of aggression questions respondents answered. When observing the types of questions asked, it can be quickly gleaned that low aggression prevalence would be a major issue. For example, the depression measures (see Appendix A) asked fairly common questions to which there were much higher responses, such as “How often in the past 12 months did you feel sad?” In comparison, the responses to the aggression measures (see Appendix A) were considerably less prevalent, especially among those questions that focused on more egregious forms of aggression like “During the past 12 months, did you shoot or stab someone?” The apparent incongruence between the intensity or severity of what was being asked led to many respondents reporting some degree of depression and few respondents reporting some degree of aggression, causing severe skewing and abnormal distributions in aggression which could not be rectified. As is always the case in any research project, the quality of the data will directly impact the quality of the results, and frankly, the poor quality of the aggression data in this research impacted the results.

Future Research

Future researchers wishing to explore the complex relationship between aggression and depression would do better to have access to much better, more accurate measures of aggression. Furthermore, it may prove useful to draw distinctions between general population surveys, such as Add Health, from other types of surveys that focus on particularly aggressive and depressive individuals. It is possible that rates of extreme forms of aggression and depression are relatively rare among the general population, but are more pronounced among smaller, more particularized groups of individuals. Future research on the relationship between aggression and depression and the moderating effects of age and biological sex on that relationship should focus on testing the hypotheses outlined in this thesis on more particularized subsets of the population.

Conclusion

Collectively, the results of this thesis identified that a relationship does exist between aggression and depression, and that the relationship does in fact change over time. The relationship significantly changed over time only when looking at specific waves among females in the sample, but not among males or all respondents combined. However, even among males and all respondents', a relationship between aggression and depression was found to exist. This finding is in line with prior research focusing on the potential relationship between aggression and depression (Baumeister et al., 2000; Muris et al., 2008; Van Praag, 2001; Winkler et al., 2005). Although not significant, the relationship between aggression and depression for all respondents combined and for males did decrease as individuals aged across the life-course. Generally, this decreasing trend could be the result of individuals developing an initial condition marked by the co-

occurrence of aggression and depression which then subsides as individuals get older. This would suggest Moffitt's (1993) theoretical suggestions regarding adolescent-limited offenders might explain the lack of stability and persistence of aggression and depression over time. In other words, perhaps the relationship between aggression and depression is limited to adolescents, and this relationship then decreases as individuals' age. It is also possible that as individuals age, life circumstances change which inhibit depression and aggression from increasing, such as the development of prosocial bonds, positive life experiences, stable jobs, familial bonds, and other stakes in conformity (Agnew, 2005; Laub & Sampson, 2003). These pillars in conformity may have positive influences in individuals' lives which then temper the occurrence of both depressive and aggressive behaviors across the life-course.

It is also important to note that when observing the relationship between aggression and depression among all participants in the study, the strength of the relationship was never greater than the first wave measured. That is, when looking at all respondents combined, the strength of the relationship was consistently strongest among younger individuals and then steadily declined across waves. This might suggest that younger individuals experience more aggression and depression during periods of rapid physiological, cognitive, and social maturation. For example, younger individuals (due to the developmental status they occupy) may be more susceptible to social pressures, social and peer labels, negative and ruminative thinking, and distorted views of personal identity, self-worth, and value. These kinds of susceptibility could potentially facilitate depression, aggression, or both, which then subside as susceptibility decreases with age.

Perhaps it is the case that young individuals, especially in their formative years, are more susceptible to depression and aggression than are older individuals.

This study is relevant for two reasons. First, it is always important to empirically test whether relationships do or do not exist between different variables. In this case, it is easy to see that assumptions could be made about the incongruence between aggression and depression: that they share nothing in common; that they are completely separate and unrelated behaviors; that only aggression is important in understanding potentially criminal and/or violent behavior. The litany of research showing the commonalities between these two behaviors exposes the inaccuracies of those assumptions. If nothing else, this thesis has supported prior research by identifying that these two seemingly different behaviors do, in fact, share a relationship, and that the relationship is present in both women and men. Second, it is important to recognize that when assessing and/or diagnosing individuals as either aggressive or depressive, it is possible that one behavior facilitates the existence of the other, suggesting that the causal mechanisms of one are actually dependent upon the other. This notion shatters the idea that aggression and depression are completely separate behaviors that do not interact. By using empirical methods to establish the existence of a shared relationship, a deeper understanding of maladaptive behavior (and even perhaps some severe forms of criminal behavior) can aid in properly identifying and responding to different behaviors and symptoms which generate negative human behavior. It should be accepted that when observing an aggressive individual, depression may be lurking in the darkness, generating what observers see as aggressive behavior. This should also be recognized in the opposite direction. It is possible that individuals experience aggressive issues which influence the

etiology of their depression. Recognizing that a relationship exists, practitioners and researchers alike will be in a better position to understand the complex nature of aggression and depression, how they are different, and how they interact across the life-course.

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APPENDIX A

Add Health Measures of Respondent Depression

Depression Measures: How often was the following true during the past week?

1. You were bothered by things that don't usually bother you.

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

2. You felt depressed.

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

3. You had trouble keeping your mind on what you were doing.

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

4. You felt that you were too tired to do things.

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

5. You felt that you could not shake off the blues, even with help from your family and your friends.

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

6. You felt that people disliked you

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

7. You felt sad.

0 = never/rarely 1 = sometimes 2 = a lot of the time 3 = most/all the time

8. You felt you were just as good as other people.

0 = most/all the time 1 = a lot of the time 2 = sometimes 3 = never/rarely

9. You enjoyed life?

0 = most/all the time 1 = a lot of the time 2 = sometimes 3 = never/rarely

10. During the past 12 months, did you ever seriously think about committing suicide?

0 = No 1 = Yes

Add Health Measures of Respondent Aggression

Aggression Measures: During the past 12 months, how often did?

1. You use or threaten to use a weapon to get something from someone?

0 = never 1 = one to two times 2 = three to four times 3 = five or more times

2. You take part in a fight where a group of your friends was against another group?

0 = never 1 = one to two times 2 = three to four times 3 = five or more times

3. You deliberately damage property that didn't belong to you?

0 = never 1 = one to two times 2 = three to four times 3 = five or more times

4. During the past 12 months, did you pull a knife or gun on someone?

0 = No 1 = Yes

5. During the past 12 months, did you shoot or stab someone?

0 = No 1 = Yes