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HYPERACTIVITY-IMPULSIVITY-ATTENTION DEFICIT AND AGGRESSION IN RELATION TO VIOLENT AND CHRONIC OFFENDING

By

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ABSTRACT

HYPERACTIVITY-IMPULSIVITY-ATTENTION DEFICIT AND AGGRESSION IN RELATION TO VIOLENT AND CHRONIC OFFENDING

By

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The diagnosis of hyperactivity-impulsivity-inattention deficit (HIA-deficit) and its comorbid relationship with other dysfunctional behaviors such as aggression have found to exist frequently in the delinquent population. In this study the interaction of HIA-deficit and aggression was hypothesized to increase the amount of violence and chronic offending compared to individuals with HIA-deficit or aggression alone. Using the Cambridge Study in Delinquent Development by Dr. David Farrington, inconclusive evidence was found on the significance of comorbid HIA-deficit and aggression on a boy's violent and chronic offending. While the interaction of HIA-deficit and aggression increased more violence in the boys during their teenage years, analysis of variance provided no evidence of the interaction having caused more chronic offending or violence in the childhood and adult years compared to a diagnosis of HIA-deficit or aggression alone. The findings suggest the need for longitudinal treatment of those diagnosed with HIA-deficit or aggression.

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INTRODUCTION

Social science research contends that there exists at least two identifiable points of interest with respect to childhood behavior. First, research suggests that childhood behavior seems to have a high degree of continuity over time (Loeber & Dishon, 1983). The second point of interest which exhibits itself through research is that there are consistent social situations which pose the greatest risk for delinquent behavior (Loeber & Dishon, 1983). The significance of these two discoveries is that we should presumably be able to determine childhood delinquency risk factors. Furthermore, we could infer that by determining those delinquent risk factors that not only could we quell delinquent activity considerably, be we would also have gained the capacity to thwart childhood delinquent activities from amplifying into acts of violence. Unfortunately, research has presented us with another conclusion about childhood risk factors: reliable childhood behavior, accompanied by situations that are consistently risky in terms of delinquent activity, does not necessarily yield valid risk factors.

Indeed, risk factors for childhood delinquency are criticized as often as they are researched. However, amidst all the scrutiny of viable risk factors, the search for these childhood factors has shown to provide a fair number of convincing and reliable factors for different types of crimes. Both violent and chronic offending are two particular areas which have identified a large number of risk factors. The search for, and confirmation of,

childhood risk factors as they relate to violence and chronic offending is the general focus of this thesis.

Hyperactivity-impulsivity-attention deficit (HIA-deficit) disorder is one risk factor that has been found to predispose children to delinquent behavior[1]. Indeed, HIA-deficit has been a growing topic of debate as of late. However, this disorder has not become an interest by virtue of an ubiquitous nature. According to the American Psychiatric Association (1994), HIA-deficit has a prevalence rate among schoolchildren at around 3-5% of the general population. Although these numbers point to the rare occurrence of this neurological deficit in the general population, in the delinquent population HIA-deficit has been found to occur exceedingly more often (af Klinteberg, Anderson, Magnusson, & Stattin, 1993; Barkley, Anastopoulos, Guevremont, & Fletcher, 1991; Gittleman, Mannuzza, Shenker, & Bonagura, 1985; Loeber, Stouthamer-Loeber, & Green, 1991; Scatterfield, Hoppe, & Schell, 1982). This augmented prevalence of those diagnosed as HIA-deficit, within not only the delinquent population but the adult criminal population as well, will be an area of concentration for my study.

When using HIA-deficit as a predictor variable for violent and chronic offending, it is necessary to consider other variables that might relate significantly to an HIA-deficit diagnosis. The reasoning behind this lies with the fact that there is a common occurrence of comorbidity, or combined existence, of two or more disorders in those diagnosed with HIA-deficit.

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[1] The American Psychiatric Association (APA) diagnostic manual describes this neurological disorder as ADHD, or Attention-Deficit/Hyperactivity Disorder (1994). However, I prefer using what Loeber et al. (1991) has described as the symptom factors that make up ADHD as HIA-deficit (hyperactivity-impulsivity-attention deficit). Thus, I will be using this HIA-deficit nomenclature primarily for the remainder of my thesis.

One construct which research has found frequently to exist in conjunction with HIA-deficit is aggression. Aggression has been defined as various forms of behavior, from a physical, violent response to a more verbally abusive, non-physical type of conduct. Magnussen, Stattin, & Duner (1983) used a definition of aggression which supports the non-physical mode of aggressive behavior, and is comparable to this study's concept of aggression:

> They (the boys) were aggressive against teachers and classmates. They may be impertinent and impudent, actively obstructive or incite to rebellion. They like disturbing and quarrelling with classmates (p.284).

Found within the realm of conduct disorder (APA, 1994), aggression has been found to be a risk factor for violent crimes as well. The literature that exists on aggression and violence will be reviewed, along with the significance of comorbid HIA-deficit and aggression as risk factors for delinquency and violent offending.

A theory which is of particular importance to the test of HIA-deficit, aggression, and delinquency is Moffitt's Developmental Theory (Moffitt, 1993). In her theory, Moffitt asserts there are two types of criminal offenders: *life-course-persistent* and *adolescent-limiting* individuals. Moffitt's Theory is intended convey the fact that "antisocial behavior is remarkably stable across time and circumstance for some persons but decidedly unstable for most other people" (1993, p.676). It is a group of life-course-persistent offenders which tend to engage is a continuous level of antisocial behavior throughout their lifetime. Conversely, the adolescent-limiting individuals are those who commit crimes for a shorter duration (primarily during adolescence), and basically are considered to have more temporary involvement in antisocial behaviors. Moffitt's

Developmental Theory will be used as the theoretical basis to test my hypotheses, and will be discussed later in further detail.

The variables used in the analyses are taken from the Cambridge Study in Delinquent Development by Dr. David Farrington (Farrington, 1994). This longitudinal study allows for adequate operationalization of HIA-deficit, aggression, and violent and chronic offending. Additionally, this study allows me to test the levels of offending in differing age groups. Past research using the Cambridge Study's data has compared aggression and violence (Farrington, 1989), while another has looked at the relationship of hyperactivity and conduct disorder in childhood and its long-term criminal outcomes (Farrington, Loeber, & Van Kammen, 1990). Indeed, it seems research from the Cambridge Study is lacking in the area of HIA-deficit and aggression interaction. Therefore, I conclude the Cambridge Study will allow my findings to bring a fresh and practical angle to the research world of violence and chronic offending. A more thorough description of the Cambridge Study will be presented in the "DATA" chapter.

Once again, the hypotheses I will be testing include the concepts of HIA-deficit, aggression, and violent and chronic offending. An exorbitant amount of research has covered the link between HIA-deficit, aggression, and criminal behavior. Nonetheless, the diverse nature of operationalization in concepts such as HIA-deficit and aggression in existing research leads to my interest in conducting another study on this topic.

My hypotheses for the study will be as follows:

Hypotheses #1: Those diagnosed with comorbid HIA-deficit and aggression will commit more violent crimes than those diagnosed with HIA-deficit or aggression alone.

Hypotheses #2: Those diagnosed with comorbid HIA-deficit and aggression will be more likely to be chronic offenders than those diagnosed with HIA-deficit or aggression alone.

Included in hypothesis #2 is the assumption that chronic offending encompasses not only a high number of crimes, but a long period of time as well. I am contending that because HIA-deficit is diagnosed in most cases early in life (before age seven) (APA, 1994), and this in turn augments aggressive behavior (Barkley, DuPaul, & McMurray, 1990), that the bulk of early deviant behavior will consist of children with HIA-deficit and aggression. Subsequently, the early offending in life as a result of comorbid HIA-deficit and aggression will persist and amplify, thrusting those children into a life of chronic, or *life-course-persistent* offending.

Over the years, the definition of HIA-deficit has fluctuated quite often in DSM-III (APA, 1980), DSM-III-R (APA, 1987), and into today's DSM-IV (APA, 1994). Because of this, there has been tremendous variation across studies in their operationalization due to the changing diagnostic criteria (Barkley, Fischer, Edelbrock, & Smallish, 1990). To compound this incongruity, researchers have been far from consistent with respect to their labeling of hyperactivity-impulsivity-attention deficit. Although in recent studies it has been referred to as hyperactivity (Bartusch, Lynam, Moffitt, & Silva, 1997), ADHD (August et al., 1996), and HIA-deficit (Loeber et al., 1991), the symptoms and diagnoses of the disorder by the researchers have remained virtually the same.

Currently, HIA-deficit is diagnosed with the essential features of inattention and/or hyperactivity-impulsivity. The APA (1994) defines HIA-deficit, or ADHD, with five criteria. First, HIA-deficit has the characteristic of "a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable lever of development (Criterion A)" (p.78). The second criterion to HIA-deficit is that some of the symptoms that cause impairment must have been present before age seven (Criterion B). Third, some impairment from the symptoms must be present in at least two different settings (e.g. at school and at home) (Criterion C). Fourth, to be diagnosed as HIA-deficit, there must be clear evidence of the disturbance

interfering with the ability of the youth to express appropriate social, academic, or occupational functioning (Criterion D). Lastly, the disturbance caused can not occur exclusively with other Psychotic Disorders, and HIA-deficit can not be better accounted for by another mental disorder (Criterion E).

Found within Criterion A are the symptoms defining inattention and/or hyperactivity/impulsivity. A diagnoses of HIA-deficit (ADHD) can be characterized into one of three subtypes according to Criteria A. These three subtypes include: ADHD combined; ADHD predominantly inattention; and ADHD predominantly hyperactive-impulsive. A diagnosis of ADHD combined type requires six (or more) symptoms of inattention and six (or more) symptoms of hyperactivity-impulsivity to have persisted for at least six months (APA, 1994). This combined ADHD, with emphasis on both inattention and hyperactivity-impulsivity, will be the focus of my hypotheses.

There are a wide range of symptoms that can be classified under inattention. Some of these symptoms for which inattention could be diagnosed include: difficulty sustaining attention to tasks; not completing schoolwork, chores, or duties in the workplace; often losing things necessary for tasks or activities; and being easily distracted by extraneous stimuli (APA, 1994). Symptoms consistent in individuals with a diagnosis of hyperactivity-impulsivity include: fidgeting with hands or feet or squirming in seat; often running, climbing, or leaving a room unexpectantly; talking excessively; blurting out answers to questions not completely spoken; difficulty in awaiting turn; and interrupting or intruding on others (e.g. during conversation and games) (APA, 1994).

Researchers assert that problems do exist, however, in the symptoms defining HIA-deficit. Indeed, the diagnostic validity of HIA-deficit has been questioned (Szatmari,

Boyle, & Offord, 1989). Specifically, some researchers believe that certain criterion are not pertinent to the definition of HIA-deficit and its relationship to delinquency. Inattention is one such symptom that has been attacked. It has been concluded that inattention is not unique to those diagnosed with HIA-deficit (Halperin et al., 1992). This point leads to the issue of mislabeling. The fact that inattention is characteristic of youth with cognitive deficits may lead clinicians to mislabel children with other psychotic disorders as being HIA-deficit (Halperin et al., 1992). This finding bring to light the considerable argument as to whether HIA-deficit is a "taxonomically valid disorder distinct from other, better established diagnosis, such as conduct disorder" (Szatmari et al., 1989, p.865). Consequently, some contend that, for purposes of differential diagnosis, clinicians need to concentrate more on the presence of hyperactivity and impulsive symptoms (Halperin et al., 1992).

Another symptom factor of HIA-deficit which has been discussed vehemently is hyperactivity. Research has compared the various subtypes of HIA-deficit, specifically with or without hyperactivity (ADD+H and ADD-H), to test their distinct predictability to delinquency. It was found that those with hyperactivity, compared to those without the hyperactive characteristic, had more pervasive conduct problems at home, seemed more impulsive, and were more aggressive and delinquent (Barkley, DuPaul, et al., 1990). This finding lends support not only to the fact that hyperactivity should be concentrated on more when diagnosing this disorder, but perhaps HIA-deficit is not a taxonomically distinct disorder from other firmly established disorders.

Previously reviewed literature has provided evidence that the symptom factors such as inattention and hyperactivity are not particularly distinct to HIA-deficit. However,

other examinations have found certain HIA-deficit symptoms, such as hyperactivity, to be "independent" with respect to other cognitive deficits. One study provided evidence that hyperactivity is indeed a syndrome which exists absent of aggressiveness, disobedience, and antisocial behavior (Stewart, Cummings, Singer, & de Blois, 1981). Other research lends support to the distinctness of HIA-deficit by concluding that measures of aggression do not differentiate among HIA-deficit and normal controls (Thompson, Riggs, Mikulich, & Crowley, 1996). Beyond the clinical properties, it has been asserted that HIA-deficit not only varies with other afflictions such as conduct disorder in terms of their clinical features, but also external variables (Biederman, Newcorn, & Sprich, 1991). For example, HIA-deficit has the outcome of cognitive dysfunction, whereas conduct disorder has the outcomes of aggression, antisocial behaviors, and delinquency (Biederman et al., 1991). Similarly, HIA-deficit tends to vary with other disorders in terms of etiological factors and psychosocial and developmental correlates (Biederman et al., 1991).

In conclusion, it seems there is much debate as to the validity of HIA-deficit as a clinically specific disorder. Inherently, if we are to circumvent any arguments as to the validity of an HIA-deficit diagnosis, we must develop appropriate conceptualizations of this and similar disorders to obtain a valid clinical diagnosis. This in turn can only help to avoid future misdiagnoses and mislabeling (Barkley, 1990).

There have been significant findings that symptoms associated with HIA-deficit are correlated with delinquency and violence. One symptom in particular, hyperactivity, has been found to significantly predict violence (af Klinteberg et al., 1993). In retrospective studies it has been found that multiple offenders, characterized as hyperactive in childhood, were rated as both antisocial and hard-to-handle by both parents and teachers

(Loeber et al., 1991; Scatterfield, 1987). Other behaviors which are characteristic of those with HIA-deficit are a lack of concentration (inattention) and a need to be daring (hyperactive). Both of these HIA-deficit defining behaviors have been found to significantly predict violence in boys aged 12-14 years (Farrington, 1989). Furthermore, in this same study of adolescent boys it was found that high restlessness (a criteria for defining HIA-deficit) significantly predicted all three measures of violence (teenage violence, adult violence, and convicted violence) (Farrington, 1989).

The components of HIA-deficit have not only been found to be a childhood risk-factor for more violent crimes, but also a risk for increased adult institutionalization rates (Scatterfield et al., 1982). It seems those who are characterized as being hyperactive, impulsive, and inattentive in childhood are at a greater risk for activities leading to an arrest, conviction, and incarceration in late adolescence and into early adulthood (Mannuzza, Klein, Konig, & Giampino, 1989). In another study of 110 hyperactive and normal controls if was found the offender rates for serious offenses (robbery, burglary, grand theft, grand theft auto, and assault with a deadly weapon), multiple offenses, and institutionalization were all statistically significant for the hyperactives compared to the normal controls (Scatterfield, 1987).

Although the symptoms of HIA-deficit have been found to be risk factors for later violent crimes, other childhood variables play a role in violent crime as well. One distinct variable that should be reviewed in the company of HIA-deficit is childhood aggressiveness (Loeber & Dishon, 1983). Paralleling HIA-deficit's relation to delinquent and violent offending is aggression's likelihood as a risk factor antisocial and problem behavior (Pulkkinen, 1983). In a study of adolescent boys, Magnussen et al. (1983) found

that high test scores of aggression significantly related to not only to criminal convictions, but were strongly correlated to more serious crimes as well. Similar results were found in a longitudinal follow-up of 8 year-olds (Pulkkinen, 1983). This research documented that at the age of 14 and 20, both alcohol and violent offenses were correlated significantly with aggression (Pulkkinen, 1983). Further evidence of aggression's relation comes from the Cambridge Study of Delinquent Development. Once more aggression's relation to violent behavior was found to be significant (Farrington, 1989).

DEVELOPMENTAL THEORY

The theoretical basis for the hypotheses in this paper is Developmental Theory as composed by Terrie E. Moffitt. The premise of Moffitt's theory is that there are basically two kinds of antisocial behavior: *adolescence-limiting* and *life-course-persistent* behaviors (Moffitt, 1993). Moffitt (1993) contends that this dual taxonomy is needed to describe antisocial behavior because two incompatible facts exist: antisocial behavior shows continuity over age, but that "its prevalence changes dramatically over age, increasing almost 10-fold temporarily during adolescence" (p.674). Therefore, with respect to juvenile delinquency, two distinct categories of individuals exist, each with its own theoretical explanation.

The first theoretical assumption in Moffitt's Developmental Theory is that there is a small group of individuals who engage in antisocial behavior at every stage of their lives. This group, labeled life-course-persistent, reflects roughly 5-6% of the offender population. This percentage was found to be true in other studies, where 6% of the offenders in a sample accounted for more than 50% of the crimes committed (Wolfgang, 1972). It is theorized that early individual differences, such as poor neuropsychological functioning, may persist from infancy and continue into adulthood. It is these neuropsychological differences that may instigate antisocial behavior. Combined with this deficit is a "failed" social development. It is at each stage of human development that

these life-course-persistent individuals fail to practice, even acquire pro-social alternatives. The point of this "failed" development is that if "social and academic skills are not mastered is childhood, it is very difficult to later recover lost opportunities" (Moffitt, 1993, p.684). In summary, it is the lack of development at an early stage in life which makes life-course-persistent individuals behavior essentially inflexible and refractory to changing environments.

The other half to this taxonomy is the antisocial behavior labeled as adolescent-limiting. Unlike life-course-persistent, adolescence-limiting antisocial behavior is theorized to be ubiquitous. These types of delinquents seem to show no continuity to their antisocial behavior, and their delinquent behavior is often abrupt across age. Moffitt (1993) contends this type of antisocial behavior is essentially a "social mimicry" of their life-course-persistent offending counterpart (p.686). Moffitt asserts that today's teens are trapped in a "maturity gap," the difference between the biological and social age. Similarly, it is between the ages of 10 to 15 that children's self-perception of autonomy and self-reliance takes a dramatic shift. Children at this time begin seeing their life-course-persistent counterparts suffering less from the maturity gap. The adolescence-limiting individual perceives the quick rewards (e.g. clothes, cars, drugs) obtained by the life-course-persistent individual, albeit through illegitimate means, as coveted assets. The adolescence-limiting individual wants to relieve the stress from maturity gap, and believes this can be accomplished through the attainment of "mature" status and privileges. Subsequently, in an attempt to cut the gap and reap the rewards, the adolescence-limiting individuals begin to mimic the behavior of life-course-persistent individuals through delinquent activity. However, unlike their life-course-persistent counterparts,

adolescence-limiting individuals are able to limit and discontinue their delinquent behaviors. Because of their "successful" development in childhood, the adolescent-limiting individual is able to recognize reinforcement and punishment contingencies, thus reducing, in fact ceasing, any pattern of delinquent and/or chronic offending.

Moffitt's Developmental Theory is important to my hypotheses for several reasons. To reiterate, my hypotheses include the concepts of chronic and violent offending. Bartusch et al. (1997) found in a study of Moffitt's Developmental Theory that the younger age of onset, the more likely an individual will be a chronic offender. I am hypothesizing that HIA-deficit and aggression play an imperative role in the age of onset of criminal behavior. My hypotheses implicate a young age of onset, because as Developmental Theory posits, and as other research has found, there is a marked reduction of functional problems between the ages of 13 and 18 for those with HIA-deficit symptoms (Gittelman et al., 1985; Moffitt, Lynam, and Silva, 1994). In terms of violent offending, Bartusch et al. (1997) found that "childhood antisocial behavior was significantly associated with convictions for violence, while adolescent antisocial behavior was significantly more strongly associated with convictions for nonviolent offenses" (p.14). Thus, much research has concluded that not only is early onset of delinquency predictive of chronic offending, but is also an excellent predictor of seriousness (Loeber & Dishon, 1983; Moffitt, 1993; Tolan, 1987; Wolfgang, 1972). These findings are consistent with my hypothetical statements. With the early initiation of HIA-deficit and aggression in childhood, and the early onset of crime leading to more violent offending, HIA-deficit and aggression will be significant in predicting violence.

Moffitt's Developmental Theory is also a key to my hypotheses in that this theory asserts childhood neuropsychological problems are a key component to antisocial behavior (Moffitt, 1993). I have previously reviewed the literature on HIA-deficit's relationship to violence. However, a dominant feature emerges when researching HIA-deficit's relationship in predicting delinquent behavior. This feature is the prevailing nature of HIA-deficit's comorbidity with other disorders. There seems to be a high-risk for HIA-deficit individuals to be diagnosed with other childhood disorders, some of which include aggression, oppositional defiant disorder, and conduct disorder (August et al., 1996; Barkley, DuPaul, et al., 1990). One study found those experiencing HIA-deficit to have three times the likelihood of comorbid oppositional defiant disorder, and four times more likely to have comorbid conduct disorder, compared to a control group of HIA-deficit teens (Barkley et al., 1991). Of particular significance to this study is the diagnosis of conduct disorder. A criteria of conduct disorder, aggressive behavior exhibited in children at the strongest levels also tended to be diagnosed with hyperactivity (Prinz, Connor, & Wilson, 1981). One study concluded that as much as three-fourths of children diagnosed with hyperactivity also experienced aggressive conduct disorder (Stewart et al., 1981). In another study it was found that of the 93 boys scoring the worst on measures of HIA-deficit, almost 64% scored equally bad on measures of conduct disorder (Farrington et al., 1990). Furthermore, there is evidence that those diagnosed with HIA-deficit not only self-report more conduct disorders, but also experience conduct disorder at an earlier age, compared to non-HIA diagnosed individuals (Thompson et al., 1996).

Since the frequent nature of comorbid HIA-deficit and aggression in delinquent individuals exists, a dichotomy of sorts with respect to predictability has emerged. On one

end of the spectrum are those researchers who are skeptical about the validity of HIA-deficit to independently predict antisocial behavior. These researchers' findings has led them to concluded that the presence of HIA-deficit in delinquent cases is solely contingent upon the existence of aggressive traits (Moffitt & Silva, 1988; Vitelli, 1996). It was found that as a predictor variable, aggression was the behavior that carried the greatest risk for continued problems for preschool boys with hyperactivity (Stormont-Spurgin & Zentall, 1995). Additionally, in a study of schoolchildren aged one through five, it was concluded those children characterized as not easy to handle by both mom and teacher were represented only among those children with combined aggression and HIA, versus HIA alone (Loeber et al., 1991).

Contradicting these researchers' findings are those studies that have found it is the continuation of HIA symptoms, and not aggressive behavior, that poses the greatest risk factor for the development of antisocial behavior into adolescence (Gittelman et al., 1985; Klein & Mannuzza, 1991; Thompson et al., 1996). It has been contended that early conduct disorder is not only associated, but also initiated by, the development of HIA-deficit (Lambert, 1988; Thompson et al., 1996). Likewise, should the original symptoms of hyperactivity alone be maintained through adolescence, the chances of developing conduct disorder were almost four-fold (Gittelman et al., 1985). Also supporting the predictability of HIA-deficit is the fact that only when combined with HIA-deficit characteristics was aggression found to be related to delinquency outcomes, as opposed to aggression without the HIA-deficit characteristics (Loeber et al., 1991).

The argument as to the validity of HIA-deficit as a risk factor is not limited strictly to juvenile delinquency. The significance of HIA-deficit as it relates to criminal behavior

lies in the fact that research has found the continuation of hyperactivity-impulsivity-inattention symptoms into young adulthood to be quite common (Weiss & Hechtman, 1986). Barkley (1990) found that up to 70 -80% of children with HIA-deficit are likely to display some of these symptoms into adulthood "to an extent inappropriate for their age group" (p.114). One study indicated the continuation of the full HIA-deficit syndrome in about one-third of the cases (Gittelman et al., 1985). Once again, however, the continuation of HIA-deficit again brings forth a contemptuous belief of HIA-deficit's inability to predict adult criminality. One study concluded that regardless of the continuation of HIA symptoms into adulthood, that HIA-deficit does not incur greater risk for law enforcement contact (Mannuzza et al., 1989). Therefore, some research has contended it is the presence of antisocial and aggressive behavior in young adulthood, and not HIA symptoms, that leads to criminal behavior (Magnussen, 1983; Mannuzza et al., 1989). Yet, the bulk of the research has determined that in both childhood and adolescence, the continuation of comorbid HIA-deficit and aggression heightens the probability of criminal outcomes into adulthood (Barkley et al., 1991; Lambert, 1988).

In conclusion, it seems that the "overlap" of HIA-deficit and aggressive conduct disorder has been found to be quite high. Because the early onset of HIA-deficit is consistent with Moffitt's Developmental Theory, I am led to believe that this study will also find frequent cases of comorbid HIA-deficit and aggressive behavior to appear early in life. In terms of a risk factor for delinquent and violent offending, there appears to be disagreement as to HIA-deficit's predictive capability towards offending. However, the present hypotheses will support past findings (Farrington et al., 1990; Moffitt, 1990), those findings being that the combination of hyperactivity, impulsivity, and inattention can

substantially predict criminal and antisocial behavior independently of childhood aggression. In addition, past research has found the attributes of hyperactivity-impulsivity-attention deficit, when accompanied with aggression, to be strong correlates of delinquency (Brier, 1995). Accordingly, it is my proposition that these two entities, when combined, will promote greater predictability towards juvenile and adult violence.

I am confident my hypotheses work well within Moffitt's Developmental Theory. With an emphasis on violent and chronic offending, as well as comorbid HIA-deficit and aggression, my thesis is well grounded in the roots of a widely researched and significant theory of antisocial behavior.

DATA

This paper looked at the variables of HIA-deficit and aggression as they related to violent and chronic offending. Once again, the hypotheses I tested were as follows:

Hypothesis #1: Those diagnosed with combined HIA-deficit and aggression will commit more violent crimes than those diagnosed with either HIA-deficit or aggression alone.

Hypothesis #2: Those diagnosed with combined HIA-deficit and aggression will be more likely to be chronic offenders than those diagnosed with either HIA-deficit or aggression alone.

For this study, the research that was used to test the hypotheses is the Cambridge Study of Delinquent Development by Dr. David Farrington (Farrington, 1994). This 20-year longitudinal study of 411 males began with surveys of the boys at age 8-9 years. Primarily a group of boys from the working class in London, this sample was drafted from the registers of the six state primary schools located near the researcher's office. Included with the 399 males from these six schools were 12 males from a local school for the educationally subnormal. The sample primarily comprised of white, urban, working class boys of British origin.

The interview process consisted of the boys interviewed by psychologists near the ages of 8, 10, and 14 years of age. The boys also tested in their school during these same years. In addition to the interviews and testing, the researcher also interviewed the parents of the boys, beginning at age 8 and continuing every year up to the boys' age of 14-15 years. Teacher questionnaires were also obtained about the boys behavior in school

at the ages of 8, 10, 12, and 14 years. Both the parent and teacher questionnaires were completed in great numbers, having 97.1 and 94 percent return rates, respectively.

The aim of the interview process, up to the age of 18 years, was to meet with every one of boys. The second set of interviewing, beginning at the age of 21, was intended for only those boys who had been convicted and a similar sized sample of unconvicted boys.

DEPENDENT VARIABLES

The dependent variables used in this analysis are violence and chronic offending. There are three items which measured the amount of violence taken at the age of 14, while the same items measured age 16 violence. Adult violence also incorporated four items similar to those measuring childhood and teenage violence. After these items were recoded into nominal level form they were put into a scale, with each scale measuring violence at their respective "stage" of development.

Measures of violence

The edition of the Cambridge Study in Delinquent Development to which I had access for use in this thesis did not include a list of specific offenses. Traditionally, research has suggested that the phrase "violent behavior" include such crimes as physical and sexual assault, robbery, and other such violent behavior. However, because of the unavailability of such crime definition, my operationalization of violence was not as specific. Nonetheless, I believe that the variables used to define my concept of violence in this thesis were definitionally sound. Table 1A in the Appendix lists the original item frequencies and their recoded scale frequencies for violence.

Violence at age 14 and 16:

Carried a weapon. At the ages of 14 and 16 the boy was asked if he had ever carried some kind of weapon like a knife or a cosh in case it was needed in a fight. If the boy has ever carried some kind of weapon for fighting purposes he was considered violent. The frequency of the boys carrying a knife at age 14 totaled 84, or 20.7% of the sample, while at age 16, a greater number of boys said they had carried a weapon (101, or 25.4%) in case it was needed in a fight.

Used a weapon. At the ages of 14 and 16 the boy was asked if he had ever used any kind of weapon (knife, cosh, razor, broken bottle) in a fight. This item was considered violent if the boy had ever used such a device in a fight. At age 14, a total of 49 (12.1%) boys had used a weapon at least once, while 68 (17.1%) said they had used a weapon in a fight at age 16.

Struggled/fought to get away from law official. As with the two previous questions, the boy was asked at the ages of 14 and 16 if he had ever struggled or fought to get away from a policeman. At both ages, a boy was considered violent in this respect if he had ever struggled or fought to get away from a policeman. The total number of boys in this category for age 14 and 16 was 28 (6.9%) and 51 (12.9%), respectively.

Adult Violence:

Involved in fights. This question was asked of the boy at the age of 18 years. This item was used to measure violence if the boy had ever gotten into a fight at all (excluding getting beaten up in a fight and not retaliating). A total of 145 (37.3%) of the boys had said they had gotten into at least one fight in the past year.

Started fights. Also asked at the age of 18, this question was used as a measure of adult violence if the boy had started a fight. In all, a total of 90 (23.1%) boys said they had started at least one fight.

Carried a weapon. Taken at age 18, this item was measured as the number of days the boy carried a weapon (knife, razor, cosh, hammer, gun, glasses, axes, for example). Only 32 (.8%) of the boys had carried a weapon at least one day in case it was needed in a fight.

Used a weapon. Also taken at age 18, this item was measured as the number of times the boy has used a weapon (as in above reference) in a fight. This item was used to measure violence not only if the boy had ever used a weapon in a fight, but also if he had ever used a weapon as a threatening device. In this sample, 32 (.8%) of the boys had used the weapon in a fight or threatened to use a weapon as least one time.

Measures of chronic offending

Much like violent offending, chronic offending was divided into three stages of life: offending at the age of 14, age 16, and adult offending covering the years 17-24. The two variables of age 14 (Youth at 14) offending and age 16 (Youth at 16) offending encompassed both self-reported delinquency and official convictions, while adult offending used official convictions as its sole measure of chronic offending. Table 2A in the Appendix lists the original item frequencies and their recoded scale frequencies for chronic offending.

Offending at age 14. Measured prior to and into age 14, this item included both measures of self-reported delinquency and official convictions. The self-reported delinquency item was measured at the age of 14. This item scored the number of different

of delinquent acts committed out of 38. Only those boys who self-reported "14 or more" were included as a measure of chronic offending. In addition to self-reported delinquency, official convictions were used to measure violence at this age. The item "Convicted 10-13" refers to convictions for offenses committed between the tenth and fourteenth birthday. If the boy had any convictions during this time span, he was considered a childhood offender. In this sample, a total number of 94 (23.2%) boys self-reported in the "14 or more" category, while 35 (8.5%) had at least one official conviction.

Offending at age 16. This variable measured the amount of delinquency committed at the age of 16. Similar to childhood offending, both self-reported delinquency and official convictions were used to gauge this level of chronic offending. The self-reported delinquency item was measured at the age of 16 and again measures the number of offenses committed out of 38 acts. Only those self-reporting "17 or more" delinquent acts were defined as part of the chronic offending group. Also measuring violence at age 16 was the item "Convicted 14-16," which refers to convictions after the fourteenth birthday up to the seventeenth birthday. A total of 92 (22.3%) of the boys self-reported delinquency in the "17 or more" category, while 74 (18%) had at least one official conviction.

Adult offending. This measure incorporated only official records, namely because there was no self-reported offending measure taken after the age of 18. Adult offending measured those acts which took place from the ages of 17-24. Thus, the items "Convicted 17-20" and "Convicted 21-24" were used to measure the amount of adult offending. The amount of boys reporting in the "Convicted 17-20" and "Convicted 21-24" items were 95 (23.6%) and 46 (11.6%), respectively.

INDEPENDENT VARIABLES

There were two independent variables analyzed in this research as well. Both HIA-deficit disorder and aggression were operationalized according to the items listed below. Table 3A in the Appendix lists the original item frequencies and their recoded scale frequencies for both HIA-deficit and aggression.

Measures of hyperactivity-impulsivity-attention deficit (HIA-deficit)

Because of the early initiation of HIA-deficit, it was important to measure this disorder early in childhood. The Cambridge Study collected information concerning measures of HIA-deficit from the boys at the ages of 8 and 10. Previously used in part to operationalize HIA-deficit from the Cambridge Study (Farrington et al., 1990), these three measures seemed to appropriately define what can be considered the symptoms of this disorder.

Inattention. At the ages of 8 and 10, the teacher rated the boy in terms of amount of concentration or restlessness the boy exhibited in the classroom. Used as a measure of the boy's inattention, this item contributed to the diagnosis of HIA-deficit if "yes" was the response from the teacher. At age 8, 135 (33.7%) boys were seen as has having a lack of concentration. At age 10, 145 (37.8%) boys were considered inattentive.

Hyperactivity. Based on a parent interview, this question gauged the amount of physical activity (such as climbing, traffic, exploring) by the boy at the age of 8. This item was used to measure the amount of hyperactivity only if the parent's response was "Takes many risks." Of the 379 responses, 75 (19.8%) of the parents believed their boy had taken many risks.

Impulsivity. At age eight, the boys were given four tests of psychomotor performance: the Porteus Q, Tapping, Spiral Maze Error, and Body Sway Ataxia. A sum of the scores on these tests were scored as a 1(0-1), 2(2), 3(3), 4(4), or a 5(5 or above). High scores on these tests were intended to reflect careless, clumsy, or impulsive behavior. Poor impulse control was also measured at age ten. Adding the raw scores of the Spiral Maze Errors combined, Porteus, Maze Q combined, and Tapping Score combined derived the psychomotor clumsiness score at this age. For my thesis, poor impulse control was considered applicable only if the boy scored a "5" (5 or above) at age 8 and a "high" at age 10. A total of 77 (18.9%) of age 8 boys were considered having poor impulse control, while 104 (25.3%) of the boys had poor impulse control at age 10.

Measures of aggression

My intention was to measure aggression not only in the childhood years, when HIA-deficit is traditionally diagnosed, but to measure aggression subsequent to a diagnosis of HIA-deficit as well. The reason for measuring aggression at different ages was to see if aggression stems out of HIA-deficit, as I have asserted previously. However, it was also important to measure aggression in the years to which children are developing their social networks and are controlling their environment much more than in their pre-adolescent years. Thus, in addition to measuring aggression at the ages of 12 and 13, levels of aggression were also operationalized at the ages of 14 and 15. The measure of aggression before age 14 was considered "Childhood Aggression," while aggression at age 14 and beyond characterized "Adolescent Aggression."

Aggressiveness. This item measuring aggressiveness combined 6 areas of behavior as rated by the teacher. These areas included disobedience, difficulty to discipline, unduly

rough, over-competitive, quarrelsome and aggressive, and resentful to criticism. Measured at the ages of 12 and 14, these behaviors were all rated on a scale of 1-3 (with a 3 being aggressive) and then summed. My concept of aggressive behavior included only those boys who scored as "most aggressive." A total of 86 (21.3%) boys scored as aggressive in childhood for this item (age 12), while 89 (23.5%) of the boys at age 14 were considered aggressive in adolescence.

CONTROL VARIABLES

The nature of this study will undoubtedly encompass many other social and psychological factors. The primary variables that one should consider in most studies dealing with humans are the variables of sex, age, race, and income. However, for this study it was not be necessary to control for these variables because of the nature of the sample. In the Cambridge Study, Dr. Farrington (1994) used a longitudinal sample composed strictly of boys, all of who were initially interviewed when they were 8 and 9 years old. These boys were also predominately all white Caucasian (only 12 boys had at least one parent of West Indian origin). Additionally, the sample was limited to those boys who were from urban, middle class neighborhoods, with the majority of the families classified as working class (93.7% of the families were located within the category of III, IV, or V on the registrar general's scale of occupational prestige). Because of the attributes of the sample, I did not need to control for the variables of age, sex, race, and income.

METHODS

There are five items that constitute the scale measuring HIA-deficit. Scales are not created to measure aggression because only one item is used to measure the amount of aggression each boy exhibits in childhood (ages 12-13) and adolescence (ages 14-15). Scales measuring violence and chronic offending are also created. Childhood violence and teenage violence are measured at the ages of 14 and 16, respectively, while adult offending is measured at the ages of 17-24 years. There are three items measuring childhood (Violence at 14) and teenage (Violence at 16) violence, while four items are used to measure violence at age 18 (adult violence). Chronic offending encompasses six items covering the span of childhood, teenage, and adult offending. Both official and self-reported delinquency are used to measure childhood (Youth at 14) and teenage offending (Youth at 16), while official convictions covering the years of 17-24 measure adult offending.

I am using bivariate correlation and analysis of variance (ANOVA) to assess the relationship between variables. Bivariate correlations not only test the significance of HIA-deficit and aggression on violence and chronic offending, but test the relationship between HIA-deficit and aggression as well. This examination is used to support or counteract findings of aggressive behavior originating from early HIA-deficit symptoms (Barkley, DuPaul, et al., 1990). Further tests involving analysis of variance allow for the

determination of how the interaction effect of HIA-deficit and aggression affects violent and chronic offending. These tests incorporating ANOVA are essentially the focus of my analysis: that HIA-deficit and aggression have an interactive effect on delinquency.

The first scale created is the HIA-deficit scale (Table 1). A reliability analysis on these five items shows fair reliability among the items (alpha = .5231), based on the standard accepted value of .6. A factor analysis is performed on the items to examine their strength in measuring HIA-deficit. This analysis shows that the five items are not very strong measures of the same concept. In particular, the items which measure a boy's hyperactivity (adventureness) and inattention (lack of concentration) do not seem to be strong measures of HIA-deficit. However, psychomotor impulsivity is very strong in measuring the concept. Both the reliability and factor analyses lend support to perceived difficulty in the operationalization of HIA-deficit. Although logically these items seem to be good measures of HIA-deficit, they appear to be borderline insignificant as to their reliability and strength in measuring the same factor. A frequency distribution of the HIA-deficit measures shows that very few of the boys test positive for three items (9.6%), four items (5.8%), or all five items (1.5%).

To help clarify the low number of HIA-deficit symptoms, a logical step might be to examine the items measuring "psychomotor clumsiness" and "lack of concentration." Because these two items are used as HIA-deficit measures at two different ages, there is the possibility of the behaviors manifesting themselves at different times. If this is the case, then there are issues of age-specific initiation and cessation points for impulsivity and/or concentration to be considered. A contingency table on the items finds them to be somewhat evenly distributed. For example, 5.1% of the boys test "yes" for impulsivity at

Table 1. Mean Measures of HIA-deficit and Aggression

Item	Mean	Range	Factor Loadings
HIA-deficit:			
Adventureness (age 8)	.1979	0 – 1	.443
Psychomotor Clumsiness (8)	.1887	0 – 1	.731
Lack of Concentration (8)	.3367	0 – 1	.528
Psychomotor Clumsiness (10)	.2530	0 – 1	.704
Lack of Concentration (10)	.3776	0 – 1	.506
HIA-deficit scale*:	**1.2653**	**0 – 5**	
No measures (valid %):	33.2%		
One measure:	32.7		
Two measures:	17.2		
Three measures:	9.6		
Four measures:	5.8		
Five measures:	1.5		
***Alpha = .5231**			
Aggression:			
Aggressive 12-13:	**.2129**	**0 – 1**	
Aggressive 14-15:	**.2354**	**0 – 1**	

age 8 but not age 10, while 11.8% score "yes" at age 10 but not age 8. A total of 13.7% measure "yes" at both age groups. Similar findings occur for the item "lack of concentration." Because the crosstabs indicate the distribution of these items to be evenly distributed, support is provided that these two symptoms are neither age specific when they start nor when they end. However, the findings do support measures of impulsivity and inattention which are more common at age 10 as compared to age 8.

Aggression is measured on two different levels for this analysis: childhood aggressive behavior at age 12-13 and adolescent aggression at age 14-15 (Table 1). These two items seem to be good measures of aggression because they encompass six different types of aggressive behaviors. Frequency distributions of aggression at each age group indicate that in both groups, over one-fifth of the sample has aggressive tendencies.

The Violence at age 14 scale consists of three items measured at the age of 14 (Table 2). Originally an item measuring the seriousness of fighting at age 14 was included as a measure of childhood violence. However, upon further analysis, it was found that this item does not measure violence as well as the other three items measured at age 14. A factor analysis indicates that fighting at 14 does not load well with the other items of carried a weapon, used a weapon, and struggled/fought with a police officer. The three items measuring Violence at 14 seem to be reliable, as indicated by an alpha of .6337. Furthermore, a factor analysis finds these items to be good measures of childhood violence, with loadings all above .6. A frequency distribution of Violence at 14 indicates that a fair amount of boys score on measures of childhood violence (Table 2).

Much like the previous items used for childhood violence, the items measuring Violence at 16 also appear to be reliable measures (alpha = .6197) (Table 2).

Table 2. Mean Measures of Violence

Item	Mean	Range	Factor Loadings
Violence at Age 14:			
Carried a weapon	.2074	0 – 1	.808
Used a weapon	.1210	0 – 1	.843
Struggled/fought with police	.0069	0 – 1	.620
Violence @ 14 scale*:	**.3975**	**0 – 3**	
No measures (valid %):	74.1%		
One measure:	15.3		
Two measures:	7.4		
Three measures:	3.2		
***Alpha = .6337**			
Violence at Age 16:			
Carried a weapon	.2544	0 – 1	.799
Used a weapon	.1713	0 – 1	.839
Struggled/fought with police	.1285	0 – 1	.610
Violence @ 16 scale*:	**.5542**	**0 – 3**	
No Measures:	65.2%		
One measure:	18.9		
Two measures:	11.1		
Three measures:	4.8		
***Alpha = .6197**			
Adult Violence:			
Involved in fights (age 18)	.3728	0 – 1	.823
Started fights	.2314	0 – 1	.826
Carried a weapon	.0082	0 – 1	.631
Used a weapon	.0082	0 – 1	.685
Violence @ Adult scale*:	**1.4424**	**0 – 4**	
No measures:	60.9%		
One measure:	12.9		
Two measures:	18.3		
Three measures:	4.4		
Four measures:	3.6		
***Alpha = .7269**			

Additionally, the items are strong factors of teenage violence, as indicated by a factor analysis. As expected, the frequency of violence at this age is higher than violence occurring at age fourteen.

The adult violence scale consists of 4 items. These items seem not only to be very reliable (alpha = .7269), but each item's factor loading is greater than .6 (Table 2). An interesting point to view with the adult violence scale is the high level of offending indicated by the mean score (1.4424). Although specific to violent offending, this finding seems contrary to Moffitt's Developmental Theory, which supports a definition of offending that occurs more in the teenage years compared to the adult years.

In conclusion, it appears that the items used in measuring the levels of violent offending are reliable and strong measures of the concept. A central focus of this conclusion lies with the items measuring violence. While the items of carried and used a weapon are good factors of childhood and teenage violence, this is not the case for adult violence. Conversely, fighting is a good measure of violence in adulthood, but not for measuring childhood violence. Thus, a conclusion to take from the analyses is that it appears violent children utilize weapons moreso than violent adults. This finding tends to agree with the traditional view of childhood violent offenders using weapons to settle disputes. Unaware of the dangers of such actions, children believe that carrying and using weapons will give them a "dangerous" and "powerful" demeanor not to be reckon with. The findings from the analyses do not necessarily echo such sentiments, but they do help to support the significant relationship of violent children and weapons.

Chronic offending incorporates the three measures of childhood, teenage, and adult offending. According to Moffitt's Developmental Theory, a life-course persistent offender will commit crime throughout the life-course, whereas an adolescent-limited offender will only commit crime in the adolescent or teenage years (Moffitt, 1994). Thus, we expect a life-course persistent individual to commit crime in the childhood, teenage, and adult years of their lives. For this analysis, a boy who scores "chronic" within the childhood, teenage, and adult offending periods is considered a life-course persistent offender.

A contingency table of offending in childhood (Youth at 14) finds that many boys self-reported a "high" amount of delinquency and no convictions. Conversely, few of the boys report a conviction and no "high" self-reported delinquency, and few score "high" on self-reports and have at least one conviction between the ages of 10-13. In all about a fourth (26.7%) of the boys report a high amount of self-reported delinquency and/or have at least one official conviction between the ages of 10 and 13 years.

As expected, teenage boys exhibit a greater percentage of offenders compared to childhood boys. However, the analysis does not find the teenage boys more willing to self-report delinquent behavior compared to boys aged 10-13 years. In the sample, only 13.6% of the teenage boys are "high" on self-report delinquency with no conviction (compared to 18.3% of childhood offenders). Although not willing to admit delinquent behavior, a frequency distribution of teenage offending indicates that nearly one-third (31.5%) of the boys measure chronic offending scores by having a high amount of self-reported delinquency and/or at least one conviction between the ages of 14 and 16 years.

The last item to measure chronic offending is adult offending (Adult 1). Unlike the previous two measures, adult offending only takes into account official convictions for the two age groups of 17-20 and 21-24 years. The analysis finds that more convictions took place in the earlier age group (17-20) compared to the later adult years (21-14). In total, a fairly large number of boys (26.0%) score as "chronic" in their adult years by having at least one conviction in either age group.

Once again, chronic offending is scaled to include all three life periods of offending (Table 3). Reliability analysis of the recoded items Youth at 14, Youth at 16 and Adult 1 finds the items are reliable (alpha = .6805). Further analysis shows that the items are compatible with one another, as a factor analysis of Chronic Offender scores the items all above .6. A final distribution of Chronic Offender indicates that 44 (11.5%) of the boys are considered offenders in all three periods (childhood, teenage, and adult), thus are considered persistent, or chronic offenders in this study.

Table 3. Mean Measures of Chronic Offending

Item	Mean	Range	Factor Loadings
Chronic Offender (Self-Reports):			
Self-report 14(valid %) (Score out of 38 acts)	.2321	0 – 1	
Self-report 16 (Score out of 38 items)	.2317	0 – 1	
Chronic Offender (Official Reports):			
Convictions 10 – 13	.0085	0 – 1	
Convictions 14 – 16	.1800	0 – 1	
Convictions 17 – 20	.2363	0 – 1	
Convictions 21 – 24	.1165	0 – 1	
Youth 14 (S-report 14 + Official 10/13)	.2667	0 – 1	.783
Youth 16 (S-report 16 + Official 14/16)	.3149	0 – 1	.833
Adult 1 (Official 17/20 + Official 21/24)	.2709	0 – 1	.725
Chronic Offender Scale*:	**.1155**	**0 – 1**	

*** Alpha = .6805**

ANALYSIS

The analyses for the two hypotheses incorporated the use of both bivariate correlation and analysis of variance (ANOVA) techniques to determine the relationship between the independent and dependent variables. The bivariate correlations were the first set of analyses performed on the variables[2]. The correlations indicated that virtually all of the variables were significant at the .01 level (Table 4). In view of the correlation analysis on HIA-deficit, the findings support that relationships do exist between this variable and both aggression variables. It is important to keep in mind that while this significant relationship does suggest an interaction exists between HIA-deficit and aggression, a correlation's significance does not explain an interaction's influence upon offending.

According to the correlations, HIA-deficit was significantly related to violence at the childhood, teenage, and adult stages of life. With correlations of .179, .218, and .216, respectively, the statistics point to the probability of HIA-deficit's impact being more significant in terms of violence as a boy got older, rather than a risk factor in childhood violence. In terms of chronic offending, HIA-deficit was also significantly related to all "stages" of offending. Much like violent offending, HIA-deficit appeared to have a stronger relationship to overall offending as a boy got older. The correlations performed

[2] Analyses not presented indicated that all relationships were positive.

Table 4. Bivariate Correlations of the Independent and Dependent Variables

Variable	HIA-deficit	Sign	Aggressive 12-13	Sign	Aggressive 14-15	Sign
HIA-deficit	1.000	....	.200*	.000	.159*	.005
Aggressive 12-13	.200*	.000	1.000	....	.328*	.000
Aggressive 14-15	.159*	.005	.328*	.000	1.000	....
Violence 14	.179*	.001	.132*	.008	.263*	.000
Violence 16	.218*	.000	.061	.226	.224*	.000
Adult Violence	.216*	.000	.192*	.000	.251*	.000
Youth 14	.169*	.002	.199*	.000	.326*	.000
Youth 16	.236*	.000	.198*	.000	.313*	.000
Adult 1	.227*	.000	.149*	.003	.221*	.000
Chronic offender	.232*	.000	.161*	.002	.263*	.000

* Correlation is significant at the .01 level (2-tailed).

with HIA-deficit were stronger with respect to teenage and adult offending than with childhood offending. The final correlation to discuss includes overall offending, in which case HIA-deficit did indeed have a significant correlation with chronic offending (.232).

The correlations performed on Aggression at 12-13 appeared to have more variance compared to HIA-deficit's relation to violence and chronic offending. Although a boy who has aggressive tendencies was correlated with early violence at 14 years and adult violence, this finding did not hold true for violence measured at age 16. In fact, of all the correlations performed, the childhood aggression and teenage violence correlation was the only non-significant relationship to exist (sig. = .226). This finding is even more intriguing considering fact that adolescent aggression was significantly related to teenage violence (sig. = .000). Lastly, it appears that childhood aggression had a declining effect on chronic offending. That is, aggression at the ages of 12-13 was more highly correlated with childhood and teenage offending than with chronic offending in adulthood.

Aggression at the ages 14-15 measured the amount of adolescent aggression. According to the correlations, aggression at this stage of life was more highly correlated with all ages of violent offending, and for the most part all ages of chronic offending, than either childhood aggression or HIA-deficit. The finding that a variable measured in adolescence has more correlation to delinquency compared to concepts measured in childhood tends to suggest that adolescent measures are stronger predictors of delinquency and violent offending.

In conclusion, the bivariate correlations indicated that HIA-deficit was significantly related to all measures of aggression, violence, and chronic offending. This finding helps to confirm previous literature that symptoms of HIA-deficit are significantly related to

violence and general delinquency. Further support was found between the relationship of aggression and delinquency. That is, on most levels, aggression was significantly related to violence and chronic offending. Finally, these findings provide evidence of a relationship existing between HIA-deficit and aggression. This relationship alone does not allow us to reject our null hypotheses. However, these findings do support my anticipation of the HIA-deficit and aggression interaction increasing violent and chronic offending. Support for the increased effect of comorbid HIA-deficit and aggression on violence and chronic offending was determined from the ANOVA tests.

There were two sets of ANOVA statistics run for the HIA-deficit and aggression variables. HIA-deficit and childhood aggression was run against the dependent variables of Violence at 14, Violence at 16, Adult Violence, and Chronic Offender. Likewise, HIA-deficit and adolescent aggression was run against these same dependent variables.

The analysis of variance did not indicate the same support as provided by our previous correlation analyses as to the significance of the independent variables (Table 5). While controlling for childhood aggression, HIA-deficit exhibited an F ratio that increased with each level of violent offending. In other words, HIA-deficit was less related to childhood violence (F = 1.742) compared to teenage (F = 2.280) and adult violence (F = 4.337). Furthermore, while HIA-deficit exhibited a significant relationship with teenage and adult violence, this was not the case with childhood violence (sig. = .125). Controlling for aggression, HIA-deficit also did not increase the amount of chronic offending (sig. = .085).

An overview of childhood aggression found that its significant relationship to violence and chronic offending was just the opposite of what HIA-deficit's findings

Table 5. Two-way ANOVA: HIA-deficit and Aggression at 12-13 Years

Variable	F	Significance	No. of Cases Included	Excluded
Violence 14			333	79
HIA-deficit	1.742	.125		
Aggressive 12/13	3.044	.029*		
2-Way Interaction	1.465	.123		
Violence 16			326	86
HIA-deficit	2.280	.047*		
Aggressive 12/13	.592	.621		
2-Way Interaction	1.878	.028*		
Adult Violence			322	90
HIA-deficit	4.337	.001*		
Aggressive 12/13	2.093	.101		
2-Way Interaction	1.770	.042*		
Chronic Offender			313	99
HIA-deficit	1.957	.085		
Aggressive 12/13	7.486	.000*		
2-Way Interaction	1.264	.229		

* Significant at the .05 level.

provided. For instance, while controlling for HIA-deficit, childhood aggression was significantly related to childhood violence (sig. = .029) and chronic offending (sig. = .000). Conversely, childhood aggression was not found to increase the amount of teenage violence (sig. = .621) or adult violence (sig. = .101). This last finding was similar to a correlation's finding in that ANOVA determined the relationship between childhood aggression and teenage violence to be non-significant.

In view of the previous findings, the important statistics now involved the two-way interaction effect of HIA-deficit and aggression on violent and chronic offending. As hypothesized, it appeared that the interaction of HIA-deficit and childhood aggression had a significant effect on several variables (Table 5). In terms of violence at age 16, the interaction of HIA-deficit and aggression increased the amount of teenage violence compared to HIA-deficit or aggression alone. This interaction effect was significant at the .05 level (sig. = .028). Another significant finding related to adult violence, in that the interaction of HIA-deficit and aggression was significantly related to violence at age 18 (sig. = .042). However, the interaction effect did not influence adult violence as much compared to HIA-deficit alone (sig. = .001). Finally, the influence of comorbid HIA-deficit and aggression was not found to significantly influence the amounts of childhood violence (sig. = .123) or chronic offending (sig. = .229). In fact, the influence of HIA-deficit and aggression alone increased the amount of chronic offending, and for the most part teenage violence, compared to the HIA-deficit and aggression interaction.

In summarizing the first set of ANOVA statistics, it is important to note several findings. First, it is interesting to note the reciprocal nature of the significance of HIA-deficit and aggression alone on violence and chronic offending. HIA-deficit alone was

found to be significantly related to teenage and adult violence, but not to childhood violence or chronic offending. Conversely, by itself aggression was significantly related to childhood violence and chronic offending, but not to teenage or adult violence. Another interesting finding related to the interaction effect of the variables. As mentioned before, singular HIA-deficit was found to be significantly related to both teenage and adult violence. Similarly, the interaction effect of HIA-deficit and childhood aggression was significantly related to teenage and adult violence. Furthermore, just as HIA-deficit alone was found to be insignificantly related to childhood violence and chronic offending, so too was the interaction of HIA-deficit and aggression insignificant. Needless to say, the ANOVA analysis conveyed just the opposite findings for childhood aggression. That is, when childhood aggression alone exhibited significance, the interaction did not, and vice versa.

The second set of ANOVA performed included the variables of HIA-deficit and adolescent aggression (Table 6). In this set we found that HIA-deficit alone did not appear to be statistically related to most dependent variables. In fact, when controlling for adolescent aggression, HIA-deficit was only found to be significant with adult violence (sig. = .002). On the other hand, adolescent aggression was very much an influential variable on delinquency. Aggression at ages 14-15 was significantly related to childhood violence (sig. = .001), teenage violence (sig. = .048), and chronic offending (sig. = .000), when controlling for HIA-deficit. Furthermore, in terms of adult violence, although insignificant, adolescent aggression still displayed a strong F ratio (2.139) almost worthy of a significant relationship (sig. = .096).

Table 6. Two-way ANOVA: HIA-deficit and Aggression at 14-15 Years

Variable	F	Significance	No. of Cases Included	Excluded
Violence 14			313	99
HIA-deficit	1.227	.296		
Aggressive 14/15	5.838	.001*		
2-Way Interaction	.902	.557		
Violence 16			305	107
HIA-deficit	1.713	.132		
Aggressive 14/15	2.672	.048*		
2-Way Interaction	1.792	.039*		
Adult Violence			301	111
HIA-deficit	3.916	.002*		
Aggressive 14/15	2.139	.096		
2-Way Interaction	1.566	.088		
Chronic Offender			295	117
HIA-deficit	1.722	.130		
Aggressive 14/15	7.807	.000*		
2-Way Interaction	.801	.668		

* Significant at the .05 level.

The two-way interaction effect of HIA-deficit and adolescent aggression did not appear to be as strong as the previous interaction effect of HIA-deficit and childhood aggression. The only interaction effect of HIA-deficit and aggression which increased delinquency more compared to HIA-deficit or aggression alone was with teenage violence (sig. = .039). This relationship to teenage violence was the only significant finding of the ANOVA analyses on comorbid HIA-deficit and adolescent aggression. The interaction effect of HIA-deficit and adolescent aggression was not significantly related to childhood violence (sig. = .557), adult violence (sig. = .088), or chronic offending (sig. = .668). In fact, on both measures of childhood violence and chronic offending, the effect of HIA-deficit and aggression alone increased the amount of delinquency moreso compared to comorbid HIA-deficit and adolescent aggression.

One interesting point to mention on ANOVA is the resemblance of the two sets of interaction analyses. Generally speaking, in both instances the interaction effect of HIA-deficit and aggression did not increase the amount of childhood violence or chronic offending compared to HIA-deficit or aggression alone. However, the interaction effect did increase the amount of teenage violence compared to HIA-deficit or aggression alone. In terms of adult violence, the analyses found that the interaction of HIA-deficit and childhood aggression, not adolescent aggression, was significantly related. Despite its significance in both analyses, the interaction of comorbid HIA-deficit and aggression did not increase the amount of adult violence compared to HIA-deficit alone. However, the HIA-deficit and aggression interaction did increase the amount of adult violence more than childhood or adolescent aggression alone.

DISCUSSION

It seems there exists some clear and consistent findings in these analyses. According to the bivariate correlations, HIA-deficit has a significant relationship with both measures of aggression, violence at all three age groups, and chronic offending. Likewise, adolescent aggression measures significant relationships on all measures of violent and chronic offending. The same holds true for childhood aggression, with the exception of teenage violence, in which case the relationship is not significant.

Perhaps the most influential finding of the correlation analysis relates to the aggression variables. It is my finding that on every correlation measure, adolescent aggression is more strongly related to all variables compared to childhood aggression. This is significant because the as one would anticipate, aggressive behavior developed early in childhood would advance and amplify in adolescence. Stated more simply, we anticipate adolescent aggression will have more influence upon a boy's life as he gets older. In my case, the analyses finds aggression's influence to exhibit itself through increased levels of violent and chronic offending.

Adolescent aggression is included in this analysis because of the potential for the findings just discussed. Perhaps the finding that adolescent aggression is more significantly related to violence and chronic offending compared to childhood aggression signifies the importance of "social networking." That is, as a boy becomes more

acclimated towards a group or network of friends, the influence of his aggressive behavior becomes heightened due to the socialization process with those friends. Social networks also introduce the concept of peer pressure. Thus, if a boy already has the characteristics of disobedience, is overly competitive, and has a tendency to play rough, the network system can be a venue for not only displaying those characteristics, but also compounding them. At any rate, these findings support a behavior that, in relation to violent and chronic offending, becomes more significant as a boy gets older.

According to ANOVA, there appears to be several conclusive statements about the comorbid HIA-deficit and aggression influence on violent behavior. One conclusion would be that those with comorbid HIA-deficit and aggression are more likely to commit teenage violence than those diagnosed with HIA-deficit or aggression alone. This conclusion is applicable to both measures of aggression. However, these same conclusions are not applicable to violence in the childhood years. I found that the interaction of HIA-deficit and aggression at both age groups does not increase the amount of childhood violence. In fact, the findings support definitions of aggression that influence violent offending in childhood considerable more than does its interaction effect with HIA-deficit. The same finding holds true for HIA-deficit. Compared to its counterpart comorbid HIA-deficit/adolescent aggression, HIA-deficit increases moreso the level of childhood violence.

In terms of adult violence, I am able to make several conclusions based on the analyses of the HIA-deficit and aggression interaction. In both analyses, the interaction effect of HIA-deficit and aggression does not increase the amount of adult violence more than HIA-deficit alone. However, the interaction in both analyses does lead to more adult

violence compared to childhood or adolescent aggression. Having many of the same results when compared to teenage violence, it appears that HIA-deficit has much more influence in adult violence compared to aggression.

Generally speaking, I conclude that those with comorbid HIA-deficit and aggression will commit more violence in their teenage years compared to those with HIA-deficit or aggression alone. Additionally, the relationship of comorbid HIA-deficit and aggression to childhood and adult violence lends support to a definition of comorbid HIA-deficit and aggression which may have more of an influence, in terms of violent offending, as a boy gets older. How much influence each variable has upon violent offending can be proposed from these findings as well. I find that comorbid HIA-deficit and childhood aggression is stronger upon adult violence compared to comorbid HIA-deficit and adolescent aggression. This finding lends support to aggression's decreasing influence in violent behavior. Because of this decreasing influence, it might be that a boy's aggression does not have as much influence on violent offending as does his level of HIA-deficit, regardless of his age. This proposal is backed from the finding that childhood aggression alone is not significant to adult offending, but its interaction with HIA-deficit is significant.

In terms of chronic offending, a more general conclusion can be made regarding the influence of comorbid HIA-deficit and aggression. I must fail to reject my null hypothesis and conclude that those diagnosed with comorbid HIA-deficit and aggression will not be more likely to be chronic, or life-course persistent offenders. Using both measures of aggression, the interaction effect with HIA-deficit does not incur a greater number of chronic offenders. Yet this conclusion does not necessarily contradict Moffitt's Developmental Theory, that an early neuropsychological deficit increases the likelihood of

someone becoming a life-course persistent offender. Rather, it provides evidence to the fact HIA-deficit may have more of an influence upon violent offending rather than general offending. However, in terms of aggression there are significant findings relating to Moffitt's Theory. Based on the findings from the correlations and from ANOVA, I conclude that those with childhood and adolescent aggression will increase the amount of chronic offending compared to comorbid HIA-deficit and aggression or HIA-deficit alone. Thus, is appears that those having fit my definition of aggression, that being a non-physical and verbally abusive type of behavior, are at risk for becoming chronic, or life-course persistent offenders.

One point must be made with regard to these findings on violent and chronic offending. The results on the influence of HIA-deficit and aggression on violence and chronic offending almost seem contradictory to previous understandings of general offending. It seems logical that those who are characterized as chronic offenders are usually prone towards more violent behavior. This study does not support such traditional understandings. The finding that comorbid HIA-deficit and aggression is significantly related to violence and not with chronic offending is definitely a unique concept.

In light of the findings, one point must be made with regards to accepting or rejecting my hypothetical statements. It appears that the evidence used in the decision to reject or fail to reject the null hypothesis, which states those diagnosed with comorbid HIA-deficit and aggression will *not* commit more violent crimes, is indecisive. Whereas one finding holds the null hypothesis to be rejected because a significant relationship exists between comorbid HIA-deficit and aggression upon teenage violence, other findings from the interaction of HIA-deficit and aggression on childhood violence indicate otherwise. In

addition, the decision revolving around the adult violence measure is unclear. HIA-deficit and childhood aggression interact significantly with adult violence, but HIA-deficit and adolescent aggression do not. Because of these findings, I am not able to make generalizing statements to the significance of comorbid HIA-deficit and aggression upon violence. I am only able to say that comorbid HIA-deficit and aggression increases teenage violent offending compared to HIA-deficit and aggression alone, and for the most part, is significantly related to adult violence.

In reaching these conclusions about our hypothetical statements, I should point to other directions researchers should take with regards to further studies on this topic. A first point to consider is the admittingly over-generalized hypothetical statements. It is my contention that this study's conclusions are better suited towards more distinct points of interest, rather than overall violent offending. This study also exposes the difficulty in operationalizing a disorder such as HIA-deficit. For example, limitations in the data do not permit for a "full-symptom" diagnosis of HIA-deficit at several age groups. The current findings not only suggest other operationalization techniques for HIA-deficit, but more importantly suggest that future studies of violence should include "full-symptom" diagnoses of HIA-deficit at both an early and late childhood age. Another direction to consider involves the operationalization of a chronic offender. The rate of chronic offending used in this study was 11.5 percent. Previous findings have supported a chronic offending rate around 5-6% (Moffitt, 1993; Wolfgang, 1972). It could be that the use of both self-reported delinquency and official convictions in measuring chronic offending over-reported the actual amount of offending. There is no question as to the dangers in using self-reported questionnaires to gauge delinquent activity, those dangers being the

under- and over-reporting of delinquent behavior. However, in some instances, as it was in this case, the necessity to include more cases was imperative. Thus, in measuring the childhood and teenage offender both self-reported offending and official convictions were used. These items were not combined, however, to measure adult offending. This is no doubt a limitation to my study. Further studies measuring levels of offending should be consistent with respect to the items used in measuring a variable.

What do my findings mean for practitioners who treat the young male offender? If we consider the fact that anywhere from 30 to 80 percent of children with HIA-deficit will continue to display the symptoms into adulthood (Barkley, 1990; Weiss and Hechtman, 1986), then we should be greatly concerned with future violence and offending from this population. Since HIA-deficit appears to manifest itself more in teenage and adult violence, it is sensible to conclude that any treatment for this neuropsychological deficit should extend further than the current repercussions of today's juvenile offending. For example, if practitioners are to initiate treatment based on these findings, then those delinquents diagnosed with HIA-deficit will receive regular check-ups from social workers and medical professionals throughout adolescence. Furthermore, should the childhood HIA-deficit symptoms continue to persist, as some researchers have found, then follow-up checks should carry through into adulthood.

In terms of chronic offending, the findings conclude that we should be more aware of those with aggressive behavior than with HIA-deficit. According to the findings, aggressive behavior needs to be examined at a relatively early age (12 years). The previous analyses point to the existence of aggressive traits in early childhood, which are significantly related to early violence. Furthermore, the analyses also point to the

significant contribution of aggression to chronic offending. Because of these findings, it is my contention that those with aggressive characteristics will be more likely to be committing crimes throughout their lifetimes. For this type of delinquency, I would suggest that treatments involving aggressive boys must involve behavior modification. Modification is necessary because the assumption is that anyone involved in a persistent cycle of offending needs to be taught how to break that cycle. In this case, the aggressive individual will need to learn how to control his temper and verbal aggression in a conflict situation. The findings also indicate that any type of treatment for childhood aggression should notice the significance of adolescent aggression on all types of offending. Thus, treatments for childhood aggression should also incorporate follow-ups through adolescence.

With that in mind, it is my contention that treating those with HIA-deficit will be much more difficult than treating aggressive delinquents. My basis for this assumption is that violence, for the most part, is an unpredictable act involving no premeditated thinking. Conversely, an individual who is a chronic offender makes a living through thought-out methods of illegitimate opportunity. In other words, this type of behavior is predictable in the sense we can assume the individual will continue to obtain privileges through illegal means. In my view, it is much easier to change the predictable chronic offender than the unpredictable violent offender. Include the fact that the violent offender has a neuropsychological deficit and the potential for treatment will dissipate even more.

In conclusion, my findings support a diagnosis of comorbid HIA-deficit and aggression which does not increase the amount of chronic offending, but does increase the amount of teenage violence, compared to either HIA-deficit or aggression alone.

Whatever the direction taken in researching these topics, my conclusions point to several objectives. First, the difficulty in operationalizing HIA-deficit and aggression encourages more research in these areas, particularly in the childhood years. Second, an objective should be made to study the effects of longitudinal treatment for those with HIA-deficit and aggression. Finally, the influence of comorbid HIA-deficit and aggression upon violence should also be viewed in relation to other variables which might have an influence in the late childhood, early teenage years of development.

APPENDIX

Table 1A: Violence Classifications and Codings

Variable	Code (and frequencies)	Recode (and frequencies)
Violence 14:		
Carried a weapon (age 14)	0 = never (79.3%) 1 = once/twice (4.0) 2 = sometimes (8.9) 3 = frequently (7.9)	0 = no (79.3) 1 = yes (20.7)
Used a weapon (14)	0 = never (87.9) 1 = once/twice (4.4) 2 = sometimes (5.2) 3 = frequently (2.5)	0 = no (87.9) 1 = yes (12.1)
Struggled/fought w/police (14)	0 = never (93.1) 1 = once/twice (4.0) 2 = sometimes (2.2) 3 = frequently (0.7)	0 = no (93.1) 1 = yes (6.9)
Violence 16:		
Carried a weapon (16) *	1 = no (74.6) 2 = yes (25.4)	
Used a weapon (16) *	1 = no (82.9) 2 = yes (17.1)	
Struggled/fought w/police (16) *	1 = no (87.2) 2 = yes (12.8)	
Adult Violence:		
Involved in fights (18)	0 = no fights (60.7) 1 = 1 fight (13.6) 2 = 2 fights (10.5) . 60 = 60 fights (0.3) Beat up-no retaliation (2.1)	0 = no (62.7) 1 = yes (37.3)
Started fights (18)	0 = no fights (76.9) 1 = 1 fight (13.6) 2 = 2 fights (3.3) . 25 = 25 fights (0.5)	0 = no (76.9) 1 = yes (23.1)

* Recoding was not necessary.

Table 1A (cont'd):

Adult Violence:	**Code** (and frequencies)	**Recode** (and frequencies)
Carried a weapon (18)	0 = never (91.8%) 1 = 1 day (2.1) 2 = 2 days (0.5) . 365 = 365 days (0.3)	0 = no (91.8) 1 = yes (8.2)
Used a weapon (18)	0 = never (91.8) 1 = 1 time (3.3) 2 = 2 times (0.8) . 25 = 25 times (0.3) Only to threaten/not hurt = (2.6)	0 = no (91.8) 1 = yes (8.2)
Final Variables	**Items** (and frequencies)	**Measures** (and frequencies)
Violence at 14	Carried a weapon (20.7%) Used a weapon (12.1) Struggled/fought w/police (6.9)	0 = no measures (74.1 %) 1 = 1 measure (15.3) 2 = 2 measures (7.4) 3 = 3 measures (3.2)
Violence at 16	Carried a weapon (25.4) Used a weapon (17.1) Struggled/fought w/police (12.8)	0 = 0 measures (65.2) 1 = 1 measure (18.9) 2 = 2 measures (11.1) 3 = 3 measures (4.8)
Adult Violence	Involved in fights (37.3) Started fights (23.1) Carried a weapon (8.2) Used a weapon (8.2)	0 = 0 measures (60.9) 1 = 1 measure (12.9) 2 = 2 measures (18.3) 3 = 3 measures (4.4) 4 = 4 measures (3.6)

Table 2A. Chronic Offending Classification and Codings

Variable	Code (and frequencies)	Recode (and frequencies)
Convicted 10/13 *	1 = no (91.5%) 2 = yes (8.5)	
Convicted 14/16 *	1 = no (82.0) 2 = yes (18.0)	
Convicted 17/20 *	1 = no (76.4) 2 = yes (23.6)	
Convicted 21/24 *	1 = no (88.4) 2 = yes (11.6)	
Self-report delinquency (age 14)	1 = 0-5 (26.2) 2 = 6-8 (24.0) 3 = 9-13 (26.7) 4 = 14 or more (23.2)	0 = no (76.8%) 1 = yes (23.2)
Self-report delinquency (16)	1 = 0-9 (28.2) 2 = 10-12 (26.2) 3 = 13-16 (22.4) 4 = 17 or more (23.2)	0 = no (76.8) 1 = yes (23.2)
Final Variables	**Items** (and frequencies)	**Measures** (and frequencies)
Youth at 14	Self-reported (23.2%) Convicted 10/13 (8.5)	0 = no (73.3%) 1 = yes (26.7)
Youth at 16	Self-reported (23.2) Convicted 14/16 (18.0)	0 = no (68.5) 1 = yes (31.5)
Adult offending 17-24	Convicted 17-20 (23.6) Convicted 21-24 (11.6)	0 = no (72.9) 1 = yes (27.1)
Chronic Offender	Youth at 14 (26.7) Youth at 16 (31.5) Adult Offending (27.1)	0 = no (88.5) 1 = yes (11.5)

* Recoding was not necessary.

Table 3A: Independent Variable Classifications and Codings

Variable	Coding (and frequencies)	Recode (and frequencies)
HIA-deficit:		
Adventureness (age 8)	1 = cautious (16.1%) 2 = average (64.1) 3 = many risks (19.8)	0 = no (80.2%) 1 = yes (19.8)
Psychomotor Clumsiness (8)	1 = 0-1 score (18.6) 2 = 2 (19.1) 3 = 3 (25.0) 4 = 4 (18.4) 5 = 5 or more (18.9)	0 = no (81.1) 1 = yes (18.9)
Concentration (8) *	1 = no (66.3) 2 = yes (33.7)	
Psychomotor Clumsiness (10)	1 = low (25.3) 2 = low average (24.6) 3 = high average (24.8) 4 = high (25.3)	0 = no (74.7) 1 = yes (25.3)
Concentration (10) *	1 = no (62.2) 2 = yes (37.8)	
Final Variables	**Items** (and frequencies)	**Measures** (and frequencies)
HIA-deficit	Adventureness (19.8%) Psychomotor 8 (18.9) Concentration 8 (33.7) Psychomotor 10 (25.3) Concentration 10 (37.8)	0 = no measures (33.2%) 1 = 1 measure (32.7) 2 = 2 measures (17.2) 3 = 3 measures (9.6) 4 = 4 measures (5.8) 5 = 5 measures (1.5)
Aggression 12-13 Years *	1 = 6-9 – least aggressive (18.6) 2 = 10 (39.9) 3 = 11 (20.3) 4 = 12 + - most aggressive (21.3)	
Aggression 14-15 Years *	1 = 6-9 – least aggressive (19.8) 2 = 10 (40.2) 3 = 11 (16.4) 4 = 12 + - most aggressive (23.5)	

* Recoding was not necessary.

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