PUBERTAL DEVELOPMENT AND SUBSTANCE USE AMONG ADOLESCENT GIRLS: THE IMPORTANCE OF SOCIAL INTERACTIONS AND SOCIAL CONTEXTS

By

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This dissertation is dedicated to all of the young girls and women struggling with issues related to substance use, as well as to the researchers and practitioners committed to improving their lives.

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CHAPTER I

PUBERTAL DEVELOPMENT AND SUBSTANCE USE: AN OVERVIEW

INTRODUCTION

The societal cost of substance abuse is well over $100 billion and steadily increasing (Office of National Drug Control Policy 2001).[1] The costs associated with substance abuse are largely due to lost productivity, criminal justice and social welfare costs, and health costs associated with morbidity and mortality. For instance, alcohol abuse is related to almost two million deaths worldwide (3% of total), and associated with almost $185 billion in costs related to treatment, prevention, law enforcement, productivity lost, and life years lost in the United States (World Health Organization 2004). Tobacco is also extremely costly; between 2000 and 2004 tobacco use and exposure to tobacco smoke were related to at least 443,000 premature deaths and $96.8 billion in lost productivity in the United States (Centers for Disease Control and Prevention 2008).

Thus it should come as little surprise that substance use among adolescents is a major public health concern in the United States. Substance use during adolescence is correlated with death by injury, suicide, physical/sexual assault, high-risk sexual behavior, academic problems, neuro-cognitive impairment, and also has adverse effects on the bones, liver, and reproductive functions of adolescents. Researchers and practitioners are particularly interested in substance use during adolescence given that age of onset of substance use is

[1] Substance use refers to any consumption of licit or illicit substances, whereas substance abuse is defined as use that leads to clinically significant impairment or distress manifested in a failure to fulfill major role obligations, recurrent use in hazardous situations, recurrent substance-related legal problems, or continued use despite recurrent social or interpersonal problems related to the substance (DSM IV; American Psychiatric Association 1994).

highly correlated with substance use career length (Dennis et al. 2005), and is thus linked to adult psychiatric and substance use disorders, criminal behavior, educational attainment, physical health, and life satisfaction (e.g., DuRant et al. 1999; Georgiades and Boyle 2007; Lynskey and Hall 2000; Mathers et al. 2006; McGee et al. 2000).

Given high relapse rates among adolescents after substance abuse treatment (Brown, Vik, and Creamer 1989; Catalano et al. 1991; Kennedy and Minami 1993), increased attention has been devoted toward interventions aimed at preventing the onset of adolescent substance use and hence likely progression to more problematic levels of abuse or dependence. The field of adolescent substance use prevention is largely guided by an examination of risk factors, or factors that increase the likelihood of substance use. Historically, however, research on adolescent substance use risk has neglected gender-specific risk factors, particularly for adolescent girls. This omission is probably because adolescent boys generally exhibit higher prevalence rates of substance use than adolescent girls.

But the social epidemiological literature indicates that the gender gap in adolescent substance use has been narrowing for specific substances. For instance, the latest estimates from the 2006 National Survey on Drug Use and Health indicate that 6.8% of boys and 6.4% of girls age 12 to 17 report using marijuana in the past month (Substance Abuse and Mental Health 2006: 25). Further, 8.1% of girls age 12 to 17 report past year substance abuse or dependence, compared to 8.0% of boys (p. 73). Results from the Monitoring the Future Study also suggest that adolescent girls in 8^{th}, 10^{th}, and 12^{th} grade generally have prevalence rates of alcohol and cigarette use equivalent to, or slightly higher than those of boys (Johnston et al. 2008). Given this narrowing gender gap in adolescent substance use, the argument that

research should not focus on substance use risk factors for adolescent girls because they do not use substances is now an outdated argument. Because the pathways and processes related to adolescent substance use often vary by gender (Amaro et al. 2001), the prevalence estimates noted here point to the importance of understanding gender-specific risk factors that could be addressed in targeted substance use intervention programs for adolescent girls. One risk factor for substance use that is particularly salient for adolescent girls is pubertal development, or the bodily changes that occur during the developmental stage of puberty. The process of puberty, although a physiological process of reproductive maturation, is heavily imbued with cultural, psychological, and social significance for adolescent girls.

To address the need for research on gender-specific risk factors for substance use among adolescent girls, this dissertation examines and "unpacks" the substance use risk associated with pubertal development among adolescent girls, focusing on the reasons why and how this relationship occurs. Specifically, this dissertation addresses the following broad questions: Does the relationship between pubertal development and substance use vary for girls with different body weights? Do adolescent girls' self-appraisals and social relationships explain why pubertal development is positively related to substance use? And finally, how do neighborhood contexts and race/ethnicity influence the relationship between pubertal development and adolescent girls' substance use?

With that as background, the following sections theorize and discuss: (a) puberty as a developmental transition, (b) the link between pubertal development and adolescent girls' substance use, and (c) the importance of body weight, self-appraisals, social relationships, neighborhood contexts, and race/ethnicity in the pubertal development-substance use

relationship. The chapter will end with an overview of the research questions and implications of the three empirical studies in the dissertation.

PUBERTY AS A DEVELOMPENTAL TRANSITION

Adolescence has been aptly described as a phase of the life course that begins in biology and ends in society (Conger and Petersen 1984:92). Academic study of adolescence and puberty has shifted attention from biological to socio-cultural factors over the last century. Hall's (1904) classic treatise originally defined adolescence as a unique developmental period characterized by 'storm and stress' (e.g., conflict with parents, mood disruptions, risky behaviors) resulting from rising levels of hormones during puberty. Some psychoanalytic theorists (e.g., Freud 1958) even argued that a lack of adolescent storm and stress signified psychopathology. Cultural theorists (e.g., Mead 1950), however, maintained that adolescent storm and stress is a phenomenon attributable to the discontinuous roles and responsibilities between childhood and adulthood in modern societies. Consequently, contemporary researchers acknowledge the importance of cultural context for adolescent storm and stress, and concede that most adolescents successfully cope with emerging emotions and conflicts during puberty. So, storm and stress does not occur for all adolescents, but for those whom it does occur, it results from a combination of rising levels of hormones, brain development, and changing social contexts during puberty (Arnett 1999; Dahl and Hariri 2005). Researchers studying adolescents have thus shifted attention to person-context interactions, emphasizing that puberty's impact on psychosocial development depends on the individual, the social context, and the interaction between the two (Schulenberg and Maggs 2001).

Puberty is therefore best conceptualized as a developmental transition, or path connecting individuals to transformed selves, where "the occurrence and meaning of developmental transitions originate in the interaction of physical maturational processes, cultural influences and expectations, and personal values and goals" (Schulenberg and Maggs 2001:11). Developmental transitions are embedded in socio-cultural contexts that provide normative social timetables and age-related expectations for role transitions. The developmental transition of puberty uniquely challenges youth as they adapt to their emerging physical appearance and associated shifts in peer and family relations. The influence of pubertal development on outcomes such as substance use must therefore be understood as embedded within and related to the person-context interactions that occur during this developmental transition.

PUBERTAL DEVELOPMENT AND ADOLESCENT GIRLS' SUBSTANCE USE

Although pubertal development is a physiological process, it is consequential for adolescent girls' behaviors such as substance use given the larger socio-cultural context in which this developmental transition occurs. Stage termination theory (Stattin and Magnusson 1990) posits that adolescent girls who experience early pubertal development are (perceived to be) developmentally and socially deviant. Early developers may experiment with substances because they are not psychologically or socially prepared to cope with this anormative developmental stage (Caspi and Moffitt 1991; Petersen and Taylor 1980; Stattin and Magnusson 1990). The maturity gap or autonomy hypothesis (Moffitt 1993) similarly suggests that puberty leads to biological maturity before social maturity in western societies and is particularly pronounced for early developers. Adolescents might use substances in an

attempt to bridge this maturity gap and establish symbolic autonomy from parents and other authority figures. Thus both stage termination and maturity gap theories indicate that early pubertal development might be positively associated with substance use among adolescent girls due to the gap between their biological and social maturity.

Stage termination and maturity gap theories suggest that the impact of pubertal development on substance use is attributable to socio-cultural rather than biological contexts. Yet these developmental theories fail to explicate for whom the link between early pubertal development and substance use applies, or the specific social psychological or relational processes that may explain why this relationship occurs. Three categories of social psychological and relational factors that might play a role in the link between pubertal development and adolescent girls' substance use are: (1) body weight; (2) self-appraisals and social relationships; and (3) neighborhood contexts and race/ethnicity, which are discussed below in turn.

The Moderating Role of Body Weight

Stage termination and maturity gap theories suggest that the substance use risk associated with pubertal development may be largely due to shifts in adolescent girls' perceptions of their body weight—problematic because these changes in body weight distance girls from same-age peers and necessitate shifts in identities in relation to peers and society. In line with social psychological theories of identity control (Burke 1991; Burke and Cast 1997), girls with different body weights may therefore experience and internalize the meanings associated with the pubertal body in very different ways. Pubertal development may have less importance as a substance use risk factor for overweight girls, who may not

experience extremely visible changes in body size and shape during puberty. Conversely, underweight or average weight girls may be at greater risk of using substances if they develop early, because bodily changes that occur during puberty will be more visibly noticeable and thus force an early negotiation of the meanings associated with the pubertal body. So the relationship between pubertal development and substance use may vary for girls with different body weights due to differences in the negotiation and attempted stabilization of identities as they relate to pubertal changes in the body. To date, however, no empirical research has examined the potential moderating role of body weight on the association between pubertal development and substance use. The first empirical study in my dissertation addresses this gap in the literature by modeling differences in the link between pubertal development and adolescent girls' substance use for girls who are under-, average, and over-weight.

The Mediating Role of Self-appraisals and Social Relationships

Stage termination theory highlights the importance of socio-cultural contexts in the association between pubertal development and substance use, but does not explicate the precise social psychological or relational mechanisms that produce this relationship. Theories of social interaction and social development in adolescence (Catalano and Hawkins 1996; Frost 2005; Hawkins and Weis 1985; Lee 1994) point to two categories of mechanisms that might explain why pubertal development is related to substance use: intrapersonal self-appraisals and interpersonal social relationships. The reason why pubertal development correlates with adolescent girls' substance use may be due to intrapersonal self-appraisals, or girls' reflections of how others see them. In light of cultural beauty ideals that value thin pre-

pubescent body types, girls who experience puberty earlier than their peers may be dissatisfied with their body weight and have low self-esteem. Yet the link between pubertal development and substance use might alternatively be explained by adolescent girls' social relationships with agents of socialization, specifically peers and parents. Early developers may be at greater risk of substance use due to autonomy from parents and association with deviant peers.

Prior research suggests that weight dissatisfaction and self-esteem are related to both pubertal development (Attie and Brooks-Gunn 1989; Blyth, Simmons, and Zakin 1985; Ge et al. 2001; Harter 1993; Richards et al. 1990; Simmons and Blyth 1987) and substance use (Boles and Johnson 2001; Crow et al. 2006; French et al. 1994; Granillo, Jones-Rodriguez, and Carvajal 2005; Nieri et al. 2005; Palmqvist and Santavirta 2006), but to date no studies have examined the potential mediating role of these intrapersonal self-appraisals. Research also shows that autonomy from parents and deviant peer association are linked to pubertal development (Duncan et al. 1998; Steinberg 1987) as well as substance use (Dishion, Nelson, and Bullock 2004; Duncan et al. 1998; Schulenberg et al. 1999; Wills and Cleary 1999). Although previous studies have established that these two social relationships may indeed mediate the relationship between pubertal development and substance use (Patton et al. 2004; Wichstrom 2001), research to date has only been conducted on mixed-gender or boy-only samples. To address these gaps in the literature, the second empirical study examines whether intrapersonal self-appraisals and interpersonal social relationships fully explain or mediate the relationship between pubertal development and adolescent girls' substance use.

The Importance of Neighborhood Contexts and Race/Ethnicity

The association between pubertal development and substance use does not happen within a cultural or social vacuum, but rather is embedded within the larger ecological context of adolescent girls' lived experiences. Adolescents' limited geographic mobility means that neighborhoods are particularly important contexts for framing their experiences and behaviors. The relationship between pubertal development and substance use may be conditional upon neighborhood dislocations like concentrated disadvantage, residential instability, and social disorder. These neighborhood dislocations, or characteristics referring to the potential deleterious structural and economic organization of a neighborhood, influence community norms related to substance use, opportunities to use substances, and networks of adults willing to enforce social control (Crum, Lillie-Blanton, and Anthony 1996; Jencks and Mayer 1990; Kadushin et al. 1998; Sampson, Raudenbush, and Earls 1997). Persistent patterns of residential racial segregation (Massey and Denton 1993) may mean that neighborhood dislocations play a larger role in the effect of pubertal development on the substance use of young adolescent girls of color (Crane 1991; Hogan and Kitagawa 1985; Kulis et al. 2007).

With few exceptions (e.g., Abdelrahman et al. 1998; Allison et al. 1999; Chuang et al. 2005; Ennett et al. 1997), the empirical literature documents a positive relationship between neighborhood dislocations and substance use among adolescents and adults (Bernstein et al. 2007; Beyers et al. 2004; Boardman et al. 2001; Crum et al. 1996; Hill and Angel 2005; Kulis et al. 2007; Lambert et al. 2004; Wardle et al. 2003). Further, neighborhood dislocations have been found to moderate the relationship between pubertal development and adolescents' behavioral outcomes such as externalizing and internalizing symptoms, and

violent behavior (Ge et al. 2002; Obeidallah et al. 2004). One study found that neighborhood contexts did not influence the link between pubertal development and substance use, but used a regional sample with potentially flawed psychometric measurement properties (Foshee et al. 2007). Further, no studies to date have examined the complex interactions between pubertal development, neighborhood dislocations, race/ethnicity, and adolescent girls' substance use. The third study in this dissertation addresses these gaps in the literature and employs an ecologically sensitive approach to examine how neighborhood contexts and race/ethnicity simultaneously and interactively influence the relationship between pubertal development and adolescent girls' substance use.

RESEARCH QUESTIONS

This dissertation presents three empirical studies examining how and why pubertal development is associated with adolescent girls' substance use. All three studies use data from the first three waves of the National Longitudinal Study of Adolescent Health and explore potential moderators and mediators of the relationship between pubertal development and adolescent girls' substance use. Motivated by the aforementioned weaknesses in the literatures, the studies address the following five research questions:

1. Does the association between pubertal development and adolescent girls' substance use persist beyond early adolescence?

2. Does body weight moderate the relationship between pubertal development and adolescent girls' substance use?

3. Do intrapersonal self-appraisals and interpersonal social relationships explain or mediate the relationship between pubertal development and adolescent girls' substance use?

4. Do neighborhood dislocations moderate the relationship between pubertal development and adolescent girls' substance use?

5. Does race/ethnicity moderate the relationships between pubertal development, neighborhood dislocations, and adolescent girls' substance use?

STUDY SIGNIFICANCE

This dissertation contributes to the fields of medical sociology and prevention science by highlighting the importance of social psychological and contextual factors for the health of adolescents and emerging adults. First, by examining social psychological and relational processes that mediate and moderate the relationship between physical processes (pubertal development) and health behaviors (substance use), this dissertation advances theory involving the interpretation and management of changing bodies and identities during key developmental transitions in the life course. Second, by drawing attention to the contextual specificity of substance use risk factors for adolescents girls from different racial/ethnic backgrounds, this dissertation illustrates how gender, race, and ethnicity as social locations within contemporary status hierarchies, influence the interpretation and management of identities during developmental transitions. Such knowledge can be used to advance theoretical understandings of similar influences during other normative social timetables in the life course (e.g., age at first intercourse, first childbirth). Third, this dissertation advances theoretical understandings of the complex interactions between individual, family, peer, and

neighborhood contexts to influence adolescent health outcomes. By presenting an ecologically sensitive framework with which to understand the substance use risk associated with adolescent girls' pubertal development, this study should encourage additional research that accounts for the multiple levels of ecological correlates that play a role in the health of adolescents and emerging adults.

Finally, this dissertation advances the field of prevention science and adolescent health by unpacking and contextualizing the substance use risk associated with pubertal development. Intervention programs aimed at promoting abstinence and/or reducing substance use among adolescent girls can be guided by the knowledge of potentially gender- and race/ethnicity-specific risk. For instance, it may be useful for intervention programs to reduce the perceived stigma associated with early pubertal development among adolescent girls in specific social locations. Intervention programs obviously cannot alter pubertal development, but by understanding the importance of reflected appraisals, social relationships, neighborhood contexts, and race/ethnicity, these programs can address the key mechanisms that link pubertal development with adolescent girls' substance use. Thus this dissertation should be informative for researchers and practitioners interested in reducing the substance use risk associated with pubertal development, but should also be used to advance further examination of the complex interactions between individual and contextual factors as they relate to health outcomes like substance use.

OUTLINE

This dissertation includes three empirical studies using data from the first three waves of the National Longitudinal Study of Adolescent Health. It examines why and how pubertal

development is related to adolescent girls' substance use. The three empirical studies, although linked by this common theme, are also intended to be stand-alone pieces. Therefore, the analytic samples, operational definitions of key measures, and analytic strategies may vary across the three studies. Chapter 2 examines the moderating role of body weight on the association between pubertal development and adolescent girls' substance use, and emphasizes the changing relationship between pubertal development, substance use, and body weight as girls mature from early adolescence to late adolescence. Chapter 3 addresses why pubertal development is positively associated with adolescent girls' substance use, and focuses on the mediational role of intrapersonal self-appraisals and interpersonal social relationships. Finally, Chapter 4 highlights the importance of social context by modeling the complex moderating roles of neighborhood contexts and race/ethnicity in the relationship between pubertal development and adolescent girls' substance use. Chapter 5 summarizes the three empirical studies and discusses the theoretical and policy implications of the dissertation as a whole.

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CHAPTER II

NEGOTIATING THE EARLY DEVELOPING BODY: PUBERTAL DEVELOPMENT, BODY WEIGHT, AND ADOLESCENT GIRLS' SUBSTANCE USE

Adolescence, defined as the second decade of life (World Health Organization 2007), is a tumultuous period during which emerging young adults must negotiate important developmental transitions with the goal of establishing their identity in relation to peers, family, and society. One such developmental transition is puberty. Although puberty is the process of physical reproductive maturation, it is heavily imbued with cultural, psychological, and social significance. Body changes (e.g., breast and hip development) that occur during puberty among adolescent girls introduce social roles, responsibilities, and expectations associated with the newly sexualized and potentially fecund body (Lee 1994; Martin 1996). The timing of puberty thus has important consequences for adolescent girls given that early transitions may force a premature negotiation of emergent meanings and identities associated with the pubertal body.

Adolescent girls that experience puberty earlier than their peers may be at risk due to their perceived social deviance from same-age peers. To negotiate the meanings and labels associated with the pubertal body, early developing girls may be more likely than peers to engage in risky health behaviors such as substance use. Compared to boys, early pubertal development may be risky for girls because they are more likely to use substances to reduce negative affect (Newcomb et al. 1988; Opland, Winters, and Stinchfield 1995). Boys are also more likely to be satisfied with the pubertal body given its increased weight and muscle

mass, whereas girls primarily gain fatty adipose tissue during puberty that conflicts with thin beauty ideals (Blyth, Simmons, and Zakin 1985). It follows that early developing girls who are already over-weight may more easily incorporate the meanings associated with developing breasts and hips into their extant identities as 'chunky,' 'thick,' or developmentally advanced. Early pubertal development may be more risky for under- and average weight girls, however, because bodily changes during puberty may be more noticeable to others and therefore more likely to necessitate a negotiation of self.

In response to Schulenberg, Maggs, and Hurrelmann's (1997: 7) call for research that focuses on the individual and contextual conditions under which developmental transitions contribute to health risks and opportunities, I examine the moderating role of body weight in both the immediate (i.e., cross-sectional) and long-term (i.e., longitudinal) associations between pubertal development and adolescent girls' substance use. Prior to outlining my research hypotheses, I discuss the theoretical and empirical literatures linking pubertal development, body weight, and substance use among adolescent girls.

THEORY

Developmental transitions such as puberty are consequential largely due to the 'mismatches' or discontinuities associated with them—discontinuities in expected social roles and behaviors, perceived self and body image, and actual and expected individual characteristics (Schulenberg et al. 1997). Adaptive outcomes during developmental transitions are therefore more likely to occur when there is a match or fit between an individual's characteristics and the demands of a social setting (Lerner 1976: 14). Both developmental and identity theories address the potential mismatches that may occur for the

early developing adolescent girl, and how those mismatches may lead to risky health behaviors such as substance use.

Based in a developmental approach, stage termination theory (Stattin and Magnusson 1990) posits that adolescent girls who experience early pubertal development are perceived, or perceive themselves to be, developmentally and socially deviant from same-age peers. Early developers may experiment with substance use because they are not psychologically or socially prepared to cope with this developmental mismatch (Caspi and Moffitt 1991; Petersen and Taylor 1980; Stattin and Magnusson 1990). Peers, teachers, and adults may interact differently with early developing girls, such as wrongly labeling them as sexually precocious or deviant (Lee 1994; Martin 1996; Thorne 1993). Early developers may be unhappy with the weight gain and bodily changes associated with puberty that distance them from same-age peers. Subsequently, early developers may seek access to older peer networks (including older boys) and hence opportunities to use substances, but also have less experience managing such opportunities (i.e., learning to reject offers of substances).

Implicit in stage termination theory is that maladaptive outcomes, including risky health behaviors such as substance use, result from the developmental mismatch between the psychosocially immature yet physically mature(ing) adolescent. Theories of identity further explicate the social psychological processes involved in adolescent girls' and boys' negotiation of this mismatch. A critical developmental task of adolescence is the establishment and stabilization of identity (Erikson 1968), broadly defined as "parts of a self composed of the meanings that persons attach to the multiple roles they typically play" (Stryker and Burke 2000: 284). Identities are comprised of standards, or subjective culturally prescribed meanings (Burke 1991; Cast and Burke 2002), that define a role identity in a

specific situation. Standards serve as bases of comparison for social input such as self-attributions (i.e., how individuals perceive themselves) and reflected appraisals (i.e., how individuals think others perceive them). Identity control theory posits that during social interaction individuals attempt to verify identities by acting consistently with their perceived identity and counteracting meanings inconsistent with their perceived identity (Burke 1991; Burke 2004; Burke and Cast 1997). Through the process of self-verification individuals attempt to create stable identities by aligning/matching perceived relevant meanings from a social situation with their identity standards. When individuals are unable to match inputs and standards, however, they will adjust their behavior until social inputs become aligned with their identity standards, change the identity standard itself, or engage in risky behaviors to reduce negative affect (Burke 1991; Burke and Cast 1997).

Thus, identity control theory posits that individuals undergo a continual process of self-verification to create stable identities, and mismatch between inputs and standards can encourage risky behaviors. The process of self-verification may be particularly salient for early developers given adolescent girls' heightened sensitivity to social comparisons and peer relationships for identity development (Brown 1990; Silbereisen and Noack 1988). Early developing adolescent girls may therefore be at greater risk of substance use due to a forced premature negotiation of reflected appraisals associated with the developing, womanly body and its irreconcilability with a pre-adolescent identity. Mismatch in inputs and standards associated with the early developing body may be less extreme (and hence less risky) for over-weight girls, however. Over-weight girls may have already internalized the meanings associated with a larger more womanly body, and/or may be less sexualized by peers and adults. Conversely, this mismatch may be most pronounced for adolescent girls who are

under- or average weight and therefore must negotiate the meanings associated with more noticeable changes in the pubertal body.

PREVIOUS RESEARCH

Consistent with predictions from stage termination and identity control theories, research has established a robust positive association between early pubertal development and adolescent girls' substance use (see Aro and Taipale 1987; Biehl, Natsuaki, and Ge 2007; Chung, Park, and Lanza 2005; Deardorff et al. 2005; Dick et al. 2000; Lanza and Collins 2002; Magnusson 1988; Michaud, Suris, and Deppen 2006; Stice, Presnell, and Bearman 2001; Wilson et al. 1994). Specifically, studies among U.S. and European girls with earlier age at menarche and earlier body development compared to peers indicate that they are more likely to use tobacco, alcohol, marijuana, and other substances. Most prior studies have relied on cross-sectional or retrospective data; several European studies with longitudinal data, however, indicate that the substance use risk associated with early pubertal development may attenuate in late adolescence (e.g., Aro and Taipale 1987; Magnusson 1988). Thus, gaps remain in the literature regarding whether the positive association between early pubertal development and substance use persists beyond early adolescence and whether it varies for girls of different body weights.

Studies conducted in Europe suggest that the substance use risk associated with early pubertal development is most pronounced in early to mid-adolescence but attenuates over time. Using longitudinal data from 506 Swedish girls beginning in third grade in 1965, Magnusson (1988) found that early developers, defined as those who experienced menarche before age 12, reported higher levels and frequencies of alcohol and marijuana use by age

14.5. By age 26, early developers still exhibited higher marijuana use levels but differences in alcohol use were attenuated (see also Stattin and Magnusson 1990). Another study (Aro and Taipale 1987) used 1981 data from 935 eighth-grade Finnish girls and found that early developers, defined as those who experienced menarche before age 12, exhibited higher levels of alcohol use at age 14. By age 16, however, early developers no longer drank significantly more than their peers. A more recent study (Dick et al. 2000) using data from 1,903 Finnish twins born between 1975 and 1979 found that girls who experienced menarche before age 12 were more likely than their peers to begin cigarette/alcohol use at an early age; these differences persisted at age 16. By age 18.5 the effect of early pubertal development on drinking was lessened but still present.

In contrast, most U.S. studies have relied on cross-sectional samples and have not examined the longitudinal relationship between early pubertal development and adolescent girls' substance use (e.g., Deardorff et al. 2005; Stice et al. 2001; Wilson et al. 1994). Further, those U.S. studies with longitudinal data have only short follow-up periods (e.g., one to two years) and have not found the attenuation of effects observed in the aforementioned European studies. For instance, using data from the first two waves of the National Longitudinal Study of Adolescent Health (a subset of the data used in the present study), Lanza and Collins (2002) found that early developers, defined as those with substantial changes in breast and body curve development since grade school, exhibited the most advanced stage of substance use in 7^{th} grade. They also found that early developers were more likely to progress from abstinence to using substances between 7^{th} and 8^{th} grade, and more likely to increase substance use in general regardless of their baseline use level. Using similar data and measures, Chung et al. (2005) found that 12 to 15 year old girls that reported

substantial changes in breast and body curve development (i.e., early developers) were more likely to advance in level of substance use compared to late developers. These studies, however, did not include a long-term follow-up period to examine whether the association between early pubertal development and substance use persisted beyond mid-adolescence. The only U.S. study with a long-term (i.e., 7 year) follow-up period (Biehl et al. 2007) examined the association between pubertal development and alcohol use for adolescent girls and boys using data from the National Longitudinal Study of Adolescent Health. They found that early developers, or those scoring one standard deviation above the mean on a multi-item pubertal development scale, exhibited higher alcohol use trajectories, but this effect dissipated slightly over time for girls. This study only examined alcohol use trajectories, however, and did not examine the association between adolescent girls' pubertal development and other substances such as tobacco.

In sum, research has consistently provided support for a cross-sectional association between early pubertal development and adolescent girls' substance use, yet evidence for a longitudinal association is more limited, particularly among U.S. girls. More research is needed to examine the potential attenuation of the effect of early pubertal development and substance use among adolescent girls in the United States. Further, to date no empirical research has examined whether the substance use risk associated with adolescent girls' early pubertal development varies by body weight. As a consequence, systematic research is needed to test predictions derived from developmental and identity theories regarding the moderating role of body weight in the association between early pubertal development and substance use. In particular, more research is needed to examine whether the substance use

risk associated with early pubertal development is lower for over-weight girls compared to under- and average weight girls.

STUDY SIGNIFIANCE

This study contributes to the literature in several ways. First, I highlight how body weight, one salient individual condition during adolescence, influences the way in which the early developmental transition of puberty contributes to a specific risky health behavior among adolescent girls—substance use. The focus on the moderating role of body weight helps develop sensitive understandings of how risk factors are situated in social contexts. However, this analysis is exploratory given the lack of previous research on whether the developmental mismatch associated with the early developing body may be less extreme (and hence less risky) for over-weight girls. Second, I examine both the cross-sectional and longitudinal associations between early pubertal development and substance use among adolescent girls. With seven-year follow-up data, I investigate whether the immediate consequences of early pubertal development on substance use risk persist over time. Finally, I use a national probability sample from the United States and improve upon previous U.S. studies that have relied on regional or non-probability samples (e.g., Deardorff et al. 2005; Stice et al. 2001; Wilson et al. 1994), have focused on early adolescence only (e.g., Chung et al. 2005), or have focused only on alcohol use outcomes (Biehl et al. 2007).

HYPOTHESES

This study will address the following research questions. First, what is the relationship between early pubertal development and adolescent girls' substance use?

Second, how does body weight moderate the relationship between early pubertal development and adolescent girls' substance use? Finally, do the associations between early pubertal development, body weight, and substance use persist over time? The following hypotheses will be tested:[2]

H1: Early developing adolescent girls will have higher levels of substance use than on-time developers.

H2: The positive relationship between early pubertal development and substance use will attenuate over time.

H3: Over-weight early developing adolescent girls will have lower levels of substance use than under- and average-weight early developing girls.

H4: The moderating role of body weight described in H3 will attenuate over time.

H5: Early developing adolescent girls will be more likely to be heavy substance users than on-time developers.

H6: The positive relationship between early pubertal development and heavy substance use will attenuate over time.

H7: Over-weight early developing adolescent girls will be less likely to be heavy substance users than under- and average-weight early developing girls.

H8: The moderating role of body weight described in H7 will attenuate over time.

[2] Note that hypotheses 3, 4, 7, and 8 are exploratory given the lack of previous empirical research on the moderating role of body weight in the relationship between early pubertal development and substance use.

METHODS

Sample

This study used longitudinal restricted-use data from three waves of the National Longitudinal Study of Adolescent Health (Add Health) (http://www.cpc.unc.edu/addhealth). Add Health used a multistage, stratified, school-based sampling design to collect data representative of U.S. adolescents attending grades 7–12 during the 1994–1995 school year. At Wave I, 90,118 students completed brief in-school questionnaires and 145 school administrators completed questionnaires. Drawn from the participating school rosters and in-school survey participants, a pool of 20,745 adolescents completed more in-depth in-home interviews in 1995. The in-home sample consisted of a nationally representative "core" sample; over-samples of high-education blacks, Cubans, Puerto Ricans, Chinese, and disabled respondents; and a twin/sibling sample. Parents/guardians of the in-home interview participants were also interviewed at Wave I. At Wave II a representative sample of students was surveyed from Wave I participants, supplemented by a sample of students absent at Wave I. This resulted in a sample of 14,738 students interviewed in their homes in 1996. The sampling design for Wave III was similar to that for Wave II and resulted in a sample of 15,197 respondents and 1,507 romantic partners of respondents interviewed in their homes in 2001-2002. See Udry, Bearman, and Harris (2006) for more detailed sampling design information.

Add Health employed a clustered sampling design with schools serving as primary sampling units (PSUs) and region of the country (Northeast, Midwest, South, and West) as a stratification variable. A total of 132 PSUs (80 high schools and 52 "feeder" middle schools)

were selected from a sample frame of 26,666 schools, and probability sampling weights were used to adjust for non-response and unequal probability of selection.

The analytic sample used herein included girls age 10 to 15 at Wave I with non-missing sampling weights.[3] This age group, which corresponds to the Centers for Disease Control's definition of early adolescence, was intended to capture the developmental stage characterized by major changes in the female pubertal body. There were 5,591 young adolescent girls with non-missing sampling weights at Wave I, 4,732 at Wave II, and 4,827 at Wave III. In addition, values coded as "refused", "don't know", or "missing" were treated as missing and cases were deleted listwise. The percent of cases lost due to missing data was less than 10% for all self-reported variables; 12% of cases from the parent-reported variables at Wave I were lost due to missing data.

Measures

Dependent variables of interest were number of substances tried and heavy substance use. Number of substances tried was a count variable of self-reported total number of substances a respondent had tried during her lifetime, including tobacco, alcohol, marijuana, cocaine, injection drugs, inhalants, and other drugs (e.g., LSD, PCP, ecstasy, mushrooms, speed, ice, heroin, or pills without a prescription); it ranged from 0 (none) to 7 (7 or more substances). The number of substances tried outcome was measured at all three waves of data collection. Heavy substance use was a dummy variable constructed for respondents who reported trying at least one substance (1=yes; else=0) and met at least one of the following

[3] Approximately 8-10% of cases sampled at each wave had missing sampling weights. These respondents were selected outside of the sampling frame at Wave I to be included in the twin/sibling sample, and were sampled to ensure large enough sample sizes to obtain genetically related respondents. Because these cases were sampled outside of the sampling frame they did not receive sampling weights and were excluded from all analyses (see Chantala 2006).

criteria: (a) smoked cigarettes every day in the past month, (b) reported binge-drinking once a week or more during the past year,[4] (c) smoked marijuana at least once a day in the past month, or (d) reported any cocaine, injection, or other drug use in the past month. These two dependent variables were chosen to capture both counts and levels of substance use, and to additionally provide sensitivity analyses as to whether the substance use risk associated with early pubertal development and body weight is robust across different substance use outcomes.

Independent variables of interest were pubertal development and body weight. Pubertal development was trichotomized into early, on-time, and late development based on the upper 20th, middle 60th, and lower 20th percentile (respectively) scores according to a scale created from three measures of perceived pubertal development.[5] Add Health included a version of Petersen et al.'s (1988) Pubertal Development Scale (PDS) that asked girls about: increases in breast size since grade school, increases in body curviness since grade school, and physical development compared to same-age peers.[6] These items were rated on a scale of 1 to 5 with lower scores representing less physical development; the items exhibited high internal consistency (Cronbach's α =.68). Respondents scoring in the upper 20th percentile of the PDS scale were categorized as "early" developers, those in the middle 60th

[4] The Add Health does not include measures of binge-drinking in the past month, and so the past year measure of binge-drinking is included here.

[5] Pubertal development was trichotomized to distinguish between early, on-time, and late developers given that theoretical predictions specify that early developers are at increased risk compared to on-time (rather than late) developers. A measure of late development was included to provide additional exploratory analyses that speak to the theoretical and empirical literatures addressing the substance use risk and/or protection associated with late pubertal development (e.g., see Brooks-Gunn and Warren 1985; Graber et al. 1997; Graber et al. 2004; Petersen and Taylor 1980; Tschann et al. 1994; Williams and Dunlop 1999).

[6] Another common measure of adolescent girls' pubertal development is age at menarche. Although this measure was available in Add Health, I chose to use the PDS items as they more fully capture changes in visible maturation that are noticeable by others and therefore more likely to elicit changes in a girl's identity. Menarche, on the other hand, is a non-visible marker of development that can be hidden from others. Additional analyses using age at menarche as a control variable significantly decreased the listwise sample size, but did not substantively alter the results reported here.

were "on-time" developers, and those in the lower 20th were "late" developers.[7] Analyses used pubertal development variables from Waves I & II; pubertal development was not measured in the Add Health at Wave III.

Body weight was trichotomized into under-, average, and over-weight based on the lower 20th, middle 60th, and upper 20th percentile (respectively) scores from respondents' standardized body mass index score (Z-BMI). The Z-BMI score is an anthropometric indicator of adolescent girls' body mass index relative to other U.S. girls of the same age. Z-BMI scores are calculated as the standard deviation score of a respondent's body mass index compared to the median value[8] of body mass index for girls of the same age based on the 2000 CDC growth charts (see Quantitative Techniques 2003 for more information). Girls in the lower 20th percentile of the Z-BMI distribution were categorized as under-weight, those in the middle 60th were average,[9] and those in the upper 20th were categorized as over-weight. Analyses included body weight variables from Waves I & II.

Control variables included parental education, family structure, substance using friends, age, race/ethnicity, and nativity status. These control variables were included because previous research (Arim et al. 2007; Blake et al. 2001; Flewelling and Bauman 1990; Goodman and Huang 2002; Hawkins, Catalano, and Miller 1992; Kaplowitz et al. 2001) has established their connection to pubertal development and substance use among adolescents. Parental education was a parent-reported ordinal variable measuring the maximum level of education attained by either of the respondent's parents, and ranged from 0 (no school) to 9

[7] Sensitivity analyses also used early and late cutoffs calculated as +/- 1 standard deviation above/below the mean of the PDS scale. This alternate cutoff specification did not substantively alter the findings reported herein.

[8] Note that the Z-BMI values are calculated as relative to the median value (rather than mean) due to skew in the variable.

[9] Note that this nomenclature refers to a statistical "average" relative to the other girls in the sample rather than an average weight based on national standards of healthy body weights for adolescent girls.

(professional degree). Family structure was measured by parent reports of marital status and recoded into a dummy variable (non-married) so that 1=non-married household and 0=married. Substance using friends was a dummy variable created so that 1=respondent reported any friends that use tobacco, alcohol, or marijuana and 0=no substance using friends. Age was an ordinal measure of respondents' self-reported age in years. Race/ethnicity was recoded into four indicator variables representing non-Latina black (1=yes), Mexican American (1=yes), non-Latina white (1=yes), and other race/ethnicity (1=yes). Nativity status was a dummy variable (immigrant) created so that 1=born outside of United States and 0=born inside United States. Analyses included parental education, family structure, race/ethnicity, and nativity status measured at Wave I, and substance using friends and age from Waves I & II.

Analytic Strategies

Given that the number of substances tried outcome was a non-negative count variable with severe positive skew, methods such as Ordinary Least Squares that assume normality and homoskedasticity of residuals could introduce inefficiency, inconsistency, and bias into regression models (Long 1997: 217).[10] Poisson regression models, which are appropriate when modeling non-negative count outcomes containing zero-values and strong positive skews, were used for the number of substances tried outcome (Long 1997). For the Poisson regression model with discrete outcome Y, observed frequencies y_i (i = 1, …., N), and covariates $\mathbf{X}_i$, the distribution of observations is represented as

$$P\,[Y = y_i] = e^{-\lambda i}\,\lambda_i{}^{yi} / y_i!,$$

[10] In fact, mathematical transformations intended to reduce severe positive skew such as the logarithmic and negative reciprocal root transformations failed to produce normally distributed outcomes for the number of substances tried measure, and thus more appropriate methods for the dependent variable were chosen.

where λ_i is the mean (or the rate) of y_i. The Poisson model can thus be written as

$$\lambda_i = \exp(\mathbf{X}_i * \boldsymbol{\beta})$$

where $\mathbf{X}_i$ is the matrix of independent variables on individuals i and $\boldsymbol{\beta}$ is a vector of regression coefficients relating independent variables to the mean number of substances tried rate. The Poisson regression coefficients can be interpreted such that $\exp[\beta_j]$ is the incidence rate ratio or predicted rate ratio (i.e., number of events per time) for a one-unit increase in predictor j, holding other variables in the model constant. Incidence rates can also be interpreted so that $[(\exp[\beta_j]-1)*100]$ is the predicted percentage change in the number of substances tried incidence rate for a unit increase in a predictor j, net of the other variables in the model (Long 1997).

With binary outcomes such as the heavy substance use outcome, linear models that assume normality and homoskedasticity of residuals, linear functional form, and allow predicted values unbound by [0,1] can introduce inefficiency, inconsistency, and bias in the models (Long 1997: 39). Logistic regression models, which are appropriate for modeling binary dependent variables, were used for the heavy substance use outcome. With the dependent variable Y coded so that 1=heavy substance use, the logistic regression model can be written as

$$P(Y = 1 \mid x) = \frac{\exp(\mathbf{X}_i * \boldsymbol{\beta})}{1 + \exp(\mathbf{X}_i * \boldsymbol{\beta})}$$

where $\mathbf{X}_i$ is the matrix of independent variables on individuals i and $\boldsymbol{\beta}$ is a vector of regression coefficients relating independent variables to the probability of heavy substance use. The logistic regression coefficients can be interpreted such that $\exp[\beta_j]$ is the odds (or in the case of a set of dummy variables with an excluded category, the odds ratio) of heavy

substance use associated with a one-unit increase in predictor j, holding other variables in the model constant. Odds can also be interpreted so that [(exp[β_j]-1)*100] is the predicted percentage change in the odds of heavy use for a unit increase in a predictor j, net of the other variables in the model.

The clustered sample design and hence correlated error structure of the Add Health data required statistical corrections for design effects and unequal probabilities of selection (Chantala and Tabor 1999). A Taylor Series variance estimation method was used to estimate standard errors and insured consistency in the probability of Type I errors. For the cross-sectional Wave I and Wave II models and longitudinal models, probability weights (variables GSWGT1, GSWGT2, GSWGT3_2, respectively), strata (variable REGION), and primary sampling units (variable PSUSCID) were specified in Stata 9.2 for use with the survey-based Poisson and logistic regression procedures.

The Poisson regression model assumes that events are independent and that the conditional mean and variance of the outcome are equal (i.e., the assumption of equidispersion). Results indicated that some of the reported models violated the assumption of equidispersion; however, this violation did not substantively alter the findings and therefore the Results section presents findings from the Poisson regression models. Namely, estimation of probability-weighted negative binomial regression models indicated that the assumption of equidispersion was satisfied for the cross-sectional models at Wave I. For the remaining models (cross-sectional Wave II, longitudinal Wave II, and longitudinal Wave III), there was evidence of slight overdispersion (i.e., the conditional variance exceeded the conditional mean). Thus, survey adjusted negative binomial models would be more appropriate models for these overdispersed complex survey data. However, variance

estimates for the survey adjusted negative binomial regression models were incalculable due to the small sub-population sample sizes (i.e., young females by body weight) relative to the overall population. Despite these problems with convergence, the substantive and statistical findings of the survey adjusted Poisson models were similar to results from probability-weighted negative binomial regression models. Results from the survey adjusted Poisson models are therefore reported in the Results section.

RESULTS

Descriptive Statistics

Table 2.1 shows the means, standard deviations, and ranges of substance use, pubertal development, and social location variables by body weight and wave of data collection. As shown in the top panel of Table 2.1, at Wave I girls had tried only two substances on average; under-weight girls tried the most substances followed by average and over-weight girls. So in early adolescence, the typical girl had only tried two substances (usually tobacco and alcohol); under-weight and average weight girls had tried more substances than over-weight girls, perhaps due to increased availability at social events or parties. Approximately 17% of girls who reported using substances were heavy users, with under-weight girls most likely to be heavy users. Although the average adolescent girl had only tried a few substances, a minority of these girls were heavily using substances in the past month. Finally, at Wave I approximately 19% of girls were early developers and 14% were late developers.[11]

[11] Note that these percentages are slightly less than the 20% cut-offs for the early and late pubertal development scale due to differences in the percentile cut-points and plausible values on the PDS scale.

For social location variables, standardized body mass index (Z-BMI) scores averaged .57 for the aggregated sample of girls at Wave I (see Table 2.1). On average, girls' parents had a GED or attended vocational/trade school; the average level of parental education was highest for under-weight girls. As shown in Table 2.1, approximately 32% of respondents lived in non-married households; 35% of over-weight girls but only 26% of under-weight girls lived in non-married parent households. The majority (77%) of girls reported having friends who used substances; average weight girls were the most likely to report having substance using friends, which may be due to their increased popularity and peer networks. The average age of respondents at Wave I was 13.82 and was statistically equivalent across body-weight categories. Finally, approximately 13% of respondents were non-Latina black, 6% were Mexican American, and 73% were non-Latina white. Of the three body weight categories, black girls were most likely to be over-weight, Mexican American girls most likely to be of average weight, and white girls were most likely to be under-weight. Finally, less than 4% of the girls were born outside of the United States and immigrant girls were more likely to be under-weight than average or over-weight.

As shown in the middle panel of Table 2.1, the number of substances tried remained low for girls at Wave II. The average number of substances tried was slightly lower at Wave II than Wave I (2.14 vs. 2.09 for all girls). This decrease was relatively small, however, and may merely be an artifact of recall bias, the timing of specific substance use questions during data collection (e.g., use in the past month may decrease during months with increased parental supervision, such as December), and the result of the fact that girls who attritted in Wave II had a slightly higher average number of substances tried than those retained in the second wave. By Wave II 25% of respondents that used substances were heavy users and

there were no significant differences in heavy use by body weight category. This may be because girls with different body weights have differential access to substances to try, but whether they actually become heavy users is due to factors other than body weight (e.g., availability). Similar to Wave I, under-weight girls were least likely to be early developers and over-weight girls were least likely to be late developers at Wave II. Z-BMI scores averaged .40 for the aggregated sample of girls at Wave II. Most respondents had friends who used substances at Wave II; average weight girls were most likely to have substance using friends but there were no significant differences across body weight categories. The average age of respondents at Wave II was 15.

Table 2.1. Means, Standard Deviations, and Ranges of Substance Use, Pubertal Development, and Social Location Controls by Body Weight and Wave of Collection[ab]

	All Girls	Under-weight	Average	Over-weight	
	M (SD)	M (SD)	M (SD)	M (SD)	Range
Wave I[c]					
Substance Use					
Number of substances tried**	2.14 (1.36)	2.21 (1.39)	2.17 (1.38)	1.95 (1.30)	0-7
Heavy substance use (1=yes)*	16.52% (.36)	19.64% (.38)	17.36% (.37)	9.95% (.34)	0-1
Pubertal Development					
Early (1=yes)***	18.52% (.36)	8.23% (.22)	18.23% (.36)	30.45% (.43)	0-1
Late (1=yes)***	13.50% (.39)	20.19% (.45)	11.74% (.37)	12.22% (.35)	0-1
Social Location					
Z-BMI***	.57 (.89)	-.71 (.50)	.55 (.44)	1.79 (.34)	-4.68-2.89
Parental education***	5.85 (2.31)	6.13 (2.23)	5.90 (2.28)	5.46 (2.36)	0-9
Non-married parent (1=yes)***	31.97% (.46)	26.03% (.43)	32.64% (.46)	34.76% (.49)	0-1
Substance using friends (1=yes)*	76.52% (.49)	76.91% (.50)	77.15% (.49)	75.10% (.49)	0-1
Age†	13.82 (1.09)	13.90 (1.10)	13.85 (1.08)	13.65 (1.11)	10-15
Non-Latina black (1=yes)***	12.97% (.42)	5.35% (.35)	12.74% (.42)	20.63% (.47)	0-1
Mexican American (1=yes)	5.84% (.26)	5.04% (.22)	5.97% (.25)	5.68% (.28)	0-1
Non-Latina white (1=yes)***	72.65% (.50)	80.35% (.48)	73.00% (.49)	65.57% (.50)	0-1
Immigrant (1=yes)***	3.50% (.25)	5.82% (.29)	3.33% (.25)	1.47% (.19)	0-1
Wave II[c]					
Substance Use					
Number of substances tried**	2.09 (1.31)	2.09 (1.34)	2.12 (1.32)	1.99 (1.27)	0-7
Heavy substance use (1=yes)	25.30% (.43)	28.87% (.44)	24.44% (.42)	24.36% (.43)	0-1

Table 2.1 cont.

	All Girls	Under-weight	Average	Over-weight	
	M (SD)	M (SD)	M (SD)	M (SD)	Range
Pubertal Development					
Early (1=yes)***	17.70% (.36)	9.01% (.23)	16.23% (.35)	30.68% (.43)	0-1
Late (1=yes)***	15.42% (.39)	26.46% (.45)	13.11% (.37)	11.62% (.34)	0-1
Social Location					
Z-BMI***	.40 (.96)	-.97 (.56)	.40 (.44)	1.73 (.36)	-4.51-2.80
Substance using friends (1=yes)	84.14% (.47)	82.16% (.49)	85.21% (.47)	82.86% (.47)	0-1
Age***	15.26 (1.19)	15.50 (1.18)	15.21 (1.19)	15.20 (1.19)	11-17
Wave III[c]					
Substance Use					
Number of substances tried*	2.56 (1.53)	2.50 (1.52)	2.61 (1.55)	2.48 (1.47)	0-7
Heavy substance use (1=yes)	35.57% (.47)	33.43% (.46)	36.56% (.46)	36.13% (.47)	0-1

Notes. [a]Standard deviations not adjusted for complex survey design. [b]Equality of means across body weight categories tested with F-statistics from probability weighted one-way ANOVAs for continuous variables and survey adjusted Design-based F-statistics from two-way contingency tables for categorical variables. [c]Wave I estimates are split by body weight at Wave I; Wave II estimates are split by body weight at Wave II; Wave III estimate are split by body weight at Wave II.
† p<.10. * p<.05. ** p<.01. *** p<.001.

Finally, as shown in the last panel of Table 2.1, respondents reported having tried an average of 2.56 substances at Wave III. This is not surprising given that some respondents were over age 18 and would have legal access to cigarettes (and in some cases, alcohol). Girls who were of average weight (versus under- or over-weight) at Wave II had tried the most substances by Wave III. This is consistent with findings from Wave II, such that average weight girls had tried the highest number of substances. At Wave III almost 36% of substance users were heavy users, although there were no differences in heavy substance use by body weight category. These changes across time in substance use by body weight category suggest developmentally specific associations between body weight and substance use. Whereas over-weight is consistently associated with less substance use, under-weight is associated with greater substance use in early adolescence but average weight is associated with greater substance use in mid-adolescence. These relationships may be due to shifting peer contexts and opportunities to use substances for girls of different body weights. For example, during mid-adolescence average weight girls may experience increased popularity and invitations to parties where alcohol and other substances are present.

Figure 2.1 displays the smoothed kernel density estimates for pubertal development and number of substances tried at Wave I. Late developers were most likely and early developers least likely to have abstained from any substances. Both on-time and late developers were more likely than early developers to have tried only one substance. Early developers were the most likely to have tried more than one substance, followed by on-time and late developers respectively. Thus, despite the low prevalence of substance use among respondents, early developers were at greater risk of having tried more substances compared to on-time and late developers. Consistent with stage termination theory, Figure 2.1 indicates

that early developers, compared to on-time and late developers, are at greater risk for substance use. Similar patterns emerge when examining heavy substance use patterns. Table 2.2 shows the distribution of heavy substance use by pubertal development. As shown in Table 2.2, among those girls who did report using substances, pubertal development was significantly associated with heavy substance use at Wave I ($F= 13.97$; $p<.001$). The odds of an early developer being a heavy versus light user were 1.66 times the odds of an on-time or late developer being a heavy user.

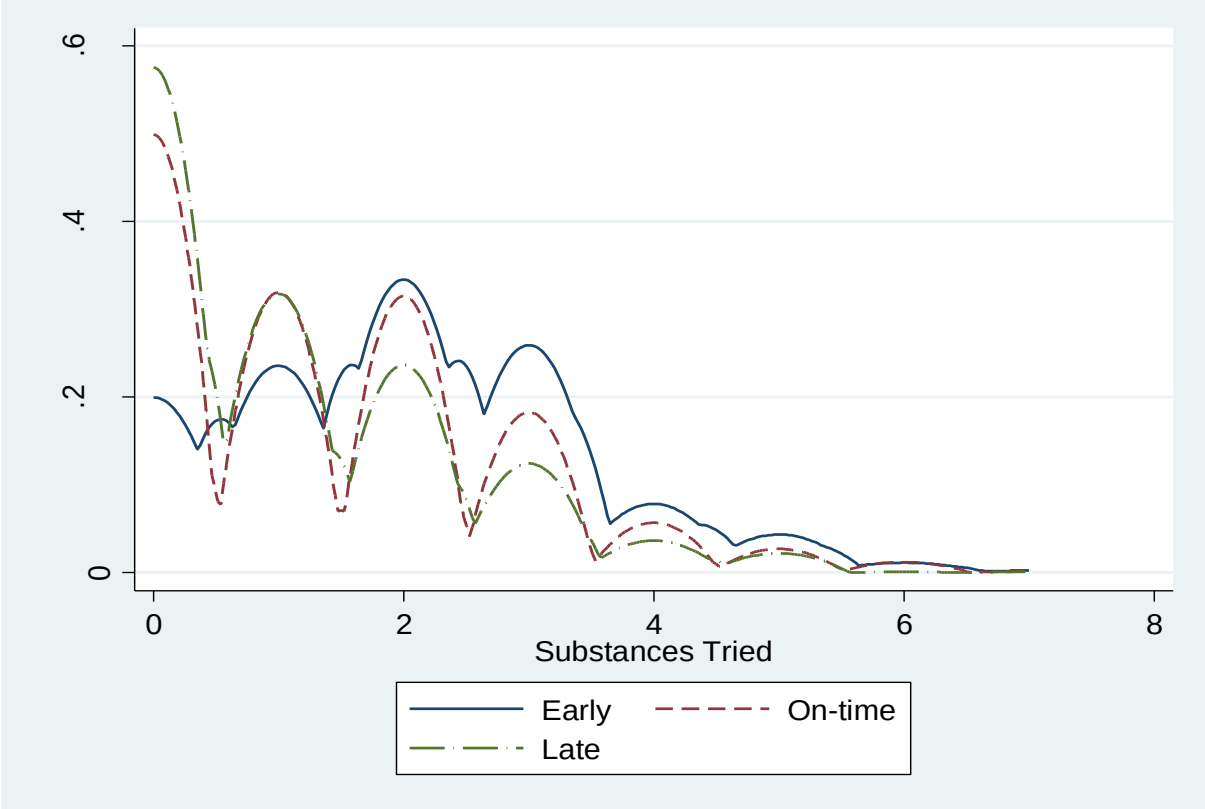

Figure 2.1. Smoothed Kernel Density Estimates of Number of Substances Tried by Pubertal Development: Add Health Wave I

Table 2.2. Distribution of Heavy Substance Use by Pubertal Development: Add Health Wave I

Cell count Column percentage	Early	On-time	Late
Light user	509 78.07%	2,006 84.75%	454 89.19%
Heavy user	143 21.93%	361 15.25%	55 10.81%
Total	652 100%	2,367 100%	509 100%

Note. Survey adjusted chi-squared test of row and column independence $\chi^2(2) = 270.71$, $p < .001$. Survey adjusted design-based $F(1.81, 231.67) = 13.97$, $p < .001$.

Table 2.3 shows the zero-order correlations between the substance use, pubertal development, and body weight measures.[12] Early pubertal development at Wave I was positively correlated with the number of substances tried at all waves (.23, .18, and .08, respectively), whereas late development was negatively associated with the number of substances tried at each wave (-.27, -.22, and -.11, respectively). Body weight, however, was only marginally correlated with the number of substances tried outcome. Finally, as would be expected from Table 2.1, over-weight was positively correlated with early development but negatively correlated with late development (.28 and -.15, respectively at Wave I). Conversely, under-weight was negatively correlated with early development and positively correlated with late development (-.36 and .25, respectively at Wave I).

[12] See Kolenikov and Angeles (2004: 16) for a detailed description of the likelihood functions used to estimate the polychoric, tetrachoric, and polyserial correlations shown in Table 2.3.

Table 2.3. Zero-order Polychoric, Tetrachoric, and Polyserial Correlation Matrix of Substance Use, Pubertal Development, and Body Weight: Add Health Waves I-III[a]

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Substances tried, Wave I	1.00												
2. Substances tried, Wave II	.72	1.00											
3. Substances tried, Wave III	.35	.40	1.00										
4. Early PD, Wave I	.23	.18	.08	1.00									
5. Late PD, Wave I	-.27	-.22	-.11	.00	1.00								
6. Early PD, Wave II	.23	.18	.08	1.00	.00	1.00							
7. Late PD, Wave II	-.27	-.22	-.11	-.97	1.00	.00	1.00						
8. Under-weight, Wave I	-.04	-.01	-.01	-.36	.25	-.36	.25	1.00					
9. Average weight, Wave I	.04	.03	.01	.02	-.13	.02	-.13	-.85	1.00				
10. Over-weight, Wave I	-.00	-.01	-.02	.28	-.15	.28	-.15	.00	-.87	1.00			
11. Under-weight, Wave II	-.03	-.01	.00	-.33	.25	-.33	.25	.86	-.54	-.72	1.00		
12. Average weight, Wave II	.00	.01	.00	-.01	-.11	-.01	-.11	-.54	.81	-.67	.00	1.00	
13. Over-weight, Wave II	.02	-.00	-.01	.27	-.13	.27	-.13	-.76	-.66	.93	-.98	-1.00	1.00

Note: [a]All correlations $\geq$ |.05| are significant at p<.05 or less.

Predicting Number of Substances Tried

Cross-sectional results. Table 2.4 presents cross-sectional results from survey adjusted Poisson regression models predicting number of substances tried for the aggregated sample and then split by respondents' body weight. Results from the cross-sectional Wave I models (i.e., 1994-1995) are shown in the left panel and results from the cross-sectional Wave II models (i.e., 1996) are shown in the right panel.[13] The first column in each panel presents results for the aggregated sample of girls, with the next three columns presenting results split by body weight. Models controlled for standardized body mass index, parental education, family structure, substance using friends, age, race/ethnicity, and nativity status.

As shown in the first column in the left panel of Table 2.4, for the aggregated sample of girls, early development was associated with a higher number of substances tried at both Wave I and Wave II. At Wave I and net of the other predictors, being an early rather than on-time developer was associated with a 16% ([1.16-1]*100) increase in the incidence rate of substances tried. Conversely, late development was associated with fewer substances tried at Waves I and II. At Wave I and net of the other predictors, being a late as compared to on-time developer was associated with a 24% decrease in the incidence rate of substances tried. By Wave II, the incidence rate of substances tried was 18% higher for early than on-time developers, and 14% lower for late developers than on-time developers.

[13] There are no cross-sectional Wave III models because pubertal development was not measured at this wave.

Table 2.4. Incidence Rate Ratios and Standard Errors from Survey Adjusted Poisson Regressions Predicting Cross-sectional Lifetime Number of Substances Tried by Body Weight: Add Health Waves I-II[ab]

	Cross-sectional Wave I[c]				Cross-sectional Wave II[c]			
	All Girls	Under-weight	Average	Over-weight	All Girls	Under-weight	Average	Over-weight
Pubertal Development								
Early	1.16***	1.21†	1.17**	1.12†	1.18**	1.14	1.21**	1.09
	(.05)	(.13)	(.06)	(.08)	(.07)	(.17)	(.08)	(.11)
Late	.76***	.77*	.76***	.74**	.86*	.92	.85†	.80
	(.04)	(.08)	(.05)	(.08)	(.05)	(.08)	(.07)	(.15)
Social Location								
Z-BMI	.96*				.96†			
	(.02)				(.02)			
Parental education	.99	.98	1.00	1.01	1.00	.97†	1.02†	.99
	(.01)	(.02)	(.01)	(.02)	(.01)	(.02)	(.01)	(.02)
Non-married parent	1.20***	1.15†	1.24***	1.14†	1.17***	1.20*	1.18***	1.08
	(.05)	(.08)	(.06)	(.08)	(.04)	(.10)	(.05)	(.09)
Substance using friends	3.14***	3.71***	3.09***	2.82***	3.88***	4.40***	3.81***	3.50***
	(.18)	(.47)	(.20)	(.29)	(.29)	(.62)	(.35)	(.46)
Age	1.11***	1.17***	1.12***	1.03	1.04*	1.07	1.04†	1.03
	(.02)	(.04)	(.02)	(.04)	(.02)	(.04)	(.02)	(.04)
Non-Latina black	.77***	.59**	.70***	.96	.65***	.39***	.65***	.72*
	(.06)	(.11)	(.05)	(.12)	(.06)	(.09)	(.08)	(.11)
Mexican American	1.11	1.14	1.02	1.32	1.07	.59†	1.08	1.25
	(.11)	(.25)	(.12)	(.24)	(.12)	(.16)	(.13)	(.23)
Non-Latina white	1.03	1.10	.94	1.18	1.05	1.15	1.09	.88
	(.06)	(.17)	(.06)	(.16)	(.08)	(.16)	(.11)	(.15)
Immigrant	.79†	.98	.71*	.61	.79*	.71†	.86	.57
	(.11)	(.25)	(.10)	(.27)	(.09)	(.14)	(.12)	(.23)

Table 2.4 cont.

	Cross-sectional Wave I[c]				Cross-sectional Wave II[c]			
	All Girls	Under-weight	Average	Over-weight	All Girls	Under-weight	Average	Over-weight
Constant	-1.97***	-2.78***	-2.02***	-1.06*	-1.41***	-1.79**	-1.49***	-1.03†
	(.23)	(.57)	(.27)	(.51)	(.28)	(.64)	(.33)	(.57)
N	4,570	901	2,926	909	3,946	765	2,405	779
F Statistic	77.90***	24.00***	68.16***	15.82***	47.48***	23.80***	30.85***	18.22***

Notes: [a]Incidence rate ratio = $\exp(\beta_j)$. [b]Standard errors for incidence rate ratios in parentheses. [c]Cross-sectional Wave I models split by body weight at Wave I; cross-sectional Wave II models split by body weight at Wave II.
† p<.10. * p<.05. ** p<.01. *** p<.001.

The split sample models provide a more nuanced description of the health risk associated with early pubertal development such that the changing pubertal body may be less consequential for over-weight girls. At Wave I, pubertal development was a consistent predictor of number of substances tried for under-weight, average, and over-weight girls (see columns 2 through 4 in left panel, Table 2.4). However, by Wave II the risk associated with early development for under-weight and over-weight girls was attenuated and remained significant only for girls of average weight (see columns 2 through 4 in right panel, Table 2.4). The protection associated with late development was also attenuated for under-weight and over-weight girls at Wave II, and only marginally associated with fewer substances tried for average weight girls. Additional models at Wave I and Wave II (not shown here) were run to test for moderation that included multiplicative interaction terms between the body weight and pubertal development categories; none of the interactions were statistically significant. This suggests that although the incidence rate ratios differed numerically across groups, the effects of pubertal development on number of substances tried was not substantively distinguishable between body weight groups. In addition, generalized Hausman specification tests were used to test equality of the early and late pubertal development coefficients across the three body weight models. The adjusted Wald tests from Wave I indicated no significant difference in coefficients for early (F=.26; p=.77) and late (F=.03; p=.97) developers across the body weight groups. The Wald tests from Wave II also indicated no significant difference for the early (F=.43; p=.65) and late (F=.32; p=.73) coefficients across the body weight groups. So although the split sample models indicated that the effect of pubertal development on number of substances tried varied by body weight, this moderation was not statistically significant.[14]

[14] The lack of statistical significance in the multiplicative interaction terms and tests of cross-model coefficient

Figures 2.2 through 2.5 depict these relationships by showing the predicted probability of number of substances tried by body weight.[15] Figure 2.2 shows the predicted probabilities of number of substances tried at Wave I for early developers by body weight. Early developing over-weight girls had the highest predicted probability of abstaining from substances at Wave I, whereas average weight girls had the lowest probability of abstaining. Early developing over-weight girls had the lowest predicted probabilities of having tried more substances. This is consistent with predictions that over-weight girls may experience less difficulty negotiating the identity mismatch precipitated by the early developing body because they have already internalized the social expectations associated with a larger body. The predicted probabilities for higher numbers of substances tried were similar for early developing under-weight and average weight girls, although early developing under-weight girls had slightly higher probabilities of trying four or more substances.

equality could be due to low statistical power and the disparate sample sizes across the three body weight groups.

[15] Note that the y-axes of these graphs have been restricted to better represent the slight variations in predicted probabilities across groups. All graphs were calculated based on predicted probabilities calculated from the split sample models across the three body weight groups.

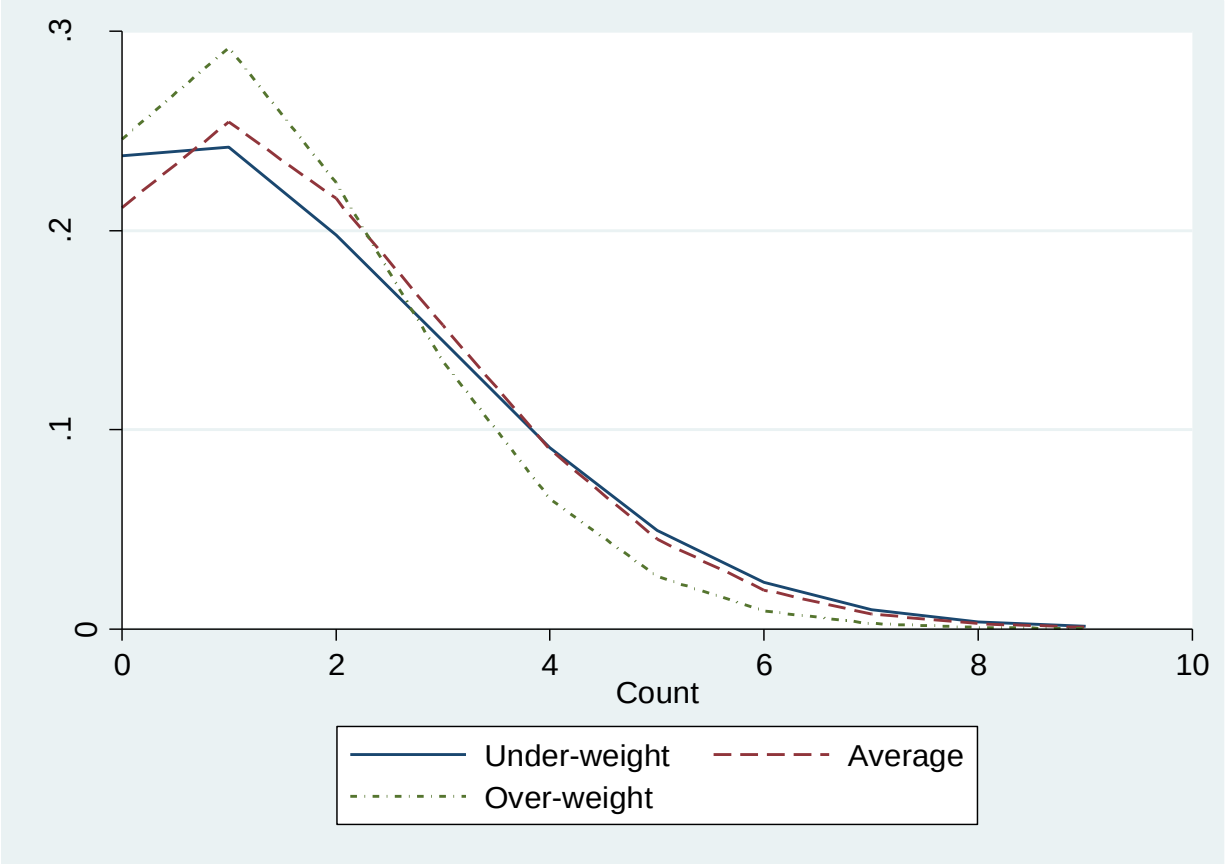

Figure 2.2. Predicted Probabilities of Early Developers' Number of Substances Tried by Body Weight: Cross-sectional Wave I

Figure 2.3 shows the predicted probabilities of number of substances tried at Wave II for early developers by body weight. Early developing under-weight girls were the most likely to abstain from substances at Wave II, with average weight girls again being the least likely to abstain. Similar to results from Wave I, early developing over-weight girls had the lowest predicted probabilities of trying more than two substances at Wave II. Again, the predicted probabilities of higher counts of number of substances tried were similar for early developing under-weight and average weight girls, but slightly higher for under-weight girls.

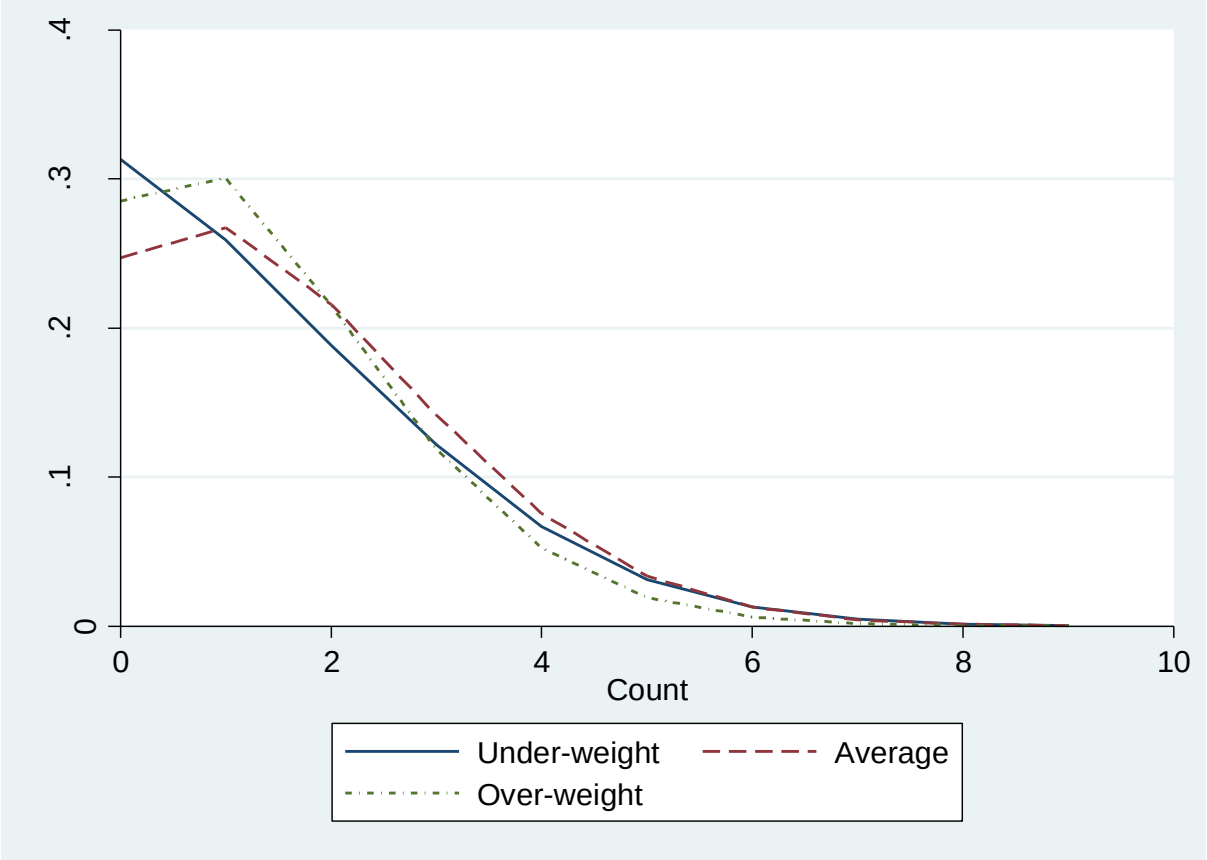

Figure 2.3. Predicted Probabilities of Early Developers' Number of Substances Tried by Body Weight: Cross-sectional Wave II

Figure 2.4 shows predicted probabilities for number of substances tried at Wave I for late developers by body weight. The predicted probabilities were similar for the three groups across the range of number of substances tried. Under-weight late developers had a slightly higher predicted probability of abstaining from substances at Wave II, whereas over-weight late developers had slightly lower probabilities of having tried two or more substances. These differences were relatively small, however, and magnified given the restricted range of the y-axis. The predicted probabilities from the cross-sectional Wave II models yielded similar results (see Figure 2.5).

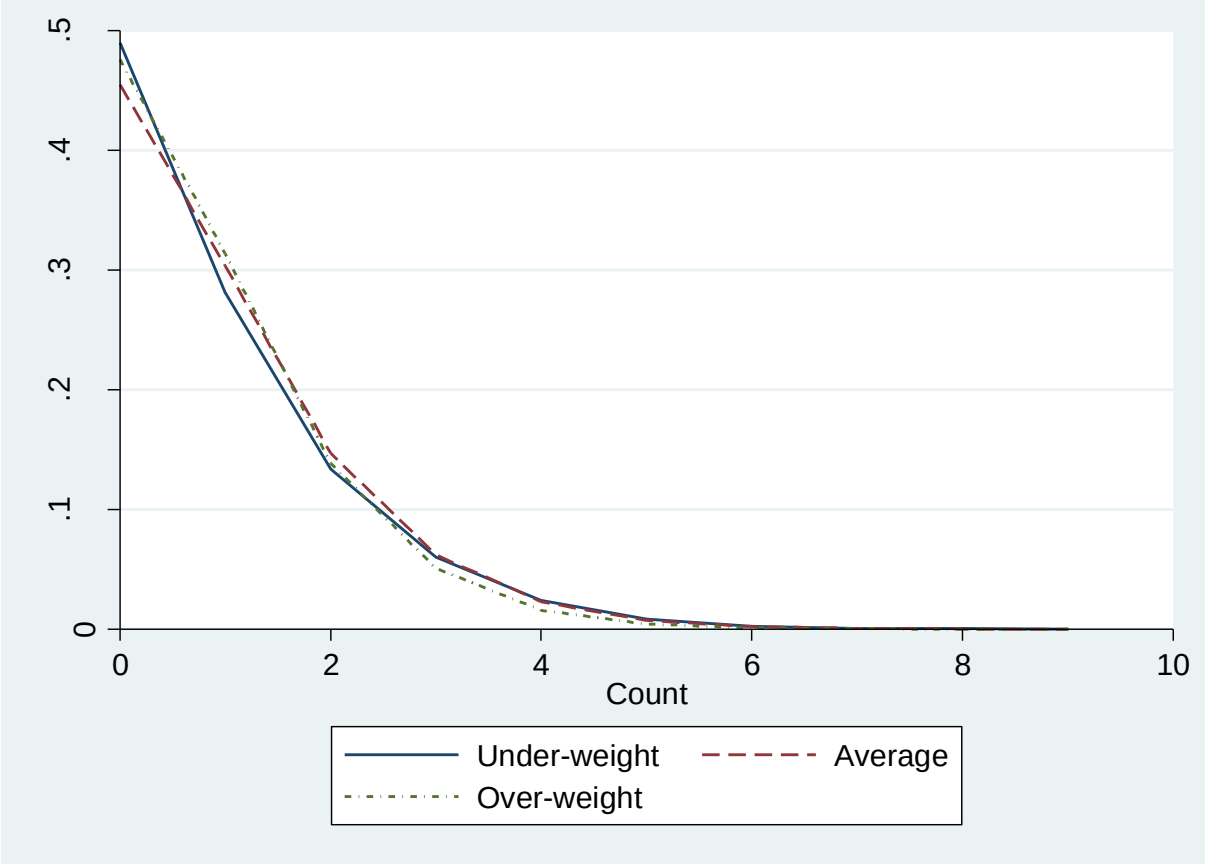

Figure 2.4. Predicted Probabilities of Late Developers' Number of Substances Tried by Body Weight: Cross-sectional Wave I

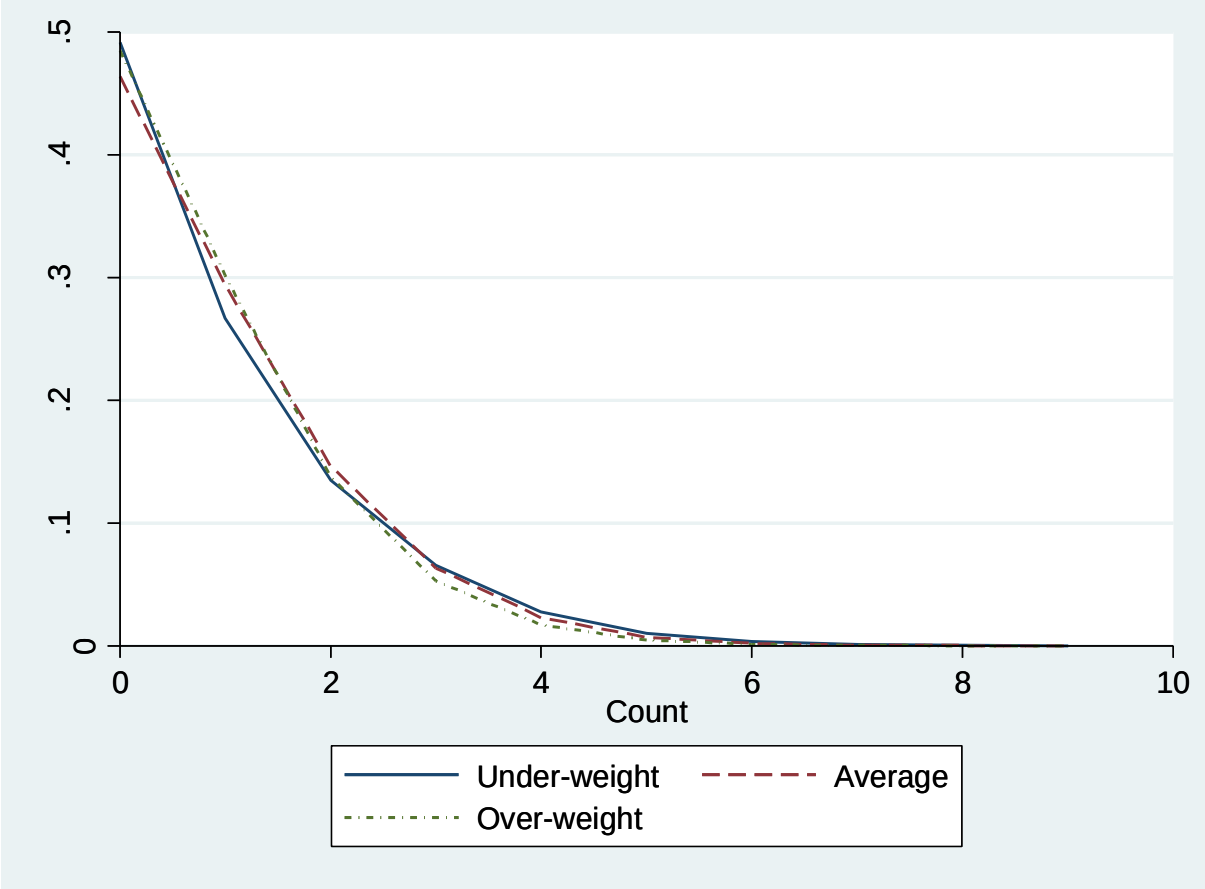

Figure 2.5. Predicted Probabilities of Late Developers' Number of Substances Tried by Body Weight: Cross-sectional Wave II

Longitudinal results. Table 2.5 presents longitudinal results from survey adjusted Poisson regression models predicting number of substances tried for the aggregated sample and then split by respondents' body weight. Results from the longitudinal models predicting Wave II (i.e., 1996) outcomes are shown in the left panel and results predicting Wave III outcomes (i.e., 2001-2002) are shown in the right panel.[16] The first column in each panel presents results for the aggregated sample of girls, with the next three columns presenting results split by body weight. Models controlled for prior substance use, standardized body

[16] Additional analyses predicting Wave III outcomes with Wave II predictors were conducted to examine potential time dependent longitudinal effects from mid-adolescence to late adolescence. These models provided statistically and substantively similar results and are therefore not reported here.

mass index, parental education, family structure, substance using friends, age, race/ethnicity, and nativity status.

As shown in the first column of the left panel in Table 2.5, substance use risk associated with early pubertal development for all girls remained significant at Wave II such that the incidence rate of substances tried was 12% higher for early versus on-time developers. Contrary to the cross-sectional findings (see Table 2.4), the risk of early development in the longitudinal Wave II model was significant only for under- and over-weight girls (see columns 2 through 4 in left panel, Table 2.5). So the substance use risk associated with early development for average weight girls at Wave II is entirely mediated or explained by their previous levels of substance use.[17] Thus, early developing average weight girls are at risk for substance use in early adolescence, with their initial levels of use then influencing their substance use in middle adolescence. Yet as shown in Table 2.5, the incidence rate of substances tried at Wave II was approximately 32% greater for under-weight girls who were early versus on-time developers at Wave I, and 24% higher for over-weight girls who were early developers.[18] Contrary to predictions, early developing over-weight girls tried more substances than average weight girls. Perhaps over-weight girls do not experience immediate risk associated the early developing body, but instead experience delayed risk if they choose to internalize the labels of deviance and hyper-sexuality often

[17] Indeed, models predicting substances tried at Wave II that did not control for baseline substance use indicated that early development was a significant risk factor for all categories of girls, and late development was protective for all girls combined as well as average weight girls. Similar models at Wave III showed no significant effects for early development, but late development was a significant protective factor for all girls combined and average weight girls.

[18] Given that tobacco use becomes more normative as girls develop in late adolescence, sensitivity analyses were conducted to examine whether the multivariate results predicting the number of substances tried were robust when tobacco use was excluded. Results from the cross-sectional and longitudinal models predicting number of non-tobacco substances tried were substantively similar to those reported in Tables 2.4 and 2.5. The only exceptions were that the early development coefficient for all girls in the longitudinal Wave II model was no longer statistically significant (IRR= 1.12; p=.14), and the late development coefficient for average weight girls in the longitudinal Wave II model was marginally significant (IRR=.84; p=.08).

attributed to early developing bodies. Internalization of such labels and embracement of such identities may be a way for early developing over-weight girls to seek/attain peer popularity and overcome their previously stigmatized status as overweight.

Given that the incidence rate ratios differed numerically for girls with different body weights, multiplicative interaction terms between body weight and pubertal development categories were used to statistically test for moderation. The multiplicative interaction term for early developing over-weight girls at Wave II attained marginal significance (exp(B)=1.22; p=.09) but the cross-model Wald coefficients indicated no significant difference between the early (F=1.55; p=.22) and late coefficients (F=.38; p=.69) across the body weight groups. By Wave III the longitudinal risk associated with early development was attenuated to non-significance. Thus, early development may have an initial impact on substance use among adolescent girls, influencing their initial path to substance use. These substance use histories, rather than early development, then largely determine substance use in late adolescence. Finally, although the multiplicative interaction term for early developing over-weight girls at Wave III attained marginal significance (exp(B)=1.14; p=.07) the cross-model Wald coefficients indicated no significant difference between the early (F=2.39; p=.10) and late coefficients (F=.15; p=.86). So again, although the split sample models indicated that the effect of pubertal development on number of substances tried differed slightly by body weight, this moderation was only marginally significant.

Table 2.5. Incidence Rate Ratios and Standard Errors from Survey Adjusted Poisson Regressions Predicting Longitudinal Lifetime Number of Substances Tried by Body Weight: Add Health Waves I-III[ab]

	Longitudinal Wave II[c]				Longitudinal Wave III[c]			
	All Girls	Under-weight	Average	Over-weight	All Girls	Under-weight	Average	Over-weight
Number of substances tried, Wave I	1.42***	1.42***	1.41***	1.51***	1.19***	1.21***	1.19***	1.18***
	(.02)	(.04)	(.02)	(.05)	(.01)	(.03)	(.02)	(.03)
Pubertal Development								
Early	1.12*	1.32†	1.07	1.24*	1.00	.88	.95	1.08
	(.06)	(.22)	(.07)	(.11)	(.03)	(.10)	(.04)	(.06)
Late	.96	1.03	.92	1.00	.99	1.01	.97	.98
	(.05)	(.10)	(.07)	(.19)	(.03)	(.06)	(.05)	(.09)
Social Location								
Z-BMI	.98				1.02			
	(.02)				(.02)			
Parental education	1.00	.99	1.01	1.00	1.03***	1.03*	1.03**	1.04***
	(.01)	(.02)	(.01)	(.02)	(.01)	(.01)	(.01)	(.01)
Non-married parent	1.08†	1.06	1.10†	1.06	.99	1.01	1.00	1.00
	(.05)	(.12)	(.06)	(.09)	(.03)	(.06)	(.04)	(.05)
Substance using friends	1.44***	1.38*	1.50***	1.26†	1.06†	1.02	1.06	1.08
	(.09)	(.19)	(.11)	(.15)	(.04)	(.07)	(.05)	(.09)
Age	.95***	.91*	.95*	.99	.95***	.94*	.96**	.95*
	(.02)	(.03)	(.02)	(.03)	(.01)	(.03)	(.01)	(.02)
Non-Latina black	.71***	.94	.70***	.68*	.75***	.82	.81*	.65***
	(.06)	(.35)	(.06)	(.12)	(.05)	(.12)	(.07)	(.08)
Mexican American	.97	.72	.96	1.08	.99	1.31	1.03	.94
	(.10)	(.16)	(.11)	(.21)	(.06)	(.23)	(.11)	(.11)
Non-Latina white	.99	1.02	1.02	.90	1.17***	1.31*	1.26***	1.03
	(.06)	(.15)	(.07)	(.15)	(.04)	(.14)	(.07)	(.08)
Immigrant	.83†	.84	.87	.68	.94	.95	.98	.76
	(.09)	(.16)	(.12)	(.35)	(.07)	(.16)	(.08)	(.16)

Table 2.5 cont.

	Longitudinal Wave II[c]				Longitudinal Wave III[c]			
	All Girls	Under-weight	Average	Over-weight	All Girls	Under-weight	Average	Over-weight
Constant	.15	.72	.02	-.41	.96***	.92*	.86***	1.02**
	(.28)	(.59)	(.33)	(.48)	(.19)	(.38)	(.23)	(.33)
N	3,304	658	2,104	662	3,733	646	1,998	669
F Statistic	98.85***	32.55***	74.43***	25.74***	53.88***	14.63***	44.47***	10.93***

Notes: [a]Incidence rate ratio = $\exp(\beta_j)$. [b]Standard errors for incidence rate ratios in parentheses. [c]Longitudinal Wave II models split by body weight at Wave I; longitudinal Wave III models split by body weight at Wave II.

† p<.10. * p<.05. ** p<.01. *** p<.001.

Predicting Heavy Substance Use

Tables 2.4 and 2.5 examined the association between early pubertal development and number of number of substances tried by body weight but did not capture effects for heavy substance use. To explore whether the patterns found in Tables 2.4 and 2.5 were consistent when predicting heavy substance use, Table 2.6 presents odds ratios from survey adjusted logistic regressions predicting heavy substance use for those girls who reported using substances. The first column in each panel presents results for the aggregated sample of girls, with the next three columns presenting results split by body weight. All models controlled for standardized body mass index, parental education, family structure, substance using friends, age, race/ethnicity, and nativity status; odds ratios for the control variables are not presented given that they are similar in pattern to those in Tables 2.4 and 2.5.

As shown in Table 2.6, the cross-sectional models for the aggregated sample of girls indicate that the odds of being a heavy user were significantly higher for early as compared to on-time developers but significantly lower for late developers at both Wave I and Wave II.[19] Similar to the split-sample models predicting counts of substance use, early development consistently predicted higher odds of heavy substance use by body weight category at Wave I. However, at Wave II early development did not significantly predict heavy substance use in any of the split-sample models. As shown in the split-sample models in Table 2.6, late development was protective only for average weight girls at Wave I, and under-weight girls at Wave II. By Wave III, however, all significant effects for pubertal development were attenuated to non-significance.

[19] Multiplicative interaction terms were not significant in any of the models shown in Table 2.6; Wald tests were as follows: for the cross-sectional Wave I models for early (F=.12; p=.89) and late (F=.02; p=.98); cross-sectional Wave II for early (F=.07; p=.93) and late (F=1.29; p=.28); longitudinal Wave II for early (F=1.38; p=.26) and late (F=.05; p=.95); and longitudinal Wave III for early (F=.04; p=.96) and late (F=.80; p=.45).

Table 2.6. Odds Ratios and Standard Errors from Survey Adjusted Logistic Regressions Predicting Heavy Substance Use by Body Weight: Add Health Waves I-III[abcd]

	Cross-sectional Wave I				Cross-sectional Wave II			
	All Girls	Under-weight	Average	Over-weight	All Girls	Under-weight	Average	Over-weight
Pubertal Development								
Early	1.79**	2.14†	1.74*	1.79†	1.43*	1.22	1.47	1.33
	(.35)	(.94)	(.42)	(.59)	(.25)	(.67)	(.35)	(.35)
Late	.60*	.60	.56*	.59	.62*	.40*	.78	.74
	(.14)	(.28)	(.15)	(.38)	(.15)	(.15)	(.24)	(.42)
	Longitudinal Wave II				Longitudinal Wave III			
	All Girls	Under-weight	Average	Over-weight	All Girls	Under-weight	Average	Over-weight
Pubertal Development								
Early	1.17	2.43	.95	.96	1.09	1.18	1.03	1.11
	(.22)	(1.31)	(.24)	(.29)	(.17)	(.61)	(.20)	(.31)
Late	.89	.76	.90	.88	.78	1.13	.69	.60
	(.26)	(.33)	(.37)	(.56)	(.15)	(.38)	(.18)	(.30)

Notes: [a]Odds ratio = $\exp(\beta_j)$. [b]Standard errors for odds ratios in parentheses. [c]Cross-sectional Wave I models split by body weight at Wave I; cross-sectional Wave II models split by body weight at Wave II. Longitudinal Wave II models split by body weight at Wave I; longitudinal Wave III models split by body weight at Wave II. [d]All models controlled for standardized body mass index, parental education, family structure, substance using friends, age, race/ethnicity, and nativity status; longitudinal models additionally control for heavy substance use at Wave II.
† p<.10. * p<.05. ** p<.01

In sum, early pubertal development consistently predicted greater number of substances tried and heavy substance use in early adolescence for under-weight, average, and over-weight adolescent girls. After controlling for initial substance use, however, the substance use risk associated with early development varied for girls of different body weights during mid-adolescence, and all effects disappeared by late adolescence. This pattern of findings is likely due to adolescent girls' movement into and out of normative and anormative body types during a developmental period characterized by sensitivity to social comparisons, which is discussed in the subsequent section.

DISCUSSION

The present study used longitudinal data from a national probability sample of U.S. adolescents to examine the effect of early pubertal development on adolescent girls' substance use, and the moderating role played by body weight. Results provided mixed support for the research hypotheses. Overall, adolescent girls who developed earlier than their peers tried more substances and were more likely to be heavy substance users, but these effects were attenuated by late adolescence. The substance use risk associated with early pubertal development was different for under-, average, and over-weight girls. However, differences across body weight categories were relatively small and often insignificant when tested with interaction terms and tests of cross-model coefficient equality. Enumerated below are the results for each of the research hypotheses.

H1: Early developing adolescent girls will have higher levels of substance use than on-time developers. Results provided strong support for Hypothesis 1 in that early developing adolescent girls had tried significantly more substances than on-time and late

developers (see left and right panels in Table 2.4). Cross-sectional findings were consistent for Waves I and II. Thus, findings support previous cross-sectional research that suggests early pubertal development is associated with greater substance use among adolescent girls (e.g., Deardorff et al. 2005; Stice et al. 2001; Wilson et al. 1994).

H2: The positive relationship between early pubertal development and substance use will attenuate over time. The longitudinal results (see Table 2.5) provided partial support for Hypothesis 2. Although early pubertal development at Wave I significantly predicted a higher number of substances tried at Wave II, this effect was attenuated by Wave III. These results support previous research outside of the United States that suggests the association between early pubertal development and adolescent girls' substance use does not persist into early adulthood (e.g., Aro and Taipale 1987; Dick et al. 2000; Magnusson 1988; Michaud et al. 2006). My findings indicate that the substance use risk associated with early pubertal development may be of import only during the short developmental time period (i.e., early to mid-adolescence) during which the changing body is particularly salient for identity formation and peer relationships. The developmental mismatch associated with early pubertal development is likely heightened in early adolescence—the period when girls must face the premature negotiation of reflected appraisals associated with their developing body and their pre-adolescent identity. After on-time and late developers have "caught-up" to early developers, the direct risk associated with early development may be attenuated so that its lasting effect is only through its impact on previous substance use or other mediating social contextual variables.

H3: Over-weight early developing adolescent girls will have lower levels of substance use than under- and average-weight early developing girls. Results provided

partial support for this exploratory hypothesis. The split-sample cross-sectional models (see Table 2.4) indicated that over-weight early developers had tried fewer substances than under- and average weight girls. However, statistical tests of moderation indicated no significant differences by body weight in the association between early pubertal development and number of substances tried. So although the developmental mismatch associated with early development may be less extreme for over-weight girls, the associated decrease in risk is relatively small.

H4: The moderating role of body weight described in H3 will attenuate over time. The longitudinal models (see Table 2.5) provided partial support for Hypothesis 4. At Wave II, over-weight early developers had tried fewer substances than under-weight girls but had also tried more substances than average weight girls. Further, by the last wave of data collection over-weight early developers had actually tried more substances than both under- and average weight girls. These theoretically unanticipated findings hint to the dynamic nature of the substance risk associated with early pubertal development for adolescent girls of different body weights. Two concepts that may help explain these findings are body weight satisfaction and peer popularity/acceptance. Early development may have an immediately risky effect on under- and average weight girls due to low levels of body weight satisfaction. Under- and average weight early developers may feel most threatened by changes in the pubertal body due to cultural beauty standards that value thin pre-pubescent body types (Bartky 2002; Frost 2005; Hesse-Biber 1996; Lovejoy 2001). As such, under- and average weight early developers may view budding breasts and hips as potential markers of bodies that are quickly diverging from current beauty ideals, so that early development signals a shift from a normative to anormative body. Conversely, over-weight early

developers, who may have been subject to peer teasing and exclusion prior to puberty, may come to embrace the newly sexualized body and gain increased peer popularity with boys and older peers who perceive them as developmentally advanced—such that early development signals a shift from an anormative to normative body relative to the peer group. These girls may in turn experience a delayed risk in substance use that results from an increased association with older (potentially more deviant) peers. Over-weight early developers may also experience a delayed risk in substance use if they begin to smoke cigarettes in an attempt to lose/manage body weight (Fulkerson and French 2003). As such, the pathways by which early pubertal development translates into a risk factor for substance use may vary for girls of different body weights due to differing interpretations and negotiations of the developing body and its normativity relative to peers.

H5: Early developing adolescent girls will be more likely to be heavy substance users than on-time developers. Results supported Hypothesis 5 in that early developers were more likely to be heavy substance users (see Table 2.6, top panels). Cross-sectional findings were consistent for Waves I and II. Thus, results indicate that the substance use risk associated with early pubertal development (see Hypothesis 1) is robust across type of substance use outcome.

H6: The positive relationship between early pubertal development and heavy substance use will attenuate over time. Hypothesis 6 was supported in that the association between early pubertal development and heavy substance use was attenuated in both of the longitudinal models (see Table 2.6, bottom panels). Overall, the lack of longitudinal findings for both the number of substances tried and heavy substance use models speaks to the temporality of the risk associated with pubertal development. The consequential effects of

pubertal development on risky health behaviors such as substance use are situated within complex social contexts that often change substantially during adolescence. The meaning and import of this transition has its largest impact during specific developmental periods for girls of different body weights. When girls experience this transition has an immediate effect on the developmental pathways that they then traverse.

H7: Over-weight early developing adolescent girls will be less likely to be heavy substance users than under- and average-weight early developing girls. Hypothesis 7 was partially supported in that over-weight early developers were less likely to be heavy substance users than under-weight, but not average weight early developers at Wave I. By Wave II, results were contrary to Hypothesis 7 in that over-weight early developers were less likely to be heavy substance users than average weight, but not under-weight girls.

H8: The moderating role of body weight described in H7 will attenuate over time. Results did not support Hypothesis 8 in that over-weight early developers were less likely to be heavy users than under-weight girls in both of the longitudinal models.

Overall, findings support stage termination theory in that early pubertal development predicted more substances tried as well as heavy substance use among adolescent girls. Early pubertal development has an immediate impact on adolescent girls' substance use, but these effects dissipate over time as prior levels of substance use then influence later substance use. However, contrary to predictions from identity control theory, over-weight girls were not consistently protected from the substance use risk associated with early development. Neither stage termination nor identity control theory predicted that the substance use risk associated with early development among over-weight girls would shift over time. So why does early

development shift from a protective, to risk, to protective factor for over-weight girls during the course of adolescence?

One explanation may be due to social mediator variables implicit in stage termination theory, which suggests that early developers are at greater risk because they attain a new developmental stage before they are capable of handling the roles and responsibilities that come with it. From this broad explanation, we could devise two hypotheses regarding why shifts in risk for girls of different body weights occur. For example, early developers may be more likely to engage in risky health behaviors because they come to see the developing body as distasteful in light of cultural beauty standards that value a thin pre-pubescent body type. Self-devaluation may then lead to risk behaviors in an attempt to cope with such negative self-appraisals. This explanation might indicate why under- and average weight early developers are at immediate risk of trying more substances—because they are likely to see the changes in their body as unwelcome changes that will magnify their deviance and distance them from same-age peers. So girls of certain weights may be more likely to experience dissatisfaction associated with the developing body and engage in risky health behaviors such as substance use in an attempt to negotiate and alleviate such dissatisfaction. Indeed, research suggests that many adolescent girls smoke cigarettes in an attempt to lose/manage body weight (Fulkerson and French 2003).

However, the substance use risk associated with early development may actually be due to girls' increased satisfaction with physical changes in the body that make them more popular with boys and older peers. Given their perceived physical maturity, early developers may be more likely to associate with older peers and boyfriends and thus have increased access to illicit substances. This explanation might explain why over-weight early developers

are at delayed risk of trying more substances—if over-weight early developers internalize the sexualized meanings associated with their newly developed body, they may recognize their new attractiveness to older boys and peers and change their peer group. More research is needed to investigate these and other potential mediators that might explain how social psychological and social contextual factors explain how the physical process of puberty influences the social behavior of substance use.

Study Limitations

Despite its strengths, this study has several limitations. First, as noted by other researchers, adolescent substance use prevalence in the Add Health data is lower than that found in other national studies (Substance Abuse and Mental Health 2002). Response bias issues may therefore underestimate the prevalence of adolescent girls' substance use and hence the potential risk associated with pubertal development and body weight. Additional research is needed to replicate these findings in samples with higher substance use prevalence. Second, I used measures of girls' actual body weight but did not incorporate measures of girls' perceived body weight or satisfaction with body weight. Future studies should investigate measures of perceived body weight or satisfaction with body weight. Third, more research is needed to explicate the social mechanisms by which the developmental transition of puberty—particularly when it occurs off-time—translates into risky health behaviors such as substance use. Fourth, potential racial/ethnic differences in the association between early pubertal development and adolescent girls' substance use should be investigated. Girls from different racial/ethnic backgrounds may not interpret and negotiate the meanings associated with the early developing body in similar ways. Indeed,

non-Latina white adolescent girls may be the most likely to accept and internalize cultural beauty standards of thinness (Casper and Offer 1990; Kelly et al. 2005; Neff et al. 1997; Neumark-Sztainer et al. 2002; Parnell et al. 1996). Thus, research should examine how race and ethnicity further moderate the relationship between early pubertal development and adolescent girls' substance use. Finally, I also only examined measures of substance use that aggregated tobacco, alcohol, marijuana, and other drugs. More research is need to address whether the main effects shown here, as well as the mediating mechanisms suggested in the previous paragraphs, are robust across different substances. It is likely that this will not be the case, given that adolescents use different substances in different social contexts (e.g., alcohol is used in largely social settings with other friends; cigarettes and marijuana may be more likely to used alone or with only a few friends) as well as for different goals (e.g., social lubricant versus weight control).

Policy Implications

Intervention programs are crucial in addressing the adolescent substance use problem in the United States. Indeed, although there is increasing availability of substance treatment programs specifically for adolescents, the majority of adolescents relapse within three months of discharge from treatment (Brown, Vik, and Creamer 1989; Catalano et al. 1991; Kennedy and Minami 1993). Given such high relapse rates after treatment, it is optimal to intervene and prevent substance use initiation and/or progression at an early age. Further, earlier age of onset is associated with longer substance use careers (Dennis et al. 2005), and adolescent substance use correlates with mental health, educational attainment, income, life satisfaction, and criminal behavior later in life (Georgiades and Boyle 2007; Lynskey and

Hall 2000; Lynskey et al. 2003; Mathers et al. 2006; McGee et al. 2000; Wells, Horwood, Ferguson 2004). Thus, intervention programs play a critical role in creating and maintaining a healthy population.

Given that gender gaps in substance use among adolescents are narrowing due to increased substance use among adolescent girls (Johnston, O'Malley, and Bachman 1998), and based on findings reported here, I advocate for gender-specific substance use intervention programs that occur during the short developmental time period during which early pubertal development is a significant risk factor for substance use. Programs that target 10 to 12 year old girls are uniquely situated to address the substance use risk associated with changes in the pubertal body. Creating programs specifically for young girls will allow practitioners to focus on the gender-specific context of risk associated with pubertal development and identity issues surrounding the body. For instance, prevention programs could prepare pre-adolescents for the bodily changes associated with puberty by combining educational components that emphasize how girls on different developmental paths (i.e., early, on-time, late developers) converge by mid- to late adolescence with self-esteem enhancement components designed to promote body acceptance (e.g., see O'Dea and Abraham 2000). Providing young girls with affirming messages about the pubertal body may help counteract negative messages from peers, adults, and media about developed bodies. Akin to recommendations by Amaro and colleagues (2001), gender-specific substance intervention programs should take an integrative ecological approach to not only improve young girls' body weight satisfaction and overall self-esteem, but also provide life-skills training, promote other healthy behaviors such as diet, strengthen family relationships, and address sexual/assault trauma that is increasingly prevalent among adolescent girls.

Continuing investigation into the social psychological and social contextual factors that explain substance use among adolescent girls will further our knowledge of key factors to address in substance use intervention programs.

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CHAPTER III

PUBERTAL DEVELOPMENT AND PATHWAYS OF RISK: SELF-APPRAISALS, SOCIAL RELATIONSHIPS, AND ADOLESCENT GIRLS' SUBSTANCE USE

Pubertal development, or the process of pubertal maturation in adolescence, is consequential for adolescent girls not only in terms of changing secondary sex characteristics but also in terms of changing exposure to risk. Indeed, pubertal development is well established as a risk factor for problem behaviors such as delinquency, externalizing behaviors, and substance use (e.g., Caspi et al. 1993; Chung, Park, and Lanza 2005; Ge et al. 2006; Haynie 2003). Scholars have perhaps paid most attention to the association between pubertal development and substance use in adolescence given its long-term impact on adult mental health, educational attainment, income, life satisfaction, and criminal behavior (Georgiades and Boyle 2007; Lynskey and Hall 2000; Lynskey et al. 2003; Mathers et al. 2006; McGee et al. 2000; Wells, Horwood, Ferguson 2004). Yet despite consistent evidence that adolescent girls who experience puberty earlier than their peers are at greater risk of substance use, it is still unclear how the social contexts in which adolescent girls develop may explain the pathway of risk between pubertal development and substance use. Of interest to sociologists and social psychologists then, is how do social factors, such as intrapersonal self-appraisals and interpersonal social relationships, mediate this relationship?

Social interaction theory maintains that self-appraisals, or how we view ourselves, are often based on reflected appraisals, or how we think others view us (Cooley 1902; Mead 1934). Understanding how intrapersonal self-appraisals are situated within a broader social context may help explain the effect of pubertal development on adolescent girls' substance

use. Contemporary beauty ideals in the United States are often based on a thin slender body type (Bartky 2002; Hesse-Biber 1996). Developing girls who have internalized these beauty ideals may experience weight dissatisfaction or low self-esteem as they gain fatty adipose tissue while other peers remain in pre-pubertal bodies (Frost 2005; Lovejoy 2001). Internalization of beauty ideals may therefore contribute to developing adolescent girls' devalued sense of self. Early pubertal developers may use substances such as tobacco or alcohol as a reaction to this devalued sense of self. As such, two key intrapersonal self-appraisals that might partially explain the link between pubertal development and adolescent girls' substance use are weight dissatisfaction and self-esteem.

Yet the potential mediating role of intrapersonal self-appraisals in the link between pubertal development and adolescent girls' substance use may be less powerful than the role played by factors related to a girls' relationships with potential agents of informal social control or socialization during adolescence (Catalano and Hawkins 1996; Elliott, Huizinga, and Ageton 1985; Farrington and Hawkins 1991; Hawkins and Weis 1985; Sampson and Laub 1993). If early pubertal developers feel physically and socially distanced from same-age peers they may seek older potentially more deviant peers to find an in-group with similar physical characteristics (Magnusson, Stattin, and Allen 1986; Stattin and Magnusson 1990). Interactions with deviant peers may influence girls' internalization of deviant norms and hence their risk of substance use. Simultaneously, association with deviant peers may co-occur with a detachment or weakening of social relationships with parents (Dishion, Nelson, and Bullock 2004). Early pubertal developers may actively seek or be granted greater autonomy from parents due to a newfound perceived maturity, which may in turn increase their access to/use of substances. Given that these interpersonal social relationships with

peers and parents are more theoretically proximal substance use risks than intrapersonal self-appraisals (i.e., the former deal with access to/internalization of deviant norms and the latter deal with general self-concept), we would expect more proximal mediators of the pubertal development-substance use link to "wash out" the effects of the less proximal mediators when accounted for in a sequential matter.

To more fully explore the potential mediating role of intrapersonal self-appraisals and interpersonal social relationships in the association between pubertal development and adolescent girls' substance use, this study addresses the following research questions: Does the link between pubertal development and adolescent girls' substance use disappear after controlling for intrapersonal self-appraisals and interpersonal social relationships? Further, does this disappearance occur sequentially with the introduction of intrapersonal self-appraisals, followed by interpersonal social relationships? Preliminary to testing four related hypotheses, the following sections discuss the theoretical and empirical roles of intrapersonal self-appraisals and interpersonal social relationships in the link between pubertal development and adolescent girls' substance use.

PUBERTAL DEVELOPMENT AND ADOLESCENT GIRLS' SUBSTANCE USE

The most common theoretical perspective applied to the study of pubertal development and adolescent girls' substance use is stage termination theory (Stattin and Magnusson 1990). According to stage termination theory, or the "early timing" hypothesis, girls who experience puberty earlier than their peers are at higher risk for problem behaviors such as substance use because they have attained physical maturity prior to social and psychological maturity (Caspi and Moffitt 1991; Petersen and Taylor 1980; Stattin and

Magnusson 1990). Because early developers are not socially or psychologically prepared to handle the roles and expectations associated with a developed adult-like body, they are more susceptible to environmental stressors such as peer pressure. Other theorists have labeled this potentially problematic stage of early pubertal development as one of "pseudomaturity," in which adolescents have physical adult status but not adult privileges such as independence (Galambos, Barker, and Tilton-Weaver 2003). Pseudomature adolescents may attempt to gain autonomy and overcome this "maturity gap" by engaging in problem behavior that mimics adult behavior such as drinking alcohol or smoking cigarettes (Moffitt 1993). It follows that early developers would be at higher risk for problem behaviors such as substance use given their forced early negotiation of this maturity gap.

A large body of empirical literature supports stage termination theory, providing consistent evidence that pubertal development, and early development compared to peers, is linked with substance use initiation, progression, and level of use. The association between pubertal development and adolescent girls' substance use has generally been consistent across different measurements of pubertal development, including measures of perceived physical development or pubertal maturity (e.g., Biehl, Natsuaki, and Ge 2007; Chung et al. 2005; Dick et al. 2001; Lanza and Collins 2002; Martin et al. 2002; Michaud, Suris, and Deppen 2006; Patton et al. 2004; Wiesner and Ittel 2002) and self-reported age at menarche (e.g., Aro and Taipale 1987; Caspi and Moffitt 1991; Caspi et al. 1993; Deardorff et al. 2005; Dick et al. 2000; Graber et al. 1997; Graber et al. 2004; Kaltiala-Heino et al. 2003; Magnusson et al. 1986; Simon et al. 2003; Stice, Presnell, and Bearman 2001; Tschann et al. 1994). Despite such consistent empirical support, several studies have found that the relationship between pubertal development and adolescent girls' substance use disappears by

late adolescence and early adulthood, whereby on-time and late developers "catch-up" with early developers (Aro and Taipale 1987; Magnusson et al. 1986; Tanner-Smith, 2009).

Although stage termination theory provides a useful theoretical framework to understand the main association between pubertal development and adolescent girls' substance use, it only partially theorizes this relationship within a broader social context. Indeed, an important call for research in this area is for study of potential mediators to explain the relationship between pubertal development and adolescent girls' substance use (Deardorff et al. 2005; Halpern, Kaestle, and Hallfors 2007). Two additional theoretical frameworks that may be useful in this regard are feminist social interaction theory and the social development model. Although both theoretical perspectives highlight the importance of the larger social context in which adolescent girls develop, the former emphasizes social psychological factors associated with interpreting the internalized meanings of a developing body, and the latter highlights the role of social relationships with potential agents of socialization.

Intrapersonal Self-appraisals

Key to the feminist social interaction perspective are the notions of the cult of thinness and tyranny of slenderness. The cult of thinness refers to the contemporary cultural context where capitalism and patriarchy have created a culture where many U.S. women are obsessed with obtaining and maintaining thinness (Hesse-Biber 1996). A related concept is the tyranny of slenderness, which posits that social interactional rituals in everyday life forbid U.S. women to be physically large; if women become physically large they are then stigmatized and experience shame due to their loss of status (Bartky 2002; Bordo 1993;

Chernin 1981). Although primarily used to explain women's body hatred, these theoretical perspectives are useful to apply to adolescent girls who may similarly be concerned with thinness and slenderness during a developmental transition when the physical body is most salient—puberty (Tobin-Richards, Boxer, and Petersen 1983).

Drawing on the notion of the cult of thinness/tyranny of slenderness, as well as Goffman's theory of stigma and shame (1963), feminist social interactionists theorize that the pubertal body is potentially shaming for adolescent girls given its incongruence with cultural beauty ideals of thinness (Frost 2005; Lee 1994; Martin 1996). Adolescent girls are a subgroup for whom body size is a particularly important component of social status with peers (Crosnoe, Mueller, and Frank 2008). If early developing girls interpret their self in terms of others' perceptions and judgments of their body, these intrapersonal self-appraisals will likely result in a devalued sense of self, reflected in weight dissatisfaction and low self-esteem. Wearing baggy clothes and having bad posture may be tangible forms of concealment that developing girls engage in (Frost 2005; Summers-Effler 2004), but substance use may represent a symbolic concealment of the self in response to such self-appraisals. The intrapersonal self-appraisals associated with pubertal development may therefore be driving the observed association between pubertal development and adolescent girls' substance use. So, after accounting for weight dissatisfaction and self-esteem, pubertal development may no longer be an important predictor of adolescent girls' substance use.

The potential mediating role of weight dissatisfaction and self-esteem in the link between pubertal development and adolescent girls' substance use has not been explored in empirical research to date. Rather, studies have focused on only one set of these variables at a time. For instance, prior research shows that pubertal development is positively associated

with low levels of weight satisfaction, body image, or body esteem among adolescent girls (e.g., Attie and Brooks-Gunn 1989; Blyth, Simmons, and Zakin 1985; Ge et al. 2001; Harter 1993; Richards et al. 1990; Simmons and Blyth 1987), and that early pubertal development is linked with disordered eating and weight control behaviors (Cauffman and Steinberg 1996; Killen et al. 1992; McCarthy 1990). Further, adolescent girls with low levels of weight satisfaction, body image, or body esteem are more likely to experiment with and use substances (e.g., Boles and Johnson 2001; Crow et al. 2006; French et al. 1994; Granillo, Jones-Rodriguez, and Carvajal 2005; Nieri et al. 2005; Ohring, Graber, and Brooks-Gunn 2002; Page, Scanlan, and Allen 1995; Palmqvist and Santavirta 2006; Wild et al. 2004). Similarly, more advanced pubertal development has been linked to lower self-esteem (Benjet and Hernandez-Guzman 2002; Simmons et al. 1979; Williams and Currie 2000), and lower self-esteem has been linked to higher levels of substance use among adolescent girls (Scheier et al. 2000; Wild et al. 2004). Although previous research has established that intrapersonal self-appraisals are key correlates of pubertal development and substance use, no studies have simultaneously accounted for them and the potential mediating role they have as proximal risk factors in the pubertal development-substance use connection.

Given this gap in the literature, and drawing on feminist social interaction theory, the first and second research hypotheses for this study are:

> *H1a: After controlling for weight dissatisfaction and self-esteem, there will be no association between body development and adolescent girls' alcohol use.*
>
> *H1b: After controlling for weight dissatisfaction and self-esteem, there will be no association between age at menarche and adolescent girls' alcohol use.*

H2a: After controlling for weight dissatisfaction and self-esteem, there will be no association between body development and adolescent girls' tobacco use.

H2b: After controlling for weight dissatisfaction and self-esteem, there will be no association between age at menarche and adolescent girls' tobacco use.

Interpersonal Social Relationships

In addition to intrapersonal self-appraisals, interpersonal social relationships may play a key role in explaining the link between pubertal development and adolescent girls' substance use. As a synthesis of social control, differential association, and social learning theories, the social development model argues that children and adolescents are socialized to prosocial and/or antisocial behavioral patterns from family, peers, school, religious, and other community organizations. The role of risk and protective factors are often developmentally specific, however, and family and peers are the key agents of socialization during childhood and adolescence (Catalano and Hawkins 1996; Elliott et al. 1985; Farrington and Hawkins 1991; Hawkins and Weis 1985; Sampson and Laub 1993). Relationships with, or attachments to, these agents of socialization play a large role in influencing problem behavior such as substance use among adolescents. Specifically, the probability of engaging in substance use depends on normative behaviors and values of the socializing agents to which the adolescent has social relationships. If early developers seek in-groups with similar levels of development, they are likely to associate with older potentially more deviant peer networks, including older boyfriends (Magnusson et al. 1986; Marin et al. 2000; Stattin and Magnusson 1990; Young and d'Arcy 2005). Older peer networks are more likely to have access to substances and therefore more likely to provide models for substance use and norms

accepting of substance use (Kandel 1980; Stice, Barrera, and Chassin 1998; Tschann et al. 1994). Similarly, if early developers seek autonomy from their parents (or if their parents grant them more autonomy) in search for social maturity as a response to their newfound physical maturity, adolescents' relationships with parents may weaken. Although obtaining autonomy from parents is a developmental process crucial to adolescence, what is detrimental and predictive of problem behavior is a "premature autonomy" (Dishion et al. 2004) characterized by an early detachment between the adolescent and parent(s), accompanied by a strengthened attachment to peers.

Previous research based on mixed gender and boy-only samples does indeed suggest that pubertal development is positively associated with autonomy from parents and deviant peer associations (Duncan et al. 1998; Steinberg 1987); and that autonomy and deviant peer association predict higher levels of substance use (Dishion et al. 2004; Duncan et al. 1998; Schulenberg et al. 1999; Wills and Cleary 1999). However, few studies to date have examined the potential mediating role of these social relationships in the link between pubertal development and substance use among girls.

Among a handful of studies examining the mediating role of social relationships in the association between pubertal development and adolescent girls' substance use, Wichstrom (2001) found that friends' problem behavior partially attenuated the association between early pubertal development and alcohol use among a sample of 7-12th grade adolescent girls in Norway. However, this study did not include any measures of social relationships with parents. There is only one study of which I am aware that has examined the potential mediating role of both family and peer social relationships in the pubertal development-substance use connection (Patton et al. 2004). Results from a large bi-national

sample of U.S. and Australian youth indicated that family and school relationships slightly reduced the impact of pubertal development on adolescent substance use, and substance-using friends largely reduced its impact (Patton et al. 2004). However, these results were based on a mixed gender sample, and therefore it is unclear whether similar processes occur for girls, which is questionable given that previous research suggests the impact of pubertal development on psychosocial outcomes often varies by gender (Dorn, Susman, and Ponirakis 2003; O'Dea and Abraham 2000; Tobin-Richards et al. 1983). Thus, there is initial evidence that interpersonal social relationships with family, and especially peers, may partially mediate the association between pubertal development and substance use among girls. Previous empirical research has not examined the sequential mediating effect of these interpersonal social relationships in tandem with intrapersonal self-appraisals, so more research is needed to simultaneously examine these two sets of potential mediators.

Drawing on social development model, the third and fourth research hypotheses for this study are:

H3a: After controlling for deviant peer association and autonomy from parents, there will be no association between body development and adolescent girls' alcohol use.

H3b: After controlling for deviant peer association and autonomy from parents, there will be no association between age at menarche and adolescent girls' alcohol use.

H4a: After controlling for deviant peer association and autonomy from parents, there will be no association between body development and adolescent girls' tobacco use.

H4b: After controlling for deviant peer association and autonomy from parents, there will be no association between age at menarche and adolescent girls' tobacco use.

In sum, despite robust evidence that pubertal development is positively associated with adolescent girls' substance use risk, several gaps in the literature remain as to the mediating role of intrapersonal self-appraisals and interpersonal social relationships. Namely, after accounting for more theoretically proximal substance use risk variables—weight dissatisfaction, self-esteem, autonomy from parents, and deviant peer association—does the association between pubertal development and adolescent girls' substance use disappear? Although previous studies have examined isolated sets of these variables, I am not aware of any that have collectively examined them. Research is needed to investigate how the pathway of risk between pubertal development and adolescent girls' substance use may be explained after sequentially accounting for less proximal intrapersonal self-appraisals and more proximal interpersonal social relationships.

STUDY SIGNIFICANCE

By contextualizing the ways in which pubertal development is linked to adolescent girls' substance use, this study makes several contributions to the literature. First, it simultaneously examines the role of four potential mediators in the association between pubertal development and substance use: weight dissatisfaction, self-esteem, autonomy from parents, and deviant peer association. By drawing attention to these intrapersonal self-appraisals and interpersonal social relationships I hope to further our understanding of the complex nexus between physical bodies and social contexts (as called for by Celio, Karnik, and Steiner 2006). By modeling the sequential pathway of risk associated with pubertal development, this study elucidates which proximal risk factors are key in explaining the substance use risk associated with adolescent girls' pubertal development. Methodologically,

this study contributes to the literature by distinguishing between two indicators of pubertal development: body development and age at menarche. Although both operationalizations of pubertal development have been used in prior studies, they have rarely been used together. Including both measures addresses whether measurement of pubertal development influences its association with adolescent girls' substance use, as well as the role of social contextual mediators in that association. Finally, this study uses nationally representative longitudinal data from a large sample of U.S. adolescent girls.

METHODS

Sample

This study analyzed restricted-use data from the first three waves of the National Longitudinal Study of Adolescent Health (Add Health) (http://www.cpc.unc.edu/addhealth), which used a multistage, stratified, and school-based sampling design. The clustered sampling design used schools as primary sampling units (PSUs) and region of the country (Northeast, Midwest, South, and West) as a stratification variable. A total of 132 PSUs (80 high schools and 52 "feeder" middle schools) were selected from a sample frame of 26,666 schools, and probability weights were used to adjust for non-response and unequal probabilities of selection.

The Add Health data are representative of 7-12th graders attending school in the United States during the 1994-1995 school year. At Wave I (1994-1995), in-school questionnaire data were collected on 90,118 students and 20,745 students completed in-home surveys. At Wave II (1996), in-home surveys were collected on 14,738 students sampled

from Wave I participants and non-participants. At Wave III (2001), in-home surveys were collected on over 15,000 students sampled from Wave I participants and non-participants (see Udry, Bearman, and Harris 2006 for more detailed sampling design information).

The analytic sample included black, Mexican-American, and white girls age 10-15 at Wave I with non-missing sampling weights.[20] These girls represent the three largest racial and ethnic groups in the Add Health data in the developmental stage of early adolescence, a stage characterized by the most dramatic changes in the female pubertal body. There were a total of 4,798 black, Mexican-American, and white girls age 10-15 with non-missing sampling weights. Values coded as "refused", "don't know", or "missing" were treated as missing and cases were deleted listwise. The amount of missing data in the variables ranged from less than 1% for geographic variables to 14% for age at menarche; on average 10% of self-reported data were missing and 12% of parent-reported data were missing.

Measures

The two dependent measures were self-reported alcohol use and tobacco use. Three items were used to calculate an average scale score of alcohol use: frequency of alcohol consumption in the past year; frequency of drinking five or more drinks in one setting in the past year; and frequency of drinking to drunkenness in the past year (1=none; 7=every day or almost every day). These three measures were averaged to create a scale of alcohol use that ranged from 1-7. Tobacco use was measured as the number of cigarettes smoked per day in

[20] Approximately 8-10% of cases sampled at each wave had missing sampling weights. These respondents were selected outside of the sampling frame at Wave I to be included in the twin/sibling sample, and were sampled to ensure large enough sample sizes to obtain genetically related respondents. Because these cases were sampled outside of the sampling frame they did not receive sampling weights and were excluded from all analyses (see Chantala 2006).

the past month where 0=abstinence. The alcohol use and tobacco use items were measured at Waves I, II, and III.

Pubertal development came from items measuring body development and age at menarche. Body development was measured by factor scores from a confirmatory maximum likelihood factor analysis based on three self-reported items: breast development since grade school, body curve development since grade school, and physical development relative to peers (1=less developed; 5=more developed). These three items of body development had high internal consistency (Cronbach's α =.70). Age at menarche was an interval variable measuring girls' self-reported age at first menstruation. The body development and age at menarche items were measured at Wave I.

Weight dissatisfaction and self-esteem were the intrapersonal self-appraisals of interest. Weight dissatisfaction was measured by factor scores from a confirmatory maximum likelihood factor analysis based on three items: whether a respondent was trying to lose weight (1=yes); perception of body weight (1=very underweight; 5=very overweight); and number of weight control behaviors in the past week—dieting, exercising, purging, diet pills, laxatives, other (0-6). The three weight dissatisfaction measures had high internal consistency (Cronbach's α =.72). Self-esteem was measured with factor scores from a confirmatory maximum likelihood factor analysis based on six items from Rosenberg's (1965) self-esteem inventory. These six items (1=strongly disagree; 5=strongly agree) asked respondents whether they have good qualities, have a lot to be proud of, like themselves as they are, always do things right, feel socially accepted, and feel loved and wanted. The six self-esteem measures had high internal consistency (Cronbach's α =.85). The weight dissatisfaction and self-esteem items were measured at Wave I.

Interpersonal social relationships were captured with measures of autonomy from parents and association with substance-using friends. Autonomy from parents was measured with factor scores from a confirmatory maximum likelihood factor analysis based on seven dichotomous (yes/no) items of perceived autonomy that asked respondents: "Do your parents let you make your own decisions about…the time you must be home on weekend nights, the people you hang around with, what you wear, how much television you watch, which television programs you watch, and what time you go to bed on weeknights, and what you eat". These seven items had moderate internal consistency (Cronbach's α =.57). Substance-using friends was a summative scale from three items asking respondents whether any of their three closest friends used tobacco, alcohol, or marijuana; the scale ranged from 0 (no substance-using friends) to 3 (at least three of their closest friends use tobacco, alcohol, and marijuana). The autonomy and substance-using friends items were measured at Wave I.

Control variables included age, standardized body mass index (Z-BMI), race/ethnicity, family structure, urbanicity, and region. These control variables were included because previous research (Arim et al. 2007; Blake et al. 2001; Flewelling and Bauman 1990; Goodman and Huang 2002; Hawkins, Catalano, and Miller 1992; Kaplowitz et al. 2001) has established their connection to pubertal development and adolescent substance use. Age was a continuous measure of respondents' self-reported age in years. The Z-BMI score was an anthropometric indicator of adolescent girls' body mass index relative to other U.S. girls of the same age. Z-BMI scores were calculated as the standard deviation score of a respondent's body mass index compared to the median value of body mass index for girls of the same age based on the 2000 CDC growth charts (see Quantitative Techniques 2003 for more information).

Race/ethnicity was recoded into three indicator variables representing non-Latina black (1=yes), Mexican-American (1=yes), and non-Latina white (1=yes). Family structure was measured by parental reports of marital status and recoded into a dummy variable so that 1=non-married parent household and 0=married. Urbanicity was measured with three dummy variables (1=yes) for urban, suburban, and rural area of residence. Finally, region was measured with four dummy variables (1=yes) for West, Midwest, Northeast, and South. All control variables were measured at Wave I.

Analytic Strategies

Given that the alcohol use outcome ranged from 1-7, models predicting alcohol use were estimated using Ordinary Least Squares (OLS) regression. OLS assumes independence and normality of residuals, homoskedasticity, and relationship linearity (Allison 2002). Diagnostics indicated that none of the models grossly violated these assumptions. Results from ordinal logistic regression models provided substantively similar results, and so results from OLS models are presented for the ease of interpretation.

The tobacco use outcome was a non-negative count outcome, and therefore violated OLS assumptions of normality and homoskedasticity of residuals (Long 1997). Negative binomial regression models were used for the tobacco use outcome. Negative binomial regression is an appropriate multivariate statistical technique when modeling non-negative and overdispersed (i.e., variance greater than the mean) count outcomes with strong positive skew. The negative binomial regression model can be expressed mathematically as

$$\lambda_i = \exp(\mathbf{X}_i * \boldsymbol{\beta}) * \exp(\varepsilon_1)$$

where $\mathbf{X}_i$ is the matrix of independent variables on individuals i and $\boldsymbol{\beta}$ is a vector of regression coefficients relating independent variables to the mean number of cigarettes smoked rate. The error term $\exp(\varepsilon_1)$ is distributed as $\Gamma(1, \alpha)$, where α is the dispersion parameter that scales the relationship between the mean and variance of the outcome. The NB1 (Cameron and Trivedi 1998) formulation of the negative binomial model was used to specify a linear mean-variance relationship.[21] The negative binomial regression coefficients can be interpreted such that $\exp[\beta_j]$ is the incidence rate ratio or predicted rate ratio (i.e., number of events per time) for a one-unit increase in predictor j, holding other variables in the model constant. Incidence rates can also be interpreted so that $[(\exp[\beta_j]-1)*100]$ is the predicted percentage change in the number of cigarettes smoked incidence rate for a unit increase in a predictor j, net of the other variables in the model.[22]

The clustered sample design and correlated error structure of the Add Health data required statistical corrections for design effects and unequal probability of selection (Chantala and Tabor 1999). A Taylor Series variance estimation method was used to estimate standard errors and insured consistency in the probability of Type I errors. For the cross-sectional Wave I and Wave II models and longitudinal models, probability weights (variables GSWGT1, GSWGT2, GSWGT3_2, respectively), strata (variable REGION), and primary sampling units (variable PSUSCID) were specified in Stata 9.2 for use with the survey-based OLS and negative binomial regression procedures.

[21] The NB1 model formulation was chosen over the NB2 specification given that the former exhibited the larger log-likelihood values in negative binomial models unadjusted for the complex survey design.

[22] Given that the Add Health data include multiple measures of the key dependent and independent variables used here, Structural Equation Modeling (SEM) may have also been an appropriate modeling strategy. SEM models were estimated using polychoric correlation matrices, but the data did not fit the model well when attempting to predict alcohol and tobacco use simultaneously, and the models did not converge after 1,000 iterations. Further, given the theoretically implied correlated errors between intrapersonal self-appraisals and interpersonal social relationships, the specified SEM model including all variables did not meet necessary and sufficient conditions for identification.

RESULTS

Descriptive Statistics

Table 3.1 shows the survey adjusted means, standard errors, and ranges of substance use, pubertal development, intrapersonal self-appraisal, interpersonal social relationships, and social location variables by wave of data collection. As shown in the top panel of Table 3.1, at Wave I girls reported drinking alcohol on average only a few times per year, and smoking just over one cigarette per day. Most girls (77%) did not report smoking any cigarettes, however. Among those who did smoke, the average number of cigarettes smoked per day was 5.65. In early adolescence, most girls drank only a few times a year and did not smoke cigarettes.

At Wave I, the average body development score was .08 and the average age at menarche was 11.92. This sample's average age at menarche was slightly younger than national estimates of age at first menstruation, which are between 12.43 and 12.76 (Herman-Giddens, Kaplowitz, and Wasserman 2004). The average factor score for weight dissatisfaction was .02 and .02 for self-esteem. For the interpersonal social relationship variables, the average autonomy score was .02, and most girls reported that at least one of their three closest friends used tobacco, alcohol, or marijuana. For social location variables, the average age of respondents was 13.78, and standardized body mass index (Z-BMI) scores averaged .58. Approximately 29% of girls came from non-married parent households. The majority (60.52%) of girls lived in suburban areas, and resided in the Midwest (37.05%) or the South (36.33%).

As shown in the middle panel of Table 3.1, by Wave II girls reported using more

alcohol and tobacco. The average level of alcohol use increased slightly to 1.70, and the mean number of cigarettes smoked per day increased to 2.09. Again, most girls (70%) reported smoking no cigarettes; among those who did, the average number of cigarettes smoked per day was 6.41. So as would be expected, by Wave II girls were drinking alcohol and smoking cigarettes more frequently than at Wave I.

Table 3.1. Means, Standard Errors, and Ranges of Substance Use, Pubertal Development, Self-appraisal, Social Relationship, and Social Location Variables by Wave of Collection[a]

	Mean	Standard Error	Range
Wave I			
Substance Use			
Alcohol use	1.64	.03	1-7
Tobacco use	1.57	.14	0-60
Pubertal Development			
Body development	.08	.02	-2.18-1.67
Age at menarche	11.92	.04	7-15
Intrapersonal Self-appraisals			
Weight dissatisfaction	.02	.02	-1.42-2.18
Self-esteem	.02	.03	-1.34-4.62
Interpersonal Social Relationships			
Autonomy	.02	.03	-2.61-.87
Substance-using friends	1.27	.04	0-3
Social Location Variables			
Age	13.78	.08	10-15
Z-BMI	.58	.02	-4.68-2.89
Non-Latina black (1=yes)	15.98%	.02	0-1
Mexican American (1=yes)	6.29%	.01	0-1
Non-Latina white (1=yes)	78.50%	.03	0-1
Non-married parent (1=yes)	29.31%	.02	0-1
Urban (1=yes)	22.95%	.04	0-1
Suburban (1=yes)	60.52%	.05	0-1
Rural (1=yes)	16.53%	.04	0-1
West (1=yes)	14.44%	.01	0-1
Midwest (1=yes)	37.05%	.03	0-1
Northeast (1=yes)	12.18%	.01	0-1
South (1=yes)	36.33%	.02	0-1
Wave II			
Substance Use			
Alcohol use	1.70	.04	1-7
Tobacco use	2.09	.16	0-60
Wave III			
Substance Use			
Alcohol use	2.35	.05	1-7
Tobacco use	3.61	.23	0-90

Notes. [a]Means and standard errors adjusted for complex survey design.

Finally, as shown in the last panel of Table 3.1, girls used the most alcohol and tobacco at Wave III. This is not surprising given that some respondents were over age 18 and would have legal access to cigarettes (and in some cases, alcohol). Specifically, the average level of alcohol use increased to 2.35, and the mean number of cigarettes smoked per day (for all girls) increased to 3.61. Most girls (69%) abstained from tobacco, but when examining only those girls who reported smoking cigarettes, the average increased to 10.49. So by Wave III girls were drinking more frequently (at least once a month) and smoking more frequently as well.

Prior to presenting multivariate models predicting adolescent girls' alcohol and tobacco use, I now briefly describe bivariate correlations between the key variables of interest. As shown in Table 3.2, alcohol use was positively correlated with tobacco use (e.g., .44 at Wave I). Alcohol use was also positively associated with body development, weight dissatisfaction, self-esteem, autonomy, and substance-using friends (e.g., .15, .06, .21, .14, and .54, respectively at Wave I); alcohol use was not significantly correlated with age at menarche. Tobacco use was positively correlated with body development, self-esteem, autonomy, and substance-using friends; and negatively correlated with age at menarche. Body development was associated with younger age at menarche, more weight dissatisfaction, greater autonomy, and more substance-using friends. Age at menarche, however, was not significantly associated with self-esteem, autonomy, nor substance-using friends, and was negatively correlated with weight dissatisfaction. Finally, the intrapersonal self-appraisal variables were positively correlated with each other as were the interpersonal social relationship variables.

Table 3.2. Zero-order Polychoric, Tetrachoric, and Polyserial Correlation Matrix of Substance Use, Pubertal Development, Self-appraisal, and Social Relationship Variables: Add Health Waves I-III[a]

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Alcohol use, Wave I	1.00											
2. Alcohol use, Wave II	.53	1.00										
3. Alcohol use, Wave III	.17	.24	1.00									
4. Tobacco use, Wave I	.44	.28	.06	1.00								
5. Tobacco use, Wave II	.39	.35	.07	.64	1.00							
6. Tobacco use, Wave III	.22	.23	.19	.35	.44	1.00						
7. Body development, Wave I	.15	.13	.05	.10	.10	.09	1.00					
8. Age at menarche, Wave I	.00	.02	.13	-.04	-.06	-.05	-.12	1.00				
9. Weight dissatisfaction, Wave I	.06	.03	.00	.01	.01	.08	.23	-.11	1.00			
10. Self-esteem, Wave I	.21	.14	.04	.15	.14	.12	.04	.02	.18	1.00		
11. Autonomy, Wave I	.14	.12	.10	.07	.04	.06	.10	.02	.00	.03	1.00	
12. Substance-using friends, Wave I	.54	.44	.14	.43	.41	.31	.18	-.03	.05	.26	.14	1.00

Note: [a]All correlations $\geq$ |.04| are significant at $p<.05$ or less.

Pubertal Development and Alcohol Use

Table 3.3 presents results from hierarchically nested survey adjusted OLS regression models predicting alcohol use. All models control for age, Z-BMI, race/ethnicity, family structure, urbanicity, and region. The left panel presents results from the Wave I models (i.e., 1994-1995); Model 1 includes pubertal development, Model 2 adds the self-appraisal variables, and Model 3 adds the more proximal social relationship variables. The middle panel in Table 3.3 presents similar nested models (Models 4 through 6) predicting alcohol use at Wave II (1996), and also controls for Wave I alcohol use. Finally, the right panel in Table 3.3 presents nested models (Models 7 through 9) predicting alcohol use at Wave III (2001) and controls for Wave I alcohol use.

As shown in Model 1 in Table 3.3, at Wave I body development was positively associated with alcohol use after controlling for social location variables, so that each additional unit increase in the body development factor scores was associated with a .16 increase in the alcohol use scale. This model, which excludes self-appraisals and social relationships, accounted for approximately 7% of the variance in the alcohol use outcome. Model 2 adds the less proximal intrapersonal self-appraisal risk factors, and provided a minimal increase in predictive power such that the model accounted for approximately 10% of the variance in the alcohol use outcome. Adding self-appraisals to the model slightly attenuated the effect of body development on alcohol use, reducing the size of the coefficient by approximately 6%, but body development was still a significant predictor of alcohol use. Thus a small portion of the effect of body development on alcohol use may be due to self-appraisals, but these self-appraisals also have independent and positive effects on alcohol use.

Table 3.3. Coefficients and Standard Errors from Survey Adjusted OLS Regressions Predicting Alcohol Use: Add Health Waves I-III[abc]

	Wave I			Wave II			Wave III		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Body development	.16***	.15***	.07**	.11***	.10***	.08**	.05	.04	.03
	(.03)	(.03)	(.02)	(.03)	(.03)	(.03)	(.04)	(.04)	(.04)
Age at menarche	-.02	-.02	.01	.01	.02	.02	.11***	.10***	.10***
	(.02)	(.02)	(.02)	(.02)	(.02)	(.02)	(.02)	(.02)	(.03)
Intrapersonal Self-appraisals									
Weight dissatisfaction		.07*	.07**		.06†	.07*		.07†	.08†
		(.03)	(.03)		(.03)	(.03)		(.04)	(.04)
Self-esteem		.18***	.07**		.08**	.05†		-.11*	-.11*
		(.03)	(.02)		(.03)	(.03)		(.04)	(.05)
Interpersonal Social Relationships									
Autonomy			.03			.08*			.15**
			(.05)			(.03)			(.05)
Substance-using friends			.40***			.15***			.01
			(.03)			(.02)			(.03)
Constant	-.40	-.10	.23	.72†	.88*	1.17**	.77	.69	1.13†
	(.37)	(.35)	(.37)	(.37)	(.38)	(.41)	(.58)	(.58)	(.61)
N	3,509	3,502	3,481	2,997	2,991	2,972	2,849	2,844	2,826
R^2	.07	.10	.28	.32	.33	.34	.11	.12	.13
F Statistic	11.13***	11.50***	26.12***	34.09***	33.52***	44.14***	21.11***	19.41***	17.58***

Notes: [a]Standard errors in parentheses. [b]All models control for age, Z-BMI, race/ethnicity, family structure, urbanicity, and region. [c]Wave II and Wave III models control for alcohol use at Wave I.
† p<.10. * p<.05. ** p<.01. *** p<.001.

If the interpersonal social relationship risk factors are more theoretically proximal to substance use, we would expect that upon entering them into the regression models that the effect of pubertal development on alcohol use would be completely attenuated, as would the effects of the intrapersonal self-appraisals. But as shown in Model 3 in Table 3.3, although adding the more proximal social relationship variables to the model attenuated the body development coefficient by 53%, they did not reduce the effect to non-significance. And although Model 3 had greater predictive power than the first two models ($R^2 = .28$), weight dissatisfaction and self-esteem remained significant predictors of alcohol use at Wave I. Models 1 through 3 also indicate that age at menarche was not a significant predictor of alcohol use at Wave I. Thus in the cross-sectional models at Wave I, net of social location variables, alcohol use was high for girls with high levels of body development, weight dissatisfaction, self-esteem, and more substance-using friends. Although interpersonal social relationships, and specifically substance-using friends, partially attenuated the effect of body development on alcohol use, they did not entirely mediate this relationship for girls in early adolescence.

At Wave II, a similar pattern emerges. As shown in Model 4, body development was positively associated with higher alcohol use net of social location variables. Entering the intrapersonal self-appraisals into the model again only reduced the size of the body development coefficient by approximately 9%, and did little to increase the predictive power of the model (see Model 5). Similar to findings from Wave I, higher levels of self-esteem were associated with higher levels of alcohol use at Wave II. After including the interpersonal social relationship variables (see Model 6), the coefficient for body development was again attenuated an additional 20%, but remained statistically significant.

Further, after controlling for the more theoretically proximal interpersonal social relationship variables, the effect of self-esteem on alcohol use was also attenuated to marginal significance whereas weight dissatisfaction emerged as a significant predictor of alcohol use. Similar to results from Wave I, age at menarche was not a significant predictor of alcohol use at Wave II. Thus after controlling for social location variables and prior alcohol use, alcohol use at Wave II was high for girls with high weight dissatisfaction, more autonomy from parents, and more substance-using friends.

At Wave III a different pattern emerged such that body development did not significantly predict alcohol use, but later age at menarche predicted higher levels of alcohol use. Including the two intrapersonal self-appraisals attenuated the coefficient for age at menarche by 9%, but it remained statistically significant. Finally, and as shown in Model 9, adding the two interpersonal social relationship variables had no influence on the age at menarche coefficient and little influence on the predictive power of the model. Thus by Wave III, after controlling for social location variables as well as prior alcohol use, girls with later ages at menarche, lower self-esteem, and more autonomy from parents had higher levels of predicted alcohol use.

Overall, results in Table 3.3 indicate that intrapersonal self-appraisals, and to a greater extent, interpersonal social relationships, explain only a part of the association between pubertal development and adolescent girls' alcohol use. So these variables appear to be important correlates of adolescent girls' alcohol use, rather than mediators of the pathways of risk associated with pubertal development. These models also provide evidence that the alcohol use risk associated with body development disappears when girls age and progress beyond puberty. Conversely, the alcohol use risk associated with older age at menarche may

have a delayed effect, emerging as girls enter late adolescence. Possible theoretical explanations for these findings will be explored in the Discussion section.

Pubertal Development and Tobacco Use

Table 3.4 presents results from hierarchically nested survey adjusted negative binomial regression models predicting tobacco use. All models control for age, Z-BMI, race/ethnicity, family structure, urbanicity, and region. The left panel presents results from the Wave I models (i.e., 1994-1995); Model 1 includes pubertal development, Model 2 adds the two intrapersonal self-appraisal variables, and Model 3 adds the two interpersonal social relationship variables. The middle panel in Table 3.3 presents similar nested models (Models 4 through 6) predicting tobacco use at Wave II (1996), and also controls for Wave I tobacco use. Finally, the right panel in Table 3.3 presents nested models (Models 7 through 9) predicting tobacco use at Wave III (2001) and controls for Wave I tobacco use.

As shown in Model 1 in Table 3.4, at Wave I body development was positively associated with tobacco use after controlling for social location variables, where each additional unit increase in the body development factor score was associated with a predicted 21% increase in the tobacco use incidence rate. Simultaneously, earlier age at menarche was associated with higher levels of tobacco use such that each additional year associated with age at menarche predicted an 11% decrease in the tobacco use incidence rate. Adding the two intrapersonal self-appraisal variables had little impact on the body development and age at menarche incidence rate ratios, such that both remained statistically significant (see Model 2). This was in spite of the positive and significant impact of self-esteem on tobacco use. Consistent with Hypothesis 3a, introducing the more proximal interpersonal social

relationship variables into the model attenuated the body development incidence rate ratio to non-significance. The attenuation of the body development effect appears to be largely due to substance-using friends, so that net of the other variables in the model each additional substance-using friend a girl reported was associated with a predicted 128% increase in the tobacco use incidence rate. Including the social relationship variables in Model 3 did not attenuate the age at menarche effect, however, which actually increased by approximately 4% upon the addition of autonomy and substance-using friends variables. So at Wave I, net of social location controls, girls with younger age at menarche, high self-esteem, and more substance-using friends had high predicted levels of tobacco use.

Results for Wave II were generally similar in that the body development incidence rate ratio remained significant after the introduction of intrapersonal self-appraisals, but was completely attenuated after accounting for social relationships. Again this effect was largely driven by substance-using friends. Unlike the results from Wave I, however, age at menarche did not have a significant impact on tobacco use at Wave II. So by Wave II, net of social location controls, the only important predictor of tobacco use was substance-using friends.

Table 3.4. Incidence Rate Ratios and Standard Errors from Survey Adjusted Negative Binomial Regressions Predicting Tobacco Use: Add Health Waves I-III[abcd]

	Wave I			Wave II			Wave III		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Body development	1.21**	1.20**	1.04	1.17*	1.16*	1.07	1.26***	1.25***	1.19**
	(.08)	(.08)	(.06)	(.07)	(.08)	(.08)	(.07)	(.07)	(.07)
Age at menarche	.89**	.89*	.93*	1.00	.99	1.00	.94	.95	.95
	(.04)	(.04)	(.03)	(.03)	(.03)	(.03)	(.04)	(.04)	(.03)
Intrapersonal Self-appraisals									
Weight dissatisfaction		.99	1.01		1.09	1.01		1.17*	1.14*
		(.06)	(.06)		(.08)	(.06)		(.07)	(.07)
Self-esteem		1.42***	1.17***		1.17**	1.02		1.16**	1.08
		(.06)	(.05)		(.06)	(.06)		(.06)	(.05)
Interpersonal Social Relationships									
Autonomy			1.07			.99			1.12†
			(.07)			(.05)			(.07)
Substance-using friends			2.28***			1.70***			1.33**
			(.12)			(.07)			(.06)
Constant	-2.76**	-2.37***	-2.60***	-.39	-.01	.16	3.52***	3.84***	4.26***
	(.89)	(.59)	(.72)	(.68)	(.70)	(.63)	(.70)	(.70)	(.71)
N	3,507	3,500	3,478	3,002	2,997	2,977	2,870	2,865	2,847
F Statistic	14.82***	20.78***	34.40***	29.29***	26.74***	50.48***	17.27***	15.78***	18.56***

Notes: [a]Incidence rate ratio = $\exp(\beta_j)$. [b]Standard errors for incidence rate ratios in parentheses. [c]All models control for age, Z-BMI, race/ethnicity, family structure, urbanicity, and region. [d]Wave II and Wave III models control for tobacco use at Wave I.
† p<.10. * p<.05. ** p<.01. *** p<.001.

Finally, at Wave III, body development remained an important predictor of tobacco use, even after controlling for self-appraisals and social relationships. Specifically, introducing intrapersonal self-appraisals reduced the body development effect by only 1%, despite the fact that both weight dissatisfaction and self-esteem had significant positive relationships with tobacco use. Controlling for social relationships reduced the body development effect an additional 5%, but body development remained a significant predictor of tobacco use. Similar to Wave II models, at Wave III age at menarche did not significantly predict tobacco use. So net of social location controls, higher levels of body development, weight dissatisfaction, and more substance-using friends predicted higher levels of tobacco use at Wave III.

In sum, findings for tobacco were somewhat similar to those for alcohol in suggesting that although intrapersonal self-appraisals and interpersonal social relationships are important correlates of adolescent girls' substance use, they inconsistently mediate the relationship between pubertal development and substance use. Social relationships with deviant peers have the greatest explanatory power as mediators, particularly given their effect on tobacco use outcomes (see Models 3 and 6 in Table 3.4), yet their meditational effect did not persist in late adolescence. Further, results in Table 3.4 indicated that contrary to the findings for alcohol use (see Models 7 through 9 in Table 3.3), younger age at menarche predicted higher incidence rates of tobacco use in early adolescence, but did not significantly influence tobacco use in middle and late adolescence. These findings suggest that the pathways of substance use risk associated with different dimensions of adolescent girls' pubertal development are more complicated than originally theorized. In the Discussion section

below, I explore potential explanations for these results in relation to theories of maturational deviance, embodiment, and social control.

DISCUSSION AND CONCLUSIONS

This study examined whether introducing theoretically proximal substance use risk factors—intrapersonal self-appraisals and interpersonal social relationships—would explain why pubertal development was associated with adolescent girls' substance use. Results did not provide consistent support for the research hypotheses. Specifically, the effects of body development and age at menarche on alcohol use did not disappear after controlling for self-appraisals and social relationships (see Hypotheses 1, 3). Although self-appraisals were associated with tobacco use during adolescence, neither self-esteem nor weight dissatisfaction completely mediated the link between pubertal development and tobacco use (see Hypothesis 2). Consistent with Hypothesis 4a, the association between body development and tobacco use in early and middle adolescence disappeared after controlling for interpersonal social relationships; this mediating effect did not hold in the Wave III models, however. Finally and contrary to expectations, later age at menarche was associated with higher levels of alcohol use in late adolescence.

Despite inconsistent support for the research hypotheses, results suggest several important conclusions. First, the substance use risk associated with pubertal development varies at different points during the course of adolescent development. The pathways of substance use risk associated with body development are developmentally specific such that the risk is most acute during a short window of early adolescence. This is consistent with previous research that documents a "catch-up" effect so that early development is most

immediately risky for young adolescent girls (Dick et al. 2000; Stattin and Magnusson 1990). A second conclusion is that the substance use risk associated with adolescent girls' pubertal development may vary for different types of substances. Studies examining adolescent substance use often focus on "substance use" in general, rather than disaggregating findings by specific substances. Although it is important for theory development to explore various "problem behavior syndromes," this should not preclude examining the substance-specific pathways of risk like those found in the current study. Indeed, the reasons that adolescent girls use tobacco may be quite different (e.g., for weight control) than reasons for using alcohol (e.g., peer camaraderie).

A third conclusion from the current study is that the substance use risk associated with pubertal development may also depend on how one measures pubertal development. Body development may have the most immediate substance use risk given the salience of the physical body during puberty and adolescence. Results suggest that early body developers may associate with deviant peers in early adolescence, which explains their higher levels of tobacco and alcohol use. Conversely and contrary to previous research, older age at menarche (i.e., late pubertal development) may have a long-term impact on alcohol use, predicting higher levels of alcohol use in late adolescence. We must look beyond stage termination theory to explain the finding that later age at menarche predicts higher levels of alcohol use in late adolescence.

One possible explanation for this finding is that maturational deviance, or off-time development, rather than early development places adolescent girls at risk for substance use (Brooks-Gunn, Petersen, and Eichorn 1985; Petersen and Taylor 1980). Although later age at menarche was associated with elevated alcohol use levels in late adolescence, the

maturational deviance hypothesis is too simplistic to explain these results given that the risk associated with early and late pubertal development varied by type of substance as well as measurement. As such, continued theoretical development is needed to explain the dynamic relationship between different components of pubertal development and substance use. For instance, one potentially useful theoretical concept is that of embodiment, or the recognition that physical bodies are lived bodies situated within social contexts. Although age at menarche is a physiological event, how an adolescent girl experiences and interprets menarche is shaped by her social environment, which in turn may affect bodily changes (Fingerson 2006). So in the case of age at menarche and alcohol use, there may be evidence of a type of "suppression effect" such that in early adolescence, late developers may engage in fewer heterosocial activities involving alcohol due to their perceived physical and social immaturity (Dubas, Graber, and Petersen 1991). Once late developers catch-up to their peers in late adolescence, they may engage in more heterosocial activities involving alcohol as an attempt to overcome potentially missed social opportunities in early adolescence. Continued empirical research is needed to test this theoretical proposition.

A final conclusion from this study is that intrapersonal self-appraisals and interpersonal social relationships are probably best viewed as key correlates, rather than full mediators of, pubertal development and adolescent girls' alcohol use. Consistent with previous empirical research (Patton et al. 2004), interpersonal social relationships with parents and peers emerge as the most important mediating factors in the association between pubertal development and substance use. Specifically, weak ties to parents and strong ties to deviant peers partially explain why body development is positively associated with substance use. However, interpersonal social relationships only fully mediated the association between

body development and tobacco use in early and middle adolescence. Thus more research is needed to explore other potential mediators to explain the association between pubertal development and adolescent girls' substance use, while being sensitive to the methodological and measurement issues noted above.

Limitations and Directions for Future Research

Several limitations of this study should be acknowledged. First, this study did not disaggregate findings for girls from different racial/ethnic and socioeconomic backgrounds. Adolescent girls' interpretation of the developing body may vary by race/ethnicity as well as socioeconomic status, as might the pathways of risk linking pubertal development with substance use. More studies are needed to disentangle these effects across racial/ethnic and socioeconomic groups of adolescent girls. Second, here I only focused on alcohol and tobacco use outcomes, and did not examine "harder" substances such as marijuana or cocaine. Although prevalence rates for these other substances are quite low among adolescent girls, future research should examine the pathways of risk associated with other substances given that hard substance use may be more strongly linked to negative life outcomes. Third, I only presented these variables as theoretically proximal predictors of pubertal development and adolescent girls' substance use. More research is needed to test hypothesized causal pathways among these substance use risk factors for adolescent girls. Finally, more research is needed to unpack the substance use risk associated with adolescent girls' pubertal development. Specifically, in addition to interpersonal social relationships, what other social contextual and social psychological factors may explain or mediate this relationship? Taken together with results from other studies (Halpern et al. 2007; Patton et al.

2004) additional mediators may be attachment to older or more deviant in-groups and de-identification with same age peers.

Intervention efforts aimed at preventing substance use among pre-adolescent girls may therefore benefit by focusing on content that enhances girls' self-concept and sense of self-worth so that early (and late) developers are not devalued by same-age peers, but rather come to view their developed bodies in a positive rather than stigmatizing and differentiating way (Sigall and Pabst 2005). Although individual level intervention programs may help girls learn to personally negotiate the meanings associated with their new woman-like bodies, larger structural changes may be necessary for long term change—for instance, reducing gender inequality and the sexualization and devaluation of the adult woman body.

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CHAPTER IV

PUBERTAL DEVELOPMENT AND ADOLESCENT GIRLS' SUBSTANCE USE: RACE, ETHNICITY, AND NEIGHBORHOOD CONTEXTS OF VULNERABILITY

The social epidemiological literature on adolescent substance use has long focused on individual level risk factors such as affective states, behavioral skills, cognitive perceptions, and personality traits (Bachman, Johnston, and O'Malley 1998; Jessor and Jessor 1977; Petraitis, Flay, and Miller 1995). Although this body of literature has contributed greatly to an individual-centered understanding of adolescent substance use, ecological perspectives are needed to examine the interactivity between individual and environmental, or structural risk factors (as called for by Celio, Karnik, and Steiner 2006; Jessor 1998; Kawachi and Berkman 2003; Schulenberg and Maggs 2001; Wen, Van Duker, and Olson 2008). The social ecological model (Bronfenbrenner 1979) posits that multiple ecological contexts—including individual, peer, family, and neighborhood contexts— interact to influence individual level health behaviors like adolescent substance use. Although scholars have extensively studied the interactive effect of individual, peer, and family contexts on adolescent substance use (see Hawkins, Catalano, and Miller 1992), less is known about the interplay between neighborhood and individual contexts as they relate to adolescent substance use (but see Chuang et al. 2005). Given the impact of neighborhood contexts on myriad health outcomes such as chronic illness, heart disease, mortality, and self-reported health (Diez-Roux et al. 1997; Jones and Duncan 1995; LeClere, Rogers, and Peters 1998; Robert 1988; Ross and Mirowsky 2001; Sloggett and Joshi 1998), neighborhoods likely provide larger contexts of vulnerability that influence individual level predictors of adolescent substance use.

One important individual level predictor of substance use among adolescent girls is the timing of pubertal development; early development compared to peers is positively associated with substance use whereas late development is negatively related to substance use (e.g., Chung, Park, and Lanza 2005; Magnusson, Stattin, and Allen 1986; Tanner-Smith 2009a; Wilson et al. 1994). However, there is a lack of research regarding whether and how neighborhood contexts influence the relationship between pubertal development and adolescent girls' substance use. For although pubertal development is a physiological process occurring at the individual level, it is also a developmental transition given meaning only by the socio-cultural context in which it is embedded (Schulenberg and Maggs 2001). The role of pubertal development in adolescent girls' healthful transition into and out of adolescence therefore relates to their broader ecological surroundings. Neighborhood "social dislocations", or characteristics referring to the potential deleterious structural and economic organization of a neighborhood, arise from unequal access to resources and opportunities (Wilson 1987) and may create ecological contexts of vulnerability that exacerbate individual level substance use risk factors (e.g., early pubertal development) while simultaneously eroding protective factors (e.g., late pubertal development). Such ecological contexts of vulnerability may be more detrimental to the health of young girls of color (Crane 1991; Hogan and Kitagawa 1985), given patterns of residential segregation that force poor minority families into concentrated, decaying neighborhoods (Massey and Denton 1993; Wilson 1987).

Drawing on the social ecological model, the current study is in response to calls for more research on the interactivity between individual and structural risk factors for adolescent substance use (Celio et al. 2006). This study addresses the following research

questions: Do neighborhood dislocations moderate the association between pubertal development and adolescent girls' substance use? And do the relationships between pubertal development, neighborhood dislocations, and adolescent girls' substance use vary by race/ethnicity? Before outlining the specific research hypotheses, I will discuss the theoretical and empirical literatures linking pubertal development, neighborhood contexts, and race/ethnicity with adolescent substance use.

PUBERTAL DEVELOPMENT, NEIGHBORHOOD CONTEXTS, AND SUBSTANCE USE

Pubertal Development and Substance Use

When a girl experiences puberty relative to her peers is an important individual level predictor of substance use. The maturational deviance or off-timing hypothesis posits that both early and late pubertal developers are more likely to engage in risky behaviors like substance use because they are physically and socially deviant from same-age peers (Brooks-Gunn, Petersen, and Eichorn 1985; Tschann et al. 1994). A competing perspective is the stage termination or early timing hypothesis. According to the early timing hypothesis, only early pubertal developers are at risk for behaviors like substance use because they are psychologically and socially unprepared to handle the new roles and responsibilities associated with the developed, adult-like body (Brooks-Gunn et al. 1985; Petersen and Taylor 1980; Stattin and Magnusson 1990). Early developers experience a maturity gap, or state of "pseudomaturity" in which they have physically adult bodies but are not granted adult privileges of independence (Galambos, Barker, and Tilton-Weaver 2003). In an attempt to gain autonomy and overcome this maturity gap, some early developers may engage in behaviors that mimic adult behavior such as drinking alcohol or smoking cigarettes (Caspi

and Moffitt 1991; Moffitt 1993). From the early timing perspective, girls who experience puberty later than their peers do not face a maturity gap and should be less likely to use substances given their delayed entry into adult-like status. Empirical research generally supports the early timing hypothesis, indicating that early pubertal development predicts higher levels of substance use among adolescent girls (e.g., Chung et al. 2005; Dick et al. 2000; Lanza and Collins 2002; Stice, Presnell, and Bearman 2001; Tanner-Smith 2009a; Wilson et al. 1994). Findings on the effect of late pubertal development on substance use have been less consistent, however, with some studies reporting a long-term substance use disorder risk associated with late development (Graber et al. 1997; Graber et al. 2004), and others reporting a protective effect associated with late development (e.g., Dick et al. 2001; Wilson et al. 1994).

To date, there is a limited understanding of how neighborhood contexts influence the relationship between pubertal development and adolescent girls' substance use. Rather than focusing on single individual level risk factors, it is more helpful to conceptualize the potential accumulation of risk and protective factors at various ecological levels (Rutter 1979; Sameroff et al. 1998). Adolescents become increasingly vulnerable to risky health behaviors as their exposure to individual, familial, and neighborhood level stressors accumulates (Appleyard et al. 2005; Sameroff 2000). Further, when children are exposed to a high number of risk factors, individual level protective factors often lose their protective effect (Sameroff et al. 1998). Thus we would expect that the substance use risk/protection associated with pubertal development should vary depending on neighborhood contexts of vulnerability.

Neighborhood Contexts and Substance Use

Neighborhoods are important ecological contexts in which adolescents' lived experiences are embedded, particularly given adolescents' constrained geographic mobility. Early Chicago-school theorists attributed the influence of neighborhood organization on behaviors like delinquency and substance use to issues of socioeconomic disadvantage, residential instability, and population heterogeneity (Shaw and McKay 1942). Building on social disorganization theory, contemporary theorists argue that the health risks associated with neighborhood organizational characteristics are largely attributable to issues of social cohesion and social control (Coleman 1988; Kubrin and Weitzer 2003; Sampson and Groves 1989; Sampson, Raudenbush, and Earls 1997). Three key neighborhood dislocations related to social cohesion and social control are: concentrated disadvantage, residential instability, and social disorder. These three structural characteristics of neighborhoods influence the social organization of a community, and in turn have implications for adolescent health behaviors like substance use.

Concentrated disadvantage may be risky for adolescent substance use due to issues of drug availability, community norms, and social control. For instance, adolescents in disadvantaged neighborhoods have greater exposure to the bars and liquor stores that are more highly concentrated in disadvantaged neighborhoods (Duncan, Duncan, and Strycker 2002; LaVeist and Wallace 2000). Adolescents in poor neighborhoods may also have increased exposure to deviant peer networks of drug dealers or drug users, and hence more opportunities or perceived opportunities to acquire drugs (Beyers et al. 2004; Crum, Lillie-Blanton, and Anthony 1996; Ge et al. 2002; Wilson 1996). Adolescents growing up in disadvantaged neighborhoods may therefore be collectively socialized to norms and values

tolerant of substance use (Jencks and Mayer 1990; Kadushin et al. 1998), and have limited access to positive social role models (Wilson 1987). But perhaps most important is that poorer neighborhoods often lack the necessary resources to prevent substance use or implement sanctions against substance users (Anderson 1990). Disadvantaged neighborhoods often lack necessary local institutions such as schools and neighborhood organizations that exercise informal social control on adolescents. Thus the chronic stress and strain, along with potentially disrupted social networks in disadvantaged neighborhoods all contribute to the risk of adolescent substance use (Boardman et al. 2001; Kowaleski-Jones 2000; Lynch and Cicchetti 1998).

Similarly, residential instability may be risky for adolescent substance use due to its association with the social cohesion and informal social control available within a neighborhood. Home owners have a vested interest in maintaining and improving the neighborhood; high turnover among neighborhood residents and low levels of home ownership decrease social relationships/ties and hence the sense of mutual trust within a neighborhood (Kasarda and Janowitz 1974; Kawachi and Berkman 2000). Similar to neighborhoods with high levels of concentrated disadvantage, those with high levels of residential instability have fewer structures in place to maintain effective social control (Sampson et al. 1997). The eroded social control that accompanies residential instability is problematic given its impact on normative climates that allow or tolerate disorderly behavior such as substance use. Adults living in residentially unstable neighborhoods may be less willing or able to manage youth in the neighborhood (Sampson et al. 1997).

The third and related neighborhood dislocation of interest is social disorder. Neighborhoods with high levels of concentrated disadvantage and residential instability are

generally plagued by overall social disorder, or what others have termed "ambient hazards" (Aneshensel and Sucoff 1996). Social disorder refers to the observable signs of decay or disorder within a neighborhood, such as crime, graffiti, litter, public intoxication, and other incivilities. Social disorder signifies the lack of social control and cohesion within a neighborhood, indicating an alienated community (Massey 1996) that is unwilling or unable to supervise and maintain an orderly environment. As such, these cues may influence adolescent substance use by providing access to substances, community norms tolerant of substance use, and role modeling of substance use.

In sum, concentrated disadvantage, residential instability, and social disorder are three key neighborhood level risk factors for adolescent substance use. However, research on the link between neighborhood dislocations and substance use has yielded inconsistent findings. Although some studies suggest that these neighborhood dislocations are associated with higher levels of substance use or access to substances (Bernstein et al. 2007; Beyers et al. 2004; Boardman et al. 2001; Crum et al. 1996; Hill and Angel 2005; Kulis et al. 2007; Lambert et al. 2004; Wardle et al. 2003), others studies have found no relationship (Abdelrahman et al. 1998; Allison et al. 1999), or even a negative relationship (Chuang et al. 2005; Ennett et al. 1997). And with few exceptions (e.g., Chuang et al. 2005; Crum et al. 1996; Foshee et al. 2007), the empirical literature linking neighborhood dislocations with substance use has focused on adults only or aggregated samples of adolescents and adults.

Despite the inconsistent findings linking neighborhood dislocations with adolescent substance use, research does suggest that neighborhood contextual factors influence or moderate the impact of individual level risk factors on behavioral outcomes (Brooks-Gunn and Warren 1985; Caspi et al. 1993; Dick et al. 2000; Leventhal and Brooks-Gunn 2000).

Further, several studies have indicated that the effect of pubertal development on various behavioral outcomes is conditional upon neighborhood contexts (Foshee et al. 2007; Ge et al. 2002; Obeidallah et al. 2004). For instance, in a multi-state study of the relationship between pubertal development and problem behavior among African American adolescents, Ge et al. (2002) found that early developing adolescents residing in disadvantaged neighborhoods and those with less supportive/involved parents had more affiliations with deviant peers. In another study examining the effect of neighborhoods on the link between pubertal development and girls' violent behavior in Chicago, Obeidallah et al. (2004) found that early pubertal development was related to violent behavior only for girls living in disadvantaged neighborhoods. There is only one study to date that has examined the moderating role of neighborhood contexts on the association between pubertal development and adolescent substance use. Among a sample of suburban middle school students in North Carolina, Foshee et al. (2007) found that the effect of pubertal development on cigarette and alcohol use among girls was moderated by peer contexts, but not neighborhood contexts. However, Foshee et al. (2007) speculate that the lack of observed neighborhood effects in their study may have been due to problems with the validity of their pubertal development measure. So, there is a paucity of empirical research examining the moderating role of neighborhood dislocations on the association between pubertal development and adolescent girls' substance use, and the available studies have been limited to specific geographic areas or limited by psychometrically suspect measures. More systematic research is needed to examine the role of neighborhood dislocations in the pubertal development-substance use relationship.

The interactivity between pubertal development, neighborhood dislocations, and substance use among adolescent girls may not be equivalent for girls from different

racial/ethnic backgrounds, however. These ecological contexts of vulnerability likely vary by race and ethnicity given group differences in the predictors and consequences of pubertal development, neighborhood dislocations, and substance use.

Racial/Ethnic Differences

It is problematic and premature for social epidemiological researchers to assume that the risk and protective factors associated with adolescent substance use are similar across racial/ethnic groups (Brown, Miller, and Clayton 2004; Gottfredson and Koper 1996; Wallace and Muroff 2002). As to the research question at hand, it is important to note racial/ethnic differences in the predictors and consequences of pubertal development, substance use, and neighborhood dislocations that may influence different patterns of relationships as they relate to adolescent girls' substance use.

First, research indicates that girls of color, particularly African American girls, experience puberty earlier than white girls (Herman-Giddens, Kaplowitz, and Wasserman 2004; Schreiber et al. 1996; Wu et al. 2001), and have lower substance use prevalence than white girls (e.g., Johnston et al. 2008; MacKay and Duran 2007). If girls of color tend to be early developers, then their reference groups of same-race same-age peers will likely be composed of other early developers. So, "early" development may be less risky for girls of color if it does not correspond with any pronounced physical or social deviance from their reference group. Most of the research examining the substance use risk/protection associated with pubertal development has relied on samples of white adolescent girls, therefore precluding investigation of racial/ethnic differences in the substance use risk associated with pubertal development. One exception was the work of Wilson and colleagues (1994), which

indicated that the substance use risk associated with early development was most pronounced for white adolescent girls compared to Hispanic adolescent girls.

Although adolescent girls of color may be protected from the detrimental effect of pubertal development on substance use, neighborhood risk factors may actually be magnified for these girls (Gruenewald et al. 2000; Lambert et al. 2004). Racial segregation in residence patterns contributes to a concentration of poverty within certain neighborhoods with large populations of racial/ethnic minorities (Massey and Denton 1993). Members of minority communities are more likely to be exposed to advertising for alcohol and tobacco (Alaniz and Wilkes 1995; Douglas, Esmundo, and Bloom 2000; Hackbarth, Silvestri, and Cosper 1995), and thus collective socialization to norms and values tolerant of substance use may be more pronounced for young girls of color. Given these larger patterns of racial segregation in housing, young adolescent girls of color may be more likely to live in segregated, disadvantaged, and decaying neighborhoods with low levels of collective trust and shared norms—neighborhoods victim to perpetual cycles of social disorder and social withdrawal that are risky for adolescent substance use. Empirical research suggests that contextual, neighborhood level risk factors are more detrimental to the health and well-being of adolescents of color than they are to white adolescents (Crane 1991; Hogan and Kitagawa 1985; Kulis et al. 2007), but that those with supportive family ecosystems may be resilient to the detrimental impact of neighborhood dislocations (Yabiku et al. 2007). Thus, adolescent girls of color may be resilient to the effects of pubertal development on substance use but be at greater risk if they reside in disadvantaged neighborhoods. No studies to date have examined racial/ethnic variation in the substance use risk associated with pubertal development, neighborhood dislocations, and the interaction between these risk factors.

THE CURRENT STUDY

Theory and previous research suggest that the neighborhood dislocations of concentrated disadvantage, residential instability, and social disorder may moderate the effect of pubertal development on adolescent girls' substance use, but these effects may vary by race/ethnicity. The current study addresses several gaps in the literature linking pubertal development, neighborhood dislocations, race/ethnicity, and substance use among adolescent girls. For example, previous studies examining the relationship between pubertal development have either focused on white girls only, or aggregated girls across different racial groups (e.g., Chung et al. 2005; Dick et al. 2000; Lanza and Collins 2002; Stice et al. 2001). This study examines whether these patterns of association vary for non-Hispanic black, Hispanic, and non-Hispanic white adolescent girls. Second, although previous research indicates that individual and neighborhood characteristics interact to influence health outcomes (see Leventhal and Brooks-Gunn 2000), the current study is the first to examine the moderating role of neighborhood dislocations on the association between pubertal development and substance use with a large, nationally representative sample of adolescent girls. Finally, in response to calls for an emphasis on social contexts (Celio et al. 2006), this study furthers our understanding of the complex connections between individual and neighborhood level risk factors for adolescent substance use. The current study tests the following research hypotheses:

H1: The effect of pubertal development on adolescent girls' substance use will vary by level of neighborhood dislocations.

H2: The effect of pubertal development on adolescent girls' substance use will vary by race/ethnicity.

H3: The effect of neighborhood dislocations on adolescent girls' substance use will vary by race/ethnicity.

H4: The moderating effect of neighborhood dislocations on the relationship between pubertal development and adolescent girls' substance use will vary by race/ethnicity.

METHODS

Sample

This study used restricted-use data from the first and third waves of the National Longitudinal Study of Adolescent Health (Add Health) (http://www.cpc.unc.edu/addhealth). The Add Health used a multistage, stratified, school-based sampling design with schools as primary sampling units (PSUs) and region of the country (Northeast, Midwest, South, and West) as a stratification variable. From a sample frame of 26,666 schools, 132 PSUs (80 high schools and 52 "feeder" middle schools) were selected and probability weights were used to adjust for non-response and unequal probabilities of selection.

The Add Health data are representative of U.S. students in 7-12th grade during the 1994-1995 school year. At Wave I (1994-1195), in-school questionnaire data were collected on 90,118 students and 20,745 students completed in-home surveys. At Wave II (1996), in-home surveys were collected on 14,738 students sampled from Wave I participants and non-participants. At Wave III (2001), in-home surveys were collected on 15,170 students sampled from Wave I participants and non-participants (see Udry, Bearman, and Harris 2006 for more detailed sampling design information). The analytic sample was limited to adolescent girls

age 10-15 at Wave I with no missing sampling weights.[23] Among these 5,591 girls, 1,266 self-identified as non-Hispanic black, 842 as Hispanic, 3,093 as non-Hispanic white, and 390 of other race/ethnicity. In all analyses, values coded as "refused", "don't know", or "missing" were treated as missing and cases were deleted listwise.

Measures

Four dependent variables were used to measure substance use. The first dependent variable was alcohol use, calculated as an average scale score from three items (Cronbach's α =.88): frequency of alcohol consumption; frequency of drinking five or more drinks in one setting; and frequency of drinking to drunkenness, all in the past year (1=none; 7=every day or almost every day). The second dependent variable was tobacco use, measured as the number of days a girl smoked cigarettes in the past month. The third dependent variable was alcohol abuse, calculated as an average scale score from five items (Cronbach's α =.72): trouble with parents due to drinking, trouble with school/school work due to drinking, problems with friends due to drinking, problems with dating partner due to drinking, and regrets over behavior that occurred when drinking, all in the past year (1=never; 4=5 or more times). The fourth dependent variable was marijuana use, measured with one item indicating the number of times used marijuana in the past 30 days. All substance use items were self-reported and measured at Waves I and III.

The key independent variables were pubertal development, neighborhood dislocations, and race/ethnicity. Pubertal development was measured with three items that

[23] Approximately 8-10% of cases sampled at each wave had missing sampling weights. These respondents were selected outside of the sampling frame at Wave I to be included in the twin/sibling sample, and were sampled to ensure large enough sample sizes to obtain genetically related respondents. Because these cases were sampled outside of the sampling frame they did not receive sampling weights and were excluded from all analyses (see Chantala 2006).

asked girls about increased breast size since grade school, body curviness since grade school, and physical development compared to same-age peers (1=less developed; 5=most developed). These three items (Cronbach's α =.70) were used to create an average scale score ranging from 1-5. Respondents scoring in the upper 20th percentile of the pubertal development scale were categorized as early (1=yes), those in the middle 60th were on-time (1=yes), and those in the lower 20th were late (1=yes).[24] Pubertal development was measured at Wave I.

Three variables were used to capture neighborhood dislocations. The first was concentrated disadvantage, calculated as factor scores from a confirmatory maximum likelihood factor analysis based on five items (Cronbach's α =.85): the proportion of single female headed households with children, proportion of households with public assistance income, proportion of persons below the poverty level, proportion of persons over age 25 with no high school degree, and the unemployment rate in the respondent's block group census area. The second neighborhood dislocation variable was residential instability, calculated as an average scale score based on two items (Cronbach's α =.56): proportion of residents not living in the same house from 1985 to 1990, and proportion of occupied housing units not owner occupied. The concentrated disadvantage and residential instability items came from the Add Health Contextual Database, which geocoded Add Health respondents' home locations to link with their block group census area from the 1990 Census of Population and Housing. The third neighborhood dislocation variable was social disorder, calculated as an average scale score based on two parent-reported items (Cronbach's α =.66): problem with litter or trash on the streets or sidewalks of the neighborhood, and problem with

[24] Sensitivity analyses using early and late cutoffs calculated as +/- 1 standard deviation above/below the mean of the pubertal development scale did not substantively alter the reported findings.

drug dealers and drug users in the neighborhood (1=no problem at all; 3=a big problem). All neighborhood dislocation variables were measured at Wave I.

Self-reported race/ethnicity was recoded into four indicator variables representing non-Hispanic black (1=yes), Hispanic (1=yes), non-Hispanic white (1=yes), and other race/ethnicity (1=yes). The remaining control variables included age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition. These control variables were included because previous research has established their connection to pubertal development, neighborhood dislocations, and adolescent substance use (e.g., Arim et al. 2007; Goodman and Huang 2002; Hawkins et al.1992; Kulis et al. 2007; Sampson et al. 1997; Tanner-Smith 2009b). Age was a continuous measure of respondents' self-reported age in years. Substance-using friends was based on three items indicating how many of a girls' three closest friends used tobacco, alcohol, and marijuana. These three items ranged from 0-3 and were summed to form a scale ranging from 0-9. Parental caring was measured with one item indicating how much a girl perceived her parents cared about her (1=not at all; 5=very much). Parental closeness was measured as the maximum of two items measuring how close a girl felt to her mother or father (1=not at all; 5=very much). Parent-reported parental education was the highest level of education obtained by the respondent's parent or parent's partner, and ranged from 0 (no school) to 8 (professional degree).[25] Collective efficacy was calculated as an average scale score based on two parent-reported items: would tell neighbor if their child got into trouble, and neighbor would tell if my child got into trouble (1=definitely would not;

[25] Because 33% of valid cases were missing parent-reported parental education data, missing cases were imputed with child-reported parental education when available (n=419).Although child-reported parental education was not an ideal measure, the correlation between child- and parent-reported parental education in the analytic sample was .70.

5=definitely would). Urbanicity was measured with three dummy variables (1=yes) for urban, suburban, and rural area of residence. Region was measured with four dummy variables (1=yes) for West, Midwest, Northeast, and South. Finally, neighborhood racial composition was measured with two items from the Add Health Contextual Database: proportion of black residents in neighborhood, and proportion of Hispanic residents in the neighborhood. All control variables were measured at Wave I.

Analytic Strategies

Nested regression models with multiplicative interaction terms were used to test the potential moderating effect of neighborhood dislocations on the relationship between pubertal development and adolescent girls' substance use. Additional split sample models for black, Hispanic, and white adolescent girls were used to explore whether race/ethnicity further moderated this relationship.[26] To test whether results were robust across measurement of substance use, all models predicted the four substance outcomes: alcohol use, tobacco use, alcohol abuse, and marijuana use. Finally, all outcomes were predicted with a cross-sectional model with all variables measured at Wave I, and a lagged longitudinal model predicting substance use at Wave III with Wave I predictors.

Ordinary Least Squares (OLS) regression models were used to predict the two continuously measured alcohol outcomes. OLS assumes independence and normality of residuals, homoskedasticity, and relationship linearity (Allison 2002). Diagnostics indicated that none of the alcohol models grossly violated these assumptions. The tobacco and marijuana outcomes were both non-negative count outcomes, however, and thus violated

[26]Split sample models for the "other" racial/ethnic category were omitted due to the potential theoretical and statistical heterogeneity within this group.

OLS assumptions of normality and homoskedasticity of residuals (Long 1997). Negative binomial (NB) regression models were used to predict the tobacco and marijuana outcomes. NB regression is an appropriate multivariate statistical technique when modeling non-negative and overdispersed count outcomes with strong positive skew. The NB regression model can be expressed mathematically as

$$\lambda_i = \exp(\mathbf{X}_i * \boldsymbol{\beta})*\exp(\varepsilon_1)$$

where $\mathbf{X}_i$ is the matrix of independent variables on individuals i and $\boldsymbol{\beta}$ is a vector of regression coefficients relating independent variables to the mean substance use rate. The error term $\exp(\varepsilon_1)$ is distributed as $\Gamma(1, \alpha)$, where α is the dispersion parameter that scales the relationship between the mean and variance of the outcome. The NB1 (Cameron and Trivedi 1998) parameterization was used to specify a linear mean-variance relationship for models predicting tobacco and marijuana use, with the following formulation:[27]

$$\text{var}(Y_i \mid \mathbf{X}_i) = \lambda_i(1 + \alpha)$$

The clustered sample design and correlated error structure of the Add Health data required statistical corrections for design effects and unequal probability of selection (Chantala and Tabor 1999). A Taylor Series variance estimation method was used to estimate standard errors and insured consistency in the probability of Type I errors. Probability weights (variables GSWGT1, GSWGT3_2), strata (variable REGION), and primary sampling units (variable PSUSCID) were specified in Stata 10.1 for use with the survey-based OLS and NB1 regression procedures using the subpopulation commands.

[27] The NB1 parameterization was chosen because it exhibited the larger log-likelihood values in negative binomial models unadjusted for the complex survey design.

RESULTS

Descriptive Statistics

Table 4.1 presents descriptive statistics for the substance use, pubertal development, neighborhood dislocations, and control variables. As shown in Table 4.1, on average girls used low levels of alcohol (1.56); alcohol use increased slightly over time but remained relatively infrequent at Wave III (2.31). Alcohol use varied significantly by race/ethnicity, with white girls consistently reporting the highest alcohol use followed by Hispanic and black girls. At Wave I, girls reported smoking cigarettes an average of 4 days in the past month, which increased to over 8 days per month at Wave III. White girls reported the highest levels of tobacco use at both waves, followed by Hispanic and black girls. Alcohol abuse was relatively low among this sample of adolescent girls, although overall abuse levels increased from .10 at Wave I to .13 at Wave III. Alcohol abuse varied significantly by race/ethnicity; Hispanic and white girls reported higher levels of alcohol abuse than black girls. The average girl used marijuana only once in the past 30 days at Wave I, which increased to over twice a month by Wave III. White girls reported the highest levels of marijuana use, followed by Hispanic and black adolescent girls.

Approximately 15% of girls were early developers, and 18% were late developers.[28] Pubertal development varied significantly by race/ethnicity; black girls were more likely to be categorized as early developers or late developers (as opposed to on-time). In terms of neighborhood dislocations, black and Hispanic girls lived in significantly higher risk

[28] These percentages are slightly less than the 20% cut-offs for the early and late pubertal development scale due to differences in the percentile cut-points and plausible values on the pubertal development scale.

neighborhoods characterized by concentrated disadvantage, residential instability, and social disorder.

The average age of girls in the sample was 14, and age did not vary by racial/ethnic group. The average girl had two substance-using friends, with white girls reporting more substance-using friends than black and Hispanic girls (2.19, 1.67, and 2.02, respectively). Most girls felt their parents cared about them quite a bit to a lot, and this measure did not vary by race/ethnicity. The average girl also felt very close with at least one of her parents, and black girls reported the highest level of parental closeness. The average level of parental education was between vocational/technical school and some college, and was highest among parents of white girls. Black girls lived in neighborhoods with significantly higher average levels of collective efficacy, with Hispanic girls living in those with the lowest levels of collective efficacy (4.35 and 3.93, respectively). Most girls lived in suburban or urban areas, although white girls were more likely to reside in suburban areas (63%) and Hispanic girls more likely to live in urban areas (84%). There was substantial geographic variability among the sample, but most girls lived in either the Midwest or the South (33% and 36%, respectively). Region of residence varied by race/ethnicity; black girls were most likely to live in the South, Hispanic girls in the West, and white girls in the Midwest. Finally, on average girls lived in neighborhoods with few black (13%) or Hispanic (7%) residents. Neighborhood racial composition varied by race/ethnicity such that black girls lived in neighborhoods with an average of 57% black residents, and Hispanic girls lived in neighborhoods with an average of 32% Hispanic residents.

Table 4.1. Means, Standard Deviations, and Ranges of Substance Use, Pubertal Development, Neighborhood Dislocations, and Control Variables[ab]

	All Girls (N =5,591)	Black Girls (N =1,266)	Hispanic Girls (N = 842)	White Girls (N =3,093)	Range
Substance Use					
Alcohol use, Wave I***	1.56	1.38	1.56	1.61	1-7
	(.99)	(.82)	(1.01)	(1.03)	
Wave III***	2.31	1.57	2.00	2.55	
	(1.29)	(.93)	(1.19)	(1.35)	
Tobacco use, Wave I***	3.77	.47	2.61	4.66	0-30
	(8.38)	(2.56)	(7.04)	(9.91)	
Wave III***	8.40	3.12	5.92	10.21	
	(12.35)	(8.31)	(10.81)	(13.54)	
Alcohol abuse, Wave I***	.10	.05	.14	.11	0-4
	(.29)	(.18)	(.35)	(.29)	
Wave III***	.13	.04	.08	.16	
	(.39)	(.21)	(.31)	(.45)	
Marijuana use, Wave I	.98	.66	.80	1.10	0-100
	(4.81)	(5.06)	(3.59)	(4.80)	
Wave III***	2.49	1.50	1.73	2.88	
	(7.74)	(6.34)	(6.63)	(8.37)	
Pubertal Development					
Early (1=yes)*	14.67%	18.66%	11.99%	14.50%	0-1
	(.36)	(.38)	(.33)	(.36)	
Late (1=yes)***	17.78%	23.46%	21.18%	15.60%	0-1
	(.39)	(.41)	(.39)	(.37)	
Neighborhood Dislocations					
Concentrated	-.05	.89	.25	-.28	-1.30-
disadvantage***	(.98)	(1.19)	(.97)	(.68)	5.72
Residential instability***	.37	.43	.49	.34	.01-
	(.16)	(.17)	(.18)	(.14)	.99
Social disorder***	1.51	1.70	1.53	1.47	1-3
	(.56)	(.62)	(.61)	(.50)	
Control Variables					
Age	13.62	13.68	13.60	13.62	10-15
	(1.09)	(1.07)	(1.07)	(1.09)	
Substance-using	2.09	1.67	2.02	2.19	0-9
friends***	(2.48)	(2.15)	(2.50)	(2.58)	
Parental caring	4.83	4.83	4.81	4.83	1-5
	(.55)	(.55)	(.61)	(.53)	

Table 4.1 cont.

	All Girls (N =5,591)	Black Girls (N =1,266)	Hispanic Girls (N = 842)	White Girls (N =3,093)	Range
Parental closeness*	4.60	4.66	4.58	4.60	1-5
	(.75)	(.76)	(.83)	(.71)	
Parental education***	5.35	4.81	4.29	5.62	0-8
	(1.92)	(1.92)	(2.26)	(1.69)	
Collective efficacy***	4.11	4.35	3.93	4.09	1-5
	(.79)	(.77)	(.93)	(.74)	
Suburban (1=yes)**	59.38%	57.19%	36.69%	63.17%	0-1
	(.50)	(.50)	(.49)	(.50)	
Urban (1=yes)***	50.91%	60.12%	83.73%	43.06%	0-1
	(.50)	(.48)	(.37)	(.49)	
Rural (1=yes)	15.23%	10.60%	4.79%	18.25%	0-1
	(.37)	(.32)	(.21)	(.42)	
West (1=yes)***	17.75%	8.54%	41.42%	13.94%	0-1
	(.42)	(.34)	(.50)	(.37)	
Midwest (1=yes)**	32.80%	23.78%	9.44%	39.00%	0-1
	(.43)	(.39)	(.26)	(.47)	
Northeast (1=yes)	13.66%	5.43%	14.61%	14.89%	0-1
	(.35)	(.26)	(.37)	(.38)	
South (1=yes)**	35.79%	62.25%	34.53%	32.17%	0-1
	(.49)	(.49)	(.47)	(.47)	
Proportion black***	.13	.57	.10	.05	0-1
	(.29)	(.33)	(.18)	(.11)	
Proportion Hispanic***	.07	.04	.32	.03	0-1
	(.19)	(.11)	(.32)	(.08)	

Notes. [a]Standard deviations not adjusted for complex survey design. [b]Equality of means across racial/ethnic groups tested with F-statistics from probability weighted one-way ANOVAs for continuous variables and survey adjusted Design-based F-statistics from two-way contingency tables for categorical variables.
* p<.05. ** p<.01. *** p<.001.

Pubertal Development, Neighborhood Dislocations, and Substance Use

Table 4.2 presents coefficients and standard errors from eight survey adjusted regression models predicting the main effects of pubertal development and neighborhood dislocations on substance use among all adolescent girls. The models predict each outcome at Wave I and Wave III using predictors measured at Wave I. Results for the alcohol use and alcohol abuse models (1st and 3rd panels of Table 4.2) show coefficients and standard errors from ordinary least squares regression models; results for the tobacco and marijuana use models (2nd and 4th panels of Table 4.2) are from negative binomial regression models. All models control for race/ethnicity, age, substance-using peers, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood composition. The Wave III models are lagged longitudinal models and so additionally control for baseline level of substance use at Wave I.

As shown in the first panel of Table 4.2, at the first wave of data collection early developers had slightly (but not significantly) higher predicted levels of alcohol use compared to on-time developers,[29] whereas late developers had significantly lower predicted levels of alcohol use. For instance, the average adolescent girl who was an early developer had a predicted alcohol use level of 1.76, compared to 1.71 for an on-time developer, and 1.64 for a late developer.[30] In terms of neighborhood dislocations, concentrated disadvantage, residential instability, and social disorder did not significantly predict adolescent girls' alcohol use at Wave I net of the control variables. By Wave III, late pubertal development no

[29] This finding is driven largely by the mediating effect of substance using friends on the relationship between pubertal development and substance use (see Tanner-Smith, 2009b). Indeed, if the substance-using friends control variable is omitted from the model, early pubertal development coefficient reaches statistical significance (early b=.19, p=.01).

[30] All predicted values are calculated with continuous covariates held at mean values and categorical control variables held at modal categories.

longer significantly influenced alcohol use, and concentrated disadvantage actually had a negative relationship with alcohol use. Girls who lived in more disadvantaged neighborhoods during adolescence had lower predicted levels of alcohol use by the time they reached late adolescence at Wave III. Given that the lagged longitudinal models at Wave III predict change in alcohol use levels between Waves I and Wave III, results in the first panel of Table 4.2 show that late pubertal development predicted initial levels of, but not changes in, alcohol use whereas concentrated disadvantaged predicted changes in, but not initial levels of, alcohol use among adolescent girls.

The second panel of Table 4.2 predicts adolescent girls' tobacco use. Results suggest that late developers had significantly lower predicted levels of tobacco use at Wave I compared to on-time developers (b=-.29; p=.04), and early developers (Wald $F_{2,127} = 3.54$; p=.03).[31] Although concentrated disadvantage and social disorder did not significantly influence adolescent girls' tobacco use, residential instability was significantly and positively related to tobacco use at Wave I. At Wave III, late development retained its significant protective effect on tobacco use, but none of the neighborhood dislocation variables predicted changes in tobacco use between Waves I and Wave III.

[31] Again, the lack of a significant effect for early development coefficient is due to the mediating effect of substance-using friends; the early pubertal development coefficient is significant when the substance-using friends control variable is omitted from the model (early b=.38, p<.001).

Table 4.2. Coefficients and Standard Errors from Survey Adjusted Main Effects Models Predicting Substance Use for All Girls, by Type of Substance and Wave of Data Collection[ab]

	Alcohol Use		Tobacco Use		Alcohol Abuse		Marijuana Use	
	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III
Early development	.05	-.09	.10	-.02	.00	-.03	-.03	.06
	(.05)	(.07)	(.10)	(.12)	(.01)	(.02)	(.15)	(.16)
Late development	-.08*	.00	-.29*	-.25*	-.01	-.04**	-.24	.02
	(.04)	(.07)	(.14)	(.12)	(.01)	(.02)	(.20)	(.14)
Concentrated disadvantage	- .01	-.09*	.03	.03	.00	-.02	.05	.11
	(.03)	(.04)	(.05)	(.05)	(.01)	(.01)	(.08)	(.07)
Residential instability	.16	-.06	.69*	-.53	.02	.02	.96*	-.21
	(.12)	(.21)	(.28)	(.28)	(.04)	(.06)	(.39)	(.38)
Social disorder	-.06	-.02	-.11	-.01	.00	.01	-.14	.03
	(.05)	(.07)	(.08)	(.08)	(.01)	(.02)	(.10)	(.10)
Constant	.72	.96	-1.39	3.51***	-.01	.05	-4.18***	2.87**
	(.37)	(.60)	(.97)	(.91)	(.01)	(.14)	(1.25)	(1.03)
N	4,503	3,685	4,494	3,666	4,505	3,678	4,497	3,666
F Statistic	28.73***	15.75***	69.80***	25.66***	12.48***	4.15***	34.12***	5.13***
R-squared	.35	.12	--	--	.18	.04	--	--

Notes: [a]Standard errors in parentheses. Standard errors for negative binomial coefficients in parentheses for tobacco use and marijuana use models. [b]All models control for race/ethnicity, age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I.
* p<.05. ** p<.01. *** p<.001.

The third and fourth panels of Table 4.2 present results for more advanced or problematic levels of substance use: alcohol abuse and marijuana use. As shown in the third panel of Table 4.2, at Wave I pubertal development did not significantly predict alcohol abuse.[32] None of the neighborhood dislocation variables influenced adolescent girls' alcohol abuse scores at Wave I. At Wave III, late development emerged as a significant protective factor against alcohol abuse. So, late pubertal development significantly influenced changes in alcohol abuse, but neighborhood dislocations did not have any significant main effects on initial levels or changes in alcohol abuse. The fourth panel of Table 4.2 presents results for marijuana use and shows that net of the control variables, pubertal development did not significantly influence marijuana use at either wave of data collection.[33] The only neighborhood characteristic that significantly predicted marijuana use was residential instability such that girls living in more unstable neighborhoods had higher predicted levels of marijuana use at Wave I; however, residential instability did not predict changes in marijuana use between Waves I and III.

The lack of significant main effects in Table 4.2 could be partly due to the interaction between pubertal development and neighborhood dislocations. To examine the potential moderating effect of neighborhood dislocations on the substance use risk/protection associated with pubertal development, multiplicative interaction terms were estimated for the six pubertal development by neighborhood dislocation combinations (early*concentrated disadvantage; late*concentrated disadvantage; early*residential instability, etc.). The eight

[32] When the substance-using friends control variable is omitted from the model, the pubertal development coefficients reach statistical significance (early b=.03, p=.03; late b=-.03; p=.01).
[33] This finding is again driven largely by the mediating effect of substance using friends. If the substance-using friends control variable is omitted from the marijuana models, pubertal development is a significant predictor at Wave I (early b=.29, p=.08; late b=.44; p=.04).

models shown in Table 4.2 were replicated six times to include each of the multiplicative interaction terms.

With only one exception, none of the multiplicative interaction terms were significant at the .05 level. The only significant interaction was between late pubertal development and concentrated disadvantage in the model predicting alcohol abuse at Wave III (late b=-.05, p<.00; disadvantage b= -.03, p=.01; late*disadvantage b=.03; p=.02); concentrated disadvantage weakened the protective effect of late pubertal development on alcohol abuse in late adolescence. Figure 4.1 depicts this interaction by showing the predicted Wave III alcohol abuse scores by level of concentrated disadvantaged and pubertal development. As shown in Figure 4.1, late developing girls generally had lower levels of alcohol abuse than early and on-time developers. However, late developers' alcohol abuse levels were higher in more disadvantaged neighborhoods. Late developers living in neighborhoods with a high level of concentrated disadvantage had higher predicted alcohol abuse scores (.04) than early developing girls in disadvantaged neighborhoods (.02). Although neighborhood disadvantage reduced the protection associated with late pubertal development, Figure 4.1 also shows that neighborhood disadvantage reduced the risk associated with early development. These findings should be regarded as tentative, however, given that we would expect to find at least one significant interaction due to chance among this many regression models.

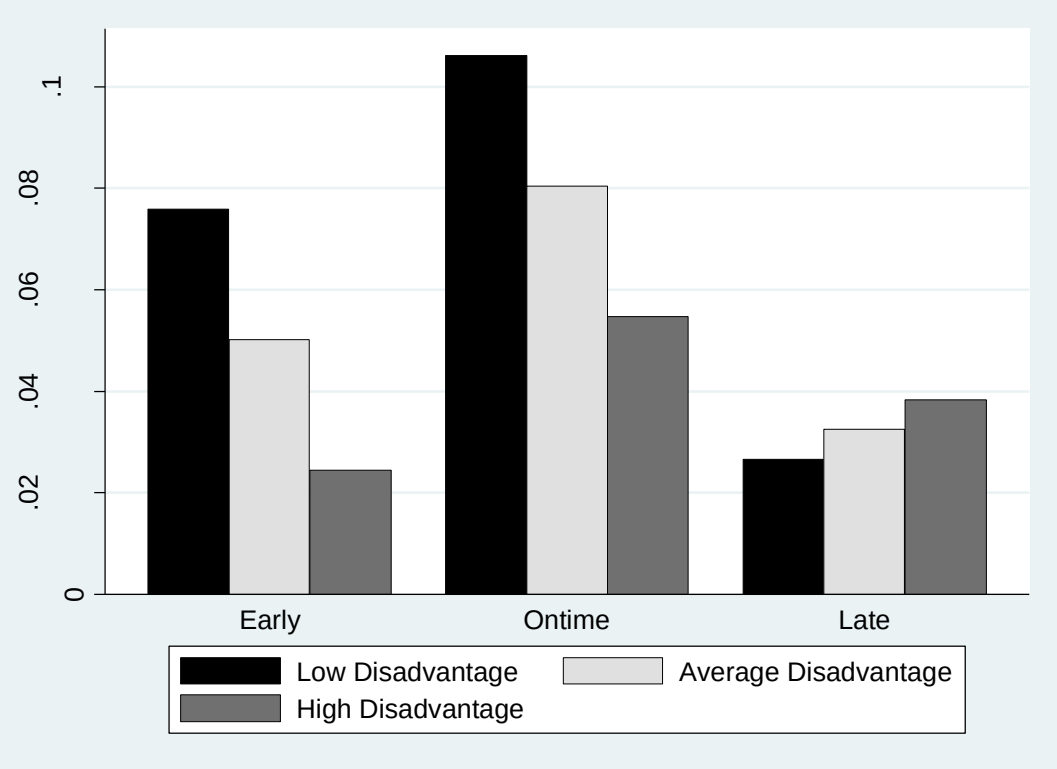

Figure 4.1. Predicted Wave III Alcohol Abuse Scores for All Girls, by Level of Concentrated Disadvantage and Pubertal Development

Note: All continuous covariates held at mean values and all categorical control variables held at modal values.

Results in Table 4.2 aggregated girls from various racial/ethnic backgrounds and therefore masked differences in the contextual specificity of risk/protection associated with pubertal development and neighborhood dislocations. Girls from different racial/ethnic backgrounds may not perceive pubertal changes in the body similarly if they have different reference groups with which to compare themselves. Further, the types of neighborhoods in which young girls live also likely vary by race/ethnicity, and thus operate differently as substance use risk factors. The remaining analyses compares black, Hispanic, and white adolescent girls to ascertain the moderating role of race/ethnicity in the relationships between pubertal development, neighborhood dislocations, and adolescent girls' substance use.

The Moderating Role of Race/Ethnicity

Tables 4.3 through 4.5 present coefficients and standard errors from survey adjusted regression models predicting the main effects of pubertal development and neighborhood characteristics on substance use for black, Hispanic, and white girls, respectively. The models predict each outcome at Wave I and Wave III using predictors measured at Wave I. Results for the alcohol use and alcohol abuse models (1st and 3rd panels) are coefficients and standard errors from ordinary least squares regression models; results for the tobacco use and marijuana use models (2nd and 4th panels) are from negative binomial regression models. All models control for race/ethnicity, age, substance-using peers, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood composition. The Wave III models additionally control for baseline level of substance use at Wave I.

As shown in Table 4.3, neither early pubertal development nor late pubertal development predicted substance use among black adolescent girls. This finding was consistent across all substance use outcomes and waves of data collection. Neighborhood dislocations, however, did occasionally influence black girls' substance use levels. Concentrated disadvantage was negatively related to marijuana use levels at Wave I, but by Wave III black girls who grew up in disadvantaged neighborhoods had higher predicted levels of alcohol and marijuana use. Social disorder also had a significant positive relationship with black girls' initial level of marijuana use (b=.59; p=.003), but did not significantly influence changes in marijuana use between Waves I and III (b=-.15; p=.66). Results in Table 4.3 therefore indicate that race/ethnicity does indeed moderate the main effects of pubertal development and neighborhood dislocations on adolescent girls' substance

use. Among black girls, pubertal development had no main effect on substance use, and concentrated disadvantage was associated with higher alcohol and marijuana use in late adolescence.

Results in Tables 4.4 and 4.5 additionally suggest that race/ethnicity moderates the effect of pubertal development and neighborhood dislocations on adolescent girls' substance use. As shown in Table 4.4, Hispanic girls who were early developers had higher predicted levels of alcohol use at both Waves I and III. Late development did not have a protective effect among Hispanic girls, however. Social disorder was the only neighborhood dislocation variable that influenced Hispanic girls' substance use, indicating that Hispanic girls living in neighborhoods characterized by high levels of social disorder had lower levels of alcohol use at Wave I.

Table 4.3. Coefficients and Standard Errors from Survey Adjusted Main Effects Models Predicting Substance Use for Black Girls, by Type of Substance and Wave of Data Collection[ab]

	Alcohol Use		Tobacco Use		Alcohol Abuse		Marijuana Use	
	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III
Early development	.08	-.01	.03	.10	.01	.00	-.28	.33
	(.09)	(.09)	(.43)	(.32)	(.02)	(.02)	(.47)	(.36)
Late development	-.04	-.03	-.28	-.28	.00	.02	-.95	-.35
	(.07)	(.11)	(.47)	(.29)	(.01)	(.03)	(.57)	(.43)
Concentrated disadvantage	.01	.09*	-.06	.19	.01	.00	-.33*	.38**
	(.04)	(.04)	(.26)	(.12)	(.01)	(.01)	(.15)	(.15)
Residential instability	.26	- .54	1.21	-.59	.01	.00	1.16	- .93
	(.23)	(.31)	(1.33)	(1.06)	(.04)	(.11)	(.96)	(1.49)
Social disorder	-.04	.02	.27	-.11	.00	-.02	.59**	-.15
	(.06)	(.07)	(.27)	(.24)	(.01)	(.02)	(.20)	(.34)
Constant	1.69	2.00**	-2.75	4.16	-.17	.23	-1.61	4.71
	(.92)	(.76)	(5.15)	(2.15)	(.14)	(.13)	(.72)	(3.20)
N	947	782	945	777	947	779	946	779
F Statistic	6.85***	4.90***	11.70***	7.99***	3.83*	1.46	11.70***	7.54***
R-squared	.25	.09	--	--	.12	.05	--	--

Notes: [a]Standard errors in parentheses. Standard errors for negative binomial coefficients in parentheses for tobacco use and marijuana use models. [b]All models control for age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I.

* p<.05. ** p<.01. *** p<.001.

Table 4.4. Coefficients and Standard Errors from Survey Adjusted Main Effects Models Predicting Substance Use for Hispanic Girls, by Type of Substance and Wave of Data Collection[ab]

	Alcohol Use		Tobacco Use		Alcohol Abuse		Marijuana Use	
	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III
Early development	.35*	.50*	.55	-.13	.05	.00	.19	.51
	(.14)	(.25)	(.36)	(.41)	(.06)	(.05)	(.38)	(.50)
Late development	.03	-.19	-.41	-.22	-.01	-.03	-.23	.11
	(.08)	(.16)	(.35)	(.41)	(.03)	(.03)	(.62)	(.34)
Concentrated disadvantage	.07	-.07	-.16	.13	.01	.00	.41	-.15
	(.08)	(.12)	(.29)	(.24)	(.02)	(.03)	(.29)	(.28)
Residential instability	- .09	- .33	.09	-.58	-.01	.04	-.44	-.18
	(.25)	(.47)	(.65)	(.92)	(.11)	(.11)	(1.00)	(1.13)
Social disorder	-.19*	-.07	.37	.05	-.02	-.02	-.04	.02
	(.08)	(.12)	(.24)	(.31)	(.04)	(.02)	(.26)	(.23)
Constant	1.22	2.11	-.56	.37	.24	.28	-2.34	-2.60
	(.76)	(1.50)	(2.73)	(3.13)	(.28)	(.22)	(3.04)	(3.26)
N	669	505	669	503	669	503	669	503
F Statistic	32.39***	2.74**	6.98***	13.48***	7.72***	1.79*	8.52***	3.75***
R-squared	.32	.09	--	--	.15	.05	--	--

Notes: [a]Standard errors in parentheses. Standard errors for negative binomial coefficients in parentheses for tobacco use and marijuana use models. [b]All models control for age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I.

* p<.05. ** p<.01. *** p<.001.

Among white adolescent girls, early development predicted lower levels of alcohol use at Wave III. Late pubertal development had a protective effect on white adolescent girls' initial levels of alcohol use, and changes in tobacco use and alcohol abuse over time. These main effects appear to be driving the pattern of results reported in Table 4.2 among the aggregated sample of girls. In terms of neighborhood dislocations, concentrated disadvantage was negatively related to white adolescent girls' alcohol use and abuse at Wave III (again, driving the results shown in Table 4.2). So, white adolescent girls living in more advantaged neighborhoods had higher levels of alcohol use/abuse. Residential instability also predicted higher initial levels of tobacco use and marijuana use, but did not influence changes in white girls' substance use over time. Finally, social disorder was negatively related to white girls' initial tobacco use, but did not influence any other outcomes. Results from Tables 4.3 through 4.5 therefore show that aggregating girls across race/ethnicity masks variation in the substance use risk/protection associated with pubertal development and neighborhood dislocations. The risk/protection associated with pubertal development was most pronounced for white and Hispanic girls' alcohol use/abuse, whereas neighborhood dislocations were risky for black girls' long-term substance use but protective for white girls' long-term substance use.

Table 4.5. Coefficients and Standard Errors from Survey Adjusted Main Effects Models Predicting Substance Use for White Girls, by Type of Substance and Wave of Data Collection[ab]

	Alcohol Use		Tobacco Use		Alcohol Abuse		Marijuana Use	
	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III	Wave I	Wave III
Early development	-.02	-.18*	.05	-.07	-.01	-.04	-.07	.04
	(.07)	(.09)	(.11)	(.14)	(.02)	(.03)	(.16)	(.19)
Late development	-.12*	-.01	-.26	-.28*	-.02	-.07***	-.20	.07
	(.05)	(.10)	(.17)	(.13)	(.02)	(.02)	(.26)	(.17)
Concentrated disadvantage	-.04	-.19***	.06	-.01	-.01	-.04**	.09	.10
	(.05)	(.05)	(.05)	(.06)	(.01)	(.01)	(.11)	(.08)
Residential instability	.19	.10	.66*	-.52	.05	.02	1.23**	-.42
	(.18)	(.29)	(.32)	(.35)	(.06)	(.08)	(.47)	(.46)
Social disorder	-.03	-.01	-.21*	.02	.00	.02	-.26	.02
	(.06)	(.09)	(.09)	(.10)	(.01)	(.02)	(.13)	(.10)
Constant	.53	.86	-1.00	3.74***	-.15	.00	-4.33***	2.94*
	(.46)	(.79)	(.97)	(.86)	(.13)	(.19)	(1.32)	(1.21)
N	2,634	2,196	2,629	2,186	2,636	2,195	2,629	2,182
F Statistic	29.40***	5.98***	51.36***	18.52***	17.03***	2.83***	32.20***	3.85***
R-squared	.38	.07	--	--	.20	.03	--	--

Notes: [a]Standard errors in parentheses. Standard errors for negative binomial coefficients in parentheses for tobacco use and marijuana use models. [b]All models control for age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I.

* p<.05. ** p<.01. *** p<.001.

The models in Tables 4.3 through 4.5 are main effects models only, and do not show whether neighborhood dislocations similarly moderate the relationship between pubertal development and substance use among black, Hispanic, and white girls. To examine these three-way interactions, multiplicative interaction terms were estimated for the six different combinations of pubertal development and neighborhood dislocations for black, Hispanic, and white girls. The models shown in Tables 4.3 through 4.5 were each replicated six times with the addition of one multiplicative interaction term. For the models with multiplicative interaction terms significant at the .05 level, joint linear combination coefficients and standard errors were calculated estimating the effect of early/late pubertal development on the substance use outcome for low, average, and high levels of the specified neighborhood dislocations. "Low" estimates were calculated at one standard deviation below the sample mean, "average" estimates at the mean, and "high" estimates at one standard deviation above the sample mean value.

Table 4.6 shows the joint linear combination coefficients and standard errors for the models with significant interaction effects predicting substance use among black girls. These joint linear combination coefficients represent the coefficients for early and late pubertal development for girls at low, average, and high levels of the neighborhood dislocations to illustrate the three level interactions between pubertal development, neighborhood dislocations, and race/ethnicity.[34] The top half of Table 4.6 shows that the effect of early pubertal development on black girls' tobacco use at Wave I was conditional upon level of

[34] For instance, in the model predicting tobacco use with a multiplicative interaction term between early pubertal development and residential instability: early development coefficient = -3.36; interaction coefficient = 6.00; mean residential instability = .38; standard deviation residential instability = .16. The joint linear combination coefficients at one standard deviation below the mean of residential stability = [-3.36 + .22(6.00) = -2.04]; at mean of residential instability = [-3.36 + .38(6.00) = -1.08]; and at one standard deviation above the mean of residential stability = [-3.36 + .54(6.00) = -.12].

residential instability as well as social disorder. Black early developers had consistently lower levels of tobacco use than on-time developers (b=-2.04; b=-1.08; b=-.12 at low, average, and high levels of residential instability), but tobacco use among early developers increased with residential instability. So for instance, the predicted rate of tobacco use among early developing black girls was .01 for girls living in neighborhoods with low levels of residential instability, .06 at average levels, and .55 at high levels. Conversely, black early developers living in neighborhoods with low levels of social disorder had the highest level of tobacco use at Wave I; tobacco use among these early developers decreased as social disorder increased. Figure 4.2 shows this interaction, illustrating that although tobacco use among black girls increased with social disorder among on-time and late developers, the opposite trend occurred among early developers. So, the neighborhood dislocation variable of social disorder actually ameliorated or lessened the substance use risk associated with early development. Or stated differently (and given the lack of significant main effects in Table 4.3), early pubertal development was only risky for tobacco use among black girls who lived in neighborhoods with low levels of social disorder or high levels of residential instability.

The bottom half of Table 4.6 shows that the effect of late pubertal development on black girls' substance use also varied by level of residential instability and social disorder. Late pubertal development was protective for black girls' marijuana use at Wave III only for girls living in residentially unstable neighborhoods, but was risky for girls living in residentially stable neighborhoods. Similarly, late pubertal development was protective for black girls' alcohol use at Wave I only for those living in highly disordered neighborhoods, and was risky for girls living in neighborhoods with low levels of social disorder. In sum,

residential stability magnified the risk of both early and late pubertal development on black girls' tobacco and marijuana use, whereas social disorder lessened the risk of early and late pubertal development on black girls' alcohol and tobacco use.

Results for Hispanic girls indicate that neighborhood dislocations decreased the substance use protection associated with late pubertal development. As shown in the bottom half of Table 4.7, late developing Hispanic girls had lower levels of alcohol use than on-time developers if they lived in neighborhoods with lower levels of disadvantage and disorder. The protective effect of late development was eroded by neighborhood dislocations, however, in that late developing Hispanic girls living in neighborhoods with high levels of disadvantage and social disorder used more alcohol than on-time developers. As shown in Figure 4.3, Hispanic girls' predicted alcohol use increased with neighborhood concentrated disadvantage. Although this positive relationship was consistent across all three categories of pubertal development it was most pronounced among late developers; late developers living in neighborhoods with high levels of concentrated disadvantage had higher predicted alcohol use scores than on-time developers. A similar pattern emerged for the moderating role of social disorder. The only exception to this pattern was for marijuana use and residential instability, where late development was only protective for Hispanic girls living in residentially unstable neighborhoods (similar to the finding for black girls in Table 4.6). Thus, with the exception of marijuana, neighborhood dislocations eroded the substance use protection associated with late pubertal development among Hispanic girls.

Table 4.6. Joint Linear Combination Coefficients and Standard Errors from Survey Adjusted Multiplicative Interaction Models Predicting Substance Use for Black Girls, by Level of Neighborhood Dislocation, Pubertal Development, Type of Substance, and Wave of Data Collection[abcd]

		Concentrated Disadvantage			Residential Instability			Social Disorder		
Early Development		Low	Avg	High	Low	Avg	High	Low	Avg	High
Alcohol use,	Wave I									
	Wave III									
Tobacco use,	Wave I				-2.04** (.69)	-1.08* (.43)	-.12 (.32)	1.66*** (.32)	.25 (.25)	-1.15** (.36)
	Wave III									
Alcohol abuse,	Wave I									
	Wave III									
Marijuana use,	Wave I									
	Wave III									
Late Development		Low	Avg	High	Low	Avg	High	Low	Avg	High
Alcohol use,	Wave I							.23 (.15)	.04 (.09)	-.16* (.08)
	Wave III									
Tobacco use,	Wave I									
	Wave III									
Alcohol abuse,	Wave I									
	Wave III									
Marijuana use,	Wave I									
	Wave III				2.43*** (.63)	.72 (.42)	-.99 (.57)			

Notes: [a]Standard errors for linear combinations in parentheses. [b]All models control for age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I. [c]Results presented only for interaction terms significant at the .05 level. [d]Low, average, high levels of neighborhood dislocations defined as one standard deviation below the mean, the mean, and one standard deviation above the mean value in the total analytic sample.
* p<.05. ** p<.01. *** p<.001.

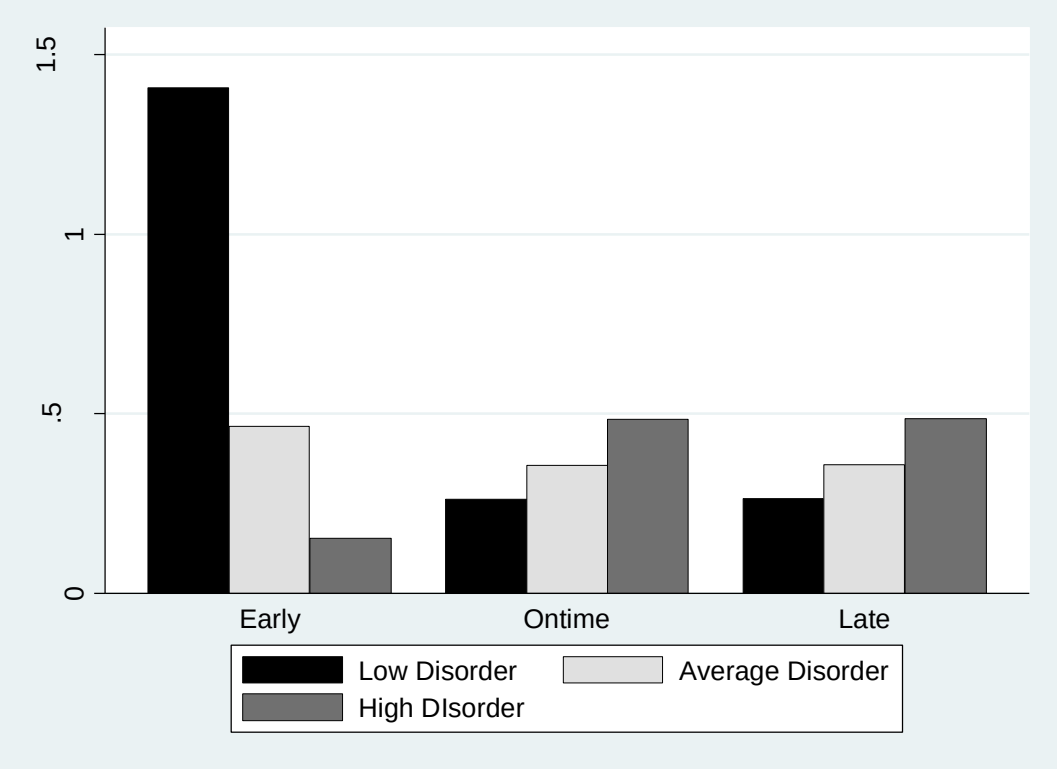

Figure 4.2. Predicted Wave I Tobacco Use Rates for Black Girls, by Level of Social Disorder and Pubertal Development

Note: All continuous covariates held at mean values and all categorical control variables held at modal values.

Table 4.7. Joint Linear Combination Coefficients and Standard Errors from Survey Adjusted Multiplicative Interaction Models Predicting Substance Use for Hispanic Girls, by Level of Neighborhood Dislocation, Pubertal Development, Type of Substance, and Wave of Data Collection[abcd]

		Concentrated Disadvantage			Residential Instability			Social Disorder		
Early Development		Low	Avg	High	Low	Avg	High	Low	Avg	High
Alcohol use,	Wave I									
	Wave III									
Tobacco use,	Wave I									
	Wave III									
Alcohol abuse,	Wave I									
	Wave III									
Marijuana use,	Wave I									
	Wave III									
Late Development		Low	Avg	High	Low	Avg	High	Low	Avg	High
Alcohol use,	Wave I	-.14 (.11)	-.03 (.08)	.09 (.09)				-.13 (.12)	.03 (.08)	.20* (.10)
	Wave III	-.48* (.24)	-.27 (.17)	-.05 (.15)						
Tobacco use,	Wave I									
	Wave III									
Alcohol abuse,	Wave I									
	Wave III									
Marijuana use,	Wave I				1.13* (.55)	.23 (.52)	-.67 (.74)	-1.27 (.83)	-.35 (.60)	.56 (.45)
	Wave III									

Notes: [a]Standard errors for linear combinations in parentheses. [b]All models control for age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I. [c]Results presented only for interaction terms significant at the .05 level. [d]Low, average, high levels of neighborhood dislocations defined as one standard deviation below the mean, the mean, and one standard deviation above the mean value in the total analytic sample.
* p<.05. ** p<.01. *** p<.001.

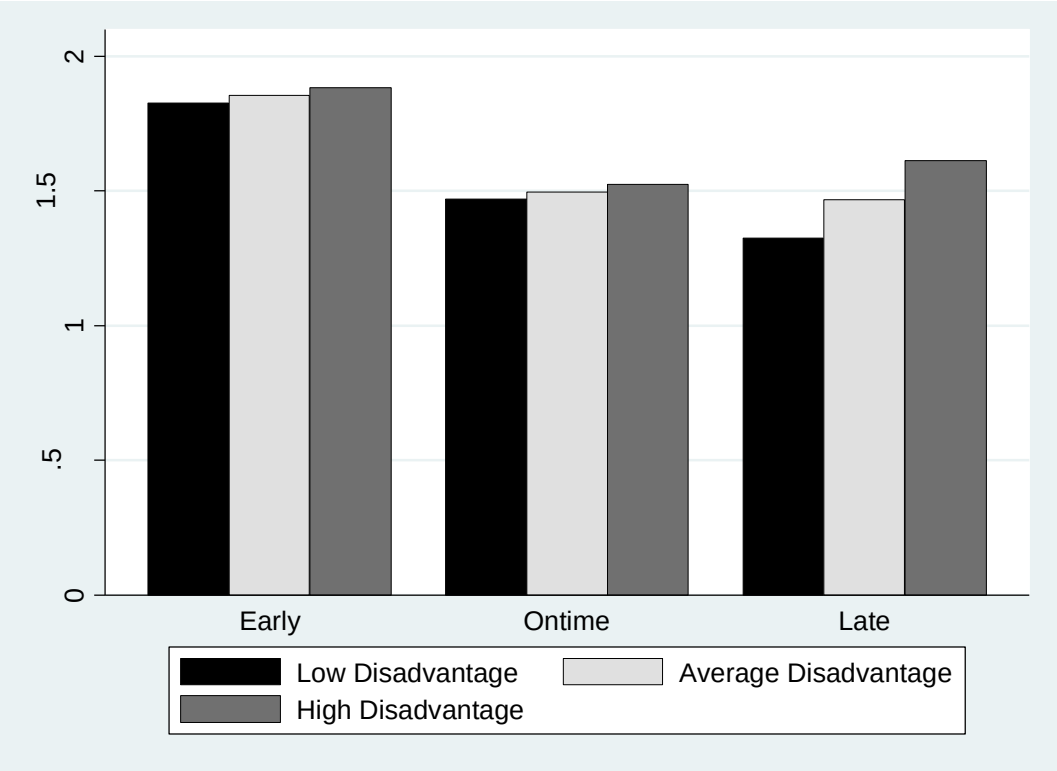

Figure 4.3. Predicted Wave I Alcohol Use Scores for Hispanic Girls, by Level of Concentrated Disadvantage and Pubertal Development

Note: All continuous covariates held at mean values and all categorical control variables held at modal values.

Table 4.8 shows the joint linear combination coefficients and standard errors for the models with significant interaction effects predicting substance use among white girls. These results indicate a pattern similar to the findings for black girls. Early development was most risky for white girls' substance use in relatively advantaged, stable, and orderly neighborhoods. Similar to the interaction graphed in Figure 4.2 for black girls, early developing white girls living in neighborhoods with high levels of disorder had lower levels of substance use than on-time developers. For instance, the predicted marijuana use rates for

early developing white girls living in neighborhoods with low, average, and high levels of concentrated disadvantage were .23, .14, and .08, respectively (see Figure 4.4). Neighborhood dislocations generally had little impact on the substance use protection associated with late pubertal development among white girls, but one model indicated that late pubertal development was only protective against white girls' initial levels of alcohol abuse for those living in disadvantaged neighborhoods.

The following discussion section summarizes and interprets the pattern of findings for the relationships between pubertal development, neighborhood dislocations, race/ethnicity, and adolescent girls' substance use, drawing on the concepts of pseudomaturity and reference groups as they relate to the population specific substance use risk associated with pubertal development.

Table 4.8. Joint Linear Combination Coefficients and Standard Errors from Survey Adjusted Multiplicative Interaction Models Predicting Substance Use for White Girls, by Level of Neighborhood Dislocation, Pubertal Development, Type of Substance, and Wave of Data Collection[abcd]

		Concentrated Disadvantage			Residential Instability			Social Disorder		
Early Development		Low	Avg	High	Low	Avg	High	Low	Avg	High
Alcohol use,	Wave I	.04 (.07)	-.06 (.07)	-.17 (.09)						
	Wave III									
Tobacco use,	Wave I									
	Wave III	.08 (.15)	-.14 (.14)	-.37 (.20)	.24 (.18)	-.06 (.14)	-.37 (.20)	.25 (.16)	-.06 (.11)	-.38* (.18)
Alcohol abuse,	Wave I				.10 (.06)	.06 (.04)	.02 (.02)			
	Wave III									
Marijuana use,	Wave I	.15 (.18)	-.25 (.18)	-.64* (.32)						
	Wave III									
Late Development		Low	Avg	High	Low	Avg	High	Low	Avg	High
Alcohol use,	Wave I									
	Wave III									
Tobacco use,	Wave I									
	Wave III									
Alcohol abuse,	Wave I	.00 (.02)	-.03* (.01)	-.05** (.02)						
	Wave III									
Marijuana use,	Wave I									
	Wave III									

Notes: [a]Standard errors for linear combinations in parentheses. [b]All models control for age, substance-using friends, parental caring, parental closeness, parental education, collective efficacy, urbanicity, region, and neighborhood racial composition; Wave III models control for baseline substance use at Wave I. [c]Results presented only for interaction terms significant at the .05 level. [d]Low, average, high levels of neighborhood dislocations defined as one standard deviation below the mean, the mean, and one standard deviation above the mean value in the total analytic sample.
* p<.05. ** p<.01. *** p<.001.

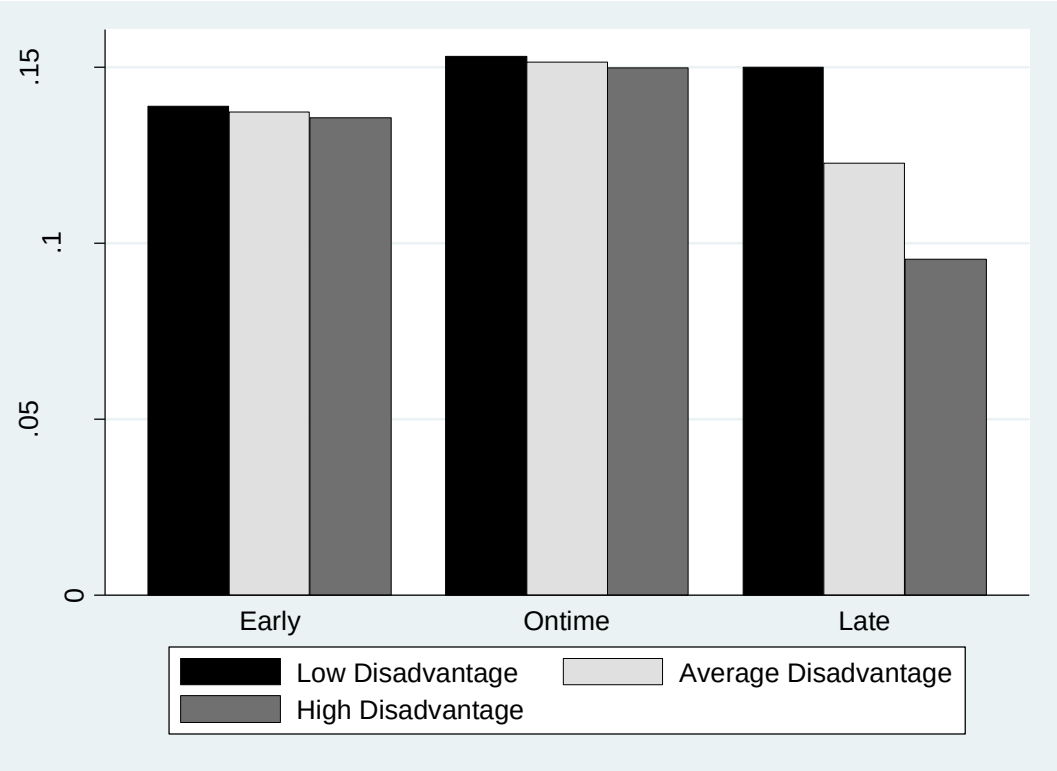

Figure 4.4. Predicted Wave I Alcohol Abuse Scores for White Girls, by Level of Concentrated Disadvantage and Pubertal Development

Note: All continuous covariates held at mean values and all categorical control variables held at modal values.

DISCUSSION AND CONCLUSIONS

This study examined the complex interactions between pubertal development, neighborhood dislocations, race/ethnicity, and adolescent girls' substance use. By emphasizing contextual sensitivity of substance use risk factors, this study contributes to the growing body of literature examining interactive effects of individual and neighborhood level risk factors for health outcomes. Overall, results indicated that aggregating girls from different racial/ethnic backgrounds masked the variation in substance use risk and protection

associated with pubertal development and neighborhood dislocations. Models with all girls combined suggested that pubertal development was most predictive of alcohol use and abuse, that residential instability was the only important neighborhood dislocation in predicting substance use, and that the effect of pubertal development on substance use was not conditional upon level of neighborhood dislocations (see *H1*). Results from split sample models for black, Hispanic, and white adolescent girls revealed a very different but interesting pattern of findings.

First, and in support of *H2* and *H3*, the main effects of pubertal development and neighborhood dislocations on substance use varied for black, Hispanic, and white adolescent girls. For example, pubertal development had no significant effect on black adolescent girls' substance use. Early pubertal development was risky only for Hispanic adolescent girls, whereas late pubertal development was protective only for white girls. This suggests that previous research relying on samples of primarily white adolescents may have overstated the risk/protection associated with pubertal development. A somewhat unexpected finding, however, was that white girls who were early developers had significantly lower levels of alcohol use in late adolescence compared to on-time developers. Although this finding is contrary to both the off-timing and early-timing hypotheses, it indicates a possible "catch-up" effect previously documented in the literature (e.g., Dick et al. 2000; Stattin and Magnusson 1990). Early developers may initially be at higher risk of substance use during early adolescence, but over the course of adolescence on-time and late developers begin drinking alcohol as is developmentally normative. It is likely that this catch-up effect only applies to alcohol, as alcohol use is what becomes most normative when adolescents transition from adolescence to early adulthood (Schulenberg and Maggs 2001). Due to data limitations it is

impossible to explore whether this catch-up effect persists into later adulthood, and thus more research is needed to examine these long-term health effects associated with pubertal development.

Results also indicated that the effects of concentrated disadvantage, residential instability, and social disorder on substance use varied for black, Hispanic, and white adolescent girls (see *H3*). Neighborhood dislocations had few main effects on Hispanic girls' substance use, but were most risky for black girls, and somewhat protective for white girls. Results for black girls therefore support findings from previous research that indicate neighborhood structural characteristics may be more influential for the health and well-being of girls of color (Crane 1991; Hogan and Kitagawa 1985; Kulis et al. 2007). On the other hand, the finding that alcohol use in late adolescence was higher for white girls who grew up in more advantaged neighborhoods may be linked to the associations between neighborhood affluence, college attendance, and alcohol use. In general, college-bound students use less alcohol during high school than non-college bound students, but then have much higher rates of alcohol and marijuana use when in college (Bachman et al. 2002; Schulenberg and Maggs 2001). So the negative relationship between neighborhood disadvantage and alcohol use for white girls at Wave III may be in part due to these girls' educational trajectories.

Finally, and perhaps of most import, this study shows that the moderating effect of neighborhood dislocations on the relationship between pubertal development and substance use varied by race/ethnicity (see *H4*). Among Hispanic adolescent girls there was a pattern of 'double-jeopardy' or cumulative risk such that neighborhood dislocations eroded the substance use protection associated with late pubertal development. This is consistent with other literatures indicating that cumulative risk factors at various ecological levels are

detrimental to numerous health outcomes (Brooks-Gunn and Warren 1985; Caspi et al. 1993; Dick et al. 2000; Ge et al. 2002; Leventhal and Brooks-Gunn 2000; Obeidallah et al. 2004; Stockdale et al. 2007). Among black and white adolescent girls, however, results suggested an alternative pattern of population specific risk, or what might be termed "advantaged" risk. Rather than neighborhood dislocations exacerbating and/or eroding individual level risk/protective factors, girls residing in relatively advantaged or affluent neighborhoods were at greater risk for substance use. Although this effect might be conceptualized as a type of resilience, such that black and white girls with both individual and neighborhood level risk factors were resilient to such risk, it may be more appropriately framed as an issue of contextualized, or population specific risk. Specifically, and building upon findings from the main effects models, pubertal development was influential for adolescent girls' substance use only in specific contexts: it was a risk factor largely relevant only for white and/or affluent adolescent girls. Why might pubertal development be important only for the advantaged?

Two possible social-psychological explanations for the population specific or "advantaged" risk associated with pubertal development relate to the concepts of pseudomaturity and reference groups. Historical, social, and economic changes in the last century have lengthened the period of adolescence and delayed the transition to adulthood and its associated role transitions related to career and family formation (Crockett 1999). Within the contemporary United States, pubertal development (and particularly early or asynchronous development) thus uniquely challenges youth as they adapt to this developmental transition and shifts in role expectations. Theorists of adolescent development have proposed three clusters of adolescent maturity: pseudomature, immature, and mature (Galambos et al. 2003; Tilton-Weaver, Vitunski, and Galambos 2001). Pseudomature

adolescents, sometimes referred to as adultoid adolescents, are those who have attained social maturity (i.e., generally as related to autonomy) but lack psychological maturity. Immature adolescents lack both social and psychological maturity whereas genuinely mature adolescents have both. Early pubertal developers characterized by pseudomaturity may thus be more likely to use substances like tobacco and alcohol as a route to adulthood to seemingly bridge the gap between their physical and social psychological maturity (Moffitt 1993). Adolescent girls living in affluent, advantaged neighborhoods may be more likely to be pseudomature given the lack of structural constraints imposed upon their daily routines and hence maturity level. For instance, due to possible economic strains within the family, early developing girls in less affluent neighborhoods may be expected to help with family chores, child-rearing, and even contribute to the family's expenses through employment earnings. These responsibilities would likely engender genuine maturity as a girl learned to handle new adult roles and responsibilities (Tilton-Weaver et al. 2001). In contrast, girls in affluent neighborhoods may be free from such expectations and therefore more likely to be pseudomature and engage in seemingly mature behaviors (e.g., smoking, drinking) while delaying psychological maturity.

Thus perhaps the reason that early development is most risky for white and black adolescent girls living in affluent neighborhoods is these girls' pseudomaturity. However, given racial/ethnic differences in the timing of pubertal development, it is possible that pseudomaturity accounts for this effect only among white girls. For black girls, the risk associated with early pubertal development in affluent neighborhoods may instead be tied to reference groups. In general, black girls reach puberty earlier than white girls (Herman-Giddens et al. 2004), and so "early" pubertal development as it is defined in the current study

(i.e., relative to the entire sample of adolescent girls) may not generally be risky for black girls if their reference groups are composed of black peers who are also developing early. As such, early developing black girls may not be perceived, or perceive themselves to be, physically or socially deviant from same-age peers. Given racial segregation patterns in residence, black girls in affluent neighborhoods may have fewer black peers and thus have a primarily white (i.e., later developing) reference group of peers. So, early pubertal development may only be risky for black girls' substance use if they live in affluent neighborhoods where early changes in body development greatly distance them from same-age peers.[35]

Study Implications

This study's findings of population specific risk as it relates to pubertal development, neighborhood dislocations, and adolescent girls' substance use must be understood within a larger framework of structural disadvantage and the transition to adulthood. For instance, some might interpret these results as evidence that living in a disadvantaged neighborhood may actually be good for black and white adolescent girls, given that a disadvantaged neighborhood context appears to reduce the substance use risk associated with pubertal development. However, such a conclusion would be unwarranted and intellectually reckless. Neighborhood disadvantage is overwhelmingly linked with lower levels of physical, mental,

[35] Another argument, related to more biological and physiological, rather than social psychological mechanisms may relate to the associations between socioeconomic status, body weight, and pubertal timing. Adolescent girls' body weight is negatively correlated with socioeconomic status (Delva, O'Malley and Johnston 2006), and the onset and maintenance of puberty, particularly menstruation, are related to body weight (Frisch and Revelle 1970; Kaplowitz et al. 2001).So it is possible that girls living in less affluent neighborhoods may have higher body weights, and therefore experience puberty earlier. If "early" development is normative for these girls, then it may lack salience as a risk factor given girls' reference groups. Conversely, girls living in more affluent neighborhoods may weigh less, experience puberty later, and therefore "early" developers would be more distanced from same-age peers.

and social well-being (Aneshensel and Sucoff 1996; Baumer and South 2001; Morenoff et al. 2007), not to mention harmful to individuals' life chances related to educational and occupational opportunities as well as residential mobility. Rather, the findings from this study should be taken only as evidence that adolescent girls living in disadvantaged neighborhoods may be protected from certain risk factors given expectations of role transitions that are necessitated by structural inequities, whereas girls in more affluent neighborhoods experience magnified risk due to states of pseudomaturity or competing reference groups.

A larger theoretical as well as methodological implication of the current study is that concepts of risk, protection, and resilience are contextually specific. Literature focusing on risk associated with pubertal development (whether for substance use, mental health, sexual activity, delinquency, victimization, etc.) is expanding. But the risk associated with early pubertal development may have been overstated in the literature due to a reliance on solely or primarily white, relatively affluent, participants. Pubertal development may indeed be an important risk factor for substance use among adolescent girls, but not universally. This is not to say all research on the topic should cease, but rather that researchers should be aware of the contextual specificity of this risk when conducting research. Such knowledge will be useful to targeted or secondary prevention programs aimed at addressing risk associated with pubertal development. Otherwise, if research devoid of context is used to inform universal preventive intervention programs, biased findings may waste valuable research monies better spent on targeted interventions, or more importantly, universal risk/protective factors with larger effect sizes (e.g., affiliation with substance using peers).

Limitations and Directions for Future Research

There are several limitations to the current study that should be addressed in future research. First, for data security issues the Add Health does not include unique neighborhood level identification codes, precluding multilevel analyses or standard error adjustments for neighborhood clusters. Although it is possible that the standard errors reported herein may be underestimated, the standard error adjustments for the complex survey design would likely minimize any such bias. Future studies examining the interactivity between individual and neighborhood level characteristics using multilevel models can contribute to the literature by modeling contextual effects of pubertal development, and perhaps examining neighborhood level pubertal development effects as they relate to the issue of reference groups mentioned in the prior section. Further, due to data limitations, the current study used census block groups as neighborhoods. We would not expect census block groups to perfectly overlap with actual neighborhoods or communities. Therefore, continued research is needed to replicate these findings using more meaningful community boundaries as identified by city residents or local officials.

The current study, grounded in the social ecological model, examined the interactions between pubertal development, neighborhood dislocations, race/ethnicity, and adolescent girls' substance use, but did not focus on mediational models to explain the social processes driving these conditional effects. Following other research in the neighborhood effects literature (Baumer and South 2001; Chuang et al. 2005; Sampson, Morenoff, and Gannon-Rowley 2002), future studies are needed to examine the complex causal pathways and social processes that might explain why the risk of pubertal development is most salient for white and/or affluent adolescent girls. For instance, an interesting line of research would employ

in-depth interviews to examine adolescent girls' lived experiences of puberty and how their reference groups vary across neighborhood contexts, and the role of reference groups in the relationship between pubertal development and substance use.

Finally, the results of this study indicated that Hispanic girls were subject to cumulative risk such that living in neighborhoods with high levels of dislocation eroded the substance use protection associated with late pubertal development. However, due to small sample sizes it was not possible to disaggregate Hispanic girls into other, more meaningful ethnic groups such as Mexican Americans, Puerto Ricans, and Cubans. Aggregating girls of Hispanic ethnicity masks the potential variation in effects among these quite diverse groups of young girls. Thus in the interest of understanding the population specificity of substance use risk, more research is needed to examine these effects among disaggregated groups of Hispanic girls.

Continued research is needed to propel contextually sensitive understandings of risk, protection, and resilience in the social epidemiological literature on adolescent substance use. Investigating the etiology of substance use among adolescents who have been historically underrepresented in empirical research, and recognizing the importance of larger social ecological contexts is crucial for the advancement of the field. By understanding when, where, and for whom risk is relevant, researchers will maximize their contributions to applied settings and inform the design of effective substance use preventive intervention programs.

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CHAPTER V

SUMMARY AND SYNTHESIS

This chapter summarizes findings from the three studies, outlines their theoretical and policy implications, reviews limitations and directions for future research, and concludes with a discussion of the larger significance of the dissertation.

DISCUSSION

Summary of Findings

The three studies in this dissertation contextualized and unpacked the substance use risk associated with adolescent girls' pubertal development. Chapter 2 examined the short- and long-term associations between early pubertal development and substance use among adolescent girls, and whether body weight moderated this relationship. Developmental and identity theories predicted that the developmental mismatch associated with early pubertal development would be most risky for under- and average-weight girls. Although cross-sectional results indicated that early pubertal development predicted a higher number of substances tried and heavy substance use for all adolescent girls, lagged longitudinal models suggested that this relationship was attenuated after controlling for baseline levels of substance use. Results did not consistently support predictions that over-weight girls would be protected from the substance use risk associated with early pubertal development. Early pubertal development was a significant predictor of substance use for average-weight girls in

cross-sectional models, but significant only for over-weight girls in lagged longitudinal models. However, tests for moderation indicated that the substance use risk associated with early pubertal development was not significantly different for under-, average, and over-weight adolescent girls. So the first study indicated that the substance use risk associated with pubertal development only varied slightly by adolescent girls' body weight. Further, as one of the first empirical studies in the United States to examine the long-term effect of pubertal development on adolescent girls' substance use, results indicated that the substance use risk associated with pubertal development was most pronounced during a short developmental window in early adolescence.

Chapter 3 focused on potential explanatory mechanisms that might mediate the relationship between pubertal development and adolescent girls' substance use. Feminist social interaction theory predicted that weight dissatisfaction and self-esteem were two intrapersonal self-appraisals that might mediate this relationship, because both are responses to reflected appraisals associated with the devalued female pubertal body. Social development theory predicted that two potential social relationship mediators were autonomy from parents and deviant peer association, both as potential agents of socialization. Results indicated that the explanatory power of these mediators depended on the operational definitions of pubertal development and substance use, as well as timing of the substance use outcome. For instance, the effect of body development on alcohol use during early adolescence was partially attenuated by the mediators, and the effect of body development on tobacco use during early adolescence was completely attenuated by substance-using friends. However, the effect of age at menarche on alcohol use was unaffected by the theoretically derived mediators. The second study thus suggested that the substance use risk associated

with pubertal development among adolescent girls was partially explained by social relationships (largely substance-using friends) during early adolescence. Although weight dissatisfaction, self-esteem, and autonomy from parents were important correlates of pubertal development and adolescent girls' substance use, they were not important mediators of this relationship.

Chapter 4 examined the interactions between pubertal development, neighborhood dislocations, race/ethnicity, and adolescent girls' substance use. Drawing on the social ecological model, the influence of pubertal development on adolescent girls' substance use was predicted to vary across different neighborhood contexts, as well as by race/ethnicity. Results indicated that the substance use risk associated with pubertal development and neighborhood dislocations varied for non-Hispanic black, Hispanic, and non-Hispanic white adolescent girls. Pubertal development was influential for substance use among black adolescent girls residing in affluent, advantaged neighborhoods. Early and late pubertal development were generally associated with higher substance use among black girls living in affluent neighborhoods. Conversely, early pubertal development was risky for Hispanic girls' alcohol use, and neighborhood dislocations eroded the substance use protection associated with late pubertal development among Hispanic girls. Finally, early pubertal development was most risky for white adolescent girls residing in affluent, advantaged neighborhoods. Results from the third study therefore suggested that pubertal development is a population specific risk factor and its effect on adolescent girls' substance use depends on girls' neighborhood contexts as well as their racial/ethnic background.

Theoretical Implications

By examining the substance use risk associated with pubertal development among adolescent girls, this dissertation makes several theoretical contributions to the fields of medical sociology and prevention science. Specifically, by highlighting how current theoretical explanations of the substance use risk associated with pubertal development are overly simplistic, this dissertation contributes to theory development in terms of causal pathways of risk, issues of population specificity and developmental specificity, and the importance of construct measurement.

First, theories of adolescent substance use risk should place greater emphasis on the pathways and processes that lead to risk. Stage termination and maturity gap theories are two principal theoretical frameworks used to describe the relationship between pubertal development and adolescent girls' substance use (Caspi and Moffitt 1991; Petersen and Taylor 1980; Stattin and Magnusson 1990). These theories suggest that early developing adolescents are at greater risk of behaviors such as substance use due to the mismatch between their biological and social maturity. Although these theories provide a broad explanatory framework for why pubertal development may be a substance use risk factor for adolescent girls, they do not fully explicate the social psychological and relational mechanisms that underlie the causal pathway of risk. Understanding the social mechanisms or pathways that connect risk factors with substance use outcomes is crucial if the ultimate goal of research is to inform substance use intervention programs. This is particularly true for the substance use risk associated with pubertal development, given that intervention programs cannot alter the course of puberty. Rather, only by identifying pathways that connect pubertal development to substance use risk will we gain knowledge about mediating

mechanisms that can be manipulated in intervention programs. The second study (Chapter 3) addressed this issue by examining the role of self-appraisals and social relationships in the pathway of substance use risk associated with adolescent girls' pubertal development. Because results provided only partial support for the explanatory role of social relationships with deviant peers, other studies should draw on these findings to continue examination of the mediating processes related to substance use risk factors such as pubertal development.

For instance, the second study yielded no support for predictions based on feminist social interaction theory that weight dissatisfaction mediated the relationship between pubertal development and adolescent girls' substance use. This finding, however, could be due to masked heterogeneity in perceptions of the pubertal body among adolescent girls in the analytic sample. For instance, many black women counteract stigmatization with the development of strong positive self-valuations and alternative beauty standards (Collins 1990; hooks 1993; Mama 1995). As a consequence, many black girls may be part of social networks that model positive evaluations of bodies failing to meet dominant societal beauty standards. Embedded in such social networks, some black girls may be less likely than Hispanic or white adolescent girls to endorse the ideal body type perpetuated by the 'cult of thinness' or 'tyranny of slenderness' implicated by feminist social interaction theory. Empirical research indeed suggests that black girls consistently report higher levels of body weight satisfaction, heavier ideal body sizes, less concern about weight, and fewer weight control behaviors than their Hispanic and white peers (Casper and Offer 1990; Kelly et al. 2005; Neff et al. 1997; Neumark-Sztainer et al. 2002; Parnell et al. 1996).

To further explore this lack of support for feminist social interaction theory in the second study, I conducted additional analyses to examine whether the mediating role of

weight dissatisfaction varied for girls from different racial/ethnic backgrounds. Using the analytic sample and variables from the second study, multiple group comparisons between generally weighted least squares structural equation models were used to test whether the causal pathways linking pubertal development, weight dissatisfaction, and alcohol use varied for black, Mexican-American, and white adolescent girls (i.e., a test for moderated mediation; see Knoke 2005; Muller, Judd, and Yzerbyt 2005).[36] Table 5.1 shows the unstandardized coefficients and standard errors from the structural equation models. The top half of the table presents coefficients from the measurement model and the bottom half shows coefficients from the structural model. The first column presents estimates for the multiple group model constraining the structural coefficients to be equal across the three racial/ethnic groups.[37] The second through fourth columns present estimates for the multiple group models allowing all coefficients to vary across racial/ethnic groups. All models controlled for age, standardized body mass index, substance-using friends, parental education, Southern region of residence, and urban area of residence.

The difference in nested chi-squared values was used to test for moderated mediation, or racial/ethnic variation in the mediating role of weight dissatisfaction. Results indicated that constraining the structural coefficients to be equal across the three racial/ethnic groups did not significantly worsen the model fit, and thus there was no statistical support for the hypothesized moderated mediation ($\Delta\chi^2_6 = 2.13$; $p = .91$). It should be noted, however, that the lack of support for moderated mediation could be due to low statistical power given the

[36] Additional models were estimated for cross-sectional models at Wave II and lagged longitudinal models at Wave III, as well as for tobacco use outcomes. Findings were substantively similar and thus omitted here in the interest of parsimony.

[37] The measurement model coefficients are not included in this model as they were allowed to vary across the three racial/ethnic groups. It should be noted, however, that a multi-group comparison of nested models testing for factorial invariance of the pubertal development, weight dissatisfaction, and alcohol use constructs across the three racial/ethnic groups also indicated no significant difference in the measurement models across the three groups.

small sample sizes for the black and Mexican-American subsamples. Indeed, examination of the direction and magnitude of the structural coefficients (see bottom panel of Table 5.1) indicate that the findings for all girls combined were largely driven by the results for white girls. With only one exception, pubertal development and weight dissatisfaction had no influence on black and Mexican American girls' alcohol use. These exploratory results are thus one example of an expansion of the findings from Chapter 3 that attempt to more fully explicate the mechanisms linking pubertal development with adolescent girls' substance use.

This leads to the second theoretical implication of the dissertation, which is that theories of substance use risk should account for potential population specificity of risk. As noted above, the lack of support for feminist social interaction theory in Chapter 3 could in fact be due to racial/ethnic heterogeneity in adolescent girls' social psychological responses to pubertal development. Weight dissatisfaction may actually explain why pubertal development is associated with white adolescent girls' substance use, but has little explanatory power for black and/or Mexican-American adolescent girls.

Table 5.1. Unstandardized Coefficients and Standard Errors for Multi-group Structural Equation Models: Add Health Wave I[abcd]

Parameter Estimates	All Girls (n = 5,289)	Black Girls (n = 1,418)	Mexican American Girls (n = 444)	White Girls (n = 3,427)
Measurement Model				
Pubertal Development → breast development		1.00	1.00	1.00
Pubertal Development → body curve development		1.01 (.06)***	.87 (.08)***	.85 (.03)***
Pubertal Development → physical development		.60 (.04)***	.59 (.06)***	.57 (.02)***
Weight Dissatisfaction → overweight perception		1.00	1.00	1.00
Weight Dissatisfaction → weight control behaviors		.77 (.10)***	.74 (.19)***	.56 (.08)***
Alcohol Use → alcohol use frequency		1.00	1.00	1.00
Alcohol Use → binge drinking frequency		.96 (.05)***	1.28 (.10)***	.97 (.02)***
Alcohol use → drunkenness frequency		.96 (.05)***	.91 (.07)***	.77 (.02)***
Structural Model				
Pubertal Development → Alcohol Use	-.05 (.02)*	-.03 (.04)	-.01 (.10)	-.06 (.03)*
Pubertal Development → Weight Dissatisfaction	.12 (.05)*	.11 (.09)	.27 (.43)	.11 (.05)*
Weight Dissatisfaction → Alcohol Use	.34 (.04)***	.13 (.07)	.27 (.12)*	.44 (.05)***
Indirect Effect of Pubertal Development → Alcohol Use	.04 (.02)*	.01 (.02)	.07 (.13)	.05 (.48)*

Notes: [a]Standard errors in parentheses. [b]All models control for age, standardized body mass index, substance-using friends, parental education, Southern region of residence, and urban area of residence. [c]Parameter estimates calculated with maximum likelihood estimation. [d]Multi-group model: Satorra-Benter Scaled $\chi^2(141) = 56.92$, $p = 1.00$; GFI = .95; RMSEA = .00; RMR = .04. Multi-group model constraining structural parameters to be equal: Satorra-Benter Scaled $\chi^2(147) = 59.05$, $p = 1.00$; GFI = .95; RMSEA = .00; RMR = .04; $\Delta\chi^2(6) = 2.13$; $p = .91$.

Universal theories of substance use risk are obviously appealing due to their conceptual simplicity and parsimony, but theorists and researchers alike should beware the masking of effects that can occur. Indeed, findings from the third study (Chapter 4) also suggested that the substance use risk associated with pubertal development is most pronounced for white adolescent girls, or black adolescent girls residing in affluent, advantaged neighborhoods. As such, predictions from stage termination and maturity gap theories are likely biased toward white and/or affluent populations. This bias reflects the historic and problematic trend of racial and class normativity in social theory development as well as in empirical research. Relying on small, regional, or convenience samples of primarily white, affluent adolescents to test theories of substance use risk not only masks variation of risk across different groups of adolescents, but also perpetuates a racial and class hegemony in the theoretical conceptualization of risk. By failing to acknowledge, or at least examine, potential group differences in the processes and effects of substance use risk factors, researchers may unwittingly perpetuate a type of colorblind social theory development that legitimates privileged discourses of risk. Theories of adolescent substance use should therefore be sensitive to the potential population specificity of substance use risk factors, and be explicit about a theory's (lack of) universality for different groups of adolescents.

Related to the issue of population specificity, a third theoretical contribution of the dissertation is the notion that many risk factors for substance use are developmentally specific (see also Hawkins, Catalano, and Miller 1992). Results from all three studies indicated that the substance use risk associated with pubertal development was most salient during a short developmental window in early adolescence, a time when sensitivity to bodily

changes may be heightened for adolescents. In its original presentation, stage termination theory did indeed highlight the developmental specificity of substance use risk associated with pubertal development by suggesting that this risk was magnified during early adolescence (Magnusson 1988; Stattin and Magnusson 1990). Unfortunately, other researchers' reliance on cross-sectional or retrospective data may have overstated the substance use risk associated with pubertal development during late adolescence and early adulthood. Although accounting for the developmental specificity of substance use risk factors may seem to be a methodological concern only, it is actually an important theoretical issue as well. If theories of adolescent substance use are to be effective informants for substance use intervention programs, they need to specify not only for whom risk factors are relevant (population specificity), but also when these risk factors are relevant and thus should be targeted by such programs (developmental specificity).

A final theoretical implication of this dissertation, and as others have noted before (Dorn et al. 2006), is the need for more theoretical clarity regarding construct measurement. For instance, how should researchers define pubertal development, particularly "early" development? Previous research has largely relied on measures of age at menarche or body development, the two of which are quite conceptually distinct. Menarche is a non-visible marker of pubertal development that can be hidden from others, whereas body development is a more noticeable marker of maturation likely to elicit changes in girls' self-perceptions. Exploratory factor analyses conducted on the analytic sample from the second study (Chapter 3) indicated that age at menarche was not empirically equivalent to the other measures of body development in the Add Health data (see Table 5.2). Further, results from the second study (Chapter 3) indicated that the substance use risk associated with "pubertal

development" was quite different when captured with measures of age at menarche versus body development. Thus more theoretical development is needed to clarify how these different measures of pubertal development operate as substance use risk factors, specifically focusing on their meanings to adolescent girls.

Table 5.2. Summary of Exploratory Factor Analysis and Reliability Analysis Results for Measures of Pubertal Development: Add Health Wave I (N = 4,088)

Item	Factor Loadings	Cronbach's α if item is deleted
Breast development	.80	.47
Body curve development	.72	.48
Physical development compared to peers	.44	.51
Age at menarche	-.15	.70
Eigenvalue	1.37	
Cronbach's α	.62	

Notes: Based on analytic sample from Chapter 3. Factor loadings calculated with maximum likelihood estimation.

Similarly, theories of adolescent substance use should be sensitive to potential differences in risk processes for various substance use outcomes such as tobacco, alcohol, marijuana, and other substances. Results from the three studies all highlight that despite similarities in patterns for different substances, the pathways of risk associated with pubertal development may vary by type of substance. Further, many theories of substance use risk (e.g., stage termination theory) are unclear as to whether theoretical predictions equally apply

to experimental substance use, regular use, abuse, and/or dependence. The third study (see Chapter 4) indeed provides initial evidence that pubertal development may not be risky for alcohol abuse outcomes. Theorists should thus also strive for conceptual clarity regarding the measurement of substance use constructs, and to which substance use outcomes a theory applies.

In sum, this dissertation has several implications for theories of adolescent substance use, particularly in terms of specifying causal pathways of risk, population and developmental specificity of risk, and finally the importance of construct measurement. Several policy implications follow from these theoretical implications, which are outlined below.

Policy Implications

The social epidemiological literature on adolescent substance use is largely concerned with identifying risk factors that can be addressed in substance use intervention programs. Effective substance use intervention programs should be guided by theories of causation and prevention that hypothesize the causal relationships between risk factors and the outcomes of interest. This requires identifying said risk factors, identifying the predictors of these risk factors, identifying the time point at which to interrupt the development of substance use, and specifying the appropriate intervention to prevent onset of use (Hawkins et al. 1992). As such, this dissertation yields several policy implications regarding causal pathways of risk and the specificity of risk associated with pubertal development.

First, and as noted in the theoretical implications, intervention programs cannot change the physiological process of pubertal development. Thus it is critical for researchers

and practitioners to understand the social mechanisms linking pubertal development to substance use that can be manipulated in substance use intervention programs. Results from this dissertation suggest that peer relationships may be an important target for intervention programs attempting to reduce the substance use risk related to adolescent girls' pubertal development. If a portion of the substance use risk associated with pubertal development is associated with deviant peer association, programs may attempt to minimize the perceived psychological and social distance between early developers and their same-age peers. Providing opportunities for early developing adolescent girls to socialize and befriend conventional same-age peers may alleviate some of the risk associated with deviant peer associations. Further, these targeted intervention programs geared toward early developers might focus specifically on adolescents living in affluent, advantaged neighborhood contexts where the substance use risk associated with pubertal development is greatest.

Second, intervention programs attempting to ameliorate the substance use risk associated with pubertal development among adolescent girls should occur during early adolescence, particularly between the ages of 10-12. This window of early adolescence is when pubertal changes in the body are beginning to surface, and when adolescent girls' sensitivity to such changes may be heightened. Because pubertal development largely influences initial levels of substance use, rather than changes in substance use over time, intervention programs that reach adolescent girls prior to puberty may be most effective.

Finally, intervention programs designed to address the substance use risk associated with pubertal development may need to be gender specific and address identity issues surrounding the body. Intervention programs should be particularly sensitive to different social and cultural expectations for girls and boys (e.g., value of appearance, dependency,

submissiveness) that may influence the substance use risk associated with pubertal development. Situating these goals within an ecological framework, programs should strive to improve young girls' social psychological perceptions of the self and body, in addition to promoting overall health and well-being, and addressing other co-occurring illnesses or traumas that may contribute to substance use among adolescent girls.

Limitations of the Studies

Despite the strengths of the three studies, several limitations should be acknowledged. First, due to data limitations none of the studies accounted for mother's age at menarche or other in-utero environmental factors. Recent research in the field of economics proposes that the link between puberty and adolescent behavior may be largely due to the correlation between body weight and prenatal and newborn environmental factors such as mother's health and nutritional status (Salsberry and Reagan 2005; Reagan 2008). Unfortunately these types of data were not available in the Add Health and so some of the models may have been misspecified because these variables were omitted. However, controlling for family structure and parental education likely reduced any potential omitted variable bias given these variables' correlations with prenatal and newborn environmental factors.

A second limitation is that although these studies controlled for adolescent girls' parental education levels, they did not explicitly examine differences of effects for girls from different family socioeconomic backgrounds. Puberty may be interpreted differently for girls from different family socioeconomic backgrounds. Results from the third study (Chapter 4) indicated that the substance use risk associated with pubertal development may be most pronounced for girls living in affluent, advantaged neighborhoods. It remains unclear

whether this neighborhood level effect is indeed a contextual effect, or simply the effect of family level socioeconomic status.

A third limitation is that the effect of pubertal development on adolescent girls' "hard" substance use (e.g., cocaine) was not examined. This was due largely to data limitations, given that the reported prevalence of adolescent substance use in the Add Health is lower than that reported in other national studies (Substance Abuse and Mental Health 2002). This problem is even more pronounced among adolescent girls. Only a small percentage of adolescent girls in the Add Health data reported using substances other than tobacco or alcohol (see Table 5.3). The small number of adolescent girls reporting hard substance use therefore precluded any reliable parameter estimation in multivariate statistical models.

Table 5.3. Survey Adjusted Percentage of Adolescent Girls Using "Hard" Substances: Add Health Wave I (N = 4,797)

Item	% Yes	Standard Error
Ever used marijuana	20.64	.01
Ever used cocaine	1.96	.00
Ever used inhalants	6.41	.01
Ever used drugs other than tobacco, alcohol, cocaine, or inhalants	6.95	.01
Ever used injection drugs	.19	.00

Note: Based on analytic sample from Chapter 3.

Directions for Future Research

Given the aforementioned limitations, there are several promising directions for future research examining the substance use risk associated with adolescent girls' pubertal development. First, future research should explore the role of intrauterine environmental factors on the relationship between pubertal development and adolescent girls' substance use, and whether these factors influence the mediating and moderating roles of substance-using friends, neighborhood dislocations, and race/ethnicity. Further, researchers interested in the complex interactions between biology and environment should explore whether intrauterine environmental factors are differentially influential when pubertal development is measured with age at menarche versus body development measures.

Additional research is also needed to examine whether the effects of pubertal development on adolescent girls' substance use varies for girls from different family socioeconomic backgrounds. Results from Chapter 4 indicate that the substance use risk may be greatest for girls living in advantaged neighborhood contexts, and so additional research is needed to explore whether family socioeconomic status is just as important. If the substance use risk associated with pubertal development is heightened for more affluent girls, it may lend support for the notion that the cult of thinness is most pronounced for women in advantaged socioeconomic locations. Again, due to data limitations, this postulate cannot be adequately addressed with the Add Health data, so additional work is needed using other data sources.

Finally, an important direction for future research on the substance use risk associated with pubertal development, as well as for the broader field of the social epidemiology of adolescent substance use is a focus on different pathways of risk for substance use, abuse,

and dependence (Kandel 1998). Understanding risk factors for adolescent substance use is important for intervention programs, but it is also important to know whether those risk factors are similarly predictive for more problematic levels of use (i.e., abuse, dependence). Longitudinal studies on high-risk samples of adolescent girls should be uniquely situated to address these issues.

CONCLUSION

The substance use risk associated with adolescent girls' pubertal development is not entirely due to something inherent in the biological processes associated with puberty. Rather, the meaning and import of puberty on outcomes such as substance use depends on social context. With pubertal bodies as the most visible aspect of the self, early developing adolescent girls are forced to negotiate new roles and expectations associated with their now adult-like, and explicitly gendered, body. An adolescent girl's social psychological and hence behavioral response to pubertal development will be guided largely by her ecological surroundings. Peer, family, and neighborhood contexts provide social comparison groups, group norms, and belief systems in which adolescent girls' lived experiences are embedded. By understanding these larger social contexts, social scientists can identify those risk factors that threaten the health and well-being of adolescent girls, knowledge which can then be used to help girls lead healthy and productive lives.

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