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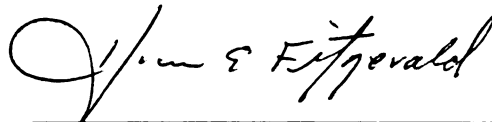
FIRST ALCOHOL USE AND THE DEVELOPMENT OF
ANTISOCIAL BEHAVIOR PROBLEMS FROM PRESCHOOL
THROUGH EARLY ADOLESCENCE

presented by

RONI MAYZER

has been accepted towards fulfillment
of the requirements for the

Doctoral degree in Psychology & Criminal Justice



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**FIRST ALCOHOL USE AND THE DEVELOPMENT OF ANTISOCIAL BEHAVIOR
PROBLEMS FROM PRESCHOOL THROUGH EARLY ADOLESCENCE**

By

Roni Mayzer

A DISSERTATION

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ABSTRACT

FIRST ALCOHOL USE AND THE DEVELOPMENT OF ANTISOCIAL BEHAVIOR PROBLEMS FROM PRESCHOOL THROUGH EARLY ADOLESCENCE

By

Roni Mayzer

Despite the fact that early Age of First Drink (AFD) is a robust predictor of negative adult outcomes, few researchers have traced its occurrence to childhood antecedents in order to fully delineate the longitudinal pathway leading to as well as from this risk marker. The current study examined the relationship between early AFD and antisocial behavior from preschool (ages 3-5) through early adolescence (ages 12-14), hypothesizing that early drinking is associated with a long-term pattern of aggression and delinquent behavior. It also attempted to explain the development of these behavior problems, using risk factors from preschool (temperament, parental psychopathology) and middle childhood (academic achievement, self-esteem, social problems, family environment). Participants were 220 male children and their parents in the Michigan Longitudinal Study. Early AFD was defined as having had a first drink by 12-14 years of age.

Results showed that early AFD was associated with delinquent behavior, not aggression. Compared to those who never tried alcohol, early drinkers were rated as more delinquent at ages 3-5, 6-8, and 12-14 – with a temporary convergence at 9-11. There were also group differences in the developmental trajectories for delinquent behavior. Particularly interesting was the stronger direct effect of preschool predispositions on adolescents' delinquent behavior for the alcohol-initiated, supporting the idea of hard-continuity in problem behavior for early AFD individuals. In a

simplified model, children were classified as high-high, low-low, low-high, or high-low on each type of antisocial behavior at 3-5 and 12-14, respectively. Early drinking was disproportionately likely for those classified as high-high in delinquent behavior (and disproportionately uncommon for those classified as low-low).

Risk profiles during preschool and middle childhood were most favorable for the low-low groups, least favorable for the high-high groups, and intermediate for the change groups (i.e., low-high and high-low). Earlier antisocial behaviors were the best predictors of later antisocial behaviors. Whereas delinquent behavior was self-perpetuated, adolescent aggression could also be uniquely explained by personal (e.g., self-esteem) and contextual (e.g., family cohesion) characteristics during middle childhood. In conclusion, there are important reasons to look at aggression and delinquent behavior as often co-morbid but distinct – conceptually and developmentally.

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CHAPTER 1 INTRODUCTION

Identification of the Problem

Although drinking during adolescence is not uncommon (and generally not a precursor to future abuse or dependence), early onset of alcohol use may be a marker for more serious developmental outcomes. Indeed, there is a substantial body of literature demonstrating heightened risk for alcohol-related difficulties with an early age of first drink (Grant & Dawson, 1997; Gruber, DiClemente, Anderson, & Lodico, 1996; McGue, Iacono, Legrand, Malone, & Elkins, 2001b; Prescott & Kendler, 1999; Schukit & Russell, 1983). An important question in trying to understand the developmental chain of events underlying this association is whether early drinking onset reflects a longitudinal pattern of behavior traceable from adolescence to younger ages. Considerable evidence exists that a hard-continuity, risk-aggregation model may describe development for a small subset of troubled individuals (Fitzgerald, Puttler, Mun, & Zucker, 2000; Zucker, Fitzgerald, & Moses, 1995; Moffitt, 1993, 1997).

Conceptual Framework

According to the hard-continuity paradigm (detailed in Table 1), risky characteristics and risky environments from infancy onward funnel the individual into a pathway of risk aggregation and cumulative disadvantage – leading to a greater likelihood for early onset of alcohol and other drug use, delinquency, and depression in adolescence as well as substance abuse and other psychopathology throughout the adult years (Fitzgerald et al., 2000). Negative outcomes are not sudden but emergent, or epigenetic, with dynamic structures that gain momentum as they organize over

developmental time (Zucker et al., 1995). Key etiological influences are already in place well before drinking onset (Zucker et al., 1995).

Table 1

Hypothesized Developmental Pathway of Enhanced Risk for Substance Abuse and Co-morbid Psychopathology

<u>Age Period</u>	<u>Possible Expression</u>
Core Diathesis	Genetically mediated neural deficits
Prenatal	Exposure to alcohol
Infancy	Difficult temperament
Preschool	Externalizing behavior problems, Social withdrawal,
Childhood	Behavior problems, Oppositional behavior, Impulsivity, Social withdrawal, Poor school performance
Adolescence	Earlier onset of alcohol and other drug involvement, Heavier alcohol and other drug use problems, Delinquency, Depression
Adulthood	Antisocial personality disorder, Mood disorder, Substance abuse disorder

Note. Adapted with permission from Fitzgerald, H. E., Puttler, L. I., Mun, E-Y., & Zucker, R. A. (2000). Prenatal and postnatal exposure to parental alcohol use and abuse. In J. D. Osofsky & H. E. Fitzgerald (Eds.). WAIMH handbook of infant mental health: Volume 4. Infant mental health in groups at high risk (pp. 123-159). New York: Wiley.

The concept of developmental contextualism, encompassed by Developmental Systems Theory (DST; Ford & Lerner, 1992; see also Dixon & Lerner, 1992; Lerner, 1980; Lerner, Castellino, Terry, Villarruel, & McKinney 1995), can be used as a framework for understanding the evolution of behavior problems. Of particular importance is the idea of multiple influences on human development, from many levels of human ecology (see also Bronfenbrenner, 1977). These influences range from genes to culture, all inclusive (Ford & Lerner, 1992). Specifically,

There is no single cause of the individual's functioning or development. Neither within-person variables (e.g., biological or psychological ones), nor interpersonal variables (such as peer group or personal relations), nor extrapersonal variables (such as institutional or environmental ones) are sufficient in and of themselves.

(Dixon & Lerner, 1992, p. 37-8)

The fact that the developing individual is embedded in an ecological, multivariate, multilevel constellation of influences means that research designs need to look at contextual risk factors as well as individual predispositions.

There is a core genetic diathesis as well as contextual influences that predispose towards negative developmental outcomes, but these are also nested within one another (Zucker et al., 1995). Risky personal characteristics and behavior typically occur within a socialization context that reinforces and models those attributes. Children with behavior problems are frequently reared in a sub-optimal environment characterized by high parental psychopathology (Mun, Fitzgerald, von Eye, Puttler, & Zucker., 2001; Moffitt, 1993). Their parents are the most likely to be the least capable of dealing with a difficult child, and may in fact share many of the same characteristics as their offspring

(e.g., in terms of temperament, personality, or cognitive ability) because of the disproportionate prevalence of biopsychosocial deficits in disadvantaged homes (Moffitt, 1993).

A second contribution made by DST is the idea of dynamic interaction within and between all levels of the organization that makes up human life: “the structure or pattern of relations among these levels of analysis produces the individual’s behaviors; and changes in the form (the configuration) of these relations produce developmental change” (Dixon & Lerner, 1992, p. 37-8). Person and context interact in a reciprocal and transforming pattern of exchange (Ford & Lerner, 1992). Many influences are naturally co-occurring anyway, or related to one another because individuals elicit and seek out certain environmental responses or contexts (Wachs, 1996). Individuals serve as stimuli in their own development (Lerner, 1985), with transactional feedback from the interpersonal context that can exacerbate initial tendencies (Moffitt, 1993) – as might be the case if parents respond with hostility or neglect. Finally, each moment of development exists not in isolation but as part of an emerging temporal history (e.g., Lerner, 1980) shaped by past events and future aspirations.

This person-in-context approach to developmental outcomes with its appreciation of dynamic interaction (see Ford & Lerner, 1992; Lerner, 1985) emphasizes the fact that biopsychosocial and environmental characteristics are inter-related in such a way as to intensify risk. In fact, this aggregation and exacerbation of risk across a variety of sources serves as a scaffold for development over time – becoming a crystallized maintenance-structure for continuity in problem behaviors (Zucker et al., 1995).

A key point of this argument is that, although there is always the potential for change, individuals who are chronically exposed to developmental risk factors find it increasingly difficult to recover. The concept of experiential canalization (Gottlieb, 1991c) refers to hard-continuity that takes place not through the inheritance of species-specific acquired characteristics (cf. Waddington, 1942), apropos of the original conceptualization of “canalization,” but because of immediate influences. Canalization at the behavioral level – driven by internal (e.g., genetic) or external (e.g., environmental) experiences – can constrain behavior over time as individuals are steered towards or remain in an epigenetic rut. From this vantage point, “canalization is a narrowing of responsiveness as a consequence of experience” (Gottlieb, 2001, p. 4). Human speech perception provides one example: “During the first year of postnatal life, human infants are responsive to the universal range of phonemes that occur in all languages. However, by the end of their first year, they are responsive to only the phonemes of their native language” (Gottlieb, 2001, p. 5). With regard to developmental psychopathology, canalization refers to the rutting of *maladaptation* over time (e.g., Zucker et al., 1995).

Another implication of the risk-cumulative, nested model (Zucker et al., 1995) is that early drinking is likely to be associated with other behavior problems and experiences that characterize the personal and ecological (e.g., family) landscape of concurrent psychological influence. Jessor and Jessor (1975, 1977) were among the first to argue – according to what is now known as Problem Behavior Theory – that multiple indicators of behavioral deviance (e.g., delinquency, substance use, smoking, sexual activity, school failure) co-vary in adolescence and are so interrelated as to comprise a syndrome, constellation, or class of conduct. They also suggested that substance use

occurring before age-based population norms could be described as early transition-marking behavior and predicted from an extant pattern of psychosocial characteristics called “transition-proneness.” For example, the order in which high school students began to drink was found to be in parallel (and inversely related) to their ranking on academic achievement values (Jessor & Jessor, 1975, 1977).

Although Jessor and Jessor (1975, 1977) were early in articulating the theme of co-occurring problem behaviors, the work of other researchers supports their perspective. For example, Elliott, Huizinga, and Menard (1989) have described “multiple problem youth” and the joint occurrence of delinquency, substance use, and mental health problems. Similarly, Dryfoos (1990, 1998) has found that there is a great deal of overlap among four risk behaviors: substance use, sexual activity, delinquency, and school problems.

In addition to their co-occurrences, there are likely to be etiological connections between these problem behaviors. For example, researchers have found that early substance use increases the likelihood of precocious transitions to adult statuses such as dropping out of school and parenthood (Krohn, Lizotte, & Perez, 1997; Perez McCluskey, Krohn, Lizotte, & Rodriguez, 2002), and that – in turn – these precocious transitions contribute to later substance use (Krohn et al., 1997).

This linkage of multiple problem behaviors may be at the core of what can be described as an antisocial behavior pathway into early onset of alcohol use. Just as substance use in adolescence is related to a cascading pattern of other problem behaviors and substance use during young adulthood (Krohn et al., 1997; Perez McCluskey et al., 2002), behavior problems from preschool onward may be precipitating factors.

Recalling the hard-continuity model presented in Table 1, the premise here is that early drinking is but one marker on the developmental path followed by children who demonstrate persistent difficulties with aggression and delinquent behavior.

Purpose of the Study

The current study seeks to advance research on Age of First Drink (AFD) and continuity in antisocial behavior, tying the two together. There are several weaknesses in the extant literature that will be addressed. First, although numerous researchers have examined the relative contributions of ecological, social, and individual factors to the initiation and escalation of alcohol use, considerably less attention has been paid to AFD as a risk marker. This is true despite the fact that the relationship between AFD and alcoholic outcomes is quite robust (e.g., Grant & Dawson, 1997). Early onset of regular drinking patterns is obviously more serious, but identification of at-risk individuals before drinking habits become ingrained would increase the likelihood of success for intervention and prevention programs. Likewise, the argument can be made that intervention and prevention programs would benefit from a better understanding of the risk factors that predispose very young children towards an early AFD. Unfortunately, studies on AFD have primarily focused on downstream sequelae (e.g., drunkenness or alcoholic diagnosis) rather than precursors to drinking onset. Is early AFD also a robust indicator for early behavioral difficulties? Proving this would strengthen the argument that there is an underlying risk trajectory for alcohol problems that can be observed even before alcohol use becomes part of the behavioral repertoire.

Second and relatedly, although continuity in antisocial behavior has been associated with greater risk for alcohol-related outcomes, samples are typically first

observed during middle childhood or early adolescence. Because behavior is emergent, there are likely to be important preschool substrates. It is therefore important to look at behavioral difficulties in very young children, and to connect this with later behavior problems as well as drinking onset.

Third, research on antisocial behavior has focused on continuity with relatively less attention accorded to discontinuity. Although hard-continuity may be the stronger trend, behavior is not predetermined. Risk is a process, not a stable trait or state. The dynamic interaction that exists between individuals and their ecological contexts can facilitate change. Risk adheres to the principles of probabilistic epigenesis. Epigenesis is a developmental unfolding of events leading to increased complexity and organization with the emergence of new structures and functions during the course of ontogeny (Gottlieb, 1991c; Kitchener, 1978), and the fact that it is probabilistic means that continuity cannot be assumed. As Werner (1986, p. 18) stated so strikingly, “risk factors are not black boxes into which one fits children to be neatly labeled and safely stored away. They are probability statements, the odds of a gamble whose stakes change with time and place.” There are many factors and processes at play, and it is the interplay between them all – not just risk factors in isolation – that shapes developmental trajectories. Resilience – the “dynamic process encompassing positive adaptation within the context of significant adversity” (Luthar, Cicchetti, and Becker, 2000b, p. 543) – matters too. Moreover, this interplay is constant: Person and context are transformed, minutely or dramatically, by their ongoing relationship with one another. The potential for change (i.e., plasticity) means that the balance between risk and resiliency isn’t immutable. Risk can increase or decrease; resiliency can be bolstered or weakened. In

turn, behavioral difficulties may remit or escalate during childhood. Prevention and intervention programs can capitalize on a better understanding of the natural processes that inhibit or bring about change.

To address these concerns, the current study will look at behavioral difficulties upstream from serious alcohol-specific outcomes, beginning with preschool. It will also recognize that – although continuity in aggression and delinquent behavior may be the stronger trend with respect to predicting drinking onset – discontinuity is also possible. The purposes of the current study are four-fold. Specifically, this research hopes to demonstrate that 1) there are marked differences in antisocial behavior between early drinkers and non-drinkers by early adolescence that are in evidence from preschool, onward; 2) high and low antisocial behavior problem groups in preschool and early adolescence (high-high, low-low, low-high, high-low) constitute a useful classification scheme, with strong continuity (high-high and low-low) as the main characteristic distinguishing drinking onset from onset avoidance; 3) discontinuity patterns reflect a less common but intermediate risk profile, generally; and 4) antisocial behavior is influenced by risk and promotive factors during middle childhood.

CHAPTER 2

LITERATURE REVIEW

Early Onset of Alcohol Use

“Early” drinking onset is typically defined as alcohol use before age 14 (e.g., McGue et al., 2001b), but occasionally as young as 10-12 years of age (Gruber et al., 1996). In general, the preadolescent period seems to be a particularly useful boundary for distinguishing problematic drinking (i.e., early initiation) from normative drinking behavior during the teenage years (i.e., during mid-to-late adolescence) – and for targeting those at the most elevated risk for alcoholism.

Alcohol consumption and alcohol-related difficulties in adulthood tend to vary inversely with Age of First Drink (AFD), even in well-functioning university-based samples (Schuckit & Russell, 1983). Among more than 27,000 ever-drinking adults, Grant and Dawson (1997) found that the rate of lifetime alcohol dependence was four times higher for those who started to drink by age 14 compared to those who started to drink after age 20. Other research studies (e.g., McGue et al., 2001b; Prescott & Kendler, 1999) have reported similar findings. Early AFD has also been associated with disinhibitory behaviors and diagnoses related to nicotine, drug use, and psychiatric disorders (McGue et al., 2001b); as well as injuries, violence, absenteeism from school or work, and drunk driving (Gruber et al., 1996). It should be noted that although having a first drink can be distinguished from “drinking onset” per se, which implies a regular pattern of alcohol use, it is still a very robust indication of alcoholic risk.

Like Jessor and Jessor (1975, 1977), some researchers have used the idea of a “syndrome” of problem behavior propensities to explain the relationship between onset of drinking and a later alcoholism diagnosis. Specifically, early onset of drinking and

alcoholism may be common symptoms of the same underlying vulnerability (McGue et al., 2001b; Prescott & Kendler, 1999). In a study of nearly 9000 adult twins, Prescott and Kendler (1999; see also McGue, Iacono, Legrand, & Elkins, 2001a) found that genetic factors and a shared family environment explained both. This supports the linkage of early drinking with risk for later alcoholism as well as the person-in-context approach to understanding risk.

Early drinking, for example, does not occur in a vacuum but, rather, is related to a number of personal and contextual factors. Marked differences in the “matrix of social relationships” have been described for adolescent drinkers versus non-drinkers (Margulies, Kessler, & Kandel, 1977, p. 898). Modeling and the quality of interpersonal relationships are both recognized as important correlates of drinking behavior (Margulies, Kessler, & Kandel, 1977). Exposure to alcohol use by parents and peers has been associated with alcohol initiation (Ellickson & Hays, 1991; Felton, Parsons, Ward, Pate, Saunders, Dowda, & Trost., 1999). Onset of regular drinking can be predicted from the density of alcoholism within families (Hill & Yuan, 1999). Substance use has been associated with concurrent levels of family conflict as well (Hops, Davis, & Lewin, 1999) – with initiation less likely among adolescents from highly organized families (Hussong & Chassin, 1997) and positive parent-child relationships (Cohen, Richardson, & LaBree, 1994). This may reflect amplified tensions/disruptions during adolescence in families with problem children, or hard-continuity at the ecological level in terms of risky individuals nested in risky environments over time. In general, although cognitions and expectancies become more central to the development of regular and continued drinking

patterns, *onset* appears to be mediated by social factors and social processes (Ellickson & Hays, 1991).

Personal characteristics also play an important role. Personality and temperament, in particular, have been linked to adolescent substance use. Of relevance are traits such as high activity level and less positive mood (Wills, DuHamel, & Vaccaro, 1995), undercontrol (Caspi et al., 1996), high novelty seeking and low harm avoidance (Cloninger, Sigvardson, & Bohman, 1988; Masse & Tremblay, 1997). Wills et al. (1995) reported a unique contribution of temperamental factors on frequency of substance use among seventh graders in metropolitan New York. In a longitudinal study by Caspi et al. (1996), undercontrolled boys at age three were 2.7 times as likely to be diagnosed with alcohol dependence by age 21. High novelty-seeking (e.g., impulsive, excitable, quick-tempered, distractible, curious) and low harm avoidance (e.g., uninhibited, carefree, unapprehensive) at age eleven were the most strongly predictive of alcohol abuse at age 27 in a longitudinal study by Cloninger and colleagues (1988), with 75% of males with the highest scores on these two dimensions at observed risk for alcohol abuse compared to only 4% of those with the lowest scores. Similarly, using data from a longitudinal study of 1034 kindergarten students in Montreal, Masse and Tremblay (1997) found a predictive relationship for teacher ratings of high novelty-seeking and low harm avoidance at ages 6 and 10 with self-reported drunkenness in the past 12 months at 10-15 years of age. Based on stability in personality ratings over time, Masse and Tremblay (1997) even argued that personality must be relatively stable by age 6 and that children at risk for early onset of substance use can be identified at the time of school entry.

The relationship between temperament and substance use is complex. Temperament may aggravate the influence of environmental risk. For example, researchers have suggested that the relationship between parental alcoholism and adolescent drinking is mediated by personality or temperament (Chassin, Pillow, Curran, Molina, & Barrera, 1993). In general, what matters are ongoing transactions between temperamental characteristics and the social environment (Tarter & Vanyukov, 1994). According to Tarter (1988), early temperament is part of a vulnerability structure for substance abuse that depends on intervening factors to activate the pathway into problem drinking and drug use. In this diathesis-stress model, a certain behavioral diathesis – or predisposition (e.g., being highly excitable, emotionally intense) – will elicit negative responses from others (e.g., attenuating the quality of parent-child relations). These interactions place “stress” on the diathesis and increase the likelihood of a deviant style of social adjustment. The environment is provocative (McGue, 1997) and exacerbating (Moffitt, 1993). This, in turn, predisposes towards negative developmental outcomes related to alcohol use and other problem behaviors.

Typologies of Alcoholism: An Antisocial Substrate

An early AFD is predictive of later alcohol problems (Grant & Dawson, 1997; McGue et al., 2001b; Prescott & Kendler, 1999; Schukit & Russell, 1983). However, alcoholism is a heterogeneous disorder. There are several etiological pathways into diagnoses of abuse or dependence. Of particular relevance to a developmental perspective is the distinction between individuals with transient manifestations of alcoholism and individuals with long-term, persistent alcohol problems. Several researchers (e.g., Zucker, 1987; Zucker et al., 1995; Cloninger, Sigvardsson, & Bohman,

1996) have proposed typologies to address this issue. These typologies demonstrate the relevance of antisocial behavior problems to alcoholism characterized by early onset.

Cloninger et al. (1996) described two alcoholism trajectories in data from the Stockholm Adoption Study. According to this scheme, Type I alcoholics are those with both genetic (biological) and environmental (including adoptive) risks, onset after age 25, and a personality structure characterized by high harm avoidance, low novelty seeking, and high reward dependence (Cloninger et al., 1996). Type II alcoholics have early onset and a primarily genetic diathesis that is often associated with a paternal history of severe alcoholism and criminality. The personality profile for type II alcoholics is the inversed combination of traits (low harm avoidance, high novelty seeking, low reward dependence) – traits that are also correlated with antisocial personality disorder (Cloninger et al., 1996).

Zucker (1987, 1994) has proposed at least four types of alcoholism. Antisocial alcoholism is defined by early onset of alcohol problems combined with a history of antisocial behavior (Zucker, 1987). Antisocial alcoholics tend to be male and from the lower economic stratum, with a family history positive for alcoholism and greater symptom severity. For these individuals, risk appears to be genetically mediated. From conduct problems in childhood to social/personal/legal difficulties that continue into adulthood, antisocial alcoholism most strongly reflects the hard-continuity model of problem behavior. Developmentally limited alcoholism, on the other hand, involves frequent, episodic heavy drinking that is life-stage specific (Zucker, 1987, 1994). Specifically, alcohol use for this subtype may be a normative process of adolescence for a large subset of individuals – peaking, along with other delinquent behaviors, during

young adulthood but followed by abatement to social drinking patterns when adult responsibilities of work and family present themselves.

Risk factors for developmentally cumulative alcoholism (Zucker, 1987) are also environmentally-located, with difficulties stemming from alcohol use rather than from a co-morbid psychopathology and with abuse/dependence developing subsequent to heavy drinking during adolescence. This subtype is the result of adolescent drinking patterns that do not remit. To point,

In the term developmentally cumulative alcoholism the notion of developmental cumulation implies that risk is more closely tied to normal, culturally prescribed processes of drinking and problem drinking than in antisocial alcoholism but that the additive process has, over the life course, become sufficiently cumulative so that thereafter it has a different trajectory than if it were simply regulated by normative developmental trends in the culture (Zucker, 1987, p. 67).

In his later work, Zucker (1994) renamed this subtype “Primary Alcoholism III” to reflect more fine distinctions in the etiologic processes leading to an environmentally mediated, alcohol-specific (i.e., not driven by co-morbid psychopathology) disorder. Added to the typology were isolated, single “incidents” of alcohol abuse (Primary Alcoholism I) that often coincide with major life stresses such as divorce, and episodic symptomatology (Primary Alcoholism II). A fourth major subtype described by Zucker et al. (1995), negative affect alcoholism, is linked to anxiety and depression. Unlike the other alcoholisms, internalizing symptomatology drives the etiologic process.

A hard-continuity model applies to Cloninger’s Type II and Zucker’s Antisocial Alcoholism. Specifically, the long-term, persisting, and pervasive problem behaviors

characteristic of these alcoholism subtypes help to demonstrate the critical role of co-morbid antisocial behavior in the development of risk for alcohol abuse or dependence. Evidence for the existence of at least two alcoholisms comes from empirical studies showing that Type II alcoholics have more severe symptoms, and that this severity is predicted from antisociality in childhood and adulthood among Type II, but not Type I, men (Zucker, Ellis, & Fitzgerald, 1994). Zucker et al. (1994) also classified individuals by level and severity of child and adult antisocial symptoms into two groups: antisocial alcoholics (AAs) and non-antisocial alcoholics (NAAs). This dimensional approach revealed marked group differences. Compared to NAAs, AAs had an earlier onset of alcohol-related difficulties, more symptom variety, lower socioeconomic achievement, more depression, and a more dense family alcoholism history.

Findings from six longitudinal studies of childhood precursors to adulthood alcoholism were summarized by Zucker and Gombert (1986). Strong consistencies in the extant literature were reported. During childhood and adolescence, individuals who developed alcohol-related difficulties in adulthood had 1) patterns of antisocial behavior, 2) achievement-related difficulties, 3) attenuated interpersonal relationships, 4) homes with heightened marital conflict 5) higher activity levels, 6) inadequate parenting and lack of parental contact, 7) alcoholic, antisocial, or sexually deviant parents, and 8) Irish or Italian ethnic heritages. Together, these constituted a network of heightened risk that was in evidence by childhood – in fact, as early as the preschool years (Zucker et al., 1995, p. 694). Of key interest (e.g., Zucker, 1987), therefore, is the relationship between early developmental processes and alcoholic etiology.

In addition, Zucker (1987) proposed a hypothetical model linking preschool antecedents to adult outcomes. Specifically, intra-individual differences in temperament and impulsivity combined with family socialization to aggression, negative affect, and alcohol schemas are the earliest precursors. The effects of this infrastructure are amplified by parents who model aggression, negative affect, and alcohol use. This heightens the risk for poor school performance, involvement with a deviant peer group, social problems, and problem behavior (including aggression, alcohol use, and other drug use) in middle childhood and adolescence – with difficulties continuing into adulthood and culminating in substance abuse (Zucker, 1987). Continuity in antisocial and other problem behaviors is therefore central to the course of alcoholism for some individuals.

Developmental Theories on Antisocial Behavior

One model for individual variation in antisocial behavior and its timing over the life course is offered by Terrie Moffitt (1993, 1997). Specifically, Moffitt suggests that there are two distinct etiologic patterns: adolescent-limited (AL) and life-course-persistent (LCP). Although indistinguishable during adolescence, individuals falling into the two groups have decidedly different behavioral trajectories.

For a small (LCP) minority, juvenile delinquency is just a snapshot within a lifetime of antisocial activity. Although behavioral manifestations may change (e.g., from conduct disorder to crime and substance abuse), there is a constant propensity. This same argument is made by Gottfredson and Hirschi (1990), who then implicate low self-control – resulting, in large part, from inadequate parenting – as the key underlying predisposition. With a slightly different emphasis, Moffitt focuses on neurological deficits in verbal ability and executive functioning (i.e., control over inattention and

impulsivity). LCP individuals are said to be born with neuro-psychological risk for cognitive deficits, difficult temperament, hyperactivity, and other behavior problems. As mentioned, their parents may share many of their characteristics and are often the least capable of dealing with a difficult child (see also Mun et al., 2001, on the relationship between parental psychopathology and behavior problems). The relationship between problem behaviors and parent-child interactions is presumably reciprocal: Behavior problems can evoke more harsh disciplinary styles from parents, and ineffective parenting can exacerbate the child's problem behaviors. Either way, behavioral difficulties are nested in a family environment and that makes later antisocial behavior more likely. The initial risk trajectory for LCP children is detailed in Figure 1.

Early vulnerabilities are aggravated by the way that others respond to difficult children – as well as the way that these individuals interpret social cues and select their environments according to their own personal style (see also Lerner, 1985). This

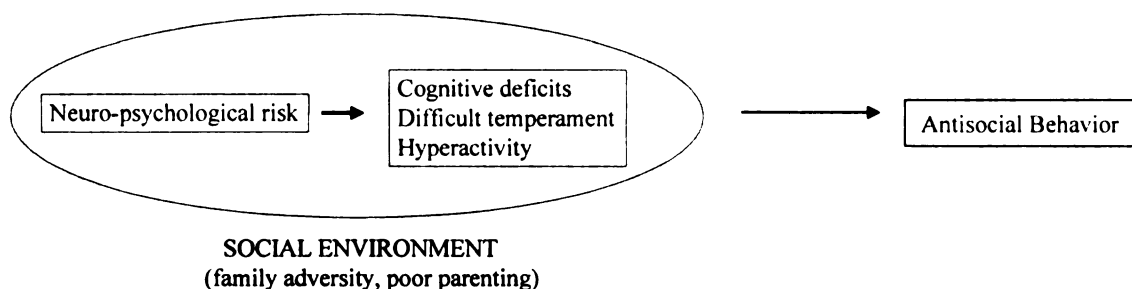


Figure 1.

Heuristic model of early etiology for Moffitt's (1993) life-course persistent antisocial behavior type.

accumulating feedback loop bears negative consequences for future opportunities, interactions, and the scope of learned behaviors in social and academic settings (Moffitt, 1993). Deficiencies in social and academic skills, from an early age, are difficult to overcome – contributing to developmental continuity and life-course persistent antisocial behavior.

In contrast, most people engage in some form of delinquent behavior during adolescence (i.e., the AL group). The impetus for this behavior, however, is thought to be both proximal (i.e., immediately relevant in time and space) and temporary. Adolescents seek individuality and “mature status.” Having biologically reached adulthood but living in a culture that still defines them as immature, there is a sense of cognitive discord. LCP peers appear independent and somehow more grown up, so their behavior is mimicked by the AL group in a “knifing-off childhood apron strings” gesture (Moffitt, 1993, p. 688). With an emphasis on motivation, reinforcement, and modeling, theories of learning are therefore thought to lie at the core of AL behavior. Misbehavior is situational and part of a time-limited developmental phase. Termination occurs – when there is no longer a need to prove one’s “mature status” and with the acquisition of new stakes in conformity (e.g., work). Never lacking in their cadre of socially-acceptable behaviors and without cognitive deficits to hinder them, they return to law-abiding lifestyles with relative ease.

Another developmental perspective especially prominent in criminology is the informal social control theory of Robert Sampson and John Laub (1993, 1997). According to this paradigm, there is generally strong continuity across the lifespan or life trajectory because of stable individual differences and a snowballing of risk (cumulative

disadvantage) which together link childhood misbehavior to adult crime. One's propensity for deviance is not seen as an invariant trait (e.g., like temperament or intelligence), however, but rather varies with the flow of personal and interpersonal attachments.

Informal social controls are constraints against crime and delinquency (Hirschi, 1969; Sampson & Laub, 1993, 1997). During childhood and adolescence, family and school are the primary socializing (or controlling) agents – with work, family, and the military as age-salient institutions in adulthood (Sampson & Laub, 1993, 1997).

According to Sampson and Laub (1993, 1997), meaningful bonds can serve as “turning points” or even termination points for misbehavior (i.e., there is both stability and change in behavioral trajectories). It is the quality and strength of these social bonds (i.e., social capital, stakes in conformity) – not just their presence or absence – that matter (Sampson & Laub, 1993). However, although social bonds at each age explain contemporaneous behaviors, the accumulation of consequences from past behaviors (“cumulative continuity”) and the responses that are evoked from others (“interactional continuity”) can repeatedly attenuate these bonds in what Sampson and Laub (1997) argue is an inherently social process.

The perspective of Sampson and Laub is consistent with Moffitt's developmental theory in terms of recognizing continuity in antisocial behavior. Both acknowledge the fact that, for some individuals, early problems are both predictive of and contributing factors to persistent deviant behavior. As mentioned, also shared is the idea of cumulative consequences (Moffitt, 1993) or cumulative disadvantage (Sampson & Laub, 1997): “Over time, accumulating consequences of the youngster's personality problems

and academic problems prune away the options for change” (Moffitt, 1993, p. 684). One difference between perspectives is that Moffitt largely implicates neurological deficits as the initial “cause” of this risk trajectory (although negative environmental influences can play an exacerbating role) whereas Sampson and Laub (1993) focus on both parenting processes and social structure influences as a core etiology. Most likely it is some combination of psychological, biological, and social factors that helps to shape the continuous or LCP pathway. With this stance, theoretical differences can be seen as complementary rather than contradictory. Unlike Moffitt, however, Sampson and Laub (1993) also explicitly described influences that may contribute to desistence among even the most at-risk individuals. With enough new social capital, informal social control can disrupt the original risk trajectory.

Evidence of Continuity in Antisocial Behavior

The extant literature on continuity has primarily focused on conduct disorder (e.g., Fergusson, Lynskey, & Nagin, 1996), crime (e.g., Farrington, 1995; Sampson & Laub, 1993), and aggression (e.g., Campbell, Ewing, Breaux, & Szumowski, 1986; Campbell & Ewing, 1990; Campbell, 1994; Campbell, Pierce, March, Ewing, & Szumowski, 1994; Eron, Huesmann, Dubow, Romanoff, & Yarmel, 1987). There is robust support for continuity in antisocial behavior across samples (Robins, 1978). For example, Fergusson et al. (1996) found that those with conduct problems at age eight were 16.1 times more likely to meet diagnosis for adolescent conduct disorder or oppositional defiant disorder at 15-16 years old. Reviews of the literature (e.g., Loeber, 1982) suggest that chronic delinquents tend to show persistent and pervasive patterns of antisocial behavior with onset of delinquency by preadolescence.

The Cambridge Study in Delinquent Development (Farrington, 1995) was a longitudinal survey of 411 inner-city South London boys that relied on information from multiple sources (youth, parent, teacher, criminal records) to describe the development of delinquent and criminal behavior. Antisocial behavior at age eight (e.g., troublesomeness, dishonesty, aggression) was the best predictor of official juvenile delinquency at 10-16 and convictions up to age 32 (Farrington, 1995). Twenty-two percent of the most troublesome boys were later convicted for violence, compared to only 4.9% of those in the least troublesome group (Farrington, 1997).

The longitudinal research of Sampson and Laub (1993) is also quite informative. Within their follow-up studies on 500 official delinquents and 500 non-delinquents, there were significant relationships between antisocial behavior at ages 10-17 (official delinquency, unofficial [self, parent, teacher] reports of delinquency, temper tantrums) and adult misbehavior (arrest, alcohol/drug use, general deviance [e.g., gambling]) up to thirty years later, at ages 17-45.

Other studies have implicated early aggressive tendencies and hyperactivity in the etiologic pathway for delinquency. Campbell and colleagues (Campbell et al., 1986; Campbell & Ewing, 1990; Campbell, 1994; Campbell et al., 1994) described patterns of continuity in externalizing behavior, from preschool to middle childhood. Two cohorts of children and controls were identified. In the first cohort of parent-referred children and a control group with no parental behavior problem complaints (Campbell & Ewing, 1990; Campbell et al., 1986), “hard-to-manage” three year olds showed strong continuity in aggression and attention deficit disorder. According to maternal report (Campbell et al., 1986), 50% of those in the problem group continued to meet clinically-significant

criteria at age six. Teacher-ratings corroborated these differences in externalizing behavior problems. By age nine (Campbell & Ewing, 1990), children with persistent problems from 3-6 years of age were more likely to meet criteria for an externalizing disorder (67%) than children who had improved by age six (29%) and the comparison group (16%).

In the second cohort (Campbell, 1994; Campbell et al., 1994), overactive, inattentive, and impulsive preschoolers were identified by teachers and matched to classroom controls. Also included in the sample were parent-referred preschoolers. “Hard-to-manage” children at age four demonstrated problems in activity and impulsivity that were pervasive across settings (home, preschool, laboratory; Campbell et al., 1994) and a two-year time interval.

In general, early aggression tends to be the best predictor of later aggression (Eron et al., 1987; Cairns, Cairns, Neckerman, Ferguson, & Gariépy, 1989) with a degree of stability not much lower than that found in the literature on intelligence (Olweus, 1979). Patterns of hard-continuity are often evident by preschool and school entry (e.g., Campbell, 1994; Campbell et al., 1994; Olweus, 1979), with antecedents traceable to infancy (Keenan & Shaw, 1994).

Eron et al. (1987; Huesmann, Eron, Lefkowitz, & Walder 1984) conducted a study on aggressive behavior over a 22 year time period. In 1960, third graders were interviewed and rated by peer nomination on aggressive behavior. Follow-up self-report assessments were conducted in 1970 (age 19) and 1981 (age 30). Significant correlations between aggression at age 8 and antisocial behavior at age 30 were found – at least for males (Eron et al., 1987). For male children, higher aggression ratings were associated

with greater contact with the criminal justice system two decades later (e.g., with a correlation of $r=.24$ for convictions, $r=.21$ for moving violations, and $r=.29$ for driving while intoxicated [DWI]) as well as severity of punishments for one's own child ($r=.24$). Structural equation modeling revealed relatively high stability coefficients for aggression (.50 for males, .35 for females, .46 overall). Finally, regression analyses demonstrated the high predictability of early aggression for later aggression.

Cairns et al. (1989) followed 220 fourth graders over six years in an investigation of changes in aggression from childhood to early adolescence. Moderate correlations for aggression were found, especially for males, from fourth grade and ninth grade in teacher-ratings ($r=.45$ for males, $r=.33$ for females) and self-report ($r=.36$ for males, $r=.13$ for females). Of an array of variables from the fourth grade, the best predictor of eighth grade aggression was fourth grade aggression.

Interestingly, individual differences in the persistence of aggression may exist in infancy. Shaw, Keenan, and Vondra (1994) documented the existence of developmental precursors to early externalizing behavior problems, including child non-compliance at 18 months of age, in infants from low-income families. Stability from 18 to 24 months was strongest for those with pervasive aggression (i.e., more than one type of aggression exhibited in more than two situations) compared to those in the non-pervasive group – with correlations ranging from .57 to .71 versus .05 to .32 (Keenan & Shaw, 1994). Although Keenan and Shaw (1994) found few sex differences, other researchers (e.g., Cummings, Iannotti, and Zahn-Waxler, 1989) have reported strong stability in physical aggression from age two to age five for males ($r=.76$) but more moderate continuity for females ($r=.36$).

Evidence of Discontinuity in Antisocial Behavior

A wealth of studies have focused on continuity in antisocial behavior over the course of development, but few have developed models to explain escalating or abating behavioral difficulties. Most of the research on change patterns has compared early onset, persistent offending to late onset, transitory offending (as discussed by Fergusson et al., 1996). Alternately, researchers have attempted to delineate distinct developmental trajectories that take other change patterns into account, but usually without attempting to determine their underlying causal processes (e.g., Nagin & Tremblay, 1999). Fergusson et al. (1996) avoid both these limitations but begin to analyze disruptive behavior in middle childhood, not earlier. Ackerman, Brown, and Izard (2003) covered an even more circumscribed developmental time period: from first grade (7 years old) to third grade (9 years old).

Nagin and Tremblay (1999) identified a four-fold typology based on teacher-ratings for three different externalizing problems: physical aggression, opposition, and hyperactivity from ages 6 to 15. The four behavioral trajectories were Chronic, High Level Near Desister, Moderate Level Desister, and Low. Less than 5% of the sample was characterized as Chronic, with consistently high problem behavior scores.

Approximately 20-30% were classified as High Level Near Desisters due to initially high levels of problem behavior that diminished (but didn't disappear) with age. The majority (50%) were Moderate Level Desisters, with modest levels of problem behavior that largely subsided by 10-12 years of age. Problem behaviors were rare for the 10-25% who fell into the Low group. The chronic trajectory for aggression and opposition led to a greater risk of juvenile delinquency than did the chronic trajectory for hyperactivity.

In one of the few studies to include an escalation pattern (Fergusson, Lynskey, & Horwood, 1996), researchers identified factors that distinguished between no onset, early onset followed by remission, late onset, and persistent antisocial behavior groups. Discontinuous pathways were associated with intermediate levels of risk. Risk factors included social disadvantage, family dysfunction, attention deficit behaviors, lower IQ, poor school achievement, and low self-esteem. Children with no conduct problems at 7-9 and 14-16 were those with the most favorable profiles, children with persistent conduct problems were those with the least favorable profiles, and children with remitting or late onset patterns typically had profiles in-between these two extremes.

Finally, Ackerman et al. (2003) sought to explain continuity and discontinuity in teacher-rated externalizing behavior for children from impoverished homes. Children were classified into one of four groups based on the presence or absence of high levels of symptomatology in the first and third grades: persistent problem (i.e., high at both grades), improver (i.e., high only in first grade), new problem (i.e., high only in third grade) and unproblematic (i.e., low at both grades). Child, parent, and family variables were explored as distinguishing characteristics. Unproblematic individuals had lower levels of impulsivity compared to everyone else, and less parental maladjustment compared to those with high levels of externalizing behaviors in the first grade (i.e., improvers and persistent problems). Vocabulary scores were lowest for those with problems in the third grade (persistent and new problem groups). Harsh punishment was higher for the persistent problem group than for those without problems in the third grade (i.e., improvers and the unproblematic group); the new problem group was at intermediate risk. Some support was therefore found for the idea that verbal ability and

harsh parenting (along with family instability) were related to changes in externalizing during the early school years – but with the emergence of new problems in particular.

Risk and Resiliency

The topic of risk and resiliency in human development is one of the most interesting and popular in the field today. Moreover, as a framework for understanding developmental processes, it is central to this discourse on continuity versus escalation or abatement. Risk can be conceptualized as an internal or external adversity that increases the likelihood of undesirable developmental outcomes (i.e., the probability of harm). According to Luthar, Cicchetti, and Becker (2000b, p. 543), “resilience refers to a dynamic process encompassing positive adaptation within the context of significant adversity.” To be labelled “resilient”, an individual must be exposed to “significant threat or severe adversity” and demonstrate positive adaptation despite that exposure (Luthar et al., 2000b, p. 543; Masten, Best, & Garmezy, 1990; Masten & Coatsworth, 1998). Resiliency has often been used synonymously with competence (Garmezy, Masten, & Tellegen, 1984; Luthar, 1993; Masten & Coatsworth, 1998; Masten, Hubbard, Gest, Tellegen, Garmezy, & Ramirez., 1999; Sameroff, 2000) and “stress resistance” (Garmezy et al., 1984; Masten et al., 1990), as well as with “buffers” and “protective factors.”

Although discussions of risk have been historically ubiquitous, resiliency is a relatively new concept that nonetheless has enjoyed much attention in recent years. It complements studies of risk, because it is well known that not all individuals exposed to adversity will suffer from the “push” towards maladaptive outcomes. For example, not all children with difficult temperament, neurological deficits, or physical handicaps; and

not all children exposed to poverty, parental alcoholism, parental psychopathology, domestic violence, violent neighborhoods, and so forth will manifest problematic cognitive, social, physical, or emotional impairments. What differentiates those who do from those who don't? Studies of resiliency hope to clarify *how* and *for whom* risks do and don't exert their predicted effects on development. The theoretical and empirical literature on risk and resilience is therefore a critical starting point for a comprehensive understanding of individual differences in developmental trajectories.

Risk Factors. Risk factors in development were recognized even before psychology became an established intellectual discipline. For example, Rutter (1985, p. 598) references the work of an early predecessor to Freud: Nearly 200 years ago, Pinel wrote about the psychiatric risks associated with unexpected reverses or adverse circumstances, and it is reported that his initial question to newly admitted psychiatric patients was: "Have you suffered vexation, grief, or reverse of fortune?" Interestingly, Pinel is sometimes referred to as the father of scientific psychiatry (Hothersall, 1995). The idea of measuring risk actually originated with maritime insurance; merchants and ship owners would negotiate a payment based on the risk of a disaster at sea (Werner, 1986). There is an important message carried over from these commercial bargains, which is just as relevant for understanding the epigenesis of human behavior – namely, that the "identification of risk factors is an exercise in estimating probabilities" (Sameroff, 2000, p. 7).

Only in recent decades has the concept of risk really come into the limelight of scholarly inquiry, however (Rutter, 1985). It first gained prominence in epidemiology and preventative medicine (Werner, 1986). Researchers were interested in understanding

susceptibility to cardiovascular disease (e.g., in the Framingham Heart Study; see Sameroff, 2000) and other maladies. The disease model, applied to psychology, led to an initial perspective of “reproductive casualty” in which risk was viewed as internal or extending from the birth process (Sameroff & Seifer, 1983). Eventually, “caretaking casualty” was added to the risk model. Studies of infants and young children exposed to pre-, peri-, and post-natal risks (e.g., chromosomal disorders, nutritional deprivation, infections, accidents, illiteracy, poverty) emerged during the late 1950’s and 1960’s (Werner, 1986). Because these forays examined constitutional and environmental influences independently, findings lacked predictive power (Werner, 1986).

There is now general consensus among developmental psychologists that a more ecological “person-in-context,” dynamic interaction approach is needed to address the complexities of human development. Developmental Systems Theory (DST) is a guiding paradigm in this respect. Within and across levels of influence, it is important to recognize that there is usually not a single factor but a set of factors that contributes to outcome (Sameroff, 2000). Given equifinality in developmental trajectories, there may be a different combination of variables at work for each individual (Sameroff, 2000) despite the fact that outcomes are quite similar. Also, risk factors tend to co-occur (Masten, Best, & Garnezy, 1990; Moffitt, 1993; Zucker et al., 1995) and aren’t random (Rutter, 1987). To varying degrees, these social forces shape the human ecology. In addition, individuals select and elicit their own environments in active and evocative ways (Ford & Lerner, 1992; Lerner, 1985; Rutter, 1997).

Not surprisingly, accumulated risk appears to be most detrimental – as measured by the number of risk factors to which individuals are exposed (Ackerman, Izard, Schoff,

Youngstrom, & Kogos., 1999; Barocas, Seifer, & Sameroff, 1985; Bry, McKeon, & Pandina, 1982; Newcomb, Maddahian, & Bentler, 1986; Sameroff, 2000; Werner, 1986, 1989; Werner & Smith, 1982). For example, Bry and colleagues (1982) and Newcomb et al. (1986) found that risk load was the best predictor of drug use among high school students. Of the children in Werner and Smith's (1982) longitudinal study on the island of Kauai, approximately one-third experienced chronic poverty and prenatal or congenital insults and were raised by uneducated mothers in troubled families. Among those with four or more risk factors present by age two, three-quarters "developed either serious learning or behavior problems by age 10, or had delinquency records and/or serious mental health problems by age 18" (Werner, 1986, p. 13; Werner, 1989). With data from the Rochester Longitudinal Study, Sameroff (2000) found that the number of risk factors was the crucial measurement in predicting psychiatric disorder. It is worth mentioning here that Sameroff (2000; Sameroff & Seifer, 1983; Barocas et al., 1985) is one of the most outspoken advocates of looking at the quantity – rather than the quality or exact type – of risk, especially in ecologically oriented models.

Yet, given the complexities of understanding risk, there is a growing appreciation of the processes leading to negative developmental outcomes (Rutter, 1987). Rather than just identifying risk factors, researchers have sought an understanding of when and how risks impinge upon human adaptation. For example, Rutter (1985) has looked at family discord (which mediates parental psychopathology; see also Barocas et al., 1985); parental loss (which exerts its effect through quality of care and family functioning); and difficult temperament (eliciting more parental criticism and hostility in already harsh contexts; see also Hetherington, 1989), among other risks.

Risk factor studies are among the most prevalent in the literature on developmental psychology and psychopathology. In fact, it is fair to say that the word “risk” is ubiquitous therein. Apart from longitudinal studies focused on development in general, a search for risk factors has been initiated with regard to a plethora of specific outcomes of concern to public health and psychiatric teams. This includes alcohol problems (Ellis, Zucker, & Fitzgerald, 1997; Wong, Zucker, Puttler, & Fitzgerald, 1999; Zucker, 1994), binge drinking (Schulenberg, Wadsworth, O’Malley, Bachman, & Johnston, 1996), drug use (Newcomb et al., 1986), depression (Petersen et al., 1993), and problem behavior (Ackerman et al., 1999).

Protective/Promotive Factors. Not all individuals exposed to risk will demonstrate developmental failures, however. In fact, many don’t. Is there a self-righting tendency, located internally or in the care-taking environment, which maintains adaptive behavior (canalized for the species, in the traditional sense of the word)? Who are the winners, losers, and survivors of adversity (Hetherington, 1989)? For many years, Garmezy and colleagues studied the effects of parental schizophrenia – finding that a large number of offspring evidenced negligible signs of pathology and incompetence (for an overview, see Garmezy, Masten, & Tellegen, 1984; Masten et al., 1990). Interest in why some individuals thrive when others wither led to the birth of studies on resiliency. From these early beginnings – rooted deeply in the field of developmental psychopathology – the resiliency literature has burgeoned and diversified. Emmy Werner’s (1986; Werner & Smith, 1982) incredibly multifaceted investigations of risk and resiliency among children in Kauai have often been credited for this diversification (Luthar et al., 2000b).

Early work often focused on individual characteristics thought to provide an internal buffer against environmental adversity (Garmezy et al., 1984; Luthar et al., 2000b; Rutter, 1985). This is consistent perhaps with organismic ideas about the nature of development. More specifically, the dominant idea was that of “invulnerable” children who were “so constitutionally tough that they could not give way under the pressures of stress and adversity” (Rutter, 1985, p. 599; Luthar et al., 2000b). However, the contextualizing of developmental psychology in the 1970’s led to recognition of ecological resiliency. As with risk, resilience can be both biological/genetic and social/experiential (Rutter, 1985). Nature and nurture both matter; in fact, they are enmeshed.

Intelligence, personal competencies, and support in the caregiving environment have been identified as primary proximate buffers against maladaptation (Egeland, Carlson, & Sroufe, 1993; Masten & Coatsworth, 1998; Masten et al., 1999; Werner, 1986; Werner & Smith, 1982; Wyman, Cowen, Work, Hoyt-Meyers, Magnus, & Fagen, 1999). Of the high-risk children in Werner’s study, one of four “escaped the ill effects of such multiple risks and developed into stable, mature, and competent young adults” (Werner, 1986, p. 13; Werner, 1989). Resilient children tended to be first-born, well-tempered infants who “met the world on their own terms” as toddlers, had many hobbies/interests and effectively maximized their communication/problem-solving skills during middle childhood, believed in their own self-efficacy and self-worth during adolescence, and were achievement-oriented (Werner, 1986, p. 14; Werner, 1989). Resilient children also had a strong bond to a primary caretaker during infancy (Werner,

1986, 1989). This was often a parental substitute, such as a grandparent or older sibling (Werner, 1989).

Similar reports are found elsewhere. In general, commonly identified resiliency factors include having a close relationship with a competent adult, healthy self-esteem and self-efficacy, positive experiences at school, and a good-natured disposition (Masten et al., 1990; Rutter, 1985, 1987). These buffers are also highly interrelated. For example, individuals' academic success can lead to "enhanced self-esteem and a feeling of self-efficacy, enabling them to cope more successfully with the subsequent life challenges and adaptations" (Rutter, 1985, p. 604). Distal factors appear to work through more proximate contexts – for example, with exosystem adversities (e.g., living in a violent community) eroding the quality of functioning in the microsystem (e.g., the family) (Richters & Martinez, 1993).

As with studies of risk and in recognition of probabilistic epigenesis, the resiliency literature is also moving away from static conceptualizations of developmental influence. Scholarly interest has evolved from discussions of "invulnerable" children (i.e., absolute protection) to "resilient" children (i.e., relative protection) for whom risk and protection can fluctuate over the course of development (Luthar et al., 2000b; Rutter, 1985). This is consistent with Lerner's (1980, p. 781) more global assertion that a focus on the process and, particularly, on the process involved in the changing relations between individuals and their contexts, is at the cutting edge of contemporary developmental theory and, as such, is the prominent conceptual frame for research in the study of human development.

Resiliency is a process, not a trait (Egeland et al., 1993; Luthar et al., 2000b; Rutter, 1985). One's resilience can change over time, may operate in behavioral chain reactions, and may be dependent on the timing of risks throughout life course. For example, parents' divorce experienced during infancy is not the same as divorce experienced during childhood, or adolescence, or young adulthood (Rutter, 1985). The social environment also expands (e.g., Coie & Jacobs, 1993; Dekovic, 1999; Masten & Coatsworth, 1998) as individuals become involved in institutions such as school and work. Therefore, although initial scientific forays sought to identify protective factors, researchers are increasingly interested in understanding how or when these factors exert an effect (Luthar et al., 2000b; Gest, Neemann, Hubbard, Masten, & Tellegen, 1993), and how or when these factors are acquired in the first place. This parallels recent trends in the risk literature. As researchers interested in the how's of development (e.g., Anastasi, 1958) – not just the what's – we ask: "Is it chance, the spin of the roulette wheel of life, or did prior circumstances, occurrences, or actions serve to bring about this desirable state of affairs?" (Rutter, 1987, p. 316). Looking at stability and change in multiple levels over time also is consistent with life-span theories and longitudinal methods for analyzing developmental phenomena (Luthar et al., 2000b; Staudinger, Marsiske, & Baltes, 1993; von Eye & Schuster, 2000).

Because the field is relatively new, there are still major disagreements about how best to measure resiliency. The most controversial issue is whether to look for main effects or interactions. Early conceptualizations referred to "protective" factors in interaction terms – that is, as moderators (see Baron & Kenny, 1986) of the relationship between risk and outcome. Specifically, these implied that resiliency modifies (i.e.,

nullifies or lessens) the effect that adversity would otherwise have on development (see Luthar et al., 2000b; Rutter, 1985; Garmezy et al., 1984; Masten et al., 1990; Rutter, 1987). In other words, protective factors should be catalytic (Rutter, 1987). Interactions are still the dominant focus of inquiries – as some argue they should be, by definition (above; see also Roosa, 2000; Rutter, 1987). Although interaction effects tend to be small and unstable (Luthar et al., 2000b), this is attributed to homogeneity on the risk dimension within samples (e.g., Dekovic, 1999; Roosa, 2000).

The main effects of protective “factors” (or processes) have also been of interest to some. Regardless of risk level, some influences may encourage more positive adaptation. An interesting question is whether resiliency only helps those exposed to adversity, helps those more than non-exposed others, or helps everyone regardless of risk status (e.g., Luthar et al., 2000b; see also methodological issues as reviewed by von Eye & Schuster, 2000). Some researchers have suggested, for example, that “resilience is an integral part of normal development that every child must achieve” to the extent that “resilience is a name for the capacity of the child to meet a challenge and use it for psychological growth” (Baldwin, Baldwin, Kasser, Zax, Sameroff, & Seifer., 1993, p. 743). To clarify the semantic confusion, Sameroff (2000) suggested that modifying factors be distinguished from factors that benefit all children (i.e., that lie at the positive pole of the risk dimension) by referring to the latter as “promotive” instead.

Risk and promotive factors would therefore be opposite ends of the same spectrum, with protective factors having an interactive effect. This makes intuitive sense, and may relieve some of the tension between researchers talking at each other rather than to each other – using the same terminology but answering different questions. Other

solutions, such as adding elaborated labels to interactive effects (e.g., “protective-enhancing”; Luthar, 1993), have been suggested but are no less confusing.

Generalizability across domains of resilience is another methodological concern. Luthar et al. (2000b, p. 548) provided the most reasonable position on this issue: “In studies of resilience, we believe that there should undoubtedly be some uniformity across theoretically similar adjustment domains, but not across those that are conceptually distinct” given that “unevenness in functioning across domains is a common occurrence in the process of ontogenesis.” Researchers need to be specific in discussing outcomes of relevance to which their data apply – for example, referring to “educational resilience,” “emotional resilience,” “behavioral resilience,” and so forth (Luthar et al., 2000b). Because adversity is often intrinsic to the definition of resiliency, care must be taken to identify exactly what individuals are expected to be resilient against, and how that would reveal itself. Social competency is not always associated with better emotional adjustment. In fact, otherwise resilient individuals sometimes report more emotional distress over time (Luthar, Doernberger, & Zigler, 1993).

Finally, how positive do adaptations need to be to qualify as resilience? Luthar et al. (2000b) suggest that maintaining near-average functioning is sufficient evidence when extreme and severe stressors are at play, but that superior functioning may need to be witnessed when the stressors are of a more moderate nature. However, superior functioning may be asking too much (Robinson, 2000). A more appropriate standard may be “trajectories that are ‘unexpectedly positive,’” with specific cut-offs determined by the researcher (Luthar, Cicchetti, & Becker, 2000a, p. 574).

It is obvious that risk and resiliency are intertwined, by definition. Many risk

factors are distal in nature – exerting their effect through more intimate milieus. Environmental (e.g., low socioeconomic status, social disorganization) or even interpersonal (e.g., parental psychopathology) risks may be offset by proximal “protections” such as having a supportive family or doing well in school. Not everyone in a risk category will actually experience the same amount of adversity. For example, high-risk children with easy temperaments may elicit more positive attention from parents, meaning that they are potentially exposed to less adversity (Masten et al., 1990). Children with difficult temperaments may become the target of parental hostility, criticism, and annoyance – possibly exacerbating an already aversive home life (Rutter, 1985, 1987). Consequently, “it cannot be assumed that any environmental risk factor – wherever it falls on the distal-proximal continuum – carries equivalent levels of risk to all children exposed to it” (Luthar, 1993, p. 443-4).

Researchers have even debated whether or not risk and protection/promotion are actually distinct constructs, or opposite poles of the same phenomenon. Sometimes empirical differences between risk and protective/promotive factors become blurred, to the extent that reverse coding and categorizing would also make sense (e.g., see Costa, Jessor, & Turbin, 1999). However, it is not necessarily the case that resilience can be equated with “lack of risk.” Having a coach who encourages positive self-esteem and academic pursuits in the face of other adversity (e.g., parental psychopathology) is equivalent to the presence of an influence; the absence of a caring and supportive adult outside the family isn’t always as meaningful, especially when parent-child relationships aren’t attenuated in any way (e.g., in poor, but cohesive, families). Even if assets are the counterparts of risk (Gilgun, 2000), the term “protection” or “promotion” may be

important if only to explicitly acknowledge that main effects come from the positive end of the variable (Rutter, 1987, p. 319) or that the two extremes are not equally potent.

“For instance, perhaps high religiosity is more related to low drug use than low religiosity is associated with high drug use” (Newcomb & Felix-Ortiz, 1992, p. 281).

Initial Risk Trajectory for Antisocial Behavior

A distinction needs to be made between early predispositions (herein called the initial “risk trajectory”) and factors within the personal and social surround that are subsequently involved in risk or promotion. The former are assumed to be foundational; they may interplay with life experience but also constitute the backdrop against which later development can be understood. Temperament and lifetime parental psychopathology (i.e., characteristics of parents that are long-standing, assumed to predate the parenting experience, and possibly genetic) are two examples. Both also have strong links to child behavior problems, as demonstrated in the extant literature, and are therefore highly relevant to the present discourse.

Temperament. As a means of studying individual differences in risk predisposition during the early years of child development (Rothbart, 1981), temperament offers one framework for research. Temperament theory has been used in academic inquiries and intervention (e.g., nursing) in terms of providing parents and investigators with an appreciation of the unique characteristics of each child and insight into expectations about child-rearing, specialized hospital care for children, the etiology of problem behaviors, developmental trajectories, and maternal mental health/fatigue (Tomlinson, Harbaugh, & Anderson, 1996).

Theories of temperament are both numerous and diverse. Unfortunately, different conceptualizations abound about what “temperament” really means. Researchers have focused on models with a variety of different theoretical emphases, including behavioral style and the “how of behavior” (Chess & Thomas, 1989); stable, heritable, and evolutionary-adaptive aspects of personality (Buss, 1991); and the psychobiological nature of reactivity and self-regulation (Rothbart, 1981), among others.

The first attempt to describe temperament is generally attributed to Thomas, Chess, and colleagues through the New York Longitudinal Study (NYLS). Analyzing parent interviews, these researchers ultimately postulated nine dimensions of temperament: activity level, rhythmicity, approach, adaptability, threshold, intensity, attention span, distractibility, and persistence (Chess & Thomas, 1989). Where children fall on these nine dimensions is presumed to reflect a threshold characteristic that crosses all emotional and behavioral processes across situational contexts: “the typical way in which a child pursues activities and responds to the demands of the environment” (Chess & Thomas, 1989, p. 166). Parenting responses can reinforce certain temperamental tendencies, such that stability may even be the result of environmental influences rather than a static psychological attribute (Chess & Thomas, 1989). In other words, there is reciprocal, or dynamic, interaction between child temperament and its context. Moreover, the environment evolves as the individual matures: “the changing context of the child’s or adult’s behavior and the emergence of new forms of behavior at later developmental periods may give the same characteristic very different forms of expression” (Chess & Thomas, 1989). Tendencies or dispositions may remain quite stable, but the ways in which they are manifested can vary with situation, maturation, and

interplay within changing social relationships (Strelau & Angleitner, 1991). Related to this is “goodness of fit” – the idea that what matters is match or mismatch between child characteristics and parenting practices or the environment (e.g., Chess & Thomas, 1989).

A second perspective under the umbrella of temperament theory comes from Buss and Plomin (Buss, 1991). Whereas some researchers (e.g., Chess & Thomas, 1989) have argued that temperament may help to shape the individual’s personality structure – in transaction with the environment – but that the two are not the same, Buss and Plomin argued that temperament is a subset of personality traits (Buss, 1991; Worobey, 1999). Specifically, it is defined as such on the basis of three major assumptions: an appearance during the first year of life; persistence through adulthood, at least in terms of prediction; and its inheritance as part of the genotype of humans and other animals (Buss, 1991). Buss (1991) proposed three dimensions of temperament, known as EAS: emotionality (distress and autonomic arousal accompanying both fear and anger), activity (movement or physical expenditure of energy), and sociability (a preference for being with others); activity is the only one of the three related to behavioral style. EAS only posits a limited and restricting transactional approach: the child is *the* driving force – capable of “choosing” environments, “setting the tone for others in social interaction,” and modifying the interpersonal environment or its impact (Buss, 1991, p. 47).

A third perspective proposes a more biological, Pavlovian psychophysiology definition of temperament. According to Rothbart (1981), temperament is related to the autonomic and somatic responses of the nervous and endocrine systems (i.e., reactivity) and the strategies adopted to modulate these psychobiological processes (i.e., self-regulation). This has “a constitutional basis, with ‘constitutional’ defined as the

relatively enduring biological makeup of the individual, influenced over time by the interaction of heredity, life experience, and maturation” (Rothbart, 1981, p. 569). For example, self-regulation becomes more and more organized over time (Strelau & Angleitner, 1991), developing from “other” regulation shaped externally by the caregiver into an internalized control structure that the child can then activate on his/her own (Rothbart, 1991). Although the theoretical assumptions here are not generally compatible with the NYLS model, two facets of temperament are recognized by both: its emergent nature, and the importance of the caregiving relationship (for Rothbart, at least initially).

Despite theoretical differences in the conceptualization and measurement of temperament, there is some consensus. In general, and at its most basic or synthesized level, the concept of temperament describes individual differences with a constellation of traits that constitute general dispositions to respond in certain ways, that have at least some biological influence, that are relatively stable, and yet are subject to change (Worobey, 1999). As such, temperament is a predisposition that precedes or underlies behavior, and can be considered a precursor to outcomes in childhood, adolescence, and adulthood.

For example, temperament has been linked to behavior problems (Campbell & Ewing, 1990; Caspi et al., 1995; Jansen, Fitzgerald, Ham, & Zucker, 1995; Loukas, Fitzgerald, Zucker, & von Eye, 2001; Wong, Zucker, Puttler, & Fitzgerald, 1999), crime/delinquency (Caspi et al., 1996; Henry, Caspi, Moffitt, & Silva, 1996; Tremblay, Pihl, Vitaro, and Dobkin, 1994; White et al., 1990), and substance use (Masse and Tremblay, 1997; Tarter, 1988, Tarter & Vanyokov, 1994) over the life-course. Details

from a few of these studies help to demonstrate the pervasiveness of temperamental difficulties in predicting the persistence of antisocial behavior.

Most strikingly from a long-term perspective are reports from the Dunedin Multidisciplinary Health and Development Study which suggest that adolescent delinquency (White et al., 1990) and behavior problems (Caspi et al., 1995) as well as adult psychiatric outcomes, crime, and alcohol dependence (Caspi et al., 1996) can be predicted by the behavioral styles of children as young as age three. The Dunedin Multidisciplinary Health and Development Study of Caspi and colleagues has been following a complete cohort from New Zealand born between April 1, 1972, and March 31, 1973, with assessments at ages 3, 5, 7, 9, 11, 13, 15, 18, and 21. Caspi et al. (1995) found that temperament dimensions at 3 and 5 years of age were correlated with externalizing behavior problems at 9, 11, 13, and 15 years of age. Specifically, there were consistent positive correlations between externalizing symptomatology and lack of control, a factor comprised of items related to emotional lability, restlessness, short attention span, and negativism.

In other research by Caspi and colleagues (1996), children were classified into one of 5 clusters at age three: undercontrolled (irritable, impulsive, impersistent, and emotionally labile), inhibited (fearful, socially reticent), well-adjusted (within normal limits), confident (zealous, eager to explore, easily adjusting), and reserved (timid, somewhat uncomfortable during testing). Undercontrolled and inhibited preschoolers were the most likely to develop an adult psychiatric disorder by age 21. Compared to their well-adjusted peers, undercontrolled children were 2.9 times as likely to be diagnosed with antisocial personality disorder, 2.2 times as likely to be a recidivistic

offender, 4.5 times as likely to be convicted for a violent offense, and 2.7 times as likely to be diagnosed with alcohol dependence.

The predictive utility of early undercontrol is also demonstrated by Tremblay, Pihl, Vitaro, and Dobkin (1994). For a large sample of boys attending French public schools in Quebec, teacher-rated impulsivity in kindergarten was the personality dimension most associated with self-reported delinquency at 10-13 years of age.

There are several possible explanations for the robust relationship between temperament and behavior problems. Difficult temperament may be an early substrate of behavior problems, reflecting the same individual differences but with one as a subclinical predecessor to the other (Caspi et al., 1995). Alternately, the relationship can be explained by dynamic interaction between person and environment (Caspi et al., 1995). Children with difficult temperament may evoke negative responses from the interpersonal environment (e.g., if parents respond with harshness, neglect, or impatience) and may react to that adversity in ways that elaborate their initial behavior schemas. They may also be more susceptible to family adversity (e.g., parental psychopathology) because of their core risk diathesis, with this environmental stress activating the temperamental vulnerability to produce a behavior problem reaction. The idea of temperament as a mediator of environmental risk and outcomes has been tested and verified (Loukas et al., 2001). Problem behaviors may even be a coping mechanism for dealing with unfavorable experiences. Interplay is also emphasized by Henry et al. (1996), who argued that risk for antisocial behavior is determined by two components of behavioral regulation: social regulation provided by the family environment, and individual differences in self-regulation (e.g., lack of control). Again, it is the nesting of

undercontrolled children in disorganized social contexts that characterizes the developmental pathway into persistent antisocial behavior.

Parental Psychopathology. Parental psychopathology has also been extensively studied in relation to the development of child behavior problems. Children of antisocial, alcoholic, and depressed parents are at particularly heightened risk. In fact, these psychopathologies are often co-morbid (Zucker, 1987, 1994; Zucker et al., 1995).

Longitudinal studies of the development of delinquent behavior have often implicated parental antisociality as an important risk factor. For example, juvenile delinquents are more likely to have parents who have been convicted, have committed crimes, or met diagnostic criteria for antisocial personality disorder (Elkins, Iacono, Doyle, & McGue, 1997; Farrington, 1995; Loeber & Dishion, 1983; Sampson & Laub, 1993). In Fitzgerald, Zucker, and Yang (1995), both maternal and paternal antisociality predicted child aggression at three to five years of age. Interestingly, the effect for maternal antisociality was stronger.

Greater severity in behavior problems has also been found among children of alcoholics (Fitzgerald, Sullivan, Ham, Zucker, Bruckel, & Schneider, 1993; Loukas, 1997). In Fitzgerald et al. (1993), a higher percentage of children from alcoholic families scored in the extreme clinical range for behavior problems. As mentioned, parental alcoholism is also indirectly related to externalizing behavior problems through temperament (Loukas et al., 2001). Fitzgerald, Zucker, & Yang (1995) reported pervasive differences between male children of alcoholics and control children – including deficits in fine-motor, personal-social, language and adaptive skills, intelligence, behavior problems, and the reactivity dimension of temperament.

Another form of parental psychopathology associated with child behavior problems in the extant literature is depression – especially maternal. Campbell (1994) found that initial levels of maternal depression were higher for persistently “hard-to-manage” boys two years after study entry. Fitzgerald et al. (1993) reported that mother’s depression (and alcohol problems) predicted her ratings for son’s externalizing behavior problems at age three.

In general, there may be a genetic vulnerability towards behavioral styles that predispose towards antisocial behavior, and children may learn to cope with stress or adversity through antisocial means as a result of modeling the behaviors they are exposed to by parents (e.g., aggression, abuse, negative affect expression). Fuller et al. (2003), for example, found evidence for continuity in aggression across three generations. As discussed in the next section, however, many of these effects are mediated through the more proximate family environment that accompanies parental psychopathology.

Influences in Middle Childhood

Early personal and environmentally-influenced risk trajectories do not exist in a self-sustaining vacuum but interact with processes of risk and protection/promotion over developmental time. As mentioned, among these proximate (and mediating) influences are features of the family environment that maintain, exacerbate, or temper initial risk status. Also of importance are proximate influences from other major domains. Schools, peers, and self-concept are particularly relevant to developmental tasks of middle childhood. For example, Erikson (1950) argued that the goal of development is to achieve a positive ego identity. This is accomplished by successfully mastering each of eight stages that, together, make up the life course (i.e., the “Eight Stages of Man”). Each

stage is marked by a conflict to be resolved. During middle childhood (i.e., Freud's latency period), the conflict is one of industry versus inferiority. The child "learns to win recognition by producing things" (Erikson, 1950, p. 226) – that is, by being industrious. Under this rubric, to not cultivate one's "tools and skills" leads to despair, failure to succeed at school, and later disappointments in the "total economy" (Erikson, 1950, p. 227). Similar issues are addressed by Havighurst (1972) in his discussion of developmental tasks, which in middle childhood include getting along with age-mates and building wholesome attitudes towards oneself. From a life-course perspective, it may be that competencies (or deficits) in these areas can buffer (or worsen) the effects of early risk.

Family Environment. Family management techniques and family climate have been widely studied as important predictors of child behavior problems. In Farrington's study (1995), future juvenile delinquents were more likely to have family environments characterized by economic deprivation, physical neglect, harsh or erratic discipline, parental conflict, and poor supervision. Delinquency was also associated with erratic/harsh discipline, poor supervision, parental rejection, and weaker parental attachment in the analyses conducted by Sampson and Laub (1993). Structural background factors (e.g., family disruption, low socioeconomic status, parental criminality, parental alcohol use) had an effect on delinquency, but operated almost entirely through these family process variables. The family-level risk factors for continuity in behavior problems reported by Fergusson et al. (1996) included social disadvantage and family dysfunction. In Wong et al. (1999), parents' negative affect

expression (spanking, bad mood, and sadness) was positively related to children's externalizing behavior problems.

Among the significant predictors of persistent externalizing disorders in the research of Campbell and colleagues were family stress, marital dissatisfaction, and negative maternal control (Campbell & Ewing, 1990; Campbell, 1994). In fact, given conditions of ongoing family adversity, Campbell (1994, p. 164) concluded that "findings highlight the stability of problem behaviors in young boys, the association between high symptoms and poor social functioning in young children, and the important role that family context plays in both the stability of problems and their apparent onset."

Patterson and colleagues (e.g., Patterson, DeBaryshe, & Ramsey, 1989; Larzelere & Patterson, 1990) have described a developmental perspective on antisocial behavior that focuses on the importance of the family context. A three-step etiological process was proposed to explain chronic delinquent behavior (Patterson et al., 1989), as follows: Ineffective parenting (i.e., poor parental discipline and monitoring and little positive parental involvement) leads to conduct problems, which lead to academic failure and peer rejection, which lead to involvement with a deviant peer group.

Many distal risks influence child behavior through the more proximate social environment. For example, Larzelere and Patterson (1990) found that parent management skills – which include parental monitoring and discipline – mediated the relationship between socioeconomic status and delinquency in early adolescence. One measure of discipline was "Nattering", or negative parental interactions toward the child.

Other research suggests that the effects of parental alcoholism and other parental psychopathologies on child development are mediated through the quality of family

relationships and overall dynamics in the home environment (e.g., Barrera, Li, & Chassin, 1995; Hawkins, 1997; Hecht, 1973; Henderson, Albright, Kalichman, & Dugoni, 1994; Hyphantis, Koutras, Liakos, & Marselos, 1991; Jacob, Krahn, & Leonard, 1991; Roosa, Beals, Sandler, & Pillow, 1990). According to Hecht (1973), families that seek professional help because of alcoholism do so because of conflicts at home or within other social institutions (e.g., school, work, community), not because of drinking behavior per se.

In general, alcoholism has been associated with a parenting style characterized by more negative and less positive affect. Jacob et al. (1991) found that, compared to a control group, fathers diagnosed as depressed or alcoholic demonstrated less congeniality and fewer neutral problem-solving strategies when interacting with their sons during a videotaped discussion about family issues. Notably, these authors described a general atmosphere of distress, as characterized by alcoholism or depression, which defines family “dysfunction.” Similarly, Henderson et al. (1994) suggested that co-morbidity in psychiatric diagnoses lends itself to the oft-unwarranted conclusion that there is something particular about substance use; rather, alcoholism seems to be one of several mental health problems (e.g., schizophrenia, depression, generalized anxiety disorder) that contribute to detrimental developmental outcomes among offspring.

In addition to attachment and affection, supervision and discipline are impacted by parental psychopathology. For example, the alcoholic is unlikely to assume adequate responsibility in this regard – expressing either detachment or inappropriate displays of anger. In some cases, the non-alcoholic parent in an alcoholic household may be so focused on dealing with the spouse that supervising and disciplining children becomes

lax (Hecht, 1973; cf. Hyphantis et al., 1991). At the other extreme is a harsh and inflexible style of discipline (i.e., authoritarian). Malo and Tremblay (1997) found that children of alcoholic fathers reported more frequent punishment than controls, but fewer rules for proper conduct. Children may find the inconsistencies inherent in a punitive but undefined system of punishment somewhat stressful (Malo & Tremblay, 1997).

Another correlate of parental alcoholism is family conflict or discord. In an observational study, Jacob et al. (1991) indicated that mother-father dyads in families characterized by paternal alcoholism demonstrated more negative emotionality, less positive affect, and heightened levels of general discord than their non-alcoholic counterparts. Webb and Baer (1995) also found a significant relationship between family disharmony and parental alcohol use, with the quality of parent-child interactions having a direct (and mediating) effect on outcomes such as attenuated social skills or adolescent alcohol use. Unfortunately, the issue of causality is problematic in most of these studies. Too often, given the data available, researchers cannot determine whether the drinking behavior of parents leads to greater conflict in the family or whether conflict is the impetus for alcohol use (e.g., Webb & Baer, 1995). Heightened conflict could also be a reaction to acting-out behavior by the child, another potential confound. Reiterating Hecht's (1973; see also Henderson et al., 1994) observation, however, it is still extremely important to consider the family environment in which children are embedded – not just manifestations of individual-level parental psychopathology.

In summary, the emphasis needs to be on the “functional structure of the family” (Hyphantis et al., 1991, p. 35), which is consistent with an ecological person-in-context approach to risk. Alcoholics tend to be less demonstrative with affection, and more likely

to employ a permissive or authoritarian style of parenting. Alcoholic families may also be less close-knit, with greater discord between family members. Although the emphasis here has been on mediating parental alcoholism, other forms of parental psychopathology (Henderson et al., 1994) and co-morbid psychopathology (Fitzgerald, Zucker, & Yang, 1995) have similar effects on the home environment.

Child Characteristics. As mentioned, developmental tasks for mastery of middle childhood include healthy adjustment with respect to school, peers, and self-concept. Competencies or deficits in these domains do seem to play an important role in the etiology of antisocial behavior.

In Fergusson et al. (1996), children with pervasive antisocial behavior had the lowest self-esteem whereas non-problem behavior children had the highest self-esteem; remitting or late onset children were intermediate. Using reciprocal effects analysis to take into account the fact that involvement in delinquent behavior may shape self-esteem at the same time that self-esteem influences delinquency risk (i.e., lagged and contemporaneous influences), Rosenberg, Schooler, & Schoenbach (1989) found evidence for both processes. Low self-esteem was significantly related to an increase in delinquency over two years and there was a trend, although non-significant, towards an association between earlier delinquency and increased self-esteem.

Campbell et al. (1986) found that hard-to-manage children at age six were less involved and skilled in social activities as indicated by social withdrawal, participation in organized social groups, and play with peers. By age nine, children with persistent problems from 3-6 years of age were still scoring lower in social competence and higher on peer problems than those with desisting behavioral difficulties and a control group

(Campbell & Ewing, 1990). In general, Campbell (1994, p. 163) argued that children with persisting behavioral difficulties are “not only more oppositional, impulsive, inattentive, and disorganized, but also more socially anxious and incompetent” and that this “may interfere with social development in the family and peer group.”

Academic competence is another important risk marker. In Farrington’s (1995) longitudinal study, future juvenile delinquents were more likely to have low intelligence and low school achievement (Farrington, 1995). Early aggression was also prognostic of less achievement in academic domains (e.g., lower scores for spelling, reading, arithmetic, education, and occupational status) for both males and females in the longitudinal research of Eron et al. (1987). Similarly, the individual-level risk factors for continuity in behavior problems reported by Fergusson et al. (1996) included lower IQ and poor school achievement. Verbal IQ was consistently related to delinquency in five major longitudinal studies (Elkins et al., 1997), and low IQ seems to be a useful predictor of behavior problems that persist (Elkins et al., 1997). In Moffitt (1990), delinquents with attention deficit disorder scored lower on verbal IQ and reading proficiency than controls; however, there were no differences between controls, children with only attention deficit disorder, and children with only delinquent behavior patterns. Loeber and Dishion (1983) reported on the predictive utility of educational attainment, particularly low vocabulary and poor verbal reasoning. It should be noted that although poor school performance is often cited as a contributor to later delinquency, high scholastic achievement can also be considered a promotive factor. For example, doing well in school may lead to greater “stakes in conformity” and strong conventional social bonds (Hirschi, 1969) – with a decreased willingness to engage in problem behavior that

might jeopardize the promise of educational attainment or career success. Good grades also contribute to higher levels of self-esteem (Rosenberg et al., 1989).

Summary and Hypotheses

Most likely, continuity in antisocial behavior over time is the result of transactional processes that take place between a child's constitutional nature, parental characteristics, and the social environment (Keenan & Shaw, 1994; Huesmann et al., 1984). In other words, researchers need to adopt an ecological and family systems perspective to understand the origin and persistence of childhood behavior problems. Within the studies reviewed here, key influences on the course and persistence of antisocial behaviors include early undercontrol/impulsivity/hyperactivity, temperament, intelligence, lower social competency, low educational achievement, family conflict, negative control or harsh discipline, maternal depression, parental alcoholism, parental antisociality and criminality. Alternately, intelligence, personal competencies, and support in the caregiving environment have been identified as primary proximate buffers against maladaptation (Egeland, Carlson, & Sroufe, 1993; Masten & Coatsworth, 1998; Masten et al., 1999; Werner, 1986; Werner & Smith, 1982; Wyman, Cowen, Work, Hoyt-Meyers, Magnus, & Fagen., 1999), as per the literature on resilience.

The current study will approach the issue of continuity with several key assumptions based on findings from the extant literature. First, given the influences of personal characteristics and environmental conditions, initial predispositions toward antisocial behavior must consist of individual-level (e.g., temperament) and family-level (e.g., parental psychopathology) components. Second, these risks appear to have their effect on later behavior patterns through more proximate influences, such as family

environment and academic, social, or other personal competencies. This supports the inclusion of middle childhood factors (e.g., family environment, discipline, academic competence, self-esteem, social problems) in predicting outcome. However, and third, middle childhood factors do not necessarily reflect initial risk levels. The fact that they are influenced by what preceded them, developmentally (e.g., early temperament and lifetime parental psychopathology), means that stability is probably the stronger trend; old risks and protections can maintain the behavioral trajectory. However, new risks and protections in the immediate context may serve as “turning points” (Sampson & Laub, 1993). Whether or not behavioral difficulties persist, remit, begin, or remain absent from preschool to adolescence most likely depends on whether risk persists, remits, begins, or remains absent as well as whether protection/promotion comes into play.

Finally, some factors can be both risks and protections— depending on their valence and the initial behavioral trajectory. For example, low academic achievement may be a risk factor for those without preschool adversity, whereas high academic achievement can be a “turning point” for high-risk preschoolers. Similarly, high self-esteem may protect at-risk youth from the effects of family dysfunction, whereas low self-esteem may potentiate the effects of adversity. Even within behavior trajectories, proximate influences can have a risk or promotive influence. This argument is made by Jansen, Fitzgerald, Ham, & Zucker (1995): Children with difficult temperament from chaotic, stressful, conflicted homes are more likely to be at-risk for substance use, whereas a stable, warm, non-alcoholic home can buffer the child from a risk trajectory. As Rutter (1987, p. 318) remarked, “it seems helpful to use the term ‘protective mechanism’ when what was previously a risk trajectory is changed to one with a greater

likelihood of an adaptive outcome... Conversely, the process would be labelled a vulnerability one when a previously adaptive trajectory is turned into a negative one.” Both processes are explored in the current study (e.g., for the high-low and low-high antisocial behavior group classifications). Therefore, risk and protection will be viewed as opposite ends of a single continuum, dependent on an individual’s circumstances rather than a priori determinations, for ease of interpretation.

Four hypotheses will be tested in the current study – each supported by findings from the extant literature. The first two hypotheses test a strong continuity model of problem behavior, and the idea of an antisocial behavior pathway into first alcohol use.

Hypothesis 1. The primary pathway into early first alcohol use begins with heightened levels of antisocial behavior from as early as 3-5 years of age, onward.

This hypothesis is derived from literature suggesting that an early Age of First Drink (AFD) and adolescent substance use is associated with later problem behaviors, risky personality traits, and an earlier history of behavioral undercontrol (e.g., Caspi et al., 1996; Cloninger et al., 1988; Gruber et al., 1996; Masse & Tremblay, 1997; Tarter, 1988; Wills et al., 1995); typologies linking childhood antisocial behavior to alcoholic risk (e.g., Cloninger et al., 1996; Zucker, 1987, 1994) combined with evidence that AFD is a marker for this risk (e.g., Grant & Dawson, 1997; Gruber et al., 1996; McGue et al., 2001b; Prescott & Kendler, 1999; Schukit & Russell, 1983); and literature demonstrating strong continuity in antisocial behavior problems for a subset of individuals (e.g., Cairns et al., 1989; Campbell, 1994; Campbell et al., 1986; Campbell et al., 1994; Campbell &

Ewing, 1990; Eron et al., 1987; Farrington, 1995, 1997; Fergusson et al., 1996; Robins, 1978; Sampson & Laub, 1993; Shaw et al., 1994) as early as infancy. Early drinkers are expected to have higher stability coefficients for aggression and delinquent behavior from preschool through adolescence.

Hypothesis 2. Preschool and adolescent levels of antisocial behavior problems are distinguishing characteristics along the developmental course leading to and through drinking onset – and more specifically, antisocial behavior problems in these two time periods can be used to categorize individuals into groups that differ in the likelihood of early first drink.

To determine whether this simplified classification scheme is an adequate representation of risk trajectories for early alcohol use, children will be grouped into one of four classifications: high-high, low-low, low-high, and high-low in antisocial behavior during preschool (3-5 years of age) and early adolescence (12-14 years of age), respectively. Early drinkers are expected to show persistent behavioral difficulties and therefore be disproportionately found in the high-high group for both aggression and delinquent behavior. It is expected that those without early drinking onset will be disproportionately found in the low-low group for both aggression and delinquent behavior. Both findings would provide support for the strong continuity model of development described first in Hypothesis 1.

Also of interest are change patterns. Not all difficult children will become delinquent adolescents and not all delinquent adolescents were difficult children.

Children with remitting antisocial behaviors and children with escalating antisocial behaviors over the course of development may be at intermediate risk for early onset of alcohol use. Another goal of the current study, therefore, is to determine the influences that lead to continuity versus change in the development of antisocial behavior.

As mentioned in the literature review, predispositions may establish the initial risk trajectory but risk and promotive factors over the developmental course can serve as maintenance structures or “turning points” for behavior. Important distal risk factors for behavior problem, crime/delinquency, and substance use trajectories – which, as previously described, are heavily intertwined – include temperament (e.g., Campbell & Ewing, 1990; Caspi et al., 1996; Henry et al., 1996; Jansen et al., 1995; Loukas et al., 2001; Masse & Tremblay, 1997; Tarter, 1988; Tarter & Vanyokov, 1994; Tremblay et al., 1994; White et al., 1994) and parental psychopathology (e.g., Elkins et al., 1997; Farrington, 1995; Fitzgerald et al., 1993; Fitzgerald et al., 1995; Loeber & Dishion, 1983; Loukas et al., 2001; Sampson & Laub, 1993). Important proximate middle-childhood influences on behavior problems, crime/delinquency, and substance use trajectories include facets of the family environment (e.g., Barrera et al., 1995; Campbell & Ewing, 1990; Campbell, 1994; Cohen et al., 1994; Farrington, 1995; Fergusson et al., 1996; Hawkins, 1997; Hecht, 1973; Henderson et al., 1994; Hops et al., 1999; Hussong & Chassin, 1997; Hyphantis et al., 1991; Jacob et al., 1991; Roosa et al., 1990; Sampson & Laub, 1993; Zucker & Gomberg, 1986) and personal competencies or deficits in domains such as academic achievement (e.g., Eron et al., 1987; Farrington, 1995; Fergusson et al., 1996; Loeber & Dishion, 1983; Zucker & Gomberg, 1986), self-esteem (e.g., Fergusson et al., 1996; Rosenberg et al., 1989; Werner, 1986), and social relationships/problems

(e.g., Campbell, 1994; Campbell & Ewing, 1990; Campbell et al., 1986; Zucker & Gombert, 1986). A third hypothesis therefore addresses continuity versus discontinuity in antisocial behavior problems within the context of initial risk profiles and middle childhood risk/promotive factors:

Hypothesis 3: On all measures of risk from preschool (temperament, parental psychopathology) and middle childhood (academic achievement, self-esteem, social problems, family cohesion, family expressiveness, family conflict, physical discipline/negative control), scores will be least favorable for the high-high group, moderate for the low-high and high-low groups, and most favorable for the low-low group. If supported, this would corroborate the findings of Fergusson et al. (1996).

The high-high group is expected to have the following characteristics relative to the low-low group: more difficult temperament (i.e., high activity, short attention span, more reactivity) and more parental psychopathology (mothers and fathers) at 3-5 years of age; as well as less academic achievement (i.e., lower scores on reading, spelling, and arithmetic), lower self-esteem, more social problems, less family cohesion, less family expressiveness, more family conflict, and more physical/negative punishment at 6-8 and 9-11 years of age. The high-low and low-high groups are expected to have in-between scores on each of these dimensions.

In addition, what are the relative contributions of these early risk and middle childhood factors for explaining levels of antisocial behavior? Are early risks more important than later influences, or do later – more proximate – influences take precedence? The fourth hypothesis speaks to these issues:

Hypothesis 4: Initial risk trajectory variables will predict aggression and delinquent behavior during the preschool years, and initial risk trajectory variables along with middle childhood variables will predict adolescent aggression and adolescent delinquent behavior. Middle childhood factors will boost the explanatory power of the model (i.e., significantly add to the R^2) and eclipse some of the early indicators of risk – recognizing both that risk is fluid (i.e., early indicators don't always pan out), and that proximate influences are likely to have greater effect.

With regard to the initial risk load, antisocial behavior in preschool and adolescence will be predicted from more difficult temperament (i.e., higher activity levels, shorter attention span, and greater reactivity) and exposure to more parental psychopathology. Adolescent antisocial behavior will also be predicted by child characteristics (less academic achievement, lower self-esteem, and more social problems) and the family environment (i.e., less cohesion, less expressiveness, more conflict, and more physical punishment/negative control) in middle childhood.

A heuristic model is depicted in Figure 2. In this model, early risks determine the level of antisocial behavior observed during preschool. However, these behavior patterns can either be maintained by experiences in middle childhood or modified by them (i.e., with respect to “turning points”). Middle childhood variables are therefore seen as mediators in the relationship between early risks and later antisocial behaviors. Arrows for antisocial behavior are intended to represent an ongoing developmental trajectory that moves to and through middle childhood, into adolescence.

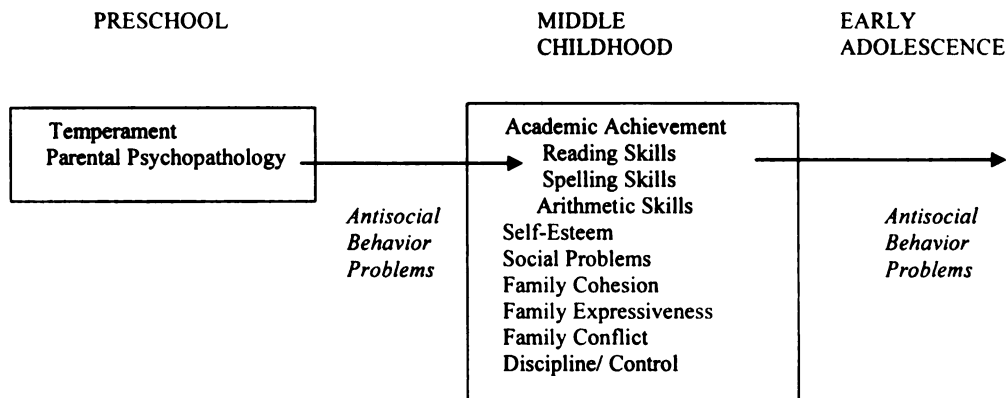


Figure 2.

Heuristic model of the relationship between the initial risk trajectory and middle childhood factors to antisocial behavior problems from preschool through early adolescence.

CHAPTER 3 METHODOLOGY

Participants

Participants were 220 male adolescents and their parents from the prospective Michigan Longitudinal Study (Zucker, Fitzgerald, Reif, Puttler, Pallas, & Ellis, 2000). This ongoing project utilized population-based recruitment strategies to identify alcoholic and ecologically-matched comparison families. Families needed to be intact and biological, with at least one male child between the ages of 3-0 and 5-11 at the time of recruitment. Almost all participants in the Michigan Longitudinal Study are non-Hispanic European. All were residents of the mid-Michigan area at the initial assessment, with income levels mostly within the low-middle class range (see Zucker et al., 2000, for additional details). The data used herein were collected at recruitment and when the children were approximately 3-5 (Wave 1), 6-8 (Wave 2), 9-11 (Wave 3), and 12-14 (Wave 4) years of age.

Alcoholic families were identified on the basis of father's drinking status. Although maternal drinking was assessed, maternal alcoholism was neither a requirement nor a basis for exclusion. However, families with children manifesting characteristics associated with a diagnosis of fetal alcohol syndrome, or with mothers who consumed six or more drinks per day during pregnancy, were excluded from participation. Alcoholic fathers were recruited from drunk driving records or neighborhood recruitment.

Of all convicted drunk drivers in a four county Mid-Michigan area, all males meeting the study criteria of a male child between 3-0 and 5-11 in an intact biological household with a blood alcohol concentration (BAC) of 0.15% (150 mg/100 ml) or higher when arrested – or a BAC of 0.12% with a history of prior alcohol-related driving

offenses – were asked permission for contact by study staff. At initial contact, a positive alcoholism diagnosis was established using the short Michigan Alcohol Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975) and later verified with the NIMH Diagnostic Interview Schedule – Version III (DIS; Robins, Helzer, Croughan, & Ratcliff, 1980) and the Drinking and Drug History questionnaire (Zucker, Fitzgerald, & Noll, 1990). All of these men met a “definite” or “probable” criterion for alcoholism using the Feighner Diagnostic Criteria (Feighner, Robins, Winokur, Guze et al., 1972).

Alcoholic fathers were also recruited from the neighborhoods in which the drunk driver alcoholic fathers resided. Community alcoholics were identified during neighborhood canvassing for non-alcoholic control families (see below) by positive alcoholism screenings. These men also met Feighner criteria for probable or definite alcoholism, and met the same inclusion criteria as the drunk-driving group for age of a biological male target child and coupled status.

In addition to alcoholic families, a group of community control families were recruited via door-to-door community surveys. These families were matched to a subset of alcoholic families on the basis of age for the male target child (within six months) and socio-economic status (from the same neighborhood or in a similar census tract). Neither mother nor father met Feighner criteria for alcoholism or other drug abuse/dependence.

For inclusion in the current sample, there were two criteria. The first was the availability of behavior problem ratings at Wave 1 and Wave 4. These constituted major dependent variables in analyses herein, so the decision was made to avoid missing data imputation on these key measures. Second, families must have participated in at least one of the middle childhood waves – Wave 2 and/or Wave 3. This requirement helped to

ensure that missing data imputation for middle childhood measures was at least partially based on other middle childhood measures (i.e., using Wave 2 information to help impute Wave 3 values, or using Wave 3 information to help impute Wave 2 values). Of the 333 families from whom some data have been collected as part of the larger Michigan Longitudinal Study, 220 cases met the current criteria. All 220 valid cases had information on age of first drink, so it was not necessary to use the availability of onset information as a criterion for inclusion or exclusion.

Unfortunately, sibling data collection did not begin at the time of family recruitment. A majority of females with data on onset of drinking were ages 6 to 11 when they were invited to participate in the longitudinal study. Only a small number of girls provided information at both Wave 1 and Wave 4 (approximate $n = 20$). This is inadequate for a statistical analysis of gender differences. However, there are documented gender differences in the development of antisocial behavior – for example, in the stability of aggression (e.g., Cairns et al., 1989; Cummings et al., 1989; Eron et al., 1987). It is for this reason that females were excluded.

Procedure

Data were collected by trained project staff blind to family alcoholism status. At each wave, the visits involved approximately 15 hours of contact time for each parent and seven hours of time for the target child. Contacts included questionnaire sessions, semi-structured interviews, and interactive tasks. Families were compensated for their participation.

Child Measures

First Drink. Alcohol use was measured by self-reports from adolescents on a Drinking and Drug History Form for Children questionnaire at 12-14 years of age (Wave 4). This questionnaire is the child's version of the Drinking and Drug History Form for Adults (Zucker, Fitzgerald, & Noll, 1990). The adult questionnaire incorporates already much tested items from the 1978 NIDA Survey (Johnston et al, 1979), from the American Drinking Practices Survey (Cahalan, Cisson & Crossley, 1969), and from the V.A. Medical Center (University of California) San Diego, Research Questionnaire for Alcoholics (Schuckit, 1978). All of the items have been extensively used in a variety of survey and clinical settings. They provide data on quantity, frequency and variability of alcohol consumption, frequency of drug use, and multiple questions on consequences and troubles related to the use of these substances. Items on the child version have been modified to be relevant for children and youth, but they cover the same substantive areas. They also cover expectancies about use, extent of exposure to alcohol-related incidents among parents and peers, and perceived availability.

Regarding onset, adolescents were asked at what age they first tried alcohol, excluding just a "sip" from an adult. If any age was listed, onset was categorized as "yes" and adolescents were included in the "first drink" group. Indications of not yet having tried alcohol led to a "no first drink" classification. Onset is therefore a dichotomous variable (0 = no first drink by 12-14; 1 = first drink by 12-14). Onset information at Wave 4 was missing in two cases, but could be constructed from data available at other waves. In the first case, the individual reported having had a first drink at 12 years of age on the same instrument administered at Wave 5 (15-17 years) as well

as during separate annual assessments at ages 14 and 15; this resulted in a “first drink” classification. In the second case, the individual reported not having had a first drink at the Wave 3 assessment as well as the annual assessment at age 14; this resulted in a “no first drink” classification. In total, 25% of the sample ($n = 55$) had a first drink by 12-14 years of age, whereas 75% had not ($n = 165$).

Antisocial Behavior. Child antisocial behavior was measured with the delinquent behavior and aggressive behavior symptom subscales of the Child Behavior Checklist (Achenbach, 1991; Achenbach & Edelbrock, 1983) at all waves. Maternal ratings were used. Items in each subscale are listed in Table 2. The 118-item CBCL is a parent-report measure of the prevalence and severity of child behavior problems. It is intended to provide an objective assessment of the target child’s social and emotional functioning. The CBCL yields raw and standardized scores on eight narrowband symptom scales, two broadband subscales on externalizing and internalizing behaviors, and a total behavior problems score. Narrowband subscales are comprised of items that co-occur in ratings among clinically-referred children and load together in principal components analysis (Achenbach, 1991).

The CBCL is a widely used instrument, with high validity and reliability (Achenbach, 1991). Although the CBCL was normed on children 4 to 16 years old, it is also administered to parents of children under four years of age because the Michigan Longitudinal Study began before the publication of the CBCL for 2-3 year olds. Scores for these younger children must be interpreted with caution, but Fitzgerald et al. (1993) found no substantive differences in three-year-old children's scores and those obtained by four- and five-year-olds (Reider, 1991). Test-retest reliability of item scores on the

CBCL range from .95 at a one-week interval, to .84 at a three-month interval (Achenbach, 1991). Parent agreement in the item scores was .99. Similar reliabilities were established on scale scores and the total problem score with one-week test-retest of .89. The median parent agreement on scale scores was .66. Adequate construct validity was established by correlations between CBCL scores and scores on a wide range of other measures of child behavior problems.

Behavior problems are rated on a three-point scale (0 = Not true; 1 = Somewhat or sometimes true; 2 = Very true or often true). Items are summed for each narrowband subscale, with higher scores reflecting more behavior problems. One item (alcohol and other drug use) was omitted from the delinquent behavior subscale to prevent artificial inflation of the correlation between delinquent behavior and drinking onset status. There are twenty aggressive behavior items (for a possible score range of 0-40) and 12 delinquent behavior items (for a possible score range of 0-24).

Continuity and discontinuity in aggressive behavior and delinquency were operationalized, independently, by division of children into four groups: those who were high-high, low-low, low-high, and high-low on the subscale score at Wave 1 (3-5) and Wave 4 (12-14), respectively. Mean splits were used to classify subjects as high or low on antisocial behavior at each wave. This allowed for adequate sample size in each cell of the 2 x 2 classification (see Figure 3).

Table 2

Items on the CBCL Delinquent Behavior and Aggressive Behavior Subscales

Delinquent Behavior

- 26. Doesn't seem to feel guilty after misbehaving
- 39. Hangs around with others who get in trouble
- 43. Lying or cheating
- 63. Prefers being with older kids
- 67. Runs away from home
- 72. Sets fires
- 81. Steals at home
- 82. Steals outside the home
- 90. Swearing or obscene language
- 96. Thinks about sex too much
- 101. Truancy, skips school
- 106. Vandalism

Aggressive Behavior

- 3. Argues a lot
- 7. Bragging, boasting
- 16. Cruelty, bullying, or meanness to others
- 19. Demands a lot of attention
- 20. Destroys his/her own things
- 21. Destroys things belonging to his/her family or others
- 22. Disobedient at home
- 23. Disobedient at school
- 27. Easily jealous
- 37. Gets in many fights
- 57. Physically attacks people
- 68. Screams a lot
- 74. Showing off or clowning
- 86. Stubborn, sullen, or irritable
- 87. Sudden changes in mood or feeling
- 93. Talks too much
- 94. Teases a lot
- 95. Temper tantrums or hot temper
- 97. Threatens people
- 104. Unusually loud

Note. One item from the delinquent behavior subscale was dropped (105. Uses alcohol or drugs for non-medicinal purposes) because it is a confound in analyses linking antisocial behavior problems with first drink onset.

		Behavior Problems in Adolescence	
		Low	High
Behavior Problems in Preschool	Low	<i>Low-Low</i>	<i>Low-High</i>
	High	<i>High-Low</i>	<i>High-High</i>

Figure 3.

Continuity and discontinuity classification groups for aggression and delinquent behavior.

Means for aggressive behavior were 10.65 at Wave 1 and 7.06 at Wave 4 (with observed scores ranging from 0-27 and 0-26, respectively). Means for delinquent behavior were 1.88 at Wave 1 and 1.85 at Wave 4 (with observed scores ranging from 0-10 and 0-12, respectively). The distribution of groups is presented in Table 3 in the marginals, along with the overlap between classification categories. Numbers on the diagonal represent consistency across behavior problem domains for severity at each time period (e.g., low-low on both aggressive and delinquent behavior). Although consistency is the stronger trend, there is also considerable disagreement of classifications across domains. For example, twenty children classified as low-low on aggressive behavior were classified as high-low on delinquent behavior. This supports the separation of the two behavior problems for analysis.

Table 3

Distribution of Groups Based on Sample Means at Wave 1 and Wave 4 for Aggression and Delinquent Behavior

		Delinquent Behavior				
		Low-Low	Low-High	High-Low	High-High	<i>Total N</i>
Aggression						
Low-Low	51	7	20	11	89	
Low-High	6	11	1	9	27	
High-Low	11	1	17	10	39	
High-High	8	7	9	41	65	
<i>Total N</i>	76	26	47	71	220	

Table 4 shows the correlations between aggression and delinquent behavior ratings at Wave 1 and Wave 4. From preschool to early adolescence, there are moderately strong auto-correlations for aggression and delinquent behavior. Even higher correlations are found within each time period between aggression and delinquent behavior, especially during early adolescence. However, correlation isn't equivalence. For example, despite being conceptually different, height and weight are highly related; ASPD and alcoholism are often co-morbid. Also, although there is considerable shared variance (with $r^2 = .21$, or 21%, at Wave 1 and $r^2 = .49$, or 49%, at Wave 4), there is considerable variance unique to each behavior problem as well; even in early adolescence, where there is the most convergence, more than half is left unexplained (i.e., $1 - r^2 = .51$, or 51%).

Table 4

Correlations Between Aggression and Delinquent Behavior

Variable	1	2	3	4
1. Wave 1 Aggression	1.00			
2. Wave 1 Delinquent Behavior	.456 **	1.00		
3. Wave 4 Aggression	.444 **	.295 **	1.00	
4. Wave 4 Delinquent Behavior	.341 **	.448 **	.701 **	1.00

** $p < .01$

Temperament: Activity, Attention Span, Reactivity. Temperament was measured by the Dimensions of Temperament Survey (DOTS; Lerner, Palermo, Spiro III, & Nesselroade, 1982) at Wave 1 and Wave 2. The DOTS is a 34-item questionnaire that measures five dimensions of temperament: activity level, attention span/distractability, adaptability/approach-withdrawal, rhythmicity, and reactivity. The dimension scores are based on sums of item ratings (1 = True; 0 = False) for each scale. Ratings from mother on child for activity (3 items; e.g., “My child moves a great deal in his/her sleep”), attention span/distractability (11 items; “My child persists at a task until it’s finished”), and reactivity (6 items; “My child reacts intensely when hurt”) were used. These dimensions were chosen because of their relevance to externalizing behavior problems in other studies (e.g., Mun, Fitzgerald, von Eye, Puttler, & Zucker, 2001; Wong, Zucker, Puttler, & Fitzgerald, 1999) and because they tap into constructs similar to attention

deficit disorder with hyperactivity, which has been associated with aggression and other problem behaviors in the extant literature (e.g., Campbell et al., 1986). Higher scores reflect more activity, longer attention span, and greater reactivity.

At Wave 3, the revised DOTS-R (Windle & Lerner, 1986) was used to measure temperament because of a change in protocol. The DOTS-R has 54 items rated on a four point scale, from “usually false” to “usually true” about the child. There are nine attributes assessed; of these, three overlap considerably with the DOTS attributes used in this study: Activity-general (7 items), activity-sleep (4 items), and task orientation (8 items). Reactivity is not assessed by the DOTS-R but some of the DOTS items measuring reactivity overlap with the DOTS-R dimension activity-general (e.g., “My child can’t sit still for long”).

Academic Achievement in Middle Childhood. Academic achievement was measured with three subscales on the Wide Range Achievement Test – 3rd Edition (WRAT; Wilkinson, 1993) from Wave 2 and Wave 3, using standardized scores. The WRAT 3 assesses three areas of academic competence: Reading, Spelling, and Arithmetic. It was standardized on 5000 individuals ranging in age from 5 to 74. Test-retest reliability is .91 to .98. The WRAT 3 is an established screening tool for identifying children at risk for learning disabilities.

Global Self-Esteem in Middle Childhood. The Harter Perceived Competence Scale for Children (PCSC; Harter, 1978, 1979) was used to measure global self-esteem at Wave 2 and Wave 3. This instrument was designed to measure the child's sense of his or her own abilities and stature vis-à-vis other children. There are 36 items on this “What I Am Like” questionnaire, six for each of six subscales: scholastic competence, social

acceptance, athletic competence, physical appearance, behavioral conduct, and global self-worth. Responses are scored from 1 to 4. Statements represent feelings of competency (e.g., “Some kids are very happy being the way they are BUT other kids often wish they were different”). The child is asked to decide which type of kid is most like him, and whether this is only sort of true or really true for him. Scale scores are averages. Items were coded such that high scores reflect more positive self-evaluations.

The PCSC correlates low (.09) with a children’s social desirability scale (Crandall et al., 1965). Internal consistency shows reliability alphas ranging from .73 to .86 across all samples. Three month test-retest reliability ranges from .70 to .87 on the various subscales. Over nine months, reliabilities were between .69 and .80.

Social Problems in Middle Childhood. Social problems, thought to represent peer rejection or interpersonal awkwardness, were measured by maternal ratings on the social problems subscale of the Child Behavior Checklist (Achenbach, 1991; Achenbach & Edelbrock, 1983) for Wave 2 and Wave 3. Acting too young, not getting along with peers, and gets teased are examples of the statements on the social problems subscale. Items are rated on a three-point scale (0 = Not true; 1 = Somewhat or sometimes true; 2 = Very true or often true) and summed.

Parent Measures

Early/Lifetime Parental Psychopathology. Maternal and paternal psychopathology was assessed with a psychopathology index that is a composite of three measures from Wave 1: alcohol diagnosis, antisocial personality disorder, and depression. Together they comprise a baseline measure reflecting the parental component of an initial risk trajectory, whether this be environmentally or genetically

determined; the possibility of the latter is included via consideration of lifetime diagnoses.

As described, alcoholism was screened with the short Michigan Alcohol Screening Test (Selzer et al., 1975) and verified with the Diagnostic Interview Schedule – Version III (DIS; Robins et al, 1980) and Drinking and Drug History questionnaire (Zucker et al, 1990). On the basis of this information, a trained clinician made diagnoses for alcohol abuse and dependence using several coding strategies. For this study, the DSM IV diagnosis was used and diagnoses were scored as follows: 0 = no lifetime diagnosis, 1=alcohol abuse/dependence diagnosis but not in the last three years, and 2 = alcohol abuse/dependence diagnosis in the last three years (see also Zucker, Wong, Puttler, & Fitzgerald, 2003).

Parental lifetime antisocial personality disorder (ASPD) was assessed with the DIS and the Antisocial Behavior Inventory (Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996; Zucker, Noll, Ham, Fitzgerald, & Sullivan, 1994). Information on the Antisocial Behavior Inventory was used to supplement DIS data in establishing a diagnosis. For this study, ASPD was scored as follows: 0 = no diagnosis, 2 = presence of diagnosis (see also Zucker, Wong, Puttler, & Fitzgerald, 2003). Only the lifetime indicator was used here because diagnosis for this personality disorder requires that a long course of behavior over childhood and adulthood be established (Zucker et al., 2003). A score of 2 was also deemed appropriate given the seriousness of ASPD and its possible influence on child development; with this scoring system, having ASPD is accorded the same weight as recent alcohol abuse/dependence.

Depression was measured by the Beck Depression Inventory (BDI; Beck et al., 1961). The BDI is a widely used phenomenological measure of depressive state that has received extensive validation (Carroll et al., 1973). The short form of the BDI is a 12-item self-report instrument that assesses cognitive, emotional, motivational, and physical manifestations of depression on a scale of 0 to 3. Beck reports a split-half reliability of .93. Scores on the long and short forms of the BDI correlate between .89 and .97 (Beck, Steer, & Garbin, 1988). A meta-analysis of 25 years of data on the BDI yielded an internal consistency mean coefficient alpha of .86 for psychiatric patients and .81 for non-psychiatric subjects. According to Beck and Beck (1972), scores of 0-4 demonstrate no or minimal depression, scores of 5-7 demonstrate mild depression, scores of 8-15 demonstrate moderate depression, and scores greater than 15 demonstrate severe depression. For this study, scores were recoded as follows, based on these guidelines: 0 = no or minimal depression, 1 = mild depression, and 2 = moderate depression. There were no cases meeting criteria for severe depression in this sample.

The parental psychopathology index was computed by summing the alcohol diagnosis score, ASPD score, and depression score for each parent. Consequently, scores can range from 0 to 6 for mother and for father. Distributions are presented in Table 5.

Contextual Measures

Family Environment in Middle Childhood: Cohesion, Expressiveness, and Conflict. Family environment was measured with the Moos Family Environment Scale (FES; Moos, 1974; Moos & Moos, 1976) at Wave 2 and Wave 3. The FES is an empirically based taxonomy of family social environments as perceived by family

Table 5

Distribution of Scores on Psychopathology Dimensions for Mothers and Fathers

	Mother <u>N</u>	Father <u>N</u>
Alcohol Diagnosis		
0 = no	148	66
1 = yes, not last three years	31	31
2 = yes, within last three years	41	123
Antisocial Personality Disorder (ASPD)		
0 = no	214	185
2 = yes	6	35
Depression		
0 = no	167	177
1 = mild	32	27
2 = moderate	21	16

members. Maternal reports were used because the youth-report version is not available until Wave 3. Mothers had at least joint custody of the male target child in all but one case. This is important because it means that the family environment being reported was the one to which the child was primarily exposed. For the one case, maternal reports were retained at Wave 2 because the mother reported living with the child for 2 to 2.5 of the last three years – but were excluded at Wave 3 because the maternal report was for an environment that too infrequently included the child (i.e., 7-12 months over the three year time period).

The FES consists of ten scales that describe dimensions of family climate: Cohesion, Organization, Expressiveness, Conflict, Independence, Control, Achievement, Intellectual-Cultural Orientation, Active-Recreational Orientation, and Moral-Religious Emphasis. Cohesion, expressiveness, and conflict were used here. Cohesion refers to the extent to which family members are concerned about, committed to, and supportive of one another (e.g., “There is a feeling of togetherness in our family”). In expressive homes, family members are allowed to act openly and express their feelings (e.g., “There are a lot of spontaneous discussions in our family”). Family conflict describes the degree to which anger, aggression, and conflict interactions are characteristic of the home (e.g., “We fight a lot in our family”).

Each scale has nine items, and respondents are asked whether the statement for each item is more true or false for their family. Responses are summed, with higher scale scores representing more cohesion, expressiveness, and conflict.

Discipline in Middle Childhood. The Parent Daily Report (PDR) was used to measure physical discipline. The PDR is a revision of the same-named instrument at the Oregon Social Learning Center (Chamberlain, 1980). Parents reported a “yes” or “no” for the occurrence of a set of behaviors during the previous 24 hour during daily phone calls over a six day time period (3 to mother and 3 to father, on alternating days). Information is requested about 12 daily behaviors – six positive behaviors and six negative behaviors. At Wave 2, the item used to measure negative control/harsh punishment was, “Within the last 24 hours, did you spank or use other physical discipline with (your child)?” At Wave 3, the most similar item was, “Within the last 24 hours, did you use physical discipline with (your child)?” Both items were scored as no-yes responses (0 = no; 1 = yes). A

maximum score (1 or 0) was computed for each adult informant across their three interview days. If any spanking/physical discipline was reported by an informant, this resulted in an overall score of 1 = “yes” for that informant. Next, a maximum score (1 = any spanking or physical discipline; 0 = none) was computed *across* informants, including step-parents. If any spanking/physical discipline was reported on any day by any respondent, the final PDR score was a 1 = “yes”. Final PDR scores therefore reflect whether or not the target child experienced physical discipline by any parent during the report periods.

An additional instrument measured negative control exerted by parents in relationships with their children but was only administered at Wave 3: the Parent Perception Inventory. Because it provides more information about discipline style than the PDR, it was also included in Wave 3 analyses.

The Parent Perception Inventory (PPI; Hazzard, Christensen, & Margolin, 1983) for parents is an 18-item questionnaire developed to assess parenting styles. Half the items describe parenting behaviors with a positive orientation (positive reinforcement, comfort, talk time, involvement in decision making, time spent together, positive evaluation, allowing independence, assistance, and non-verbal affection); these are alternated with half that suggest a negative orientation (privilege removal, criticism, command, physical punishment, yelling, threatening, time-out, nagging, and ignoring). Responses are based on a five-point Likert scale (0=never; 4 = a lot). A summed total for the negative orientation items on the PPI were used to measure negative control or harsh punishment at Wave 3. The highest negative orientation scale score among all the

biological and step-parents who completed the PPI on the child and have adequate contact with the target child were included in the analyses.

Dropped Cases

Most of the missing data from the Michigan Longitudinal Study are missing by design. Data from some families were initially not collected due to funding restraints; when funding issues were resolved, data were then not collected from families with children who were too old for the study wave in question (particularly at Wave 2 and Wave 3). Much fewer data are missing due to non-administration or non-response.

Based on the criteria for inclusion in the study sample, described earlier, there were 220 valid cases. Of the 113 cases with insufficient data, 85 had less than three waves of (at least partially) collected data, CBCL inclusive.¹ Seven of those meeting the three-wave criteria were missing the CBCL at Wave 1, and 21 were missing the CBCL at Wave 4.

Screening for differences between the study sample ($n = 220$) and those dropped due to insufficient data ($n = 113$) revealed some bias. Chi-square tests showed significant differences for paternal alcoholism, $\chi^2 (2, 333) = 8.93, p = .012$; paternal antisocial personality disorder, $\chi^2 (1, 322) = 15.24, p = .000$; and maternal depression, $\chi^2 (3, 328) = 8.15, p = .043$.² Cases that were dropped were characterized by fathers who were more alcoholic (85% with at least a lifetime diagnosis, versus 70% in this sample) and more antisocial (35%, versus 16% in this sample) – as well as mothers who were more

¹ Two of the 220 cases included in the study were missing both middle childhood CBCL scores (i.e., Waves 2 and 3), but otherwise had nearly-complete data for at least three waves. Because there were actually very few missing data points for these cases, and the missing CBCL scores were from neither Wave 1 nor Wave 4, the data were retained.

² Non-significant differences were found for maternal alcoholism, $\chi^2 (2, 333) = .28, p = .872$; maternal ASPD, $\chi^2 (1, 332) = 3.58, p = .058$; and paternal depression, $\chi^2 (3, 326) = 6.89, p = .075$.

depressed (17% of with at least moderate depression, versus 10% in this sample). Note that sample size fluctuations across analyses here derive from missing values in the original data set by which the two groups can be compared (i.e., before imputation of missing values for the study sample).

Regarding child behavior problems, there were also significant differences in maternal ratings of aggression at Wave 4, $t(248) = 2.20$, $p = .029$ – with a mean of 9.4 for those dropped and 7.1 in the study sample. However, the two groups were statistically similar on Wave 1 aggression (mean of 10.93 versus 10.65, respectively; $t[319] = .39$, $p = .695$), Wave 1 delinquent behavior (mean of 2.33 versus 1.88, respectively; $t[319] = 1.95$, $p = .105$), and Wave 4 delinquent behavior (mean of 2.47 versus 1.85, respectively; $t[248] = 1.44$, $p = .152$). Nonetheless, for each of these variables, the means for the dropped cases were higher.

In general, the 220 cases included in the study sample are less at-risk than the families excluded from imputation due to insufficient data. In some ways, then, this will be a conservative test of the research hypotheses. Interpreted conversely, the study sample is slightly more normative than the larger Michigan Longitudinal Study subject pool.

Missing Data Imputation

For the 220 valid cases, the proportion of missing values for each variable is listed in Table 6. The range of missing data was from 0 – 20%, with a total of 8.6%. The average percentage of missing data was .3% at Wave 1, 18.1% at Wave 2, and 6.9% at Wave 3. There were no missing data at Wave 4, by virtue of the criteria used for inclusion in the sample (i.e., behavior problem ratings at Wave 1 and Wave 4 as well as

information on drinking onset). Recall that another criterion was participation in at least one of the middle childhood waves (Wave 2 or Wave 3) to ensure that missing data imputation for middle childhood measures were at least partially based on other middle childhood measures.

Missing values were imputed using the EM (expectation-maximization) method available in most statistical packages. The EM algorithm is an iterative procedure to find the maximum likelihood estimates of parameters for missing values using data from all observed variables.

Each iteration involves an E step and an M step. In the E step, conditional expectations for the missing data (i.e., a “best guess”) are derived from known values on observed variables and the current estimate of parameters. In the M step, expected values are substituted for the missing data and maximum likelihood estimation is used to produce new parameter estimates. These updated parameter estimates are considered in the next E step, with iterations repeating until convergence (i.e., when negligible change in parameter estimates from iteration to iteration is obtained). The result is that set of imputed values which best fits the existing covariance matrix – thereby maintaining observed relationships between variables in the dataset. Stated another way, “the basic principle of ML estimation is to choose as estimates those values that, if true, would maximize the probability of observing what has, in fact, been observed” (Allison, 2002, p. 13).

The EM method produces less bias in the results and is generally superior to methods such as list-wise deletion, mean substitution, and regression imputation (Collins, Schafer, & Kam, 2001; Graham & Hofer, 2000; Rovine & Delaney, 1990; Schafer &

Table 6

Proportion of Missing Cases by Variable

Variable	Number of Intact Cases	Proportion Missing
<u>Wave 1</u>		
Aggression	220	--
Delinquent Behavior	220	--
Activity	218	.01
Attention	219	<.01
Reactivity	219	<.01
Age	220	--
Maternal Alcoholism and ASPD	220	--
Paternal Alcoholism and ASPD	220	--
Maternal Depression	218	.01
Paternal Depression	219	<.01
<u>Wave 2</u>		
Aggression	183	.17
Delinquent Behavior	183	.17
Reading	180	.18
Spelling	178	.19
Arithmetic	180	.18
Global Self-Esteem	176	.20
Social Problems	183	.17
Family Cohesion	179	.19
Family Expressiveness	179	.19
Family Conflict	179	.19
Spanking	180	.18
Activity	181	.18
Attention	181	.18
Reactivity	181	.18
<u>Wave 3</u>		
Aggression	208	.05
Delinquent Behavior	208	.05
Reading	202	.08
Spelling	202	.08
Arithmetic	202	.08
Global Self-Esteem	206	.06

(table continues)

Table 6 (cont'd).

Variable	Number of Intact Cases	Proportion Missing
<u>Wave 3 (Continued)</u>		
Social Problems	208	.05
Family Cohesion	207	.06
Family Expressiveness	207	.06
Family Conflict	207	.06
Physical Discipline	195	.11
Negative Control	204	.07
Activity – General	205	.07
Activity – Sleep	205	.07
Task Orientation	205	.07
<u>Wave 4</u>		
Onset Group	220	--
Aggression	220	--
Delinquent Behavior	220	--

Graham, 2002; West, 2001). In order to estimate missing values accurately, data must be missing at random (MAR). Data are MAR for a variable if the pattern of missing values does not depend on the value of that variable (Allison, 2002). Missing completely at random (MCAR) involves an even stronger assumption: that the pattern of missing values doesn't depend on the value of the variable itself or any other variable in the data set (Allison, 2002).

Little's (1988) MCAR test uses all of the available data to produce a global chi-square statistic which speaks to the issue of whether the data are MCAR. Because the data in this study are presumed to be MAR (because of planned missingness and the role of age), not MCAR, this is a conservative test. Little's MCAR test resulted in a chi-

square = 1122.217 (df = 1078; $p = .17$) which indicates that no identifiable pattern exists to the missing data; the null hypothesis against random missingness is rejected, and missingness is considered ignorable for likelihood inferences (which require a minimum of MAR; Little, 1988).

Analytical Strategy

To test Hypothesis 1 (that at the core of differences associated with drinking onset status is a pattern of higher antisocial behavior from preschool through adolescence), levels of antisocial behavior from preschool to adolescence were compared between early drinkers (i.e., first drink group) and those who had not tried alcohol by 12-14 years of age (i.e., no first drink group). Next an autoregressive two-group (first drink, no first drink) stacked model in LISREL 8.52 was run with aggression at Wave 1, Wave 2, Wave 3, and Wave 4 measured by parental report on the CBCL (see Figure 4). This analysis was repeated for delinquent behavior problems. Preliminary analyses suggested a unique contribution of preschool antisocial behavior on later antisocial behavior – that is, that a path from Wave 1 to Wave 4 would need to be included in the model for adequate fit. This fits with the assumption of initial predispositions (internal or externally-driven) that contribute to the developmental process. Antisocial behavior becomes more normative in adolescence, but relative severity rankings from preschool difficulties are expected to remain stable from a hard-continuity perspective.

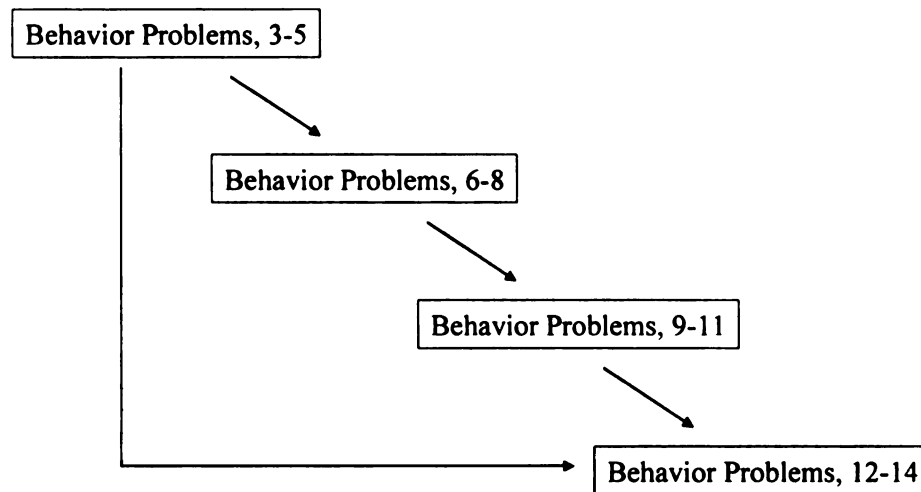


Figure 4.

Stacked (multi-group) model comparing early drinkers to non-drinkers at 12-14.

Configural frequency analysis (CFA; von Eye, 1990, 2002) was used to test Hypothesis 2 (that classification into high-high, low-low, low-high, and high-low groups using preschool and early adolescent antisocial behavior scores significantly overlap with early drinking onset – such that early drinking is disproportionately associated with strong continuity at the high end [i.e., high-high] and avoidance of drinking onset is associated with strong continuity at the low end [i.e., low-low]). The four longitudinal behavior classifications (e.g., recall Figure 3) were cross-tabbed with drinking onset in a 4 (low-low, low-high, high-low, high-high) x 2 (drinking onset: no, yes) configuration. Separate analyses were run for aggressive behavior classifications and the delinquent behavior classifications.

Using CFA, researchers ask whether cells contain more or fewer cases than expected from some chance model. A cell with more cases than expected is a “type;” a cell with fewer cases than expected is an “antitype.” Data were analyzed under the

assumption of total independence which dictates that the classifications are not related at all; if this is true, neither types nor antitypes will be revealed. Lehmacher's test (L, Lehmacher, 1981) was used for significance testing of types and antitypes with a Bonferroni-adjusted alpha level ($\alpha^* = 0.05/8 \text{ cells} = .00625$). If Hypothesis 2 is supported, greater than expected frequencies will be found for the high-high and low-low groups with respect to first drink (i.e., there will be more cases than expected in the high-high drinking onset cell, and there will be more cases than expected in the low-low no-onset cell).

To test Hypothesis 3 (that discontinuity patterns are marked by intermediate levels of risk), group differences on variables from preschool and middle childhood were examined with Analysis of Variance (ANOVA). Analyses were run separately for aggression and delinquent behavior classifications. Exact *p*-values are reported (except where $p < .001$) so that the reader can evaluate the risk of Type 1 error.

Finally, regressions were employed to test Hypothesis 4 (that early risk load will predict Wave 1 antisocial behavior, that early risk factors *as well as* middle childhood variables will predict Wave 4 antisocial behavior, and that more proximate influences will have a stronger effect than more distal influences).

Age at Wave 1 (from the CBCL administration) was included as a control variable. Since participants were re-interviewed every three years, this is a proxy measure for age at each wave. Recall that each wave covers a range (i.e., Wave 1, 3-5; Wave 2, 6-8; Wave 3, 9-11; Wave 4, 12-14); if any of the measures have age-related variance, including age in the model will help control for it. Regressions were run

separately for aggression and delinquent behavior, and separately within each for Wave 2 and Wave 3 predictors.

The *Baseline Model* includes child temperament and the composite parental psychopathology measure for mothers and fathers. These variables reflect the initial risk trajectory. It is especially important to include parental diagnostic variables in the model because of the nature of the sample (i.e., non-alcoholic families, alcoholic families with fathers convicted of drunk-driving, and community alcoholic families without a criminal history). Antisocial behavior is likely to be elevated among children of alcoholics – especially with co-morbid antisociality or depression.

The *Contextual Model* consists of risk and protective factors from the family environment during middle childhood. Predictors include family cohesion, expressiveness, and conflict, as well as discipline (i.e., negative control and/or physical punishment). The *Child Characteristics Model* consists of individual-level risk and protective factors from middle childhood. Predictors include academic achievement in three domains (reading, spelling, arithmetic), self-esteem, and social problems.

The Baseline Model alone was used in a regression for Wave 1 aggression and delinquent behavior. For Wave 4 aggression and delinquent behavior, it was tested in a first run to determine whether characteristics from preschool are sufficient to predict later behavioral trajectories. The other two models were then added in a second run. This second run 1) controlled for the influence of initial characteristics on later characteristics and 2) helped determine which variables are the most predictive of each antisocial behavior pattern. If only baseline predictors are significant in this second run, it would suggest that risk for hard-continuity or change is established by preschool and that

subsequent influences do little to disrupt the risk trajectory.

Finally, three additional sets of control variables were added in a hierarchical procedure. Temperament variables concurrent with the other middle childhood predictor variables were entered to control for the possibility of temperamental variability over time.³ Next, Wave 1 antisocial behavior was included to see whether early antisocial behavior predicts later antisocial behavior above and beyond more proximate influences, and to see whether other variables explain these outcomes above and beyond what is known about behavior problems from as early as three years old. The new temperament variables were expected to attenuate the relationship between Wave 1 temperament and antisocial behavior, and the inclusion of Wave 1 antisocial behavior was expected to make a significant contribution to explained variance. Later, the effect of controlling for contemporaneous (middle childhood) behavior problems was explored with similar expectations.

The rule of thumb for statistical power with regression is at least 10 cases per independent variable (VanVoorhis & Morgan, 2001). One alternate criteria is $N > 50 + 8m$ (where m is the number of predictors) for testing the multiple correlation and $N > 104 + m$ for testing partial correlations for individual predictors, assuming a medium-sized relationship between the dependent and independent variables (Green, 1991; VanVoorhis & Morgan, 2001). The largest model in this study contained 21 variables, with 220 cases (i.e., 10.5 cases per predictor, $50 + 8m = 218$, and $104 + m = 125$). Sample size therefore should not be prohibitive, although some caution is warranted.

³ Parental psychopathology – the other initial risk trajectory component – may also change over time but these changes should be accounted for by family environment characteristics (see literature review).

CHAPTER 4

RESULTS

Onset of First Drink

Hypothesis 1. The primary pathway into early first alcohol use begins with heightened levels of antisocial behavior from as early as 3-5 years of age, onward.

Means and standard deviations for each variable by drinking onset status can be found in Table 7. Graphical depictions can be found in Figure 5 for aggression means and Figure 6 for delinquent behavior means; recall that the possible range of CBCL scores was 0 to 40 for aggression (20 items), and 0 to 24 for delinquent behavior (12 items). T-tests revealed significant differences in symptomatology by drinking onset group for Wave 4 aggression and for delinquent behavior at three time periods: Wave 1, Wave 2, and Wave 4 (see Table 7). Figures 5-6 show much more marked group differences for delinquent behavior than for aggression.

In general, levels of aggression declined for both groups from 3-5 to 12-14 years of age. The only group difference was for aggression at 12-14, with early drinkers rated by their mothers as more aggressive. On the other hand, early drinkers were more delinquent than their never-drinking peers at all ages except 9-11—when the level of symptomatology for early drinkers appears to diminish and converge with that of those who have never tried alcohol, before rising again in early adolescence. Early drinkers therefore engaged in more delinquent activities at most time periods, with distinctly more aggression and delinquent behavior in the transition from late childhood to early adolescence.

To more precisely specify developmental differences in antisocial behavior

Table 7

Means (and Standard Deviations) by Drinking Onset Group for Aggression and Delinquent Behavior

Variable	Wave	<i>No First Drink</i> <u>M</u> (SD)	<i>First Drink</i> <u>M</u> (SD)	<i>t</i>
Aggression	1	10.47 (5.57)	11.20 (5.51)	-.85
	2	8.61 (5.14)	10.15 (5.40)	-1.90
	3	8.21 (5.69)	8.31 (5.87)	-.11
	4	6.58 (5.12)	8.47 (5.96)	-2.27 *
Delinquent Behavior	1	1.67 (1.28)	2.53 (1.96)	-3.75 **
	2	1.76 (1.32)	2.96 (1.99)	-5.01 ***
	3	1.57 (1.43)	1.99 (1.96)	-1.72
	4	1.45 (1.77)	3.05 (2.70)	-5.00 ***

Note. *** $p < .001$. ** $p < .01$. * $p < .05$; $df = 218$ for all tests; the same pattern of significance was found when equal variances were not assumed.

between early drinkers and non-drinkers, structural equation modeling was used to examine the influences of early antisocial behavior on later antisocial behavior. An autoregressive two-group (first drink, no first drink) stacked model in LISREL 8.52 was run separately for aggression and delinquent behavior problems at Wave 1 – 4, testing the model from Figure 4. All parameters were first allowed to vary across drinking onset groups. When a tenable model was obtained, these parameters were constrained to be equal in an invariant model to test for significant group differences. Covariance matrices for the first drink and no first drink groups are presented in Table 8 for aggression and Table 9 for delinquent behavior.

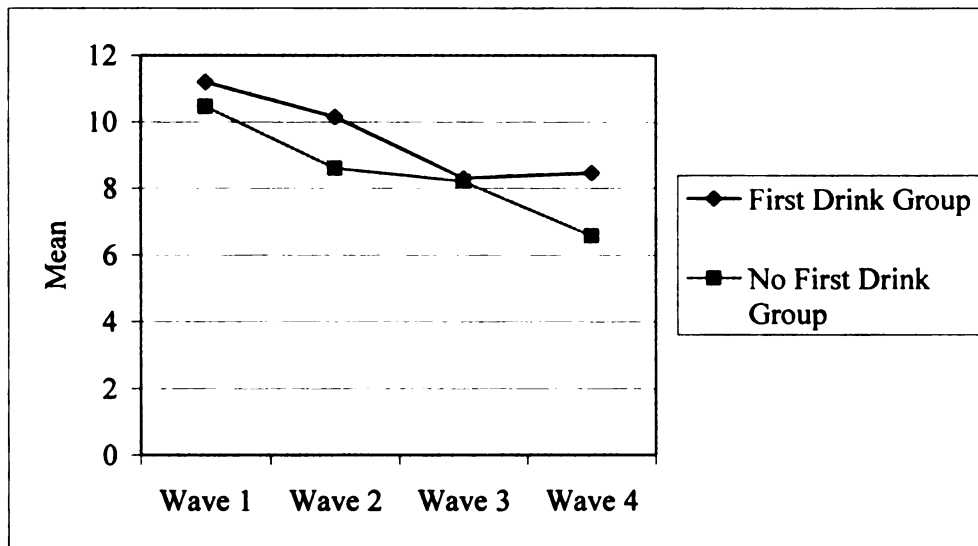


Figure 5.

Mean aggression scores from Waves 1-4 for first drink versus no first drink group.

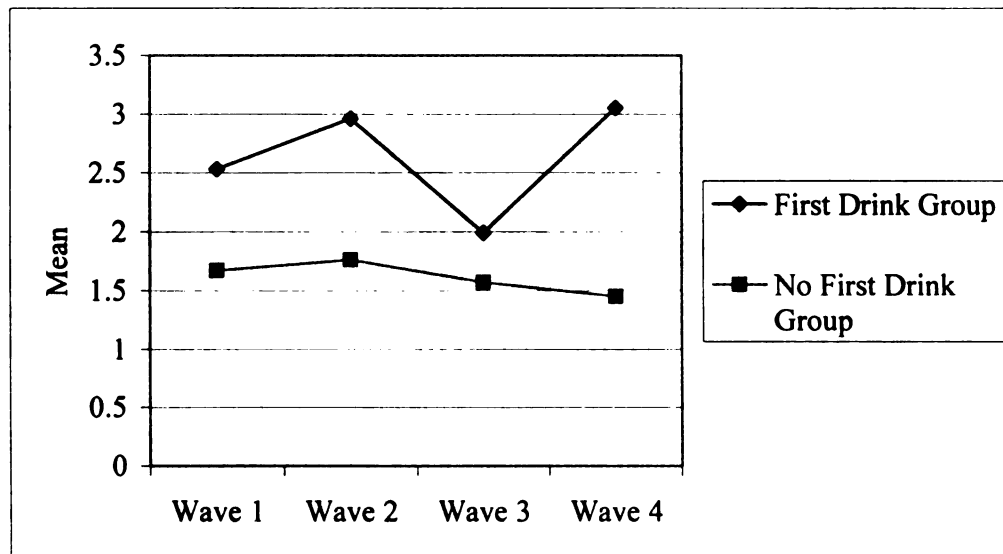


Figure 6.

Mean delinquent behavior scores from Waves 1-4 for first drink versus no first drink group.

Table 8

Covariance Matrix for Aggression by First Drink Onset Group

FIRST DRINK = NO	Wave 1	Wave 2	Wave 3	Wave 4
Aggression Wave 1	30.9943			
Aggression Wave 2	15.8370	26.4615		
Aggression Wave 3	12.0781	21.0290	21.3235	
Aggression Wave 4	11.4691	15.7427	19.9636	26.1991
FIRST DRINK = YES	Wave 1	Wave 2	Wave 3	Wave 4
Aggression Wave 1	30.3481			
Aggression Wave 2	19.9652	29.1121		
Aggression Wave 3	17.6258	22.6642	34.5139	
Aggression Wave 4	17.9778	16.9324	20.9610	35.5502

Table 9

Covariance Matrix for Delinquent Behavior by First Drink Onset Group

FIRST DRINK = NO	Wave 1	Wave 2	Wave 3	Wave 4
Delinquent Wave 1	1.6260			
Delinquent Wave 2	.7679	1.7528		
Delinquent Wave 3	.6228	1.0534	2.0516	
Delinquent Wave 4	.7012	1.2922	1.4708	3.1519
FIRST DRINK = YES	Wave 1	Wave 2	Wave 3	Wave 4
Delinquent Wave 1	3.8465			
Delinquent Wave 2	1.8846	3.9576		
Delinquent Wave 3	1.3157	2.8008	3.8338	
Delinquent Wave 4	2.7664	3.3493	2.6278	7.2862

The maximum likelihood (ML) method of estimating free parameters was employed; ML is a standard procedure used throughout much of the literature. Several criteria will be used to evaluate model fit. Of primary interest is the chi-square (χ^2) test, which measures discrepancies between the actual sample covariance matrix and the one generated by the estimated (fitted) model; χ^2 values should be low with $p > .05$ (relative to the degrees of freedom), indicating that the data and model matrices are not significantly different. Notably, it was the normal theory weighted least squares chi-square that was used for purposes of evaluation. If the model is tenable, the root mean square error of approximation (RMSEA) – which is a measure of discrepancy – should be less than .05. In addition, a value of at least .90 should be obtained for the normed fit index (NFI) and the comparative fit index (CFI), which indicate how much better the model fits (i.e., explains the observed variances and covariances) relative to a null model.

For aggression, the first run tested a model in which paths exist between each adjacent wave (i.e., without the path from Wave 1 to Wave 4). The result was poor model fit: $\chi^2 = 16.41$, $df = 6$, $p = .01$, RMSEA = .126, NFI = .96, CFI = .97. The path from Wave 1 to Wave 4 aggression was then added, resulting in a model with acceptable fit statistics: $\chi^2 = 3.79$, $df = 4$, $p = .43$, RMSEA = .000, NFI = .99, CFI = 1.00. This specification was a significant improvement, $\Delta \chi^2 = 12.62$, $\Delta df = 2$, $p < .01$.

For delinquent behavior, the first run tested a model in which paths exist between each adjacent wave (i.e., without the path from Wave 1 to Wave 4). The result was poor model fit: $\chi^2 = 36.17$, $df = 6$, $p < .001$, RMSEA = .215, NFI = .87, CFI = .89. The path from Wave 1 to Wave 4 delinquent behavior was then added, resulting in a model with the following fit statistics: $\chi^2 = 24.81$, $df = 4$, $p < .001$, RMSEA = .219, NFI = .92, CFI =

.93. This specification was a significant improvement, such that $\Delta \chi^2 = 11.36$, $\Delta df = 2$, $p < .01$, but additional changes were needed to meet acceptable fit criteria. Modification indices suggested a direct path between Wave 2 and Wave 4 delinquent behavior. A tenable model was achieved when this parameter was included in the model: $\chi^2 = 2.34$, $df = 2$, $p = .31$, RMSEA = .039, NFI = .99, CFI = 1.00.⁴ It was also a significant improvement in fit with $\Delta \chi^2 = 9.02$, $\Delta df = 2$, $p < .05$.

The common metric completely standardized solution can be found in Figure 7 for aggression and Figure 8 for delinquent behavior. Parameter estimates for the group with no first drink by 12-14 years of age are above each arrow; parameter estimates for the first drink group are below each arrow.⁵

For aggression, the severity of behavior problems at each wave significantly contributed to severity at adjacent waves for both groups. In addition, the severity of problems at 3-5 significantly predicted the severity of problems at 12-14 for both groups. This link between aggression in preschool and early adolescence was needed to achieve good model fit.

For delinquent behavior, one additional path was required to obtain adequate model fit: a link between delinquent behavior at 6-8 and delinquent behavior at 12-14. Although not hypothesized, this path makes sense given the developmental trajectories

⁴ When this path was added to the aggression model, $\chi^2 = 0.96$, $df = 2$, $p = .62$, RMSEA = .000, NFI = 1.00, CFI = 1.00. It was not an improvement in fit, however, with $\Delta \chi^2 = 2.83$, $\Delta df = 2$, $p > .05$. The path from Wave 2 → Wave 4 also was not statistically significant for either group.

⁵ Error terms are not depicted. The parameter estimates for the error terms were 1.01, .68, .48, and .47 for aggression at Waves 1-4, respectively, for the no first drink group and .98, .59, .51, and .72 for aggression at Waves 1-4, respectively, for the first drink group. Parameter estimates for the error terms were .75, .60, .57, and .45 for delinquent behavior at Waves 1-4, respectively, for the no first drink group and 1.77, 1.32, .74, and .95 for delinquent behavior at Waves 1-4 for the first drink group.

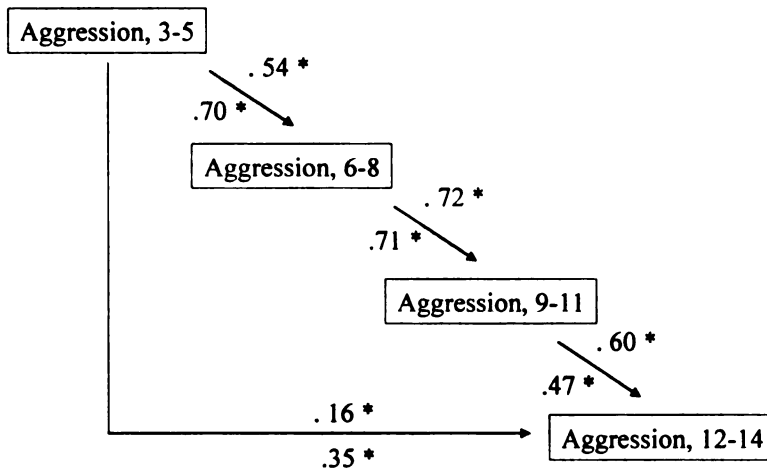


Figure 7.

Common metric standardized solution for aggression by drinking onset group.

Note: parameter estimates are above arrows for the no drink group and below arrows for the first drink group; * $p < .05$.

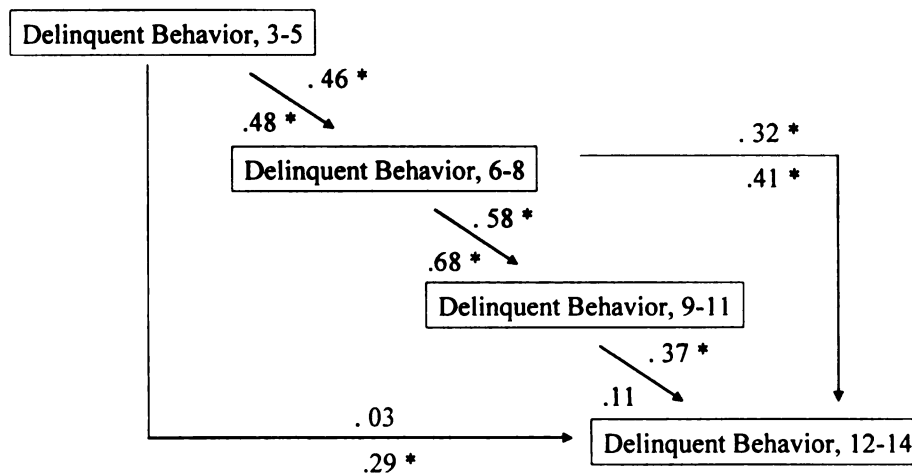


Figure 8.

Common metric standardized solution for delinquent behavior by drinking onset

group. Note: parameter estimates are above arrows for the no drink group and below arrows for the first drink group; * $p < .05$.

for delinquent behavior depicted in Figure 6; levels of delinquent behavior converged for the two groups at Wave 3 (9-11). The added path bypasses this convergence and links the elevated levels of delinquent behavior observed among early drinkers at 6-8 to their elevated levels at 12-14. The decline in delinquent behavior during late middle childhood among early drinkers also probably explains why the path from Wave 3 to Wave 4 delinquent behavior was not significant for this group.

Interestingly, the path from Wave 1 to Wave 4 delinquent behavior was significant for early drinkers only. This direct path represents the influence of early predispositions on adolescent outcomes and supports the idea of hard-continuity for this subset of individuals.

Comparing each model to an invariance model (i.e., parameters between groups constrained to be equal) revealed significant differences in the covariance structure by first drink onset status for delinquent behavior but not aggression. For aggression, the invariance model yielded $\chi^2 = 12.35$, $df = 12$, $p = .42$, RMSEA = .012, NFI = .97, CFI = 1.00. Relative to the free parameter model, $\Delta \chi^2 = 8.60$, $\Delta df = 8$, $p > .05$. For delinquent behavior, the invariance model yielded $\chi^2 = 68.99$, $df = 11$, $p = .00$, RMSEA = .220, NFI = .82, CFI = .85. The free parameter model for delinquent behavior showed significantly better fit: $\Delta \chi^2 = 66.65$, $\Delta df = 9$, $p < .01$. Again, group differences in this developmental model were much more marked for delinquent behavior than aggression.

Finally, the relative strength of indirect and direct effects were examined across drinking onset groups for the added paths of Wave 2 on Wave 4 behavior (for delinquent) and Wave 1 on Wave 4 behavior (for delinquent and aggressive). Standardized effects are reported in Table 10. The most striking differences between groups were in the

effects of preschool symptomatology on delinquent behavior during early adolescence. The total effect was much stronger among early drinkers (.51 versus .28). This disparity was largely the result of differences in the direct effect (i.e., Wave 1 → Wave 4) rather than the indirect effect (i.e., Wave 1 through the middle childhood waves to Wave 4). The direct effect of Wave 1 on Wave 4 delinquent behavior was .29 for early drinkers compared to .04 for those who had not yet tried alcohol by 12-14 years of age.

Table 10

Direct and Indirect Effects of Paths from Non-Adjacent Waves in Structural Equation Models for Aggression and Delinquent Behavior

Relationship	Group	Standardized Effect		
		Direct	Indirect	Total
<u>Aggression</u>				
Wave 1 to Wave 4	No First Drink	.16	.25	.41
	First Drink	.31	.21	.52
<u>Delinquent Behavior</u>				
Wave 1 to Wave 4	No First Drink	.04	.24	.28
	First Drink	.29	.23	.52
Wave 2 to Wave 4	No First Drink	.32	.22	.54
	First Drink	.41	.07	.48

Hypothesis 2. Preschool and adolescent levels of antisocial behavior problems are distinguishing characteristics along the developmental course leading to and through drinking onset – and more specifically, antisocial behavior problems in these two time periods can be used to categorize individuals into groups that differ in the likelihood of early first drink.

Results from the Configural Frequency Analysis (CFA) are presented in Table 11 for aggression and Table 12 for delinquent behavior. The CFA was significant for delinquent behavior (Pearson's $\chi^2 = 18.38$ for $df = 3$, $p < .001$) but not for aggression (Pearson's $\chi^2 = 6.31$ for $df = 3$, $p > .05$).

There were neither types nor antitypes for aggression. For delinquent behavior, there were more cases than expected by chance for those who were low in delinquent behavior during both preschool and early adolescence and who had not had a first drink by 12-14 years of age (i.e., the “11” type). There were fewer cases than expected by chance for low-low early drinkers (i.e., the “12” antitype). The opposite pattern was found for those who were high in delinquent behavior during both time periods. Specifically, there were more cases than expected by chance for the high-high group with drinking onset (i.e., the “42” type), and fewer cases than expected by chance for high-high children who had not yet tried alcohol (i.e., the “41” antitype). In other words, those in the high-high delinquent behavior group were more likely than chance to be early drinkers, whereas those in the low-low delinquent behavior group were less likely than chance to have tried alcohol by 12-14 years of age. This observed relationship between

continuity in delinquent behaviors and likelihood of early AFD is consistent with the hard-continuity model of problem behavior.

Table 11

Configurations for Aggression Group and First Drink Onset

AO	Observed Frequency	Expected Frequency	$\underline{L}$	Type/Antitype
11	70	66.75	1.03	--
12	19	22.25	-1.03	--
21	20	20.25	-0.12	--
22	7	6.75	0.12	--
31	33	29.25	1.53	--
32	6	9.75	-1.53	--
41	42	48.75	-2.30	--
42	23	16.25	2.30	--

Note. A = aggression group; O = first drink onset. Numerals in AO column represent ordered pairs of variable categories. Response categories for aggression were 1 = low-low, 2 = low-high, 3 = high-low, and 4 = high-high. For first drink onset, 1 = no onset and 2 = onset. $\underline{L}$ stands for Lehmacher's (1981) test statistic; Bonferroni adjusted alpha, $\alpha^* = .0062500$ was used; * significant at $\alpha^* = .0062500$.

Table 12

Configurations for Delinquent Behavior Group and First Drink Onset

DO	Observed Frequency	Expected Frequency	<u>L</u>	Type/Antitype
11	65	57.00	2.61 *	Type
12	11	19.00	-2.61 *	Antitype
21	19	19.50	-0.24	--
22	7	6.50	0.24	--
31	40	35.25	1.80	--
32	7	11.75	-1.80	--
41	41	53.25	-4.07 *	Antitype
42	30	17.75	4.07 *	Type

Note. D = delinquent behavior group; O = first drink onset. Numerals in DO column represent ordered pairs of variable categories. Response categories for delinquent behavior were 1 = low-low, 2 = low-high, 3 = high-low, and 4 = high-high. For first drink onset, 1 = no onset and 2 = onset. L stands for Lehmacher's (1981) test statistic; Bonferroni adjusted alpha, $\alpha^* = .0062500$ was used; * significant at $\alpha^* = .0062500$.

Summary. Hypothesis 1 was partially supported for delinquent behavior but not supported for aggression. Contrary to expectations, there was a convergence of delinquent behavior problem ratings between early drinkers and those who had not tried alcohol by 12-14 years of age during late-middle childhood (9-11), and few differences by drinking onset status for aggression from preschool to adolescence. Hypothesis 2 was fully supported for delinquent behaviors only.

The Development of Antisocial Behavior

Hypothesis 3: On all measures of risk from preschool (temperament, parental psychopathology) and middle childhood (academic achievement, self-esteem, social problems, family cohesion, family expressiveness, family conflict, physical discipline/negative control), scores will be least favorable for the high-high group, moderate for the low-high and high-low groups, and most favorable for the low-low group. If supported, this would corroborate the findings of Fergusson et al. (1996).

The high-high group is expected to have the following characteristics relative to the low-low group: more difficult temperament (i.e., high activity, short attention span, more reactivity) and more parental psychopathology (mothers and fathers) at 3-5 years of age; as well as less academic achievement (i.e., lower scores on reading, spelling, and arithmetic), lower self-esteem, more social problems, less family cohesion, less family expressiveness, more family conflict, and more physical/negative punishment at 6-8 and 9-11 years of age. The high-low and low-high groups are expected to have in-between scores on each of these dimensions.

Before proceeding to risk profile comparisons, it may be useful to examine aggression and delinquent behavior groups on the variable that defines them – that is, on aggressive and delinquent behavior, respectively. The developmental patterns of aggression by group are depicted in Figure 9, and the developmental patterns of

delinquent behavior by group are depicted in Figure 10; again, the possible range of CBCL scores was 0 to 40 for aggression (20 items), and 0 to 24 for delinquent behavior (12 items). Means and standard deviations for aggression and delinquent behavior by wave and behavior problem group can be found in Table 13.

There were behavior problem classification group differences for all aggression and delinquent behavior scores from Wave 1 to Wave 4, inclusive (see Table 13). Specifically, there were significant group differences between aggression groups at Wave 1 ($F [3, 216] = 167.36, p < .001$), Wave 2 ($F [(3, 216] = 32.77, p < .001$), Wave 3 ($F [3, 216] = 27.84, p < .001$), and Wave 4 ($F [3, 216] = 138.53, p < .001$). There were also significant group differences between delinquent behavior groups at Wave 1 ($F [3, 216] = 98.76, p < .001$), Wave 2 ($F [(3, 216] = 32.29, p < .001$), Wave 3 ($F [3, 216] = 24.66, p < .001$), and Wave 4 ($F [3, 216] = 91.88, p < .001$).

Bonferroni post-hoc comparison tests revealed significant differences between pairs of groups as well (at $\alpha = .05$). At Wave 1, all groups differed except for high-high versus high-low aggression and for low-low versus low-high delinquent behavior. Stated conversely, there were no significant baseline differences between the high-high versus high-low groups for aggression and the low-low versus low-high groups for delinquent behavior.

High ratings of aggression and low ratings of delinquent behavior at Wave 1 meant the same across classifications. On the other hand, it appears that the low-high group for aggression was already more aggressive than the low-low group. Similarly, the high-low group for delinquent behavior was already less delinquent than the high-

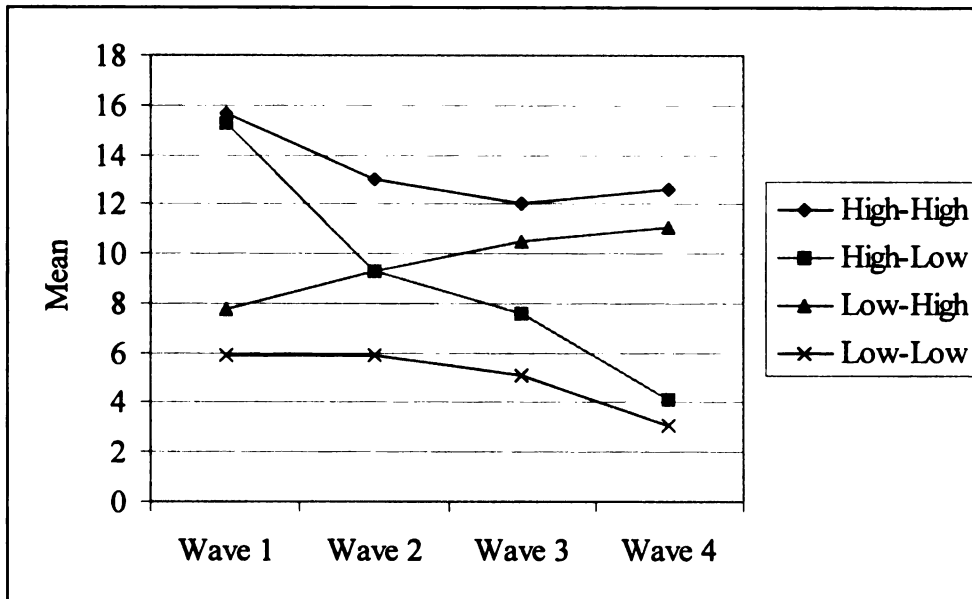


Figure 9.

Mean CBCL aggression scores from Wave 1 to Wave 4, by aggression group.

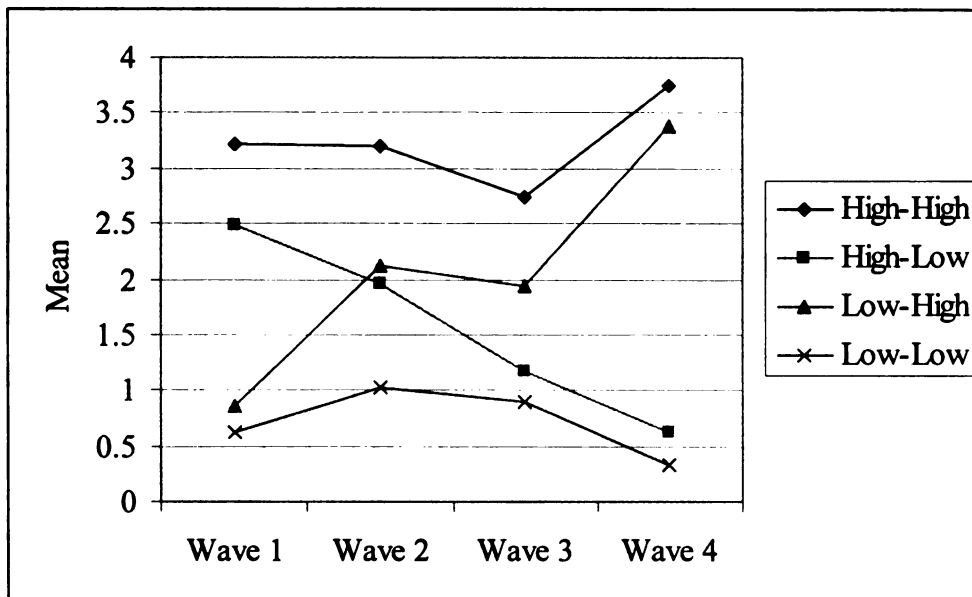


Figure 10.

Mean CBCL delinquent behavior scores from Wave 1 to Wave 4, by delinquent behavior group.

Table 13

Means (and Standard Deviations) for Aggression by Aggression Group and Delinquent Behavior by Delinquent Behavior Group

Variable	Low-Low M (SD)	Low-High M (SD)	High-Low M (SD)	High-High M (SD)
Aggression				
Wave 1 *** C	5.87 (2.70)	7.74 (1.83)	15.28 (3.55)	15.63 (3.59)
Wave 2 *** F	5.90 (4.19)	9.31 (3.75)	9.25 (4.59)	12.96 (4.71)
Wave 3 *** B E F	5.06 (4.17)	10.47 (4.53)	7.55 (4.53)	12.06 (6.02)
Wave 4 *** B E	3.08 (2.22)	11.07 (3.42)	4.10 (1.68)	12.61 (4.56)
Delinquent Behavior				
Wave 1 *** D	.62 (0.49)	.85 (0.37)	2.49 (0.72)	3.21 (1.55)
Wave 2 *** F	1.03 (1.01)	2.11 (1.34)	1.95 (1.21)	3.20 (1.68)
Wave 3 *** B E F	.90 (0.95)	1.94 (1.67)	1.17 (1.08)	2.74 (1.77)
Wave 4 *** B E	.32 (0.47)	3.38 (1.55)	.62 (0.49)	3.75 (2.26)

A High-High versus Low-Low, $p \geq .05$ B High-High versus Low-High, $p \geq .05$ C High-High versus High-Low, $p \geq .05$ D Low-Low versus Low-High, $p \geq .05$ E Low-Low versus High-Low, $p \geq .05$ F Low-High versus High-Low, $p \geq .05$ *** $p < .001$. ** $p < .01$. * $p < .05$

high group. Some caution in interpreting differences between these group pairs is therefore warranted as initial problem behavior severity was not truly equivalent.

At subsequent waves, classifications fit with expected patterns. At Wave 2, all groups differed except for low-high and high-low on both aggression and delinquent behavior. These groups were also not statistically significant at Wave 3. This reflects the crossing over of these groups on behavior problem severity during middle childhood. At Wave 3, two additional pairs of groups were not statistically different: high-high versus low-high and low-low versus high-low for both aggression and delinquent behavior. These groups were also statistically similar at Wave 4. The implication here is that the cross-over has already taken place by Wave 3; groups high and low in behavior problems at Wave 4 have converged by 9-11 years of age.

Means and standard deviations by aggression group on the risk factors can be found in Table 14 (Wave 1 variables), Table 15 (Wave 2 variables), and Table 16 (Wave 3 variables). Corresponding information for the delinquent behavior groups are in Tables 17-19. Group differences on each variable were tested using a one-way analysis of variance (ANOVA). Temperament variables from middle childhood were also examined because of their inclusion as control variables in the testing of Hypothesis 4 (discussed later).

Aggression. At Wave 1, there were significant aggression group differences for reactivity ($F [3, 216] = 11.36, p < .001$), maternal psychopathology ($F [3, 216] = 5.60, p = .001$), and paternal psychopathology ($F [3, 216] = 2.66, p = .049$).⁶ At Wave 2, there were significant aggression group differences for reading ($F [3, 216] = 2.78, p = .042$),

⁶ Non-significant differences for Wave 1 aggression groups were as follows: For age, $F [3, 216] = .37, p = .773$; for activity, $F [3, 216] = 1.23, p = .300$; and for attention, $F [3, 216] = 2.29, p = .079$.

global self-esteem ($F [3, 216] = 2.96, p = .033$), social problems ($F [3, 216] = 10.36, p < .001$), family conflict ($F [3, 216] = 5.05, p = .002$), and all three temperament variables: activity ($F [3, 216] = 2.66, p = .049$), attention ($F [3, 216] = 3.97, p = .009$), and reactivity ($F [3, 216] = 14.61, p < .001$).⁷ At Wave 3, there were significant aggression group differences for social problems ($F [3, 216] = 7.06, p < .001$), family conflict ($F [3, 216] = 4.57, p = .004$), negative control ($F [3, 216] = 7.65, p < .001$), and all three temperament variables: activity-general ($F [3, 216] = 10.15, p < .001$), activity-sleep ($F [3, 216] = 3.44, p = .018$), and task orientation ($F [3, 216] = 3.61, p = .014$).⁸

Pair-wise comparisons were investigated using Bonferroni post-hoc tests (with $\alpha = .05$). At Wave 1 (see Table 14), more aggressive children (i.e., high-high and high-low individuals) were more reactive than those with initially lower levels of aggression (i.e., low-low and low-high). Maternal psychopathology was lower for the low-low aggression group compared to all others, and paternal psychopathology was lower for the low-low aggression group than the high-high aggression group.

At Wave 2 (see Table 15), there were no pair-wise differences on reading or activity despite significant overall ANOVAs. Reactivity was higher in the high-high group than for all others. The high-high group could also be distinguished from the low-low group by more social problems and family conflict as well as a shorter attention span. Fewer social problems were associated with a remitting pattern of aggressive behavior (i.e., high-low versus high-high) and so good peer relations may be an important

⁷ Non-significant differences for Wave 2 aggression groups were as follows: For spelling, $F [3, 216] = 2.11, p = .100$; arithmetic, $F [3, 216] = 1.11, p = .346$; family cohesion, $F [3, 216] = 2.39, p = .070$; and family expressiveness, $F [3, 216] = 1.31, p = .273$.

⁸ Non-significant differences for Wave 3 aggression groups were as follows: reading, $F [3, 216] = .57, p = .638$; spelling, $F [3, 216] = 1.36, p = .256$; arithmetic, $F [3, 216] = 1.15, p = .331$; global self-esteem,

promotive factor – or, alternatively, poor peer relations may be a risk factor that maintains the developmental trajectory among highly aggressive children. Lower self-esteem was associated with an escalating pattern of aggressive behavior (i.e., low-high versus low-low) and therefore may be an important risk factor.

At Wave 3 (see Table 16), the high-high aggression group could be distinguished from the low-low group by more social problems, family conflict, negative parental control, and general activity. Fewer social problems were again associated with a remitting pattern of aggressive behavior (i.e., high-low versus high-high), as was a lower activity-general level. More family conflict and activity-general combined with less task orientation were characteristics of an escalating pattern of aggressive behavior (i.e., low-low versus low-high).

Because of its dichotomous nature, mean group differences on spanking and physical discipline could not be examined. Chi-square goodness-of-fit tests were therefore employed. At Wave 2, there was a negligible relationship between aggression group and spanking, $\chi^2(3, 220) = 2.13, p = .546$. At Wave 3, the relationship between aggression group and physical discipline was significant, $\chi^2(3, 220) = 14.61, p = .002$. This was such that 4.5% of the low-low group, 25.9% of the low-high group, 5.1% of the high-low group, and 18.5% of the high-high group had parents who reported using physical discipline at 9-11 years of age.

$F[3, 216] = 2.35, p = .074$; family cohesion, $F[3, 216] = 2.29, p = .079$; and family expressiveness, $F[3, 216] = .90, p = .444$.

Table 14

Means (and Standard Deviations) by Aggression Group for Wave 1 Variables

Variable	<i>Low-Low</i>		<i>Low-High</i>		<i>High-Low</i>		<i>High-High</i>	
	<u>M</u>	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)
Age	4.32	(0.98)	4.14	(0.94)	4.24	(1.09)	4.19	(0.96)
Activity	1.26	(1.41)	1.33	(1.41)	1.69	(1.34)	1.60	(1.48)
Attention	5.76	(2.85)	5.22	(2.58)	4.46	(3.03)	4.85	(2.98)
Reactivity *** ^A ^B ^E ^F	2.73	(1.62)	2.15	(1.51)	3.56	(1.64)	3.85	(1.38)
Maternal Psych. ** ^A ^D ^E	0.53	(0.89)	1.30	(1.23)	1.15	(1.33)	1.11	(1.28)
Paternal Psych. * ^A	1.56	(1.45)	1.70	(1.51)	1.92	(1.58)	2.25	(1.67)

^A High-High versus Low-Low, $p < .05$ ^B High-High versus Low-High, $p < .05$ ^C High-High versus High-Low, $p < .05$ ^D Low-Low versus Low-High, $p < .05$ ^E Low-Low versus High-Low, $p < .05$ ^F Low-High versus High-Low, $p < .05$ *** $p < .001$. ** $p < .01$. * $p < .05$

Table 15

Means (and Standard Deviations) by Aggression Group for Wave 2 Variables

Variable	Low-Low		Low-High		High-Low		High-High	
	<u>M</u>	(SD)	<u>M</u>	(SD)	<u>M</u>	(SD)	<u>M</u>	(SD)
Reading *	98.97	(15.06)	99.01	(11.76)	92.97	(13.35)	93.29	(16.57)
Spelling	95.59	(15.45)	94.94	(11.82)	91.27	(15.20)	89.89	(15.66)
Arithmetic	97.65	(15.87)	97.67	(12.48)	97.36	(10.77)	93.77	(14.03)
Global Self-Esteem * ^D	21.16	(2.65)	19.24	(3.63)	21.04	(2.84)	20.42	(3.62)
Social Problems *** ^{A C}	1.39	(1.74)	2.01	(1.85)	2.01	(1.59)	3.08	(2.15)
Family Cohesion	7.76	(1.50)	7.03	(2.09)	7.09	(1.75)	7.38	(1.49)
Family Expressiveness	6.46	(1.47)	6.65	(1.36)	6.68	(1.42)	6.92	(1.42)
Family Conflict ** ^A	3.00	(1.89)	4.03	(2.24)	3.82	(2.00)	4.18	(2.05)
Activity *	1.04	(1.26)	1.10	(1.35)	1.00	(1.21)	1.57	(1.32)
Attention ** ^A	6.87	(2.98)	5.95	(2.87)	5.93	(2.84)	5.29	(2.73)
Reactivity *** ^{A B C}	2.07	(1.30)	2.14	(1.59)	2.41	(1.23)	3.50	(1.50)

^A High-High versus Low-Low, $p < .05$ ^B High-High versus Low-High, $p < .05$ ^C High-High versus High-Low, $p < .05$ ^D Low-Low versus Low-High, $p < .05$ ^E Low-Low versus High-Low, $p < .05$ ^F Low-High versus High-Low, $p < .05$

Table 16

Means (and Standard Deviations) by Aggression Group for Wave 3 Variables

Variable	Low-Low		Low-High		High-Low		High-High	
	<u>M</u>	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)
Reading	101.75	(15.58)	102.15	(12.89)	99.15	(11.42)	99.30	(16.32)
Spelling	99.89	(14.43)	98.10	(12.72)	96.79	(17.61)	94.84	(16.92)
Arithmetic	100.59	(17.18)	102.10	(15.05)	96.67	(13.08)	97.30	(16.32)
Global Self-Esteem	20.98	(3.15)	19.31	(3.41)	20.95	(2.86)	20.43	(2.74)
Social Problems *** ^{A C}	1.41	(1.69)	2.11	(1.58)	1.71	(1.63)	2.82	(2.42)
Family Cohesion	7.86	(1.58)	7.26	(2.26)	7.59	(1.31)	7.15	(1.99)
Family Expressiveness	6.53	(1.69)	6.69	(1.79)	6.17	(2.10)	6.73	(1.62)
Family Conflict ** ^{A D}	3.04	(1.95)	4.40	(2.00)	3.86	(1.90)	4.02	(2.33)
Negative Control *** ^A	10.68	(3.31)	12.29	(2.97)	11.95	(2.57)	13.07	(3.24)
Activity-General *** ^{A C D}	15.16	(4.36)	18.12	(4.42)	15.88	(4.05)	19.02	(5.18)
Activity-Sleep *	9.31	(3.24)	10.55	(3.14)	8.76	(3.00)	10.54	(3.64)
Task Orientation * ^D	20.78	(4.12)	18.11	(5.20)	20.95	(4.28)	19.41	(4.39)

^A High-High versus Low-Low, $p < .05$ ^B High-High versus Low-High, $p < .05$ ^C High-High versus High-Low, $p < .05$ ^D Low-Low versus Low-High, $p < .05$ ^E Low-Low versus High-Low, $p < .05$ ^F Low-High versus High-Low, $p < .05$

Delinquent Behavior. At Wave 1, there were significant delinquent behavior group differences for attention ($F [3, 216] = 3.08, p = .029$), reactivity ($F [3, 216] = 5.89, p = .001$), and maternal psychopathology ($F [3, 216] = 4.54, p = .004$).⁹ At Wave 2 there were significant delinquent behavior group differences for social problems ($F [3, 216] = 6.77, p < .001$), family conflict ($F [3, 216] = 5.18, p = .002$), and all three temperament variables: activity ($F [3, 216] = 3.02, p = .031$), attention ($F [3, 216] = 4.83, p = .003$), and reactivity ($F [3, 216] = 6.36, p < .001$).¹⁰ At Wave 3, there were significant delinquent behavior group differences for social problems ($F [3, 216] = 3.30, p = .021$), family cohesion ($F [3, 216] = 3.66, p = .013$), negative control ($F [3, 216] = 5.87, p = .001$), and activity-general ($F [3, 216] = 8.63, p < .001$).¹¹

Pair-wise comparisons were investigated using Bonferroni post-hoc tests (with $\alpha = .05$). At Wave 1 (see Table 17), there were no group differences for attention despite a significant overall ANOVA. The high-high delinquent behavior group was more reactive than those with initially low levels of delinquent symptomatology (i.e., low-low and low-high) and experienced more maternal psychopathology than the low-low group.

At Wave 2 (see Table 18), there were no group differences on activity despite a significant overall ANOVA. The high-high group experienced more social problems and family conflict, had a shorter attention span, and was more reactive compared to the low-

⁹ Non-significant differences for Wave 1 delinquent behavior groups were as follows: age, $F [3, 216] = 1.56, p = .201$; activity, $F [3, 216] = .30, p = .825$; and paternal psychopathology, $F [3, 216] = 1.31, p = .271$.

¹⁰ Non-significant differences for Wave 2 delinquent behavior groups were as follows: reading, $F [3, 216] = .76, p = .520$; spelling, $F [3, 216] = 1.51, p = .214$; arithmetic, $F [3, 216] = 1.16, p = .327$; global-self-esteem, $F [3, 216] = .08, p = .972$; family cohesion, $F [3, 216] = 2.29, p = .080$; and family expressiveness, $F [3, 216] = .35, p = .789$.

low group. More family conflict and shorter attention spans were also associated with persisting rather than remitting delinquent behaviors (i.e., high-high versus high-low), and may therefore be risk factors that maintain the developmental trajectory for children who already exhibit elevated symptomatology (or conversely, improvement is more likely in the absence of these risks). The high-high group also had more social problems compared to the low-high group.

At Wave 3 (see Table 19), the high-high group had more social problems, negative control, and activity-general but less family cohesion than the low-low group. More negative control and activity-general were also associated with the persisting rather than remitting pattern (i.e., high-high versus high-low).

Because of its dichotomous nature, mean group differences on spanking and physical discipline could not be examined. Chi-square goodness-of-fit tests were therefore employed. At Wave 2, there was a negligible relationship between delinquent behavior group and spanking, $\chi^2(3, 220) = 1.10, p = .778$. At Wave 3, the relationship between delinquent behavior group and physical discipline was significant, $\chi^2(3, 220) = 12.11, p = .007$. This was such that 5.3% of the low-low group, 23.1% of the low-high group, 4.3% of the high-low group, and 18.3% of the high-high group had parents who reported using physical discipline at 9-11 years of age.

¹¹ Non-significant differences for Wave 3 delinquent behavior groups were as follows: reading, $F[3, 216] = 1.25, p = .293$; spelling, $F[3, 216] = 1.90, p = .130$; arithmetic, $F[3, 216] = 1.74, p = .159$; global self-esteem, $F[3, 216] = .79, p = .502$; family expressiveness, $F[3, 216] = .21, p = .891$; family conflict, $F[3, 216] = 2.24, p = .085$; activity-sleep, $F[3, 216] = 1.60, p = .190$; task orientation, $F[3, 216] = 1.93, p = .126$.

Table 17

Means (and Standard Deviations) by Delinquent Behavior Group for Wave 1 Variables

Variable	Low-Low		Low-High		High-Low		High-High	
	<u>M</u>	(SD)	<u>M</u>	(SD)	<u>M</u>	(SD)	<u>M</u>	(SD)
Age	4.15	(0.97)	3.98	(1.04)	4.33	(0.95)	4.40	(0.99)
Activity	1.35	(1.44)	1.35	(1.50)	1.57	(1.43)	1.49	(1.39)
Attention *	5.52	(3.03)	6.04	(2.25)	5.43	(3.20)	4.38	(2.67)
Reactivity ** ^{A B}	2.71	(1.61)	2.54	(1.42)	3.36	(1.71)	3.66	(1.57)
Maternal Psych. ** ^A	0.54	(1.50)	1.12	(1.26)	0.91	(1.78)	1.21	(1.58)
Paternal Psych.	1.71	(1.44)	1.65	(1.50)	1.72	(1.43)	2.14	(1.39)

^A High-High versus Low-Low, $p < .05$ ^B High-High versus Low-High, $p < .05$ ^C High-High versus High-Low, $p < .05$ ^D Low-Low versus Low-High, $p < .05$ ^E Low-Low versus High-Low, $p < .05$ ^F Low-High versus High-Low, $p < .05$ *** $p < .001$. ** $p < .01$. * $p < .05$

Table 18

Means (and Standard Deviations) by Delinquent Behavior Group for Wave 2 Variables

Variable	Low-Low		Low-High		High-Low		High-High	
	<u>M</u>	<u>(SD)</u>	<u>M</u>	<u>(SD)</u>	<u>M</u>	<u>(SD)</u>	<u>M</u>	<u>(SD)</u>
Reading	96.63	(14.45)	98.17	(12.14)	97.68	(14.35)	94.15	(17.09)
Spelling	92.56	(15.10)	98.00	(10.59)	94.29	(15.27)	90.97	(16.42)
Arithmetic	97.55	(15.35)	98.06	(12.18)	97.66	(13.00)	93.89	(14.15)
Global Self-Esteem	20.73	(3.66)	20.43	(3.19)	20.78	(2.34)	20.67	(3.11)
Social Problems *** ^{A B}	1.45	(1.74)	1.54	(1.59)	2.37	(1.95)	2.75	(2.12)
Family Cohesion	7.74	(1.51)	7.06	(1.73)	7.61	(1.56)	7.15	(1.74)
Family Expressiveness	6.61	(1.41)	6.75	(1.21)	6.82	(1.37)	6.57	(1.59)
Family Conflict ** ^{A C}	3.24	(1.77)	3.68	(2.00)	3.10	(2.20)	4.35	(2.08)
Activity *	0.89	(1.21)	1.61	(1.38)	1.13	(1.30)	1.41	(1.30)
Attention ** ^{A C}	6.63	(2.91)	6.68	(2.74)	6.61	(3.02)	5.06	(2.71)
Reactivity *** ^A	2.09	(1.24)	2.37	(1.52)	2.58	(1.59)	3.13	(1.56)

^A High-High versus Low-Low, $p < .05$ ^B High-High versus Low-High, $p < .05$ ^C High-High versus High-Low, $p < .05$ ^D Low-Low versus Low-High, $p < .05$ ^E Low-Low versus High-Low, $p < .05$ ^F Low-High versus High-Low, $p < .05$ *** $p < .001$. ** $p < .01$. * $p < .05$

Table 19

Means (and Standard Deviations) by Delinquent Behavior Group for Wave 3 Variables

Variable	<i>Low-Low</i>		<i>Low-High</i>		<i>High-Low</i>		<i>High-High</i>	
	<u>M</u>	(SD)	<u>M</u>	(SD)	<u>M</u>	(SD)	<u>M</u>	(SD)
Reading	101.64	(13.22)	101.38	(19.57)	102.66	(12.58)	97.89	(15.71)
Spelling	95.66	(14.58)	101.97	(7.50)	100.68	(18.55)	96.13	(16.50)
Arithmetic	100.20	(17.64)	102.35	(13.63)	100.75	(13.66)	95.67	(16.21)
Global Self-Esteem	20.60	(3.39)	20.82	(2.92)	21.08	(2.70)	20.23	(2.92)
Social Problems * ^A	1.52	(1.87)	1.58	(1.36)	2.14	(2.16)	2.47	(2.09)
Family Cohesion * ^A	7.85	(1.57)	7.65	(1.83)	7.78	(1.03)	6.97	(2.21)
Family Expressiveness	6.49	(1.94)	6.80	(1.67)	6.52	(1.70)	6.54	(1.66)
Family Conflict	3.25	(2.09)	4.03	(2.05)	3.45	(1.96)	4.06	(2.22)
Negative Control ** ^{A C}	10.88	(3.08)	12.46	(2.92)	11.29	(3.37)	12.92	(3.20)
Activity-General *** ^{A C}	15.09	(4.28)	17.13	(3.91)	16.19	(4.86)	18.89	(5.02)
Activity-Sleep	9.41	(3.48)	9.40	(2.72)	9.33	(3.32)	10.44	(3.45)
Task Orientation	20.52	(4.30)	20.11	(4.24)	20.84	(4.03)	19.08	(4.83)

^A High-High versus Low-Low, $p < .05$ ^B High-High versus Low-High, $p < .05$ ^C High-High versus High-Low, $p < .05$ ^D Low-Low versus Low-High, $p < .05$ ^E Low-Low versus High-Low, $p < .05$ ^F Low-High versus High-Low, $p < .05$

Hypothesis 4: Initial risk trajectory variables will predict aggression and delinquent behavior during the preschool years, and initial risk trajectory variables along with middle childhood variables will predict adolescent aggression and adolescent delinquent behavior. Middle childhood factors will boost the explanatory power of the model (i.e., significantly add to the R^2) and eclipse some of the early indicators of risk – recognizing both that risk is fluid (i.e., early indicators don't always pan out), and that proximate influences are likely to have greater effect.

With regard to the initial risk load, antisocial behavior in preschool and adolescence will be predicted from more difficult temperament (i.e., higher activity levels, shorter attention span, and greater reactivity) and exposure to more parental psychopathology. Adolescent antisocial behavior will also be predicted by child characteristics (less academic achievement, lower self-esteem, and more social problems) and the family environment (i.e., less cohesion, less expressiveness, more conflict, and more physical punishment/negative control) in middle childhood.

Hierarchical regression results for Wave 1 variables predicting Wave 1 aggression and delinquent behavior are presented in Table 20. Results for variables predicting Wave 4 aggression are in Table 21 (with Wave 2 middle childhood variables) and Table 22 (with Wave 3 middle childhood variables). Results for variables predicting Wave 4 delinquent behavior can be found in Table 23 (with Wave 2 middle childhood variables) and Table 24 (with Wave 3 middle childhood variables). Given the large number of

independent variables in each final model, multicollinearity diagnostics were examined. Tolerance is a statistic that reflects the proportion of a variable's variance not accounted for by other predictors in the equation (i.e., $1 - R^2$ for the variable predicted by all others). A variable with a tolerance close to 0 contributes little to the model and represents a problem of multicollinearity. Tolerances for each variable in regression equations using Wave 1 and Wave 2 predictors can be found in Table 25; tolerances for each variable in regression equations using Wave 1 and Wave 3 predictors can be found in Table 26. Tolerances ranged from .252 - .907; no multicollinearity problems were detected.

Predicting Wave 1 Aggression and Delinquent Behavior. Regression analyses for Wave 1 behavior problems with Wave 1 predictors revealed that the model significantly predicted both aggression, $F(6, 213) = 10.15, p < .001$; and delinquent behaviors, $F(6, 213) = 7.79, p < .001$. R^2 was .22 for aggression and .18 for delinquent behavior. Table 20 displays the standardized regression coefficients for each variable. In terms of individual relationships between the independent variables and behavior problems, reactivity (for aggression $t = 5.12, p < .001$; for delinquent behavior $t = 2.61, p = .010$) and maternal psychopathology (for aggression $t = 2.39, p = .018$; for delinquent behavior $t = 2.67, p = .008$) were significant predictors in both models. More reactivity and higher levels of maternal psychopathology were associated with more behavior problems during the preschool years. Age ($t = 4.21, p < .001$) was also a significant predictor in the regression for delinquent behavior: As age within the wave increased (i.e., a between-subjects distinction), delinquent behavior scores increased.

Table 20

Hierarchical Regression Results for Aggression and Delinquent Behavior at Wave 1

Wave	Variable	Standardized Regression Coefficient (β)	
		Aggression	Delinquent Behavior
1	Age	.013	.267 ***
1	Activity	.041	-.108
1	Attention	-.091	-.103
1	Reactivity	.333 ***	.174 *
1	Maternal Psych.	.157 *	.181 **
1	Paternal Psych.	.120	.095
Total R^2		.222	.180

*** $p < .001$, ** $p < .01$, * $p < .05$

Predicting Wave 4 Aggression. A hierarchical regression was run for Wave 4 aggression with predictors from preschool and Wave 2 middle childhood. Table 21 displays the standardized regression coefficients for each variable. Preschool measures were entered as predictors in Step 1. The model significantly explained aggression in early adolescence, $F(6, 213) = 2.96$, $p = .008$. R^2 was .08. Only maternal psychopathology ($t = 2.82$, $p = .005$) at Wave 1 predicted aggression at Wave 4, with more maternal psychopathology associated with more severe symptoms.

In Step 2, predictors from middle childhood (Wave 2) were entered. The model significantly predicted Wave 4 aggression ($F[15, 204] = 2.96$, $p = .008$), R^2 was .28, and adding these middle childhood predictors significantly increased the amount of explained variance ($F[9, 204] = 6.48$, $p < .001$) by 21%. Significant predictors were maternal psychopathology ($t = 2.09$, $p = .038$), global self-esteem ($t = -3.05$, $p = .003$), social problems ($t = 4.13$, $p < .001$), and family conflict ($t = 3.12$, $p = .002$). Adolescent

aggression was associated with more maternal psychopathology, social problems, and family conflict but lower self-esteem.

In Step 3, temperament from Wave 2 was added to the model. The model significantly predicted Wave 4 aggression ($F[18, 201] = 5.25, p < .001$), R^2 was .32, and adding these predictors significantly increased the amount of explained variance ($F[3, 201] = 3.70, p = .013$) by 4%. Significant predictors were maternal psychopathology ($t = 2.38, p = .018$), global self-esteem ($t = -2.15, p = .033$), social problems ($t = 3.28, p = .001$), family conflict ($t = 2.70, p = .008$), and Wave 2 reactivity ($t = 2.72, p = .007$). Greater reactivity at Wave 2 led to more aggressive symptomatology by adolescence.

Finally, Wave 1 (baseline) aggression was entered in Step 4. The model significantly predicted Wave 4 aggression ($F[19, 200] = 6.27, p < .001$), R^2 was .37, and adding this predictor significantly increased the amount of explained variance ($F[1, 200] = 17.04, p < .001$) by 5%. Wave 1 aggression was the strongest predictor of Wave 4 aggression, and controlling for it led to the effects of maternal psychopathology and Wave 2 reactivity becoming statistically negligible ($p > .05$). Significant predictors were global self-esteem ($t = -2.49, p = .014$), social problems ($t = 2.63, p = .009$), family conflict ($t = 2.28, p = .024$), and Wave 1 (baseline) aggression ($t = 4.13, p < .001$).

The same procedure was followed for predicting Wave 4 aggression with predictors from preschool and Wave 3 middle childhood. Table 22 displays the standardized regression coefficients for each variable. Results for Step 1 are the same as in the previous analysis. In Step 2, predictors from middle childhood (Wave 3) were entered. The model significantly predicted Wave 4 aggression ($F[16, 203] = 6.24, p < .001$), R^2 was .33, and adding these middle childhood predictors significantly increased

the amount of explained variance ($F[10, 203] = 7.64, p < .001$) by 25%. Maternal psychopathology was no longer significant. The significant predictors were social problems ($t = 2.98, p = .003$), family cohesion ($t = -2.12, p = .035$), family expressiveness ($t = 2.73, p = .007$), physical discipline ($t = 2.49, p = .013$), and negative control ($t = 3.55, p < .001$). Adolescent aggression was associated with more social problems, family expressiveness, physical discipline, and negative control but less family cohesion.

In Step 3, temperament from Wave 3 was added to the model. The model significantly predicted Wave 4 aggression ($F[19, 200] = 6.60, p = .001$), R^2 was .39, and adding these predictors significantly increased the amount of explained variance ($F[3, 200] = 6.08, p = .001$) by 6%. Significant predictors were social problems ($t = 2.33, p = .021$), family cohesion ($t = -2.44, p = .016$), family expressiveness ($t = 2.77, p = .006$), physical discipline ($t = 2.11, p = .036$), negative control ($t = 3.45, p = .001$), and Wave 3 general activity ($t = 3.52, p = .001$). Higher general activity levels at Wave 3 predicted more aggressive symptomatology in early adolescence.

Finally, Wave 1 (baseline) aggression was entered in Step 4. The model significantly predicted Wave 4 aggression ($F[20, 199] = 8.17, p < .001$), R^2 was .45, and adding this predictor significantly increased the amount of explained variance ($F[1, 199] = 23.69, p < .001$) by 7%. Wave 1 aggression was the strongest predictor of Wave 4 aggression. Social problems were no longer significant. The significant predictors were family cohesion ($t = -2.55, p = .012$), family expressiveness ($t = 2.83, p = .005$), physical discipline ($t = 2.28, p = .024$), negative control ($t = 2.57, p = .011$), Wave 3 general activity ($t = 3.33, p = .001$), and Wave 1 (baseline) aggression ($t = 4.87, p < .001$).

Table 21

Hierarchical Regression Results for Aggression at Wave 4 with Preschool (Wave 1)
and Middle Childhood (Wave 2) Predictors

Wave	Variable	Standardized Regression Coefficient (β)			
		Step 1	Step 2	Step 3	Step 4
1	Age	-.065	-.115	-.094	-.098
1	Activity	.041	.042	.033	.022
1	Attention	-.075	-.013	-.006	.012
1	Reactivity	.090	.114	.021	-.049
1	Maternal Psych.	.202 **	.142 *	.161 *	.118
1	Paternal Psych.	.014	-.063	-.082	-.095
2	Reading		-.011	.009	.048
2	Spelling		.134	.080	.077
2	Arithmetic		-.148	-.107	-.112
2	Global-Self Esteem		-.193 **	-.139 *	-.155 *
2	Social Problems		.260 ***	.210 **	.164 **
2	Family Cohesion		.026	-.006	-.014
2	Family Expressiveness		.080	.080	.048
2	Family Conflict		.235 **	.201 **	.165 *
2	Spanking		.041	.001	-.031
2	Activity			.064	.037
2	Attention			-.004	-.013
2	Reactivity			.220 **	.156
1	Aggression				.292 ***
Total R ²		.077	.282	.320	.373
ΔR^2			.205 ***	.038 *	.053 ***

*** $p < .001$, ** $p < .01$, * $p < .05$

Table 22

Hierarchical Regression Results for Aggression at Wave 4 with Preschool (Wave 1)
and Middle Childhood (Wave 3) Predictors

Wave	Variable	Standardized Regression Coefficient (β)			
		Step 1	Step 2	Step 3	Step 4
1	Age	-.065	-.084	-.100	-.100
1	Activity	.041	.051	.002	-.009
1	Attention	-.075	-.003	.008	.034
1	Reactivity	.090	.101	.057	-.036
1	Maternal Psych.	.202 **	.073	.045	.013
1	Paternal Psych.	.014	-.025	-.024	-.050
3	Reading		.054	.098	.104
3	Spelling		-.066	-.046	-.028
3	Arithmetic		-.038	-.030	-.047
3	Global-Self Esteem		-.009	-.008	-.031
3	Social Problems		.194 **	.150 *	.107
3	Family Cohesion		-.166 *	-.185 *	-.184 *
3	Family Expressiveness		.174 **	.172 **	.167 **
3	Family Conflict		.116	.099	.077
3	Physical Discipline		.156 *	.128 *	.131 *
3	Negative Control		.233 ***	.221 **	.159 *
3	Activity - General			.262 **	.235 **
3	Activity - Sleep			.071	.079
3	Task Orientation			.061	.028
1	Aggression				.305 ***
Total R ²		.077	.330	.386	.451
ΔR^2			.253 ***	.056 **	.065 ***

*** $p < .001$, ** $p < .01$, * $p < .05$

Predicting Wave 4 Delinquent Behavior. A hierarchical regression was run for Wave 4 delinquent behavior with predictors from preschool and Wave 2 middle childhood. Table 23 displays the standardized regression coefficients for each variable. Preschool measures were entered as predictors in Step 1. The model significantly explained delinquent behavior in early adolescence, $F(6, 213) = 4.24, p < .001$. R^2 was .11. Only maternal psychopathology ($t = 3.43, p = .001$) at Wave 1 predicted delinquent behavior at Wave 4, with more maternal psychopathology associated with greater severity in adolescent's delinquent behavior.

In Step 2, predictors from middle childhood (Wave 2) were entered. The model significantly predicted Wave 4 delinquent behavior ($F[15, 204] = 3.81, p < .001$), R^2 was .22, and adding these middle childhood predictors significantly increased the amount of explained variance ($F[9, 204] = 3.26, p = .001$) by 11%. Significant predictors were maternal psychopathology ($t = 2.89, p = .004$), social problems ($t = 3.97, p < .001$), and family conflict ($t = 2.40, p = .017$). More delinquent behavior was predicted from more maternal psychopathology, social problems, and family conflict.

In Step 3, temperament from Wave 2 was added to the model. The model significantly predicted Wave 4 delinquent behavior ($F[18, 201] = 3.44, p < .001$), R^2 was .24, and adding these predictors did not significantly increase the amount of explained variance ($F[3, 201] = 1.48, p > .10$) at only 2%. Significant predictors were maternal psychopathology ($t = 2.97, p = .003$), social problems ($t = 3.41, p = .001$), and family conflict ($t = 2.12, p = .036$). The configuration of significant variables was therefore unchanged from Step 2.

Finally, Wave 1 (baseline) delinquent behavior was entered in Step 4. The model significantly predicted Wave 4 delinquent behavior ($F[19, 200] = 5.072, p < .001$), R^2 was .33, and adding this predictor significantly increased the amount of explained variance ($F[1, 200] = 26.51, p < .001$) by 9%. Wave 1 delinquent behavior was the strongest predictor of Wave 4 delinquent behavior. The effect of family conflict was no longer significant. Significant predictors were maternal psychopathology ($t = 2.26, p = .025$), social problems ($t = 3.04, p = .003$), and Wave 1 (baseline) delinquent behavior ($t = 5.15, p < .001$).

The same procedure was followed for predicting Wave 4 delinquent behavior with predictors from preschool and Wave 3 middle childhood. Table 24 displays the standardized regression coefficients for each variable. Results for Step 1 are the same as in the previous analysis. In Step 2, predictors from middle childhood (Wave 2) were entered. The model significantly predicted Wave 4 delinquent behavior ($F[16, 203] = 3.76, p < .001$), R^2 was .23, and adding these middle childhood predictors significantly increased the amount of explained variance ($F[10, 203] = 3.20, p = .001$) by 12%. Significant predictors were maternal psychopathology ($t = 2.29, p = .023$) and negative control ($t = 2.14, p = .033$). Delinquent behavior in early adolescence was predicted from more maternal psychopathology and more negative control by parents.

In Step 3, temperament from Wave 3 was added to the model. The model significantly predicted Wave 4 delinquent behavior ($F[19, 200] = 4.06, p < .001$), R^2 was .28, and adding these predictors significantly increased the amount of explained variance ($F[3, 200] = 4.61, p = .004$) by 5%. Significant predictors were maternal psychopathology ($t = 2.16, p = .032$), family cohesion ($t = -2.18, p = .031$), negative

control ($t = 2.26, p = .025$), Wave 3 general activity ($t = 2.73, p = .007$), and Wave 3 task orientation ($t = 2.07, p = .039$). In addition to more maternal psychopathology and negative control, more delinquent behavior in early adolescence was predicted by higher activity levels, more task orientation, and less family cohesion.

Finally, Wave 1 (baseline) delinquent behavior was entered in Step 4. The model significantly predicted Wave 4 delinquent behavior ($F[20, 199] = 5.155, p < .001$), R^2 was .34, and adding this predictor significantly increased the amount of explained variance ($F[1, 199] = 19.04, p < .001$) by 6%. Wave 1 delinquent behavior was the strongest predictor. The effects of maternal psychopathology, family cohesion, negative control, and Wave 3 task orientation were no longer significant. The significant predictors were Wave 3 general activity levels ($t = 2.17, p = .031$) and Wave 1 (baseline) delinquent behavior ($t = 4.36, p < .001$).

Predicting Change Scores. The previous regressions predicted raw aggression and delinquent behavior scores using independent variables from preschool and middle childhood. Would it make a difference if change scores were used instead? To answer this question, Step 4 regression models were re-run with change as the dependent variable. Change was computed by subtracting scores at Wave 1 from scores at Wave 4 (i.e., Wave 4 – Wave 1). Scores at either end of the range reflect discontinuity: High change scores (>0) represent escalation and low change scores (<0) represent improvement. Scores approximating zero reflect continuity (i.e., minimal change). The same pattern of significant predictors was found for both aggression and delinquent behavior, using middle childhood predictors from either Wave 2 or Wave 3, as previously reported in Step 4 of the hierarchical regressions.

Table 23

Hierarchical Regression Results for Delinquent Behavior at Wave 4 with Preschool
(Wave 1) and Middle Childhood (Wave 2) Predictors

Wave	Variable	Standardized Regression Coefficient (β)			
		Step 1	Step 2	Step 3	Step 4
1	Age	.089	.051	.064	-.022
1	Activity	-.046	-.057	-.075	-.044
1	Attention	-.121	-.095	-.094	-.073
1	Reactivity	.097	.090	.039	-.004
1	Maternal Psych.	.242 **	.205 **	.212 **	.154 *
1	Paternal Psych.	.006	-.041	-.050	-.069
2	Reading		-.093	-.088	-.089
2	Spelling		.202	.173	.166
2	Arithmetic		-.082	-.058	-.041
2	Global-Self Esteem		-.023	.014	.015
2	Social Problems		.261 ***	.231 **	.196 **
2	Family Cohesion		.045	.022	.015
2	Family Expressiveness		-.040	-.038	-.044
2	Family Conflict		.189 *	.167 *	.145
2	Spanking		-.052	-.073	-.087
2	Activity			.075	.071
2	Attention			.000	.012
2	Reactivity			.122	.087
1	Delinquent Behavior				.339 ***
Total R ²		.107	.219	.236	.325
ΔR^2			.112 **	.017	.089 ***

*** $p < .001$, ** $p < .01$, * $p < .05$

Table 24

Hierarchical Regression Results for Delinquent Behavior at Wave 4 with Preschool
(Wave 1) and Middle Childhood (Wave 3) Predictors

Wave	Variable	Standardized Regression Coefficient (β)			
		Step 1	Step 2	Step 3	Step 4
1	Age	.089	.083	.058	-.027
1	Activity	-.046	-.049	-.102	-.066
1	Attention	-.121	-.069	-.099	-.075
1	Reactivity	.097	.103	.058	.018
1	Maternal Psych.	.242 **	.161 *	.150 *	.112
1	Paternal Psych.	.006	-.016	-.033	-.057
3	Reading		-.171	-.136	-.082
3	Spelling		.152	.173	.089
3	Arithmetic		-.059	-.056	-.005
3	Global-Self Esteem		-.026	-.042	-.076
3	Social Problems		.107	.082	.075
3	Family Cohesion		-.159	-.179 *	-.109
3	Family Expressiveness		.081	.081	.069
3	Family Conflict		.000	-.006	.048
3	Physical Discipline		.114	.085	.092
3	Negative Control		.151 *	.157 *	.083
3	Activity - General			.219 **	.170 *
3	Activity - Sleep			.109	.097
3	Task Orientation			.164 *	.130
1	Delinquent Behavior				.305 ***
Total R ²		.107	.228	.278	.341
ΔR^2			.121 **	.050 **	.063 ***

*** $p < .001$, ** $p < .01$, * $p < .05$

Table 25

Tolerances for Variables Predicting Wave 4 Aggression and Delinquent Behavior
with Preschool (Wave 1) and Middle Childhood (Wave 2) Predictors

Wave	Variable	Tolerance	
		Aggression	Delinquent Behavior
1	Age	.850	.794
1	Activity	.777	.771
1	Attention	.675	.676
1	Reactivity	.609	.637
1	Maternal Psych.	.725	.724
1	Paternal Psych.	.696	.696
2	Reading	.302	.305
2	Spelling	.252	.252
2	Arithmetic	.528	.527
2	Global-Self Esteem	.809	.812
2	Social Problems	.802	.818
2	Family Cohesion	.589	.589
2	Family Expressiveness	.866	.881
2	Family Conflict	.598	.606
2	Spanking	.815	.826
2	Activity	.773	.781
2	Attention	.552	.552
2	Reactivity	.497	.513
1	Aggression or Delinquent	.624	.776

Table 26

Tolerances for Variables Predicting Wave 4 Aggression and Delinquent Behavior
with Preschool (Wave 1) and Middle Childhood (Wave 3) Predictors

Wave	Variable	Tolerance	
		Aggression	Delinquent Behavior
1	Age	.907	.823
1	Activity	.755	.744
1	Attention	.671	.672
1	Reactivity	.697	.753
1	Maternal Psych.	.745	.742
1	Paternal Psych.	.726	.727
3	Reading	.428	.419
3	Spelling	.382	.367
3	Arithmetic	.554	.543
3	Global-Self Esteem	.770	.764
3	Social Problems	.728	.743
3	Family Cohesion	.532	.511
3	Family Expressiveness	.794	.792
3	Family Conflict	.609	.595
3	Physical Discipline	.553	.544
3	Negative Control	.693	.692
3	Activity	.570	.569
3	Attention	.833	.833
3	Reactivity	.718	.704
1	Aggression or Delinquent	.701	.677

Controlling for Contemporaneous Behaviors. It is possible that the effects of discipline and home environment during middle childhood can be explained by concurrent levels of antisocial behavior. For example, the greater level of family conflict at Wave 2 (6-8 years) experienced by those who will later show heightened levels of aggression symptomatology in early adolescence may be a function of heightened levels of aggressiveness at 6-8 years of age.

To address this issue, the four final regression models using middle childhood variables to predict Wave 4 behavior problems were re-run with contemporaneous behavior problem scores as an additional predictor (i.e., Wave 2 aggression or delinquent behavior in the models with Wave 2 middle childhood predictors, and Wave 3 aggression or delinquent behavior in the models with Wave 3 middle childhood predictors). In each run, adding contemporaneous behavior problems significantly increased the amount of explained variance ($p < .001$). Standardized regression coefficients are in Table 27 for models that include middle childhood predictors from Wave 2, and in Table 28 for models that include middle childhood predictors from Wave 3.

For the regression with Wave 2 variables predicting Wave 4 aggression controlling for Wave 2 aggression (see Table 27), the effects of social problems, family conflict, and baseline aggression become statistically insignificant. Wave 2 aggression was the strongest predictor ($t = 5.16, p < .001$), with age ($t = -2.75, p = .006$) and global self-esteem ($t = -2.65, p = .009$) also predicting aggression during early adolescence.

For the regression with Wave 3 variables predicting Wave 4 aggression controlling for Wave 3 aggression (see Table 28), physical discipline and negative control become insignificant – which suggests that contemporaneous behavior problems

may explain them. Wave 3 aggression was a significant predictor ($t = 5.97, p < .001$) but did not eclipse the effects of baseline aggression ($t = 3.41, p = .001$), Wave 3 general activity level ($t = 2.34, p = .021$), or two family environment variables: cohesion ($t = -2.25, p = .026$) and expressiveness ($t = 2.38, p = .018$). Age also was marginally significant ($t = -.98, p = .049$). Although punishments may overlap with contemporaneous behavior problems, family cohesion and expressiveness at 9-11 years of age make significant contributions to understanding later aggression.

None of the contextual variables related to family environment or discipline were still significant by Step 4 of the hierarchical regressions predicting delinquent behavior in early adolescence. However, the results do demonstrate the importance of more temporally proximate behavior problems for predicting later outcome.

For the regression with Wave 2 variables predicting Wave 4 delinquent behavior controlling for Wave 2 delinquent behavior (see Table 27), maternal psychopathology and social problems were no longer significant. In fact, the only two significant predictors were Wave 1 delinquent behavior ($t = 2.47, p = .015$) and Wave 2 delinquent behavior ($t = 7.08, p < .001$).

For the regression with Wave 3 variables predicting Wave 4 delinquent behavior controlling for Wave 3 delinquent behavior (see Table 28), Wave 3 activity level dropped from significance. Only baseline ($t = 3.10, p = .002$) and Wave 3 ($t = 5.79, p < .001$) delinquent behavior were significant predictors.

Table 27

Regression Results for Aggression and Delinquent Behavior at Wave 4 with Preschool
(Wave 1) and Middle Childhood (Wave 2) Predictors Including Contemporaneous
(Wave 2) Behavior Problems

Wave	Variable	Standardized Regression Coefficient (β)	
		Aggression	Delinquent Behavior
1	Age	-.161 **	-.052
1	Activity	.044	-.073
1	Attention	.001	-.062
1	Reactivity	-.016	.029
1	Maternal Psych.	.089	.121
1	Paternal Psych.	-.088	-.087
2	Reading	.025	-.062
2	Spelling	.034	.131
2	Arithmetic	-.129	-.045
2	Global-Self Esteem	-.155 **	-.005
2	Social Problems	.031	.035
2	Family Cohesion	.007	.067
2	Family Expressiveness	.064	-.052
2	Family Conflict	.093	.107
2	Spanking	-.054	-.056
2	Activity	.034	.089
2	Attention	.062	.051
2	Reactivity	.076	.035
1	Aggression or Delinquent	.126	.159 *
2	Aggression or Delinquent	.443 ***	.499 ***
Total R^2		.447	.461
ΔR^2 (from previous Step 4 model)		.074 ***	.136 ***

*** $p < .001$, ** $p < .01$, * $p < .05$

Table 28

Regression Results for Aggression and Delinquent Behavior at Wave 4 with Preschool
(Wave 1) and Middle Childhood (Wave 3) Predictors Including Contemporaneous
(Wave 3) Behavior Problems

Wave	Variable	Standardized Regression Coefficient (β)	
		Aggression	Delinquent Behavior
1	Age	-.101 *	.004
1	Activity	-.010	-.067
1	Attention	.023	-.088
1	Reactivity	-.025	.045
1	Maternal Psych.	.000	.098
1	Paternal Psych.	-.037	-.057
3	Reading	-.014	-.097
3	Spelling	.039	.064
3	Arithmetic	-.040	.040
3	Global-Self Esteem	-.046	-.093
3	Social Problems	-.033	-.004
3	Family Cohesion	-.150 *	-.091
3	Family Expressiveness	.130 *	.053
3	Family Conflict	-.006	-.013
3	Physical Discipline	.081	.059
3	Negative Control	.111	.063
3	Activity - General	.155 *	.119
3	Activity - Sleep	.005	.047
3	Task Orientation	.013	.132
1	Aggression or Delinquent	.205 **	.207 **
3	Aggression or Delinquent	.433 ***	.379 ***
Total R ²		.535	.437
ΔR^2 (from previous Step 4 model)		.084 ***	.095 ***

*** $p < .001$, ** $p < .01$, * $p < .05$

Changing the Order of Variable Entry. Finally, the steps in the hierarchical regression were reordered to answer one more question: Do middle childhood predictors contribute significantly to the model after we take into consideration what we know about the development of the problem behaviors themselves? In other words, is there a significant R^2 change when middle childhood predictors are added *after* Wave 1 and middle childhood aggression or delinquent behavior scores?

To address this issue, variables were entered in the following order: Wave 1 aggression or delinquent behavior (Step 1), Wave 1 predictors (Step 2), middle childhood aggression or delinquent behavior (Step 3), middle childhood predictors (Step 4), and then middle childhood temperament control variables (Step 5).

When aggression was predicted from preschool and Wave 2 variables, adding the Wave 1 predictors after the aggression scores from Wave 1 resulted in a non-significant R^2 change for Step 2 ($F[6, 212] = 1.04, p = .401$). However, adding the Wave 2 middle childhood predictors after the Wave 2 middle childhood aggression scores still led to a significant increase in explained variance at Step 4 ($F[9, 202] = 2.15, p = .027$). The same pattern was found in the model predicting aggression from preschool and Wave 3 variables. Adding the Wave 3 middle childhood predictors after the Wave 3 middle childhood aggression scores significantly increased the R^2 with Step 4 ($F[10, 201] = 1.95, p = .040$). For aggression, then, middle childhood variables take precedence over preschool contextual and temperamental factors – and add to our understanding of how aggression develops beyond what we would understand from only looking at aggression itself.

When delinquent behavior was predicted from preschool and Wave 2 variables, adding the Wave 1 predictors after the delinquent behavior scores from Wave 1 resulted in a non-significant R^2 change for Step 2 ($F[6, 212] = 1.64, p = .137$). Similarly, adding the Wave 2 middle childhood predictors after the Wave 2 middle childhood delinquent behavior scores revealed a non-significant increase in explained variance for Step 4 ($F[9, 202] = .79, p = .628$). The same pattern was found in the model predicting delinquent behavior from preschool and Wave 3 variables. Adding the Wave 3 middle childhood predictors after the Wave 3 middle childhood delinquent behavior scores yielded a non-significant change in R^2 for Step 4 ($F[10, 201] = .96, p = .479$). As in the contemporaneous models, only earlier delinquent behavior predicted delinquent behavior among early adolescents.

Summary. Hypothesis 3 was generally supported for both aggression and delinquent behavior. The high-high and low-low groups were distinguishable from one another on a number of different characteristics. Change groups were generally intermediate in their risk profiles. However, these groups did not completely occupy the “middle ground.” In some cases these groups were more similar to one continuity group than the other – or conversely, more different from one continuity group than the other.

It is worth noting that classification groups were as expected on the variable that defined them from Wave 2 through Wave 4 – with equal “highs” (i.e., for high-high and low-high) and equal “lows” (i.e., for low-low and high-low) by 9-11 years of age, and with some intermediacy in scores for the change groups during middle childhood. Interestingly, the results demonstrate that the cross-over from low to high or high to low has already occurred by Wave 3; Wave 2 seems to be the important turn-point.

There were two unexpected findings at Wave 1. For aggression, those with initially “low” levels of symptomatology were different from one another; for delinquent behavior, those with initially “high” levels of symptomatology were different from one another. Some caution is therefore warranted when describing the constellation of risks associated with the escalating pattern for aggression (i.e., low-high versus low-low) and the remitting pattern for delinquent behavior (i.e., high-low versus high-high) using univariate tests like ANOVA; it may be that baseline differences in aggression and delinquent behavior can account for some later disparities.

Hypothesis 4 was also partially supported. Of the initial trajectory risk factors, only concurrent levels of maternal psychopathology and reactivity predicted preschool levels of aggression and delinquent behavior. Adding in middle childhood risk/promotive factors helped to explain aggression and delinquent behavior during adolescence. In fact, middle childhood risk factors were much more useful than more distal preschool risk factors for predicting adolescents’ antisocial behavior; maternal psychopathology at Wave 1 was the only contributing factor from the baseline trajectory. Many significant middle childhood predictors remained significant even after controlling for baseline levels of antisocial behavior (but much more so for aggression). Contrary to expectations, school achievement was relatively unimportant.

When baseline levels of aggression and delinquent behavior were included in the model, the same predictors were identified as significant regardless of whether raw scores or change scores were used as dependent variables. Controlling for contemporaneous behavior problems in middle childhood mitigated the influence of some variables (e.g., discipline at 9-11) but not others (e.g., family cohesion and expressiveness at 9-11) for

adolescent aggression. At this stage, only previous delinquent behaviors predicted later delinquent behaviors.

CHAPTER 5 DISCUSSION

The present study examined early age of first drink (AFD) from a perspective that emphasized early alcohol use as a marker in a more longitudinal pattern of problem behaviors – specifically, aggression and delinquent behavior from preschool onward. This perspective, looking at early AFD as the outcome of an ongoing developmental trajectory characterized by antisociality, stands in contrast to studies focusing on early AFD as a precursor to other outcomes (e.g., alcoholism) as well as a literature on drinking initiation that emphasizes concurrent or more proximate influences in the personal and social surround of adolescents (e.g., research on peer influences or concomitant levels of family conflict).

Consistent with a life-course or developmental perspective on problem behavior, the present study explored the utility of aggression and delinquent behavior trajectories for explaining early AFD. Hypothesizing that hard-continuity in aggression and delinquent behaviors is associated with first drink onset by 12 to 14 years of age, early drinkers were expected to demonstrate higher levels of problem behavior symptomatology from preschool to early adolescence – with levels of symptomatology in preschool (3-5) and early adolescence (12-14) serving as particularly strong markers of the developmental trajectory and early onset risk. Although continuity was assumed to be the more common trend, change patterns were also recognized. It was hypothesized that those with either remitting or escalating behavior problems would be at intermediate risk for drinking onset and would experience intermediate levels of risk across a broad domain of preschool and middle childhood risk (or, conversely, promotive) factors.

Finally, the role of these middle childhood variables – in combination with the initial risk trajectory – was explored in terms of predicting severity of antisocial behaviors during preschool and early adolescence.

The overarching premise is that if early alcohol use is associated with longitudinal patterns of aggression and delinquent behavior, we might be able to prevent precocious drinking by disrupting this behavioral trajectory. Efforts to do so should be informed by a better understanding of the etiological factors relevant to antisocial behavior, particularly from preschool and middle childhood (i.e., early in the developmental process – before behaviors become too ingrained, before cumulative disadvantages from school and social relationships snowball into an epigenetic rut, and before regular drinking becomes part of the behavioral repertoire). Findings from the current study are summarized and discussed here in terms of 1) onset of first drink, 2) the development of antisocial behavior, 3) limitations and future directions, then 4) concluding remarks.

Onset of First Drink

The results from testing Hypothesis 1 and Hypothesis 2 support the decision to look separately at aggression and delinquent behavior. The idea that early drinkers can be distinguished from those who have not yet tried alcohol by early adolescence using a hard-continuity model of problem behavior was supported for delinquent behavior only.

In general, levels of aggression declined for both groups (early drinkers and non-drinkers alike) from 3-5 to 12-14 years of age. This fits with other literature describing a decline in aggression from preschool through adolescence (Bongers, Koot, van der Ende, & Verhulst, 2003; Stanger, Achenbach, & Verhulst, 1997; Tremblay, 2002). The only group difference in the current study was for aggression at 12-14, with early drinkers

rated by their mothers as more aggressive. In general, however, early drinkers and those who have not tried alcohol by 12-14 years of age showed more similarity than difference in the developmental trajectory of aggression. Aggression levels during preschool and early adolescence (i.e., low-low, low-high, high-low, high-high) also were not significantly related to onset of first drink.

On the other hand, early drinkers were more delinquent than their never-drinking peers at all ages except 9-11 – when the level of symptomatology for early drinkers appeared to diminish and converge with that of those who have never tried alcohol, before rising again in early adolescence. This, too, is consistent with other research. For example, Bongers et al. (2003; see also Stanger et al., 1997) found that scores on the delinquent behavior scale of the CBCL were curvilinear from 4 to 18 years of age, initially declining but then increasing in early adolescence. Interestingly, the current study revealed a developmental increase in delinquent behavior for early drinkers but not for those who hadn't yet tried alcohol by 12-14 years of age (revisit Figure 6).

Bongers et al. (2003; see also Stanger et al., 2003) tied the “normative” adolescent-increase they observed to the “normative” delinquency of adolescents described by theorists like Terrie Moffitt. This increase is also reflective of the ubiquitous “age-crime curve”: At the aggregate level, there is a steep incline in antisocial behavior between the ages of 7 and 17, followed by a steep decline from 17 to 30 (tapering off thereafter) (Moffitt, 1993). For Moffitt (1993), however, the age-crime curve is a consequence of those who engage in delinquency only during adolescence (i.e., the adolescent-limited type) temporarily accompanying those with persistent delinquent behavior problems (i.e., the life-course-persistent type) in antisocial involvement. As

Moffitt (1993, p. 675) has stated, “Actual rates of illegal behavior soar so high during adolescence that participation in delinquency appears to be a normal part of teen life.”

It was therefore surprising that, in the current study, there was no coinciding increase seen for the non-drinker group. In retrospect, however, this study only begins to look at problem behavior during adolescence, ending as it does with reports from 12-14 years of age. Those who have either persisted in their delinquent behaviors from preschool or who escalate in delinquent activity very early in adolescence (i.e., by 12-14 years of age) are probably quite different from those who initiate delinquent behavior during middle-to-late adolescence (i.e., the “normative” years). As a group, the early drinkers were therefore precocious in their first use of alcohol as well as in their delinquent involvements going into adolescence.

An alternate explanation focuses on the dip in delinquent behavior for the early drinker group at ages 9-11 rather than the subsequent increase at ages 12-14. It is possible that the predisposition to engage in markedly more delinquent behaviors remained constant but that control structures during middle childhood (e.g., socialization by the school) temporarily reduced the enactment of these tendencies – or, alternately, that mothers were less aware of these behaviors; if more autonomy is granted to pre-adolescents than young children, the observed dip may be an artifact of less supervision and parental oversight. In either case, the idea that delinquent behavior is “normative” during adolescence may have led to its return during early adolescence despite control structures or even because of them (i.e., in a “knifing-off childhood apron strings” gesture of rebelliousness; Moffitt, 1993, p. 688).

Beyond simple statistics, complex analyses comparing early drinkers to those who

had not yet tried alcohol by 12-14 years of age also supported the idea of a delinquent behavior substrate within the early drinker group. Two findings speak to this conclusion.

First, there were omnibus differences in the developmental trajectories of delinquent behavior for early drinkers and non-drinkers (revealed in a stacked autoregressive structural equation model). One of these differences was in the direct effect of preschool delinquent behavior on delinquent behavior during early adolescence. Specifically, the path from Wave 1 to Wave 4 delinquent behavior was significant for early drinkers only. The direct path represents the influence of early predispositions on adolescent outcomes and supports the idea of hard-continuity for this subset of individuals. One implication is that a high level of delinquent behavior as early as age three should serve as a “red flag” to parents. Children with this type of symptomatology are prime candidates for early intervention programs that seek to disrupt the behavioral trajectory at a young age (perhaps preventing early onset of alcohol use and related sequelae in the process).

Second, configural frequency analysis revealed that those with high levels of delinquent symptomatology in both preschool and adolescence (i.e., the high-high delinquent behavior group) were more likely than chance to have had a first drink, whereas the opposite pattern was found for those with low levels of delinquent symptomatology throughout (i.e., the low-low delinquent behavior group). In other words, there was a relationship between continuity of delinquent behaviors and likelihood of early AFD consistent with the hard-continuity model of behavior problems.

The findings presented thus far raise an important question: Why this relationship with drinking onset for delinquent behavior but not aggression? Three explanations are

offered: two conceptual, one methodological.

Conceptually, there is a difference between aggression and delinquent behavior that can account for both the distinct developmental patterns found in the current study and by other researchers (e.g., Bongers et al., 2003) as well as the relationship with first alcohol use only for delinquent behavior. This difference is one of kind: Delinquent behavior may be more “deviant” than aggression.

Aggression among preschool children is normative to some extent, as individuals learn to internalize the norms and values of conventional society and begin to master the skills of emotion regulation and behavior regulation. The ability to use language to negotiate through interpersonal relationships is acquired in a developmental process which also may parallel changes in aggression over time; indeed, aggression is expected to decline during childhood as communication skills become more proficient. CBCL items used to measure aggression include boasting, arguing, screaming, showing off, fighting, and general disobedience.

Delinquent behavior, as assessed by the CBCL, is more intrinsically connected to criminality (e.g., stealing, setting fires, and vandalism). Consider the difference between the following two individuals: One pushes people around, demands attention, destroys things in a temper tantrum, and lacks the social skill of subtlety; the other commits theft, sets fires, lies, swears, and runs away from home. Although both individuals should be of concern to parents, schools, researchers, and prevention workers, the second individual engages in less common (i.e., more “deviant”) activities: Many children bite, push, and have temper tantrums; fewer steal or set fires.¹² The development of delinquent behavior

¹² This can also be demonstrated empirically. The sample mean for aggression was 10.65 at Wave 1

also better parallels the age-crime curve (see also Bongers et al., 2003; Stanger et al., 1997).

An alternate conceptual distinction concerns developmental specificity. In other words, the difference between aggression and delinquent behavior may be outcome-dependent. Of key importance here is the distinction between the overt and covert antisocial pathways described by Loeber and colleagues (Loeber & Hay, 1997; Loeber & Stouthamer-Loeber, 1998).

According to Loeber (Loeber & Hay, 1997; Loeber & Stouthamer-Loeber, 1998), antisocial behavior can be best understood with a triple-pathway model. Each path has a unique developmental course leading to particular outcomes. The overt pathway begins with minor aggression (e.g., bullying, annoying others) and escalates through physical fighting to violent crime (e.g., rape, assault). The covert pathway begins with minor covert behaviors (e.g., shoplifting, lying) and escalates through property damage (e.g., vandalism, fire-setting) to more serious forms of delinquency (e.g., fraud, burglary, serious theft). Finally, the authority conflict pathway begins with stubborn behavior and escalates through defiant disobedience to problems associated with authority avoidance (e.g., truancy, staying out past curfew). Overt behaviors tend to decrease with age, whereas covert behaviors increase with age (Loeber & Stouthamer-Loeber, 1998).

and 7.06 at Wave 4; prorating these by a possible high score of 40 yields .226 for Wave 1 and .176 for Wave 4. The sample mean for delinquent behavior was 1.88 at Wave 1 and 1.85 at Wave 4; prorating these by a possible high score of 24 yields .078 for Wave 1 and .077 for Wave 4. Therefore, aggressive behavior problems were almost three times more common than delinquent behavior problems at Wave 1 (i.e., .226 versus .078), and still more than twice as common at Wave 4 (i.e., .176 versus .077).

Overt behaviors appear to characterize the aggression subscale of the CBCL, whereas covert behaviors seem to characterize the delinquent behavior subscale of the CBCL – in both content and development trends. Of relevance to the current study is the relationship of these behaviors to early first alcohol use. Alcohol use is not part of Loeber’s conceptual scheme, but is non-violent and likely to be associated with the covert (rather than overt) pathway. If the outcome measure for the current study was date rape, the result may have been a stronger association with aggression rather than delinquent behavior. Rather than delinquent behavior being more “deviant,” perhaps it is just more consistent with a developmental path that also includes substance use.

There is also a methodological explanation for the stronger relationship with first alcohol use found for delinquent behavior compared to aggression. The CBCL delinquent behavior syndrome scale was initially constructed to include an item on substance use. This item was dropped from the current set of analyses to avoid the obvious confound with first drink onset status. Nonetheless, CBCL scales were derived from a factor analysis. The CBCL delinquent behavior scale used in the present study, although not inclusive of substance use, therefore may be predisposed towards an association with substance use – with this scale and first drink onset status “hanging together” by virtue of the way the delinquent behavior subscale was constructed. Caution is certainly warranted in the interpretation of these findings, and future research is needed to address the question of whether delinquent behavior is in fact more strongly tied to first alcohol use than aggression when separate measures of delinquent behavior are used. However, the next section of this discussion returns to this issue of difference – offering some support for the idea that delinquent behavior is, indeed, more consistent

with the hard-continuity paradigm than aggression.

The Development of Antisocial Behavior

Given the hard-continuity paradigm and problem behavior theory perspective adopted to explain early first alcohol use, the next step in the current study was to examine the etiology of antisocial behavior itself. First, the four classification groups for aggression and delinquent behavior (i.e., low-low, low-high, high-low, high-high) were compared on general risk profiles from preschool and middle childhood. These risk (or promotive) factors were then used to predict severity of aggression and delinquent behavior in preschool (Wave 1) and early adolescence (Wave 4).

“High” and “low” antisocial behavior scores were expected to mean the same across classifications (e.g., with the low-low and low-high groups indistinguishable during preschool, but the low-high and high-high groups indistinguishable during early adolescence). Two exceptions were found at Wave 1. The low-high group for aggression was already more aggressive than the low-low group – meaning that this change pattern might be driven by more risky predispositions in addition to the presence or absence of other risk/promotive factors during middle childhood. Similarly, the high-low group for delinquent behavior was already less delinquent than the high-high group – meaning that this change pattern might be driven by less risky predispositions in conjunction with other developmental “turning points”. Some caution in interpreting differences between these group pairs is therefore warranted as initial problem behavior severity was not truly equivalent. At subsequent waves, classifications fit with expected patterns. By Wave 4, both high-high and low-high groups were indistinguishable, as were both low-low and high-low groups.

Fergusson et al. (1996) found that those with no conduct problems had the most favorable risk profiles on family and child characteristics, those with persistent conduct problems had the least favorable profiles, and children with remitting or late onset patterns showed profiles situated between these two extremes. These findings were generally replicated in the current study.

Indeed, risk profiles in the low-low group were less serious than in the high-high group for both aggression and delinquent behavior. For both aggression and delinquent behavior, the low-low group presented with a less risky profile with respect to reactivity and maternal psychopathology at Wave 1; social problems, family conflict, attention, and reactivity at Wave 2; as well as social problems, negative control, and activity-general at Wave 3.

That the low-low versus high-high comparisons were so similar across antisocial behavior subtypes speaks to the importance of these particular risk/promotive factors in the development of behavior problems from preschool to adolescence. There were several differences, however. For aggression, the low-low group also had less paternal psychopathology at Wave 1 and less family conflict at Wave 3 than the high-high group. For delinquent behavior, the low-low group had more family cohesion at Wave 3 than the high-high group.

Change groups were generally intermediate in their risk profiles. However, these groups did not completely occupy the “middle ground.” In some cases these groups were more similar to one continuity group than the other. Conversely, the continuity group they differed from in these instances reveals something about the change pattern itself (e.g., which factors are related to the diverging trajectories of those with initially high or

initially low levels of symptomatology – and therefore, to remitting or escalating antisocial behaviors).

The remitting pattern (i.e., high-low versus high-high) was associated with less reactivity at 6-8 years and fewer social problems at both middle childhood waves for aggression; less family conflict and longer attention spans at 6-8 years and less negative control at 9-11 years for delinquent behavior; as well as less general activity at 9-11 years for both aggression and delinquent behavior. At 9-11 years of age, both high-low groups seemed most like the low-low groups (i.e., and less like the high-high groups) on incidence of physical discipline.

Recall that the high-low and high-high delinquent behavior groups could be differentiated from one another at 3-5 years of age. The already-higher level of delinquent involvement among the high-high group suggests that one contributing factor to persistence rather than improvement is one's initial predisposition to some extent (although the high-low group was more delinquent than those with low levels of symptomatology during preschool – so significant improvement did take place over the developmental course nonetheless).

The finding of less difficult temperament for remitting groups during middle childhood compared to those with persistent behavioral difficulties suggests that temperament and behavior problems may be reflections of the same underlying construct or disposition. Both can change – and do, in the same direction. For example, although the high-low aggression group was more reactive than those with initially low levels of aggressive symptomatology during preschool, this difference had disappeared by 6-8 years of age. These results are consistent with some of the most popular perspectives on

temperament (e.g., Chess & Thomas, 1989; Worobey, 1999) and lend support to dynamic views of the behavior-temperament nexus. Temperament isn't immutable; its initial risk trajectory is subject to alteration. However, it cannot be determined here whether change in temperament precedes, follows, or is concordant with change in behavior problems.

Those who start of as highly antisocial but are not characterized as such by adolescence also seem to experience more favorable family environments during middle childhood (e.g., with regard to negative control, family conflict, and/or physical discipline). This can be interpreted in several ways. Conflict, negative control, and physical discipline may exacerbate the initial predispositions of aggressive or delinquent children to the extent that an adverse home context and family dynamics (e.g., inadequate parenting) become part of the maintenance structure for their behavior problems (e.g., Moffitt, 1993; Patterson et al., 1989). Conversely, the absence of this familial risk may enable some individuals to “phase out” of their early behavioral tendencies and allow them to take advantage of “turning point” moments in their development. The alternate explanation is also possible: The remitting pattern emerges first – leading to less conflict, control, and physical discipline because the child behaves better, doesn't aggravate his parents as much, and isn't involved in as many activities that require disciplinary action compared to children with persistent antisocial behaviors (i.e., doesn't “evoke” the same style of parenting or parental aggravation as persisters). The fact that the remitting pattern was associated with longer attention spans (at 6-8) for delinquent behavior and less general activity (at 9-11) for both aggression and delinquent behavior provides some support for the “less evocative” stance – because remitters do present with less risky temperaments during middle childhood. However, it is impossible to

completely disentangle causal order in the current research design.

There is a similar quagmire with respect to the causal ordering of social problems and aggression. Those with remitting aggressive behaviors had fewer social problems than those with persistent aggression. This is interesting given that the two groups started off in preschool with statistically indistinguishable levels of aggressive symptomatology. These initial levels therefore cannot account for the divergence in the quality of peer relationships; at least for aggression, preschool behaviors do not automatically “knife off” opportunities for social engagement. There are two explanations for the divergence. Either conventional peer relationships “protect” those in the remitting group and allow for a “turning point” in behavioral trajectories (conversely, that poor social relationships maintain the persistent behavior problem trajectory), or aggression begins to remit first – leading to less peer rejection and better social relationships. The likelihood is that both explanations apply: Behavior problems may evoke peer rejection; peer rejection may lead to rebellious or frustration-related problem behavior. Rejected children may also join in with antisocial others in response to their more conventional social failures (e.g., see Patterson et al., 1989). Those with remitting behavior patterns for aggression break the cycle at some point.

The remitting pattern is important to consider because it represents relative desistence from aggression or delinquent behaviors. Prevention and intervention programs can capitalize on a better understanding of the natural processes that inhibit or bring about this change. Findings from the current study suggest that programs focused on family dynamics and social problems may be beneficial in either disrupting aggressive and delinquent behavioral trajectories or disrupting their consequences socially and in the

home – thereby helping to break self-perpetuating maintenance structures of maladaptation.

Prevention efforts, and future research endeavors, might also benefit by looking beyond these familial and social risk factors to those ecological variables that, in turn, shape these interpersonal relationships (as well as individual behaviors) (Bronfenbrenner, 1977; Evans, 2003; Fraser, 1996; Jack, 2000; Tolan, Gorman-Smith, & Henry, 2003). For example, neighborhood violence and poverty are two ecological variables that have received much attention in this literature. An even broader appreciation of the contextual, risk accumulation model can be adopted than studied here – with risky child behaviors (e.g., aggression, delinquency) nested in risky immediate environments (e.g., familial, peer) which in turn are nested in risky ecological settings (e.g., community, socioeconomic). Just as it is important to consider familial and social factors as ecological influences with much explanatory power, there are wider ecological or structural domains that impinge on these. To address family dynamics and social problems, intervention/prevention efforts must keep in mind the role played by influences at all levels of human ecology.

In addition to the remitting patterns, escalating patterns are also important because they can provide some insight into what aggravates antisocial behavior. Looking beyond the risk factors that maintain the developmental trajectory of children with behavioral difficulties, which escalate the development of behavioral difficulties among others?

For aggression, the escalating pattern (i.e., low-high versus low-low) was associated with more maternal psychopathology at 3-5 years, lower self-esteem at 6-8 years, as well as more family conflict, more general activity, and less task orientation at 9-11 years. For both aggression and delinquent behavior, the low-high group seemed

most like the high-high group on incidence of physical discipline. Interestingly, the low-high delinquent group was similar to the low-low delinquent group in having fewer social problems than the high-high group at Wave 2.

Again, it should be remembered that the low-high and low-low aggression groups were already different from one another in preschool. For aggression, then, one determinant of escalation may be this riskier predisposition compared to those who remained relatively unproblematic.

Of interest is the fact that the risk/promotive factors included in the current study did little to illuminate the processes associated with escalating problems for delinquent behavior. Aside from the observation that those in the escalating delinquent behavior group experienced more physical discipline at 9-11 than the low-low delinquent behavior group (i.e., 23% versus 5%), there were no significant differences between these two classifications. The only other relevant finding was that the low-high group had fewer social problems than the high-high group at 6-8 (and was similar to the low-low group in this regard). For delinquent behavior, then, early social problems seem more dependent on initial levels of symptomatology. The low-high group did become intermediate at 9-11 years of age, however. That the cross-over from “low” to “high” had already occurred by Wave 2 for the group as a whole suggests that there is a lag between the escalation of delinquent behaviors and escalation of social problems. This suggests causal order: Peer rejection seems to be a consequence of, rather than an antecedent to, delinquent symptomatology for those with emergent delinquent behavior problems.

Many more characteristics distinguished those with escalating aggression from the low-low group. As previously mentioned, temperament often changed in sync with

behavior problems; those with escalating patterns of aggression were more active and less task oriented by ages 9-11. There was more family conflict and physical discipline at 9-11 years of age for the escalating aggression group compared to the low-low aggression group (i.e., 26% versus 5%). The same issue of ambiguity in temporal ordering discussed with respect to the remitting pattern applies here: Conflict and discipline may be a consequence of, rather than a contributing factor to, the child's aggressive behaviors. In fact, that the family environment was not more adverse until Wave 3 supports the "evocative" explanation; it was not until after the (aggregate) Wave 2 cross-over from "low" to "high" that significant differences in family environment or discipline style were observed.

Those with escalating aggression also had lower self-esteem than the unproblematic low-low group at 6-8, but not 9-11, years of age. Therefore, lower self-esteem appears to temporarily accompany the transition from "low" to "high." It's unclear from the current research design whether lower self-esteem is a consequence or cause of escalating aggression. Nonetheless, this is an intriguing finding that deserves future attention. If low self-esteem leads to aggression, programs geared toward fostering positive feelings about one's self might prevent this escalation. If increased aggression leads to lower self-esteem (mediated, perhaps, by changes in the family environment or parents' reactions), this could fuel a child's frustration and perpetuate his problems.

There are several interpretations for the finding that maternal psychopathology was lower for the low-low aggression group than the low-high aggression group – and all others. Maternal psychopathology was also lower for the low-low delinquent behavior group than the high-high delinquent behavior group. It may be that the low-low groups

are at the least risk in terms of less exposure to alcoholic, antisocial, and/or depressed mothers – and that this relative absence of maternal risk is what maintains the low-aggression or low-delinquent behavioral trajectory. However, it may also be the case that the other groups are rated high on aggression or delinquent behavior at some point because psychopathology has affected mothers' perceptions of their children's behavior problems (rather than because of the behavior problems per se). In other words, mothers with more psychopathology may be more critical or less tolerant of their children's behavior than mothers with less psychopathology.

Finally, beyond specific issues related to remitting or escalating behavior problems, the current study revealed an interesting developmental pattern in the relationship between temperament and antisocial behavior. Among young children, there seems to be a connection between reactivity and antisocial behavior. Those high on aggression during preschool were more reactive than those low on aggression during preschool. Those with persistent delinquent behaviors were more reactive than those low in delinquent behaviors during preschool (although those with remitting delinquent behaviors were not). There were no group differences on other temperament dimensions at 3-5 years of age.

Other dimensions of temperament came into play during middle childhood. Reactivity remained an important risk marker; those with persistent aggression problems were more reactive than all others by Wave 2, and those with persistent delinquent behavior problems were more reactive than the low-low group at Wave 2. Now attention had emerged as another distinguishing characteristic. Those with persistent aggression problems had shorter attention than those in the low-low aggression group; those with

persistent delinquent behavior problems had shorter attention than either group with low levels of adolescent symptomatology. By Wave 3 (9-11), the major distinguishing dimension of temperament was general activity. Those with persistent behavior problems were more active than either group with low levels of adolescent symptomatology for both aggression and delinquent behavior. In addition, those with escalating aggression problems were more active and less task oriented than the low-low group.

To interpret these findings, remember that there was a change in instrumentation between the second and third wave of data collection (from the DOTS to the DOTS-R). The general activity subscale of the DOTS-R overlaps somewhat with the reactivity scale of the DOTS. That said, persistent behavior problems were associated with more reactivity over the developmental course as well as attenuated attention and higher activity levels during middle childhood. Like communication skills and self-regulation, the ability to focus one's attention is learned with time. For all groups, scores for attention increased from Wave 1 to Wave 2. Preschoolers are expected to have attention-related deficits because of their age. Differences in attention really become relevant after school entry. By middle childhood, children should already be mastering attention skills. It makes sense that those who have trouble with this developmental task are also those with other behavioral difficulties, and that differences in attention become evident only during this time period. That general activity became the risk marker at ages 9-11 can be explained by its overlap with reactivity, its absence in earlier assessments (i.e., activity at Wave 1 and Wave 2 was sleep-oriented), and the refinement of self-regulatory skills over time. As with attention deficits, high activity levels are normative for young children but become more restrained over the course of development. Again, it makes sense that

over-active children – those who haven't mastered self-regulation quite as well as other children – would have other behavior problems.

In the final set of analyses, the risk/promotive factors already discussed were used to explain the initial risk trajectory and adolescent outcomes. These analyses address some of the previous concerns about causal order in that regression procedures allowed for an examination of the effect of each variable *controlling for all others*.

Reactivity and maternal psychopathology were the only significant predictors of initial levels of problem behavior severity. This reaffirms the importance of reactivity as an early risk marker. More maternal psychopathology was associated with more aggression and delinquent behavior during preschool, even controlling for maternal ratings of child temperament – a possible substrate of behavior. Although this does not negate the possible confound of maternal psychopathology and maternal behavior problem ratings, it does provide some support for the contention that maternal alcoholism, antisociality, and depression play a role in establishing the initial risk trajectory. Similar findings have been reported by other researchers from the Michigan Longitudinal Study (Fitzgerald et al., 1993; Fitzgerald et al., 1995).

Why maternal psychopathology but not paternal psychopathology? This may be an artifact of sample characteristics. Paternal psychopathology was over-represented among these children because the Michigan Longitudinal Study is a population-based study of paternal alcoholism. A disproportionate number of alcoholic fathers (i.e., approximately two-thirds) were recruited for this reason. Therefore, there may be a saturation effect with respect to paternal psychopathology. What probably makes a difference is whether children are exposed to psychopathology from both parents versus

one or none. This echoes the conclusion drawn by Mun (2002) for the influence of only one versus both parents' smoking and alcoholism on early onset smoking by offspring. Having a mother low in psychopathology may serve as an important protective factor in the child's development (directly buffering the effects of paternal deviance, or with an alternate set of behaviors being modeled).

Early behavior problems were the best predictors of adolescent behavior problems when predicting adolescent outcomes. This is strong evidence in support of the hard-continuity model. In addition, risk/promotion factors from middle childhood were much more influential than other facets of the initial risk trajectory. For example, preschool reactivity was no longer a key influence when predicting this distal outcome. These findings speak to the viability of prevention and intervention efforts which don't get started until school entry: Middle childhood matters; it's not too late to mitigate some of the risk associated with adolescent behavior problems.

Controlling for initial levels of aggression, key predictors of aggression in early adolescence also included global self-esteem, social problems, and family conflict at ages 6-8; as well as family cohesion, family expressiveness, physical discipline, negative control, and general activity at ages 9-11. When contemporaneous behavior problems were taken into consideration, only global self-esteem at ages 6-8 also remained significant. At ages 9-11, the punishment variables (physical discipline, negative control) dropped from significance but all other predictors remained the same.

Controlling for initial levels of delinquent behavior, other key predictors of delinquent behavior in early adolescence included maternal psychopathology and social

problems at ages 6-8; but only general activity at ages 9-11. Only the delinquent behavior control variables remained significant in the contemporaneous model.

Of these findings, several are particularly intriguing. Social problems at 6-8 were a significant predictor of later aggression and delinquent behavior. However, social problems were no longer significant at 9-11 years of age (once initial levels of aggression and delinquent behavior were taken into account). This reaffirms the importance of peer relations as a key marker in the developmental trajectory. As mentioned, social problems have featured prominently in other research on behavioral difficulties (Campbell, 1994; Campbell et al., 1986, Campbell & Ewing, 1990) and theories of delinquency – including the work of Patterson et al. (1989) as well as other criminological perspectives (e.g., social control theory and attachment to conventional others, social learning theory and differential exposure to definitions for versus against delinquent behavior, and general strain theory with peer rejection as a noxious experience). However, what really seemed to matter here were *early* social problems. Rather than focusing on social problems in adolescence or adolescent peer groups, then, prevention/intervention efforts need to direct their attention developmentally upstream. “First impressions” can have a lasting impact in the social circles of school children, and peer rejection can exacerbate behavior problems above and beyond the effect of initial behavior problem symptomatology (albeit not contemporaneous symptomatology). Programs that redress deficits in social skills might help – especially if they are implemented in the first few years of school, or earlier.

It is also noteworthy that lower self-esteem at ages 6-8 also predicted more adolescent aggression (even controlling for contemporaneous aggression). The same was

not true for delinquent behavior. Self-esteem therefore seems to play a unique role in the development of the aggressive behavior repertoire. Along with social skills training, early prevention/intervention programs can capitalize on the importance of self-esteem by fostering healthy, positive self-perceptions as well as healthy, positive social relationships.

Regression results helped clarify the relationship between contextual variables and antisocial behavior. In particular, controlling for contemporaneous aggression at Wave 3 negated the effects of the punishment variables (physical discipline, negative control) on later aggression. This suggests that level and style of punishment may in fact have been in response to the severity of aggression exhibited by children – that is, the evocative explanation. However, other family environment variables remained significant: family cohesion and family expressiveness. Therefore, even taking into account concurrent levels of aggressive symptomatology, less cohesion and more expressiveness at 9-11 predicted more aggression at 12-14 years of age. Lack of cohesion and more expressiveness may be important risk factors that either maintain the developmental trajectory for those with behavioral difficulties or “turn the tide” for vulnerable others.

The fact that greater family expressiveness predicted more aggression during early adolescence seems counterintuitive. Expressiveness was originally conceptualized to reflect openness to discussion as characteristic of a warm home environment. However, items such as “ We tell each other about our personal problems”, “Money and paying bills is openly talked about in our family,” and “There are a lot of spontaneous discussion in our family” may not uniquely capture the positive side of expressiveness.

In disadvantaged homes, homes with parental psychopathology, or homes with difficult children, expressiveness may actually contribute to more conflict and tension. In other words, perhaps the valence for expressiveness is conditioned by the larger social context.

Finally, there is a very striking difference between aggression and delinquent behavior in the models controlling for *contemporaneous* behavior problems. Although some contextual and child characteristics remained important in the prediction of aggression, only earlier delinquent behavior predicted later delinquent behavior. The implication here is that aggression is partially mediated by middle childhood risk/promotive factors whereas delinquent behavior is driven by its own core diathesis. The results of the analyses linking onset of first alcohol use to delinquent behavior but not aggression led to the suggestion that delinquent behavior might be more “deviant.” Whether or not it is in fact more serious, delinquent behavior does seem to follow the hard-continuity paradigm more conservatively (i.e., have at its center a harder “core”).

Of relevance here may be the distinction Moffitt (1993) makes between cumulative consequences and contemporary consequences. Cumulative consequences are those that snowball from early individual differences: Aggressive or delinquent children may experience attenuated parent-child interactions, social rejection, and academic difficulties that exacerbate their initial predispositions – leading to a progressive deterioration of personal and social assets or more deep epigenetic ruts. Contemporary consequences are those that result from ongoing or current behavior problems. Cumulative consequences may be more important for aggression, with interplay in the personal and social surround determining later outcomes; contemporary consequences may be more important for delinquent behavior, with this underlying

constellation of traits serving as the dominant driving force in its own behavioral trajectory.

One implication of this finding is that adolescent aggression may be more amenable to prevention/intervention programs that address not only the use of aggression itself but also personal and contextual factors that have a unique effect on later aggression (above and beyond initial and contemporaneous symptomatology). On the other hand, prevention/intervention programs for delinquent behavior would be best served by focusing on the “hard-cored” nature of delinquent behavior itself.

Limitations and Future Directions

The current study has several limitations. These relate to 1) the nature of the sample, 2) the operationalization of key constructs, 3) use of maternal report, and 4) factors not included in analyses.

The sample used for this study was racially and geographically homogeneous. Participants were mostly Caucasian and from the mid-Michigan area. It would be desirable to replicate this research in other geographic locations and with a more diverse population. In particular, there may be racial, ethnic, or cultural differences in the factors that contribute to the maintenance of behavior problem trajectories or that contribute to “turning points” in development. This was also a high-risk sample, heavily characterized by paternal psychopathology. It would be useful to replicate these findings in a general-population sample, such as the National Longitudinal Survey of Youth (NLSY). In addition, families were intact at recruitment. Although some decoupling occurred after the first wave, and step-parents were added to the assessment protocol, there may be important differences between these families and families with different

structures (e.g., single-parent, earlier-divorced, non-biological, etc.)

Another sample-related limitation involves the exclusive focus of this study on male children. Do the same risk factors buttress the developmental process for girls and boys? There were insufficient data on daughters to allow for inclusion in the sample or to look at gender differences because of the importance of Wave 1 and Wave 4 variables to the theoretical models tested. However, daughters from the Michigan Longitudinal study were started at later assessments; some were young enough to be assessed at 3-5 years old and will eventually reach their own Wave 4 (12-14). This will enable the types of analyses that were untenable for the current study. Alternatively, other samples could be used to investigate sex differences in the development of antisocial behavior as well as its relationship to first alcohol use.

A second area of limitation involves the operationalization of key variables. One issue is with the way that high and low levels of aggression or delinquent behavior were defined. Because of the relatively small sample size, mean splits were used to define “high” and “low” levels of symptomatology. “High” and “low” therefore were not extreme classifications. Different results might have obtained if more strict definitions of “high” and “low” were used. For example, it would be useful to examine those who reach clinical thresholds for aggression or delinquent behavior or who are at least one standard deviation above the mean. Future research needs to look at the difference between sub-clinical ratings and more conservative classification schemes. In addition, it would be useful to replicate these findings using a more pure delinquency scale, not constructed originally to include substance use (as in the CBCL) and focusing on delinquent acts more than a syndrome or constellation of behaviors (e.g., having older

friends) that “hang together” with delinquency.

Another operationalization issue concerns the use of the DOTS to measure activity level. In the DOTS, items used to assess activity level are sleep-oriented. Activity during sleep may not be the best indicator of general activity level (i.e., hyperactivity, which been implicated in the etiology of antisocial behavior; Moffitt, 1993). This may explain the lack of significance of activity level as a key initial risk trajectory marker in the current study. The DOTS-R includes both activity-general and activity-sleep, and it is when the DOTS-R is employed that marked group differences in activity-general were observed. Unfortunately, the DOTS-R was added to the assessment protocol subsequent to the first few waves (i.e., in the middle of Wave 2) and complete DOTS-R data weren’t available until Wave 3.

The third major limitation of the current study involves the use of maternal reports for describing child and family characteristics. Youth self-reports were generally not available until Wave 4. Maternal reports were chosen over paternal reports because this is common in the child development literature and because of a priori knowledge about the prevalence of psychopathology among fathers in the Michigan Longitudinal Study (e.g., paternal alcoholism was a criterion for recruitment in the majority of families, but maternal alcoholism was neither a requirement for study inclusion nor a basis for exclusion). In addition, there was decoupling in families after the first wave of data collection. Mothers were the primary caretakers in these decoupled families, and therefore could provide more accurate information about the family environment to which children were exposed during middle childhood. Also using data from the Michigan Longitudinal Study, Mun (2002) found that maternal ratings of temperament

and behavior problems were more predictive of smoking onset in children than paternal ratings. The use of averaged maternal and paternal reports was considered but deemed inadvisable for several reasons. Specifically, there was no guarantee that mothers and fathers would have the same relationships with their children or perceive their children similarly; averaging their ratings might therefore create more “white noise” than meaningful amalgams.

There were indeed differences between maternal and paternal ratings in this study. With $n = 220$ at Wave 1 and $n = 186$ at Wave 4, a retrospective analysis revealed that mother-father correlations were moderately strong in early adolescence but weaker when their children were of preschool age. For aggression, $r = .36$ at Wave 1 and $r = .52$ at Wave 4. For delinquent behavior, $r = .27$ at Wave 1 and $r = .40$ at Wave 4.¹³ At most, here, maternal and paternal CBCL scores shared 27% of their variance (i.e., $r^2 = .27$ for Wave 4 aggression). Note that sample size varies because these data were pre-imputation (given that paternal data were not included in missing data estimation).

Future research on the difference between maternal and paternal reports would be interesting and may help to illuminate any effect of rater on the results obtained. More ideal would be a study utilizing child self-reports. It may be that the child’s own perceptions drive his own behavior more – and would be more accurate for this reason – than the feelings his parents have about family environment, discipline, or the social problems experienced by their sons.

¹³ Similar results were obtained when the correlations were run for all male target children in the Michigan Longitudinal Study: for aggression, $r = .39$ at Wave 1 ($n = 321$) and $r = .54$ at Wave 4 ($n = 209$); for delinquent behavior, $r = .25$ at Wave 1 ($n = 321$) and $r = .46$ at Wave 4 ($n = 209$).

Factors not included in the current study (e.g., parental monitoring, peer substance use, peer delinquency, attachment to school and family, neighborhood violence, poverty) represent a fourth limitation. Parental monitoring and delinquent peers have been implicated in a number of criminological models (e.g., Patterson et al., 1989) but are not directly assessed with this sample until Wave 4. The Michigan Longitudinal Study loads heavily on the individual and parent-child causal aspects of risk accumulation, but broader structural and environmental influences (e.g., violent neighborhoods, poverty) might also help to explain the development of antisocial behavior. Neighborhood violence was not assessed, and socioeconomic status was not included in the current analyses. These variables likely exert their effect through more proximate processes (e.g., parental psychopathology, parent-child relationships, family conflict, use of harsh discipline, self-esteem, school achievement), but their inclusion in other research endeavors would allow for further delineation of the underlying causal matrix. Finally, attachments to school and family were not measured to the extent required for tests of social control theory (Hirschi, 1969; Sampson & Laub, 1993). These are likely to be important etiological influences in the development of aggression and delinquent behaviors, and future research is needed to assess their predictive utility within the larger framework adopted by the present study. Too often these variables are studied with a cross-sectional research design or without consideration of the continuity versus change perspective adopted here and in recent work within developmental criminology.

Despite these limitations, the findings from the current study are robust in their consistency. Results point to several potentially viable foci for intervention. For aggression especially, these include personal and contextual factors such as self-esteem,

social problems, and family functioning. The aggression and delinquent behaviors themselves should also be addressed, given that their substrates are apparent quite early and buttress an emergent, self-perpetuating developmental trajectory. A logical question might now be: where should we go from here?

Ideally, the utility of different intervention strategies would be assessed in a randomized clinical trial. However, in contrast to studies that compare an intervention group to a follow-up only group, more complex research designs are recommended. For example, one might randomly assign preschoolers to one of several conditions: intervention addressing problem behaviors directly – perhaps through cognitive restructuring, behavior modification techniques, or training in self-regulation/self-control; intervention addressing family functioning (e.g., the quality of parent-child relations, discipline); intervention addressing personal competencies (e.g., self-esteem, social skills); interventions combining two or three of the above; and no intervention/follow-up only. This methodology would help researchers disentangle the many causal factors now under consideration, and help to identify those most efficacious to problem behavior intervention/prevention.

The appeal of randomized trials in the developmental sciences is evidenced by recent efforts to adopt this research design – including the Early Head Start National Research and Evaluation Project (Love et al., 2002), among others (see Brooks-Gunn, 2003, for some examples). It is interesting to mentally map out how this model might be used to test the implications of the current study, and a randomized trial would definitely be the most scientifically rigorous option. More important than whether future research adopts a randomized or quasi-experimental design, however, is that movement continue

toward a better understanding of prevention and intervention factors. This call to action can be summarized as follows: “It is to be hoped that the next generation of studies will get further inside the ‘black box’ of program design to tell us what features of these programs are key to successful early intervention” (Currie, 2003, p. 5). Another progressive step would be the creation of a national registry of prevention trials for the behavioral sciences, to improve knowledge dissemination and facilitate meta-analyses (Biglan, Mrazek, Carnine, & Flay, 2003).

In Conclusion

Early onset of drinking is more strongly tied to the developmental course of delinquent behavior than aggression. It appears to be part of a longitudinal pattern of delinquent behavior problems traceable to preschool – that is, as young as 3 years of age. Preschool levels of delinquent behavior had a direct effect on later levels of symptomatology for early drinkers only, supporting the hard-continuity model and the role of initial predispositions on later outcomes for a small subset of individuals.

One implication of this finding is that intervention and prevention strategies geared towards early alcohol use should focus on this longitudinal pattern of problem behavior. Programs that focus only on trying to delay first alcohol use will probably fail to prevent its sequelae (e.g., alcohol problems) because it is likely that both are part of a larger developmental trajectory. Unless this larger developmental trajectory is addressed, intervention and prevention efforts will be mere ‘band-aids’ rather than “cures”. On the other hand, a true disruption of the developmental trajectory may dismantle the structure that supports early alcohol use.

A second consistent finding is in the importance of proximate versus distal risk

factors in the continuity of aggression and delinquent behavior from preschool to adolescence. The initial risk trajectory or core diathesis is modified or maintained by current influences in the personal and social surround of young children. For example, temperament, social problems, and the family environment during middle childhood are better predictors of an adolescent's aggression or delinquent behavior than temperament during preschool. This points to two key conclusions. First, risk is not immutable or set in stone during infancy. What matters are ongoing person-environment interactions and the developmental process. Risk may accumulate to become more deeply rutted, or change and provide the opportunity for a behavioral "turning point." Second, prevention/intervention programs that include middle childhood risk/promotive factors can have an effect on later outcomes; they may offer "windows of opportunity" for change.

The hard-continuity model applied more to delinquent behavior than aggression. The influence of preschool on adolescent levels of delinquent behavior was especially pronounced among early drinkers. In addition, delinquent behavior during adolescence was driven more exclusively by prior delinquent behavior than the parallel etiology for aggression. Stated conversely, middle childhood risk/promotive factors have a stronger influence on aggression (controlling for initial and contemporaneous levels of aggression) than they do on delinquent behavior. Prevention efforts for delinquent behavior therefore need to more directly attack the delinquent behavior trajectory, whereas prevention efforts for aggression may benefit from efforts to control aggression as well as efforts to ameliorate other personal and contextual influences. Delinquent behavior (e.g., lying, stealing, setting fires) seems to be more 'hard-cored' than aggression (e.g., bullying,

fighting, showing off).

Finally, the current study reaches the same conclusion as Stanger et al. (1997, p. 55): “These findings suggest that failure to distinguish between these patterns of problems in developmental research could generate misleading conclusions and limit the generalizability of results.” Aggression and delinquent behavior are often aggregated together under the overarching label of “externalizing” or “antisocial” problems, but major developmental differences were observed here. Delinquent behavior and aggression followed different age-related trajectories, were differentially associated with other problem behaviors (i.e., early drinking), and were differentially predicted by individual, interpersonal, and familial variables in this study. Ultimately, there may be very important reasons to look at these behaviors as often co-morbid but distinct – conceptually and developmentally.

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