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A Test of Biosocial Models of Adolescent Cigarette and Alcohol Involvement

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Abstract

We tested biosocial models that posit interactions between biological variables (testosterone, estradiol, pubertal status, and pubertal timing) and social context variables (family, peer, school, and neighborhood) in predicting adolescent involvement with cigarettes and alcohol in a sample of 409 adolescents in grades 6 and 8. Models including the biological and contextual variables and their interactions explained significantly more variance in adolescent cigarette and alcohol involvement than did models including only the main effects of the biological and contextual variables. Post-hoc analyses of significant interactions suggested that, in most cases, moderation occurred in the hypothesized direction. Consistent with dual hazards models of adolescent antisocial behaviors, the relationships between the biological and substance use variables became positive and stronger as the context became more harmful. Considerations of adolescent substance use, and perhaps other problem behaviors, should recognize the possible role of biological variables and how their influence may vary by social context.

Keywords

hormones; adolescent cigarette use; adolescent alcohol use; biosocial models

Research on adolescent problem behaviors has largely been limited to examining the influence of psychological and social factors. However, several theoretical perspectives suggest that biological factors also play a role in the development of adolescent problem behaviors (Flay & Petraitis, 1994; Huba, Wingard, & Bentler, 1980; Jessor, 1991). Collectively, these theories suggest the need to consider biological, psychological, and social factors together to better understand problem behaviors that begin and accelerate during early adolescence. Moreover, they emphasize that individuals are embedded in social contexts and that there is dynamic interaction between the contexts and biological factors. However, most research does not simultaneously consider both contextual and biological variables. In this study, we consider the interaction of biological factors and social contexts as they relate to adolescent involvement with cigarettes and alcohol. The biological variables

to be examined include hormonal processes and characteristics of pubertal development. The social contexts considered include family, peers, school, and neighborhood, which are considered key contexts in the lives of adolescents (Bronfenbrenner, 1979; Brooks-Gunn, 1987). Our hypotheses are guided by dual-hazards models of antisocial behavior which propose that a biological propensity for antisocial behavior will be exacerbated in harmful social contexts.

The hormones examined are testosterone for both boys and girls, and estradiol for girls. Testosterone and estradiol are sex steroids from two endocrine groups: androgens (testosterone) and estrogens (estradiol). They are responsible for the sexual maturation changes associated with puberty that typically begin around age 11 for girls and age 12 for boys (Lee, 1980; Tanner, 1962). Two aspects of pubertal development are examined: pubertal status and pubertal timing. Pubertal status is the current level of physical development based on the appearance of somatic characteristics, such as changes in body hair, skin, height, and genitalia (Dubas, Graber, & Petersen, 1991). Pubertal timing captures whether an adolescent's pubertal development is occurring on time, early, or late relative to his or her peers (Dubas et al., 1991). Although hormones stimulate somatic changes associated with pubertal development, a delay may occur between the initial change in hormone levels and somatic changes (Richards, Abell, & Petersen, 1993). Thus, the hormones and the characteristics of pubertal are distinctly different biological variables.

We propose that adolescent involvements with cigarettes and alcohol is influenced by hormone levels and characteristics of pubertal development: the higher the hormone level, the more advanced the pubertal development status, or the earlier the pubertal development, the greater the involvement with cigarettes and alcohol. These predictions are based on positive associations between testosterone and adolescent and child problem behaviors (Bauman, Foshee, Koch, Haley, & Downton, 1989; Dabbs, Jurkovic, & Frady, 1991; Finkelstein et al., 1997; Olweus, Mattsson, Schalling, & Low, 1980, 1988; Susman et al., 1998), estradiol and adolescent problem behaviors and affective problems (Inoff-Germain et al., 1988), and pubertal status and adolescent problem behaviors (Ge, Conger, & Elder, 2001; Magnusson, Stattin, & Allen, 1986; Resnick et al., 1997; Silbereisen, Petersen, Albrecht, & Kracke, 1989). Studies also have found that early maturing adolescents are more involved in problem behaviors than adolescents who are on time or late in pubertal development (Cota-Robles, Neiss, & Rowe, 2002; Dick, Rose, Viken, & Kaprio, 2000; Flannery, Rowe, & Gulley, 1993; Halpern, Udry, Campbell, & Suchindran, 1993). We propose, further, that family, peer, school, and neighborhood contexts will moderate the associations between the biological factors and adolescent involvement with cigarettes and alcohol. Dual hazards models of adolescent antisocial behavior guide specific predictions about the proposed nature of the moderation, as described below.

Theoretical Framework and Hypotheses

In addition to the theories cited above that include biological influences, several prominent researchers of adolescent development espouse models that specify interactions between biological and social factors. As early as 1973, Hill (1983) emphasized that biological and psychosocial variables interact to influence the development of adolescents. Hamburg (1974) suggested that the significance of any intra-individual change that occurs during early adolescence, such as pubertal development, lies in its relation to all other areas of change in the multiple contexts in which the adolescent lives. Lerner (1987) uses the term "embeddedness" to suggest that events during adolescence at any one level (e.g., inner-biological, individual-psychological, interpersonal, societal) do not occur in isolation, but rather need to be considered in combination with events at the other levels. Lerner and Foch (1987) suggest that a key issue in the study of the basis of early adolescent development is

the understanding of the conditions under which biological functioning contributes to or constrains the early adolescent's psychological functioning. Applying these ideas specifically to adolescent pubertal development, Petersen (1987) suggests that the influence of pubertal development on adolescent behavior must be considered along with other changes in the developing individual and the several contexts in which that development takes place.

Although those researchers suggest that biological and contextual variables interact to influence adolescent development, they do not identify precise patterns for the interactions. For example, they do not indicate the specific social contexts within which the association between hormone levels and adolescent behaviors are expected to be relatively large or small, or the exact differential directions of the relationships. Dual hazards models of antisocial behavior, which were developed in the field of criminology, provide guidance in specifying the expected nature of the interactions.

Dual hazards models of antisocial behavior propose that a biological propensity for antisocial behavior in combination with negative social environments leads to criminal outcomes (Brennan & Raine, 1997). Brennan and Raine (1997) reviewed the tenets and empirical support for three dual hazards models of antisocial behavior: Eysenck's (1964) biosocial theory of crime, Mednick's (1977) biosocial theory, and Buikhuisen's (1988) biosocial theory of chronic juvenile delinquency. The main tenet of Eysenck's biosocial theory of crime is that biologically-based personality characteristics increase one's risk for antisocial outcomes, given a particular social upbringing. Mednick's biosocial theory predicts that children with deficits in the autonomic nervous system (the biological variable) who are raised in inadequate social environments (family context) are to be considered at highest risk for antisocial outcomes. Inadequate social environments include, for example, those in which punishment for aggressive behavior by parents is inadequate or inconsistent. Buikhuisen's biosocial theory of chronic juvenile delinquency suggests that chronic criminal behavior is the product of factors in both the person and his or her environment. "Person" factors include biological factors and "environment" factors include those from the family, peer, neighborhood, and social-political contexts. The theory predicts that a chronic delinquent outcome is most likely when the adolescent has both biological and social deficits.

The biological variables considered in the above dual-hazards theories are primarily autonomic, central nervous system, or neuropsychological deficits. There is some evidence that the dual-hazards concept also is supported when the biological variables are hormones and characteristics of pubertal development, the outcomes are adolescent problem behaviors and attributes, and the moderators are from the family, peer, school, and neighborhood context (Raine, 2002).

When considering the family context, Booth and colleagues (Booth, Johnson, Granger, Crouter, & McHale, 2003) found an interaction between testosterone and the parent-child relationship when predicting depression: As the quality of the parent-child relationship decreased, the positive associations between testosterone and adjustment problems of the child increased. In another study, pubertal timing interacted with parenting practices in predicting externalizing behaviors of children (Ge, Brody, Conger, Simons, Murry, 2002). The negative effects of early maturation on externalizing problems were exacerbated when the adolescent was exposed to harsh and inconsistent parenting. In the only empirical analyses to examine factors that moderate the relationship between testosterone and adolescent cigarette use, Bauman and associates (Bauman, Foshee, & Haley, 1992) found a significant interaction between testosterone and mother smoking for girls; testosterone influenced adolescent smoking only when the mother smoked cigarettes.

With regard to the peer context, Bauman et al. (Bauman et al., 1992) found a significant interaction for boys between testosterone and having a friend that smokes. For boys testosterone influenced smoking only when they were exposed to peers who smoked cigarettes. Rowe and colleagues (Rowe, Maughan, Worthman, Costello, & Angold, in press) found that peer deviance moderated the association between testosterone and adolescent non-aggressive conduct disorder symptoms, with the strongest positive association being among the boys who had deviant peers. In a study of adolescent sexual behavior, Smith and associates (Smith, Udry, & Morris, 1985) found that the association between pubertal status and adolescent sexual behavior was moderated by friend's sexual behavior, such that those at highest risk for sexual behavior were those with advanced pubertal development who had friends who were sexually active.

In studies examining school factors, Udry (1991) found that the association between testosterone and alcohol use by adolescents was moderated by school achievement, such that those adolescents at highest risk for alcohol use were those with both high testosterone and low school grades. In addition, Caspi et al. (Caspi, Lynam, Moffitt, & Silva, 1993) found that the association between pubertal timing and delinquency was moderated by characteristics of the school.

Finally, with regard to neighborhoods, Obeidallah et al. (Obeidallah, Brennan, Brooks-Gunn, & Earls, 2004) found that early maturing girls living in disadvantaged neighborhoods were three times more likely to engage in violent behaviors than early maturing girls who did not live in disadvantaged neighborhoods, controlling for individual-level socioeconomic status and race. Another study found that early maturation was associated with drinking frequency by girls who lived in urban settings but not by girls living in rural settings (Dick et al., 2000). Although not an examination of neighborhood characteristics per se, in another study, testosterone was positively associated with delinquency for boys who were of low socioeconomic status (SES) but there was no association for boys of high SES (Dabbs & Morris, 1990).

As noted above, dual-hazards models propose that a harmful context exacerbates the impact of a risk on the outcome. Other theoretical perspectives, especially theories of resiliency, suggest that a protective context neutralizes the impact of a risk on the outcome (Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Garmezy, Masten, & Tellegen, 1984; Hollister-Wagner, Foshee, & Jackson, 2001). Thus, specific to our study, in more protective contexts, the biological variables would not be expected to be associated with the adolescent's involvement with substances, but consistent with expectations of dual-hazards models, we propose that the biological propensity to smoke cigarettes and drink alcohol, as indicated by hormone levels and characteristics of pubertal development, will be exacerbated when the adolescent is exposed to harmful social contexts. Thus, we propose the following four hypotheses:

The relationships between the biological variables and cigarette and alcohol involvement will become positive and more significant as the context (family [H1], peer [H2], school [H3], and neighborhood [H4]) becomes more harmful.

Based on the findings from studies that have examined associations between various aspects of the contexts considered and adolescent problem behaviors, we characterize harmful contexts in the following ways. Characteristics of a harmful family context include having parents who are not responsive to the needs and emotions of their children (Baumrind, 1985, 1991; Jackson, Henriksen, & Foshee, 1998), do not set and enforce clear standards of behavior (Baumrind, 1985, 1991; Jackson et al., 1998), express approval or do not express disapproval of cigarette and alcohol use (Andrews, Hops, Ary, Tildesley, & Harris, 1993; Brook, Whiteman, Gordon, & Cohen, 1986; Kandel & Andrews, 1987), do not actively

discourage use (Andrews et al., 1993; Komro et al., 2001), and do not state expectations and consequences related to substance use (Komro et al., 2001).

Peer substance use and approval of use have been shown empirically to be among the strongest correlates of adolescent substance use (Botvin, Baker, Goldberg, Dusenbury, & Botvin, 1992; Hawkins, Catalano, & Miller, 1992). Thus, the peer context will become more harmful as the proportion of peers who smoke or drink alcohol or who approve of using those substances increases.

Characteristics of a harmful school context are that substance use is perceived as being very prevalent by students in the school, substance users are given high status in the school, and the school is not viewed as a caring community (Ennett, Flewelling, Lindrooth, & Norton, 1997; Komro et al., 2001). Finally, a harmful neighborhood context has been defined as one in which the residents do not feel safe, adults are not willing to monitor the behavior of the teens living there, and social integration is low (Brook, Nomura, & Cohen, 1989; Sampson, Raudenbush, & Earls, 1997).

The study hypotheses are tested with a sample of early adolescents who range in age from 11 to 14 years old. Although cigarette and alcohol use do not peak until later in adolescence (Jackson, Sher, Cooper, & Wood, 2002), early involvement in substances, especially use by 11-to 13-year-olds, repeatedly has been found to be a predictor of substance abuse and substance use disorders in later adolescence and adulthood (Anthony & Petronis, 1995; DeWit, Adlaf, Offord, & Ogborne, 2000; Sung, Erkanli, Angold, & Costello, 2004). Thus, identifying factors associated with early involvement with cigarettes and alcohol is important for understanding how problematic substance use begins and may be prevented.

Methods

Study Design and Sample

Data were collected in Spring 2001 in a middle school located in a suburban community in central North Carolina. Letters sent to parents via first class mail and also sent home from school with the child invited all sixth and eighth grade students enrolled in the school to participate in the study. Parents who did not want their child to participate returned a signed refusal form or called a toll-free number. Adolescents also had the opportunity to refuse participation when written informed assent was obtained in the classroom. The study was approved by the Institutional Review Boards at the University of North Carolina at Chapel Hill School of Public Health and Wake Forest University School of Medicine.

Of 513 eligible students, 424 (82.7%) participated. There were no differences in response rates for the sixth (83.7%) and eighth grade (81.3%) cohorts ($p = .49$). Of the 89 adolescents who did not participate, 60 did not participate because their parents refused participation, 27 did not provide written assent, and 2 were absent on both data collection days. Of the 424 participants, 418 provided saliva samples for hormone assays. We screened all saliva samples for blood contamination, which can invalidate salivary hormone estimates (Worthman & Stallings, 1997), and eliminated nine adolescents with possible blood contamination from the analyses (Kivlighan, Granger, & Schwartz, 2005; Kivlighan, Granger, Schwartz, Nelson, & Curran, 2004), leaving 409 adolescents in the analysis sample. Although there were only a few instances when data were missing on the study variables, imputation procedures (Rubin, 1987) using SAS PROC MI were performed to generate a data set that allowed us to use all 409 adolescents in the analyses.

Of the 409 adolescents in the study sample, half (51.3%) were boys. Thirteen of the 409 adolescents (3.2%) indicated they were of Hispanic origin. The racial distribution of the 409

adolescents was 72.4% white, 17.5% black, 4.2% mixed race, less than 1% Asian, 2.2% of other races, and the race of a few participants (0.3%) was unknown. Approximately 45.0% of the students indicated that their mother had a college degree, and approximately 76.0% of the adolescents lived in two-parent households. The average age was 11.6 years ($SD = .57$) for sixth graders and 13.6 years ($SD = .61$) for eighth graders.

Data Collection Procedures

Data were collected in schools in homeroom classes. After excusing students whose parents had refused permission for participation, research staff distributed packets containing study materials, described the study, obtained written adolescent assent, collected saliva samples, and administered questionnaires. To control for diurnal variation in hormones, saliva samples always were collected before adolescents completed questionnaires. Thus, hormone samples were collected between 8:10 A.M and 10:20 A.M., with 90% of samples being collected between 8:15 and 10:05. Several procedures were used to increase the likelihood of valid self-reports of substance use. Trained data collectors (rather than teachers) were used to administer the questionnaires, and teachers were asked not to walk around the room or to answer student questions related to the questionnaires during data collection. Adolescents were spread out during data collection, and two trained data collectors were in each room to decrease the likelihood that students would look at each other's answers. The confidentiality of the data was stressed when the data collectors described the study, and a protocol was used that clearly showed that names were not on the questionnaires. Each questionnaire contained a unique student identification number to allow linkage with the biological specimen. Finally, when adolescents completed the questionnaire, they put it in an envelope and sealed it before handing it to the data collector. Students were allowed approximately 2 hours to complete the self-administered questionnaires. Others have concluded that school-based data collection with procedures like those we used produce acceptably accurate self-reports of adolescent substance use (Fendrich, & Johnson, 2001; Sudman, 2001).

To collect the saliva samples, adolescents were given a straw and a 30 ml vial marked at the 5 ml level that was labeled with their subject identification number. Adolescents were instructed to spit saliva through the straw into the vial to the 5 ml line. Data collectors checked each vial as it was turned in and adolescents who had not provided a sufficient quantity were encouraged to provide more saliva. Data collectors put the completed vials in plastic bags and immediately placed the bags on dry ice in coolers. The bags were then transferred to a freezer and stored at -70°C until they were shipped on dry ice for assaying.

Measures

Biological Variables

Pubertal development: Two pubertal development variables were created: pubertal status and pubertal timing. Pubertal status was measured with the Pubertal Development Scale (PDS) (Petersen, Crockett, Richards, & Boxer, 1988), which is strongly correlated with physician ratings of adolescent physical development (Petersen et al., 1988). The PDS measures the presence of secondary sexual characteristics including body hair growth, skin changes, height, voice changes (boys only), facial hair growth (boys only), breast development (girls only), and menarche (girls only). Except for a dichotomous item assessing menarche, the response options range from 1 ("not yet started") to 4 ("seems complete"). Responses were summed, such that higher scores indicate more advanced pubertal development ($\alpha = .77$ for both boys and girls). Pubertal timing was measured with a single item: "Compared to most other adolescents your same age and same sex, do you think your physical development is much earlier, somewhat earlier, about the same, somewhat later, or much later?" (Dubas et al., 1991). This variable was coded so that higher

scores indicate earlier physical maturation. Both pubertal development variables were centered.

Testosterone: Saliva samples were analyzed for testosterone using a radioimmunoassay (Diagnostic Systems Laboratories, Webster, TX) modified for use with saliva (Granger, Schwartz, Booth & Arentz, 1999). The test uses 200 μ l of saliva (for singlet determination) and has a minimum detection limit of 0.8 pg/ml. Values from matched serum and saliva samples using this method show the expected strong linear relationship (Granger et al., 1999). The testosterone values used in the analyses were determined by assay in singlet. A random sample of 20% was assayed in duplicate to confirm reliability; the intra-assay coefficient of variation and correlation coefficient between the duplicate tests were 12.63% and $r = .99, p < .01$, respectively. Both are in the range considered acceptable (Chard, 1990). The testosterone distribution was sufficiently skewed (skew = 2.17) to warrant a log (base 10) transformation.

Estradiol: Estradiol also was assayed for girls using radioimmunoassay (Diagnostic Systems, Webster, TX) modified for use with saliva (Shirtcliff et al., 2000). The test uses 300 μ l of saliva (for singlet determination), and has a minimum detection limit of 0.25 pg/ml (range up to 7.5 pg/ml). Values from matched serum and saliva samples show the expected linear relationship for girls (Shirtcliff et al., 2000). Given the low levels of estradiol expected in this young adolescent group, all samples were assayed in duplicate. The intra-assay coefficient of variation and correlation between the duplicate tests were 16.01% and $r = .98, p < .01$, respectively. These values are within the acceptable range (Chard, 1990). The average of the duplicates was used in the analyses except in cases in which one assay detected estradiol and the other did not, in which case the detected estradiol value was used. Estradiol was coded "0" if no estradiol was detected in either assay. The estradiol distribution was sufficiently skewed (skew = 2.89) to warrant a log (base 10) transformation.

Factors that Potentially Influence Hormone Values—We examined the associations between testosterone and estradiol and a number of factors that could influence those hormone levels, including current sickness; drugs taken for asthma, diabetes, endocrine disorders, depression, anxiety, and hyperactivity; recent (within 15 minutes of saliva collection) intake of food and dairy products; steroid use; and, for girls, menarche status and intake of hormones, including birth control pills. There were no significant associations between these variables and the hormone values, and therefore we did not eliminate adolescents with any of these characteristics from the analyses.

Context Variables—The context variables each were composed of multiple scales. Responses to the scales were converted to z-scores before creating the total context scores to impose equal weights on the constructs making up the context variables. Some of the scales within the family, peer, and school context variables are substance specific. Thus, for these contexts, there are two context variables: one that incorporates the cigarette-specific items and one that incorporates the alcohol-specific items. The neighborhood context did not include any substance-specific items, and therefore there is only one neighborhood context variable. All context variables were coded so that the lower the number, the more harmful the context.

Family context: The two family context variables were each created from items that measured parental responsiveness, parental demandingness, and substance-specific parenting practices. Parental responsiveness and demandingness were measured with the 20-item Authoritative Parenting Index (Jackson et al., 1998); 10 items each assessed responsiveness and demandingness. Adolescents completed the index twice, once

referencing their mother figure and once referencing their father figure. Example responsiveness items are “She/he listens to what I have to say” and “She/he wants to hear about my problems.” Example demandingness items are “She/he has rules that I must follow” and “She/he tells me times when I must come home.” The four-point response options range from “just like her/him” to “not like her/him.” The items for each scale were summed and converted to z-scores ($\alpha = .88$ for responsiveness and $\alpha = .81$ for demandingness).

Cigarette-specific parenting practices were measured with five questions, asked in reference to the mother and then again in reference to the father. One question assessed how strongly the mother (father) would approve or disapprove of the adolescent smoking a cigarette. The other questions assessed whether in the previous 6 months the mother (father) had encouraged the adolescent to not smoke cigarettes, talked to the adolescent about rules related to cigarette use, and talked to the adolescent about the negative consequences of cigarette use. Parallel items assessed alcohol-specific parenting practices. For each scale, all of the items were summed and converted to a z-score. The alpha for both cigarette-specific parenting practices and alcohol-specific parenting practices is .90. The z-scores for the three parent constructs described above were summed to create each of the two family context variables.

Peer context: The two peer context variables included assessments of substance use in the adolescent’s friendship network as reported by friends, perceived peer substance use, and perceived peer approval of substance use. We used peer nomination procedures to obtain information about actual substance use within friendship networks. Each respondent was provided with a student roster in which each student who was in the same grade as the respondent was given an identification number. Adolescents identified their best friend and up to six other close friends using the identification number from the student roster. The adolescent’s friendship network was defined as those school friends identified by the respondent and those adolescents who identified the respondent as a friend. Because each respondent’s friends also were included in data collection, the friend’s reports of cigarette and alcohol use were used to measure friend substance use in the network. Cigarette use in the friendship network was defined as the proportion of friends in the network that indicated that they had at least puffed on a cigarette; alcohol use in the friendship network was defined as the proportion of friends in the network that indicated that they had at least sipped alcohol.

We also assessed the adolescent’s perception of peer substance use by asking, “About how many of these friends would you estimate smoke cigarettes (drink alcohol)?” The five response options ranged from “almost none” to “almost all.” We assessed peer approval of substance use by asking, “How do you think these friends would feel about you smoking cigarettes (drinking alcohol)?” The five response options ranged from “strongly approve” to “strongly disapprove.” We created z-scores for each of these variables and summed the items to create our two peer context variables, one for the peer cigarette context and one for the peer alcohol context.

Perceived school context: The two perceived school context variables included assessments of perceived prevalence of substance use in the school, status given substance users in the school, and sense of school as a caring community. To assess perceived prevalence of substance use, adolescents were asked, “At your school, about how many students your age would you estimate smoke cigarettes (drink alcohol [beer, wine, wine coolers, or liquor])?” The five response options ranged from “almost none” to “almost all.” Status afforded substance users was assessed by asking adolescents to indicate how important it is in his/her school to smoke cigarettes (drink alcohol) in order to be looked up to or to have high status.

The four response options ranged from “not at all important” to “very important.” The adolescent’s sense of his/her school as a caring community was assessed with a scale developed by Roberts, Hom, and Battistich (1995). Adolescents were asked how strongly they agreed or disagreed with a series of nine statements about their school environment. Example items are “People care about each other in this school” and “Students in my school don’t get along together very well.” The nine items were summed ($\alpha = .81$). We computed z-scores for these composite variables and summed them to create our two perceived school context variables, one for the school cigarette context and one for school alcohol context.

Neighborhood context: Perceived characteristics of the neighborhood, such as perceived safety, adult monitoring, and social integration, were measured by asking adolescents to indicate how strongly they agreed or disagreed with 11 statements about their neighborhood. Example items are “People feel safe in my neighborhood,” “Adults in my neighborhood tell other parents if their child has done something bad,” and “People in my neighborhood socialize together.” Items were summed ($\alpha = .70$) and, for consistency with the other context measures, we standardized the variable into a z-score.

Cigarette and Alcohol Involvement—Cigarette involvement was created as a composite of three questions. One question assessed the number of cigarettes the adolescent smoked the last day he or she smoked, with seven response options ranging from “never smoked” to “two packs or more.” Another assessed the number of days the adolescent smoked cigarettes during the past 6 months with six responses ranging from “0 days” to “20 days or more.” The third question asked how much the adolescent usually smoked when he or she smoked over the past 6 months, with seven response options ranging from zero to “two packs or more a day.” Responses to these three questions were summed, with higher scores indicating greater cigarette involvement.

Alcohol involvement was created as a composite of two questions. One question assessed the number of days in the past 6 months that the adolescent had one or more drinks of alcohol (including beer, wine, wine coolers, and liquor, and not including wine at church) with six responses ranging from “0 days” to “20 days or more.” The second assessed how much the adolescent usually had to drink when he or she did drink in the past 6 months, with seven responses ranging from zero to “five or more drinks” (with a drink being a glass of wine, a can of beer, a bottle or can of wine cooler, or a shot glass of liquor, or a mixed drink). Responses to these two questions were summed, with higher scores indicating greater alcohol involvement.

Data Analysis

Table 1 presents the bivariate correlations between all study variables for boys (top half) and girls (bottom half). All significant relationships are in the expected direction.

For our hypotheses to be supported, the interactions between the biological and context variables as related to each substance use outcome must be statistically significant, and the associations between the biological variables and substance use outcomes should become positive and stronger as the context becomes more harmful. We first conducted a two-step hierarchical linear regression analysis as suggested by Frazier, Tix, and Barron (2004) to identify statistically significant interactions. Step 1 included the main effects of the biological variables (hormones, pubertal status, and pubertal timing), control variables (age and, for girls, their menstrual status [whether the girl had begun menstruating] and current menstruation [whether the girl was menstruating at the time of data collection]), and the four context variables on each substance use outcome. In step 2, all of the two-way interactions

between the biological variables and the context variables were added to the step 1 model. We conducted an omnibus F test to determine if the increment in R^2 from the main effects model (step 1) to the model including the significant interactions (step 2) was statistically significant. That omnibus F test determines if there is significant gain in model prediction when testing for moderation (Aiken & West, 1991), and it helps to control the Type I error because only one test is being conducted to determine the presence of significant interactions. According to Frazier et al. (2004), if the omnibus F test is significant, then the single degree of freedom tests related to a specific interaction can be inspected and post hoc analyses can be conducted on significant interactions to determine the nature of the interaction.

To probe the nature of significant interactions, we followed post hoc analyses suggested by Bauer and Curran (in press) and Curran, Bauer, and Willoughby (in press). We used their interactive software (Preacher, Curran & Bauer, 2003) to determine the value of the context variable at which the slope associated with a biological variable and the outcome becomes significantly positive. For the results to be in accordance with the hypothesis, the direction of the slope should become positive at values of the context variable that are considered harmful, and the positive slopes should become more significant as the context becomes more harmful. We considered any value below the mean on the context variable to represent a harmful context.

Although not a primary purpose for this paper, when biological and context variables were not involved in a significant interaction, their first-order effects are described. Because all of the independent variables are centered, it is possible to interpret the first-order effects of the biological and context variables from the step 2 models that are not involved in significant interactions. However, it is important to note that those effects are conditional effects in that they represent the effects of the variables on the outcomes at the average level of the other variables in the model (Frazier et al., 2004).

Also, although our primary purpose was to examine how biological and substance use associations might be moderated by context, we conducted analyses of mediation effects—how biological variables might influence substance use through their impact on contexts. Thus, for biological variables that were not involved in significant interactions and that were significantly correlated with the substance use outcome in bivariate analyses but not in the step 2 multivariate analyses, we followed MacKinnon & Dwyer's (1993) difference of coefficients approach for identifying mediators of the associations between those biological variables and the substance use outcomes. We added one variable at a time to the model regressing the substance use outcome on the biological variable to determine if that variable attenuated the regression coefficient associated with the biological variable. We then did the analyses required to determine if the amount of attenuation, or the mediated effect, was statistically significant using the Sobel test (Sobel, 1982). Mediation is indicated if the potential mediator variable significantly attenuates the association between the biological variable and the substance use outcome, and if the biological variable is significantly associated with the potential mediator.

All analyses were stratified by gender because the variables included in the models differed for boys and girls. Specifically, estradiol, menstrual status, and current menstruation were measured only for girls.

Before conducting the analysis to test the study hypotheses, we conducted analyses using procedures described by Cohen (1988) to determine if there was sufficient power to detect statistical significance in the incremental R^2 (effect size) from the step 1 to the step 2 models in the hierarchical linear regression models. The power to detect a significant incremental R^2

ranged from .94 to .96 across the four models (boy and girl cigarette and alcohol involvement), and therefore we consider power to be adequate to detect interactions. We also tested for multicollinearity in our models by conducting eigenanalyses (Belsley, Kuh, & Welsch, 1980) on the covariate matrices associated with the step 2 models. The condition indices across the four models ranged from 3.64 to 5.66 and the VIF (variance inflation factors) ranged from 2.65 to 3.83. Because condition numbers above 30.0 and a VIF above 10.0 indicate multicollinearity, we concluded that we did not have a problem with collinearity in our models. We also checked for the impact of outliers on our interaction analyses and concluded they had no impact.

Results

Boys

Cigarette Involvement

Tests of study hypotheses for cigarette involvement by boys: In the step 1 model, 38.6% of the variance in cigarette involvement by boys is explained by the main effects of the biological, control, and context variables. The variance explained when adding the interactions between the biological and the context variables (step 2) increases to 45.5%. This incremental R^2 ($\Delta R^2 = .069$) is statistically significant ($p = .03$), thus providing the justification to examine the nature of individual interactions in post hoc analyses.

The parameter estimates from the step 2 model of the hierarchical linear regression analyses for predicting cigarette involvement by boys are presented in Table 2. Two interactions are statistically significant: the interaction between testosterone and the peer context and the interaction between pubertal timing and the peer context. Based on the post hoc analyses, the slope associated with the testosterone and cigarette involvement relationship is positive and significant for all values of the peer context that are 0.55 or greater standard deviations below the mean. Thus, the nature of this interaction is as hypothesized: A significant positive relationship between testosterone and cigarette involvement first appears when the peer context value is 0.55 standard deviations below the mean (which represents a more harmful context), and that positive association becomes more significant as the peer context becomes more harmful.

The nature of the interaction between pubertal timing and cigarette involvement, however, is the opposite of what was hypothesized. The slope associated with the pubertal timing and cigarette involvement relationship is negative and significant for all values of the peer context that are 1.29 or greater standard deviations below the mean. Thus, early pubertal development is associated with less cigarette involvement by boys in a harmful peer context.

Additional findings: Conditional main effects and mediators of the relationships between biological variables and cigarette involvement by boys: Pubertal status was not involved in a significant interaction and therefore its conditional main effect can be examined. Although pubertal status was significantly associated in bivariate analyses with cigarette involvement by boys ($b = .63, p = .01$), it was no longer associated with cigarette involvement by boys in the multivariate analyses (see Table 2). Based on our analyses for identifying mediators of the pubertal status and cigarette involvement relationship, the peer context and the family context are the only variables that met the criteria for being mediators of that relationship. The regression coefficient associated with the pubertal status and cigarette involvement relationship when controlling for the peer context ($b = .03, p = .90$) was significantly attenuated (Sobel test = 3.88, $p < .01$) from the bivariate model ($b = .63, p = .01$), and the peer context was significantly associated with cigarette involvement ($b = -.59, p < .01$) in that model. The regression coefficient associated with the pubertal status and

cigarette involvement relationship when controlling for the family context ($b = .46, p = .07$) was significantly attenuated (Sobel test = 2.23, $p = .02$) from the bivariate model ($b = .63, p = .01$), and the family context was significantly associated with cigarette involvement ($b = -.16, p < .01$) in that model.

None of the interactions involving the family, school, or neighborhood context were statistically significant, and therefore their conditional main effects can be examined. Although the family context ($b = -.19, p < .01$) and the neighborhood context ($b = -.30, p = .04$) were significantly associated with cigarette involvement by boys in bivariate analyses, those two variables were no longer associated with cigarette involvement by boys in the multivariate analyses (see Table 2). The school context was not associated with cigarette involvement by boys in bivariate ($b = -.15, p = .12$) or multivariate analyses (see Table 2).

Alcohol Involvement

Tests of study hypotheses for alcohol involvement by boys: In the step 1 model, 31.6% of the variance in alcohol involvement by boys is explained by the main effects of the biological, control, and context variables. The variance explained when adding the interactions between the biological and the context variables (step 2) increases to 39.7%. This incremental R^2 ($\Delta R^2 = .081$) is statistically significant ($p = .02$).

The parameter estimates from the step 2 model of the hierarchical linear regression analyses when predicting alcohol involvement by boys are presented in Table 2. One interaction is statistically significant: the interaction between testosterone and the peer context. As hypothesized, the slope associated with the testosterone and alcohol involvement relationship is positive and significant for all values of the peer context that are 3.11 or greater standard deviations below the mean. That positive association becomes stronger as the peer context becomes more harmful.

Additional findings: Conditional main effects and mediators of the relationships between biological variables and alcohol involvement by boys: Pubertal status was not involved in a significant interaction and therefore its conditional main effect can be examined. Although pubertal status was significantly associated in bivariate analyses with alcohol involvement by boys ($b = .77, p < .01$), it was no longer associated with alcohol involvement by boys in the multivariate analyses (see Table 2). Based on our analyses for identifying mediators of the pubertal status and alcohol involvement relationship, the peer context is the only variable that met the criteria for being a mediator of that relationship. The regression coefficient associated with the pubertal status and alcohol involvement relationship when controlling for the peer context ($b = .36, p = .06$) was significantly attenuated (Sobel test = 3.55, $p < .01$) from the bivariate model ($b = .77, p < .01$), and the peer context was significantly associated with alcohol involvement ($b = -.36, p < .01$) in that model.

Pubertal timing also was not involved in a significant interaction. That variable, however was not associated with alcohol involvement by boys in either bivariate ($b = .225, p = .10$) or multivariate analyses (see Table 2), therefore an assessment of mediators of that relationship was not warranted.

None of the interactions involving the family, school, or neighborhood context were significant and therefore their conditional main effects can be examined. The neighborhood context was significantly and negatively associated with alcohol involvement by boys in both bivariate analyses ($b = -.521, p < .01$) and multivariate analyses (see Table 2), such that the more harmful the neighborhood context the more involvement the boys have with

alcohol. Although the family context ($b = -.23, p < .01$) and the school context ($b = -.25, p < .01$) were significantly associated with alcohol involvement by boys in bivariate analyses, neither of these variables was associated with alcohol involvement by boys in multivariate analyses (see Table 2).

Girls

Cigarette Involvement

Tests of study hypotheses for cigarette involvement by girls: In the step 1 model, 40.1% of the variance in cigarette involvement by girls is explained by the main effects of the biological, control, and context variables. The variance explained when adding the interactions between the biological and the context variables (step 2) increases to 50.2%. This incremental R^2 ($\Delta R^2 = .101$) is statistically significant ($p = .02$).

The parameter estimates from the step 2 model of the hierarchical linear regression analyses when predicting cigarette involvement by girls are presented in Table 3. Two interactions are statistically significant: the interactions between pubertal status and the peer context and between estradiol and the neighborhood context. The nature of both of these interactions is as hypothesized. The slope associated with the pubertal status and cigarette involvement relationship is positive and significant for all values of the peer context that are 0.12 or greater standard deviations below the mean; that positive association becomes stronger as the peer context becomes more harmful. Likewise, the slope associated with the estrogen and cigarette involvement relationship is positive and significant for all values of the neighborhood context that are 0.38 or greater standard deviations below the mean; that positive association becomes stronger as the neighborhood context becomes more harmful.

Additional findings: Conditional main effects for cigarette involvement by girls:

Pubertal timing was not involved in a significant interaction and therefore its main effects can be examined. Pubertal timing was not associated with cigarette involvement by girls in bivariate ($b = -.022, p = .89$) or multivariate analyses (see Table 3), and therefore an assessment of the mediators of that association was not warranted.

None of the interactions involving the family or school context were significant. The school context was significantly negatively associated with cigarette involvement by girls in both bivariate analyses ($b = -.395, p < .01$) and multivariate analyses (see Table 3), such that the more harmful the school context, the more involvement the girl has with cigarettes. Although the family context was associated with cigarette involvement by girls in bivariate analyses ($b = -.19, p < .01$), it was not associated with cigarette involvement by girls in the multivariate models (see Table 3).

Alcohol Involvement

Tests of study hypotheses for alcohol involvement by girls: In the step 1 model, 34.4% of the variance in alcohol involvement by girls is explained by the main effects of the biological, control, and context variables. The variance explained when adding the interactions between the biological and the context variables (step 2) increases to 51.1%. This incremental R^2 ($\Delta R^2 = .167$) is statistically significant ($p < .01$).

The parameter estimates from the step 2 model of the hierarchical linear regression analyses predicting alcohol involvement by girls are presented in Table 3. Four interactions are statistically significant, and three of the four are of the nature hypothesized. For those that support the hypothesis, the value of the context variable at which the positive association between the biological variable and alcohol involvement becomes significant varies. The slope associated with the testosterone and alcohol involvement relationship is positive and

significant for all values of the family context that are 5.81 or greater standard deviations below the mean. The slope associated with the pubertal status and alcohol involvement relationship is positive and significant for all values of the peer context that are below the mean. Similarly, the slope associated with the estrogen and alcohol involvement relationship is positive and significant for all values of the neighborhood context that are below the mean. In all cases, the positive associations get stronger as the context gets more harmful. The fourth interaction, which is between testosterone and the peer context, is in the opposite direction from what was hypothesized. The slope associated with the testosterone and alcohol involvement relationship is negative and significant for all values of the peer context that are below 0.20 standard deviations above the mean. Thus, testosterone is associated with less alcohol involvement by girls in a harmful peer context.

Additional findings: Conditional main effects for alcohol involvement by girls: Pubertal timing was not involved in a significant interaction and therefore its main effect can be examined. Pubertal timing was not associated with alcohol involvement by girls in bivariate ($b = .074$, $p = .57$) or multivariate analyses (see Table 3), and therefore an assessment of the mediators of that association was not warranted.

The school context also was not involved in any significant interactions. In bivariate analyses, the school context is significantly associated with alcohol involvement by girls ($b = -.26$, $p < .01$) but that relationship decreases to non-significance in multivariate models (see Table 3).

Discussion

Evidence supporting the proposed interactive biosocial model is that, for both boys and girls, models including the biological and contextual variables and their interactions explained significantly more variance in adolescent cigarette and alcohol involvement than did models including only the main effects of the biological and contextual variables, and that post-hoc analyses of significant interactions suggested that, in most cases, moderation occurred in the hypothesized direction. Consistent with dual hazards models of adolescent antisocial behaviors, in most cases, the relationships between the biological and substance use variables became positive and stronger as the context became more harmful. Although the value on the context variable at which the associations between the biological variables and the outcome variables became significant varied considerably, in none of these interactions was there a significant positive association between the biological variable and the substance use outcome at values of the context variable above the mean, indicating more protective contexts. Additionally, the effect sizes for the interactions were substantial, ranging from .069 to .167, compared to typical interaction effect sizes of .020 (Frazier et al., 2004). Although many of the interactions were not statistically significant, the evidence above, coupled with the growing body of literature providing evidence of interactive biosocial models, suggests that studies examining biological influences on adolescent problem behaviors should not ignore the possibilities that those influences vary by context. The findings also suggest that studies of social influences should not ignore biological variables.

Most prior studies of the associations between biological variables and adolescent problem behaviors, however, examined main effects and did not consider the possibility that these associations could be moderated by context variables. Our findings suggest that ignoring the possibility of moderation can lead to inappropriate conclusions about the role of biological variables in the development of adolescent problem behaviors. For example, based on our bivariate main effects findings, we would have concluded that testosterone was not associated with alcohol involvement by girls. Our interaction findings, however, led us to conclude that testosterone is associated with alcohol involvement for some girls, specifically

those living in harmful family contexts and a harmful peer context, but not for other girls. As pointed out by Mazur and Booth (1998), on close inspection many studies examining the main effects of testosterone on adolescent problem behaviors found weak or no significant associations (Constantino, et al., 1993; Inoff-Germain et al., 1988; Mattsson, Schalling, Olweus, Low, & Svensson, 1980; Olweus et al., 1980, 1988; Susman et al., 1987; Udry, 1988, 1990; Udry, Billy, Morris, Groff, & Raj, 1985). Perhaps the main effects findings were weak or non-existent because the relationships between testosterone and problem behaviors vary by groups. If testosterone is associated with problem behaviors for some groups but not others, the main effect of testosterone on problem behaviors could be canceled out.

Likewise, our findings suggest that examining the main effects of context variables on adolescent problem behaviors without considering the possibility that biological variables moderate those associations also can lead to misleading conclusions. For example, we found that the neighborhood context was not associated with alcohol involvement by girls, but with the interaction analyses we were able to determine that neighborhood context is associated with alcohol use by girls who also have high estradiol levels. Our findings support the tenet from several theories of adolescent problem behaviors that the influence of both biological and social factors on adolescent problem behaviors should be considered (Flay & Petraitis, 1994; Huba et al., 1980; Jessor, 1991), as well as the models espoused by several prominent researchers of adolescent development that encourage consideration of interactions between biological and context or environmental variables (Hamburg, 1974; Hill, 1983; Lerner, 1987; Lerner & Foch, 1987; Petersen, 1987).

For six of the nine statistically significant interactions, the peer context was the moderator. The peer context moderated the relationships between biological and outcome variables for both boys and girls and when considering both cigarette and alcohol involvement. In four of these six interactions, as hypothesized, the association between the biological variable and the substance use outcome was positive and became stronger as the peer context became more harmful. The nature of those interactions suggests that a biological propensity for cigarette and alcohol use coupled with the availability of peer models of the behaviors may be particularly toxic. Other investigators also have found that the positive associations between biological variables and problem behaviors of adolescents, including early sexual initiation (Smith et al., 1985), conduct disorders (Rowe et al., in press), and cigarette use (Bauman et al., 1992), were the strongest for adolescents who had friends who were also involved in those same problem behaviors.

However, interestingly, two of the interactions involving the peer context were opposite the direction hypothesized; one suggested that boys in a harmful peer context may be protected from using cigarettes if they are early maturers and the other suggested that girls in a harmful context may be protected from using alcohol if they have high levels of testosterone. Clearly, our findings suggest that there is interplay between biology and peer interactions that needs to be considered in future research.

For two of the nine significant interactions, the neighborhood context was the moderator, and both of these interactions were in the girl models. For girls, the neighborhood context moderated the associations between estradiol and both substance use outcomes. The positive associations between estradiol and the substance use outcomes became stronger as the neighborhood context became more harmful. To our knowledge, only two other studies have examined the neighborhood context as a moderator of the relationships between biological variables and problem behaviors of adolescents. In one of those studies (Obeidallah et al., 2004), neighborhood context was measured with census indicators of neighborhood disadvantage. Early maturing girls living in disadvantaged neighborhoods were more likely to be violent than early maturing girls living in neighborhoods that were not disadvantaged,

controlling for individual-level race and socioeconomic status. One of their explanations for this finding is that disadvantaged neighborhoods offer more opportunities to associate with negative role models for problem behaviors than do neighborhoods that are not disadvantaged, and early maturing girls may attract older adolescents who may be deviant. In our analyses, neighborhood context moderated associations between biological variables and outcomes for girls even after controlling for peer substance use. However, we do not have information about the availability of older deviant role models, and therefore we were not able to examine their explanation for why biological influences on adolescent substance use vary by neighborhood characteristics. The other study found that pubertal timing was associated with alcohol use by girls living in urban areas but not by girls living in rural areas (Dick et al., 2000), such that the earlier the maturation the more likely the girls in urban areas were to drink. The authors suggested that this finding could be explained by greater access to alcohol in urban areas or by differences in attitudes towards early maturation in the two types of settings which may have influenced alcohol consumption. We do not have the variables in our data set to examine either of these possible explanations.

Surprisingly, the family context moderated only a single relationship, that between testosterone and alcohol involvement by girls. Although the interaction was in the expected direction, the point at which the positive association between testosterone and alcohol involvement become significant was 5.81 standard deviations below the mean of the family context variable, a score so extreme that only three of our subjects scored there. The more typical findings related to the family context were that it was significantly associated in bivariate analyses with cigarette and alcohol involvement for both sexes, such that the more harmful the context, the more involvement the adolescent had with cigarettes and alcohol, but that these associations decreased to non-significance in the multivariate models.

For biological variables not involved in significant interactions, we assessed their main effects and when appropriate, mediators of the associations between biological variables and substance use outcomes. That context variables might mediate effects of biological variables on adolescent behaviors is suggested by Petersen (1987) such that biological variables influence adolescent attributes and relationships in a way that increases the risk for problem behaviors. In other words, the associations between biological variables and problem behaviors are mediated by adolescent attributes and relationships with others. Because our focus was moderation, we examined this possibility only when biological variables were not involved in significant interactions. Pubertal status was not involved in any significant interactions when predicting cigarette or alcohol involvement by boys, but it was significantly positively associated with both substances in bivariate analyses and decreased to non-significance in multivariate analyses. The peer context mediated the association between pubertal status and involvement with cigarettes and alcohol by boys. One explanation for these findings is that boys of advanced pubertal development select or are selected by peers who are using and approving of cigarettes and alcohol, and that exposure to those substance-using models and substance use supporting attitudes, along with increased access to substances due to those relationships, facilitate their use of those substances. However, it is important to remember that in both the cigarette and alcohol involvement models for boys, the peer context was involved in a significant interaction with testosterone such that the influence of the peer context on substance use was conditional on the boy's testosterone level.

The association between pubertal status and cigarette involvement by boys also was mediated by the family context. As the pubertal status of the boys increased, the family context became more harmful, and as the family context became more harmful, the boys' cigarette involvement increased. Parents may respond to their son's advanced pubertal development by altering their parenting strategies, such as decreasing their monitoring,

which could lead to an increased likelihood of the boy becoming involved with drugs. Several studies report that parental monitoring and supervision decrease with increased pubertal development of boys, independent of the boys' ages (Anderson, Hetherington, & Clingempeel, 1989). Alternatively, other studies report that increased pubertal development of boys is associated with increased monitoring and supervision of boys and that this decrease in freedom can cause conflict in the parent-child relationship (Papini, Clark, Barnett, & Savage, 1989). Because our family context variable involved a composite of monitoring and parent-child interaction items, we cannot determine which of these processes may have been at work.

This study has several strengths. The first is that the hypotheses are theoretically based; the lack of theoretical guidance is a common criticism of studies examining biological influences on adolescent outcomes. Another strength is that we examined multiple biological variables. Because others have found that biological variables are correlated with each other (Booth et al., 2003; Nottelmann et al., 1987; Susman et al., 1987), we used an analysis strategy that precludes the possibility that the effect of one biological variable on the outcomes is confounded by the other biological variables. Other strengths are that we measured many more variables that could potentially contaminate hormone levels than have been measured in previous studies; we included measures of multiple social contexts important in the lives of adolescents, and the reliability of those measures is high; our outcomes are behavioral, and few studies have examined the associations between hormones and behaviors of adolescents; and the sample size is larger than many studies that have examined the associations between hormones and the behaviors and attributes of adolescents. Also, this is the first study to examine the association between estradiol levels and cigarette involvement by adolescent girls.

The primary study weakness is that the data are cross-sectional and therefore the temporality of relationships cannot be determined. Throughout this manuscript we make an assumption that biological variables influence substance use under varying contexts; that is, an adolescent with a biological propensity to use substances coupled with a harmful context is at increased risk of using substances. An alternative temporal sequence is that cigarette use and alcohol use influence biological variables and biological variables produce changes in social contexts. Several studies of adult women have examined the association between estradiol and cigarette use, and those studies tend to focus on the effect that cigarette use has on ovarian function, and thus estradiol levels and ultimately fertility. In general, adult women who smoke tend to have lower estradiol levels than adult women who do not smoke (Anderson et al., 1989; MacMahon, Trichopoulos, Cole, & Brown, 1982). We found that adolescent girls who smoke have higher estradiol levels than girls who do not smoke. Although it is possible that cigarette use influences estradiol levels of girls, the direction of the relationship found in the adult studies, coupled with the brief smoking histories of these girls due to their young ages, suggests that this is an unlikely temporal sequence. However, given the cross-sectional nature of the data, that explanation cannot be ruled out.

Also given the cross-sectional nature of the data, we examined biosocial processes operating at one specific phase of adolescence—early adolescence. Some researchers have suggested that there may be distinct biosocial processes operating at different times during adolescence that also are relevant to the study of substance use by youth and that developmental processes involve reciprocal relationships, over time, between factors at multiple levels of influence, including biological, psychological, sociological, and environmental (Lerner, 1998; Magnusson & Stattin, 1998). These dynamic biosocial models of development require longitudinal studies.

Another weakness is that for some of the context variables we assessed adolescent perceptions of their social environments rather than their actual social environments; our peer measure is a notable exception. Perceptions are more likely than objective measures of context to be influenced by the adolescent's use of substances. However, few studies are able to obtain objective measures of adolescent family, peer, school, and neighborhood contexts in one study. Also, our findings may have been influenced by the way we defined our context variables and our decision to define a harmful context at or below its mean. Each context variable was composed of several aspects of that context found in earlier studies to be associated with adolescent problem behaviors. Perhaps some of those components were more important for defining a "harmful" context than others. If so, our combined measures would have obscured that importance. However, the internal consistencies of our context measures were high and their correlations with the substance use outcomes were significant and in the expected directions for both boys and girls, lending credence to their definitions.

With respect to the biological measures, our pubertal timing variable was limited. It was a single item self-report of early, on time, or late maturation in relation to peers. Dubas et al. (1991) suggest that perceptions of pubertal timing overlap with actual pubertal timing, but that they are distinct concepts that reflect different biological and psychological processes. Because pubertal timing was not associated with cigarette or alcohol involvement in any of the bivariate analyses, because this lack of a significant effect could not be explained by the fact that pubertal timing was involved in significant interactions, and because one of the only two significant interactions that were not in the hypothesized direction involved pubertal timing, we conclude that there may have been problems with the validity of this measure.

Our findings have many implications for practice and future research. Our findings suggest that programs designed to alter the social context, especially the peer context and the neighborhood context, may be appropriate approaches to decreasing the negative impact of biological variables on adolescent substance use. Further research is needed to inform the specific content of such interventions. For example, additional studies are needed to determine which aspect of each context has the strongest moderating effect. For example our peer context variable is composed of actual peer use of substances, perceived peer use, and peer approval of use. It would be useful for developing peer directed interventions to know which of these aspects of the peer context was most responsible for exacerbating the negative impact of the biological variables on substance use. For developing specific interventions it would also be useful to know what mechanisms are responsible for exacerbating the negative impact of biological variables on substance use in harmful contexts. For example, the authors of studies that found that the association between pubertal timing and problem behaviors of girls is exacerbated in high risk neighborhoods suggested that the mechanisms responsible for the association included greater exposure to older deviant role models, greater access to substances, and differences across neighborhood contexts in the meanings attributed to early physical maturation for girls. Each of these mechanisms suggests different neighborhood intervention approaches. Also, our mediation findings have practical implications. For example we found that the association between pubertal status and cigarette involvement by boys was mediated by the family context; more advanced biological development was associated with a more harmful family context, which was associated with greater cigarette involvement. These findings, along with those of others (Anderson, Hetherington, & Clingempeel, 1989; Papini, Clark, Barnett, & Savage, 1989), suggest that parent-based substance use prevention interventions should include a component on how to respond to a child's advancing pubertal development in ways that do not increase the child's likelihood of becoming involved with substances.

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Table 1

Correlation Matrix

	(1)	(2)	(3)	(4) ^a	(5)	(6)	(7)	(8) ^a	(9) ^a	(10)	(11)	(12)	(13)	(14)	(15)	(16)
(1) Cigarette involvement	1.00		.20**	---	.18**	.03	.15**	---	---	-.22**	-.61**			-.11		-.15*
(2) Alcohol involvement		1.00	.22**	---	.24**	.13	.27**	---	---	---	-.30**		-.52**		-.21**	-.28**
(3) Testosterone	.08	-.03	1.00	---	.60**	.20**	.63**	---	---	-.32**	-.37**		-.36**	-.21**	-.17**	-.17**
(4) Estradiol ^a	.22**	.22**	.45**	1.00				---	---	---	---		---	---	---	---
(5) Pubertal status	.28**	.21**	.25**	.37**	1.00	.27**	.48**	---	---	-.26**	-.28**		-.26**	-.17**	-.17**	-.10
(6) Pubertal timing	-.01	.04	.20**	.32**	.36**	1.00	.16*	---	---	-.03	-.02		-.05	.06	.07	-.06
(7) Age	.30**	.17*	.13	.34**	.53**	.03	1.00	---	---	-.31**	-.31**		-.38**	-.20**	-.17**	-.17**
(8) Menstrual status ^a	.28**	.21**	.25**	.36**	.69**	.23**	.53**	1.00	---	---	---		---	---	---	---
(9) Current menstruation ^a	.18**	.27**	-.02	-.13	.26**	.08	.19**	.39**	1.00	---	---		---	---	---	---
(10) FC cigarettes	-.22**		-.10	-.14*	-.05	-.02	-.18**	-.17**	.01	1.00		.38**		.28**		.47**
(11) FC alcohol		-.22**	-.11	-.15*	-.05	-.02	-.17*	-.16*	.03		1.00		.45**		.32**	.48**
(12) PC cigarettes	-.58**		-.04	-.13	-.21**	.08	-.35**	-.27**	-.24**	.28**		1.00		.18**		.25**
(13) PC alcohol		-.48**	.03	-.12	-.27**	.02	-.33**	-.28**	-.24**		.23**		1.00		.18**	.28**
(14) SC cigarettes	-.32**		.08	.01	-.19**	.10	-.24**	-.20**	-.14*	.22**		.23**		1.00		.28**
(15) SC alcohol		-.24**	.09	.03	-.16*	.07	-.17*	-.22**	-.19**		.28**		.25**		1.00	.27**
(16) NC	-.20**	-.13	-.08	-.07	-.01	.03	-.08	-.02	-.06	.41**	.11	.15*	.15*	.24**	.33**	1.00

Note. Data for boys above diagonal (N=210). Data for girls below diagonal (N=199). FC = Family context. PC = Peer context. SC = School context. NC = Neighborhood context. The substance use outcomes were correlated only with the corresponding substance-specific contextual variables.

^aNot relevant for boys.

* $p < .05$.

** $p < .01$.

Table 2

Cigarette and Alcohol Involvement by Boys

	Cigarette Involvement			Alcohol Involvement		
	B	S.E. B	95% CI	B	S.E. B	95% CI
Intercept	3.7630**	0.13	3.499, 4.027	2.817***	0.12	2.573, 3.061
Testosterone	0.1789	0.15	-0.121, 0.479	-0.106	0.14	-0.382, 0.171
Pubertal status	0.0169	0.26	-0.497, 0.531	0.168	0.24	-0.310, 0.646
Pubertal timing	-0.0004	0.14	-0.284, 0.283	0.101	0.13	-0.164, 0.367
Age	-0.0204	0.01	-0.044, 0.003	0.013	0.01	-0.009, 0.034
Family (FC)	-0.0128	0.06	-0.130, 0.104	-0.023	0.06	-0.137, 0.090
Peer (PC)	-0.4605**	0.08	-0.626, -0.295	-0.189**	0.07	-0.319, -0.059
School (SC)	0.0471	0.08	-0.120, 0.214	-0.114	0.08	-0.271, 0.043
Neighborhood (NC)	-0.0858	0.14	-0.356, 0.184	-0.346**	0.12	-0.592, -0.100
Testosterone X FC	0.0643	0.07	-0.065, 0.194	0.103	0.06	-0.024, 0.230
Pubertal status X FC	0.1189	0.13	-0.144, 0.382	-0.007	0.13	-0.258, 0.243
Pubertal timing X FC	-0.0949	0.08	-0.248, 0.059	-0.049	0.07	-0.193, 0.094
Testosterone X PC	-0.1887*	0.08	-0.354, -0.024	-0.168*	0.07	-0.308, -0.028
Pubertal status X PC	-0.0797	0.15	-0.366, 0.207	-0.026	0.12	-0.256, 0.204
Pubertal timing X PC	0.1718*	0.07	0.032, 0.312	-0.012	0.06	-0.128, 0.103
Testosterone X SC	-0.0584	0.09	-0.232, 0.116	0.147	0.08	-0.018, 0.312
Pubertal status X SC	0.0003	0.18	-0.359, 0.360	-0.015	0.16	-0.337, 0.308
Pubertal timing X SC	-0.0453	0.11	-0.271, 0.180	-0.110	0.10	-0.314, 0.093
Testosterone X NC	-0.0337	0.14	-0.300, 0.233	0.109	0.13	-0.138, 0.356
Pubertal status X NC	-0.4284	0.27	-0.965, 0.108	-0.415	0.24	-0.892, 0.062
Pubertal timing X NC	0.1100	0.17	-0.222, 0.442	0.174	0.15	-0.126, 0.474

Note. CI = confidence interval. FC = Family context. PC = Peer context. SC = School context. NC = Neighborhood context.

* $p < .05$.

** $p < .01$.

Table 3

Cigarette and Alcohol Involvement by Girls.

	Cigarette Involvement			Alcohol Involvement		
	B	S.E. B	95% CI	B	S.E. B	95% CI
Intercept	3.663**	0.12	3.43, 3.90	2.45**	0.10	2.261, 2.642
Testosterone	0.039	0.21	-0.38, 0.46	-0.42*	0.18	-0.770, -0.067
Estradiol	0.146	0.16	-0.17, 0.46	0.27*	0.13	0.019, 0.516
Pubertal status	0.592	0.34	-0.08, 1.26	0.38	0.27	-0.148, 0.916
Pubertal timing	-0.060	0.15	-0.36, 0.24	0.06	0.12	-0.189, 0.299
Age	-0.008	0.01	-0.03, 0.02	-0.01	0.01	-0.033, 0.005
Menstrual status	0.145	0.35	-0.55, 0.84	-0.08	0.28	-0.630, 0.479
Current menstruation	0.007	0.35	-0.68, 0.69	0.56*	0.28	0.012, 1.117
Family (FC)	0.005	0.06	-0.11, 0.12	-0.06	0.05	-0.149, 0.034
Peer (PC)	-0.377**	0.07	-0.51, -0.24	-0.25**	0.06	-0.379, -0.128
School (SC)	-0.235**	0.08	-0.39, -0.07	-0.09	0.07	-0.221, 0.047
Neighborhood (NC)	-0.299*	0.14	-0.58, -0.02	-0.08	0.11	-0.301, 0.144
Testosterone X FC	-0.006	0.11	-0.22, 0.21	-0.20*	0.09	-0.364, -0.028
Estradiol X FC	-0.017	0.08	-0.17, 0.13	0.03	0.06	-0.091, 0.149
Pubertal status X FC	-0.087	0.15	-0.38, 0.20	-0.02	0.11	-0.240, 0.195
Pubertal timing X FC	-0.047	0.07	-0.19, 0.10	-0.07	0.06	-0.179, 0.045
Testosterone X PC	0.019	0.12	-0.21, 0.25	0.45**	0.11	0.235, 0.660
Estradiol X PC	0.048	0.08	-0.11, 0.21	-0.05	0.07	-0.185, 0.088
Pubertal status X PC	-0.384**	0.15	-0.68, -0.09	-0.34*	0.14	-0.621, -0.062
Pubertal timing X PC	0.014	0.07	-0.12, 0.15	-0.13	0.08	-0.286, 0.035
Testosterone X SC	-0.059	0.15	-0.36, 0.24	0.21	0.12	-0.020, 0.439
Estradiol X SC	0.010	0.10	-0.20, 0.22	0.01	0.09	-0.155, 0.181
Pubertal status X SC	-0.133	0.17	-0.46, 0.19	-0.24	0.13	-0.497, 0.020
Pubertal timing X SC	0.086	0.08	-0.08, 0.25	0.09	0.08	-0.063, 0.238
Testosterone X NC	-0.008	0.26	-0.53, 0.51	-0.03	0.21	-0.450, 0.381

	Cigarette Involvement				Alcohol Involvement			
	B	S.E.	B	95% CI	B	S.E.	B	95% CI
Estradiol X NC	-0.453*	0.19	-0.82, -0.08	-0.47**	0.15	-0.764, -0.177		
Pubertal status X NC	-0.183	0.31	-0.80, 0.44	0.34	0.26	-0.165, 0.850		
Pubertal timing X NC	0.275	0.16	-0.04, 0.59	0.12	0.13	-0.141, 0.378		

Note. CI = confidence interval. FC = Family context. PC = Peer context. SC = School context. NC = Neighborhood context.

* $p < .05$.

** $p < .01$.