

ABSTRACT

Title of Document: GENERAL STRAIN THEORY AND STABILITY IN OFFENDING AND SUBSTANCE USE OVER TIME: A DYNAMIC APPROACH

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One of the hallmarks of a good theory is that it can explain the known facts. Therefore, it is surprising that little research has examined whether General Strain Theory (Agnew 1992, 2006) can account for the relative continuity in antisocial behavior that individuals generally display over their time. The current study fills this void in the criminological literature by testing the ability of General Strain Theory (GST), in combination with the broader stress literature, to account for stability in offending and substance use from adolescence to adulthood. Four mechanisms that Agnew (1997, 2006) argues lead to behavioral continuity—a direct effect, evocative and active selection, passive selection, and stressor and deviance amplification—are examined using structural equation modeling. Drawing from the broader stress literature and the life-course perspective, two additional pathways—stress proliferation and the moderating effect of past exposure to stressors—are tested. This research is conducted using two unique datasets, the Collaborative Perinatal Project and the Pathways to Adulthood Study, which together provide information on the lives of 1,758 high risk individuals from birth through adulthood. Support for GST explanations of behavioral continuity is mixed, with more support for the dynamic mechanisms that do not rely on negative emotionality and low

constraint. Specifically, for both offending and substance use, there is no evidence to suggest that evocative and active selection or passive selection contribute to the stability of criminal behavior, however, stress proliferation and stressor and deviance amplification each explain a small portion of the association between adolescent and adult illegal behavior. In addition, the findings indicate that negative emotionality and low constraint condition the effect of stressors on criminal behavior, as does exposure to stressors in childhood. The findings for offending and substance use diverge only with regard to the direct effects of negative emotionality and low constraint: the direct effect of these variables on criminal behavior accounts for continuity in substance use, but not offending. It is argued that GST's emphasis on individual differences may be misplaced and that more attention should be directed to exploring the social processes through which stressors develop over time.

GENERAL STRAIN THEORY AND STABILITY IN OFFENDING AND
SUBSTANCE USE OVER TIME: A DYNAMIC APPROACH

By

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Dissertation submitted to the Faculty of the Graduate School of the
University of Maryland, College Park, in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
2007

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DEDICATION

For my family, old and new.

ACKNOWLEDGEMENTS

As I worked on this research, I couldn't help but wonder if I was destined for a life of crime, given the amount of subjective strain I experienced during the dissertation process. Thankfully, I am fortunate to have access to an extensive set of legitimate coping mechanisms—my family, friends, and colleagues—who made my life manageable.

First and foremost, I extend my gratitude to my dissertation committee. Professor Sally Simpson, my chair, advisor, “mentoreess”, boss, and friend, guided me through the dissertation process, providing theoretical direction. She exhibited great patience during the period of time when my dissertation topic changed on a daily basis. Most importantly, Sally's faith and confidence in my ability, and my desire to make her proud, always has and probably always will inspire me to do my best work.

Other members of my committee also must be thanked for their contribution to this project. Brian Johnson acted as a sounding board for my dissertation ideas and, through my outside work with him, taught me how to do careful research. Hanno Petras met with me regularly to assist me with Mplus and structural equation modeling. I am grateful for the amount of time he spent with me. Sandra Hofferth provided unique insights into the project at both the proposal and final defense. Finally, John Laub provided careful and critical feedback on the dissertation and, through our adjoining wall, much needed emotional support during the writing process.

Additional scholarly advice came from my fellow graduate students, Sue-Ming Yang and Nancy Morris, especially with regards to structural equation modeling. Equally if not more important, was the support and encouragement provided by these two

individuals. Sue-Ming was always down the hall encouraging me to keep going, while I could count on Nancy to provide me with my daily pep talk via phone. Phone encouragement was also provided by Jill Farrell and Kari Cohen, both of whom provided me with a connection to the outside world and vicarious living. Kari also had the added task of talking me off the ledge when things got rough. Erica Luetzow provided much needed inspiration—if she could give birth to her baby in an efficient and timely manner, I could do the same with my “baby”. Also, thanks to her I realized that there are many things worse than writing a dissertation! In addition Erin Bauer provided lots of nutritional sustenance and Cody Telep plied me with Jolt Cola during my all night work sessions.

Special thanks must also be given to my entire family, especially my mom and sister. Both forgave me my absences from home and dealt with panicky phone calls. My mom has always been a role model for me, and the lessons she has tried to teach me over the years—the importance of hard work and independence, the knowledge that all bad things will pass, and the necessity of letting some things go—served me well during the dissertation process. My sister Erica provided a voice of sanity and lots of laughs. And of course how could I forget “the baby”, Suka, who could be counted on to give me a warm welcome no matter what time I came home from school.

Finally, my deepest gratitude and love goes to my better half, Eric. Not only did he do all the cleaning, cooking, laundry, food shopping, dog walking, dog feeding (most of the time), and gardening over the past few months, but he sold our house. Even more amazing, he put up with my long hours and, when I was home, with me! He is one of the

most kind-hearted people I know. Without his support and love I am certain I would still be working on this dissertation.

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CHAPTER I: INTRODUCTION

In criminology, where even the “facts” are sometimes debatable, it is rare for researchers to reach a consensus; however, with regard to recognizing the stability of antisocial behavior over time, this appears to be the case (Sampson and Laub 1997). The stability of antisocial behavior is imperfect, with many delinquents growing up to be non-offenders, yet childhood antisocial behavior is generally considered a prerequisite for adult offending (Robins 1978). This has been demonstrated in numerous data sets, with samples drawn from various time periods, countries, and cultures (for a review see Loeber 1982 or Sampson and Laub 1993, 1997). After reviewing the literature, Sampson and Laub (1997, 135) conclude that “Taken as a whole, the different studies across time, space, and methods yield an impressive generalization that is rare in the social sciences.”

There is much less agreement regarding the mechanisms that produce behavioral continuity. Although numerous explanations have been put forth, they can be grouped into three general categories. The first explanation, population heterogeneity, attributes continuity in antisocial behavior to the relative stability over time of criminal propensity, making the association between past and future offending spurious (e.g. Gottfredson and Hirschi 1990; Wilson and Herrnstein 1985). In contrast, state dependence, the second explanation, causally links past to future offending. In this model, childhood antisocial behavior has a genuine effect on future offending by weakening constraints and strengthening incentives for crime. For example, Sampson and Laub (1993, 1997) argue that being labeled a delinquent can set in motion a process of cumulative disadvantage in which individuals’ social bonds are systematically weakened and their opportunities to live a conventional life are diminished. Finally, mixed models (Laub and Sampson 2003;

Ousey and Wilcox 2007) combine population heterogeneity and state-dependent explanations, allowing for both individual differences and contexts to contribute to stability in offending. Examples of this type of explanation include Moffitt's (1993) typology of adolescent-limited and life-course-persistent offenders and Sampson and Laub's (1993) age-graded theory of informal social control. Agnew's General Strain Theory (1992, 1997, 2006) also provides a way to combine state dependence and population heterogeneity to explain continuity in illicit behavior; however this theory has received considerably less attention.

General Strain Theory (GST) (Agnew 1992, 2001, 2006) conceptualizes strain as a psycho-social concept that can explain individual-level offending. The crux of this theory is that strain—caused by exposure to noxious stimuli, the removal of positive stimuli, or blocked goals—leads people to experience negative emotions. In an effort to relieve these negative emotions, individuals may use illegitimate coping mechanisms, such as delinquency, offending, or drug or alcohol use.

In its original version, GST was formulated to explain between-individual variations in offending (Agnew 1992). More recently, it has been extended to explain within-individual patterns of behavior, including stability in offending (Agnew 1997, 2006). Drawing on state dependant and population heterogeneity explanations of continuity, Agnew (1997, 2006) posits that individuals who persist in offending do so for one of two reasons: Either they are high in negative emotionality and low in constraint, which makes them more likely to encounter strains throughout their life and to cope with these strains in a criminal manner, or they have a low socio-economic status and this sets in motion an amplifying loop in which living in a disadvantaged environment results in

greater exposure to criminogenic stressors. This, in turn, increases the likelihood of delinquency and offending, which at best sustains aversive life circumstances and at worst exacerbates them, thus beginning the cycle over again.

Empirical research suggests GST may provide a viable explanation for stability in offending over time (Hoffmann and Cerbone 1999; Kim et al. 2003), yet few studies have tested its utility and those that have focused exclusively on adolescent samples and short time spans. Moreover, none of this work has explicitly tested the stability-promoting pathways outlined by GST or even considered the role of individual differences, a key factor in Agnew's explanation of stability. In fact, many of these studies use statistical models to "sweep out" the effects of unobserved heterogeneity in the propensity to offend (e.g. Hoffmann and Cerbone 1999; Kim et al. 2003; Slocum, Simpson, and Smith 2005).

One framework within which to study stability is the life-course perspective, which is concerned with "pathways through the age-differentiated life span" (Elder 1985, 17). This paradigm emphasizes social and historical context, timing, linked lives, and human agency. More specifically, it is centered around four major principles, which guide research in terms of problem selection and theoretical development: 1) individuals' lives are shaped by the historical times and places in which they develop; 2) the effects of events and transitions depend on their timing; 3) lives are interconnected, with social and historical influences being expressed through this network of shared experiences; and 4) individuals shape their paths through life via their choices and actions (Elder 1998).

Despite the relative prominence of studies using the life-course perspective and the increasing interest in GST, criminologists have paid scant attention to strain explanations of offending over the life course. This theoretical and empirical dearth is

surprising for two reasons. First, in recent years sociologists have experienced relative success in combining the life-course perspective with the study of stressors to explain within- and between-individual differences in a wide variety of outcomes, including mental and physical health (e.g. Ensel and Lin 2000; Lantz et al. 2005; Menaghan 1997; Umberson et al. 2005). Second, and more importantly, sociologists, like Pearlin and Skaff (1996, 246), have argued that there is a synergism between the study of social stress and the life-course perspective and “that each can put meat on the bones of the other,” while Agnew asserts that “new strain theories...should be part of any developmental theory of crime” (1997, 102).

As suggested by Pearlin and Skaff (1996), the life-course paradigm can do much to advance the study of strain. First, it provides strain researchers with a new set of problems and puzzles and encourages them to question what sets trajectories in motion, how are they maintained over time, and what changes their direction.

Second the life-course paradigm requires criminologists to place the study of stress within its historical and social context and forces researchers to reconnect the study of strain to underlying social conditions. In criminology, a great deal of research has examined the relationship between strain, emotions, and offending and the factors that condition these relationships (e.g. social support, peer delinquency). However, insufficient attention has been paid to determining the origins of stressors. Often times in stress research a standardized checklist of negative life events is used to measure exposure to stressors. Although this instrument provides a standardized methodology, it also gives the false impression that negative life events just happen to people. The few studies that have attempted to explain the uneven distribution of stressors across the

population have been limited in scope, focusing primarily on socio-economic status (De Coster and Heimer 2001; Hagan and Foster 2003). While socioeconomic status certainly influences exposure to stressors, the life-course paradigm and the principle of linked lives points to the possibility that parents also play a role through their behavior and actions.

Finally, the life-course perspective necessitates that researchers study the strain-crime relationship within the context of individuals' personal histories (Wheaton 1990). As such, it sensitizes researchers to the possibility that past experiences with stressors and offending may affect the types and level of stressors individuals experience as well as their responses to these stressors. Therefore past experience is not merely something to be statistically controlled for, but rather analytically and theoretically developed. Similarly, the life-course paradigm makes obvious "the need to study particular stressors in the context of accumulating prior stress exposure" (Wheaton and Gotlib 1997, 15), with stressors arrayed in a causal sequence. In this manner, experiencing stressors at a young age has the ability to set individuals on a trajectory that may be difficult to deflect.

Very little research has examined GST within a life-course perspective or looked at strain as a potential source of stability in offending and substance use, although somewhat more work has examined change. Given the natural alliance between the study of stressors and the life-course paradigm, why haven't criminologists explored the relationship between strain and continuity in illegal behavior over the life span? This oversight may be due to the fact that GST is a relatively new incarnation of strain theory and, as a result, researchers have been primarily concerned with developing and testing its core tenets. It may also result from a lack of adequate data. There are relatively few datasets that provide information on individual differences, stressors, and offending and

substance use over an extended period of time. Finally, criminologists may have shied away from the study of illegal behavior and stressors over the life span because of the lack of clearly articulated and easily testable propositions about the contribution of stressors to continuity and change in behavior. Although Agnew (1997, 2006) has several articles in which he begins to outline this process, criminologists have not given serious theoretical consideration to the relationship between stressors and illegal behavior over time. One goal of the present study is to explore how GST in particular, and stress theories in general, can explain continuity in offending and drug use from adolescence to adulthood.

THE PRESENT STUDY

In this research, I use Agnew's GST explanations of stability in conjunction with the stress literature to explore the mechanisms through which exposure to stressors can promote stability in offending and substance use across the life course. This is accomplished using structural equation modeling (SEM), which provides several advantages over traditional regression methods. This study is not intended to be exhaustive, but instead to explore the potential contribution that GST and the stress literature can make to understanding continuity in offending and drug use over time. As will become apparent in the next chapter, GST predicts multiple pathways leading to continuity and these are built on many contingencies. Therefore, this dissertation is a modest attempt to understand these mechanisms. However, given the lack of research and theoretical elaboration concerning strain and continuity in illegal behavior, the proposed study is a warranted and necessary first step.

The data used to examine stability come from two separate studies following the same group of high-risk children born in the 1960s. In the Collaborative Perinatal Project (CPP), researchers collected prospective data from birth to age 8 on a sample of children born at Johns Hopkins Hospital. These data included information on each child's family history, demographic background, and interactions with his or her mother. In addition the child's biological, neurological, neurosensory, and cognitive development, and temperament were carefully monitored.

The second study, Pathways to Adulthood (PTA), was conducted between 1992 and 1994 when the CPP children were between the ages of 27 and 33. With the assistance of a life history calendar, respondents were asked to provide extensive retrospective information on the 20 years that had elapsed since the end of the CPP. The interviews included questions on education, employment, family composition, health, income, partnerships, and neighborhood characteristics. Respondents were also asked to report on their aspirations, involvement in delinquency, substance use, reproductive history, and social relationships. Supplemental data reported by each respondent's mother is also available in both the CPP and the PTA.

The current work extends previous research in several ways. First, if GST is to remain a viable theoretical explanation of illegal behavior, it must demonstrate that it can explain continuity and change in offending and substance use over time. Although there have been some recent attempts to use this theory to explain patterns of illegal behavior over the life course, these studies have focused on explaining change, especially the normative changes in delinquency associated with adolescence, not stability (e.g. Agnew 2003; Hoffmann and Cerbone 1999; Kim et al. 2003; Slocum, Simpson, and Smith 2005).

Furthermore, in recent years, there has been an increasing focus on change in offending, or desistance. According to Menaghan (1997), “It is precisely because continuing in the same direction in which one is already headed is so expected that, in fact, we find exceptions interesting. We are fascinated as social scientists and fellow travelers from cradle to grave, when people appear to dramatically shift course, get “derailed” or “jump off the track,” reinvent themselves and their daily lives, and reshape their future prospects. In comparison, we tend to be less interested in that which makes these exceptions remarkable—the strong persistence over time, for most people, of their current circumstances” (114).

Second, in the current study the stress process is viewed over an extensive span of the life course, from birth through adulthood. Past research has tended to focus on short time periods (e.g., Slocum, Simpson, and Smith 2005), particularly those in adolescence (e.g., Hoffmann and Cerbone 1999; Paternoster and Mazerolle 1994). While it is true that most crime is committed by the young, the focus on adolescence severely limits the generalizeability of GST and ignores a population of great interest to criminologists—individuals who continue to violate the law into adulthood.

Third, structural equation modeling (SEM) is used to model the relationships between temperament, stressors, and offending and substance use across three stages of the life course—childhood (birth to age 8), late childhood through adolescence (ages 9 through 17), and adulthood (ages 18 through 32). Tests of GST typically have used ordinary least squares (OLS) regression or hierarchical modeling to examine the relationship between strain and criminal behavior over time (for an exception see Hoffmann and Miller 1998 and Hoffmann and Su 1997). SEM provides an advantage

over these more traditional techniques because it enables researchers to explicitly model measurement error, which results in more accurate estimates of the relationship between strain and offending. In addition, rather than merely controlling for the effects of key variables, like parenting, past delinquency, and social structure, SEM provides a way to model the process through which these factors influence one another by allowing one to simultaneously model the multiple pathways through which these variables influence future stressors and illegal behavior.

The final way in which the current work extends previous research is by using GST and a mixed model approach to study stability in offending and substance use. The role of difficult temperament, characterized by negative emotionality and low constraint, in producing behavioral continuity is explored. Negative emotionality and low constraint are hypothesized to have direct and indirect effects on illegal behavior, the latter operating through exposure to stressors. However, following a dynamic approach the ramifications of problem behavior for future exposure to stressors are also considered. This firmly places the study of crime and stressors within social and structural context, while simultaneously considering the role of individual differences.

Chapter II, briefly reviews the stability literature and GST. Next this chapter describes each of the four mechanisms that Agnew (1997, 2006) posits promote continuity in illegal behavior across the life span and reviews the relevant theoretical and empirical literature. Drawing on the stress literature, I also discuss possible ways that these pathways to stability can be elaborated. Finally, the implications of these mechanisms for the development of strain and problem behavior over time are discussed and the specific hypotheses that will be testing are outlined. Chapter III describes the

data collection process, sample, and key variables of interest as well as the analytic strategy, while Chapter IV presents the results of the structural equation models. Finally, in Chapter V the findings are summarized and synthesized, limitations of the study are detailed, and implications of the research for theory and practice are discussed.

CHAPTER II: THEORETICAL AND EMPIRICAL BACKGROUND

STABILITY

There is a considerable amount of stability in offending and substance use over time and across life domains (see Caspi and Moffitt 1992; Loeber 1982; Sampson and Laub 1993).¹ In fact, most criminologists are familiar with the phrase “The best predictor of future behavior is past behavior.” Despite the consensus regarding behavioral continuity, the jury is still out on the mechanism through which this stability is produced. Population heterogeneity explanations, like the General Theory of Crime (Gottfredson and Hirschi 1990), attribute continuity to time stable differences in the propensity to offend. Therefore, in these static explanations, the relationship between past and future offending is spurious. In contrast, state dependence arguments take a dynamic approach and posit that past problem behavior has a causal effect on future behavior through the negative ramifications of the behavior itself (see Laub and Sampson 2003).

The most fruitful explanations of continuity, however, appear to be ones that discard the false dichotomy between person and context. Because the life-course paradigm attributes behavior to both individual differences and social context,, it provides an alternative to these oversimplified explanations. Yet, the life-course perspective cannot explain behavior in and of itself. Rather, it provides a framework that

¹ There are multiple types of continuity (Caspi and Bem 1990), but this work primarily deals with homotypic continuity. Homotypic continuity occurs when the same behavior or phenotype is exhibited over time. For example, a person who engages in violence both in adolescence and adulthood exhibits homotypic continuity. Because the study spans approximately 30 years of the life course, it is necessary to also consider heterotypic continuity, characterized by the same latent trait manifesting itself in different ways. A person who engages in violence as an adolescent and then abuses alcohol as an adult displays heterotypic continuity, assuming that aggression and substance use are different manifestations of the same latent traits.

can be incorporated into many different theories including General Strain Theory (Agnew 1992).

GENERAL STRAIN THEORY

In 1992, Robert Agnew took traditional strain theory in a new direction with his General Strain Theory (GST), in which he conceptualized strain as a psychosocial characteristic that can be used to explain individual-level offending. In GST, strain is defined as negative relations with others (Agnew 1992) or “events or conditions that are disliked by individuals” (Agnew 2006, 4). It may be caused by exposure to noxious stimuli, the removal of positive stimuli, or blocked goals. Not all sources of strain are equally likely to result in illegal behavior. Agnew (2001) suggests that strains are most likely to lead to crime when they have the following characteristics: (1) they are seen as unjust; (2) they are viewed as high in magnitude; (3) they are associated with low social control; and (4) they create pressure or incentive to engage in deviant coping. Examples include failure to achieve goals that are easily achieved through criminal behavior and that are not the product of conventional socialization (e.g., masculine status, wealth, excitement, etc.); discrimination; criminal victimization; work in the secondary labor market; abusive peer relations; homelessness; negative secondary school experiences; parental rejection; poor parenting; and child abuse and neglect.

The intervening mechanism between strain and delinquency is negative affect. Strain leads the individual to experience negative emotions including fear, disappointment, depression, and especially anger (Agnew 1992). These feelings can lead an individual to desire corrective action, which may take the form of crime for certain

individuals. Specifically, people may (1) engage in illegitimate behavior to achieve their goals, (2) attack or escape from the source of their stress, and/or (3) manage their negative feelings (Agnew 1992, 49). For example, drugs may be used to manage feelings, while other types of illegal behavior may be used to attack or escape sources of strain or to achieve positively valued goals.

Strain has been associated with both internalizing behaviors, like substance use, and externalizing behaviors, like violence and property crimes. The emotions that mediate the relationship between strain and outwardly directed crimes may differ from those that mediate the relationship between strain and inwardly directed deviance, with anger more closely associated with property and violent crimes and depression more closely associated with substance use (Agnew and White 1992; Jang and Johnson 2003). Depression creates pressure for corrective action by decreasing the perceived costs of crime because depressed individuals feel they have nothing left to lose. Also the feelings of lethargy, powerlessness, and listlessness associated with depression decrease individuals' ability to cope with strain in a legal manner. In contrast to anger, which tends to activate and mobilize people, depression is more likely to result in inwardly directed coping mechanisms, like drug use, as opposed to aggressive and overt types of offending (Agnew 1992; Jang and Johnson 2003). For this reason, research often examines substance use as a separate outcome from other types of illegal behavior (e.g., Agnew and White 1992).

Legitimate and illegitimate coping mechanisms are not equally distributed across the population. Instead, individuals have constraints that limit their ability to access non-delinquent and delinquent coping mechanisms (Agnew 1992). Constraints may be

internal, such as goals and values, or external, like a social support system. The choice of coping strategies is also affected by an individual's disposition to delinquency.

Disposition to crime is a function of several variables, including learning history and attributions for the cause of adversity (Agnew 1992, 73); however, recently, Agnew and colleagues (2002) have argued that personality may be the most important conditioning factor.

Empirical research generally supports the main propositions of this theory.

Strain, especially presentation of noxious stimuli and negative life events, has been linked to both specific and general measures of delinquency and substance use (e.g., Agnew and White 1992; Mazerolle and Maahs 2000; Paternoster and Mazerolle 1994). Some of the effect of strain on delinquency is mediated by factors associated with social control and social learning theories. For example, using data from the National Youth Survey, Paternoster and Mazerolle (1994) found that strain had a direct effect on delinquency as well as an indirect effect via weakening social controls and increasing association with delinquent peers.

Although less prevalent and less consistent, studies suggest negative emotions may at least partially mediate the relationship between strain and crime (Broidy 2001; Jang and Johnson 2003; Mazerolle and Piquero 1998; Mazerolle, Piquero, and Capowich 2003; Piquero and Sealock 2000). Generally, evidence suggests that there is an association between strain and anger and that anger increases the likelihood of illegitimate delinquency, especially violence (e.g., Jang and Johnson 2003; Mazerolle, Piquero, and Capowich 2003). Less well studied is the association between strain, depression, and illegitimate coping. Piquero and Sealock (2004) found that in a sample

of delinquents strain was associated with depression for males, but not for females. Moreover, there was no significant relationship between depression and property offending or aggression (see also Mazerolle and Piquero 1998). Broidy (2001) found that a global measure of non-anger related negative affect did result from strain, but that this measure decreased, rather than increased the likelihood of delinquent coping. However, there is some evidence supporting a link between depression and substance use, specifically. Jang and Johnson (2003) and Drapela (2006) both found an association between depression and drug use. Jang and Johnson found that depression fully mediated the link between strain and depression, while in Drapels's work depression had a direct effect on drug use but did not mediate the effect of strain. In sum, empirical research provides support for various aspects of GST; however, the relationship between strain, negative affect, and various forms of illegal behavior may be more complex than suggested by Agnew.

Although a fair amount of work has been done to test the main tenets of GST, much less research has examined the usefulness of this theory for explaining continuity in offending and substance use over time. The only study that explicitly looked at strain as a source of continuity in criminal behavior was supportive of GST. Using four years of data from the Family Health Study, Hoffmann and Cerbone (1999) used hierarchical growth curve modeling to relate the escalation of delinquency in adolescence to increases in the number of stressful life events experienced. They found that experiencing an increasing number of life events was related to an intra-individual growth in delinquent activity and that this relationship was not conditioned by gender, family income, self-esteem, or mastery. This study, however, did not consider the mechanisms by which

strain may promote stability in offending and drug use. The next section examines this question and reviews the limited empirical findings relevant to this issue.

For the sake of conceptual clarity, it is necessary at this point to draw on the broader stress literature to more carefully define the components of the stress process as there has been little consistency in the criminological literature. Criminologists, including Agnew, often use the terms strain, stressor, stress, and distress interchangeably. In Table 1.1 of *Pressured into Crime* (Agnew 2006, 5-7), Agnew lists the types of strain examined in the literature. Included in this list of *strains* are negative life events, life stresses, neighborhood problems, negative relationships with others, and daily hassles. Rather than using the nebulous term “strain”, which seems more applicable to classic macro-level strain theories than GST, I explicitly refer to “stressors” instead. Stressors have been defined by Wheaton (1996, 32) as “conditions of threat, demand, or structural constraint that, by the very fact of their occurrence or existence, call into question the operating integrity of the organism.” This term is more appropriate than strain because it captures the broad array of events, states, and occurrences that Agnew posits are causes of offending, yet does not confound them with their effects (i.e. stress or psychological distress). It is also more appropriate for the work at hand because the measures used here, like in many other tests of GST, consist of events and conditions, as opposed to the psychological distress or strain resulting from these events and conditions.

GENERAL STRAIN THEORY EXPLANATIONS OF STABILITY

GST posits four mechanisms contribute to stability in offending and substance use, three of which rely on time stable individual differences, directly or indirectly, to

explain continuity. The first explanation attributes stability in illegal behavior over time to persistent individual differences in negative emotionality and low constraint. In this direct effect explanation, any observed association between behavior at two points in time is spurious because the behaviors are related only through their common cause. This explanation of stability is inherently static in that continuity in offending and substance use is attributed to a latent trait that is invariant over time (Sampson and Laub 1997). The second explanation for stability—active and evocative selection processes—is also based on stable individual differences; however, in this case negative emotionality and low constraint operate via selection into aversive relationships and situations. Similarly, the third explanation Agnew proposes to explain continuity, passive selection (Scarr and McCartney 1983), is also static. In this account continuity in offending and substance use is not directly produced by a latent trait, but instead by stability in aversive family conditions. However, the aversive family context is directly the result of the persistent negative emotionality and low constraint of parents, who create this rearing environment for their children. The remaining pathway is the only dynamic explanation for continuity in offending and substance use. Here stability is attributed to state dependent mechanisms in which an individual's behavior and the effects of this behavior causally influence future behavior.

The next section describes the traits which Agnew gives a primary role in the maintenance of offending and substance use over the life course—negative emotionality and low constraint. Then Agnew's three static explanations for continuity are discussed and the empirical evidence supporting or refuting each of these explanations is detailed. In addition, I propose several ways that Agnew's explanations can be extended to provide

a more dynamic account of behavioral continuity. Then, the only purely state dependent explanation put forth by Agnew to explain stability in problem behavior is described. Finally, these explanations are synthesized to produce the conceptual framework that is the focus of my analysis and the hypotheses arising from this model are outlined.

GST AND INDIVIDUAL DIFFERENCES

Original conceptualizations of GST paid scant attention to the relevance of personality for explaining the strain-crime link, but in later work, Agnew and others (Agnew 1997, 2006; Agnew et al. 2002) argue that personality traits may be the primary and most pervasive factor moderating the relationship between strain and crime.² In his 1997 article on GST and development over the life course, Agnew lists poor problem solving skills, impulsivity, low frustration tolerance, and irritability as the characteristics most pertinent to GST. For lack of a better term, he calls the confluence of these traits “aggressiveness” (107). In more recent work, however, Agnew (Agnew 2006; Agnew et al. 2002) draws from Tellegen’s (1985) three factor model of personality and explicitly identifies negative emotionality and low constraint as the central factors responsible for stability in problem behavior.³ Furthermore, he states that these traits have a fundamental conditioning effect on the relationship between strain and offending.

² Temperament and personality are often used interchangeably and their distinction remains fuzzy (Roberts and Del Vecchio 2000), but there are some notable differences between the two. Temperament refers to “behavioral styles of approach and response to novel situations” (Caspi et al. 1995: 57). It is present at birth and is generally linked to neurobiological functioning. Moreover, it is a precursor to personality, which develops as a function of temperament and the environment (Caspi and Silva 1995). When reviewing the literature on negative emotionality and low constraint I will use the terminology utilized by the researchers whose work I am discussing. For the sake of simplicity, in this work I will refer to negative emotionality and low constraint as individual differences or traits.

³ The third domain is positive emotionality.

More generally, in the stress literature, no consensus has been reached regarding the relevance of traits to the stress process. Some scholars, including Folkman, Lazarus, and colleagues (e.g. Folkman and Lazarus 1980; Folkman et al. 1986) view a trait-based approach as too static and unidimensional. They argue that trait-based studies of stress assume that coping is a function of the person and fail to consider variations in stressful situations. Others, however, believe that trait oriented approaches to stress research have been misrepresented and an “enlightened trait conception,” which conceives of behavior as a joint function of traits and situations, avoids many of these problems (Ben-Porath and Tellegen 1990, 15).

In this enlightened approach, the behavioral manifestation of traits provides a matching of traits to situations via a process of active selection and contributes to cognitive restructuring in which the situation is perceived in a way that matches individual differences (Ben-Porath and Tellegen 1990). Traits like negative emotionality and low constraint may affect how a person initially appraises the threat posed by a situation and how that person assesses his or her ability to deal effectively with the threat (Ben-Porath and Tellegen 1990), leading to a disconnect between the actual threat associated with a situation and the individual’s perception of the threat. For these reasons Ben-Porath and Tellegen (1990) argue that a trait-blind or situation blind approach to studying stress is indefensible; both must be considered to fully understand the stress process. This view dovetails with the mixed-model approach to understanding continuity.

Agnew sometimes takes Ben-Porath and Tellegen’s advice to heart. He proposes two processes—passive selection and active/evocative selection—through which traits

and situations jointly promote continuity in offending and substance use over time, but even in these behavioral stability is ultimately a result of persistent population heterogeneity. Agnew's other explanations either take a situation blind (direct effect) or trait blind (stressor and delinquency amplification) approach.

Negative Emotionality and Low Constraint

Negative emotionality (NE) "assesses individuals' tendency to experience negative emotions (e.g. fear, depression, and anger) and their tendency to breakdown under stress" (Miller and Lynam 2001, 770). It encompasses three sub-scales: aggression, alienation, and stress reaction (Tellegen 1985). Individuals high in negative emotionality have a tendency "to experience events as aversive, to attribute these events to the malicious behavior of others, to experience intense emotional reactions to these events..., and to be disposed to respond to such events in an aggressive or antisocial manner" (Agnew et al. 2002, 46). Negative emotionality overlaps with the domains of neuroticism and agreeableness described in other models of personality, such as the Five-Factor Model and's (Eysenck and Eysenck 1969) three-factor model (see Miller and Lynam 2001; Ormel and Wohlfarth 1991). According to John and colleagues (1994, 173), negative emotionality "forms the core of adult neuroticism." Because much of the research on stressors and temperament has been done with adult samples, most of the research reviewed here refers to neuroticism instead of negative emotionality.

Like negative emotionality, low constraint (LC) is drawn from Tellegen's (1985) model of personality, where it is described in its opposite form, constraint, which is composed of three subscales: traditionalism, harm avoidance, and control. Low

constraint characterizes individuals who “are impulsive, are risk-taking/sensation-seeking, reject conventional social norms, and are unconcerned with the feelings or rights of others” (Agnew et al. 2002, 46). It corresponds to the domain of conscientiousness in the Five-Factor Model (Miller and Lynam 2001) and Block, Block, and Keyes’ (1988) concept of ego under control.

Combined, NE and LC contain many of the elements of Gottfredson and Hirschi’s (1990) concept of self control, however, Agnew and colleagues aver that these traits are important for reasons that are unique to GST (2002). Specifically NE/LC “increase the likelihood that that individuals will react to strain with strong negative emotions, will have trouble coping with such strain through legitimate channels, and will find crime an attractive option” (Agnew et al. 2002, 48). Furthermore, though acknowledging that the personality domains most often related to criminal behavior—agreeableness and conscientiousness—incorporate many of the characteristics of low self-control, Miller and Lynam (2001) argue that it is more useful to keep these domains of personality distinct rather than combining them into one concept called self-control. They reason that these dimensions emerge as separate factors in most of the major structural models of personality. This leaves the possibility that they do not operate in the etiology of delinquency as one monolithic force. Caspi et al. (1994: 187) also view self-control as an overly simplistic concept because most psychological research finds that crime-proneness has two distinct components—negative emotionality and low constraint. Negative emotionality and low constraint also differ from self control in their origins. Gottfredson and Hirschi (1990) explicitly state that low self control is the result of ineffective

parenting. In contrast, negative emotionality and low constraint are believed to have biological and environmental origins.

The Origins of Negative Emotionality and Low Constraint

Negative emotionality and low constraint result from biological processes⁴ and interactions with others and the environment (Caspi et al. 1994). Negative emotionality is believed to develop when family life is chaotic and parenting is harsh and/or inconsistent. The constant threat of physical or emotional harm means that for children in these families, ambiguous situations are more likely to be founded in harmful versus benign intent. Therefore, the perceptual bias that leads children who are high in negative emotionality to see harmful intent in others' behavior where other children see none, actually serves to protect them in childhood (Caspi et al. 1994). Low constraint may have similar origins, but given its overlap with self control it may also be related to authoritative and ineffective parenting.

Biologically, increased sensitivity to stressors has been associated with elevated levels of the stress hormones cortisol and adrenocorticotrophic hormone (Fishbein 2001). Impaired executive cognitive functions have also been traced to the inability to correctly interpret social cues during interpersonal interactions (Fishbein 2001) and poor problem solving skills. The neurotransmitters serotonin, dopamine, and norepinephrine and the enzyme monoamine oxidase (MAO) have all been associated with the inability to control impulses (Fishbein 2001). Moreover, the effect of norepinephrine appears to be exacerbated under certain environmental conditions, like exposure to stressors.

⁴ It is important not to equate biology with genetics. Biological processes, like the production of neurotransmitters and hormones, may have both genetic and environmental causes (see Rutter et al. 1997 and Rutter and Silberg 2002).

Individuals with damage to the prefrontal cortex of the brain are more likely to be impulsive, insensitive to the consequences of their behavior, and irritable (Fishbein 2001).

Of course the notion that individual differences shape transitions and trajectories through the life course is not new (e.g. Caspi, Elder, and Bem 1987). However, Agnew (1997, 2006) argues that there exist pathways leading to behavioral continuity that are unique to GST. He proposes three mechanisms through which negative emotionality and low constraint promote continuity of offending and substance use over the life course (1997, 2006). These traits have a direct effect on illegal behavior, increase exposure to objective stressors (active and evocative selection), or result in a stable aversive home environment (passive selection). I discuss each of these mechanisms below. In addition, consistent with the life-course perspective I draw from the stress literature to discuss how these mechanisms can be extended to include dynamic processes.⁵

PATH 1: DIRECT EFFECT

Agnew (2006) posits that NE and LC have a direct effect on antisocial behavior and that problem behavior persists because these traits are relatively stable over time. In and of itself, this explanation does little to elucidate how strain contributes to continuity in offending. Many studies have found a direct relationship between NE/LC and child behavior problems (e.g., Caspi et al. 1995) and Gottfredson and Hirschi (1990) argue that

⁵ As compared to the criminological literature, there is a much stronger tradition of studying the effects of stressors on mental and physical health. For this reason, much of the literature I rely on draws from research that uses outcomes aside from offending and substance use. Although health and crime are certainly not interchangeable, studies have found them to be related (e.g., Laub and Vaillant 2000; Piquero et al. 2007) and, therefore, insights gleaned from one of these outcomes might be applicable to the others. In addition, Agnew (1992) himself drew heavily from the mental health literature in his initial inception of GST. Finally, because negative emotions, including depression, are a key component of GST, the mental health literature is certainly relevant.

low self control, which shares many characteristics of NE and LC, has a direct effect on offending. Rather, the unique contribution of GST comes from the manner in which these traits influence offending—by enhancing the effect of stressors on offending and substance use via reactive person-environment transactions in which people exposed to the same stimulus perceive it, and therefore react to it, differently (Caspi and Bem 1990). Specifically, individuals high in NE and LC will be more likely than others to interpret a given situation as threatening or as a result of deliberate mistreatment (Agnew 2001; Agnew et al. 2002; Ben-Porath and Tellegen 1990).

In situations where the intent of the actor is ambiguous (that is, it is not apparent whether the actor has harmful intent), individuals with high levels of negative emotionality will be more likely to attribute harmful intent to the actor, more likely to respond aggressively, and more likely to believe that aggressive responses will be effective (Coie and Dodge 1998). “They may be pre-disposed to construe events in a biased way, perceiving threat in the acts of others and menaces in the vicissitudes of everyday life” (Caspi et al. 1994, 187). Furthermore, the tendency of individuals high in NE/LC to blame others for their problems increases the likelihood that they will react to stressors with anger and this anger, in combination with poor problem solving skills, enhances the probability that an individual will respond to stressors with delinquency. Neuroticism has also been linked to avoidance coping styles, like substance use, through its effect on perceptions of stress (Kardum and Krapić 2001).

Because individuals with weak constraint tend to act quickly with little thought, low constraint may enhance the effect of negative emotionality on offending by encouraging the rapid translation of negative emotions into impulsive action (Caspi et al.

1994). Moreover, people with low levels of control may perceive stressors as more threatening or negative (Taylor and Aspinwall 1996), although this has not been well investigated.

In sum, individuals high in negative emotionality and low in constraint are theorized to be more reactive to stressors increasing the likelihood they will engage in offending and substance use. Consistent with population heterogeneity explanations of behavioral continuity, it is the stability of these traits overtime that is directly responsible for stability in illicit behavior.⁶

Path 1: Direct Effect—Empirical Work

If Agnew's population heterogeneity explanation of continuity is supported, we should expect to see two things. First, NE and LC should be relatively stable across the life course. The assumption of continuity in these traits is certainly reasonable. Research suggests that there is moderate stability in personality and temperament over the life span (see Caspi and Roberts 2001; Costa, McCrae, Arenburg 1983; Miller and Lynam 2000). Relative stability⁷ in personality, that is maintaining rank order compared to others, is more often the norm than absolute stability, which involves sustaining the level of a trait over time (McGue, Bacon, and Lykken 1993).⁸ In a meta-analysis examining the relative stability of personality, Roberts and Del Vecchio (2000) found that traits tend to change a good deal from birth to age 3, then dramatically increase in consistency until age 6, when

⁶ Followed to their logical conclusion, pure population heterogeneity explanations allow no room for real change in behavior. Although beyond the scope of this paper, this appears to be contrary to the mounting evidence that change is possible (see Laub and Sampson 2003).

⁷ This has also been called differential stability (Caspi and Roberts 1990).

⁸ Absolute and relative continuity are both forms of homotypic continuity in that they refer to stability of a particular behavior or phenotype over time (Caspi and Roberts 1990). Heterotypic continuity may also exist, which in the context of personality or individual differences, implies that the same underlying trait, like NE or LC, manifests itself in different ways.

they maintain relatively stable until the college years. Another increase in relative stability occurs through the mid-20s, with stability leveling off again during the 30s, until one final increase in the stability of personality during the 40s and 50s. Although, personality becomes more fixed over time, it always retains the possibility of changing; however, these changes are usually minor (Caspi and Roberts 2001).

Second, and of primary importance for GST, individuals high in NE/LC should be more reactive to stressors. Research from various fields, including psychology, genetics, biology, and behavioral ecology, overwhelmingly suggest that this is true (see Rutter et al. 1997). For example, a relationship between neuroticism and increased emotional distress was found by Bolger and Schilling (1991) using a community sample of married couples, who were asked to keep a daily diary of stressful experiences and emotional distress (anxiety, depression, and hostility). They found that individuals who scored above the median in neuroticism experienced higher levels of distress and that differences in neuroticism explained 8 percent of the between-individual variance in daily distress. Although part of this variability was explained by differential exposure to stressors, (individuals high in neuroticism were exposed to more daily interpersonal conflicts), decomposition of the relationship between neuroticism and distress indicated that differential reaction to stressors accounted for twice as much of the distress difference between high and low neurotics than differential exposure. Similarly, studies in the mental health literature have found that differential reactivity to stressors also explains some of the increased rate of depression for neurotics as compared to non-neurotics (e.g., Ormel, Oldehinkel, and Brilman 2001; Van Os and Jones 1999).

Only two studies examine the association between NE/LC, stress reactivity, and delinquency. In the first, Agnew and colleagues (2002) used cross-sectional data from a nationally representative sample of youth to study the moderating effect of NE/LC on the strain-delinquency relationship. As hypothesized, the relationship between self-reported strain and delinquency was stronger for individuals who were high in NE/LC as reported by their teachers and parents. Furthermore, NE/LC had virtually no effect on offending for individuals experiencing low self-reported levels of strain (i.e. individuals with strain levels that were one standard deviation below the sample mean). These results held even when relevant social control and social learning variables and prior problem behavior were included in the model.

The second study simultaneously replicates Agnew and colleagues' (2002) findings and calls them into question. Ousey and Wilcox (2007) found that peer bullying had a significantly greater effect on delinquency for individuals low in self control when the relationship was modeled using OLS. However, drawing on Osgood, Finken, and McMorris (2002) they argue that because of the censoring and skewness of delinquency measures, OLS is inappropriate and tobit, a technique appropriate for data with these characteristics, should be used. When using tobit, they find that the association between strain is in the opposite direction predicted by GST; the relationship between strain and delinquency is weaker for individuals with low self control. Ousey and Wilcox conclude that models used in previous studies were misspecified and that once this is corrected there is no support for differential reactivity to stressors by varying levels of low self-control.

Several problems with these studies limit the interpretability of the results. First, both used cross-sectional data making it difficult to establish causal ordering. Furthermore, in Agnew and colleagues' study the respondents were asked to report on current strain but past-year delinquency, consequently the researchers examined the effect of current strain on past delinquency. In addition, Ousey and Wilcox included only measures of self-control and used a very limited measure of strain, peer bullying. It is possible, therefore, that their findings with the tobit model would be different if they used a more comprehensive measure of stressors and a more appropriate measure of negative emotionality and low constraint. Finally, like most tests of GST, these studies only looked at adolescents.

In summary, there is evidence that NE and LC are associated with delinquency and that these traits are relatively stable over time. Studies examining the interaction between NE and LC and stressors support the idea that these traits moderate the stressor-mental health relationship; however tests with delinquency as outcome have been less conclusive.

Extension of Path 1: Past Experience as a Source of Population Heterogeneity

The stress literature and the life-course framework suggest that Agnew's explanation of stability can be extended to take into account differences in reactivity to stressors that are not trait based by considering the lasting effects of past experiences. Like negative emotionality and low constraint, past exposure to stressors, especially traumatic life experiences and serious chronic stressors, is believed to interact with current stressors to enhance the effect of stressors on offending and substance use (Thoits

1983).⁹ When experienced early in life, particularly before age 10, stressful experiences have the ability to modify an individual's response to later stressors (Turner and Lloyd 1995). In this manner, early stressful experiences can lead to stability in offending through their lasting effect on stress reactivity. This explanation of stability still draws on population heterogeneity to explain continuity in offending, however, the source of the time stable differences is experiential, not necessarily biological or psychological.¹⁰

There is some debate, however, about whether early exposure to severe stressors exacerbates or mitigates the effects of later stressors. Drawing on the engineering model of stress, Wheaton (1996, 36) argues that models of the stress process should consider that experiencing stressful situations may enhance an individual's coping capacity such that future strain of the same type and level will have less of an effect. Childhood stressors may also present learning opportunities that promote resilience providing an "inoculation against later stress" (Moen and Erickson 1995, 178). There has been some support for the inoculation hypothesis. For example, Wheaton and colleagues (1995) found that stressors had a weaker contemporaneous effect on mental disorder for individuals with a higher number of cumulative stressors.

In contrast, research also suggests that experiencing severe stress in childhood may exacerbate the effects of stressors on later mental health. In these situations, the size and force of a stressor can become so great as to overwhelm the capacity to deal with stress, thus decreasing the amount of additional stress an individual can withstand

⁹ Traumas are relatively rare stressors that are deeply disturbing and almost always overwhelming in their impact (Wheaton 1996).

¹⁰ Evidence suggests that an individual's prior behavior and experiences can affect his or her reactivity to stressors via multiple pathways, some sociological, like changes in relationships (Rutter et al. 1997), some biological (Fishbein 2001; McLean and Link 1994), and some psychological, such as changes in cognitive processing (Rutter et al. 1997). My point here is that differential stress reactivity due to past experiences does not require one to rely on a trait-based explanation.

(Wheaton 1996). For example, those experiencing chronic stressors may deplete their coping resources or may drive others away (Taylor and Aspinwall 1996). Using three waves of the American's Changing Lives data, a national longitudinal survey of adults between the ages of 24 and 96, Umberson et al. (2005) examined the influence of exposure to stressors in childhood on trajectories of marital quality. They found that individuals who experienced a great deal of stress in childhood were more reactive to stressors they encountered in later life. Similarly, the effect of stress on multiple mental disorders is reportedly stronger for individuals whose parents went through a divorce during their childhood (Aseltine 1996; Elder, George, and Shanahan 1996).

There could be several reasons for these divergent results. First, the direction of the effect may depend on the type of stressor that is experienced (Aseltine 1996). Second, the effect of early exposure to stressors may be contingent on the magnitude or chronicity of the stressor. For example chronic stressors may erode coping mechanisms like social support, leaving the individual with few resources to deal with later adversity (Monroe and McQuaid 1994) and enhancing the effect of future stressors on criminal behavior. Conversely, new stressors may have little additional effect on individuals who have led a life full of adversity; they are merely a drop in the bucket of life difficulties. Finally, it is also possible that social context in which the past stressors occurred may moderate its effect on current strain (Monroe and McQuaid 1994). In affluent communities, when a traumatic event, such as the death of a parent, occurs the loss may actually mobilize neighborhood resources, enhancing an individual's ability to cope with later stressors. In a disadvantaged neighborhood, where resources are already spread

thin, there may be less access to legitimate coping mechanisms to deal with aversive situations.

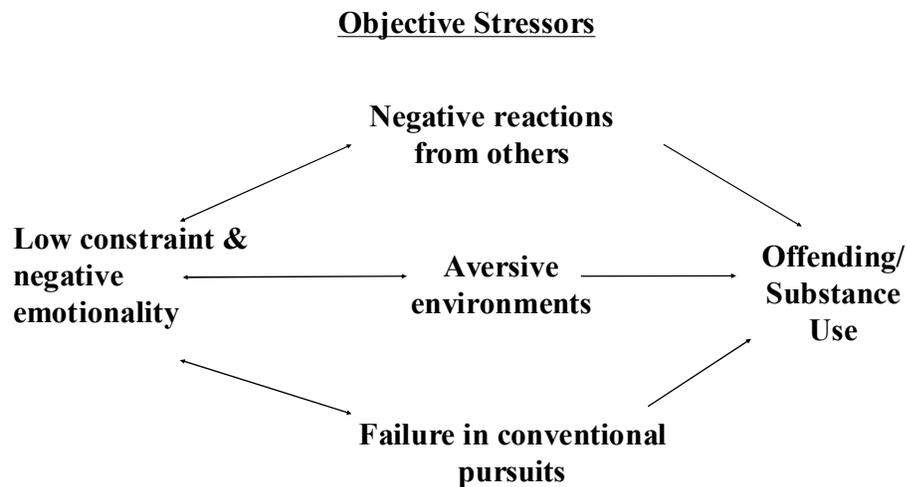
PATH 2: SELECTION INTO OBJECTIVELY AVERSIVE SITUATIONS: ACTIVE AND EVOCATIVE SELECTION

Exposure to strain is not evenly dispersed across the population (Rutter et al. 1995) or across the life span (Elder, George, and Shanahan 1996). There are likely many factors that influence the amount, magnitude, and types of stressors to which an individual is exposed with one of these being individual differences. Agnew argues that the second way negative emotionality and low constraint can account for behavioral continuity is by affecting exposure to objective stressors (i.e. stressors that most people would find aversive) (Agnew 1997, 2006), like failure to marry or bad marriages, negative school experiences, and unemployment or employment in the secondary labor market (Agnew 2005). Because individuals high in NE/LC face more stressors, and therefore more negative emotions, these individuals will be more likely to engage in criminal behavior. Experiencing stressors, in turn, contributes to the maintenance of NE/LC.

There are three central ways traits may influence exposure to objective stressors (see figure 1). First, individuals high in negative emotionality are unlikely to be successful in conventional pursuits and relationships because they have problems getting along with others and are unlikely to dedicate the time and effort necessary to excel in conventional activities (Agnew 2005, 68). In turn, failure in conventional pursuits can force those with NE/LC into strain-producing environments, like the secondary labor

market and delinquent peer groups. Second, children with traits like negative emotionality or low constraint may evoke negative reactions from their social environments (Agnew 2006). These responses may be stressful in and of themselves, or they may introduce the individual to stressful situations. This process is often referred to as evocative selection (Scarr and McCartney 1983). Finally, in the process of active selection (Scarr and McCartney 1983), individuals with these traits might actively seek interpersonal interactions with others that they find compatible and stimulating, leading to environments and relationships fraught with stressors. Because the first of these mechanisms is closely related to social control, the focus in this dissertation will be on the latter two pathways.

Figure 1. Active and Evocative Selection



Path 2—Evocative and Active Selection: Empirical Evidence

According to Magnus and colleagues (1993, 1047), “The preliminary findings in this area suggest that life events should not be treated entirely as exogenous shocks to people but they also have an endogenous component that can be predicted by certain

personality traits.” Many studies have found a relationship between neuroticism/negative emotionality and exposure to negative life events (e.g. Aldwin et al. 1989; Headey and Waring 1989; Kendler, Gardner, and Prescott 2003; Magnus et al. 1993; Van Os, Park, and Jones 1999, 2001, but see Ormel, Oldehinkel, and Brilman 2001). For example, using longitudinal data collected as part of the Medical Research Council National Survey of Health and Development (NSHD), Van Os, Park, and Jones (2001) examined the effect of neuroticism and cognitive ability measured in late adolescence on later depression and anxiety and life events. Path analysis indicated that even after controlling for the reciprocal relationship between life events and mental health, neuroticism had a significant positive relationship with both life events and anxiety/depression measured at age 36. The effect of neuroticism on life events, though significant, was substantively small.

Kendler and colleagues (2003) also found a significant association between neuroticism in adulthood and significant life events using two waves of data from a population-based sample of adult mono- and dizygotic twins. Self-reported neuroticism at wave 1 was associated with 14 out of 18 significant life events at wave 2 and this relationship was strongest for marital problems, joblessness, and financial problems. Studying twin pairs allowed the researchers to estimate the contribution of shared familial factors (both environmental and genetic) to the relationship between neuroticism and life events. Results suggest “the existence of a set of shared familial factors which both influence levels of neuroticism and predispose an individual to select themselves into or create for themselves high-risk environments” (1200). In addition a comparison of the results for mono- and dizygotic twins indicates that at least part of this shared effect is

genetic. However, Kendler and colleagues did not control for wave 1 levels of environmental adversity, raising the possibility that the effect of neuroticism on environmental adversity is spurious.

Magnus and colleagues (1993) used longitudinal data collected from a sample of college students to study the effect of neuroticism on the occurrence of objective life events. The authors limited their outcome to objective events—those that can be externally verified—because reports of subjective life events (such as made a lot of new friends or had problems getting along with co-workers) may be influenced by the independent variables of interest (extraversion and neuroticism). Focusing on objective events only (like getting married or the birth of a child) reduces response bias that may result from the independent variables of interest affect the reporting of life events. They found that neuroticism measured in college was significantly and positively associated with the number of negative objective life events a person reported experiencing 4 years later.

Finally, Champion, Rutter, and colleagues (Champion et al. 1995; Rutter, Champion, et al. 1995) found that individuals who lacked planning were more likely to experience long-term difficulties. This is consistent with the hypothesis that low constraint, one characteristic of which is impulsivity, is partially responsible for sorting individuals into stressful environments.

Overall, evidence suggests that selection into stressful situations is a viable source of continuity in behavior. Empirical findings “are inconsistent with an aetiological model of psychiatric illness in which individuals are passive recipients of environmental adversity. Individual differences in personality, which result partly from genetic

influences, significantly impact on the way in which individuals structure the world around them” (Kendler, Gardner, and Prescott 2003). However, whether the relationship between negative emotionality, low constraint, and exposure to chronic stressors can explain continuity in offending and substance use over time is still unknown.

PATH 3—STABILITY OF AVERSIVE FAMILY ENVIRONMENT: LINKED LIVES AND PASSIVE SELECTION

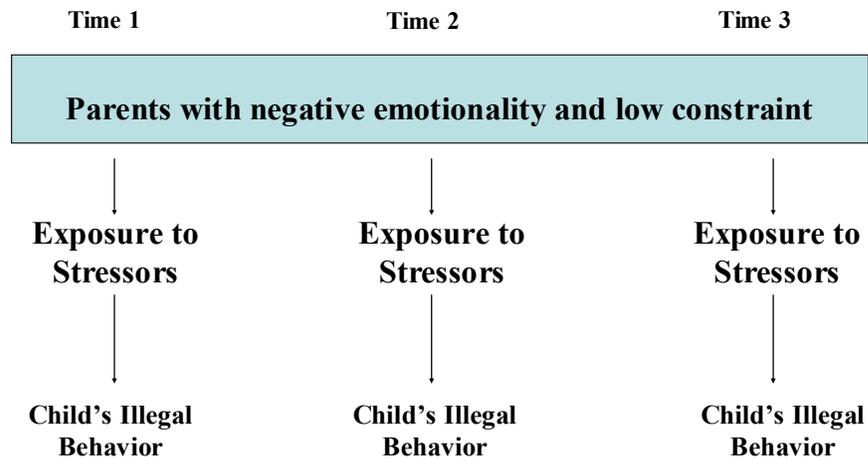
The third manner in which NE and LC contribute to continuity is through their effect on the home environment (see figure 2). According to Agnew (1997) children high in NE and LC are likely to have parents who possess these traits, partially because there is a hereditary component to these characteristics (Kendler, Gardner, and Prescott 2003; Rutter et al. 1997) and partially because parents who are high in negative emotionality and low in constraint are likely create a family environment that promotes the development of these traits in their children (see Rutter and Silberg 2002; Rutter et al. 1997, 345). Parents with these characteristics are impatient and irritable (Agnew 1997) and typically create home environments for their children that are high in objective stressors (Fishbein 2001). This is independent of any negative reactions the child may elicit from his or her parents because of the child’s own difficult temperament. Because youth rarely have recourse to change their living arrangements, a child born into a home life filled with stressors will most likely remain in that aversive environment until he or she is old enough to move out. Children who are continually exposed to stressors created by their parents’ behavior may attempt to relieve the ensuing negative emotions by using illicit coping mechanisms, like offending and substance use. Scarr and McCartney

(1983) refer to this process as passive selection, because the individual's environment is provided for him or her. Stability in antisocial behavior, therefore, is due to a correlation between parent and child traits and the effects of parental characteristics on the home environment.

Although not dynamic, this type of passive selection can be expanded to include the life-course principles of linked lives and social context. Stressors faced by parents are passed onto their children because family relationships make exposure to stressors a shared experience (Elder, George, and Shanahan 1996). Moreover, this process can be used to explain intergenerational continuity in behavior.

It is important to note that in this scenario, the likelihood that the child will use illegitimate versus legitimate coping mechanisms is increased because he or she has an increased chance of being high in NE and LC (Agnew 1997, 2006). This is partially attributable to the heritability of these traits and partially attributable to the stressful home environment, which contributes to the development of NE and LC. Therefore, an aversive environment serves not only as a wellspring of stressors, but also as a source of the very traits believed to condition the effect of stressors. However, some have pointed out that it is not clear whether environmental continuity is a cause or a consequence of negative emotionality and low constraint (Caspi and Roberts 2001).

Figure 2. Passive Selection



Path 3—Passive Selection: Empirical Evidence

Research indicates that men and women high in neuroticism tend to have poor parenting practices and create stressful environments for their children. Parents with high levels of neuroticism are more likely than others to engage in negative emotional interactions with their children, to have lower sensitivity to their children's needs, and to provide them with less cognitive stimulation (Belsky, Crnic, and Woodward 1995). Ellenbogen and Hodgins (2004) studied a sample of parents with and without bipolar disorder and their offspring. They found that parents high in neuroticism exhibited poor psychosocial functioning, ineffective coping strategies, and experienced a greater number of negative life events.¹¹ In addition these parents possessed poor parenting skills, which included a failure to provide their children with emotional support or an organized,

¹¹ These include stressors that may have been partially attributable to, and not independent from, the parents' own behavior.

predictable, and consistent home life. Not surprisingly, parents with high levels of neuroticism were significantly more likely to have children with externalizing, internalizing, social, and attention problems. Because the data were cross-sectional, it is impossible to determine the direction of causality; therefore, the observed relationship may be due to the influence of children's difficult behavior on parenting practices rather than vice versa. However, this is an empirical question.

Additional evidence of passive selection has also been found in four generations of data from the Berkley Guidance Study. Elder, Caspi, and Downey (1986) found behavioral continuities across generations such that unstable personalities led to disrupted family relations, which in turn resulted in unstable personalities in the next generation.

Extension of Path 3—Chronic Stressors and Stress Proliferation

Agnew's discussion of passive selection provides a preliminary framework for discussing how stability in aversive environments can promote continuity in offending and substance use. However, it is just that, preliminary and therefore requires further development. There are several weaknesses with Agnew's discussion. First, it is apparent that individuals may be part of other aversive environments, in addition to their family. Second, a life-course perspective makes clear the importance of studying development across the life span, not just childhood and adolescence. Therefore, the explanatory usefulness of this stability-producing mechanism could be greatly enhanced if it were extended to explain the continuity of offending and substance use into adulthood. Third, aversive environments have multiple causes, many of which are social in origin (e.g. unemployment, divorce, poverty) not just parental negative emotionality

and low constraint. Finally, this explanation is static and fails to take into account that experiencing stressors can have a causal impact on the likelihood of experiencing later stressors.

An examination of the stress literature points to a relatively simple way to generalize this pathway to other contexts and stages of life—reframe the discussion in terms of chronic stressors. Shifting to a discussion of chronic stressors has two additional advantages. First, it decouples this explanation from its reliance on negative emotionality and low constraint. Second it focuses attention on, arguably, one of the most detrimental type of stressors—chronic strain. Some researchers go so far as to posit that other types of stressors, like negative life events, affect negative outcomes solely as initiators of chronic stressors (Pearlin et al. 1981). For example, it is not losing a job that is detrimental; rather it is the long term ramifications of this event, like poverty and relationship problems due to arguments about money, that take the greatest toll. Anton Chekhov famously made this point when he stated “Any idiot can face a crisis—it’s day to day living that wears you out.”

Chronic strains are stressors that last for an extended period of time or occur repeatedly over the life course. Menaghan (1997) describes them as the continuing, non-dramatic social conditions, like economic hardship, that can maintain the path of a trajectory like “the cumulative effect of a steady, day-after-day drip of water on a rock” (114). Chronic stressors may result from persistent life difficulties; role strain; strain resulting from societal responses to characteristics of the person, such as race or ethnicity; or community-wide strain (McClellan and Link 1994, 24). However, stress researchers also posit that chronic strain is produced through a dynamic process known as

stress proliferation, where current stressors have a direct causal effect on future stressors. It is based on the idea that exposure to one trauma puts people at risk for exposure to another. Stress proliferation,¹² may be responsible for maintaining continuity in stressor exposure, and hence offending and substance use, over time. Pearlin, Aneshensel, and LeBlanc (1997, 223) define stress proliferation as “the expansion or emergence of stressors within and beyond a situation whose stressfulness was initially more circumscribed” (223). According to Elder and colleagues (2005), “the notion of stress proliferation is useful in conceptually tracing the influence of early stressful circumstances on the structuring of the life course in ways that produce additional stressful circumstances” (214).

Stress proliferation is most often triggered by a trauma. Wheaton (1996: 53) has found, for example, that the powerful impact of childhood traumas arises from their indirect effects on negative outcomes via exposure to other types of stressors. Other stressful experiences, including chronic stressors, have the ability to trigger this process (Pearlin, Aneshensel, and LeBlanc 1997; Pearlin et al. 2005). Pearlin, Aneshensel, and LeBlanc (1997) distinguish between primary stressors and secondary stressors in the proliferation process. Primary stressors are adversities that are embedded in the situation whose effects are under study and secondary stressors are those that are influenced by the stressful situation being studied but are outside that situation (Pearlin, Aneshensel, and LeBlanc 1997, 227). For example, if one is studying the effects of working in the secondary labor market, the stressors that are inherent in the job itself, like lack of autonomy, are primary stressors while stressors that arise from this situation but are not

¹² De Coster and Kort-Butler (2006) refer to this process as “stress spillover” (301).

in the same life domain, such as marital problems due to emotional distress caused by the job, are secondary stressors.

These two types of stressors correspond to the two mechanisms through which stress proliferation occurs (Pearlin, Aneshensel, and LeBlanc 1997). First, primary stressors can expand into aspects of the same life domain that were previously unaffected or increase their intensity. Using the employment example again, primary proliferation occurs if the individual is tasked with new unwanted job responsibilities or if the lack of autonomy is intensified, perhaps through the monitoring of emails. The second stress proliferation mechanism is the expansion of the primary stressor into other life domains. Following our example, if an individual is forced to work over time to complete new job responsibilities, this can lead to marital problems if the spouse requires his or her assistance at home. In this manner, primary stressors may disrupt patterns of social interactions, obligations, and expectations, and lead to problems in other life domains (Pearlin, Aneshensel, and LeBlanc 1997). This underscores the importance of recognizing that individuals are simultaneously embedded in multiple contexts (Bronfenbrenner 1975) and that actions in one context (e.g., work) can have ramifications in other contexts (e.g. the family).

Agnew (1992, 2006) also recognizes the importance of chronic strain. He argues that chronic or repeated strains may create a disposition for crime such that individuals who experience chronic stressors will be more likely to respond with criminal behavior when tempted or provoked. He also suggests that strain of longer duration will be more likely to result in criminal behavior because it not only increases the likelihood that an individual will be high in negative affect at any given time, but also leads to a hostile

attitude towards others (1992). This increases the probability that any one specific instance of strain will instigate offending. Furthermore, individuals experiencing chronic stressors may deplete their coping resources, impairing their ability to deal with strain using legitimate coping mechanisms. In sum, chronic stress “primes the brain” for maladaptive behavior (Fishbein 2001, 64).

Extension of Path 3—Chronic Stressors and Stress Proliferation: Empirical Evidence

Despite the importance attributed to chronic strain in the stress process, not much is known about how duration of exposure to stressors influences outcomes (Elder, George, and Shanahan 1996). Findings regarding the effect of chronic stressors on offending have been mixed. Paternoster and Mazerolle (1994) found that living in a disagreeable neighborhood increased delinquency regardless of the length of time the adolescent lived in that area. This suggests that the length of time one is exposed to stressors is unimportant. Slocum, Simpson, and Smith (2005) studied self-reported within-individual changes in strain and offending over a 3 year period using a sample of female chronic offenders. They found that as the number of months a woman remained above her average level of strain increased, so did the likelihood of offending and drug use. Similarly, in a study of racial differences in stress exposure, Eitle and Turner (2003) also found that chronic stressors, like job dissatisfaction, domestic issues and money problems, have a significant and positive effect on self-reported crime. In this study, stressors included in the survey were assumed to be chronic; the duration was not actually measured. More extensive work has been done with regard to the relationship between chronic strain and mental health. For example, Avison and Turner (1988) looked at the

role of self-reported discrete and chronic stressors on depressed mood using a sample of adults. Respondents were asked to report on the timing and duration of life events for a 12 month period and they defined chronic strain as stressors lasting 10 months or more. Avison and Turner found that compared to discrete events, chronic stressors explained the greatest variance in mood scores.

Although research is limited, studies generally find support for the existence of stress proliferation. Pearlin, Aneshensel, and LeBlanc (1997) found some evidence supporting stress proliferation among the caregivers of patients with AIDS. Similarly, Aseltine (1996) found support for stress proliferation in his study on the effects of parental divorce. Secondary stressors, like parental conflict, financial distress, and family disorganization fully mediated the effect of parental divorce on psychological distress. “Divorce is seen as setting off a chain of negative events and transitions that are causally related to psychological distress and may be more potent stressors than the physical separation of parents” (134).

There are many unanswered questions surrounding the study of chronic stressors. Little work has looked at its maintenance through stress proliferation and none has considered it as a source of behavioral continuity. This hole in the literature may be partially due to the difficulty associated with measuring stressors. Not only is it hard to establish the beginning and end of chronic stressors, but many times, they come in the form of daily hassles that are overlooked in the offending research.

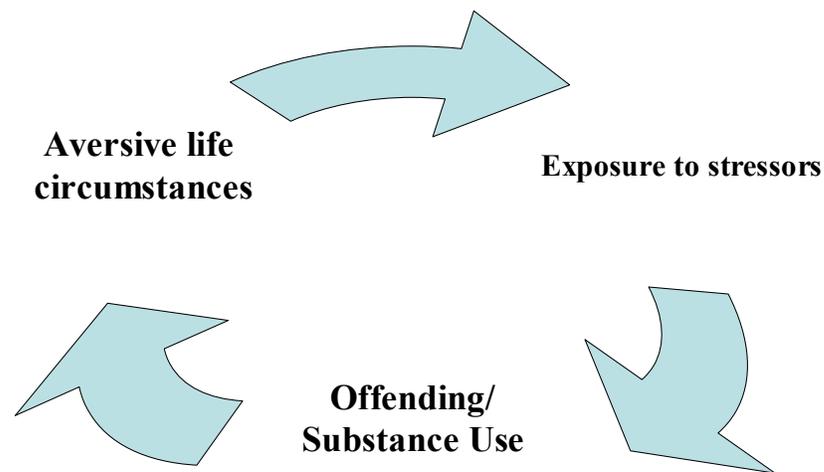
PATH 4—STRESSOR AND STRESSOR AND DELINQUENCY AMPLIFICATION

Of course, individual differences are not the only factors that influence how people shape the world around them. People are born or come into social circumstances for reasons beyond their control. Acknowledging this, the final source of continuity outlined by Agnew (1997, 2006) does not rely on the traits of negative emotionality or low constraint, but instead utilizes a state dependence argument.¹³ In this process, depicted in figure 3, individuals facing persistent poverty and underclass status are exposed to more stressors, are more sensitive to the stressors they experience, and are more likely to respond to stressors with offending. This enhanced exposure and reactivity to stressors increases the likelihood an individual will engage in offending. Offending creates its own set of consequences, like decreased social support or disrupted schooling (Sweeten 2005), and may disrupt the normative timing of life events, such as the birth of a child or marriage, enhancing the strain associated with these life changes (Elder, George, and Shanahan 1996). These aversive circumstances may then increase exposure and reactivity to stressors and make offending more likely. In essence, stressors act as mediators for the relationship between social location and social structure on the one hand, and illegal behavior on the other (Wheaton 1996). Therefore, consistent with other state dependent explanations of continuity, it is the negative ramifications an individual experiences as a result of his or her behavior that increases exposure to strain, creating a downward spiral (Agnew 1997, 2006). While Agnew posits that the “knifing off” of conventional social attachments contributes to continuity (Sampson and Laub 1993, 1997) as does associating with delinquent peers, there is a unique contribution of

¹³ Agnew draws heavily from others, including Thornberry (1987) and Sampson and Laub (1993, 1997) for the development of this process.

strain through a “...process of increasing personal and social disadvantage through the mutual reinforcement of emotional or behavioral problems and adversity” (Kim et al. 2003).

Figure 3. Amplifying Loop



Others have offered ways in which stressors may serve as the intervening mechanism between social structure and offending and substance use. Building on GST, Hagan and Foster (2003) argue that delinquency is an intervening mechanism linking stressors and anger with later alcohol problems and depression. In their sequential stress theory, they view delinquency as both a cause and consequence of stress. The full sequence they outline begins with social and economic stressors leading to anger. Some individuals choose to deal with their negative emotions by engaging in delinquency, which is a form of rebellion. Next, delinquency itself serves as source of stressors that leads to further behavioral or mental health problems, like drinking and depression. In this way, delinquency itself mediates the effect of anger on subsequent problem behavior.

De Coster and Heimer (2001) also see stressors as playing a role in maintaining the stability of offending and substance use, albeit a peripheral role. In their interactionist model, social structure influences exposure to negative life events, which increases delinquent behavior. At this point, however, stressors disappear from their model and instead, they argue that it is attachment and self appraisals that lead to continuity in behavior over time. It seems likely that although stressors are omitted from the stability promoting pathways in De Coster and Heimer's conceptual model, they still contribute to continuity.

Path 4—Stressor and Delinquency Amplification: Empirical Evidence

Evidence supports the stressor and delinquency amplification process. Using a sample of 10-year old children from schools in London who were followed up in their 20s, Champion, Goodall, and Rutter (1995) found that individuals who had experienced emotional or behavioral disturbances as children, experienced in their 20s twice the rate of severe negative events that carried a long-term contextual threat, the type of stressful event most likely to provoke psychiatric disorder. In contrast, these individuals did not experience increased rates of mildly stressful events or potentially stressful normative life changes. On average, both individuals with and without emotional and behavioral disturbances experienced the same mean number of stressful events that were a direct consequence of their actions (e.g., marital fights) and stressful events that were independent of their actions (e.g. death of a loved one from cancer); however, all events occurred at a lower frequency in the sample without disturbances. This indicates that the increase in severe negative threats cannot be entirely attributed to disturbed individuals' tendency to evoke negative reactions from others. They also found that emotional and

behavioral disturbance at age 10 was associated with an increase in severe life events, but only when the events were linked to a chronic psychosocial difficulty. In some cases, the link between the chronic psychosocial difficulty was direct (e.g., partner left respondent after chronic marital discord), but other times the relationship was less direct (e.g. both the event and the chronic difficulties were related to the individual's family of origin).

Using two waves of the Add Health data, Hagan and Foster (2003) found support for their sequential stress theory. Specifically, for both males and females family characteristics indicative of social and economic stress were associated with anger and in turn, anger was associated with delinquency. Furthermore, delinquency was linked to later depression for males and females and to drinking problems for males. As predicted, delinquency mediated, but only partially, the effect of anger on depression and alcohol problems. This work provides great insight into how stress, emotion, and problem behavior are related to each other over time, however, it could be improved upon in several ways. First, the study only covered a 2-year time period in adolescence. Therefore, although the theory provides a mechanism through which stressors contribute to stability in problem behavior, the empirical findings are only suggestive. Future studies should attempt to understand how this sequence develops over a longer period of time. Second, structural family characteristics (e.g., parent education, single parent family) were used as proxies for actual measures of stressors. Future work should include measures of actual stressors, particularly those that Agnew (2001) argues are most criminogenic. The final limitation of this study is that although Hagan and Foster postulate that stress is both a cause and consequence of problem behavior, their analytic

model measured stressors at one point in time, treating stressor exposure as only a cause, not a consequence, of delinquency.

Like Hagan and Foster (2003), Kim and colleagues (2003) also argued that problem behavior serves as a wellspring of stressors. However, unlike other researchers, Kim and associates explicitly modeled stressor exposure as both a consequence and cause of offending. They were interested in understanding the reciprocal relationship between experiencing stressors and exhibiting internalizing (depression and anxiety) and externalizing behaviors (major and minor delinquency). Specifically, they compared the social causation hypothesis (i.e., exposure to stressors leads to problem behavior) with the social selection hypothesis (i.e., problem behavior leads to increases in stressor exposure). This study used five waves of self-report data spanning early to late adolescence that were collected from a sample of white youths raised in intact families in rural Iowa.

Using cross-lagged autoregressive path models, Kim and colleagues (2003) found that negative life events and externalizing behaviors had a reciprocal relationship such that increases in one predicted increases in the other one year later. Therefore, negative life events and externalizing behaviors were both causes and consequences of each other. The researchers also found that, although the effect of stressors on externalizing behaviors was greater during early and middle adolescence, this pattern reversed in later adolescence. They suggest that stronger effect of negative life events on delinquency observed in early adolescence may be due to the extensive changes that occur during this period. In late adolescence, the negative ramifications of problem behavior begin to manifest themselves as individuals move into adulthood. This study illustrates why it is

important to consider the stage of the life course being studied; the effects of behaviors and their patterns of development may vary across the life span.

Although this study does provide a more dynamic perspective of the relationship between stressors and offending, it is not without its limitations. Specifically, their sample is not very representative of offending populations (i.e. all white, adolescents from two-parent homes living in rural Iowa). In addition, they fail to include any controls for possible time-varying confounds.

SUMMARY OF THEORETICAL AND EMPIRICAL LITERATURE

In sum, Agnew (1997, 2006) argues that stability in behavior results from complex feedback loops between individual traits, behavior, and exposure to stressors. No research has been done that examines all of these potential pathways at once, but studies of GST and development are generally consistent with Agnew's explanations of continuity. In concordance with Agnew's direct pathway, studies suggest that negative emotionality and low constraint have a direct effect on offending and substance use, are relatively stable over time, and increase reactivity to stressors. Evidence also supports evocative and active selection processes. Relatively rigorous studies have found that individuals high in negative emotionality and low in constraint are exposed to a greater number of objective stressors. Less work has explicitly examined the role of passive selection in behavioral continuity, but negative emotionality and low constraint are partially inherited and parents with these traits tend to provide chaotic home environments for their children. There is also some evidence of a relationship between chronic stressors and illegal behavior, although studies specifically examining

delinquency and offending provide more mixed results. However, none of these studies view the production of chronic stressors as part of a dynamic process. Finally, research suggests the existence of a stressor and delinquency amplification process. The above review suggests that GST theory has something to add to explanations of continuity in offending and substance use over time. However, to date there have been no attempts to simultaneously consider all these explanations. This is one of the contributions of the current study.

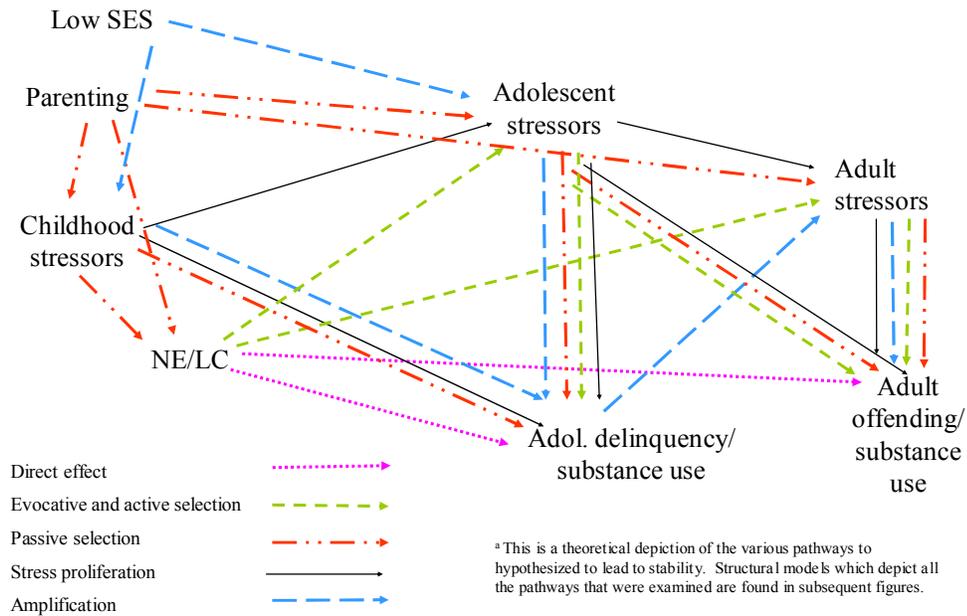
HYPOTHESES

The above description makes obvious that GST has much to offer the study of the development of stressors and illegal behavior over time. Thinking about how each of the explanations of continuity—direct effect, active and evocative selection, passive selection, stress proliferation, and stressor and delinquency amplification—applies to the development of stressors and offending/substance use over time provides a useful framework in which to study stress explanations of behavioral continuity. It also provides for the development of clear hypotheses about the relationship of negative emotionality and low constraint, stressors, and offending and substance use over time. In this section, I synthesize these pathways to form a conceptual model of continuity in stressors and illegal behavior. This theoretical model is introduced in figure 4.¹⁴ Of particular interest are the dynamic mechanisms through which behavioral continuity is maintained.¹⁵

¹⁴ Figure 4 is not meant to depict structural models (these are presented later); rather its purpose is to theoretically distinguish the pathways from one another.

¹⁵ Note that identical hypotheses are made for both offending and substance use. For simplicity, the term criminal behavior is used to refer to both of these outcomes.

Figure 4. Theoretical Model of the Development of Criminal Behavior and Stressors Over Time ^a



DIRECT EFFECT

The direct effect hypothesis states that continuity in offending and substance use is due to the stability of NE/LC, which is a direct cause of criminal behavior. Here the relationship between past and future behavior is spurious, with both resulting from negative emotionality and low constraint. In figure 4, this explanation is represented by a direct path between NE/LC and adolescent and adult criminal behavior, respectively. If continuity in offending and substance use is solely a result of population heterogeneity processes as predicted by the direct effect hypothesis then once these direct relationships are entered into the model the association between adolescent and adult criminal behavior will be reduced to 0. This leads to the first hypothesis:

Hypothesis 1: NE/LC will have a direct, positive association with adolescent and adult criminal behavior. Once these paths are controlled, there will be no significant direct association between adolescent and adult criminal behavior.

ACTIVE AND EVOCATIVE SELECTION

Continuity in offending and substance use may arise if individuals high in NE/LC select themselves into stressful circumstances, either through evocative or active selection. In this selection pathway, there are no causal effects of past exposure to stressors on present exposure. Rather, the relationship is spurious, with NE/LC causing both. In addition, there is no direct effect of NE/LC on criminal behavior; its effect is indirect via exposure to stressors.

Hypothesis 2: NE/LC will have a direct effect on exposure to stressors in adolescence and adulthood and there will be no causal relationship between childhood, adolescent, and adult exposure to stressors. The effect of NE/LC on criminal behavior will be indirect, operating through exposure to stressors.

PASSIVE SELECTION

The third source of continuity in behavior discussed by Agnew is that individuals who are high in NE/LC will have parents who are high in these same traits, making the parents likely to provide an aversive family environment. As long as the child remains in this family environment, he or she will be exposed to stressors, which promotes continuity in illegal behavior. This explanation also implies that there should be a direct association between parent and child NE/LC (a hereditary effect), as well as an indirect

relationship via exposure to stressors in childhood (an environmental effect). Ideally, measures of parental negative emotionality and low constraint would be included in the model, however, the dataset lacks these variables. Instead, poor parenting is used as a proxy because it is one of the mechanisms through which parental NE/LC is passed from parents to children, and because it contributes to a stressor-filled home environment.

Hypothesis 3: Poor parenting leads to stability in criminal behavior through its effect on NE/LC and by increasing a child's exposure to stressors. Continuity in stressor exposure, therefore, is due to continuity in poor parenting.

STRESS PROLIFERATION

Extending Agnew's explanation of continuity to include chronic stressors more generally and expanding it to include contexts aside from the family produces a more dynamic picture of the development of stressors. Chronic stressors can be divided into two types. The first results from one type of stressor lasting an extended period of time, such as a lengthy bout of unemployment. This form of chronic stressor provides little explicit information on the development of stressors over time aside from emphasizing the non-discrete nature of some types of stressors. In contrast, the second source of chronic stressors, stress proliferation, explicitly states that past stressors have a direct causal effect on future stressors independent of other factors, like temperament.

Hypothesis 4: Stressors in childhood will have a direct effect on stressors in adolescence. In turn, stressors in adolescence will have a direct effect on stressors in adulthood. The direct effect of these stressors on criminal behavior leads to continuity.

STRESSOR AND DELINQUENCY AMPLIFICATION

Using state dependence arguments to explain stability in criminal behavior, Agnew argues that low SES is associated with increased exposure to stressors. These stressors, and the ensuing negative emotions, are associated with offending and substance use, which in turn more deeply entrenches the individual in the underclass. “Delinquency has consequences which contribute to further delinquency, setting an amplifying loop in motion” (Agnew 1997, 112). In this process, the adversity resulting from criminal behavior links past exposure to stressors to present exposure. Here, offending and substance use are both a cause and consequence of stressor exposure and their effect on future criminal behavior is indirect with adult stressor exposure mediating their relationship.

Hypothesis 5: Low SES will be associated with exposure to stressors. The effect of past stressors on future stressors will be mediated by criminal behavior and the effect of adolescent criminal behavior on adult criminal behavior will be mediated by exposure to stressors.

MODERATING EFFECTS

According to Agnew (1997, 2006; Agnew et al. 2002), in addition to its direct effect on illegal behavior, NE/LC should also have a moderating effect. There should be a stronger relationship between exposure to stressors and offending and substance use for individuals who are high in NE/LC because they are more reactive to the stressors they experience. This leads to the following hypothesis:

Hypothesis 6: There will be a stronger relationship between stressors and criminal behavior for individuals who are high in NE/LC.

Drawing from the life-course framework, I extend the direct effect pathway and propose that past experiences with stressors will shape how an individual reacts to future stressors. The research is divided as to whether past exposure to severe stressors will enhance or mitigate the relationship between current stressors and illegal behavior; however only the former of these should promote behavioral continuity. Therefore, I expect the effect of stressors on offending and substance use to be stronger for individuals who have experienced severe stressors in childhood in comparison to those who have not.

Hypothesis 7: There will be a stronger relationship between stressors and criminal behavior for individuals who experienced stressors in childhood.

SUMMARY

Agnew (1997, 2006) argues that the stability of criminal behavior is primarily due to traits that develop early in life, specifically negative emotionality and low constraint. These traits may have a direct effect on offending and substance use or increase an individual's exposure to stressors through active/evocative selection and passive selection. Consistent with a population heterogeneity explanation of stability, criminal behavior remains stable because of the persistence of these traits over time. Independent of individual differences, Agnew states that offending and substance use can be maintained over time through state dependent mechanisms—criminal behavior has a causal effect on future behavior via increased exposure to stressors. I posit that two

additional mechanisms—stress proliferation and past exposure to traumatic stressors—can also promote continuity in offending and substance use.

The next chapter details the unique datasets used to test these hypotheses as well as the analytic strategy that was utilized.

CHAPTER III: METHODS

To examine the research questions outlined in the previous chapter I used data collected as part of the Collaborative Perinatal Project and the Pathways to Adulthood studies that, when combined, follow from birth to adulthood a sample of children born to high-risk mothers in the early 1960s. Most of the variables that Agnew views as central to promoting stability in illicit behavior were produced using exploratory and confirmatory factor analyses. Outcomes variables (delinquency and offending and adolescent and past year substance use) were also created using these techniques. Other constructs, like exposure to stressors and demographic characteristics, were measured with observed rather than latent variables. The hypothesized causal relationships among these variables were tested using structural equation modeling, which allows for the analysis of latent and observed data.

In this chapter, I provide more detail about the studies from which my data were drawn, the sampling procedures they utilized, and how the final sample was created for this study. I then describe this final sample, comparing it to the original sample from which data were collected. Next, the observed indicators from which my measures were created are described. Finally, I discuss structural equation modeling as an analytic technique to test the pathways hypothesized to lead to continuity in offending and substance use from adolescence to adulthood.

DATA OVERVIEW

The present study combines information collected from two different studies conducted by researchers at Johns Hopkins Hospital: the Collaborative Perinatal Project

(CPP) and the Pathways to Adulthood study (PTA).¹⁶ The CPP was a nationwide study funded by the National Institute of Health to study relationships between biological risk factors during pregnancy and birth outcomes, taking into account the effect of family and individual characteristics (Hardy 2003). CPP data were collected from 14 obstetrical or pediatric departments at 12 universities around the United States, but the study at hand uses only data from the Baltimore site (Hardy 2003).

Researchers began the data collection for the CPP in the early 1960s. Participants were pregnant women seeking prenatal care and delivery at Johns Hopkins Hospital between 1960 and 1964 (G1) and the offspring born to these women (G2). Because the participants were drawn from a clinic for low-income women, high-risk women and their children were oversampled. Prospective data were collected on the mothers and their children between 1960 and 1973. Data on the mothers include obstetrical and reproductive history, observations of the mothers with their infants, and family history, demographic and sociological information at their children's births and age 7 evaluations.

Extensive biological information was collected on the child at the time of birth and post delivery, including gestational age and birth weight as well as observations and diagnoses regarding the infant's physiological, physical, and neurological integrity. At 4 and 12 months and 7 years researchers assessed each child's neurological, neurosensory, and cognitive development, and general health and physical growth. (Hardy 2003, 305). Researchers supplemented this data with information from clinical notes and laboratory studies, and summaries of hospital admissions and medical records (Hardy 2003, 306).

¹⁶ The bibliographic citation for these data is Hardy, Janet B., and Sam Shapiro. Pathways to Adulthood: A Three Generation Urban Study 1960-1994: [Baltimore, Maryland] [Computer file]. ICPSR version. Baltimore, MD: Janet B. Hardy, Johns Hopkins University, Department of Pediatrics/Baltimore, MD: Sam Shapiro, Johns Hopkins University, Department of Health Policy and Management [producers] 1998. Ann Arbor MI: Inter-University Consortium for Political and Social Research [distributor] 1999.

In addition, psychological and behavioral data were collected at ages 8 months and 4 and 7 years by developmental psychologists following a structured protocol and at age 8 months researchers also collected observational data on maternal behavior toward the child. IQ tests were also administered at age 4 and age 7.

A great deal of care was taken in collecting high quality data. Clinical examinations were done by staff members with special training, who had no knowledge of prior findings. Data collection was reviewed by senior staff who provided feedback to the clinicians in order to resolve any problems with incompleteness or inaccuracy. Behavioral data were checked for completeness and consistency by another psychologist and finally a senior staff psychologist.

The second study, Pathways to Adulthood, was conducted between 1992 and 1994 and re-interviewed the original mothers and their children, who were then 27 to 33 years old. This study used a “catch up” design (Dempster-McClain and Moen 1998), in which participants from the CPP were relocated and re-interviewed approximately 20 years after the end of the original study. Approximately 70 percent of the interviews were conducted in-person. The remaining 30 percent were carried out via phone because the respondent lived outside of the region, resided in a dangerous area, or refused to meet in person. All respondents received a \$25 incentive for their participation.

Maternal interviews were intended to supplement and corroborate the information provided by their children. Almost of all the maternal interviews were conducted with the birth mother, with 5 percent being completed by whoever raised the child between the ages of 8 and 18. Data on the mothers include information about their education, employment, family composition, health, income, partnerships, and neighborhood

characteristics. Each mother was also asked to provide information on her child's life during the period between the original and follow-up studies, including his or her behavior, family relationships, and academics.

The PTA interviews with the children were more extensive and included questions about their aspirations, education, schooling, employment, family relationships, involvement in delinquency, substance use, reproductive history, and social relationships. A life event calendar was also used to collect yearly data on each respondent's living situation, marital status, fertility, welfare utilization, work history, and schooling during the period between the two studies. In addition, all respondents were asked to retrospectively report their perceptions of the safety of their neighborhoods and their residential addresses at birth, age 11/12, age 16/17, and at the time of the follow-up. These addresses were then matched to census tracts and census data.¹⁷

Like the CPP, collecting complete and accurate data was a high priority for the PTA study. Interviewers attended three weeks of training and each interview went through a three step editing process. First, immediately after the interview concluded, the interviewers reviewed the data. Next, the data were inspected by editors who looked for completeness and accuracy. When necessary, the editors would re-contact interviewees to get additional information. They also validated the data by calling participants and asking them a predesignated set of questions taken from the interview and comparing these phone responses to the interview data. Approximately 10 percent of the records were validated in this manner. At this time, the data were also cleaned using a program to check for out-of-range responses and invalid skip patterns. In the final stage of data

¹⁷ Additional data that were collected but not used in the present study include information on the children of the children (i.e. G3).

cleaning, researchers working with the dataset informally checked its accuracy, internal consistency, and completeness and reviewed hard copies of the interviews to resolve any problems.

SAMPLE SELECTION AND DESCRIPTION

SAMPLING PROCEDURE

The CPP sampled pregnant women seeking prenatal care and delivery at Johns Hopkins Hospital between 1960 and 1964. In the first year of the study 30 percent of the prenatal clinic registrants were randomly selected for participation based on their hospital identification numbers. The percent of women sampled increased yearly until 70 percent were sampled in 1963 and 100 percent in 1964. These women are not the sample of interest in the current study. Rather, the current work primarily uses data collected from the children born to these women, generation two (G2).

The PTA study attempted to re-interview all the CPP children born between the years of 1960 and 1965 who completed the 7 and/or 8 year assessment, as well as the mothers of these children. Of the 2,694 children who met these criteria 1,758 completed the PTA interviews, as did 1,552 of the 2,307 eligible mothers.¹⁸

SAMPLE ATTRITION

Preventing sample attrition was a major priority for the CPP. At the Johns Hopkins site, 76 percent of all surviving children attended all of the scheduled follow-up exams, with 90 percent completing the first year exam, 85 percent the 4 year exam, and 88 percent the 7 and 8 year exams. Table 1 outlines the pattern of attrition beginning

¹⁸ More children were eligible than mothers because one mother could have multiple children in the study.

with the number of children who were eligible for inclusion in the CPP ($n = 4,025$) and ending with the final sampling frame for the PTA study ($n = 2,694$).

Table 1. Sample Attrition from the Collaborative Perinatal Project to the Pathways to Adulthood Study

	Total	Percent
Total JHCPP Sample	4025	100
Died during neonatal period	122	3
Died before month 4 exam	30	0.7
Died after 4 month exam	35	0.09
Followed in CPP	3838	100
Born in 1959	427	11.1
Did not complete 7 or 8 year exams	404	10.5
Eligible for PTA	3007	1010
Pilot Study	60	2
Conflict of interest	7	0.2
Omitted replicate	247	8.2
Final Eligible Sample	2694	

Source: Hardy and Shapiro, 1999

Given the over 20 year lag between the end of the CPP and the initiation of PTA study, it is expected that sample attrition will be problematic. In fact, one of the biggest drawbacks of any prospective study design is selective attrition (Scott and Alwin 1998), which can lead to biased findings. According to Hardy and colleagues (1998), of the 3,006 children whose mothers entered the original CPP study after January 1, 1960 and who had completed the age 7 or age 8 evaluation, 312 were eliminated for budgetary reasons. Of the 2,694 remaining G2 respondents, 474 could not be located and 28 were located but not interviewed because they were deceased, out of the country, or incapable of completing the interview. Of the 2,192 fieldable interviews 135 refused to participate in the study and 157 had incomplete interviews. Data for an additional 142 G2 respondents were collected by interviewing their relatives, because the respondent was either deceased or unavailable for an interview. This leaves 1,758 of the eligible 2,694

G2 respondents with completed interviews. (See table 2, reproduced from Hardy and Shapiro 1999.)

Table 2. Sample Attrition for Respondents (G2) and Their Mothers (G1) Eligible for the Pathways to Adulthood Study

	G1	G2
Sample eligible for PTA	2306	2694
Not located	292	474
Located	2014	2220
Not fieldable		
Out of country/incapable	17	11
Deceased, no data	69	17
Fieldable sample	1928	2192
In field end of follow-up	58	157
Refused	151	135
Absent child interview	n/a	71
Deceased with data	167	71
Full interview	1552	1758
Response rate (known outcome) ^a	77.90%	71.40%
Response rate (full interview) ^b	67.30%	65.30%

^a The response rate for known outcomes was calculated using the following formula:

$$(\# \text{ located} / \text{eligible sample}) \times (\# \text{ completed} / \text{fieldable sample}) \times 100.$$

^b The response rate for the full interviews was calculated using the following formula:

$$(\# \text{ completed} / \text{eligible sample}).$$

Source: Hardy and Shapiro 1999

The G2 respondents who could not be located for the 1992 follow-up interviews differed from those who were located in several ways (Hardy et al. 1998). First, their mothers were significantly younger, more poorly educated, less likely to be married, and more likely to be poor at the time of their children's births. Second, the children who were located as adults were more likely to be female, have IQ scores 90 or higher as measured at age 7, and reading skills at or above grade level at age 8. There were no significant differences by race ($t = .171$, d.f. = 2,692, $p > .05$) or negative emotionality and low constraint ($t = 1.114$, d.f. = 2,677, $p > .05$). Those children who were located but

declined to participate in the study did not differ from those who did complete the interview (Hardy et al. 1998).

Listwise deletion of individuals who were missing data on observed variables further reduced the sample size by 288 individuals to 1,468 for the offending analysis and 1,436 for the substance use analysis. The 288 individuals with missing data did not differ significantly from those retained in the sample with regards to NE/LC ($t = .465$, d.f. = 1,774, $p > .05$), race ($t = .013$, d.f. = 1,756, $p > .05$), sex ($t = .361$, d.f. = 1,756, $p > .05$), or childhood ($t = .047$, d.f. = 1,756, $p > .05$) or adolescent ($t = .134$, d.f. = 1,756, $p > .05$) exposure to stressors. Individuals with missing data did differ from those with complete data on exposure to stressors in adulthood ($t = 2.440$, d.f. = 1,756, $p < .05$) with those in the former group exposed to a greater number of stressors on average than those in the latter group.

Because of selective attrition, individuals who, based upon childhood characteristics, are more likely to be offenders are also more likely to be excluded from the follow-up sample. This will have the unfortunate effect of dampening the variability in the independent and dependent variables possibly biasing the parameter estimates. The strategy for dealing with the bias due to attrition is discussed in the Missing Data section of the Analytic Strategy.

SAMPLE DESCRIPTION

Descriptive statistics for the samples used in the analysis as well as the entire follow-up sample can be found in table 3. In the description here, I focus on the sample included in the offending analysis, however, the sample used in the substance abuse

analysis varies only slightly. All individuals in the offending analysis were used in the substance abuse analysis minus those few individuals who were missing data on depression or substance use.

The majority of the mothers who participated in the CPP were African American (81.5 percent) with the remainder identified as white. Many of them (27.9 percent) had completed no more than eighth grade and only 30.2 percent had earned their high school diploma. As might be expected given the sampling frame, the women were relatively poor. Forty six percent lived below the poverty line, yet only 8.8 percent were receiving public assistance. Few of the women (10.3 percent) were employed at the time of their child's birth; however, the majority (72.6 percent) had worked in the past. Most of the women were married with 29.9 percent single at the birth of the study child.

The black women in the sample were very much like other black women delivering babies in the 1960s in Baltimore in terms of age, education, parity, and marital status. The white women in the sample, however, were very different from other Caucasians giving birth in Baltimore at the time of the study. They were younger, less educated, and had higher rates of infant mortality and low birth weight. Hardy and colleagues (1998, 1225) concluded that "They were more like the Black women than White Baltimore women, in general. To our knowledge, no other longitudinal study has a population that includes groups of Black and White families living in the same inner-city areas under similar socioeconomic conditions." Although the black and white participants lived within the same neighborhoods and attended the same schools, within the neighborhoods they lived in racially segregated pockets (Hardy et al. 1998).

The children differ in many respects from their mothers. First, they were better educated with 70.4 percent having completed twelfth grade. Most (68.9 percent) were employed at the time of the interview, and 27.8 percent had received public assistance as an adult. Only 32.6 percent were married at the time of the interview. In comparison, almost 70 percent of their mothers were married at the birth of their child.

Table 3. Sample Description

	Offending Sample		Substance Use Sample		Full Follow-Up Sample		
	<i>f</i>	Percent	<i>f</i>	Percent	<i>f</i>	Percent	<i>n</i>
Respondents^a							
Race							
Black	1197	81.54	1169	81.42	1434	81.57	1758
White	271	18.46	267	18.58	324	18.43	1758
Sex							
Male	670	45.64	655	45.58	807	45.90	1758
Female	798	54.36	781	54.42	951	54.10	1758
Education							
8th grade or less	62	4.23	57	3.97	75	4.27	1756
Some high school	373	25.35	362	25.21	459	26.14	1756
High school degree	1033	70.42	1016	70.82	1222	69.59	1756
Employment							
Never worked	26	1.77	26	1.81	41	2.33	1758
Worked at time of interview	1011	68.87	1001	69.94	1200	68.26	1758
Worked in past but not at time of interview	431	29.36	406	28.25	517	29.41	1758
On welfare as an adult	407	27.80	401	27.96	509	29.05	1752
	<i>n</i>	1468		1436			
Mothers of Respondents^b							
Under age 18 at 1st birth	498	39.62	487	39.50	598	40.05	1493
Single	376	29.91	367	29.76	455	31.19	1459
Father under age 18 at birth	64	5.09	64	5.19	75	5.21	1439
Education							
8th grade or less	351	27.92	343	27.82	428	29.36	1458
Some high school	527	41.92	514	41.69	606	41.56	1458
High school degree	379	30.15	376	30.49	424	29.08	1458
Received public assistance	111	8.83	109	8.84	127	8.78	1447
Employment							
Never worked	216	17.18	212	17.19	274	18.77	1460
Worked at birth of child	129	10.26	129	10.46	148	10.14	1460
Worked in past but not at birth of child	912	72.55	892	72.34	1038	71.10	1460
	<i>n</i>	1257		1233			

^a Measured at Pathways to Adulthood interview

^b Measured at birth of child

The majority (64 percent) of G2 respondents interviewed in the PTA study still resided in Baltimore City. They lived mainly in East and West Baltimore, which are two of the poorest areas in the city (Hardy and Shapiro 1999). An additional 26 percent of the sample resided in other places in Maryland, with 17 percent of the total sample living in the counties surrounding the city. The remaining 10 percent had moved out of the state.

HISTORICAL CONTEXT

The CPP and PTA data were gathered during a period of great change in Baltimore. At the commencement of the CPP in the 1960s, Baltimore neighborhoods were relatively safe and stable (Hardy and Shapiro 1999). Most families lived in small row houses, but there were some garden-style and low rise housing projects. Because there were few playgrounds or open spaces, most children played in the street. Blacks and whites lived in the same neighborhoods and therefore attended the same schools; however, within neighborhoods there was racial segregation with whites living in separate enclaves.

Families were large by today's standards and extended kin often lived together (Hardy and Shapiro 1999). Unemployment was relatively low compared to the inner city today. Men were employed in low-level jobs in industry, shipyards, and defense plants, while women worked as domestics, housekeepers, or clerks (Hardy and Shapiro 1999).

Beginning in the late 1960s, industry and manufacturing, the main source of stable employment for men, began to move out of Baltimore City. Residents who could afford to leave the city did so, resulting in neighborhoods that were relatively homogenous in terms of poverty and disadvantage (Hardy and Shapiro 1999, Wilson 1987), and leading to increased social isolation for those individuals who remained. Neighborhood shops and businesses closed and housing stock deteriorated. In addition, the number of female headed households increased as did teen pregnancy, the spread of sexually transmitted diseases, drugs, crime, and substance abuse.

VARIABLES

As described above, the measures used in this dissertation were collected from multiple sources, including interviews with the respondents and their mothers and observations recorded by trained psychologists. The variables described below draw on reports from all three of these sources. Note that in the subsequent text, childhood refers to the period from birth through age 8, adolescence covers ages 9 through 17, and adulthood ranges from age 18 through the interview date.¹⁹

The next section describes the observed variables used to create each of the latent constructs as well as variables that are composed of a single observed variable (e.g., exposure to stressors). Exact item wording, frequencies, factor loadings, and scale reliabilities can be found in tables 4 and 5. The process used to create the latent variables as well as the reliabilities for the scales are described in the discussion of the measurement model in the Results section.

DELINQUENCY AND OFFENDING

Delinquency and offending were measured as latent constructs,²⁰ each composed of dichotomous observed indicators which were coded 1 if the respondent engaged in the behavior more than one time and 0 otherwise. Because information on the age at which the respondent first and last engaged in the behavior was only recorded for individuals who engaged in the behavior more than once, only repeat offenders are captured by these

¹⁹ I recognize that the terms childhood, adolescence, and adulthood do not map onto these age ranges precisely. For example, when describing the stages of psychosocial development Erikson (1950) defines adolescence as occurring between the ages of 11 to 18 years of age, and divides the period I call childhood into infancy, the younger years, and early and middle childhood. However, because of the way some of the questions were asked, I cannot divide the data to correspond to these periods of the life course.

²⁰ The term delinquency will be used to refer to illegal behavior engaged in before age 18, while offending is reserved for the illegal behavior of those age 18 or older.

indicators. Most of the questions were adapted from the Diagnostic Interview Schedule, Version III (see Robins et al. 1981) or the National Longitudinal Study of Youth.

Measuring deviance over the life span can be difficult because it takes different forms at different ages. In this sense, deviance exhibits heterotypic, versus homotypic continuity. Drawing on Loeber and Hay (1997), LeBlanc (2006, 213) describes the progression of problem behavior from infancy to adulthood:

During infancy it takes the form of authority conflicts and physical aggression (Loeber and Hay 1997). During childhood, these behaviors change in seriousness and diversity and, in addition, the authority conflict manifests itself at school and covert behaviors are added (Loeber and Hay 1997). With adolescence, the syndrome is diversified in terms of covert, overt, and authority conflict, and reckless behaviors are added....During adulthood some criminal behaviors gradually stop and new forms of offending are introduced (tax evasion and family violence), school authority conflict is replaced by problems at work, and there are new forms of reckless behavior (compulsive gambling).

Recognizing that antisocial behavior is age-graded, the delinquency and offending measures contain different, age-graded items. The delinquency construct includes information on whether or not respondents engaged in the following behaviors before age 18: played hookey, got in trouble at school for misbehaving, carried a weapon, got in trouble at school or work for fighting, stole something worth \$50 or more, hit or seriously threatened to hit someone, sold drugs, and took a vehicle for a ride without the owner's permission.²¹ Approximately 80 percent of the sample ($n = 1,404$) engaged in at least

²¹ Information is also available on prostitution, but this behavior was dropped from the delinquency measure because of its low prevalence ($n = 10$).

one of these acts between ages 8 and 18. The mean number of behaviors engaged in was 1.85 (s.d. = 1.58). As might be expected, the most commonly reported behaviors were the least serious: playing hookey (66.9 percent; $n = 982$), getting in trouble at school or work for fighting (40.9 percent; $n = 601$), hitting or threatening to hit someone (29.2 percent; $n = 428$), and misbehaving at school (21.5 percent; $n = 316$).

The measure of adult offending captures involvement in six different illegal behaviors including carrying a weapon, theft of something worth \$50 or more, hitting or seriously threatening to hit someone, drug dealing, motor vehicle theft, and prostitution. In comparison to delinquency, offending was less prevalent with approximately 41 percent of the respondents ($n = 605$) reporting that they had committed one of these offenses as an adult.²² The average number of illicit behaviors engaged in was .75 (s.d. = 1.15). In adulthood, the most common offenses were hitting or threatening to hit someone (30.9 percent; $n = 453$); carrying a weapon (19.1 percent; $n = 281$); and selling drugs (11.9 percent; $n = 175$).²³

²² When the delinquency and offending constructs are constrained to contain the same five items (theft, hitting or seriously threatening to hit someone, drug dealing, and motor vehicle theft), the percentage of respondents who reported engaging in offending (48.88 percent, $n = 601$) is higher than for delinquency (35.51 percent, $n = 522$). This reversal is due to the larger number of less serious behaviors included in the delinquency measure. (See also footnote 24.)

²³ The decision was made not to weight the items according to seriousness for practical reasons. The questions were too general to ascertain the seriousness of the behavior as scored by Wolfgang and colleagues (1985). For example, three different severity scores could be assigned to selling drugs depending on whether the respondent sold marijuana (8.5), barbiturates (10.3), or heroin (20.6). In addition, determining a severity score for "hit or threatened to hit" would be exceedingly difficult without additional information on the circumstances surrounding the incident. Instead of weighting the variables, the analysis was rerun excluding the more minor offenses that were only available in the adolescent measure (playing hookey, getting in trouble at work or school for fighting, and getting in trouble at school for misbehaving). These results were substantively the same as those using all delinquency items; therefore, I only report the findings that were obtained using the full adolescent delinquency measure.

ADOLESCENT AND PAST YEAR SUBSTANCE USE

Extensive information about licit and illicit substance use was collected from respondents at the time of the PTA interview. With regard to illicit drugs, respondents were asked to report on their use of the following substances: marijuana, cocaine, heroin, methadone, other opiates, and inhalants (glue, gasoline, nitrous oxide, amyl nitrite, and poppers).²⁴ The exact item wordings, frequencies, and factor loadings for these items are located in table 4. If a respondent had taken any of these drugs more than five times, they were asked to report more detailed information about their usage, including the age they first used the drug and when they most recently used the drug.²⁵ In addition, they were specifically asked if they had used the drug in the past year.²⁶ It is this past year drug use that serves as one of the dependent variables in the substance use analysis.

Adolescent and past year substance use are measured as latent variables.

Adolescent substance use is composed of five dichotomous indicators coded 1 if the respondent used a given drug before age 18 and 0 otherwise. The drugs covered by this measure include marijuana, cocaine, heroin, other opiates, and inhalants. Approximately 40 percent ($n = 572$) of the sample reported using any of these drugs before the age of 18 with 35.6 percent using only one type of drug. The vast majority of substance using adolescents reported smoking marijuana (39.4 percent, $n = 572$), with the second most prevalent drug being cocaine (2.8 percent; $n = 40$).

²⁴ Alcohol use is not considered here because in these data it is not possible to disentangle periods of normative use from abuse.

²⁵ Because only individuals who used a given substance five or more times were asked to report the ages at which they first and last used the drug, those who merely experimented with illicit substances are coded as non-users in the data.

²⁶ The ideal measure of substance use would take into account both the number of different drugs used as well as the frequency of drug use. However, the instrument does not provide information on the frequency of drug use. The dataset does contain questions tapping into drug addiction, but because there is no information about the timing of these dependency problems, they cannot be incorporated into a time specific measure of substance use.

Past year drug use is measured with the same indicators, except inhalant use was replaced with illicit methadone use.²⁷ As would be expected given the shortened reference period, past year drug use was less prevalent than substance use in adolescence. Approximately 15 percent ($n = 220$) reported using illicit drugs at least once in the past year, with 10.0 percent using only one type of drug and 5.4 percent using 2 or more different drug types. Again, marijuana use was the most prevalent (10.7 percent; $n = 153$) followed by cocaine (6.9 percent; $n = 99$) and heroin (4.6 percent; $n = 66$).

Table 4. Descriptive Statistics and Factor Loadings for Delinquency, Offending, and Adolescent and Past Year Substance Use

Item Wording	<i>f</i>	Percent	Standardized Factor Loading
Adolescent Delinquency ($n = 1,468$)			
When you were in grade school or in high school, did you ever play hookey, that is, skip a full day of school without a real excuse?	982	66.89	0.511
Did you frequently get into trouble with the teacher or principal for misbehaving in grade school or high school?	316	21.53	0.584
Did you ever get into trouble at school or work for fighting?	600	40.87	0.510
Have you taken something not belonging to you worth \$50 or more?	111	7.56	0.832
Have you hit or seriously threatened to hit someone?	428	29.16	0.614
Have you sold drugs such as marijuana, heroin, crack or cocaine?	80	5.45	0.774
Have you taken a vehicle for a ride or driven it without the owner's permission?	56	3.81	0.684
Have you carried a weapon such as a gun or a switchblade knife?	150	10.22	0.796
Scale reliability	0.6431		
Adult Offending ($n = 1,468$)			
Have you taken something not belonging to you worth \$50 or more?	126	8.58	0.817
Have you hit or seriously threatened to hit someone?	453	30.86	0.697
Have you sold drugs such as marijuana, heroin, crack or cocaine?	175	11.92	0.864
Have you taken a vehicle for a ride or driven it without the owner's permission?	35	2.38	0.675
Have you carried a weapon such as a gun or a switchblade knife?	281	19.14	0.805
Have you traded sex for money, food, drugs, or other things?	39	2.66	0.657
Scale reliability	0.6572		
Adolescent Substance Use ($n = 1,436$)			
Have you ever used...			
Marijuana, hashish, pot, grass	572	39.38	0.697
Cocaine, coke, crack	40	2.79	0.852
Heroin	19	1.32	0.742
Opiates other than heroin or methadone (codeine, demerol, morphine, darvon, opium)	11	0.77	0.883
Glue, gasoline, nitrous oxide, amyl nitrite, poppers	18	1.25	0.752
Scale reliability	0.3429		
Adult Past Year Substance Use ($n = 1,436$)			
In the past year have you used...			
Marijuana, hashish, pot, grass	153	10.65	0.631
Cocaine, coke, crack	99	6.89	0.905
Heroin	66	4.59	0.949
Opiates other than heroin or methadone (codeine, demerol, morphine, darvon, opium)	13	0.90	0.877
Methadone	6	42.00	0.891
Scale reliability	0.6097		

²⁷ Inhalant use was omitted from this measure because it was used by only two respondents in the past year.

EXPOSURE TO STRESSORS

There is no consensus regarding the single best way to measure stressors and the resulting strain. Some argue that whether an event or situation produces strain is highly dependent on the context in which it occurs and, therefore, only perceptual measures of stressors can be used. Lazarus (1986) and others aver that the psychosocial situation, or the environment as perceived and reacted to, not objective experiences are the key to understanding stress processes. Therefore, two people in the same situation but with different beliefs, values, and commitments may differ in their appraisals of the stressfulness of the situation. Others disagree and suggest that it is not necessary to measure the emotional impact or the extent of life change associated with events. Some empirical evidence does indeed support this view. For example, Van Os, Park, and Jones (2001) found that in a community sample, a count of the number of life events that occurred was nearly perfectly correlated with scores that took into account the emotional impact and life change caused by these events. Moreover, many researchers, like Dohrenwend (1979, 8), argue that asking respondents to subjectively rate the impact of stressful events “is virtually guaranteed” to lead to confounding between measures of stressors and the outcome.

Similarly, using subjective measures of stressors may lead to reporting bias due to the confounding of individual traits with the reporting of life events (Kendler, Gardner, and Prescott 2003). However, some studies suggest that the effects of reporting bias for negative life events are small if non-existent (Kendler, Gardner, and Prescott 2003) and that bias is more of a concern when individuals provide subjective judgments. The measures of stressors used in the current study include both objective and perceptual

indicators in an attempt to strike a balance between the two sides of the debate. For example, dissatisfaction with work, disjunction between expectations and aspirations, and decline in the financial situation of the household over the last five years were included as subjective stressors, while death of a spouse, homelessness, and receipt of welfare were included as objectively stressful experiences.²⁸

Further complicating the measurement of strain is disagreement over what type of stressors should be examined. One camp of researchers has suggested studying only stressors that are exogenous to the problem behavior of interest, again to prevent confounding the independent variable of interest—stressors—with the dependent variable—illegal behavior; however, this is also problematic. First, many stressful events preceding problem behavior are not entirely independent of individuals' actions or control (McLean and McQuaid 1994) and many of the types of strain that Agnew (2001) lists as criminogenic tend to be at least partially attributable to individuals' own behaviors or choices. Second, omitting stressors that result from a person's own actions ignores the fact that individuals shape their environments, including their exposure to stressors, through their actions. This is contrary to the life-course perspective, which gives primacy to agency. Finally, it is very difficult to classify events as exogenous or endogenous (Kim et al. 2003), especially without information on the context of the situation. For these reasons, stressors that may be endogenous to criminal behavior are not excluded from the analysis.

A less debated finding in the stress literature is that it is the accumulation of a number of stressors that is most detrimental to human functioning (for a review see

²⁸ An objective strain is one that is "disliked by most members of a given group" (Agnew 2001, 320). This does not mean that every person will necessarily find these events stressful, but that they are considered stress producing for most people.

Thoits 1983). Agnew (1992, 2001) argues that stressors have a cumulative negative impact on individuals, with strain more likely to result in offending when stressors accumulate over time and are clustered together. For this reason, exposure to stressors is commonly measured as a count of the number of negative life events an individual has experienced in a given period of time.²⁹ Although most other key variables are measured as latent constructs, factor analysis was not used to create measures of stressor exposure. It is generally inappropriate to measure stressor exposure as a latent construct because the types of events typically included in studies—such as deaths, accidents, injuries, moving, illness, divorce or separation, burglaries or robberies, worries and crises arising from work, spousal discord, and problems with family or children—tend to be relatively independent from one another (Van Os and Jones 1999). This is evidenced in the data; when the indicators of stressor exposure were included in one scale, the reliabilities were quite low. The reliabilities (alphas) for exposure to stressors in childhood, adolescence, and adulthood were .1252, .4897, and .4864, respectively.

For these reasons, I measured exposure to stressors as a count of the number of stressful experiences each individual has experienced during the developmental period of interest. Because this count was positively skewed, these counts were transformed by adding one and taking the square root.³⁰ Finally, to ease interpretation of the results, they were grand mean centered by subtracting each individual's score from the overall mean.

²⁹ A problem that arises from measuring stressor exposure using a count of negative life events is that chronic strain cannot be distinguished from discrete events. It is likely that many of these events have lasting ramifications that are not captured by this measure. Future analysis should attempt to more carefully distinguish chronic strain from negative life events, but this is beyond the scope of the current analysis.

³⁰ An examination of the data indicated that this transformation produced variables that more approximated the normal distribution than the more commonly seen log transformation. However, skew tests still differ significantly from the normal distribution. I deal with this problem by estimating the models using weighted least squares parameter estimates with robust standard errors and mean- and error-variance adjusted χ^2 test statistics.

Ideally, exposure to stressors would be left as count variables and analyzed using Poisson or negative binomial models. Unfortunately, the complexity of the model made convergence an issue when exposure to stressors was left as a count measure.³¹

Following the life-course perspective, age-appropriate stressors were examined. This is particularly important because most tests of GST focus on adolescents, and therefore less is known about stressors in adulthood. Table 5 contains a complete list of the stressors included in the childhood, adolescent, and adult measures. Also included here is the percent of respondents experiencing each type of stressor as well as the data source from which each indicator was taken.

Although most of the stressors included in these measures are standard in the stress literature, a few require further explanation either because they are less familiar to criminologists or because they deviate from the measures commonly used in literature.³² I comment on some of these here.

Death of Mother—Ideally, I would have information on the death of either parent, however the CPP data were collected at a time when child health was viewed as primarily the responsibility of mothers. For this reason, little information was collected on the fathers and I am unable to ascertain paternal death before age 8. In the measure of exposure to stressors in adolescence, paternal death is captured with death of household member (if the father resided with G2), however, it is not captured in the adult measure of stressor exposure.

³¹ It is important to note that the transformation does not solve the issue of censoring. This is probably most problematic for the childhood stressor measure, which contains a smaller number of items than the other measures of stressors. Again, future work should replicate this analysis using Poisson or negative binomial models.

³² Although there are validated instruments used to collect measures of stressors, these were not utilized in either the CPP or PTA study.

Problems with Crime in the Neighborhood and Lack of Security from Break-ins—

Victimization is one of the types of strain that Agnew avers is most conducive to offending (2001); not only is it generally high in magnitude, but it also is viewed as unjust. Because the data do not include a measure of victimization, this item partially serves as a proxy for personal victimization. However, more importantly, this variable also captures vicarious strain, that is, strain experienced by others around the individual (Agnew 2002). Vicarious strain is most likely to lead to illicit behavior when it occurs to family, friends, or neighbors, is physically proximate, impacts the groups to which one belongs, and has a high probability of affecting one in the future (Agnew 2002). These traits are likely to characterize neighborhood crime.

*Lack of Space or Privacy—*Noise crowding and low quality housing have been associated with psychological distress in children (Evans 2001). This measure, therefore, is intended to capture strain arising from exposure to noxious environmental stimuli.

Stressors in Childhood (Birth to Age 8)

Because the focus of the CPP was primarily on perinatal and childhood health, it contained fewer measures of life events and circumstances than the PTA study. For this reason, the measure of childhood exposure to stressors is somewhat limited. This scale contains six items, some of which were taken from prospective reports in the CPP and others from retrospective reports collected as part of the PTA study. About half of the items, including death of mother, sexual abuse, and activity limiting illness or injury, were relatively rare, with each occurring in less than 4 percent of the sample. The other stressors (repeated grade, parental divorce, and moving more than three times) were more

common, with 10 to 15 percent of the sample reporting their occurrence. The average number of childhood stressors experienced was .464 (s.d. = .678), while the median and mode were both 0 and the range was 4.

Stressors in Adolescence (Ages 9-17)

This measure is a transformed count of the number of stressors that the respondent experienced between the ages of 9 and 17. Eight of the 22 items come from the maternal interviews in which the mothers were asked to report household problems that existed while the respondent was in adolescence. Two additional stressors (homelessness and moving) came from data collected with a life event calendar, while the remaining stressors came from the retrospective PTA interview with the children. All items are dichotomous and are coded 1 if the respondent experienced the stressors and 0 otherwise.³³

The number of stressors experienced in adolescence ranges from 0 to 10, with an average of 2.32 (s.d. = 1.86), a median of 2 and modes of 1 and 2.³⁴ The most common stressors were problems with crime in the neighborhood ($n = 478$; 32.5 percent) followed by serious illness or injury of a household member ($n = 354$; 24.1 percent) and repeating a grade ($n = 340$; 23.1 percent). These were followed by dropping out of high school ($n = 294$; 20.0 percent) and lack of privacy or space in the household ($n = 273$; 18.6 percent).

³³ The mothers of 243 of the respondents in the sample were not interviewed. Therefore, these individuals are missing information on eight of the stressors. These cases were included in the analysis, however, they are scored as not experiencing the eight stressors.

³⁴ The distribution is bimodal.

Table 5. Frequencies for Childhood, Adolescent, and Adulthood Stressors

	<i>f</i>	Percent	Variable Source ^a
Childhood			
Death of Mother	16	1.09	CPP
Sexual Abuse	40	2.72	PTA
Illness or injury that limits activity	46	3.13	PTA
Repeated grade	157	10.68	PTA
Parents got divorced	196	13.33	CPP
Moved > 3 times	227	15.44	CPP
Adolescent Stressors			
Death of mother	31	2.11	PTA
Death of household member (excluding mother)	148	10.07	G1 Interview
Repeated a grade	340	23.13	PTA
Dropped out of high school	294	20	PTA
Wanted to attend college but couldn't	25	1.7	PTA
Sexually abused	77	5.24	PTA
Became a teen parent	145	9.86	PTA
Unemployed	40	2.72	Calendar
Not enough money for necessities	249	16.94	PTA
Moved > 3 times	239	16.26	PTA
Homeless	190	12.93	Calendar
Beaten by parents for breaking rules	249	16.94	PTA
Lack of privacy or space in household	273	18.57	PTA
Lack of security in household	145	9.86	PTA
Problems with crime in the neighborhoods	478	32.52	PTA
Chronic illness of household member	354	24.08	G1 Interview
Trouble with friends or relatives in the household	51	3.47	G1 Interview
Trouble with other children in the household	76	5.17	G1 Interview
Trouble with men in the household	69	4.69	G1 Interview
Trouble with women in the household	12	0.82	G1 Interview
Someone in the household had work difficulties	89	6.05	G1 Interview
Other trouble in the household	56	3.81	G1 Interview

Table 5 Continued. Frequencies for Childhood, Adolescent, and Adulthood Stressors

	<i>f</i>	Percent	Variable Source ^a
Adult Stressors			
Death of mother	75	5.1	PTA
Death of spouse or live-in partner	4	0.27	Calendar
Death of child	20	1.36	PTA
Sexually abused	20	1.37	PTA
Engaged in combat	33	2.24	PTA
Separated from spouse or live-in partner	646	43.95	Calendar
Got divorced	126	8.57	Calendar
Moved > 5 times	288	19.59	Calendar
Homeless	33	2.24	Calendar
Lack of privacy or space in household	262	17.82	PTA
Lack of security in household	103	7.01	PTA
Problems with crime in the neighborhoods	460	31.29	PTA
Had a child who was abnormal at birth	93	6.33	PTA
Has child with behavior problem (in trouble at school, with the police, teen parent, etc)	499	33.95	PTA
Forced to leave school (financial reasons, bad grades, expelled, etc)	230	15.65	PTA
Dissatisfied with work	87	5.92	PTA
Unemployed	391	26.6	Calendar
On welfare	407	27.69	Calendar
Difficulty obtaining medical care	208	14.15	PTA
Health interferes with activities	181	12.31	PTA
Aspirations exceed expectations	643	43.74	PTA
Worse off than mother or father when they were your same age	149	10.14	PTA
Financial situation of household has been getting worse over the past 5 years	201	13.67	PTA
	<i>n</i> 1468		

^a CPP = Collaborative Perinatal Project

PTA = Pathways to Adulthood Study

Calendar = Data collected using a life event calendar as part of the PTA respondent (G2) interview

G1 interview = Pathways to Adulthood interview with mother (G1)

Stressors in Adulthood (18-33)

This measure contains 23 items, some of which overlap with those included in the childhood and adolescent measures (e.g., sexual abuse, death of mother, homelessness, lack of household privacy) and others of which are age-graded and apply more specifically to adulthood (e.g., dissatisfaction with work, combat experience, divorce or separation from a partner or spouse, problems with children). Also included in this measure are perceptual measures of stressors, some of which tap into classic strain measures. For example, one item measures a disjunction between aspirations and expectations (Cloward and Ohlin 1960) while two others capture perceived downward trajectories—both intragenerational (financial situation has been getting worse over past 5 years) and intergenerational (worse off than mother or father when they were your age)—in life satisfaction.

The measure of exposure to adult stressors ranges from 0 to 12 with a median of 3, a mode of 2, and a mean of 2.32 (s.d. = 1.86). The most common stressors were separation from a spouse or partner and aspirations exceeding expectations ($n = 646$ and $n = 643$, respectively), which were both reported by approximately 44 percent of the sample.

NEGATIVE EMOTIONALITY AND LOW CONSTRAINT

Unlike today, at the inception of the CPP in the late 1950s and early 1960s, there were no universally accepted standardized tools for measuring temperamental differences (Caspi et al. 1995). Therefore, NE and LC were derived from observations by trained psychologists during each child's age 7 assessment. Examiners administered a set of

standardized cognitive and motor tests to the children after which they rated the children's behavior. Behavioral ratings were measured on a five point scale with a separate code used when the children's behavior was too variable to assess.³⁵

Observational measures capturing NE/LC were recorded at ages 4 and 7, however the present study utilizes observations from the age 7 evaluation only. The age 7 observations are more likely to capture personality characteristics that persist into adolescence and adulthood because, although there is general agreement that personality may change over time, its stability increases with age (Caspi and Roberts 2001; Roberts and Del Vecchio 2000). The lengthy lapse between measures of individual differences and stressors and offending is not ideal, however, it is also not unprecedented. Caspi and Silva (1995) found that observer ratings of children's behavior taken at age 3 were significantly associated with self-reported personality at age 18 (Caspi and Silva 1995).³⁶ Moreover, the moderating effects of neuroticism measured at age 16 have been associated with an enhanced effect of significant life events on depression at ages 36 and 43 (Van Os and Jones 1999) and Wright and colleagues (2001) found that traits measured in childhood moderated the effect of social bonds on offending 10 years later.

The CPP did not contain variables created to explicitly measure negative emotionality and low constraint, although many of the items capture aspects of these traits. The six indicators that best captured the various facets of NE and LC were chosen from a more complete set of observations measuring temperament. These items included

³⁵ There were very few instances where a child's behavior was too variable to assess and so these were coded as missing.

³⁶ In the CPP there was low to moderate stability in these traits from age 4 to age 7. The correlation between the four measures common to the age 4 and age 7 assessments was .171. This is relatively low, but recall that personality usually changes a good deal up until age 6 and then remains relatively stable until young adulthood (Roberts and Del Vecchio 2000).

volatility, overactivity, impulsivity, willfulness, negativism, and hostility (see table 6 for a more complete description). Because of the relative rarity of these characteristics in the sample, the five category scale on which they were originally measured was collapsed into two categories so that anyone who scored above average on that trait was scored as 1 and all others were scored 0.

Impulsivity and overactivity were selected because of their close association with low constraint. Willfulness is also associated with low constraint because it measures a lack of concern for the feelings and rights of others. Volatility captures the sudden mood changes associated with negative emotionality, while negativism and hostility personify the tendency to respond to events in an aggressive manner, stubbornness, and irritability that are associated with this trait.

The construction of this factor is described in more detail in the description of the measurement model for offending in the Results section. It should be noted here, however, that in the end the decision was made to measure NE and LC as one construct rather than as two separate measures. Exploratory factor analysis indicated that only two of the indicators—negativism and hostility—loaded on negative emotionality and both of these characteristics were relatively rare in the sample ($n = 59$ and $n = 17$, respectively). Other items were more prevalent including, overactivity, which was observed in about 10 percent of the sample, volatility ($n = 85$), and impulsivity ($n = 78$).

MATERNAL³⁷ CHARACTERISTICS

Parenting

One of the core tenets of the life-course paradigm is the interdependency between the lives of individuals and those of their family members and friends across the lifespan (Elder 1994). Agnew (1997) attributes stability in illicit behavior partially to the linkage between the lives of parents and their children. At the most basic level, there is an association between negative emotionality and low constraint in parents and their children, with parents passing on these traits to their children through their genes. In addition, individuals with high negative emotionality and low constraint may reproduce these traits in their children via poor parenting practices. Parents high in neuroticism tend to provide significantly less support and structure for their children than other parents, engage in more negative emotional interactions with their children, have lower sensitivity to their children's needs, and provide them with less cognitive stimulation (Belsky and Barends 2002; Belsky, Crnic, and Woodward 1995; Ellenbogen and Hodgins 2004).

Unfortunately, without direct measures of these parental traits in the data, the hereditary linkage cannot be examined. Data on parenting, however, was collected by trained observers during the course of each child's 8 month evaluation. At this assessment, each mother was evaluated on her responsiveness to her child's needs, her attitude toward her child's test performance, and the physical appearance of her child as well as her physical handling of her child. The mothers' level of self absorption (versus

³⁷ Conspicuously absent from the data, especially from the CPP, is detailed information about the respondents' fathers or male caretakers. Apparently, at the time of the initial data collection fathers were not viewed as integral to the health and wellbeing of their children.

attention to her child) was also noted. Although these variables were originally measured on a scale from 1 to 5, I recoded them as binary variables scored 1 if the mother was rated above average on parenting behaviors or characteristics associated with NE/LC and 0 otherwise. As a result, these dichotomous variables indicate whether or not a mother was self-absorbed, indifferent towards her child, unresponsive to her child's needs, handled her child roughly, and failed to take care of her child's appearance.

These parenting characteristics were relatively uncommon among the mothers in the sample (see table 6). Their prevalence in the sample ranged from 3.3 percent for self absorption ($n = 49$) to 6.4 for indifference to her child's test performance ($n = 94$). Given the testing situation, which is likely to elicit socially desirable behavior from parents, the short observation period (no more than a few hours), and the relative rarity of these negative behaviors, women who exhibited these characteristics are likely to represent the most extreme cases of poor parenting.

LOW SES

According to Agnew (1997, 2006) the second way in which parents contribute to continuity in their children's criminal behavior is through their class membership. According to the amplification hypothesis, regardless of temperament, individuals with a low SES will be more likely to experience stressors and more likely to react to these stressors with illegitimate coping mechanisms, such as delinquency and substance use. Illegitimate coping itself has negative consequences that can lead to more stressors and therefore more offending.

Low SES may also serve as a rough proxy for parental negative emotionality and low constraint. Parents high in neuroticism have significantly fewer years of education, lower income, poorer psychosocial functioning, less social support, ineffective coping strategies (less task-oriented coping and more emotion-focused and avoidant coping), poorer marital adjustment, more negative life events, and more verbal aggression toward their partner and children (Ellenbogen and Hodgins 2004).

In youth, class is generally determined by parental class membership, and I include a measure of the mother's SES at her child's birth. Low SES is measured as a latent variable using behaviors that distinguish this group from the mainstream poor (Ricketts and Sawhill 1988), including failing to complete high school, becoming a mother before age 18, receiving welfare, and being unwed at the birth of her child (see table 6). In addition, one paternal indicator of low SES, father was under age 18 at the time of his child's birth, was included.³⁸ Each one of these indicators is dichotomous, scored 1 if the parent possesses the characteristic and 0 if not.

The women in the sample were overwhelmingly undereducated. Over 70 percent ($n = 1,041$) had not obtained their high school degree by the time the study child was born and many of them (40.5 percent, $n = 596$) were under 18 when their first child was born. However, in terms of marriage and public assistance, these mothers were better off than their children would be in adulthood.

³⁸ Although unemployment is often used an indicator of low SES (Ricketts and Sawhill 1988), the decision was made not to include it here because a large number of the mothers had left the work force to have their child (see table 3).

DEPRESSION

One of the key tenets of GST is that negative emotions mediate the relationship between stressors and illegitimate coping. Offending and substance use are viewed as means of coping with the negative emotions brought about by stressors. The emotion given primacy in GST is anger, because it creates pressure for corrective action, reduces access to legitimate coping mechanisms, and leads to feelings of injustice (Agnew 2006). However, Agnew identifies other emotions, like depression and fear, that may link strain to offending. Neither the CPP nor the PTA includes measures of anger, but the PTA does contain measures of depression recorded at the time of the interview.³⁹

The measure of depression was adapted from the General Health Questionnaire (Goldberg and Hillier 1979). Depression is measured with seven categorical items which ask how much in the past few weeks have you: 1) been thinking of yourself as a worthless person, 2) felt that life is entirely hopeless, 3) felt that life isn't worth living, 4) found at times you couldn't do anything because your nerves were too bad, 5) found yourself wishing you were dead and away from it all, 6) thought of the possibility that you might do away with yourself, and 7) found that the idea of taking your own life kept coming into your mind. The first five of these items, are scored on a four point scale ranging from 0 ("not at all") to 3 ("much more than usual"). The last two items are scored on a four point scale ranging from "not at all scored" coded as 0 to "you definitely have thought of the possibility" scored as 3. (See table 6.)

³⁹ Negative emotions may be either trait based or state based. Trait based emotions are those that an individual has a tendency to experience, while state based emotions are those that an individual experiences at one moment in time (Agnew 2006). Although both types are important mediating mechanisms in the production of delinquency and offending (Agnew 2001), the measures used here are trait based because of data limitations. State based measures are less common because they require that respondents report their emotions at a given point in time. While it would be ideal to have both types of emotions, evidence indicates that the two are highly correlated (Capowich, Mazerolle, and Piquero 2001).

Table 6. Item Wording, Frequencies, Factor Loadings, and Scale Reliabilities for Negative Emotionality and Low Constraint, Parenting, Underclass Status, and Control Variables

	<i>f</i>	Percent	Standardized Factor Loading
Low Constraint/Negative Emotionality			
Volatility: Extreme instability of emotional responses; marked emotional lability	85	5.78	0.91
Overactivity: Unusual or extreme amount of activity and restlessness	147	10.00	0.866
Impulsivity: Behavior frequently or extremely impulsive; explosive and uncontrolled behavior	78	5.31	0.912
Willfulness: Quite forceful or extremely assertive, willful personality	27	1.84	0.976
Negativism: Extreme negativism; resistive to demands or directions a good deal of the time	59	4.02	0.686
Hostility: Very hostile, obstructive, unusual amounts of hostility present	17	1.16	0.77
Reliability	0.7441		
Parent Characteristics at G2 Birth (Low SES)			
Mother receiving public assistance	127	8.64	0.486
G1 < 18 at first birth	596	40.54	0.833
Father < 18	73	4.97	0.69
G1 lacks high school degree	1041	70.82	0.863
G1 single	397	27.01	0.443
Reliability	0.5579		
Parenting Observations at 8 Months			
Rough handling of child	51	3.47	0.713
Unresponsive to child's needs	89	6.05	0.825
Indifferent to child's test performance	94	6.39	0.938
Self absorbed	49	3.33	0.777
Child's appearance is unkept	72	4.90	0.638
Reliability	0.6394		
Depression			
Over the past few weeks, have you...			
Been thinking of yourself as a worthless person?			
Not at all	1,207	83.99	0.736
No more than usual	146	10.16	
Rather more than usual	52	3.62	
Much more than usual	32	2.23	
Felt that life is entirely hopeless?			
Not at all	1,285	89.42	0.841
No more than usual	102	7.10	
Rather more than usual	27	1.88	
Much more than usual	23	1.60	

Table 6 Continued. Item Wording, Frequencies, Factor Loadings, and Scale Reliabilities for Negative Emotionality and Low Constraint, Parenting, Underclass Status, and Control Variables

	<i>f</i>	Percent	Standardized Factor Loading
Felt that life isn't worth living?			0.884
Not at all	1,337	93.04	
No more than usual	71	4.94	
Rather more than usual	19	1.32	
Much more than usual	10	0.70	
Thought of the possibility that you might do away with yourself?			0.824
Definitely not	1,349	93.88	
You don't think so	48	3.34	
It crossed your mind	32	2.23	
You definitely have thought of the possibility	8	0.56	
Found at times you couldn't do anything because your nerves were too bad?			0.745
Not at all	1,213	84.41	
No more than usual	130	9.05	
Rather more than usual	60	4.18	
Much more than usual	34	2.37	
Found yourself wishing you were dead and away from it all?			0.901
Not at all	1,320	91.86	
No more than usual	71	4.94	
Rather more than usual	27	1.88	
Much more than usual	19	1.32	
Found that the idea of taking your own life kept coming into your mind?			0.779
Definitely not	1,332	92.69	
You don't think so	46	3.20	
It crossed your mind	50	3.48	
You definitely have thought of the possibility	9	0.63	
Reliability	0.842		
Control Variables			
Male	672	45.71	
Black	1,199	81.56	
	Mean	SD	
Quarter Century of Birth	1963.49	1.44	
	<i>n</i>	1468	

Abbreviation: G1 = generation one (mother of respondent)

The questions ask respondents to think about how they have been feeling over the past few weeks, but recall that the adult measures of stressors and offending refer to ages 18 to 27-33. Therefore, including measures of emotions that refer to the past few weeks creates problems with causal ordering. To alleviate this problem depression is only

included in the models examining past year substance use, and not offending. Although this does not solve the problem of causal ordering, it is at least mitigated.

ANALYTIC STRATEGY

Regression models are often used in social science research to assess causality. In regression, the relationship between an exogenous and endogenous variable can be calculated while holding the effects of control variables constant. However, the analysis at hand was not done using simple regression for several reasons. First, in several of the pathways proposed by Agnew, some measures serve as both independent and dependent variables. Although this issue can be worked around by estimating multiple regression equations and decomposing total effects into direct and indirect effects, it can become tedious, and more importantly does not allow for the simultaneous estimation of multiple pathways. A better option is to use path analysis to model the stability promoting mechanisms. Path analysis is based on regression but it allows for variables to simultaneously serve as predictors and outcomes. Path analysis, however, cannot solve the second issue: several of the constructs, including low constraint and delinquency are multi-faceted and therefore cannot be measured with a single indicator. Rather they are best captured with multiple items each of which captures a different aspect of the total construct. For example, NE/LC encompasses the characteristics of impulsivity, negativity, hyperactivity, reactivity, hostility, and willfulness, each which is measured with a separate indicator. Yet it is the convergence of these traits that exemplifies NE/LC. One common solution to this problem is to create a scale of the various indicators, and include this scale as a single variable in a regression equation. While this

is often done in practice, it ignores the reality that, in all likelihood, these characteristics are not perfectly measured. This is not an issue that can be resolved in standard regression or path analysis but can be dealt with using structural equation modeling (SEM).

Structural equation modeling is “a hypothesized pattern of directional and nondirectional linear relationships among a set of measured variables and latent variables” (MacCallum and Austin 2000, 202). Directional relationships are those that imply causality, while nondirectional relationships are correlations that indicate that there is an association between variables but the relationship is not necessarily causal. At the most basic level, the purpose of SEM is to account for the variation and covariation of observed variables (MacCallum and Austin 2000, 202). SEM, however, also enables researchers to test a priori specified and theory-driven hypothesized causal relationships by comparing a hypothesized model to the data and by providing information on the degree to which they are consistent with one another. In addition, unlike regression analysis, SEM provides for the simultaneous estimation of all model pathways.

SEM entails the development of a measurement model and a structural model, which are combined to form a hybrid model or a structural regression model (Kline 2005). The measurement component, which is primarily data-driven, can be thought of as a series of confirmatory factor analyses in which observed variables, called items or indicators, are used to create latent variables that represent abstract concepts of interest. The construction of latent variables provides researchers with a technique for taking measurement error into account. This process assumes that no one indicator perfectly measures the concept, but rather each one provides information about a portion of the

concept domain. The structural model, which is theory-driven, is analogous to a path model in that it summarizes the relationships among variables and may involve direct, indirect and total effects. However, unlike path models, structural models can include both latent and observed variables, rather than observed variables only.⁴⁰

Often times SEM is done in a maximum likelihood (ML) framework. ML estimation assumes that all indicators are continuous and distributed multivariate normal. Violations of this assumption can cause estimates of standard errors to be downwardly biased and χ^2 values to be overestimated. Many of the indicators in the models that were estimated were dichotomous or categorical and therefore ML estimation was inappropriate. In addition, it becomes computationally demanding to use ML to estimate models that have categorical indicators once more than four factors are included in the model (Muthén and Muthén 2006). For these reasons, weighted least squares parameter estimates with robust standard errors and mean- and error-variance adjusted χ^2 test statistics (WLSMV) were used (Muthén and Muthén 2006, 2007).

Using WLSMV has two major drawbacks. First, the degrees of freedom are not calculated the way they are with ML estimation. Instead, this calculation “is data dependent because it draws on the estimates, their derivatives, and the asymptotic covariance matrix of the sample statistics with the aim of choosing the degrees of freedom that gives a trustworthy chi-square-based p value” (Muthén 2000).⁴¹ An explanation of the estimation of the degrees of freedom is beyond the scope of this

⁴⁰ My model is classified as “partially latent” because it includes variables that are the single-indicator of a construct as well as multiple-indicator latent variables (Kline 2005, 211).

⁴¹ A disconcerting result of this is that sometimes when a model is rerun with one parameter fixed to 0, the degrees of freedom remain unchanged rather than decreasing by 1.

dissertation. Interested readers should see appendix 4, equation 10 of the MPlus technical appendix for more information.

It is also more difficult to compare nested models using a chi-square difference test when using WLSMV versus ML estimation. This is because unlike the results obtained with ML, the difference between the chi-square statistics of two nested models is not distributed as chi-square (Satorra and Bentler 1999). Therefore, the difference test statistic cannot be obtained by computing the difference between the chi-square statistics of two models and comparing them to the chi-square distribution with degrees of freedom equal to the difference in the number of parameters estimated in the nested models. Instead, a special feature of MPlus called DIFFTEST must be used to compare the nested models (Asparouhov and Muthén 2006).

OVERVIEW OF ANALYSIS

In the following section I outline the process through which the mechanisms hypothesized to lead to continuity in offending were tested. Each step was done separately for offending and past year substance use. In the Results section, I first present all the findings for delinquency and offending, followed by the results for adolescent and past year substance use.

Step 1: Specify the Measurement Model

The first step in the analysis was to specify the measurement models for offending and substance use. Each model included all the indicators and latent variables of interest and the latent variables and observed measures of stressors were free to covary. No

causal pathways were specified at this stage. This step of the analysis was essentially data driven and its purpose was to make sure the specified measures adequately described the data. At this stage, the error variances of factor indicators were also freed to covary based on modification indices and theory.

Step 2: Specify Full Causal Base Model

Once the measurement models were specified and it was determined that they adequately fit the data, correlations between variables were changed to causal arrows as specified by theory and the age at which the variables were measured (in order to maintain the proper causal ordering). These models were used to determine which hypotheses were supported by testing for direct and indirect effects and by fixing pathways to 0. Examining indirect effects is key to learning about the processes through which independent variables affect outcomes. For example, merely controlling for the effect of exposure to stressors when examining the relationship between temperament and criminal behavior tells us nothing about how these two constructs are related or the process through which NE/LC influences offending and drug use.

According to Baron and Kenny (1986), there are three necessary, but not sufficient, conditions that must be satisfied in order to claim a mediation effect exists: 1) the exogenous variable must have a significant relationship with the mediator; 2) the mediator must have a significant relationship with the dependent variable of interest; and 3) the relationship between the exogenous variable and the outcome must be attenuated when the mediator is added to the model. If any of these criteria do not hold, then there is no evidence to suggest that a mediation effect exists.

During this stage of the analysis, chi square difference tests were used to tease out which models were supported.⁴² Paths between variables were fixed to 0 based on the pathways hypothesized to be responsible for continuity. The fit of the constrained model was then compared to that of the unconstrained model using the DIFFTEST function of Mplus. This enabled me to determine if there was empirical support for each hypothesized path. Appendix A contains a description of various fit statistics that were used to assess models fit.

Step 3: Specify the Final Model

Based on the results from the full causal model, a final causal model was developed in which paths that did not differ significantly from 0 were constrained to be 0. This model was used to test for moderating effects and to check the robustness of the results (see appendix B).

Step 4: Exploring Moderating Effects

Once the final causal model was developed, the two moderating hypotheses were tested: 1) the effect of stressors on offending and substance use is stronger for individuals higher in NE/LC, and 2) the effect of stressors on offending differs depending on individuals' experience with stressors in childhood.

Originally, multigroup analysis was used to examine these interactions. This method provides a way to formally test whether the model fits better when the pathways

⁴² There are numerous indexes that assess how well a proposed model fits the data. The chi-square fit statistic is one of the more common of these and for categorical observed variables it is the only fit statistic that has been validated with simulation studies (Muthén and Muthén 2006).

in the model are constrained to be the same or allowed to vary across the groups of interest (i.e., high vs. low NE/LC and exposure to stressors in childhood vs. no exposure). Unfortunately, the data were too thin for this type of analysis and I would have needed to drop several indicators with low cell counts in order to test for these interactions. Therefore, interaction terms were created by multiplying the measures of stressor exposure by a dichotomous indicator of high or low NE/LC and a dichotomous indicator of high or low stressor exposure, respectively. These interaction terms were included in the models as predictors of criminal behavior.

MISSING DATA

As discussed above, the follow-up sample interviewed in the PTA study differed from that of the original CPP sample on many fronts. This may lead to biased estimates of parameters. Because individuals with characteristics associated with offending were less likely to be in the follow-up sample, one likely ramification of this is less variability in the outcomes of interest. While beyond the scope of the current study, future research should attempt to correct for possible bias by using sampling weights to adjust for selection into the sample.

In addition to selective sample attrition, missing data on individual items may also be problematic. For most of the variables, this was not an issue because missing data were minimal. However, for some variables, especially those measuring parenting and temperament, missing data were more prevalent. In the main analyses, listwise deletion was used to remove cases with any missing data. This method is most effective when less than 5 percent of the cases have missing data. The obvious drawback to listwise

deletion is that, in addition to possibly biasing results, it can lead to a significant loss of cases and consequently, a loss of statistical power. Despite these limitations, listwise deletion was used in the current study for two reasons. First, in order to compare nested models, the models must retain the same sample. With other types of missing data procedures, like pairwise deletion, the sample changes as a function of the variables included in the model. Second, because of my relatively large sample size, loss of statistical power was less of a concern.

Because listwise deletion reduced my sample size in the offending analysis by 290 cases and in the substance use analysis by 322 cases, the models were re-estimated using the MISSING command in Mplus to see if the loss of cases may have biased my findings. When the MISSING feature is enabled, Mplus uses all available data to estimate parameters. For example, if a case is missing information about the age of the father at his child's birth, which is one indicator of low SES, this case will remain in the analysis and the score for low SES will be calculated based on the other four indicators of this latent variable.⁴³

The results for the offending analysis using the MISSING function were substantively identical to those obtained using listwise deletion. For substance use, the effect of childhood stressors on adult stressors dropped from $\beta = .059$ to $\beta = .058$. This small change was enough to make this coefficient non-significant; however, given that the magnitude of the change was so small, this is not problematic. Together, these results

⁴³ Future analysis should use more sophisticated methods of dealing with missing data including multiple imputation with an EM algorithm or sampling weights.

provide some evidence that my original findings were not substantially impacted by using listwise deletion.⁴⁴

⁴⁴ This demonstration does not speak to bias that may result from sample attrition between the CPP and PTA.

CHAPTER IV: RESULTS

DELINQUENCY AND OFFENDING

MEASUREMENT MODEL

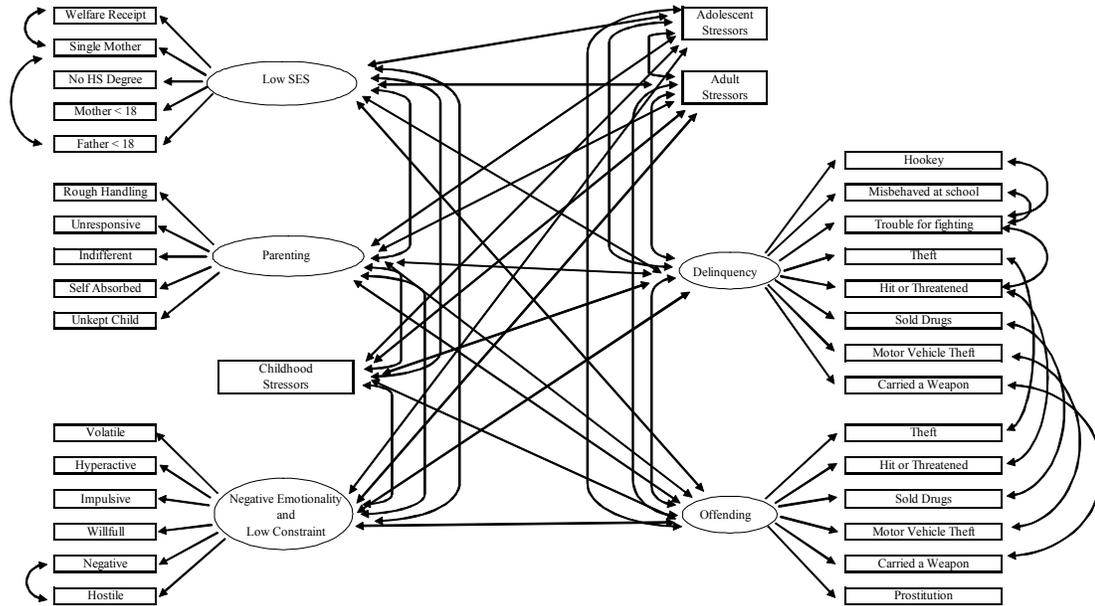
The first step in SEM is to create a measurement model, which specifies the relationship between the latent and observed variables and their errors. Ideally, the data would contain validated scales to measure key constructs. Although this was true for some factors, like depression, other latent variables needed to be constructed through a series of confirmatory factor analyses (CFA), which were informed by the results of prior exploratory factor analysis (EFA). The following process was used. First, indicators were chosen that represent the construct(s) of interest based on theory. Exploratory factor analysis was then used to assess if the indicators loaded together on one factor, or if they appeared to represent multiple constructs. CFA was then used to confirm that the indicators believed to measure the same construct loaded together and to model the covariation among the residuals of the observed indicators.⁴⁵ The measurement model then combined these multiple CFAs together into one model, controlling for race, age, and sex.

The final measurement model for offending is depicted in figure 5. Factor loadings are located in tables 4 (delinquency and offending) and 6 (parenting, low SES, NE/LC) and are interpreted as probit coefficients. The associations between variables are interpreted as covariances (unstandardized coefficients) or correlations (standardized

⁴⁵ It is not entirely appropriate to use CFA to confirm the results of EFA because EFA may capitalize on chance variation in the data. Rather it is best to validate factor structures across different. This should be done in future research.

coefficients) and can be found in table 7 along with model fit indices. All observed indicators were described previously in Chapter III.

Figure 5. Measurement Model for Delinquency and Offending



The first group of latent variables is exogenous and captures characteristics of the mother and her behavior at or around the time of the birth of her child. Low SES is composed of five dichotomous indicators that capture socio-economic characteristics of the mother and father at the birth of their child. Factor loadings are adequate but low, ranging from .443 (mother was single) to .863 (mother did not have a high school degree). The reliability of the scale is .5579.⁴⁶ The second latent variable, parenting, is captured by five dichotomous items that measure maternal parenting behavior when the child was 8 months old. Again, factor loadings are satisfactory (.638 for child is unkept to .938 for mother is indifferent towards child) as is the scale reliability (kr20 = .6394).

⁴⁶ Cronbach's alpha is not appropriate for categorical variables, therefore reliabilities were calculated using the Kuder-Richardson coefficient of reliability (KR20).

The third factor measures negative emotionality and low constraint. This factor was constructed using a two step process. First, as described above, measures were selected from the CPP observation instrument that correspond to characteristics of individuals with low constraint and high negative emotionality. Next exploratory factor analysis with promax rotation⁴⁷ was used to assess whether and how these indicators loaded together. Only one eigenvalue was greater than 1, suggesting that either a one or two factor model was appropriate. With regard to the one factor model, all the indicators had factor loadings greater than .76, however, the root mean square residual (RMSR) was greater than .05, which is usually the upper limit for acceptable model fit. The two factor model, on the other hand had a RMSR of .03, indicating the two factor structure adequately fit the data. In this factor structure, four of the indicators—volatility, overactivity, impulsivity, and willfulness—loaded onto one factor, which closely resembled low constraint. The two other indicators—hostility and negativity—loaded together on a second factor, which maps onto negative emotionality. Willfulness, while loading higher on low constraint (.597), also loaded on negative emotionality (.467).

Next, confirmatory factor analysis was used to determine if a one or two factor model better fit the data. The single factor model of temperament fit the data adequately ($\chi^2 = 48.188$ (6), $p = .000$; CFI = .976; TLF = .972; RMSEA = .069) with factor loadings ranging from .783 to .906. The two factor model, however, provided a better fit to the data ($\chi^2 = 26.434$ (6), $p = .0002$; CFI = .988; TLF = .986; RMSEA = .048) and had higher factor loadings that ranged from .861 to .978.

⁴⁷ Promax, versus varimax, rotation was used because the two factors that emerged from the exploratory analysis were highly correlated (.632).

Because, the two factor model is consistent with Agnew's assertion that negative emotionality and low constraint are two distinct traits, I initially elected to use the two factor solution despite the eigenvalues, the cross loading of willfulness, and the fact that NE was measured with only two indicators.⁴⁸ Initial analyses, however, indicated problems with the two factor model, namely negative emotionality had very low cell counts leading to problems with model convergence once it was entered into the full models. Specifically, only 59 individuals exhibited high levels of negativism and only 17 were more hostile than average. Therefore, a decision was made to use the one factor model, which had adequate reliability ($kr20 = .7441$).⁴⁹

The fourth group of variables included in the measurement model was childhood, adolescent, and adult stressors. These variables are transformed counts of the number of stressors the respondents experienced during each stage of their lives. Observed count variables rather than latent variables were used for several reasons. First, a number of the stressors are exogenous, and therefore would not necessarily be expected to co-occur or load together. For example, the death of one's mother should not necessarily be associated with experiencing sexual abuse. In the instances where several indicators of stressors did load together, they loaded on multiple factors. Practically, including multiple measures of stressors at multiple time periods would make an already complicated model even more complicated.⁵⁰ Last but not least, the stress research indicates that it is the accumulation of multiple stressors that is important (Thoits 1983).

⁴⁸ Models that contain factors with only two indicators are not always identified (Kline 2005).

⁴⁹ This decision is not without precedence; Agnew and colleagues (2002) combined negative emotionality and low constraint in their examination of the moderating effects of personality.

⁵⁰ Research indicates that the type of negative affect individuals experience and the type of coping mechanisms they utilize may vary as a function of the type of stressors they experience (e.g., Slocum, Simpson, and Smith 2005). For this reason, future research should replicate this analysis examining the differential effects of various types of stressors.

For this reason, using a count of stressors has become the standard methodology for measuring stressors.

The final group of variables included in the measurement model was adolescent delinquency and adult offending. Delinquency is a latent variable constructed from eight dichotomous items, scored 1 if the respondent engaged in the behavior and 0 otherwise. The behaviors include played hooky from school, got in trouble at school for misbehaving, carried a weapon, got in trouble at school or work for fighting, stole something worth \$50 or more, hit or seriously threatened to hit someone, sold drugs, and rode in or drove a vehicle without the owner's permission. The latent construct measuring adult offending consists of six dichotomous items: carried a weapon; stole something worth \$50 or more; hit or seriously threatened to hit someone; sold drugs; rode in or drove a vehicle without the owner's permission; and traded sex for money, drugs, food, or anything else. For delinquency, the factor loadings range from .510 (getting in trouble at work or school for fighting) to .832 (theft) and for offending they range from .657 (prostitution) to .864 (selling drugs). The scale reliabilities for delinquency and offending are .6431 and .6572, respectively (see table 4).

One of the main advantages of using SEM is the ability to model the correlations among the error terms of observed indicators, thereby controlling for the biasing effects of measurement error. When two error terms are specified to covary, the assumption is that they have something in common that is not captured by the latent variable (Kline 2005). In the measurement models, error terms were specified to correlate when this made theoretical sense and significantly improved the fit of the model.

Table 7. Parameter Estimates for the Offending Measurement Model Controlling for Race, Sex, and Age

Standardized Parameters

	Low SES	Parenting	NE/LC	Childhood Stressors	Adolescent Stressors	Adult Stressors	Delinquency	Offending
	1							
Parenting	0.327	1						
LC/NE	0.169	0.023	1					
Childhood Stressors	0.087	0.070	0.075	1				
Adolescent Stressors	0.200	0.096	0.038	0.202	1			
Adult Stressors	0.171	0.132	0.029	0.160	0.336	1		
Delinquency	0.173	0.182	0.064	0.085	0.309	0.344	1	
Offending	0.202	0.196	0.046	0.072	0.272	0.401	0.658	1

Unstandardized Parameters (Standard errors in parentheses; z-score in italics)

	Low SES	Parenting	NE/LC	Childhood Stressors	Adolescent Stressors	Adult Stressors	Delinquency	Offending
Low SES	1							
Parenting	0.113 (.025) <i>z= 4.494</i>	1						
NE/LC	0.075 (.026) <i>z= 2.917</i>	0.015 (.035) <i>z= 0.437</i>	1					
Childhood Stressors	0.011 (.004) <i>z= 2.627</i>	0.013 (.008) <i>z= 1.709</i>	0.018 (.010) <i>z= 1.824</i>	1				
Adolescent Stressors	0.049 (.009) <i>z= 5.149</i>	0.034 (.015) <i>z= 2.249</i>	0.017 (.021) <i>z= 0.823</i>	0.026 (.003) <i>z= 7.681</i>	1			
Adult Stressors	0.044 (.009) <i>z= 4.607</i>	0.049 (.016) <i>z= 3.032</i>	0.014 (.022) <i>z= 0.648</i>	0.021 (.004) <i>z= 6.017</i>	.088 (.007) <i>z= 12.598</i>	1		
Delinquency	0.045 (.012) <i>z= 3.778</i>	0.068 (.023) <i>z= 2.972</i>	0.031 (.026) <i>z= 1.204</i>	.011 (.004) <i>z= 2.629</i>	.081 (.010) <i>z= 7.884</i>	.095 (.011) <i>z= 8.403</i>	1	
Offending	0.086 (.020) <i>z= 4.316</i>	0.123 (.042) <i>z= 2.947</i>	0.037 (.057) <i>z= 0.654</i>	.016 (.007) <i>z= 2.222</i>	.119 (.015) <i>z= 8.085</i>	.185 (.017) <i>z= 11.159</i>	.306 (.031) <i>z= 9.919</i>	1

Chi-square = 333.774 (168), p = .000 CFI = .966 TLI = .968 RMSEA = .026

Notes: z-scores greater than 1.96 are significant at alpha = .05 and z-scores greater than 2.58 are significant at alpha = .01; n = 1,468

Because the same instrument was used to simultaneously obtain both adult and adolescent measures of illegal behavior, it is highly probable that the indicators capturing the same behaviors in adolescence and adulthood have correlated error terms (Hoffmann and Miller 1998; Jöreskog and Sörbom 1979). For this reason, the errors terms of items measuring the same behavior in adolescence and adulthood were allowed to correlate with each other (e.g., the error term for selling drugs as an adolescent was allowed to correlate with the error term for selling drugs as an adult). This decision makes sense theoretically and significantly improved the fit of the measurement model. Several additional correlations were added among the delinquency indicators. Specifically, the error term for got in trouble for fighting at work or school was freed to correlate with the error terms for got in trouble for misbehaving at school and playing hookey. In addition, the error term for got in trouble for fighting at school or work was specified to correlate with hit or threatened to hit. Theoretically, these decisions were based on the fact that in the former case, all the variables are related to school and in the latter case, both items capture violent behavior.

A few additional correlated error terms were specified. First, the residual for mother was single at the birth of her child was allowed to covary with father was under age 18 at the child's birth and mother received welfare. Finally, the residuals of negativism and hostility were freed to covary. These two items capture negative emotionality and, therefore, they probably share measurement error not shared by those items mapping more closely onto low constraint. Overall, allowing indicator error terms to covary significantly improved the fit of the model ($\chi_D = 543.413 (10), p = .0000$). The

χ^2 value decreased from 605.127 (d.f. = 170) in the uncorrelated model to 333.774 (d.f. = 168) in the model containing correlated error terms.⁵¹

In the next section, I describe the base model, which specifies the causal relationships among the variables of interest. For each hypothesized pathway to stability, the findings from the model are evaluated to determine if the pathway is supported by the data. In addition, for each pathway I then eliminated the relationships that are hypothesized to lead to continuity in offending by fixing their relationship to 0. Changes in the fit of the model and in the relationship between delinquency and offending and adolescent and adult stressors are used to assess whether the hypothesized relationships are supported (Klein 2005).

For each pathway there is a corresponding figure that shows the relationships that are central to the hypothesis. All other relationships have been deleted from the figure (but not from the model) for ease of interpretation. In addition, each figure contains a pathway(s) represented by a dashed arrow. This dashed arrow indicates which relationships are predicted to be affected when the relevant pathways are fixed to 0. The number in parentheses is the standardized estimate of the parameter after the relationships of interest were fixed to 0. In addition, the bottom of each figure contains information regarding the change in model fit associated with fixing the relevant pathways to 0.

⁵¹ It should be noted that SEM involves making theoretically and empirically informed decisions about how best to model the data. The results obtained from the models may vary based on these decisions. My decisions regarding correlated error terms were based on theoretical and empirical considerations and represent what I believe is the appropriate specification of the model. However, to test the robustness of this specification, the base models were rerun omitting correlated error terms for the NE/LC, low SES, and depression constructs. (The correlations between the error terms of items measuring the same behavior at two points in time, such as adolescent and adult drug selling, were not omitted because doing so would theoretically misspecify the models) In all the models that were run, removing the correlated error terms worsened the fit of the model, and negligibly altered the magnitude of the coefficients. For both outcomes, there were no substantive differences between the models presented in this dissertation and the alternate specifications.

Specifically, the fit indices of the restricted model are provided as well as the chi-square difference statistic comparing the restricted model to the base model. Once all the hypothesized pathways to continuity were examined, all nonsignificant relationships were removed and a final model was estimated.

BASE MODEL

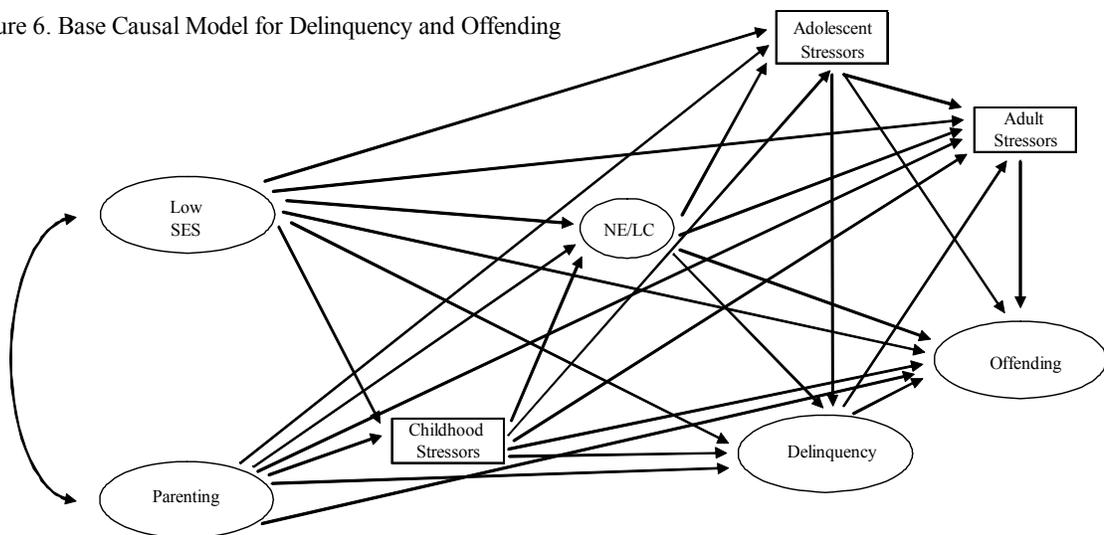
The base model used to assess all of the hypothesized pathways leading to continuity in offending is depicted in figure 6 and the standardized and unstandardized parameters are located in table 8. The base model contains the correlated residuals for the indicators as specified in the measurement model (these are not shown in the figure), and an unanalyzed correlation between parenting and low SES. In contrast to the measurement model, all other variables are now connected with causal arrows rather than correlations. The causal arrows go from variables measured at earlier points in time to variables measured later in time to ensure proper temporal order. In the instances where variables are measured simultaneously, causal ordering was determined by theory and prior empirical research. Therefore, adolescent stressor exposure is specified to cause adolescent offending and adult stressor exposure is specified to cause adult offending. The base model has adequate fit with χ^2 /d.f. that is less than 3 ($\chi^2 = 333.774$ (168), $p = .0000$); CFI = .966; TLI = .968; and RMSEA = .026 (see appendix A for an explanation of the fit indices).

There are several points that should be noted regarding the base model. First, in all likelihood there are reciprocal relationships between stressor exposure and illegal

behavior; however, because of data limitations these cannot be explicitly modeled.⁵²

Also note that childhood exposure to stressors is modeled as a cause, not an outcome, of low constraint. This specification maintains proper causal ordering because the measure of childhood stressor exposure contains events that occurred from birth through age 8, while the measure of NE/LC was taken between the ages of 7 and 8. Moreover, Agnew (1997, 2006) argues that exposure to stressors in childhood contributes to the development of NE/LC. Finally, both contemporaneous and lagged effects of stressors on delinquency and offending are included in the model.

Figure 6. Base Causal Model for Delinquency and Offending



Chi Square = 333.774 (168), $p = .0000$, CFI = .966, TLI = .968, and RMSEA = .026

Not shown: sex, race, and age were included as control variables

⁵² An instrumental variable (i.e., a variable expected to have a direct effect on stressor exposure but not illegal behavior, or vice versa) is needed to estimate the simultaneous effects of stressors on illegal behavior and illegal behavior on stressors. Otherwise, the model is not identified (Kline 2005).

Table 8. Parameter Estimates for Offending Base Model Controlling for Race, Sex, and Age

Standardized Parameters

Endogenous Variables	Exogenous Variables						
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Delinquency	Adult Stressors
Childhood Stressors	0.072	0.047					
NE/LC	0.176	-0.039	0.065				
Adolescent Stressors	0.177	0.025	0.192	-0.007			
Delinquency	0.066	0.132	0.009	0.039	0.286		
Adult Stressors	0.057	0.025	0.087	-0.017	0.203	0.345	
Offending	0.042	0.022	-0.015	-0.015	-0.028	0.813	0.124

Unstandardized Parameters (Standard errors in parentheses; z-score in italics)

Endogenous Variables	Exogenous Variables						
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Delinquency	Adult Stressors
Childhood Stressors	.038 (.020) <i>z = 1.925</i>	.017 (.017) <i>z = 0.992</i>	1				
NE/LC	.332 (.122) <i>z = 2.708</i>	-.050 (.080) <i>z = -0.625</i>	.232 (.156) <i>z = 1.485</i>	1			
Adolescent Stressors	.181 (.045) <i>z = 4.04</i>	.018 (.036) <i>z = 0.492</i>	.374 (.048) <i>z = 7.807</i>	-.004 (.026) <i>z = -0.15</i>	1		1
Delinquency	.071 (.050) <i>z = 1.427</i>	.098 (.046) <i>z = 2.116</i>	.019 (.064) <i>z = 0.297</i>	.022 (.03) <i>z = 0.731</i>	.303 (.041) <i>z = 7.417</i>		
Adult Stressors	.062 (.041) <i>z = 1.512</i>	.018 (.036) <i>z = 0.505</i>	.179 (.053) <i>z = 3.369</i>	-.01 (.027) <i>z = -0.363</i>	.214 (.029) <i>z = 7.389</i>	.342 (.056) <i>z = 6.108</i>	
Offending	.075 (.074) <i>z = 1.024</i>	.028 (.067) <i>z = 0.41</i>	-.052 (.098) <i>z = -0.534</i>	-.014 (.052) <i>z = -0.271</i>	-.050 (.051) <i>z = -0.974</i>	1.354 (.155) <i>z = 8.739</i>	.209 (.057) <i>z = 3.693</i>

Chi-square = 333.774 (168), p = .000 CFI = .966 TLI = .968 RMSEA = .026

Notes: z-scores greater than 1.96 are significant at alpha = .05 and z-scores greater than 2.58 are significant at alpha = .01; n = 1,468.

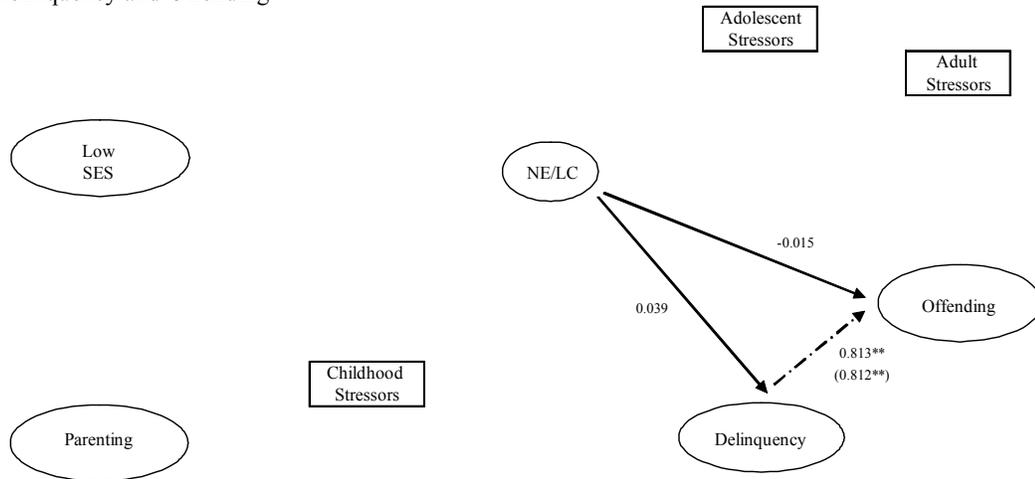
Note, as predicted by GST there is a significant contemporaneous relationship between stressors and illegal behavior. The standardized coefficient for the effect of adolescent stressors on delinquency is .286, which means that when adolescent stressor exposure is one standard deviation above the mean, delinquency is predicted to increase by .286 standard deviations. The relationship between adult stressors and offending is also moderate and significant ($\beta = .124$); However, none of the lagged effects of stressors on delinquency or offending are significant once contemporaneous stressors have been taken into account. This may be true for several reasons. First, it may be that the effects of stressors on offending are only contemporaneous and quickly fade over time. Second, the large periods of time over which my data is aggregated (8 years for childhood, 9 years for adolescence, and 10 years or more for adulthood) may obscure any lagged effects of stressors. Finally, it may be that any lagged effect of stressors is overshadowed by the more proximal effects of contemporaneous stressors (see Slocum, Simpson, and Smith 2005).

DIRECT EFFECT

The direct effect hypothesis states that NE/LC has a direct effect on both delinquency and offending and that stability in offending is due to stability in these traits. Therefore, the relationship between past and future offending is spurious with the two sharing a common cause, NE/LC. If this population heterogeneity explanation of continuity is true, several relationships should exist. First, there should be a significant direct association between NE/LC and both delinquency and offending. Second, when these relationships are constrained to be 0, model fit should be significantly worse in the

constrained model than in the unconstrained model. Moreover, when the paths from NE/LC to delinquency and NE/LC to offending are eliminated, the association between delinquency and offending should increase because their common cause is no longer being controlled.

Figure 7. Direct Effect of Negative Emotionality and Low Constraint on Delinquency and Offending



Chi square from restricted model = 297.139 (153), $p = .0000$, CFI = .970, TLI = .976, and RMSEA = .025
 Chi-square difference test statistic comparing base model to restricted model = .235 (1), $p = .6277$

Not shown : Sex, race, and age were included as control variables

* $p < .05$; ** $p < .01$

Notes : Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between NE/LC and delinquency and NE/LC and offending were constrained to be 0;

Numbers not in parentheses are the standardized coefficients from the base model presented in table 8.

From the base model, it is apparent that there was not a significant relationship between NE/LC and delinquency or offending (see Table 8 and Figure 7). A level of NE/LC one standard deviation above the mean predicted a level of delinquency that was .039 standard deviations above the mean, controlling for childhood and adolescent stressor exposure, low SES, parenting, and demographic characteristics (gender, race, age). The corresponding parameter estimate for the NE/LC-offending relationship was -.015.⁵³

⁵³ Looking at the relationship of NE/LC and delinquency and offending separately for males and females resulted in similar null findings.

The lack of a relationship between NE/LC and criminal behavior provides clear evidence against the direct effect hypothesis. As would be expected given the lack of an association between the key variables, constraining the relationships between NE/LC and delinquency and NE/LC and offending to be 0 did not significantly alter the fit of the model ($\chi^2_D = .235 (1), p = .6277$). Moreover, the association between delinquency and offending remained virtually unchanged from the unconstrained model ($\beta = .812$). In sum, there is no evidence to suggest that continuity in offending is attributable to time stable differences across individuals in NE/LC.

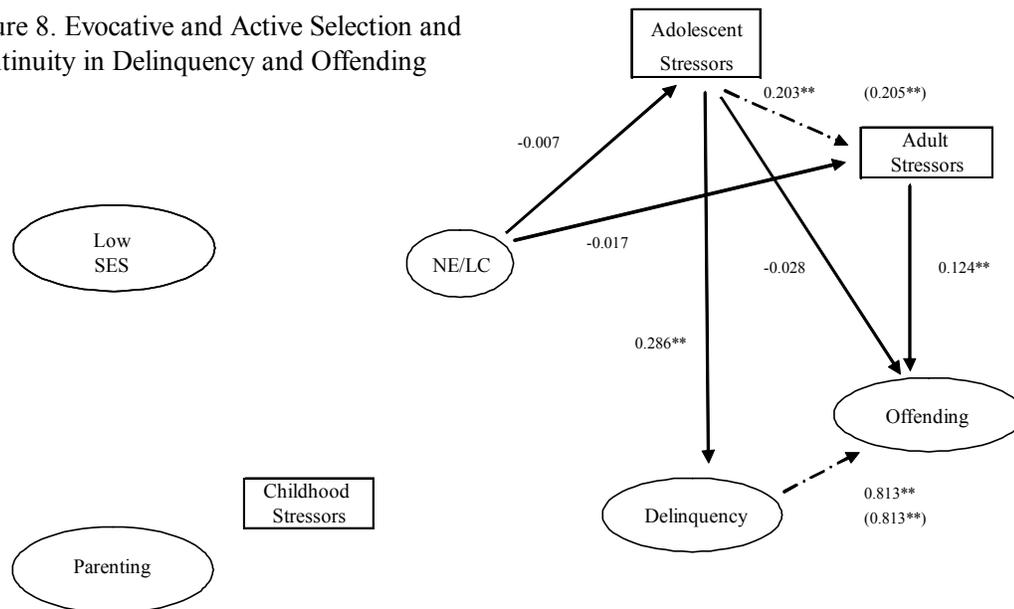
EVOCATIVE AND ACTIVE SELECTION

In evocative and active selection, individuals high in NE/LC encounter more stressors in their lives because they actively seek out environments that are likely to have a high number of stressors and because they create stressful situations through their unpleasant interactions with others. Continuity in offending from adolescence to adulthood is due to the continuing effects of NE/LC on exposure to stressors, which in turn increases the likelihood of illegitimate coping. As such, in this pathway NE/LC is expected to have a direct association with adolescent and adult stressor exposure and an indirect association with delinquency and offending via exposure to stressors. In addition, if stressor exposure in adolescence and adulthood are both associated with NE/LC, constraining the pathways between NE/LC and stressors to 0 should increase the association between the stressor measures.

Examination of the unconstrained model immediately brings the validity of the evocative and active selection hypothesis into question (see table 8 and figure 8). The

relationships between NE/LC and exposure to adolescent and adult stressors were small and nonsignificant ($\beta = -.007$ and $\beta = -.017$, respectively). Moreover, none of the indirect effects of NE/LC on delinquency and offending via exposure to stressors were significant (NE/LC→adolescent stressors→delinquency: $\beta = -.002$; NE/LC→adolescent stressors→offending: $\beta = .000$; NE/LC→adult stressors→offending: $\beta = -.002$).

Figure 8. Evocative and Active Selection and Continuity in Delinquency and Offending



Chi Square from restricted model = 330.617 (168), $p = .0000$, CFI = .966, TLI = .969, and RMSEA = .026
 Chi-square difference test statistic comparing base model to restricted model = .173 (2), $p = .9173$

Not shown: Sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes: Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between NE/LC and adolescent stressors and NE/LC and adult stressors were constrained to be 0;

Numbers not in parentheses are the standardized coefficients from the base model presented in table 8.

Next the pathways from NE/LC to adolescent and adult stressor exposure were constrained to 0. When the constrained and unconstrained models were compared, there was no significant difference in model fit ($\chi_D = .173 (2)$, $p = .9173$) and almost no difference in the relationship between adolescent and adult stressors ($\beta = .205$) or

delinquency and offending ($\beta = .813$). These findings lead to the conclusion that there is no evidence in these data that individuals with higher levels of NE and LC seek out more stressful environments or create stressors through their interactions with others and therefore there is no support for the evocative and active selection explanation of continuity.⁵⁴

PASSIVE SELECTION

Passive selection is based on the idea that children's temperaments are a function of both heredity and family environment and, therefore, children are more likely to be high in NE/LC if their parents are high in these traits. Moreover, parents characterized by NE/LC tend to engage in poor parenting practices and create stressful family environments for their children. This means that as long as children remain living with parents high in NE/LC they are at increased risk for exposure to stressors. Passive selection, then, implies that there should be an indirect effect of parental temperament on criminal behavior via the respondent's exposure to stressors.

Passive selection, as specified by Agnew, only applies to the time when youth are still living with their parents because it is based on the notion that parents "select" their children's environments for them. Thus, this path should only operate while children are living at home with their parents. However, because most of the respondents in the sample remained in their parents' houses after age 18 ($n = 949$, 65 percent), I may observe a direct relationship between parenting and adult stressors.

⁵⁴ Because the measures of stressors contain some exogenous events that are unlikely to be a result of any action on the part of the respondent (e.g. death of family member, sexual abuse), I re-estimated the model using measures of stressors that excluded these items. Still, the effects of NE/LC on stressors in adolescence and adulthood were small, negative, and nonsignificant ($\beta = -.007$ for adolescent stressors and $\beta = -.053$ for adult stressors).

Because there are no measures of parental temperament in either the CPP or the PTA study, observations of the mothers with their children, which were recorded when the children were 8 months old, were used to capture parental NE/LC. Although this is not ideal, poor parenting is one of the mechanisms through which parental temperament is hypothesized to impact child temperament and exposure to stressors. Therefore, maternal parenting behavior⁵⁵ should be an acceptable substitute for measures of NE/LC.

There are two relationships regarding the origins of NE/LC that should hold true if the passive selection hypothesis is supported. First, there should be relationship between parenting behavior and child NE/LC. If poor parenting captures aspects of parental NE/LC, then children with parents characterized by these traits should have a higher probability of inheriting these traits. In addition, poor parenting itself increases a child's probability of being high in NE/LC. Second, there should be a significant association between childhood stressors and NE/LC because experiencing stressors in childhood contributes to the development of these traits.

At the heart of passive selection is the idea that stability in illicit behavior is due to the indirect effects of parental behavior on delinquency and offending via exposure to stressors. This implies that the relationship between past and future stressors is spurious and both are attributable to parental behavior. If this is true, the association between adolescent and adult stressors should increase if their associations with parenting are constrained to 0.

Results from the base model indicate immediate problems with the passive selection explanation of stability (See figure 9 and table 8). First, neither of the variables predicted to be associated with NE/LC were significant. The relationship between

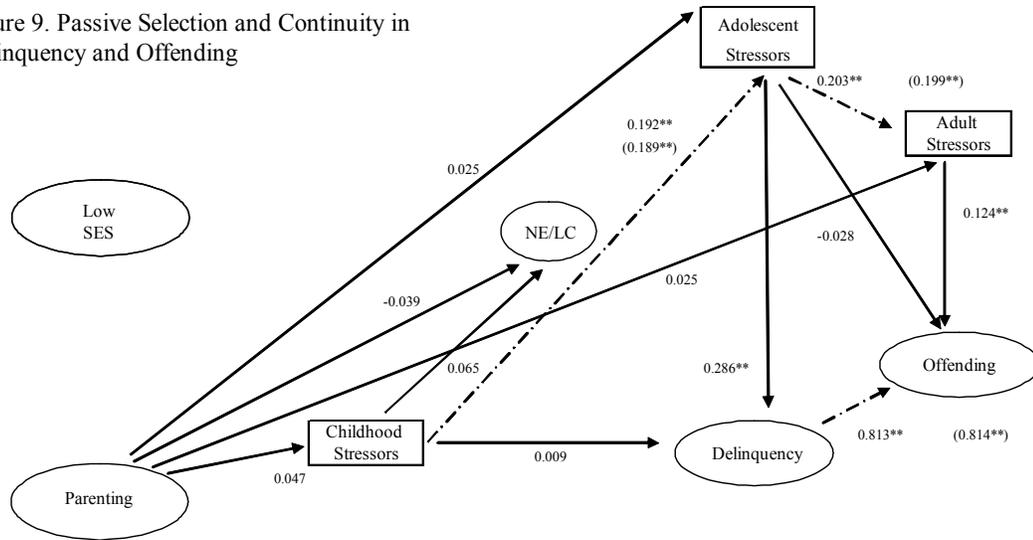
⁵⁵ Again, these measures are not available for the fathers.

parenting and NE/LC was small and in the opposite direction of what was predicted ($\beta = -.039$)⁵⁶ and there was no association between childhood stressor exposure and NE/LC ($\beta = .065$). Note, however, that there was a small to moderate relationship between NE/LC and low SES ($\beta = .176$), which was not predicted (see table 8). One possible explanation for this finding is that low SES may serve as a better measure of exposure to stressors in childhood than my limited measure. The childhood stressor measure is quite narrow in scope and includes relatively rare events. In contrast, the daily stressors associated with living in a low SES household (e.g. victimization, discrimination, poverty, aversive neighborhoods) might be more likely to lead to the development of negative emotionality and low constraint. Furthermore, chaotic family life and inconsistent discipline have been linked to the development of these traits, both of which are likely to be more prevalent in families headed by single teenage mothers with little education.

Further evidence against passive selection is that the association between parenting and exposure to stressors in childhood was relatively small ($\beta = .047$) and not significant. Similar results were found for the relationship between parenting and stressors in adolescence ($\beta = .025$) and adulthood ($\beta = .025$). Given that one of the requirements for mediation is a significant effect of the exogenous variable (in this case parenting) on the mediator (exposure to stressors), the data fail to support the hypothesis that the effect of parenting on delinquency and offending is mediated by exposure to stressors. This is further confirmed when these indirect effects are calculated (parenting→childhood stressors→delinquency: $\beta = .000$; parenting→adolescent

⁵⁶ Low SES and parenting are fairly highly correlated so it is possible that the lack of a significant association between NE/LC and parenting is due to multicollinearity. Further analysis in which low SES was removed indicated this was not the case; the relationship between NE/LC and parenting still did not reach significance.

Figure 9. Passive Selection and Continuity in Delinquency and Offending



Chi Square from restricted model = 326.867 (167), $p = .0000$, CFI = .967, TLI = .969, and RMSEA = .026
 Chi-square difference test statistic comparing base model to restricted model = 4.346 (5), $p = .5007$

Not shown : sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes : Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between all stressor measures and parenting, NE/LC and parenting, and NE/LC and childhood stressors were constrained to be 0; Numbers not in parentheses are the standardized coefficients from the base model presented in table 8.

stressors \rightarrow delinquency: $\beta = .007$; parenting \rightarrow adolescent stressors \rightarrow offending: $\beta = -.001$;
 parenting \rightarrow adult stressors \rightarrow offending: $\beta = .003$).

When the key relationships predicted by passive selection were set to 0 (i.e., parenting to childhood, adolescent, and adulthood stressors; parenting to NE/LC; and childhood stressors to NE/LC), there was no significant change in the model fit ($\chi_D = 4.346 (5)$, $p = .5007$). In addition, the associations between childhood and adolescent stressors ($\beta = .189$), adolescent and adult stressors ($\beta = .199$), and delinquency and offending ($\beta = .814$) remained virtually unchanged from the base model.

Notice, however, that there was a direct effect of parenting on delinquency ($\beta = .132$) and an indirect effect of parenting on offending via delinquency ($\beta = .107$) that remained, even after controlling for adolescent stressors, low SES, and childhood and adolescent stressors (see table 8). Parenting is clearly important, but in this sample it

does not appear to affect delinquency in the manner hypothesized by a passive selection model of continuity.⁵⁷

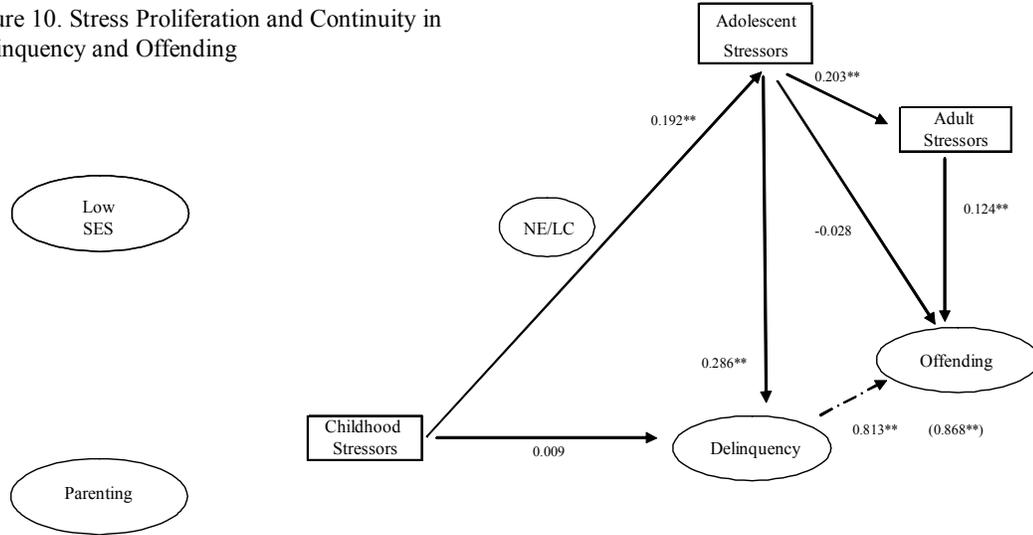
STRESS PROLIFERATION

Stress proliferation is the notion that stressors may actually create future stressors, independent of an individual's behavior. Therefore, stress proliferation predicts that controlling for delinquency and other relevant variables, there should be a direct relationship between stressors in childhood and adolescence and stressors in adolescence and adulthood. It is this continuity in stressors that is responsible for continuity in criminal behavior. This pathway also suggests the existence of several indirect effects. First, adolescent stressors should mediate the relationship between childhood stressors and delinquency and adolescent and adult stressors should mediate the relationship between childhood stressors and offending. Similarly, there should be an indirect effect of adolescent stressors on offending via adult stressors.

All of the results were consistent with the process of stress proliferation (see figure 10 and table 8). There were indeed significant moderate relationships between childhood and adolescent stressors ($\beta = .192$) and adolescent and adult stressors ($\beta = .203$). Furthermore, all the hypothesized indirect effects were significant, albeit small. Adolescent stressor exposure mediated the relationship between childhood stress exposure and delinquency ($\beta = .055$) and childhood stressors were associated with offending via adolescent and adult stressor exposure, although this effect was quite small in magnitude ($\beta = .005$). Similarly, as predicted, adult stressor exposure mediated the

⁵⁷ I tested the robustness of these results by excluding all individuals who left home before age 19. The substantive results remained unchanged.

Figure 10. Stress Proliferation and Continuity in Delinquency and Offending



Chi Square from restricted model = 366.750 (169), $p = .0000$, CFI = .959, TLI = .962, and RMSEA = .028
 Chi-square difference test statistic comparing base model to restricted model = 100.543 (2), $p = .0000$

Not shown : sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes : Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between childhood and adolescent stressors and adolescent and adult stressors were constrained to be 0; Numbers not in parentheses are the standardized coefficients from the base model presented in table 8.

relationship between adolescent exposure and offending ($\beta = .025$). These findings highlight the importance of looking at indirect effects. If only the direct effects of stressors on delinquency and offending were examined, then the importance of childhood stressors would be overlooked.

When the associations between childhood and adolescent stressors and adolescent and adult stressors were fixed to 0, the fit of the model worsened significantly ($\chi^2_D = 100.543 (2), p = .0000$) and the relationship between delinquency and offending increased from $\beta = .813$ to $\beta = .868$. This increase provides further support that stress proliferation is partially responsible for continuity in offending from adolescence to adulthood.

Finally, note that there was an unexpected direct effect of childhood stressor exposure on adult stressors. In fact, 58 percent of the total relationship between childhood and adult stressors was direct, while only 26.4 percent was indirect through adolescent stressors. It is likely that at least part of this association is due to omitted variables, like neighborhood characteristics or traits aside from NE/LC, that affect stressors across the life span. However, it is also possible that experiencing stressors early in life, especially traumatic stressors like sexual abuse and maternal death, can have a lasting impact.

AMPLIFICATION

The amplification hypothesis is the only continuity promoting mechanism that Agnew discusses which is not trait based. Here continuity is hypothesized to result from a feedback loop in which stressors lead to criminal behavior, which itself leads to more stressors and then more criminal behavior. This loop is set in motion not by NE/LC, but by low SES, because lower class status greatly increases the likelihood an individual will be exposed to stressors.

The amplification hypothesis predicts direct effects of low SES on childhood and adolescent stressor exposure because those with a lower SES are more likely to experience a variety of strains resulting from their economic position, neighborhood characteristics, and schooling experiences. Stressor exposure should, in turn, have a direct association with delinquency and offending. The key prediction of the amplification hypothesis, however, is that delinquency will have a direct effect on adult

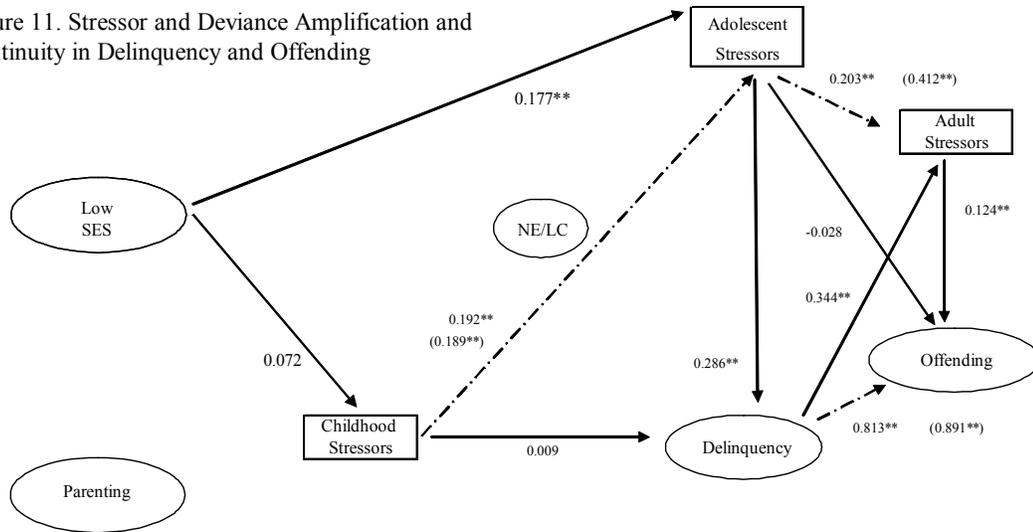
stressor exposure, controlling for past exposure to stressors, and that adult stressor exposure will be associated with offending.

Several additional indirect effects are predicted by the amplification process. Low SES is hypothesized to have an indirect effect on delinquency via exposure to adolescent and childhood stressors. The relationship between delinquency and offending should be mediated by exposure to stressors in adulthood, while the relationship between adolescent and adult stressor exposure is mediated by delinquency.

Examination of the base model (table 8 and figure 11) suggests that there is support for the amplification process. As predicted, low SES was significantly and moderately associated with adolescent exposure to stressors ($\beta = .177$). In contrast, the relationship between class and childhood stressors was small ($\beta = .072$) and not quite significant. In turn, as discussed above, contemporaneous effects of stressors on delinquency and offending were significant, while lagged effects were not. Other predicted direct relationships were significant and in the predicted direction. Delinquency was strongly associated with greater stressor exposure in adulthood ($\beta = .344$) controlling for prior stressor exposure in adolescence. Adult stressor exposure was also significantly associated with adult offending ($\beta = .124$) after controlling for delinquency.

The indirect effects also generally supported the contribution of the amplification process to continuity in offending. There was a small but significant indirect effect of low SES on delinquency via exposure to stressors in adolescence ($\beta = .051$). On the other hand, childhood stressor exposure did not mediate the effect of low SES on delinquency as it fails to meet the necessary conditions for serving as a mediator (i.e.,

Figure 11. Stressor and Deviance Amplification and Continuity in Delinquency and Offending



Chi Square from restricted model = 362.012 (169), $p = .0000$, CFI = .960, TLI = .963, and RMSEA = .028
 Chi-square difference test statistic comparing base model to restricted model = 60.8000 (1), $p = .0000$

Not shown : sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes : Numbers in parentheses are the standardized coefficients from the restricted model in which the relationship

between delinquency and adult stressors was constrained to be 0;

Numbers not in parentheses are the standardized coefficients from the base model presented in table 8.

there was not a significant relationship between the independent variable and the mediator or the mediator and the outcome). As predicted, adult exposure to stressors partially mediated the relationship between delinquency and offending ($\beta = .043$); however, this indirect effect only accounted for 5 percent of the total association between delinquency and offending. In addition, delinquency partially mediated the relationship between adolescent and adult stressor exposure ($\beta = .099$), with this indirect effect responsible for approximately 33 percent of the total relationship between adolescent and adult stressors.

Further evidence that amplification plays a role in the stability of illegal behavior comes from the fact that when the relationship between delinquency and adult stressors was fixed to 0, the model fit worsened significantly ($\chi^2_D = 60.800 (1), p = .0000$). In addition, the association between delinquency and offending increased to .891 further suggesting that exposure to adult stressors partially mediates the relationship between

delinquency and offending. Similarly, the relationship between adolescent and adult stressors increased to .412, suggesting that delinquency partially mediates the relationship between adolescent and adult stressors.

TRIMMED FINAL OFFENDING MODEL

Figure 12 depicts the final model that incorporates each of the supported pathways to continuity and has all of the nonsignificant pathways fixed to 0. The model adequately fits the data ($\chi^2 = 285.608$ (148), $p = .0000$; CFI = .972; TLI = .970; RMSEA = .025) and the model fit does not differ significantly from that of the base model ($\chi^2_D = 12.663$ (9), $p = .1784$). Parameter estimates are presented in table 9.

From this figure, it is apparent that in these data NE/LC does not play any role in maintaining delinquency and offending from adolescence to adulthood, nor do these traits affect exposure to stressors. Therefore, the direct effect and evocative and active selection models of continuity are not supported in these data. Parenting also fails to contribute to continuity in offending over time via its influence on stressor exposure as there are no direct associations between this variable and experiencing stressors in childhood, adolescence, or adulthood. These findings fail to support the passive selection model. However, there is a direct effect of parenting on delinquency ($\beta = .205$) and an indirect effect on offending via delinquency ($\beta = .170$) so, although parenting as measured here may not influence exposure to stressors, it is not irrelevant to explanations of offending and delinquency.

One model that is supported is that of stress proliferation. There were significant moderate direct effects of past stressor exposure on future exposure controlling for the

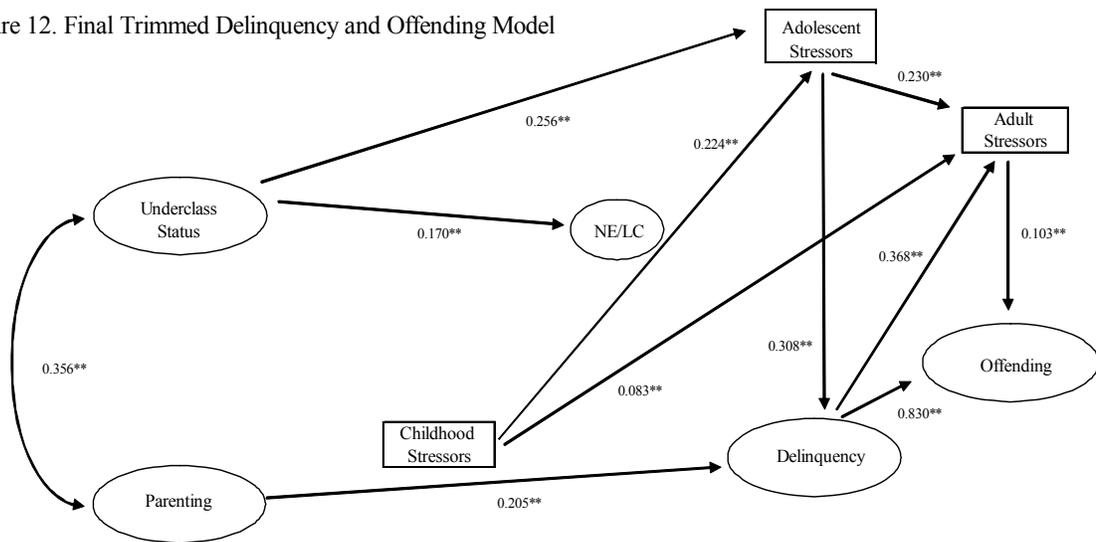
other variables in the model. Moreover, the effect of childhood stressors on delinquency was completely mediated by adolescent stressors while its effect on offending was fully mediated by adolescent and adult stressors. In the final model, fixing the direct effects of past stressors on future stressors to 0 increased the association of delinquency with offending by about 4 percent from .830 to .862. Therefore this process plays a role, albeit a modest one, in the continuity of offending.

The only other model that the data supported is amplification. As predicted, being a member of the lower class increased exposure to stressors in adolescence. This stressor exposure was associated with engaging in delinquency and then, key to the amplification process, delinquency was associated with exposure to more stressors in adulthood. Finally, adult stressor exposure was associated with adult offending, even with delinquency included as a control for time stable omitted variables. However, recall that because the non-recursive relationship between stressors and offending is not modeled, the size of this relationship is probably inflated. There also existed a significant indirect effect of delinquency on offending via exposure to stressors in adulthood ($\beta = .038$), which accounted for approximately 4 percent of the total association between delinquency and offending. In comparison, delinquency mediated a much larger portion of the total association between adolescent and adult stressors—about 33 percent. The one path in the amplification hypothesis that was not supported was the relationship between class, childhood stressors, and delinquency, and this may be a function of the measure of childhood stressors. Unlike the later stressor measures, the stressors measured in childhood tend to be rare and relatively isolated events, and not necessarily the types of events and situations that are associated with SES.

In sum, it appears as if the models outlined by Agnew to explain continuity in delinquency and offending are lacking. There is some support for stress proliferation and amplification, however these processes account for a small portion of the association between delinquency and offending. Moreover, the amplification process seems to do a better job of explaining the stability of stressors over time, than the stability of offending.

In the next section, I examine the final two hypotheses about the role of stressors in maintaining offending over time. Both of these hypotheses involve moderating effects. Specifically, NE/LC and experience with stressors in childhood are predicted to moderate the effect of stressors on illegal behavior.

Figure 12. Final Trimmed Delinquency and Offending Model



Chi-square = 285.608 (148), $p = .000$

CFI = .972 TLI = .970 RMSEA = .025

Chi-square difference test statistic comparing base model to restricted model = 12.663 (9), $p = .1784$

Not shown: sex, race, and age were included as control variables

* $p < .05$; ** $p < .01$

Note: Coefficients are standardized.

Table 9. Parameter Estimates for the Final Offending Model Controlling for Race, Sex, and Age

Standardized Parameters

Endogenous Variables	Exogenous Variables						
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Delinquency	Adult Stressors
Childhood Stressors							
NE/LC	0.17						
Adolescent Stressors	0.256		0.224				
Delinquency		0.205			0.308		
Adult Stressors			0.083		0.23	0.368	
Offending						0.83	0.103

Unstandardized Parameters (Standard errors in parentheses; z-score in italics)

Endogenous Variables	Exogenous Variables						
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Delinquency	Adult Stressors
Childhood Stressors							
NE/LC	.32 (.106) <i>z = 3.013</i>						
Adolescent Stressors	.257 (.045) <i>z = 5.675</i>		.427 (.061) <i>z = 7.049</i>				
Delinquency		.152 (.044) <i>z = 3.456</i>			.333 (.042) <i>z = 7.952</i>		
Adult Stressors			.171 (.052) <i>z = 3.283</i>		.248 (.033) <i>z = 7.484</i>	.366 (.056) <i>z = 6.538</i>	
Offending						1.382 (0.154) <i>z = 8.98</i>	.172 (.059) <i>z = 2.931</i>

Chi-square = 285.608 (148), p = .000

CFI = .972

TLI = .970

RMSEA = .025

Notes: z-scores greater than 1.96 are significant at alpha = .05 and z-scores greater than 2.58 are significant at alpha = .01; n = 1,468

MODERATING EFFECTS

Differences in the Strain-Offending Relationship by Level of NE/LC

Recall that population heterogeneity theories, such as self-control theory (Gottfredson and Hirschi 1990), are based on the idea that stability in criminal behavior is due to the continued expression of time stable traits. Therefore, GST is not alone in arguing that continuity in offending is partially due to stability in temperament over time. The unique contribution of GST to static explanations of continuity is that in addition to having a direct effect on offending, NE and LC will moderate the relationship between stressor exposure and illicit behavior such that individuals possessing higher levels of these traits will be more likely to find a given stressor aversive and more likely to use illegitimate coping to alleviate the ensuing negative emotions.

To assess the hypothesis that individuals higher in NE/LC will be more likely to respond to stressors with illicit behavior I divided the sample based on their level of NE/LC. Because the vast majority of respondents did not receive high scores on the indicators of NE/LC, the sample was divided into those possessing none of the characteristics associated with NE/LC ($n = 1,250$) and those possessing one or more of these characteristics ($n = 218$). Next I created two interaction terms by multiplying the dichotomous measure of NE/LC first by adolescent stressors and then by adult stressors. These interaction terms were then included in the model as predictors of delinquency and offending. As predicted by GST, NE/LC significantly moderated the effect of adolescent stressors on delinquency ($\beta = .076$) and adult stressors on offending ($\beta = .134$), such that individuals high in NE/LC were more likely to respond to stressors with delinquency and

offending. So even though there is no evidence to suggest that NE/LC contributes to continuity in offending via selection, it does play a moderating role.

Past Experience as a Source of Population Heterogeneity

Just as the relationship between stressors and offending may be stronger for individuals high in NE/LC, it may also be moderated by non-trait related factors. It has been suggested that experiencing chronic or traumatic stressors at an early age can permanently alter how a person reacts to stressors.

To test for the possibility of an interaction effect based on past experience I followed the same procedure used above. The sample was divided into two groups based upon their exposure to stressors in childhood. Because of the low frequency with which the observed childhood stressors were experienced by the respondents, the individuals who experienced one stressor ($n = 542$; 36.9 percent) were compared to those experiencing none ($n = 926$; 63.1 percent).

Once again the results supported the existence of an interaction effect. Individuals who experienced one or more stressors in childhood were significantly more likely than others to react to adolescent ($\beta = .136$) and adult stressors ($\beta = .190$) with delinquency and offending. This highlights the importance of studying the stressor-offending relationship within the context of individual's prior experiences.

SUMMARY OF FINDINGS

The support for GST explanations for continuity in offending from adolescence to adulthood is mixed. There is no evidence to suggest that NE/LC contributes to stability

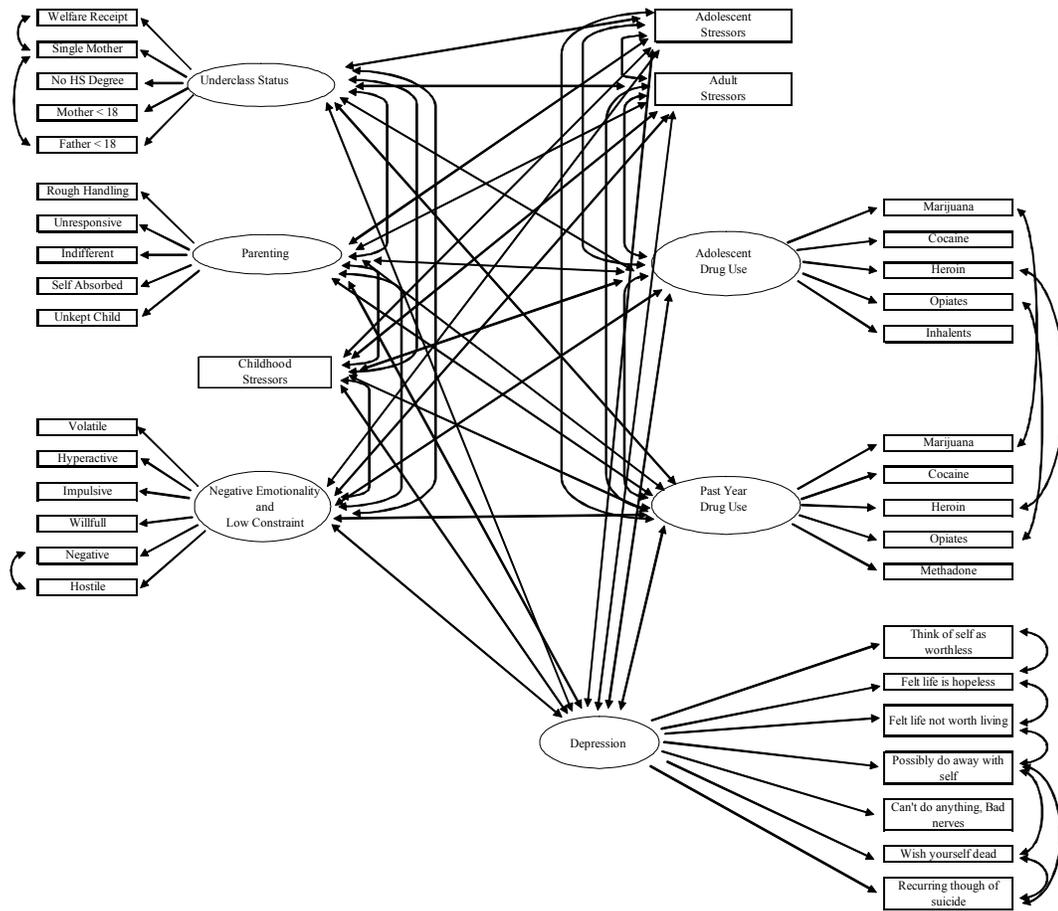
in offending through a direct time stable effect on illicit behavior or via evocative and active or passive selection. However, the findings do suggest that consistent with GST, individuals with higher NE/LC are more reactive to the stressors they experience. Non-trait based explanations of stability also appear to be promising. Stress proliferation plays a small role in maintaining offending from adolescence to adulthood, as does amplification. Yet, these processes better explain stability in stressors over time than stability in offending. There was also support for the hypothesis that past experiences can have a lasting influence on reactions to stressors. As predicted, individuals who had experienced one or more stressors in childhood were more likely to react to later stressors with offending. In the next section, I explore whether these same findings hold true for substance use.

SUBSTANCE USE

MEASUREMENT MODEL

The measurement model for adolescent and past year substance use is presented in figure 13 and the standardized and unstandardized parameter estimates are in table 10. All the latent variables are correlated with each other and with the observed stressors measures. The substance use model includes all of the variables contained in the offending measurement model, plus the addition of depression. Only the variables not included in the offending model will be described here. Please note, however, that there are slight differences in factor loadings and scale reliabilities between the substance use and offending models due to the reduced sample size and the different variables included in the substance use model.

Figure 13. Measurement Model for Adolescent and Past Year Substance Use



Depression is a latent variable composed of seven categorical indicators that measure each respondent's emotional state in the few weeks prior to the interview. The factor loadings range from .736 (think of self as worthless) to .901 (wish yourself dead) and the scale reliability is adequate at .8420.

Adolescent and past year substance use are also latent variables with dichotomous indicators scored 1 if the individual used the drug of interest at least five times and 0 otherwise (see table 4). Adolescent drug use measures the use of five drugs: marijuana, cocaine, heroin, glue, and other opiates. Although the factor loadings were adequate, ranging from .697 for marijuana to .883 for opiates, the reliability is quite low (.3207). This may be due to the relative rarity of adolescent substance use. The past year measure

Table 10. Parameter Estimates for the Substance Use Measurement Model Controlling for Race, Sex, and Age

Standardized Parameters

	Low SES	Parenting	NE/LC	Childhood Stressors	Adolescent Stressors	Adult Stressors	Adolescent Substance Use	Depression	Past Year Substance Use
Low SES	1								
Parenting	0.34	1							
NE/LC	0.166	0.03	1						
Childhood Stressors	0.077	0.072	0.071	1					
Adolescent Stressors	0.199	0.1	0.042	0.199	1				
Adult Stressors	0.171	0.133	0.036	0.157	0.335	1			
Adolescent Substance Use	0.124	0.099	0.152	0.006	0.271	0.373	1		
Depression	0.147	0.193	-0.058	0.061	0.31	0.465	0.149		
Past Year Substance Use	0.137	0.25	0.212	0.092	0.302	0.412	0.29	0.386	1

Table 10 Continued. Parameter Estimates for the Substance Use Measurement Model Controlling for Race, Sex, and Age

Unstandardized Parameters (Standard errors in parentheses; z-score in italics)

	Low SES	Parenting	NE/LC	Childhood Stressors	Adolescent Stressors	Adult Stressors	Adolescent Substance Use	Depression	Past Year Substance Use
Low SES	1								
Parenting	.126 (.026) <i>z = 4.852</i>	1							
NE/LC	.075 (.026) <i>z = 2.855</i>	.020 (.034) <i>z = .605</i>	1						
Childhood Stressors	.010 (.004) <i>z = 2.340</i>	<i>z =</i>	.016 (.010) <i>z = 1.703</i>	1					
Adolescent Stressors	.050 (.010) <i>z = 5.198</i>	.037 (.016) <i>z = 2.350</i>	.019 (.021) <i>z = .902</i>	.025 (.003) <i>z = 7.477</i>	1				
Adult Stressors	.045 (.010) <i>z = 4.645</i>	.052 (.017) <i>z = 3.016</i>	.017 (.022) <i>z = .772</i>	.021 (.004) <i>z = 5.855</i>	.088 (.007) <i>z = 12.425</i>	1			
Adolescent Substance Use	.045 (.019) <i>z = 2.387</i>	.053 (.029) <i>z = 1.826</i>	.098 (.033) <i>z = 3.012</i>	.001 (.007) <i>z = .171</i>	.097 (.015) <i>z = 6.470</i>	.140 (.016) <i>z = 8.736</i>	1		
Depression	.055 (.018) <i>z = 2.965</i>	.106 (.039) <i>z = 2.720</i>	-.039 (.034) <i>z = -1.167</i>	.012 (.007) <i>z = 1.617</i>	.115 (.014) <i>z = 8.436</i>	.182 (.015) <i>z = 12.141</i>	.149 (.029) <i>z = 5.066</i>		
Past Year Substance Use	.044 (.014) <i>z = 3.215</i>	.119 (.023) <i>z = 5.289</i>	.124 (.026) <i>z = 4.760</i>	.015 (.006) <i>z = 2.352</i>	.097 (.016) <i>z = 6.171</i>	.139 (.017) <i>z = 8.123</i>	.290 (.034) <i>z = 8.551</i>	0.185 (.027) <i>z = 6.730</i>	1

Chi-square = 390.523 (158), p = .000

CFI = .942 TLI = .944 RMSEA = .037

Notes: z-scores greater than 1.96 are significant at alpha = .05 and z-scores greater than 2.58 are significant at alpha = .01; n = 1,436

of drug use contained the same items except illegal methadone use was substituted for sniffing glue.⁵⁸ Factor loadings were also adequate (.631 for marijuana use to .949 for heroin use) as was the scale reliability (.5988).

As was the case with offending, the substance use indicators for adolescence and the past year are repeated measures and therefore likely to have correlated error terms. For this reason, I allowed the error terms for identical observed indicators to covary. However, allowing the adolescent and past year indicators of cocaine to covary created a singular matrix, which could not be estimated. Therefore, in order to estimate the model, I specified that these two error terms remain independent. The residuals of several of the depression measures were also specified to correlate with each other, as were all the error terms that had been specified to covary in the offending measurement model.

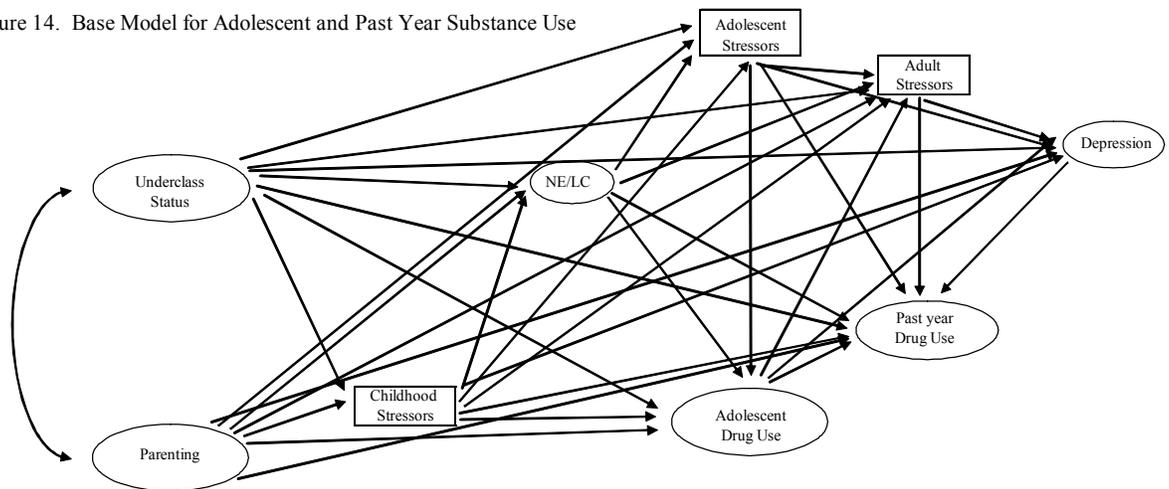
BASE MODEL

The base model for substance use is presented in figure 14 and the parameters are located in table 11. Like the base model for offending, the causal pathways were specified according to theory and proper temporal ordering. In the instances where two or more variables covered the same time period, theory dictated which variable was the cause and which was the effect. There is one exception to this, however. Even though depression refers to the past few weeks and past year substance use refers to the prior year, depression was specified to cause past year substance use. While this is not ideal, it allows for the incorporation of negative affect into the model, which is a key component of GST.

⁵⁸ The cell counts for methadone use were too low to include this item in the adolescent measure ($n = 1$, .07 percent), and the cell counts for glue sniffing were too low to include in the adult measure ($n = 2$, .14 percent).

Consistent with GST, there was a significant moderate effect of adolescent stressor exposure on adolescent substance use ($\beta = .272$), however, the lagged effect of childhood stressors was not significant ($\beta = -.068$). The relationship between adult stressor exposure and depression was significant and moderate to strong ($\beta = .363$), as predicted by the theory. In turn, depression had a significant association with past year substance use ($\beta = .159$). Note that the direct effect of stressors in adulthood on past year substance use was not significant ($\beta = .086$). Instead the effect of this variables on past year substance use was entirely mediated by depression. The indirect effect from adult stressors to past year substance use via depression was significant ($\beta = .058$) and accounted for just over 40 percent of the total effect of adult stressors on past year substance use.

Figure 14. Base Model for Adolescent and Past Year Substance Use



Not shown: sex, race, and age were included as control variables.

Table 11. Parameter Estimates for Substance Use Causal Base Model Controlling for Race, Sex, and Age

Standardized Parameters

Endogenous Variables	Exogenous Variables							
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Adolescent Substance Use	Adult Stressors	Depression
Childhood Stressors	0.06	0.052						
NE/LC	0.173	-0.033	0.063					
Adolescent Stressors	0.176	0.027	0.19	-0.001				
Adolescent Substance Use	0.03	0.063	-0.067	0.14	0.272			
Adult Stressors	0.069	0.048	0.113	-0.048	0.206	0.35		
Depression	0.019	0.117	-0.032	-0.102	0.156	0.117	0.363	
Past Year Substance Use	-0.063	0.161	0.04	0.134	0.052	0.574	0.086	0.159

Table 11 Continued. Parameter Estimates for Substance Use Causal Base Model Controlling for Race, Sex, and Age

Unstandardized Parameters

Endogenous Variables	Exogenous Variables							
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Adolescent Substance Use	Adult Stressors	Depression
Childhood Stressors	.03 (.019)	.018 (.017)	1					
	<i>z</i> = 1.581	<i>z</i> = 1.089		1				
NE/LC	.312 (.119)	-.04 (.074)	.221 (.156)		1			
	<i>z</i> = 2.62	<i>z</i> = -0.546	<i>z</i> = 1.413			1		
Adolescent Stressors	.176 (.044)	.018 (.035)	.372 (.049)	-.001 (.027)			1	
	<i>z</i> = 3.991	<i>z</i> = 0.515	<i>z</i> = 7.632	<i>z</i> = -0.03				
Adolescent Substance Use	.043 (0.09)	.061 (.061)	-.188 (.115)	.111 (.044)	.39 (.065)			
	<i>z</i> = 0.481	<i>z</i> = 0.995	<i>z</i> = -1.628	<i>z</i> = 2.51	<i>z</i> = 5.964			
Adult Stressors	.073 (.044)	.034 (.037)	.231 (.058)	-.028 (.03)	0.216 (.032)	.256 (.046)		
	<i>z</i> = 1.649	<i>z</i> = 0.914	<i>z</i> = 4.003	<i>z</i> = -0.918	<i>z</i> = 6.742	<i>z</i> = 5.629		
Depression	.028 (.085)	.117 (.074)	-.092 (.112)	-.084 (.044)	.231 (.058)	.121 (0.076)	.514 (.063)	
	<i>z</i> = 0.328	<i>z</i> = 1.578	<i>z</i> = -0.829	<i>z</i> = -1.89	<i>z</i> = 4.012	<i>z</i> = 1.597	<i>z</i> = 8.179	
Past Year Substance Use	-.081 (.065)	.014 (.04)	.1 (.104)	.096 (.024)	.067 (.063)	.516 (.069)	.105 (.075)	.138 (.061)
	<i>z</i> = -1.251	<i>z</i> = 3.499	<i>z</i> = 0.959	<i>z</i> = 3.969	<i>z</i> = 1.064	<i>z</i> = 7.493	<i>z</i> = 1.395	<i>z</i> = 2.269

Chi-square = 390.523 (158), *p* = .000

CFI = .942

TLI = .944

RMSEA = .037

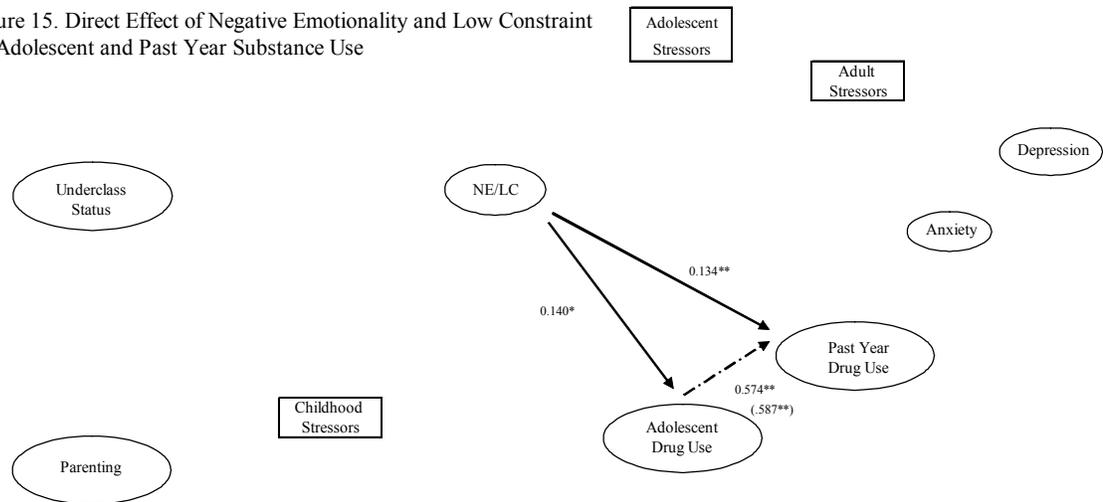
Notes: *z*-scores greater than 1.96 are significant at alpha = .05 and *z*-scores greater than 2.58 are significant at alpha = .01; *n* = 1,436.

DIRECT EFFECT

Agnew hypothesizes that negative emotionality and low constraint contribute to continuity in substance use through their direct effects on this behavior. Results indicate support for this hypothesis (see figure 15). There was a significant moderate relationship between NE/LC and adolescent substance use ($\beta = .140$) controlling for background characteristics, class, parenting, and childhood stressor exposure. Similarly, the relationship between NE/LC and past year drug use was moderate and significant ($\beta = .134$). This is fairly compelling evidence supporting the direct effect hypothesis because this relationship is independent of adolescent substance use. This should control for any time stable causes of substance use that are not included in the model and provides some protection against omitted variables bias.

There was further support for the direct effect hypothesis when the pathways from NE/LC to adolescent and past year substance use were fixed to 0. Eliminating these effects caused the standardized direct effect of adolescent substance use on past year substance use to increase from .574 to .587 and significantly worsens the fit of the model ($\chi_D = 15.442 (1), p = .0001$). While the entire association between adolescent and past year drug use is not spurious, controlling for NE/LC reduces the direct relationship between these two variables by about 2 percent.

Figure 15. Direct Effect of Negative Emotionality and Low Constraint on Adolescent and Past Year Substance Use



Chi Square from restricted model = 398.473 (128), $p = .0000$, CFI = .939, TLI = .939, and RMSEA = .038
 Chi-square difference test statistic comparing base model to restricted model = 15.442 (1), $p = .0001$

Not shown: Sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes: Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between NE/LC and adolescent substance use and NE/LC and past year drug use were constrained to be 0; Numbers not in parentheses are the standardized coefficients from the base model presented in table 11.

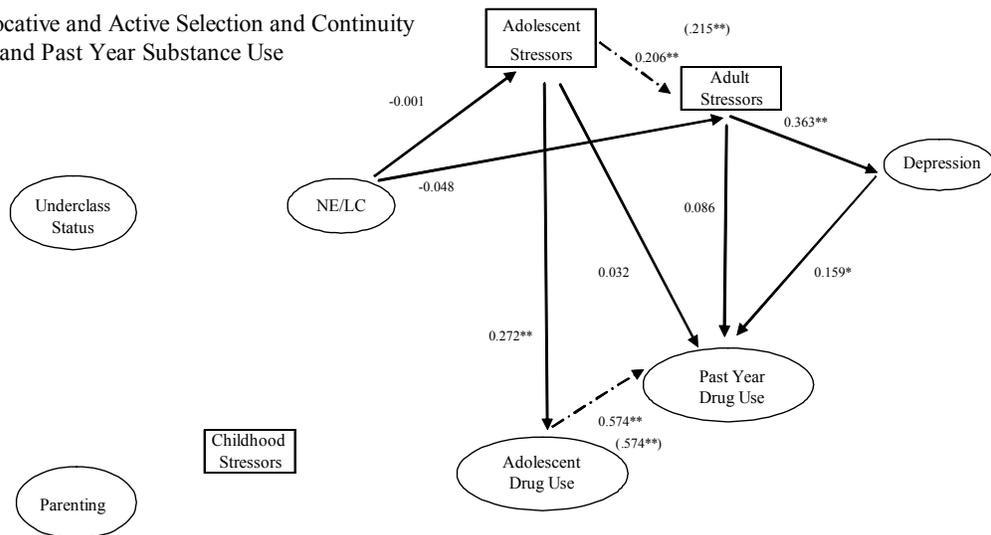
EVOCATIVE AND ACTIVE SELECTION

In evocative and active selection, individuals higher in NE/LC are more likely to elicit negative reactions from others and to actively seek out environments that are higher in stressors throughout their life span. The association between past and future substance use, therefore, is attributed to the continued indirect effect of NE/LC on drug use via exposure to stressors. Given that there was no support for this hypothesis in the offending model, it is not surprising that again there was no evidence to support the contribution of evocative and active selection to stability in substance use. First and foremost, there was no direct association between NE/LC and adolescent stressors ($\beta = -.001$) and NE/LC and adult stressors ($\beta = -.048$), and in fact, the relationship between these variables was negative and very small (see figure 16). Because there was no relationship between the exogenous variable (NE/LC) and the hypothesized mediators (adolescent and adult stressors), by definition (Baron and Kenny 1986) stressor exposure

cannot mediate the relationship between NE/LC and substance use. This was also evident in the size of the indirect effects of NE/LC on adolescent and past year substance use via exposure to stressors (NE/LC→adolescent stressors→adolescent substance use: $\beta = .000$; NE/LC→adolescent stressors→past year substance use: $\beta = .000$; NE/LC→adult stressors→past year substance use: $\beta = -.004$).

The irrelevance of evocative and active selection to continuity in substance use is further confirmed when the relationships between NE/LC and adolescent and adult stressors were fixed to 0. There was no significant change in the fit of the model to the data ($\chi^2_D = .864 (2), p = .6493$) and only a small increase in the standardized effect of adolescent stressors on adult stressors ($\beta = .215$).

Figure 16. Evocative and Active Selection and Continuity in Adolescent and Past Year Substance Use



Chi Square from restricted model = 387.585 (133), $p = .0000$, CFI = .942, TLI = .945, and RMSEA = .037
 Chi-square difference test statistic comparing base model to restricted model = .864 (2), $p = .6493$

Not shown : Sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes : Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between NE/LC and adolescent stressors and NE/LC and adult stressors were constrained to be 0;

Numbers not in parentheses are the standardized coefficients from the base model presented in table 8.

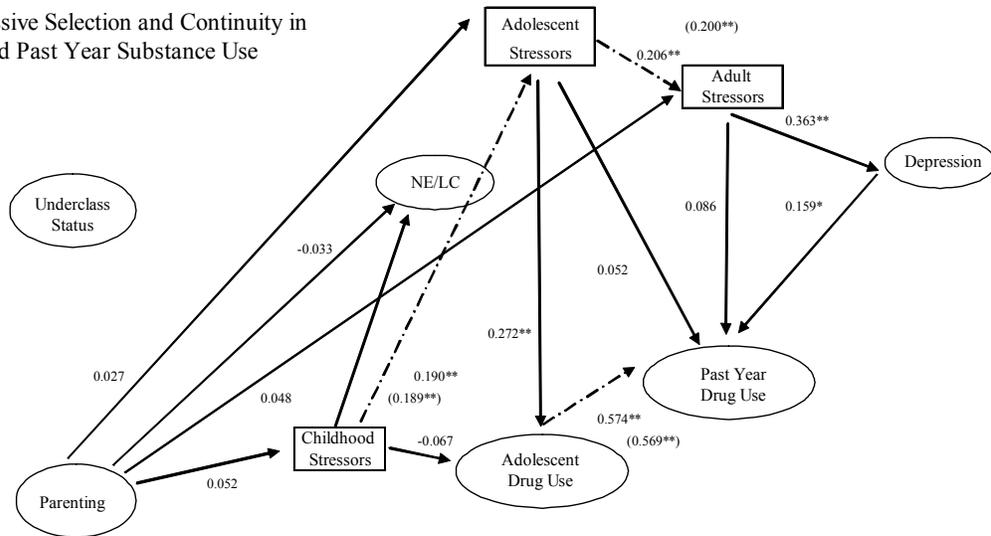
PASSIVE SELECTION

The substance use model again confirms the findings from the offending model: there is no evidence to indicate that continuity in illegal behavior is due to passive selection (see figure 17 and table 11). The data do not support any of the hypotheses regarding the origins of NE/LC. There was no direct relationship between parenting and NE/LC ($\beta = -.033$) and no indirect association via exposure to stressors in childhood ($\beta = .003$). Furthermore, there was also no support for the hypothesis that parental behavior is associated with substance use indirectly through exposure to stressors. Parenting was not related to exposure to stressors in childhood, adolescence, or adulthood ($\beta = .052$, $\beta = .027$, and $\beta = .048$, respectively).

Setting the relationships between parenting and stressor exposure to 0 provided further evidence against passive selection. There was no significant change in the fit of the model ($\chi_D = 2.822$ (3), $p = .4199$) and there was little change in the relationship between stressors or adolescent and past year substance use.

Note that even though parenting did not explain continuity in substance use over time in the manner predicted by GST, it was not totally irrelevant. Parenting did have a moderate direct association with past year substance use ($\beta = .161$), which is surprising given the long lag between the measures (see table 11). This association is not due to the effect of retrospective reporting bias because parenting was measured using observations recorded by trained psychologists and not using self reports.

Figure 17. Passive Selection and Continuity in Adolescent and Past Year Substance Use



Chi Square from restricted model = 400.863 (134), $p = .0000$, CFI = .940, TLI = .942, and RMSEA = .037
 Chi-square difference test statistic comparing base model to restricted model = 2.822 (3), $p = .4199$

Not shown: Sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes: Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between all stressor measures and parenting, NE/LC and parenting, and NE/LC and childhood stressors were constrained to be 0; Numbers not in parentheses are the standardized coefficients from the base model presented in table 11.

STRESS PROLIFERATION

Continued exposure to stressors can be a result of processes other than passive selection, such as stress proliferation. Stress proliferation is based on the idea that stressors can actually create additional stressors, controlling for an individual's actions, environment, and temperament. Thus, continuity in substance use is due again to the chronicity of stressor exposure, but it results from the causal effect of past stressors on future stressors rather than characteristics of individuals or their environments.

As was found with offending, childhood and adolescent stressor exposure and adolescent and adult stressor exposure had significant moderate relationships with one another controlling for all causally prior variables in the model ($\beta = .190$ and $\beta = .206$, respectively) (see figure 18). Also consistent with stress proliferation, childhood stressor exposure had an indirect association with adolescent substance use through adolescent

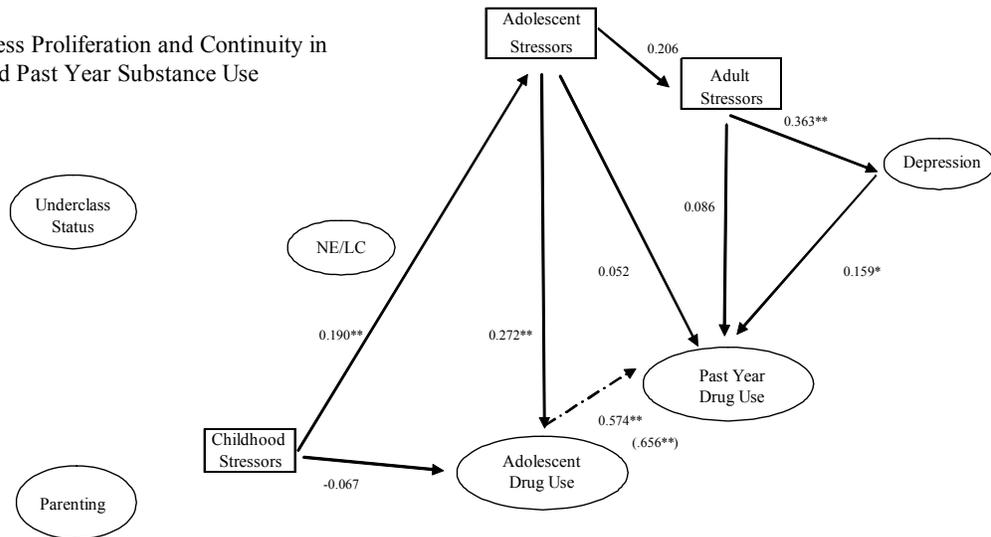
stressor exposure ($\beta = .052$), however, its relationships with past year substance use through adolescent stressors ($\beta = .010$), through adolescent and adult stressors ($\beta = .003$), and through adolescent and adult stressors and depression ($\beta = .002$) were small and non-significant. Although adolescent stressor exposure had a direct effect on exposure in adulthood, adult stressors did not significantly mediate its relationship with past year substance use.

When the relationships between childhood stressors and adolescent and adult stressors and the relationship between adolescent and adult stressors were fixed to 0, the model fit worsened significantly ($\chi_D = 84.570 (2), p = .000$) and the standardized direct relationship between adolescent and past year substance use increased from .574 to .656.

Again, even after controlling for adolescent stressors, childhood stressors continued to have a significant direct association with adult stressors ($\beta = .113$) in addition to its significant indirect effect on adult stressor exposure via exposure to stressors in adolescence ($\beta = .039$).

In sum, the findings suggest some support for the contribution of stress proliferation to continuity in illicit behavior. Exposure to stressors in childhood was significantly associated with exposure to stressors in adolescence and adulthood and stressors in adolescence were significantly associated with stressors in adulthood. Furthermore, eliminating the effects of past on future stressors worsened model fit and increased the association between substance use in adolescence and the past year, suggesting that at least part of the association between these behaviors operates through the process of stress proliferation. However, childhood and adolescent stressors failed to have an indirect effect on past year substance use via exposure to later stressors.

Figure 18. Stress Proliferation and Continuity in Adolescent and Past Year Substance Use



Chi Square from restricted model = 411.917 (134), $p = .0000$, CFI = .937, TLI = .940, and RMSEA = .038
 Chi-square difference test statistic comparing base model to restricted model = 84.570 (2), $p = .0000$

Not shown : Sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes : Numbers in parentheses are the standardized coefficients from the restricted model in which the relationships between childhood and adolescent stressors and adolescent and adult stressors were constrained to be 0; Numbers not in parentheses are the standardized coefficients from the base model presented in table 11.

AMPLIFICATION

Continuity in substance use may result from the reciprocal relationship between stressors and criminal behavior, a process which is set in motion by membership in the lower class. Here, individuals living in poverty with little education are exposed to more stressors in adolescence and childhood by virtue of their social status. Individuals may respond to stressors with criminal behavior, which in turn creates additional stressors that individuals may deal with through illegitimate coping mechanisms.

Consistent with amplification, low SES was significantly and moderately associated with adolescent stressors ($\beta = .176$), however, contrary to predictions the association between class and childhood stressors was not significant ($\beta = .060$) (see figure 19). Again, this may be a function of the types of stressors included in the

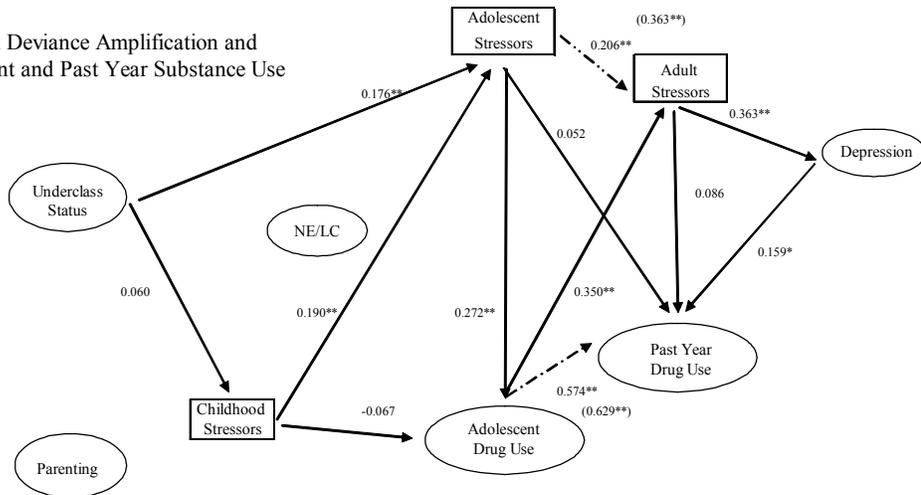
childhood stressors measure. Although some of the stressors included in this measure are probably more prevalent among the severely disadvantaged (e.g., sexual abuse, death of mother), they are also relatively exogenous and therefore we may not observe a difference in childhood stressor exposure by class status.

Importantly, as predicted by amplification, adolescent substance use had a relatively strong direct relationship with adult stressor exposure ($\beta = .350$) and it partially mediated the association between adolescent and adult stressors ($\beta = .095$). This indirect pathway accounted for almost 32 percent of the total association between adolescent and adult stressors. The indirect relationship between adolescent substance use and past year substance use was more complicated. The indirect effect of adolescent substance use on past year substance use via adult stressor exposure was not significant ($\beta = .030$) (recall that adult stressor exposure did not have a direct effect on past year drug use, and therefore cannot serve as a mediator); however, the indirect effect from adolescent to past year substance use via adult stressors and depression was significant, but smaller ($\beta = .020$). Together, these two indirect pathways account for about 8 percent of the total relationship between adolescent and past year substance use. As was the case with offending, the amplification hypothesis seems to do a better job of explaining continuity in stressors than continuity in substance use.

Fixing the relationship between delinquency and adult stressors to 0 worsened the model fit ($\chi^2_D = 43.417 (1), p = .0000$). In addition, it resulted in a moderate change in the relationship between adolescent and past year drug use. This standardized parameter increased from .574 to .629. In contrast, the change in the relationship between adolescent and adult stressors was larger going from .205 to .363 and again suggests that

the amplification hypothesis does a better job of explaining continuity in stressor exposure than continuity in substance use.

Figure 19. Stressor and Deviance Amplification and Continuity in Adolescent and Past Year Substance Use



Chi Square from restricted model = 402.371 (133), $p = .0000$, CFI = .939, TLI = .941, and RMSEA = .038
 Chi-square difference test comparing base model to restricted model = 43.417 (1), $p = .0000$

Not shown: Sex, race, and age were included as control variables.

* $p < .05$; ** $p < .01$

Notes: Numbers in parentheses are the standardized coefficients from the restricted model in which the relationship between adolescent substance use and adult stressors was constrained to be 0; Numbers not in parentheses are the standardized coefficients from the base model presented in table 11.

TRIMMED FINAL SUBSTANCE USE MODEL

All nonsignificant pathways were fixed to 0 to create the final trimmed model of substance use (figure 20 and table 12). There was a significant improvement in model fit ($\chi^2_D = 35.349 (13)$, $p = .0007$), however fit indices are ambiguous regarding the adequacy of the overall fit of the model. The ratio of the χ^2 to the degrees of freedom and the RMSEA indicate adequate model fit. The χ^2/df is 2.9, which is less than the recommended cut off value of 3 and at .036 the RMSEA is less than the suggested upper limit of .05. The CFI is .944 which is above the lower limit set by Yu and Muthén (2002) but below the lower limit of .96 recommended by Yu (2002) for dichotomous outcomes. Given that the χ^2 statistic is the only fit index for WLSMV that has been studied

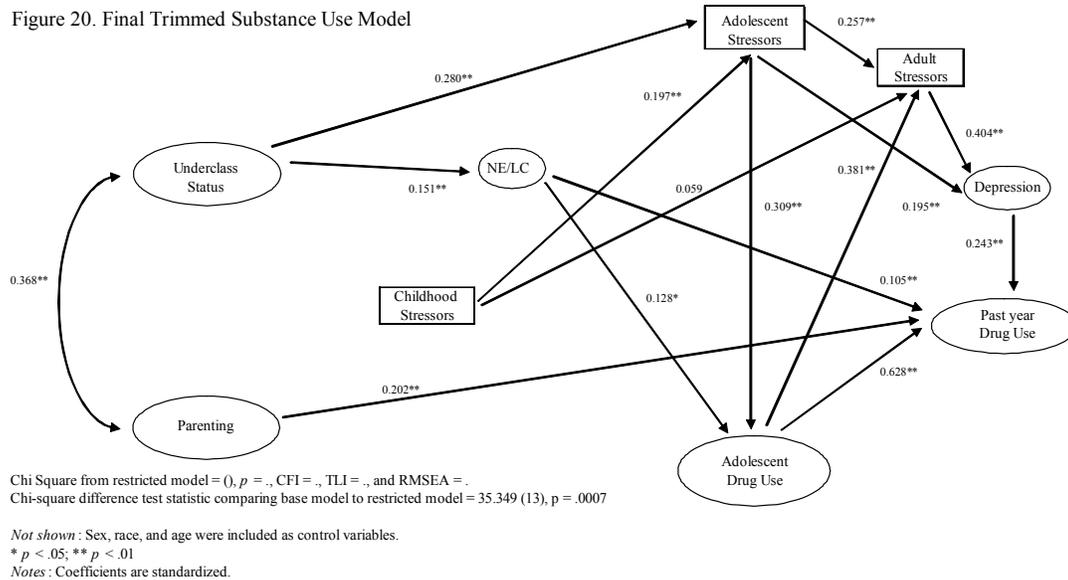
extensively with Monte Carlo simulations, (Muthén and Muthén 2006), it is probably safe to conclude that the model fit is adequate.

First and foremost, this final substance use model supports GST in that adolescent stressors were associated with adolescent substance use, and more significantly, depression mediated the effect of adult stressors on past year substance use. Of course these findings should be interpreted with caution. It is likely that the effect of stressors on drug use is overestimated because the reciprocal relationship between the two has not been taken into account. In addition, recall that depression over the last few weeks is specified to cause past year substance use. The current research cannot disentangle the causal ordering of depression and substance use, but this should be a priority of future work.

With regards to the pathways hypothesized to contribute to continuity in illegal behavior, the final trimmed model for substance use is in many ways similar to that for offending. Like the offending model, stress proliferation and amplification both explain some of the association between adolescent and past year substance use. Moreover, there is no evidence to suggest that passive selection or evocative and active selection are responsible for stability in substance use over time, at least not when selection is based on the traits of negative emotionality and low constraint. However, there is one major difference between the offending and substance use models: negative emotionality and low constraint have a direct effect on substance use in adolescence and the past year and this partially accounts for the association between these behaviors over time. Fixing the effect of NE/LC on adolescent and past year substance use to 0 increases the direct effect of adolescent substance use on adult substance use by 4.5 percent from .628 to .656. This

change is relatively small, but recall that the indicators of negative emotionality and low constraint consist of observations taken by psychologists when the respondents were 7 years old.

With the exception of the direct effect of NE/LC on substance use, the findings from the two outcome variables, drug use and offending, provide a convergent picture.⁵⁹ Both models suggest that stress proliferation and amplification contribute to continuity in criminal behavior, while evocative, active, and passive selection play little or no part in this process. In the next section, I examine whether the effects of stressors on drug use are moderated by NE/LC or exposure to stressors in childhood.



⁵⁹ Supplemental analyses in which depression was left out of the model to make the results across outcomes more comparable, lead to the same substantive conclusions. Of particular interest is that NE/LC continued to have a significant direct effect on adolescent and past year substance use. The only difference between the models is that in the model without depression, there was a direct effect of adult stressors on past year substance that was not apparent in the model including depression. This is consistent with the notion that depression mediates the effect of stressors on past year substance as predicted by GST.

Table 12. Parameter Estimates for the Final Substance Use Model Controlling for Race, Sex, and Age

Standardized
Parameters

Endogenous Variables	Exogenous Variables							
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Adolescent Drug Use	Adult Stressors	Depression
Childhood Stressors								
NE/LC	0.151							
Adolescent Stressors	0.28		0.197					
Adolescent Drug use				0.128	0.309			
Adult Stressors			0.059		0.257	0.381		
Depression							0.404	
Past Year Drug use		0.202		0.105		0.628		0.243

Table 12 Continued. Parameter Estimates for the Final Substance Use Model Controlling for Race, Sex, and Age

Unstandardized Parameters (Standard errors in parentheses; z-score in italics)

Endogenous Variables	Exogenous Variables							
	Low SES	Parenting	Childhood Stressors	NE/LC	Adolescent Stressors	Adolescent Drug Use	Adult Stressors	Depression
Childhood Stressors								
NE/LC	0.279 (.095)							
	<i>z = 2.93</i>							
Adolescent Stressors	.277 (.047)		.373 (.057)					
	<i>z = 5.89</i>		<i>z = 6.56</i>					
Adolescent Drug use				.101 (.040)	.458 (.068)			
				<i>z = 2.511</i>	<i>z = 6.777</i>			
Adult Stressors			.120 (.063)		.277 (.043)	.278 (.048)		
			<i>z = 1.97</i>		<i>z = 6.478</i>	<i>z = 5.833</i>		
Depression					.301 (.062)		0.576 (.055)	
					<i>z = 4.836</i>		<i>z = 10.462</i>	
Past Year Drug use		.176 (.042)		.075 (.022)		.564 (.067)		.210 (.049)
		<i>z = 4.167</i>		<i>z = 3.429</i>		<i>z = 28.405</i>		<i>z = 4.317</i>

Chi-square = 375.154 (129), p = .000 CFI = .944 TLI = .945 RMSEA = .036

Notes: z-scores greater than 1.96 are significant at alpha = .05 and z-scores greater than 2.58 are significant at alpha = .01; n = 1,436.

MODERATING EFFECTS

Differences in the Strain-Substance Use Relationship by Level of NE/LC

To examine the interaction of NE/LC and stressors, individuals were characterized as low or high in NE/LC based on the same criteria described in the offending analysis. Again, an interaction term was created by multiplying the dichotomous NE/LC variable (scored 1 if the individual scored high in NE/LC and 0 otherwise) by adolescent and adult stressors, respectively. The interaction term for adolescent stressors and high NE/LC was included in the model as a predictor of adolescent substance use. The interaction between adult stressors and NE/LC was included as a predictor of depression. This is because the effect of exposure to stressors in adulthood was completely mediated by this measure of negative affect.

The results only partially support the hypothesis that the effect of stressors on drug use and depression is stronger for individuals higher in NE/LC. High NE/LC did not moderate the effect of adolescent stressors on adolescent substance use ($\beta = .052$), however, the effect of exposure to stressors in adulthood on depression was stronger for individuals high in these traits ($\beta = .162$).

Past Experience as a Source of Population Heterogeneity

The moderating effect of childhood exposure to stressors on the relationship between stressors, substance use, and depression later in life was examined following the same procedure used for offending except the interaction of adult stressor exposure with childhood stressor exposure was regressed on depression rather than adult substance use. The results were in concordance with those found for offending. Exposure to at least one

childhood stressor seems to enhance the relationship between adolescent stressors and adolescent substance use ($\beta = .138$) and adult stressors and depression ($\beta = .262$).

Therefore the results for both outcomes of interest illustrate the importance of studying behavior within the context of individuals' personal histories.

SUMMARY OF FINDINGS FOR SUBSTANCE USE

The findings from the examination of substance use support the key premise of GST: Stressors are associated with increased participation in substance use and this relationship appears to be mediated, at least with regards to past year substance use, by depression. Of course these results must be interpreted with caution given the failure to account for the non-recursive relationship between strain and illegitimate coping mechanisms and the issues of causal ordering.

The key question, however, is can GST explain stability in substance use over time? The substance use findings were somewhat mixed. There was support for the hypothesis that part of the stability in substance use over time is due to time stable differences in negative emotionality and low constraint. Moreover, as predicted, the strength of the relationship between depression and adult stressors is stronger for individuals who exhibit high levels of negative emotionality and low constraint in childhood; however there was no moderating effect of these traits on the adolescent stressors-substance use relationship. In contrast, childhood exposure to stressors enhanced the effect of both adolescent and adult stressors as predicted. This implies that differences in early life experiences as well as temperament can influence a person's reaction to stressors.

There was no support for evocative and active selection or passive selection based upon the traits of negative emotionality and low constraint. However, stress proliferation and amplification both accounted for a small portion of the relationship between adolescent and past year substance use, with a large portion of the direct association between these variables left unexplained.

CHAPTER V: DISCUSSION AND CONCLUSIONS

This study is one of the first attempts to examine the applicability of GST to understanding continuity in offending over time. By simultaneously examining all four stability promoting mechanisms that have been outlined by Agnew (1997, 2006), this work explored both the social origins of stressors as well as the role of individual differences in shaping reactivity to and exposure to stressors. Overall, the findings suggest that it is the former that plays the greater role in maintaining offending over time. Furthermore, I draw from the life-course perspective to understand how stress proliferation and past experiences (Wheaton and Gotlib 1997) can promote continuity in offending.

Within the GST framework, the key to understanding continuity in offending and substance use is understanding continuity in stressors. With the exception of the direct effect hypothesis, all of the pathways through which past and future illegal behavior are linked rely on the stability of stressors over time. Where each pathway differs is to what it attributes the stability in stressors. In the evocative and proactive model, stressor exposure is hypothesized to remain stable over time because individuals with high negative emotionality and low constraint actively seek out the types of environments that tend to be high in stressors and because they elicit negative reactions from others. Continuity in stressors in the passive selection model is attributed to living in a family in which parents create a stressful home environment, while stress proliferation posits that stressors actually create new stressors, apart from any behavior on the part of the individual. In contrast, the amplification hypothesis predicts that individuals' law

violating behaviors sustain their exposure to stressors as exposure to stressors simultaneously sustains law violating behavior.

Overall this research provides mixed support for GST explanations of stability in offending and substance use over time. Generally, there is more evidence in favor of those pathways to stability that are based on sociogenic processes as opposed to individual differences. Only two findings give credence to the notion that negative emotionality and low constraint play a central role in the continuity of illegal behavior. First, results suggest that negative emotionality and low constraint may have a direct relationship with adolescent and past year substance, which accounts for some of the association between these behaviors over time. In contrast, no such direct effect was found for delinquency and offending. These findings are not without precedence. In a sample of adolescents, Kardum and Krapic (2001) found that neuroticism was associated with a tendency to engage in avoidance coping, but not emotion or problem focused coping, and that part of this effect was mediated by higher levels of subjective stress. They argue that this relationship may be explained by the tendency of those high in neuroticism to experience more stressors and to react more strongly to the stressors they experience. Avoidance coping, of which substance use is one type, provides a way for these individuals to bring their aversive feelings under control. However, given that substance use is associated with an increase in future stressors, this coping mechanism may create more stressors and psychological distress than it alleviates.⁶⁰

Similarly, these findings may indicate that individuals higher in negative emotionality and low constraint are more likely to experience inwardly directed

⁶⁰ The validity of this explanation was not tested here because analyses were not run separating “pure” drug users (those who engaged in drug use only and not other illegal behavior) from “pure” offenders (those who engaged in crime, but did not engage in substance use).

emotions, such as depression, that are associated with avoidance coping mechanisms like substance use. If this were the case I would expect to see a significant relationship between NE/LC and depression, which was not the case. However, this null finding may be a function of the long lag between the measurement of NE/LC and depression. One thing is certain, however: Future research using more appropriate lags and measures is needed to better understand why NE/LC has a direct effect on substance use but not offending.

The second way in which negative emotionality and low constraint were found to contribute to continuity in offending is by enhancing the effect of stressors on delinquency, offending, and past year substance use. This is consistent with Agnew and colleague's (2002) findings. The implication of these findings for contemporaneous effects of stressors on illegal behavior is relatively obvious— all things equal, individuals high in negative emotionality and low in constraint, should be more likely to utilize illicit coping mechanisms, either because they interpret innocuous situations as threatening or because of impulsive actions. The implication for continuity, on the other hand, is less straightforward. Individuals high in these traits may be more likely to engage in illegal behavior, which, in accordance with stress and deviance amplification, creates additional adversity and stressors. Then, because these individuals are high in negative emotionality and low constraint, they will be more likely to respond to these new stressors with illegal behavior, thus continuing the amplifying loop. Note that the effect of adolescent stressors on adolescent substance use did not vary significantly by level of negative emotionality and low constraint. It is not immediately obvious why there is a significant interaction effect for adolescent delinquency and adult offending and depression but not

for adolescent substance use. It is possible that this null finding is a period or cohort effect. Recall the sample entered adolescence during the mid 1970s, a time period when drug use, especially marijuana, was seen by many to be an acceptable form of recreation. Because the substance use measure does not account for the frequency of use, it may not be sensitive enough to distinguish recreational use from the more problematic use that might indicate avoidance behavior associated with heightened reactivity to stressors.

Support was also found for the existence of another moderating effect. There was a significant interaction between past and future stressor exposure such that individuals who experienced stressors in childhood were more likely than others to react to later stressors with substance use and offending. Similarly, the relationship between stressor exposure and depression was also enhanced for those exposed to stressors in childhood. Therefore, consistent with the life-course perspective, an individual's personal history should be taken into account when studying offending and substance use (Wheaton and Gotlib 1997).

There are several possible reasons why individuals with a history of exposure to severe stressors are more likely to engage in illegal behavior: Stressor exposure may lead to a biological change in brain functioning (Fishbein 2001); limit access to future coping mechanisms or resources, such as parental social support; or negatively affect an individual's social position. Future research is needed to determine which, if any, of these explanations hold true.

Contrary to hypotheses, no support was found for evocative and active selection. Individuals higher in NE/LC did not experience more stressors than other individuals, and this held true even when the data were reanalyzed using measures of stressors that

omitted independent or exogenous life events. Moreover, NE/LC did not account for any of the association between adolescent and adult stressors, which would be predicted by evocative and active selection.

It is possible that my null findings are due to the inclusion in my stressor measure of too many exogenous events or situations. However, this is unlikely because these null findings were replicated in supplemental analyses where exogenous stressors were omitted from the stressor measures. Moreover, Magnus and colleagues (1993) found an association between neuroticism and a measure of exogenous stressors.

It is important to note that the null finding regarding evocative selection is inconsistent with many, but not all, past studies. For example, Ormel and colleagues (2001) found no evidence to suggest that exposure to stressors mediated the relationship between neuroticism and depression, and Van Os et al. (2001) reported that while the relationship between neuroticism and negative life events was significant, it was substantively very small. If the results presented here are real findings, they imply that Agnew's focus on individual differences as the primary source of stressors may be misplaced. Instead, exposure to stressors may be more strongly associated with social position. However, further exploration is needed to understand why this study, along with several others, fails to find a relationship between temperament and exposure to stressors.

Null findings were also found for the passive selection hypothesis; parenting behavior did not have a direct effect on exposure to stressors nor were parenting behavior or exposure to stressors in childhood related to NE/LC. This may partially be attributable to my measure. First, parenting behavior probably only captures the most extreme cases

of poor parenting given the setting of data collection (a hospital clinic), which may be more likely to put parents on their best behavior. Moreover given the age at which parenting was measured (8 months), the data may not be representative of the manner in which the mother treated her child later in life. Finally, no measures of paternal parenting practices were included in the study.

Combined, the lack of support for evocative and active selection and passive selection suggest that that NE/LC may not lead to stability in offending by structuring the amount of stressors a person experiences. Rather, the significant moderating effect of NE/LC on the stressor-illegal behavior relationship indicates that individual differences may be important because they condition the effect of stressors on illicit coping. In addition, given the differences in the direct effect of NE/LC on offending versus substance use, these traits may also condition the type of illicit coping an individual uses.

In contrast to active and evocative and passive selection, stress proliferation did contribute a small amount to the explanation of continuity in offending and substance use. Results indicate that past stressors may have a causal effect on future stressors, controlling for an individual's actions. Life trajectories may be set in motion by experiences in childhood. Wheaton and Gotlib (1997) describe how chronic sexual abuse may increase the likelihood a person drops out of school, enters into an unstable marriage, or has an unsuccessful career. "The abuse changes the person's trajectory by virtue of a number of possible mechanisms, such as altering one's identity at crucial developmental junctures, or leading to leaving home at an earlier age, or through lowering self-esteem or through poorer performance in school. These mechanisms, in

turn, underscore the chain reaction notion of a trajectory: once a trajectory is defined, events tend to have accumulating consequences (Wheaton and Gotlib 1997, 3).

Although it was hypothesized that the effect of childhood stressors would be fully mediated by adolescent stressors, this was not so. In fact, the majority of the relationship between childhood and adult stressors was direct. This may be partially due to omitted variable bias. That is, part of their association may stem from a relatively time stable common cause that is not included in the model such as a different aspect of temperament, neighborhood characteristics, and the like. It is also probable that part of this association may be due to a real direct effect of childhood stressors on both adolescent and adult stressors. Stressors may lead to changes that have an enduring effect into adulthood, such as identity changes or even changes in the biological stress response (Fishbein 2001).

The amplification hypothesis, which also stresses the importance of accumulating consequences, was also supported for both offending and substance use. In the amplification process, individuals who are members of the lower class get caught in a cycle in which their own illegal behaviors increase their exposure to stressors which in turn leads to more illegal behavior. Low SES was associated with adolescent stressors. Delinquency and adolescent substance use mediated the relationship between adolescent and adult stressors, while adult stressors mediated the relationship between adolescent and adult offending. However, contrary to predictions there was no relationship between class and childhood stressors or childhood stressors and adolescent illegal behavior. The most likely reason for these null findings is the narrow scope of the childhood stressor measure. In comparison to the later measures of stressors, the childhood measure is made

up for the most part of relatively rare and severe events, like death of a mother, sexual abuse, parental divorce, and an activity limiting illness or injury. Even the more common stressors included in this measure, like moving or failing a grade, tend to be isolated events. What they all lack is an element of chronicity or the “cumulative effect of a steady, day-after-day drip of water on a rock” (Menaghan 1997, 114). It is these chronic stressors, not necessarily stressful life events, which are believed to have the most damaging effects. Not only do they deplete coping resources and reduce levels of social control, but they also contribute to negative emotional traits like anger and depression (Agnew 2006). Future research that examines GST over the life span should make a concerted effort to capture chronic stressors in childhood.

It is anticipated that these various stability producing mechanisms operate simultaneously. For example, negative emotionality and low constraint or exposure to stressors in childhood may increase the likelihood an individual engages in illicit coping when exposed to stressors. In addition, experiencing a stressor could set in motion the process of stress proliferation thereby maintaining illegal behavior via sustained stressor exposure. Simultaneously, law violating behavior may create additional life adversities which may be dealt with via offending or substance use. The original stressor may be an exogenous chance event, like the death of parent, or it could be rooted in an individual’s position in the social structure or behavior.

Overall, the findings suggest that although negative emotionality and low constraint play a role in maintaining offending from adolescence to adulthood, these traits may not take on as much importance as is attributed to them by GST. Instead, it is the

more dynamic aspects of the stress process, like proliferation and amplification, which contribute the most to stability in behavior over time.

POLICY IMPLICATION

One finding to come out of this study is the long reaching effects of childhood stressors. Experiencing stressors in childhood not only increased exposure to stressors in adolescence and adulthood, but also enhanced the effects of these stressors on offending and substance use. This suggests that intervention programs should be developed that target childhood stressors. Although it would be ideal to prevent the occurrence of childhood stressors, this may not be possible given that many of the events included in my measure are amenable to interventions (death of a parent, illness or injury). However, programs should be explored that teach pro-social coping skills.

FUTURE RESEARCH

The findings from this study suggest several areas that need to be explored further. The current work supports the notion that stressors and the negative affect associated with them play a role in maintaining stability in offending over time, partially through an amplification process. However, GST is not the only theory to use this concept to explain continuity. Sampson and Laub (1993, 1997) also describe a process of cumulative disadvantage, but the ramifications of criminal behavior attenuate social bonds (rather than increase stressors), which leads to more illegal activity. Although, in support of GST, the current work did find that offending increases exposure to stressors and that depression mediates the relationship between stressors and substance use, there

were no controls for alternate explanations. Thus, future research should begin by examining if stressors and emotions still matter once controls for social bonds have been introduced. If so, the next step is to examine how these two constructs operate in conjunction to produce stability in offending and substance use over time. For instance it is possible that the effect of illegal behavior on social bonds is mediated by exposure to stressors or that social bonds moderate the effect of offending and substance use on stressors by serving as a source of social support.

This work further confirms that the dichotomy between individual differences and context (Laub and Sampson 2003) is false. Consistent with a mixed model approach to stability, past and future offending were linked through processes that were both state dependent (i.e., stress proliferation and amplification) and population heterogenic (i.e. the direct effect of NE/LC on substance use and depression). What is not clear, however, is the manner in which these processes are linked. Do they operate simultaneously? Is there a synergism between the processes such that one enhances or maintains the effect of the other? Or, consistent with a taxonomy approach, do different processes dominate for different people? Agnew (1997, 2006) hypothesizes that these processes operate in different people: For people high in NE/LC, criminal behavior is maintained via a direct effect of these traits and passive, active, and evocative selection. In other individuals, amplification explains continuity. However, this assumes that there is some threshold of NE/LC and that for people scoring above this threshold continuity is the result of one set of processes and for those scoring below, it is the result of a separate set of processes. This explanation may be overly simplistic; it seems more likely that for almost all people, these processes are simultaneously at work, reinforcing one another.

In addition, it was beyond the scope of the current study to examine whether the findings hold for different subgroups and across different contexts. A consistent finding in the GST literature is that the strain-negative affect-offending relationship varies by gender. Gender differences have been found in the strength of the association between strain and offending (Agnew and Brezina 1997; Hay 2003), the types of stressors which are criminogenic (Mazerolle 1998), the types of emotions that result from stressors (Ostrowsky and Messner, 2005; Piquero and Sealock 2004), and the types of behaviors used to cope with emotions (Mazerolle and Piquero 1998; Ostrowsky and Messner 2005; Piquero and Sealock 2004). Consistent with these studies, exploratory analyses presented in appendix B indicate that in this sample, gender moderates the magnitude of the relationship between stressor exposure and offending, with the association between these two variables being stronger for males. Given these preliminary findings, combined with the vast literature on GST and gender differences, it is important to know if and how the effects of stressors on offending, substance use, and depression vary across the life span by gender.

It is also possible that the processes leading to stability in illegal behavior may vary by ecological context. For example, there is evidence that suggests that the effects of individual differences may differ as a function of neighborhood disadvantage. No study examines this exact issue, but some are suggestive. Lynam and colleagues (2000) found that impulsivity had a stronger relationship with delinquency in more disadvantaged neighborhoods. These authors suggest that internal constraints play a more important role in controlling behavior in environments where social controls are lacking. Other work has also found that neuropsychological factors, like IQ, have a

stronger association with delinquency in disadvantaged neighborhoods (e.g. Moffitt 1997). Although supplemental exploratory analyses found no evidence that neighborhood disadvantage moderates the stressor-offending relationship as was the case with gender (see appendix B), it is still not known if the effects of NE/LC on illegal behavior and exposure to stressors are stronger for those in more disadvantaged neighborhoods. This question should be taken up in future work.

One framework that might help make sense of the interplay of these processes and interactions discussed above is an ecological perspective. Ecological perspectives are based on the notion that development takes place through a process of progressively more complex reciprocal interactions between an organism and its immediate surroundings (e.g. people, objects, symbols). The ecological perspective “see[s] context not just in terms of variables to be controlled but as ecological niches worthy of investigation” (Moen 1995, 9). As such, behavior and development can only be understood as a joint function of the characteristics of the person and the environment. The former include traits that are biological, like genetic makeup, and psychological, like personality. The latter incorporate family, school, and neighborhood contexts, as well as broader social, cultural, and historical settings (Moen 1995).

Another cornerstone of ecological paradigms is the importance of interactions. Although the ecological perspective does emphasize the moderating effect of variables, in this case the term “interaction” refers to dynamic and reciprocal transactions between an individual and his or her environment (Caspi, Elder, and Bem 1987; Magnus et al. 1993). Through these interactions people can create and shape their environment, while context simultaneously acts upon them. Moreover, just as people change over time, so do the

environments in which they live. For example, families move from one neighborhood to another, neighborhoods evolve, and adolescents become adults, choosing their own place to live. As context changes over time, so do the types of stressors to which one is exposed. Thus, the most complete picture of offending is one that incorporates the person, time, and context as well as their interaction.

LIMITATIONS

As is the case with most research, the study limitations should be considered when evaluating the results. Covering such a large span of the life course required making several tradeoffs and what was gained in breadth was lost in specificity. Although the data cover an extensive period of the life course (birth to 27/33 years), the way in which measures of illicit behaviors were recorded forced me to divide the life course into three lengthy developmental stages: childhood, adolescence, and adulthood. It was thereby impossible to examine more nuanced relationships in the development of stressors and illegal behavior over time, especially the reciprocal relationships between stressors and offending, and it was not possible to ensure the proper causal ordering of variables by using lagged measures. As a result, it is highly likely that the estimates of the relationships between stressors and offending are inflated.

The issue of timing is especially problematic for the substance use models in which past year substance use was regressed on respondents' self reports of depression. This suggests that it may be substance use that leads to depression, not vice versa. Future work should be done to replicate the findings presented here using data that allow for the

correct causal ordering of key theoretical variables (i.e., stressors→emotions→illegal behavior) and shorter lag times separating the measures.

A second limitation of the current study is one that is common to many catch-up studies—differential attrition. Recall that individuals most likely to engage in illegal activity and to experience stressors were disproportionately less likely to be in the follow-up sample. This differential attrition may have decreased the variability in the outcome and stressor variables and, therefore, I may have underestimated the relationships between NE/LC, criminal behavior, and stressors. A second possible explanation for the failure to find significant associations between NE/LC and my key variables of interest is that NE/LC is not as stable as I assumed. Although there is research suggesting that there exists a good deal of stability in personality over time, there is also room for change. This is especially true when temperament is measured at a young age, because the stability of traits tends to increase as individuals age. The current study used a measure of NE/LC taken at age 7 and hence it is possible that the temperament of the participants changed significantly over time. Future studies should avoid this issue by measuring temperament at multiple points in time. Finally, the measure itself may be flawed. No standardized scales of temperament existed at the time of the CPP and therefore it is difficult to assess the validity and reliability of my NE/LC measure. Some may argue with the items contained in the measure, however, they do map onto Agnew's descriptions of negative emotionality and low constraint. Yet, considering the low frequency of participants scored as possessing any of these characteristics, it is likely that the measure is too conservative. This also creates a lack of variability on this trait which may have contributed to the null findings. Future research should replicate the analysis

in the current work using one of the available validated measures of temperament like Achenbach's (1974) Child Behavior Checklist.

Negative emotionality and low constraint are not the only measures that are wanting. Given that the distinguishing feature of GST is the role that negative affect plays in mediating the stressor-offending relationship, this study would be greatly enhanced with better measures of emotion, especially anger. Moreover, different emotions may be associated with the use of different types of coping mechanisms; specifically anger is more likely to be associated with outwardly directed behaviors like aggression while depression more often leads individuals to use inwardly directed or avoidance coping mechanisms. While I was able to incorporate depression into the model, proper causal ordering was not maintained as emotions over the past few weeks were used to predict drug use over the past year.

Despite these limitations, this study has built on past work by applying dynamic elements from the life-course perspective to GST in an attempt to understand continuity over time. Unlike past studies of GST, the current research spans from birth to adulthood rather than focusing on adolescence alone.

CONCLUSIONS

In his 1997 discussion of GST and continuity and change in offending, Agnew boldly asserted that "new strain theories...should be part of any developmental theory of crime" (102), yet since then almost no work has attempted to study over the life span using this theory. In the introduction, it was argued that this may be due to a lack of data or a pre-occupation with the core tenets of the theory. While these explanations may ring

true, it is also possible that the failure for new stress theories to have an impact on developmental criminology is because, with its almost exclusive focus on traits, GST provides little above and beyond what is offered by other population heterogeneity explanations of continuity. For example, the direct effect hypothesis closely mirrors Gottfredson and Hirschi's (1990) self control explanation of offending, while evocative and active selection draw heavily from Moffitt's (1993) dual taxonomy of offenders. Even when Agnew strays from trait based explanations of continuity in illegal behavior and posits that stability is the result of stressor and deviance amplification, his explanation maps closely onto those of others, including Sampson and Laub's (1997) cumulative disadvantage and Thornberry's (1987) interaction theory. To borrow from feminist criminologists, it seems like Agnew has "added strain and stirred."

Yet, the study of stressors does have much to contribute to our understanding of criminal behavior over time and this is hinted at, but never fully developed, in Agnew's work. Rather than focusing on individual differences, GST might be better served by drawing from the broader stress literature to understand how stressors themselves develop and are sustained over time. Stress proliferation is one mechanism through which this occurs. In addition, the long term effects of early experiences with stressors provide another avenue through which GST can make a unique contribution.

APPENDIX A: DESCRIPTION OF FIT STATISTICS

Fit Statistic	Description	Acceptable Fit
χ^2	Tests the null hypothesis that the specified model perfectly fits the data. The higher the value, the worse the correspondence to the data. Sensitive to sample size and model complexity. ^A	$\chi^2/df < 3^a$
CFI (Comparative Fit Index)	Assesses the improvement in fit of the specified model over the null model in which all variables are unrelated. ^A	CFI > .90 ^b
TLI (Tucker Lewis Index)	A type of comparative fit index that also takes parsimony into account. ^A	TLI > .95
RMSEA (Root Mean Square Error of Approximation)	Value of 0 indicates the best fit, with increasing values indicating worsening fit. Estimates the amount of error of approximation per model degree of freedom and takes sample size into account. ^A	RMSEA < .06 ^b

^a (Kline 2005)

^b (Hu and Bentler 1999)

APPENDIX B: PRELIMINARY ANALYSES EXPLORING THE MODERATING EFFECTS OF GENDER AND NEIGHBORHOOD DISADVANTAGE

THE MODERATING EFFECT OF GENDER

To determine if the effect of stressors vary by gender I created multiplicative interaction terms between the dichotomous gender variable and stressor exposure in adolescence and adulthood. These terms were then included as predictors of illicit behavior and depression (for adult substance use) in the final trimmed models. Given the large literature on stress and gender, it is not surprising that the effect of stressors on both delinquency ($\beta = .187$) and offending ($\beta = .218$) varied by gender, with a stronger association for males. The effects of stressors on adolescent substance use ($\beta = .180$), depression ($\beta = .329$), and past year substance use ($\beta = .165$) were also stronger for males. These findings are consistent with past research. Agnew and Brezina (1997) and Hay (2003) both found that the relationship between stressors and delinquency was stronger for males. It is also consistent with findings from Piquero and Sealock's (2004) study of high risk youth in which they found that depression was only associated with strain for males.

THE MODERATING EFFECT OF NEIGHBORHOOD DISADVANTAGE

Neighborhood disadvantage was measured using tract-level data from the 1970 Census. It was composed of the following standardized items: percent black; percent female headed households; percent of women over age 16 who are unmarried, have children and live below the poverty line; percent of population between ages 18 and 24; percent of households receiving public assistance; percent of families receiving public

assistance income; male unemployment rate; and adult unemployment rate ($\alpha = .9367$). This scale was then multiplied by adolescent stressors and adult stressors respectively and then included as a predictor of delinquency and offending. There was no significant interaction between stressors and neighborhood disadvantage for delinquency ($\beta = .010$) or offending ($\beta = .024$). Similarly, there was no difference in the effect of adolescent stressors on adolescent substance use ($\beta = -.004$), adult stressors on past year substance use ($\beta = -.006$), or depression on past year substance use ($\beta = -.017$). However, for both delinquency ($\beta = .114$) and adolescent substance use ($\beta = .144$), there was a significant direct effect of neighborhood disadvantage. Therefore, while neighborhood context measured in youth does not moderate the effect of stressors on delinquency and substance use, it does have direct effect. The null findings for adult measures of criminal behavior suggest the effect of disadvantage is contemporaneous.

WORK CITED

- Achenbach, Thomas. 1974. *Developmental Psychopathology*, 1st ed. New York: Wiley.
- Agnew, Robert. 1992. Foundation for a general strain theory of crime and delinquency. *Criminology* 30:47-87.
- Agnew, Robert. 1997. Stability and change in crime over the life course: A strain theory explanation. In *Developmental Theories of Crime and Delinquency*, ed. Terence Thornberry. New Brunswick, NJ: Transaction.
- Agnew, Robert. 2001. Building on the foundation of general strain theory: Specifying the types of strain most likely to lead to crime and delinquency. *Journal of Research in Crime and Delinquency* 38:319-361.
- Agnew, Robert. 2002. Experienced, vicarious, and anticipated strain: An exploratory study on physical victimization and delinquency. *Justice Quarterly* 19(4): 603-632.
- Agnew, Robert. 2003. An integrated theory of the adolescent peak in offending. *Youth & Society* 34:263-299.
- Agnew, Robert. 2005. *Why do Criminals Offend? A General Theory of Crime and Delinquency*. Los Angeles, CA: Roxbury.
- Agnew, Robert. 2006. *Pressured into Crime: An Overview of General Strain Theory*. Los Angeles: Roxbury Publishing Company.
- Agnew, Robert, and Helene Raskin White. 1992. An empirical test of General Strain Theory. *Criminology* 30: 474-499.
- Agnew, Robert, Timothy Brezina, John Paul Wright, and Francis T. Cullen. 2002. Strain, personality traits, and delinquency: Extending general strain theory. *Criminology* 40(1): 43-72.
- Aldwin, C. M., M.R. Levenson, A. Spiro, and R. Bosse. 1989. Does emotionality predict stress? Findings from the Normative Aging Study. *Journal of Personality and Social Psychology* 56:616-624.
- Aseltine, Robert H., Jr. 1996. Pathways linking parental divorce with adolescent depression. *Journal of Health and Social Behavior* 37:133-148.
- Asparouhov, Tihomir, and Bengt Muthen. 2006. A Comparison of Estimation Methods for Complex Survey Data.

- Avison, William R., and R. Jay Turner. 1988. Stressful life events and depressive symptoms: Disaggregating the effects of acute stressors and chronic strains. *Journal of Health and Social Behavior* 29(3): 253-264.
- Baron, Reuben M., and David A. Kenny. 1986. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology* 51:1173-1182.
- Belsky, J. and N. Barends. 2002. Personality and parenting. In *Handbook of Parenting: Volume 3. Being and Becoming a Parent*, ed. M. Bornstein. Mahwah, NJ: Erlbaum.
- Belsky, J., K. Crnic, and S. Woodworth. 1995. Personality and parenting: Exploring the mediating role of transient mood and daily hassles. *Journal of Personality* 63:905-929.
- Ben-Porath, Yossef S., and Auke Tellegen. 1990. A place for traits in stress research. *Psychological Inquiry* 1(1): 14-17.
- Block, J., Block, J. H., and Keyes, S. 1988. Longitudinally foretelling drug usage in adolescence: Early childhood personality and environmental precursors. *Child Development* 59:336-355.
- Bolger, Niall, and Elizabeth A. Schilling. 1991. Personality and the problems of everyday life: The role of neuroticism in exposure and reactivity to daily stressors. *Journal of Personality* 59(3): 355-386.
- Broidy, Lisa. M. 2001. A test of general strain theory. *Criminology* 39:9-33.
- Broidy, Lisa. M., and Robert Agnew. 1997. Gender and crime: A general strain theory perspective. *Journal of Research in Crime and Delinquency* 34:275-306.
- Bronfenbrenner, Urie. 1975. Reality and research in the ecology of human development. *Proceedings of the American Philosophical Society* 119(6): 439-469.
- Capowich, George E., Paul Mazerolle, and Alex Piquero. 2001. General strain theory, situational anger, and social networks: An assessment of conditioning influences. *Journal of Criminal Justice* 29:445-461.
- Caspi, Avshalom, and Daryl Bem. 1990. Personality continuity and change across the life course. In *Handbook of Personality: Theory and Research*, ed. Lawrence Pervin. New York: Guilford Press.

- Caspi, Avshalom, and Brent W. Roberts. 2001. Personality development over the life course: The argument for continuity and change. *Psychological Inquiry* 12(2): 49-66.
- Caspi, Avshalom, and Phil A. Silva. 1995. Temperamental qualities at age three predict personality traits in young adulthood: Longitudinal evidence from a birth cohort. *Child Development* 66:486-498.
- Caspi, Avshalom, Glen H. Elder, Jr., and Daryl Bem. 1987. Moving against the world: Life-course patterns of explosive children. *Developmental Psychology* 23(2): 308-313.
- Caspi, Avshalom, Bill Henry, Rob O. McGee, Terrie E. Moffitt, and Phil A. Silva. 1995. Temperamental origins of child and adolescent behavior problems: From age three to age fifteen. *Child Development* 66:55-68.
- Caspi, Avshalom, Terrie E. Moffitt, Phil A. Silva, Magda Stouthamer-Loeber, Robert F. Krueger, and Pamela Schmutte. 1994. Are some people crime-prone? Replications of the personality-crime relationship across countries, genders, races, and methods. *Criminology* 32(2): 163-195.
- Champion, Lorna, G. M. Goodall, and Michael Rutter. 1995. Behavioural problems in children and stressors in early adult life: A 20-year follow-up of London school children. *Psychological Medicine* 25:231-246.
- Cloward, Richard, and Lloyd Ohlin. 1960. *Delinquency and Opportunity*. Glencoe, IL: Free Press.
- Coie, John D., and Kenneth A. Dodge. 1998. Aggression and antisocial behavior. In *Handbook of Child Psychology: Social, Emotional, and Personality Development*, ed. Nancy Eisenberg. New York: Wiley.
- Costa, P. T., Jr., R. R. McCrae, and D. Arenberg. 1983. Recent longitudinal research on personality and aging. In *Longitudinal Studies of Adult Psychological Development*, ed. K. W. Schaie. New York: Guilford Press.
- De Coster, Stacy, and Lisa Kort-Butler. 2006. How general is general strain theory? Assessing determinacy and indeterminacy across life domains. *Journal of Research in Crime and Delinquency* 43(4): 297-325.
- De Coster, Stacy, and Karen Heimer. 2001. The relationship between law violation and depression: An interactionist analysis. *Criminology* 39(4): 799-836.
- Dempster-McClain, Donna, and Phyllis Moen. 1998. Finding respondents in a follow-up study. In *Methods of Life Course Research: Qualitative and Quantitative Approaches*, eds. Janet Z. Giele and Glen H. Elder Jr. Thousand Oaks, CA: Sage.

- Dohrenwend, Bruce P. 1979. Stressful life events and psychopathology: Some issues of theory and method. In *Stress and Mental Disorder*, ed. J. E. Barrett. New York: Raven Press.
- Drapela, Laurie A. 2006. The effect of negative emotion on drug use among high school dropouts: An empirical test of general strain theory. *Journal of Youth and Adolescence* 35(5): 752-767.
- Eitle, David R., and R. Jay Turner. 2003. Stress exposure, race, and young adult male crime. *Sociological Quarterly* 2:243-269.
- Elder, Glen H., Jr. 1985. Perspectives on the life course. In *Life Course Dynamics*, ed. Glen H. Elder, Jr. Ithaca, NY: Cornell University Press.
- Elder, Glen H., Jr. 1994. Time, human agency, and social change: Perspectives on the life course. *Social Psychology Quarterly* 57(1): 4-15.
- Elder, Glen H., Jr. 1998. The life course as developmental theory. *Child Development* 69:1-12.
- Elder, Glen H., Jr., Avshalom Caspi, and Geraldine Downey. 1986. Problem behavior and family relationships: Life course and intergenerational themes. In *Human Development and the Life Course*, eds. Aage B. Sorensen, Franz E. Weinert, and Lonnie R. Sherrod. Hillsdale, NJ: Erlbaum.
- Elder, Glen H., Jr., Linda K. George, and Michael J. Shanahan. 1996. Psychosocial stress over the life course. In *Psychosocial Stress: Perspectives on Structure, Theory, Life-Course, and Methods*, ed. Howard B. Kaplan. San Diego, CA: Academic Press.
- Ellenbogen, Mark A., and Sheilagh Hodgins. 2004. The impact of high neuroticism in parents on children's psychosocial functioning in a population at high risk for major affective disorder: A family-environmental pathway of intergenerational risk. *Development and Psychopathology* 16:113-136.
- Ensel, Walter M., and Nan Lin. 2000. Age, the stress process, and physical distress: The role of distal stressors. *Journal of Aging and Health* 12(2): 139-168.
- Erikson, Erik H. 1950. *Childhood and Society*. New York: Norton.
- Evans, G. W. 2001. Environmental stress and health. In *Handbook of Health Psychology*, eds Greg J. Duncan and J. E. Singer. Mahwah, NJ: Erlbaum.
- Eysenck, Hans J., and Sybil B. G. Eysneck. 1969. *Personality Structure and Measurement*. London: Routledge.

- Fishbein, Diana. 2001. *Biobehavioral Perspectives in Criminology*. Belmont, CA: Wadsworth.
- Folkman, S., and R.S. Lazarus. 1980. An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behavior* 21: 219-239.
- Folkman, S., R.S. Lazarus, C. Dunkel-Schetter, A. DeLongis, and R.J. Gruen. 1986. Dynamics of a stressful encounter: Cognitive appraisal, coping, and encounter outcomes. *Journal of Personality and Social Psychology* 50:992-1003.
- Goldberg, D.P., and V.F. Hillier. 1979. A scaled version of the General Health Questionnaire. *Psychological Medicine* 9:139-145.
- Gottfredson, Michael, and Travis Hirschi. 1990. *A General Theory of Crime*. Stanford, CA: Stanford University Press.
- Hagan, John, and Holly Foster. 2003. S/He's a rebel: Toward a sequential stress theory of delinquency and gendered pathways to disadvantage in emerging adulthood. *Social Forces* 82(1): 53-86.
- Hardy, Janet B. 2003. The Collaborative Perinatal Project: Lessons and legacy. *European Psychiatry* 13(5): 303-311.
- Hardy, Janet B., Nan M. Astone, Jeanne Brooks-Gunn, Sam Shapiro, and Therese L. Miller. 1998. Like mother, like child: Intergenerational patterns of age at first birth and associations with childhood and adolescent characteristics and adult outcomes in the second generation. *Developmental Psychology* 34(6): 1220-232.
- Hay, Carter. 2001. Parenting, self-control, and delinquency: A test of self-control theory. *Criminology* 39: 707-736.
- Headey, Bruce, and Alexander Waring. 1989. Personality, life events, and subjective well-being: Toward a dynamic equilibrium model. *Journal of Personality and Social Psychology* 57(4): 731-739.
- Hoffmann, John P., and Felice Gray Cerbone. 1999. Stressful life events and delinquency escalation in early adolescence. *Criminology* 37:343-373.
- Hoffmann, John P., and Alan S. Miller. 1998. A latent variable analysis of general strain theory. *Journal of Quantitative Criminology* 14(1): 83-110.
- Jang, Sung Joon, and Byron R. Johnson. 2003. Strain, negative emotions, and deviant coping among African Americans: A test of general strain theory. *Journal of Quantitative Criminology* 19:79-105.

- John, Oliver P., Avshalom Caspi, Richard W. Robins, Terrie Moffitt, and Magda Stouthamer-Loeber. 1994. The "little five": Exploring a nomological network of the five-factor model of personality in adolescent boys. *Child Development* 65(1): 160-178.
- Jöreskog, Karl G. and Dag Sörbom. *Advances in Factor Analysis and Structural Equation Models*. Cambridge, MA: Abt Books.
- Kardum, Igor, and Nada Krapić. 2001. Personality traits, stressful life events, and coping styles in early adolescence. *Personality and Individual Differences* 30:503-515.
- Kendler, Kenneth S., C. O. Gardner, and C. A. Prescott. 2003. Personality and the experience of environmental adversity. *Psychological Medicine* 33: 1193-1202.
- Kim, Kee Jong, Rand D. Conger, Glen H. Elder, Jr., and Frederick Lorenz. 2003. Reciprocal influences between stressful life events and adolescent internalizing and externalizing problems. *Child Development* 74(1): 127-143.
- Kline, Rex B. 2005. *Principles and Practices of Structural Equation Modeling*. New York: The Guilford Press.
- Laub, John H. and Robert J. Sampson. 2003. *Shared Beginnings, Divergent Lives: Delinquent Boys to Age 70*. Cambridge, MA: Harvard University Press.
- Laub, John H., and George E. Vaillant. 2000. Delinquency and mortality: A 50-year follow-up study of 1,000 delinquent and non-delinquent boys. *American Journal of Psychiatry* 157:96-102.
- Lazarus, R. S. 1986. Puzzles in the study of daily hassles. In *Development as Action in Context: Problem Behavior and Normal Youth Development*, eds. R. K. Silbereisen, K. Eyferth, and G. Rudinger. Berlin: Springer-Verlag.
- LeBlanc, Marc. 2006. Self-control and social control of deviant behavior in context: Development and interactions along the life course. In *The Explanation of Crime: Context, Mechanisms and Development*, eds. Per-Olaf Wikström and Robert J. Sampson. New York: Cambridge University Press.
- Loeber, Rolf. 1982. The stability of antisocial and delinquent child behavior: A review. *Child Development* 53: 1431-1446.
- Loeber, Rolf, and D. Hay. 1997. Key issues in the development of aggression and violence from childhood to early adulthood. *Annual Review in Psychology* 45:371-410.

- MacCallum, Robert C., and James T. Austin. 2000. Applications of structural equation modeling in psychological research. *Annual Review of Psychology* 51:210-226.
- Magnus, Keith, Ed Diener, Frank Fujita, and William Pavot. 1993. Extraversion and neuroticism as predictors of objective life events: A longitudinal analysis. *Journal of Personality and Social Psychology* 65(5): 1046-1053.
- Mazerolle, Paul, and Jeff Maahs. 2000. General strain and delinquency: An alternative examination of conditioning influences. *Justice Quarterly* 17:753-78.
- Mazerolle, Paul, and Alex Piquero. 1998. Linking exposure to strain with anger: An investigation of deviant adaptations. *Journal of Criminal Justice* 26:195-211.
- Mazerolle, Paul, Alex Piquero, and George E. Capowich. 2003. Examining the links between strain, situational and dispositional anger, and crime. *Youth and Society* 35:131-157.
- McGue, Matthew, Steven Bacon, and David Lykken. 1993. Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental Psychology* 29:96-109.
- McLean, Diane E., and Bruce G. Link. 1994. Unraveling complexity: Strategies to refine concepts, measures, and research designs in the study of life events and mental health. In *Stress and Mental Health: Contemporary Issues and Prospects for the Future*, eds. William R. Avison and Ian H. Gotlib. New York: Plenum Press.
- Menaghan, Elizabeth G. 1997. Intergenerational consequences of social stressors: Effects of occupational and family conditions on young mothers and their children. In *Stress and Adversity over the Life Course: Trajectories and Turning Points*, eds. Ian H. Gotlib and Blair Wheaton. Cambridge: Cambridge University Press.
- Miller, Joshua D., and Donald Lynam. 2001. Structural models of personality and their relation to antisocial behavior: A meta-analytic review. *Criminology* 39(4): 765-795.
- Moen, Phyllis. 1995. Introduction. In *Examining Lives in Context: Perspectives on the Ecology of Human Development*, eds. Phyllis Moen, Glen H. Elder, Jr., and Kurt Lüscher. Washington, DC: The American Psychological Association.
- Moen, Phyllis, and Mary Ann Erickson. 1995. Linked lives: A transgenerational approach to resilience. In *Examining Lives in Context: Perspectives on the Ecology of Human Development*, eds. Phyllis Moen, Glen H. Elder, Jr., and Kurt Lüscher. Washington, DC: The American Psychological Association.

- Moffitt, Terrie. 1993. Adolescent-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review* 100:674-701.
- Moffitt, Terrie. 1997. Neuropsychology, antisocial behavior, and neighborhood context. In *Violence and Childhood in the Inner City*, ed. Joan McCord. Cambridge: Cambridge University Press.
- Monroe, Scott M., and John R. McQuaid. 1994. Measuring life stress and assessing its impact on mental health. In *Stress and Mental Health: Contemporary Issues and Prospects for the Future*, eds. William R. Avison and Ian H. Gotlib. New York: Plenum Press.
- Muthén, Bengt O. 2000. MPlus Discussion: Structural Equation Models, Degrees of Freedom. MPlus Message Board, January 11, <http://www.statmodel.com/discussion/messages/11/21.html?1153932224>.
- Muthén, Linda K., and Bengt O. Muthén. 2006. Observed and Latent Categorical Variable Modeling Using Mplus. Mplus Short Courses Day 3.
- Muthén, Linda K., and Bengt O. Muthén. 2007. *Mplus User's Guide*. Fourth Edition. Los Angeles, California: Muthén & Muthén.
- Ormel, Johan, and Tamar Wohlfarth. 1991. How neuroticism, long-term difficulties, and life situation change influence psychological distress: A longitudinal model. *Journal of Personality and Social Psychology* 60(5): 744-755.
- Ormel, Johan, Albertine J. Oldehinkel, and Els I. Brillman. 2001. The interplay and etiological continuity of neuroticism, difficulties, and life events in the etiology of major and subsyndromal, first and recurrent depressive episodes in later life. *American Journal of Psychiatry* 158:885-891.
- Osgood, D. Wayne, Laura L. Finken, and Barbara J. McMorris. 2002. Analyzing multiple-item measures of crime and deviance II: Tobit regression analysis of transformed scores. *Journal of Quantitative Criminology* 16:117-144.
- Osgood, D. Wayne, Barbara J. McMorris, and Maria T. Potenza. 2002. Analyzing multiple-item measures of crime and deviance I: Item response theory scaling. *Journal of Quantitative Criminology* 18:267-296.
- Ostrowsky, Michael K. and Steven F. Messner. Explaining crime for a young adult population: An application of general strain theory. *Journal of Criminal Justice* 33:463-476.
- Ousey, Graham C., and Pamela Wilcox. 2007. The interaction of antisocial propensity and life-course varying predictors of delinquent behavior: Differences by methods of estimation and implications for theory. *Criminology* 45(2): 313-354.

- Paternoster, Raymond, and Paul Mazerolle. 1994. General Strain Theory and delinquency: A replication and extension. *Journal of Research in Crime and Delinquency* 31:235-264.
- Pearlin, Leonard I., and Marilyn McKean Skaff. 1996. Stress and the life course: A paradigmatic alliance. *The Gerontologist* 36(2): 239-247.
- Pearlin, Leonard I., Carol S. Aneshensel, and Allen J. LeBlanc. 1997. The forms and mechanisms of stress proliferation: The Case of AIDS caregivers. *Journal of Health and Social Behavior* 38:223-236.
- Pearlin, Leonard I., Elizabeth G. Menaghan, Morton A. Lieberman, and Joseph T. Mullan. 1981. The stress process. *Journal of Health and Social Behavior* 22(4): 337-356.
- Pearlin, Leonard I., Scott Schieman, Elena M. Fazio, and Stephen C. Meersman. 2005. Stress, health, and the life course: Some conceptual perspectives. *Journal of Health and Social Behavior* 46(June): 205-219.
- Piquero, Alex R. Leah E. Daigle, Chris Gibson, Nicole Leeper Piquero, and Stephen G. Tibbetts. 2007. Research note: Are life-course-persistent offenders at risk for adverse health outcomes? *Journal of Research in Crime and Delinquency* 44(2): 185-207.
- Piquero, Nicole L., and Miriam D. Sealock. 2000. Generalizing General Strain Theory: An examination of an offending population. *Justice Quarterly* 17:449-484.
- Ricketts, Erol R., and Isabel V. Sawhill. 1988. Defining and measuring the underclass. *Journal of Policy Analysis and Management* 7(2): 316-325.
- Roberts, Brent W., and Wendy F. Del Vecchio. 2000. The rank order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin* 126(1): 3-25.
- Robins, Helzer, Croughan and Ratcliff 1981 (methods section description of delinquency variable)
- Rutter, Michael. 1987. Temperament, personality, and personality disorder. *British Journal of Psychiatry* 150: 443-458.
- Rutter, Michael, and Judy Silberg. 2002. Gene-environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology* 53:463-490.
- Rutter, Michael, Lorna Champion, David Quinton, Barbara Maughan, and Andrew Pickles. 1995. Understanding individual differences in environmental-risk

- exposure. In *Examining Lives in Context: Perspectives on the Ecology of Human Development*, eds. Phyllis Moen, Glen H. Elder, Jr., and Kurt Lüscher. Washington, DC: The American Psychological Association.
- Rutter, Michael, Judy Dunn, Robert Plomin, Emily Simonoff, Andrew Pickels, Barbara Maughan, Johan Ormel, Joanne Meyer, and Linda Eaves. 1997. Integrating nature and nurture: Implications of person-environment correlations and interactions for developmental psychology. *Development and Psychopathology* 9:335-364.
- Sampson, Robert J., and John H. Laub. 1993. *Crime in the Making: Pathways and Turning Points through Life*. Cambridge, MA: Harvard University Press.
- Sampson, Robert J., and John H. Laub. 1997. A life-course theory of cumulative disadvantage and the stability of delinquency. In *Developmental Theories of Crime and Delinquency*, ed. Terence Thornberry. New Brunswick, NJ: Transaction.
- Satorra, Albert, and Peter M. Bentler. 1999. A scaled difference chi-square test statistics for moment structure analysis. Working Paper Number 260. UCLA Statistics Series.
- Scarr, Sandra, and Kathleen McCartney. 1983. How people make their own environments: A theory of genotype→environment effects. *Child Development* 54: 425-435.
- Scott, Jaqueline, and Duane Alwin. 1998. Retrospective versus prospective measurement of life histories in longitudinal research. In *Methods of Life Course Research: Qualitative and Quantitative Approaches*, eds. Janet Z. Giele and Glen H. Elder, Jr. Thousand Oaks, CA: Sage.
- Slocum, Lee Ann, Sally S. Simpson, and Douglas A. Smith. 2005. Strained lives and crime: Examining intra-individual variation in strain and offending in a sample of incarcerated women. *Criminology* 43(4): 1067-1110.
- Taylor, Shelley E., and Lisa G. Aspinwall. 1996. Mediating and moderating processes in psychosocial stress: Appraisal, coping, resistance, and vulnerability. In *Psychosocial Stress: Perspectives on Structure, Theory, Life-Course, and Methods*, ed. Howard B. Kaplan. San Diego, CA: Academic Press.
- Tellegen, Auke. 1985. Structures of mood and personality and their relevance to assessing anxiety with an emphasis on self-report. In *Anxiety and Anxiety Disorders*, eds A. Hussain Tuma and Jack D. Maser. Hillside, NJ: Lawrence Erlbaum Associates.

- Thoits, Peggy A. 1982. Conceptual, methodological, and theoretical problems in studying social support as a buffer against life stress. *Journal of Health and Social Behavior* 23:145-159.
- Thoits, Peggy A. 1983. Dimensions of life events that influence psychological distress: An evaluation and synthesis of the literature. In *Psychosocial Stress: Trends in Theory and Research*, ed. Howard B. Kaplan. New York: Academic Press.
- Thornberry, Terence. 1987. Toward an interactional theory of delinquency. *Criminology* 25:863-891.
- Turner, R. Jay, and Donald A. Lloyd. 1995. Lifetime traumas and mental health: The significance of cumulative adversity. *Journal of Health and Social Behavior* 36(4): 360-376.
- Turner, R. Jay, and Blair Wheaton. 1995. Checklist measurements of stressful life events. In *Measuring Stress: A Guide for Health and Social Scientists*, eds. Sheldon Cohen, Ronald C. Kessler, and Lynn Gordon. Oxford: Oxford University Press.
- Umberson, Debra, Kristi Williams, Daniel A. Powers, Hui Liu, and Belinda Needham. 2005. Stress in childhood and adulthood: Effects on marital quality over time. *Journal of Marriage and Family* 67:1332-1347.
- Van Os, Jim, S.B.G. Park, and Peter B. Jones. 1999. Early risk factors and adult person-environment relationships in affective disorder. *Psychological Medicine* 29:1055-1067.
- Van Os, Jim, S.B.G. Park, and Peter B. Jones. 2001. Neuroticism, life events and mental health: Evidence for person-environment correlation. *British Journal of Psychiatry* 178 (suppl. 40): s72-s77.
- Wheaton, Blair. 1990. Life transitions, role histories, and mental health. *American Sociological Review* 55:209-224.
- Wheaton, Blair. 1996. The domains and boundaries of stress concepts. In *Psychosocial Stress: Perspectives on Structure, Theory, Life-Course, and Methods*, ed. Howard B. Kaplan. San Diego, CA: Academic Press.
- Wheaton, Blair, and Ian H. Gotlib. 1997. Trajectories and turning points over the life course: Concepts and themes. In *Stress and Adversity over the Life Course: Trajectories and Turning Points*, eds. Ian H. Gotlib and Blair Wheaton. Cambridge: Cambridge University Press.
- Wilson, James Q., and Richard J. Herrnstein. 1985. *Crime and Human Nature*. New York: Simon and Schuster.

Wolfgang, Marvin E., Robert M. Figlio, Paul E. Tracy, and Simon I. Singer. 1985. *The National Survey of Crime Severity*. Washington, DC: U.S. Department of Justice, Bureau of Justice Statistics.

Wright, Bradley R. E., Avshalom Caspi, Terrie Moffitt, and Phil A. Silva. 2001. The effects of social ties vary by criminal propensity: A life-course model of interdependence. *Criminology* 39(2): 321-348.