A Test of Biosocial Models of Adolescent Cigarette and Alcohol Involvement

Vangie A. Foshee, Susan T. Ennett, Karl E. Bauman, Douglas A. Granger, Thad Benefield, Chirayath Suchindran, Andrea M. Hussong, Katherine J. Karriker-Jaffe and Robert H. DuRant

The Journal of Early Adolescence 2007; 27; 4
DOI: 10.1177/0272431606294830

Journal of Early Adolescence
Volume 27 Number 1
February 2007 4-39

10.1177/0272431606294830
http://jea.sagepub.com
hosted at
http://online.sagepub.com

A Test of Biosocial Models of Adolescent Cigarette and Alcohol Involvement

Vangie A. Foshee
Susan T. Ennett
Karl E. Bauman
University of North Carolina at Chapel Hill

Douglas A. Granger
Pennsylvania State University, University Park

Thad Benefield
Chirayath Suchindran
Andrea M. Hussong
Katherine J. Karriker-Jaffe
University of North Carolina at Chapel Hill

Robert H. DuRant
Wake Forest University School of Medicine, North Carolina

The authors test 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 explain significantly more variance in adolescent cigarette and alcohol involvement than do models including only the main effects of the biological and contextual variables. Post hoc analyses of significant interactions suggest 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 become positive and stronger as the context becomes more harmful. Considerations of adolescent substance use 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,

Authors' Note: This study was funded by National Cancer Institute Grant No. CA16086 administered through the Lineberger Comprehensive Cancer Center at the University of North Carolina at Chapel Hill. This study was reviewed and approved by the Institutional Review Boards at the University of North Carolina at Chapel Hill School of Public Health and the Wake Forest University School of Medicine. We would like to thank Ruth Merritt, Laurie O'Brien, Eve Schwartz, Carla Slike, and Mary Curran at Salimetrics LLC, State College, Pennsylvania, for technical assistance with saliva sample collection and assay. Correspondence concerning this article should be addressed to Vangie A. Foshee, 319B Rosenau Hall, Department of Health Behavior and Health Education, School of Public Health, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7440; e-mail: foshee@email.unc.edu.

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 involvement 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 is 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, Johnson, Granger, Crouter, and 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, Foshee, and 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. (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, Maughan, Worthman, Costello, and Angold (in press) found that peer deviance moderated the association between testosterone and adolescent nonaggressive conduct disorder symptoms, with the strongest positive association being among the boys who had deviant peers. In a study of adolescent sexual behavior, Smith, Udry, and Morris (1985) found that the association between pubertal status and adolescent sexual behavior was moderated by a 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, Lynam, Moffitt, and Silva (1993) found that the association between pubertal timing and delinquency was moderated by characteristics of the school.

Finally, with regard to neighborhoods, Obeidallah, Brennan, Brooks-Gunn, and 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 (SES) 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 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; C. Jackson, Henriksen, & Foshee, 1998), do not set and enforce clear standards of behavior (Baumrind, 1985, 1991; C. 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. Although cigarette and alcohol use do not peak until later in adolescence (K. M. 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.

Method

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 9 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 (SAS Institute, 1999) 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 (SD = .57) for sixth graders and 13.6 (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 procedures such as 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, with higher scores indicating 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, Texas) modified for use with saliva (Granger, Schwartz, Booth, & Arentz, 1999). The test uses 200 µl 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, Texas) modified for use with saliva (Shirtcliff et al., 2000). The test uses 300 ml of saliva (for singlet determination) and has a minimum detection limit of 0.25 pg/µl (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 comprised 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 (C. 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 4-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 or her school to smoke cigarettes (drink alcohol) 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 or 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 include "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 include "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 during 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" 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

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		0.20**	—	0.18**	0.03	0.15**	—	—	–0.22**		–0.61**		–0.11		–0.15*
2. Alcohol involvement		1.00	0.22**	—	0.24**	0.13	0.27**	—	—		–0.30**		–0.52**		–0.21**	–0.28**
3. Testosterone	0.08	–0.03	1.00	—	0.60**	0.20**	0.63**	—	—	–0.32**	–0.33**	–0.37**	–0.36**	–0.21**	–0.17**	–0.17**
4. Estradiol[a]	0.22**	0.22**	0.45**	1.00				—	—	—	—	—	—	—	—	—
5. Pubertal status	0.28**	0.21**	0.25**	0.37**	1.00	0.27**	0.48**	—	—	–0.26**	–0.28**	–0.28**	–0.26**	–0.17**	–0.17**	–0.10
6. Pubertal timing	–0.01	0.04	0.20**	0.32**	0.36**	1.00	0.16*	—	—	–0.03	–0.02	–0.06	–0.05	0.06	0.07	–0.06
7. Age	0.30**	0.17*	0.13	0.34**	0.53**	0.03	1.00	—	—	–0.31**	–0.31**	–0.40**	–0.38**	–0.20**	–0.17**	–0.17**
8. Menstrual status[a]	0.28**	0.21**	0.25**	0.36**	0.69**	0.23**	0.53**	1.00	—	—	—	—	—	—	—	—
9. Current menstruation[a]	0.18**	0.27**	–0.02	–0.13	0.26**	0.08	0.19**	0.39**	1.00	—	—	—	—	—	—	—
10. FC cigarettes	–0.22**		–0.10	–0.14*	–0.05	–0.02	–0.18**	–0.17**	0.01	1.00		0.38**		0.28**		0.47**
11. FC alcohol		–0.22**	–0.11	–0.15*	–0.05	–0.02	–0.17*	–0.16*	0.03		1.00		0.45**		0.32**	0.48**
12. PC cigarettes	–0.58**		–0.04	–0.13	–0.21**	0.08	–0.35**	–0.27**	–0.24**	0.28**		1.00		0.18**		0.25**
13. PC alcohol		–0.48**	0.03	–0.12	–0.27**	0.02	–0.33**	–0.28**	–0.24**		0.23**		1.00		0.18**	0.28**
14. SC cigarettes	–0.32**		0.08	0.01	–0.19**	0.10	–0.24**	–0.20**	–0.14*	0.22**		0.23**		1.00		0.28**
15. SC alcohol		–0.24**	0.09	0.03	–0.16*	0.07	–0.17*	–0.22**	–0.19**		0.28**		0.25**		1.00	0.27**
16. NC	–0.20**	–0.13	–0.08	–0.07	–0.01	0.03	–0.08	–0.02	–0.06	0.41**	0.41**	0.11	0.15*	0.24**	0.33**	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.

a. Not relevant for boys.

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

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 of this article, 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 and 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 (1982) test. 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 eigen analyses (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 (ΔR^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 .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 .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

Table 2
Cigarette and Alcohol Involvement by Boys

	Cigarette Involvement			Alcohol Involvement		
	B	*SE B*	95% CI	*B*	*SE B*	95% CI
Intercept	3.7630**	.13	3.499, 4.027	2.817**	.12	2.573, 3.061
Testosterone	0.1789	.15	–0.121, 0.479	–0.106	.14	–0.382, 0.171
Pubertal status	0.0169	.26	–0.497, 0.531	0.168	.24	–0.310, 0.646
Pubertal timing	–0.0004	.14	–0.284, 0.283	0.101	.13	–0.164, 0.367
Age	–0.0204	.01	–0.044, 0.003	0.013	.01	–0.009, 0.034
Family (FC)	–0.0128	.06	–0.130, 0.104	–0.023	.06	–0.137, 0.090
Peer (PC)	–0.4605**	.08	–0.626, –0.295	–0.189**	.07	–0.319, –0.059
School (SC)	0.0471	.08	–0.120, 0.214	–0.114	.08	–0.271, 0.043
Neighborhood (NC)	–0.0858	.14	–0.356, 0.184	–0.346**	.12	–0.592, –0.100
Testosterone × FC	0.0643	.07	–0.065, 0.194	0.103	.06	–0.024, 0.230
Pubertal Status × FC	0.1189	.13	–0.144, 0.382	–0.007	.13	–0.258, 0.243
Pubertal Timing × FC	–0.0949	.08	–0.248, 0.059	–0.049	.07	–0.193, 0.094
Testosterone × PC	–0.1887*	.08	–0.354, –0.024	–0.168*	.07	–0.308, –0.028
Pubertal Status × PC	–0.0797	.15	–0.366, 0.207	–0.026	.12	–0.256, 0.204
Pubertal Timing × PC	0.1718*	.07	0.032, 0.312	–0.012	.06	–0.128, 0.103
Testosterone × SC	–0.0584	.09	–0.232, 0.116	0.147	.08	–0.018, 0.312
Pubertal Status × SC	0.0003	.18	–0.359, 0.360	–0.015	.16	–0.337, 0.308
Pubertal Timing × SC	–0.0453	.11	–0.271, 0.180	–0.110	.10	–0.314, 0.093
Testosterone × NC	–0.0337	.14	–0.300, 0.233	0.109	.13	–0.138, 0.356
Pubertal Status × NC	–0.4284	.27	–0.965, 0.108	–0.415	.24	–0.892, 0.062
Pubertal Timing × NC	0.1100	.17	–0.222, 0.442	0.174	.15	–0.126, 0.474

Note: CI = confidence interval; FC = family context; PC = peer context; SC = school context; NC = neighborhood context.
*$p < .05$. **$p < .01$.

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 (ΔR^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 .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 .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

Table 3
Cigarette and Alcohol Involvement by Girls

	Cigarette Involvement			Alcohol Involvement		
	B	*SE B*	95% CI	*B*	*SE B*	95% CI
Intercept	3.663**	.12	3.43, 3.90	2.45**	.10	2.261, 2.642
Testosterone	0.039	.21	–0.38, 0.46	–0.42*	.18	–0.770, –0.067
Estradiol	0.146	.16	–0.17, 0.46	0.27*	.13	0.019, 0.516
Pubertal status	0.592	.34	–0.08, 1.26	0.38	.27	–0.148, 0.916
Pubertal timing	–0.060	.15	–0.36, 0.24	0.06	.12	–0.189, 0.299
Age	–0.008	.01	–0.03, 0.02	–0.01	.01	–0.033, 0.005
Menstrual status	0.145	.35	–0.55, 0.84	–0.08	.28	–0.630, 0.479
Current menstruation	0.007	.35	–0.68, 0.69	0.56*	.28	0.012, 1.117
Family (FC)	0.005	.06	–0.11, 0.12	–0.06	.05	–0.149, 0.034
Peer (PC)	–0.377**	.07	–0.51, –0.24	–0.25**	.06	–0.379, –0.128
School (SC)	–0.235**	.08	–0.39, –0.07	–0.09	.07	–0.221, 0.047
Neighborhood (NC)	–0.299*	.14	–0.58, –0.02	–0.08	.11	–0.301, 0.144
Testosterone × FC	–0.006	.11	–0.22, 0.21	–0.20*	.09	–0.364, –0.028
Estradiol × FC	–0.017	.08	–0.17, 0.13	0.03	.06	–0.091, 0.149
Pubertal Status × FC	–0.087	.15	–0.38, 0.20	–0.02	.11	–0.240, 0.195
Pubertal Timing × FC	–0.047	.07	–0.19, 0.10	–0.07	.06	–0.179, 0.045
Testosterone × PC	0.019	.12	–0.21, 0.25	0.45**	.11	0.235, 0.660
Estradiol × PC	0.048	.08	–0.11, 0.21	–0.05	.07	–0.185, 0.088
Pubertal Status × PC	–0.384**	.15	–0.68, –0.09	–0.34*	.14	–0.621, –0.062
Pubertal Timing × PC	0.014	.07	–0.12, 0.15	–0.13	.08	–0.286, 0.035
Testosterone × SC	–0.059	.15	–0.36, 0.24	0.21	.12	–0.020, 0.439
Estradiol × SC	0.010	.10	–0.20, 0.22	0.01	.09	–0.155, 0.181
Pubertal Status × SC	–0.133	.17	–0.46, 0.19	–0.24	.13	–0.497, 0.020
Pubertal Timing × SC	0.086	.08	–0.08, 0.25	0.09	.08	–0.063, 0.238
Testosterone × NC	–0.008	.26	–0.53, 0.51	–0.03	.21	–0.450, 0.381
Estradiol × NC	–0.453*	.19	–0.82, –0.08	–0.47**	.15	–0.764, –0.177
Pubertal Status × NC	–0.183	.31	–0.80, 0.44	0.34	.26	–0.165, 0.850
Pubertal Timing × NC	0.275	.16	–0.04, 0.59	0.12	.13	–0.141, 0.378

Note: CI = confidence interval; FC = family context; PC = peer context; SC = school context; NC = neighborhood context.
*$p < .05$. **$p < .01$.

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 .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 nonsignificance 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 of 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 nonexistent 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, it is interesting that 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 SES. 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 toward 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.

It is surprising that 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 nonsignificance 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 nonsignificance 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 because of 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 article, 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 because of 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 comprised 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 comprises 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 et al., 1989; Papini et al., 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.

References

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.

Anderson, E. R., Hetherington, E. M., & Clingempeel, W. G. (1989). Transformations in family relations at puberty: Effects of family context. *Journal of Early Adolescence*, *9*, 310-334.

Andrews, J. A., Hops, H., Ary, D., Tildesley, E., & Harris, J. (1993). Parental influence on early adolescent substance use: Specific and nonspecific effects. *Journal of Early Adolescence*, *13*, 285-310.

Anthony, J. C., & Petronis, K. R. (1995). Early-onset drug use and risk of later drug problems. *Drug and Alcohol Dependence*, *40*, 9-15.

Bauer, D. J., & Curran, P. J. (in press). Probing interactions in fixed and multilevel regression: Inferential and graphical techniques. *Multivariate Behavioral Research*.

Bauman, K. E., Foshee, V. A., & Haley, N. J. (1992). The interaction of sociological and biological factors in adolescent cigarette smoking. *Addictive Behaviors*, *17*, 459-467.

Bauman, K. E., Foshee, V. A., Koch, G. G., Haley, N. J., & Downton, M. I. (1989). Testosterone and cigarette smoking in early adolescence. *Journal of Behavioral Medicine*, *12*, 425-432.

Baumrind, D. (1985). Familial antecedents of adolescent drug use: A developmental perspective. In C. L. Jones & R. L. Battjes (Eds.), *Etiology of drug abuse: Implications for prevention* (NIDA Research Monograph No. 56, pp. 13-44). Rockville, MD: National Institute on Drug Abuse.

Baumrind, D. (1991). The influences of parenting style on adolescent competence and substance use. *Journal of Early Adolescence*, *11*, 56-95.

Belsley, D. A., Kuh, E., & Welsch, R. E. (1980). *Regression diagnostics: Identifying influential data and sources of collinearity*. New York: John Wiley & Sons.

Booth A., Johnson, D. R., Granger, D. A., Crouter, A. C., & McHale, S. (2003). Testosterone and child and adolescent adjustment: The moderating role of parent-child relationships. *Developmental Psychology*, *39*, 85-98.

Botvin, G. J., Baker, E., Goldberg, C. J., Dusenbury, L., & Botvin, E. M. (1992). Correlates and predictors of smoking among Black adolescents. *Addictive Behaviors*, *17*, 97-103.

Brennan, P. A., & Raine, A. (1997). Biosocial bases of antisocial behavior: Psychological, neurological, and cognitive factors. *Clinical Psychology Review*, *17*, 589-604.

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Cambridge, MA: Harvard University Press.

Brook, J. S., Brook, D. W., Gordon, A. S., Whiteman, M., & Cohen, P. (1990). The psychological etiology of drug use: A family interactional approach. *Genetic, Social, and General Psychology Monographs*, *116*, 111-127.

Brook, J., Nomura, C., & Cohen, P. (1989). A network of influences on adolescent drug involvement: Neighborhood, school, peer, and family. *Genetics, Social, and General Psychological Monographs*, *115*, 125-145.

Brook, J. S., Whiteman, M., Gordon, A. S., & Cohen, P. (1986). Dynamics of childhood and adolescent personality traits and adolescent drug use. *Developmental Psychology*, *22*, 403-414.

Brooks-Gunn, J. (1987). Pubertal processes and girl's psychological adaption. In R. M. Lerner & T. T. Foch (Eds.), *Biological-psychological interactions in early adolescence* (pp. 123-154). Hillsdale, NJ: Lawrence Erlbaum.

Buikhuisen, W. (1988). Chronic juvenile delinquency: A theory. In W. Buikhuisen & S. A. Mednick (Eds.), *Explaining criminal behavior* (pp. 27-50). Leiden, the Netherlands: E. J. Brill.

Caspi, A., Lynam, D., Moffitt, T. E., & Silva, P. A. (1993). Unraveling girls' delinquency: Biological, dispositional, and contextual contributions to adolescent misbehavior. *Developmental Psychology*, *29*(1), 19-30.

Chard, T. (1990). *An introduction to radioimmunoassay and related techniques* (4th ed.). Amsterdam: Elsevier.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum.

Constantino, J. N., Grosz, D., Saenger, P., Chandler, D. W., Nadi, R., & Earls, F. J. (1993). Testosterone and aggression in children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *32*, 1217-1222.

Cota-Robles, S., Neiss, M., & Rowe, D. C. (2002). The role of puberty in violent and nonviolent delinquency among Anglo American, Mexican American, and African American boys. *Journal of Adolescent Research*, *17*, 364-376.

Curran, P. J., Bauer, D. J., & Willoughby, M. T. (in press). Testing and probing interactions in hierarchical linear growth models. In C. S. Bergeman & S. M. Boker (Eds.), *The Notre Dame series on quantitative methodology, Volume 1: Methodological issues in aging research*. Mahwah, NJ: Lawrence Erlbaum.

Dabbs, J. M., Jurkovic, G. J., & Frady, R. L. (1991). Salivary testosterone and cortisol among late adolescent male offenders. *Journal of Abnormal Child Psychology*, *19*, 469-478.

Dabbs, J. M., & Morris, R. (1990). Testosterone, social class, and antisocial behavior in a sample of 4,462 men. *Psychological Science*, *1*, 209-211.

DeWit, D., Adlaf, E., Offord, D., & Ogborne, A. (2000). Age at first alcohol use: A risk factor for the development of alcohol disorders. *American Journal of Psychiatry*, *157*, 745-750.

Dick, D. M., Rose, R. J., Viken, R. J., & Kaprio, J. (2000). Pubertal timing and substance use: Associations between and within families across late adolescence. *Developmental Psychology*, *36*(2), 180-189.

Dubas, J. S., Graber, J. A., & Petersen, A. C. (1991). A longitudinal investigation of adolescents' changing perceptions of pubertal timing. *Developmental Psychology*, *27*(4), 580-586.

Ennett, S. T., Flewelling, R. L., Lindrooth, R. C., & Norton, E. C. (1997). School and neighborhood characteristics associated with school rates of alcohol, cigarette, and marijuana use. *Journal of Health and Social Behavior*, *38*, 55-71.

Eysenck, H. J. (1964). *Crime and personality* (1st ed.). London: Methuen.

Fendrich, M., & Johnson, T. P. (2001). Examining prevalence differences in three national surveys of youth: Impact of consent procedures, modes, and editing rules. *Journal of Drug Issues*, *31*(3), 615-642.

Finkelstein, J. W., Susman, E. J., Chinchilli, V. M., Kunselman, S. J., D'Arcangelo, M. R., Schwab, J., et al. (1997). Estrogen or testosterone increases self-reported aggressive behaviors in hypogonadal adolescents. *Journal of Clinical Endocrinology and Metabolism*, *82*, 2433-2438.

Flannery, D. J., Rowe, D. C., & Gulley, B. L. (1993). Impact of pubertal status, timing, and age on adolescent sexual experience and delinquency. *Journal of Adolescent Research*, *8*(1), 21-40.

Flay, B. R., & Petraitis, J. (1994). The theory of triadic influence: A new theory of health behavior with implications for preventive interventions. *Advances in Medical Sociology*, *4*, 19-44.

Frazier, P. A., Tix, A. P., & Barron, K. E. (2004). Testing moderator and mediator effects in counseling psychology research. *Journal of Counseling Psychology*, *51*(1), 115-134.

Garmezy, N., Masten, A. S., & Tellegen, A. (1984). The study of stress and competence in children: A building block for developmental psychopathology. *Child Development*, *55*, 97-111.

Ge, X., Brody, G., Conger, R. D., Simons, R. L., & Murry, V. M. (2002). Contextual amplification of pubertal transition effects on deviant peer affiliation and externalizing behavior among African American children. *Developmental Psychology*, *38*(1), 42-54.

Ge, X., Conger, R. D., & Elder, G. H. (2001). The relation between puberty and psychological distress in adolescent boys. *Journal of Research on Adolescence*, *11*(1), 49-70.

Granger, D. A., Schwartz, E. B., Booth, A., & Arentz, M. (1999). Salivary testosterone determination in studies of child health and development. *Hormones and Behavior*, *35*, 18-27.

Halpern, C. T., Udry, J. R., Campbell, B., & Suchindran, C. (1993). Relationships between aggression and pubertal increases in testosterone: A panel analysis of adolescent males. *Social Biology*, *40*(1-2), 8-24.

Hamburg, B. (1974). Early adolescence: A specific and stressful stage of the life cycle. In G. Coelho, D. A. Hamburg, & J. E. Adams (Eds.), *Coping and adaptation* (pp. 101-124). New York: Basic Books.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin*, *112*, 64-105.

Hill, J. P. (1983). Early adolescence: A research agenda. *Journal of Early Adolescence*, *3*(1-2), 1-21.

Hollister-Wagner, G., Foshee, V. A., & Jackson, C. (2001). Adolescent aggression: Models of resiliency. *Journal of Applied Social Psychology*, *31*, 445-466.

Huba, G. J., Wingard, J. A., & Bentler, P. M. (1980). Framework for an interactive theory of drug use. In D. J. Lettieri, M. Sayers, & H. W. Pearson (Eds.), *Theories on drug abuse: Selected contemporary perspectives* (Research Monograph 30, pp. 95-101). Rockville, MD: National Institute on Drug Abuse.

Inoff-Germain, G., Arnold, G. S., Nottelmann, E. D., Susman, E. J., Cutler, G. B., Jr., & Chrousos, G. P. (1988). Relations between hormone levels and observational measures of aggressive behavior of young adolescents in family interactions. *Developmental Psychology*, *24*, 129-139.

Jackson, C., Henriksen, L., & Foshee, V. A. (1998). The authoritative parenting index: Predicting health risk behaviors among children and adolescents. *Health Education and Behavior*, *25*, 319-337.

Jackson, K. M., Sher, K. J., Cooper, M. L., & Wood, P. K. (2002). Adolescent alcohol and tobacco use: Onset, persistence and trajectories of use across two samples. *Addiction*, *97*, 517-531.

Jessor, R. (1991). Risk behavior in adolescence: A psychosocial framework for understanding and action. *Journal of Adolescent Health*, *12*, 597-605.

Kandel, D. B., & Andrews, K. (1987). Processes of adolescent socialization by parents and peers. *International Journal of the Addictions*, *22*, 319-342.

Kivlighan, K. T., Granger, D. A., & Schwartz, E. B. (2005). Blood contamination and the measurement of salivary progesterone and estradiol. *Hormones and Behavior*, *47*, 367-370.

Kivlighan, K. T., Granger, D. A., Schwartz, E. B., Nelson, V., & Curran, M. (2004). Quantifying blood leakage into the oral mucosa and its effects on the measurement of cortisol, dehydroepiandrosterone, and testosterone in saliva. *Hormones and Behavior*, *46*, 39-46.

Komro, K. A., Perry, C. L., Williams, C. L., Stigler, M. H., Farbakhsh, K., & Veblen-Mortenson, S. (2001). How did Project Northland reduce alcohol use among young adolescents? Analysis of mediating variables. *Health Education Research*, *16*, 59-70.

Lee, P. A. (1980). Normal ages of pubertal events among American males and females. *Journal of Adolescent Health Care*, *1*, 26-29.

Lerner, R. M. (1987). A life-span perspective for early adolescence. In R. M. Lerner & T. T. Foch (Eds.), *Biological-psychological interactions in early adolescence* (pp. 9-34). Hillsdale, NJ: Lawrence Erlbaum.

Lerner, R. M. (1998). Theories of human development: Contemporary perspectives. In W. Damon (Series Ed.) & R. M. Lerner (Vol. Ed.), *Handbook of child psychology: Volume 1. Theoretical models of human development* (5th ed., pp. 1-24). New York: John Wiley & Sons.

Lerner, R. M., & Foch, T. T. (Eds.). (1987). *Biological-psychosocial interactions in early adolescence*. Hillsdale, NJ: Lawrence Erlbaum.

MacKinnon, D. P., & Dwyer, J. H. (1993). Estimating mediated effects in prevention studies. *Evaluation Review*, *17*, 144-158.

MacMahon, B., Trichopoulos, D., Cole, P., & Brown, J. (1982). Cigarette smoking and urinary estrogens. *New England Journal of Medicine*, *307*, 1062-1065.

Magnusson, D., & Stattin, H. (1998). Person-context interaction theories. In W. Damon (Series Ed.) & R. M. Lerner (Vol. Ed.), *Handbook of child psychology: Volume 1. Theoretical models of human development* (5th ed., pp. 685-759). New York: John Wiley & Sons.

Magnusson, D., Stattin, H., & Allen, V. L. (1986). Differential maturation among girls and its relation to social adjustment: A longitudinal perspective. In D. L. Featherman & R. M. Lerner (Eds.), *Life-span development and behavior, Vol. 7* (pp. 135-172). New York: Academic Press.

Mattsson, A., Schalling, D., Olweus, D., Low, H., & Svensson, A. (1980). Plasma testosterone, aggressive behavior, and personality dimensions in young male delinquents. *Journal of the American Academy of Child Psychiatry*, *19*, 476-490.

Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral and Brain Sciences*, *21*, 353-363.

Mednick, S. A. (1977). A biosocial theory of the learning of law-abiding behavior. In S. A. Mednick & K. O. Christiansen (Eds.), *Biosocial bases of criminal behavior* (pp. 1-8). New York: Gardner.

Nottelmann, E. D., Susman, E. J., Dorn, L. D., Inoff-Germain, G., Loriaux, D. L., Cutler, G. B., et al. (1987). Developmental processes in early adolescence. *Journal of Adolescent Health Care*, *8*, 246-260.

Obeidallah, D., Brennan, R. T., Brooks-Gunn, J., & Earls, F. (2004). Links between pubertal timing and neighborhood contexts: Implications for girl's violent behavior. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(2), 1460-1468.

Olweus, D., Mattsson, A., Schalling, D., & Low, H. (1980). Testosterone, aggression, physical, and personality dimensions in normal adolescent males. *Psychosomatic Medicine*, *42*, 253-269.

Olweus, D., Mattsson, A., Schalling, D., & Low, H. (1988). Circulating testosterone levels and aggression in adolescent males: A causal analysis. *Psychosomatic Medicine*, *50*, 261-272.

Papini, D., Clark, S., Barnett, J., & Savage, C. (1989). Grade, pubertal status, and gender-related variations in conflictual issues among adolescents. *Adolescence*, *24*, 977-987.

Petersen, A. C. (1987). The nature of biological-psychosocial interactions: The sample case of early adolescence. In R. M. Lerner & T. T. Foch (Eds.), *Biological-psychological interactions in early adolescence* (pp. 35-61). Hillsdale, NJ: Lawrence Erlbaum.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth & Adolescence*, *17*, 117-133.

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2003). *Probing interactions in multiple linear regression, latent curve analyses, and hierarchical linear modeling*. Retrieved November 14, 2005, from http://www.unc.edu/%7Epreacher/interact/index.html

Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and adults: A review. *Journal of Abnormal Child Psychology*, *30*, 311-326.

Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., et al. (1997). Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health. *Journal of the American Medical Association*, *278*, 823-832.

Richards, M. H., Abell, S., & Petersen, A. C. (1993). Biological development. In P. H. Tolan & B. J. Cohler (Eds.), *Handbook of clinical research and practice with adolescents* (Wiley series on personality processes, pp. 21-44). Oxford, UK: John Wiley & Sons.

Roberts, W., Hom, A., & Battistich, V. (1995, April). *Assessing students' and teachers' sense of the school as a caring community*. Paper presented at the annual meeting of the American Educational Research Association, San Francisco.

Rowe, R., Maughan, B., Worthman, C. M., Costello, E. J., & Angold, A. (in press). Testosterone, antisocial behavior and social dominance in boys: Pubertal development and biosocial interaction. *Biobehavioral Health*.

Rubin, D. B. (1987). *Multiple imputation for non-response in surveys*. New York: Wiley.

Sampson, R. J., Raudenbush, S. W., & Earls, F. (1997). Neighborhoods and violent crime: A multilevel study of collective efficacy. *Science*, *277*, 918-924.

SAS Institute. (1999). *Statistical Analysis Software (SAS) v. 8.0*. Cary, NC: Author.

Shirtcliff, E. A., Granger, D. A., Schwartz, E. B., Curran, M. J., Booth, A., & Overman, W. H. (2000). Assessing estradiol in biobehavioral studies using saliva and blood spots: Simple radioimmunoassay protocols, reliability, and comparative validity. *Hormones and Behavior*, *38*, 137-147.

Silbereisen, R. K., Petersen, A. C., Albrecht, H. T., & Kracke, B. (1989). Maturational timing and the development of problem behavior: Longitudinal studies in adolescence. *Journal of Early Adolescence*, *9*, 247-268.

Smith, E. A., Udry, J. R., & Morris, N. M. (1985). Pubertal development and friends: A biosocial explanation of adolescent sexual behavior. *Journal of Health & Social Behavior*, *26*, 183-192.

Sobel, M. E. (1982). Asymptotic intervals for indirect effects in structural equation models. In S. Leinhardt (Ed.), *Sociological methodology* (pp. 290-293). Washington, DC: American Sociological Association.

Sudman, S. (2001). Examining substance abuse data collection methodologies. *Journal of Drug Issues*, *31*, 695-716.

Sung, M., Erkanli, A., Angold, A., & Costello, E. J. (2004). Effects of age at first substance use and psychiatric comorbidity on the development of substance use disorders. *Drug and Alcohol Dependence*, *75*, 287-299.

Susman, E. J., Finkelstein, J. W., Chinchilli, V. M., Schwab, J., Liben, L. S., D'Arcangelo, M. R., et al. (1998). The effect of sex hormone replacement therapy on behavioral problems and moods in adolescents with delayed puberty. *Journal of Pediatrics*, *133*, 521-525.

Susman, E. J., Inoff-Germain, G., Nottelmann, E. D., Loriaux, D. L., Cutler, G. B., Jr., & Chrousos, G. P. (1987). Hormones, emotional dispositions, and aggressive attributes in young adolescents. *Child Development*, *58*, 1114-1134.

Tanner, J. M. (1962). *Growth at adolescence*. Oxford, UK: Blackwell.

Udry, J. R. (1988). Biological predispositions and social control in adolescent sexual behavior. *American Sociological Review*, *53*, 709-722.

Udry, J. R. (1990). Biosocial models of adolescent problem behaviors. *Social Biology*, *37*, 1-10.

Udry, J. R. (1991). Predicting alcohol use by adolescent males. *Journal of Biosocial Science*, *23*, 381-386.

Udry, J. R., Billy, J., Morris, N., Groff, T., & Raj, M. (1985). Serum androgenic hormones motivate sexual behavior in adolescent boys. *Fertility and Sterility*, *43*, 90-94.

Worthman, C. M., & Stallings, J. F. (1997). Hormone measures in finger-prick blood spot samples: New field methods for reproductive endocrinology. *American Journal of Physical Anthropology*, *104*, 1-21.

Vangie A. Foshee is an associate professor in the Department of Health Behavior and Health Education from the School of Public Health at the University of North Carolina at Chapel Hill.

Her research focus is on adolescent problem behaviors, particularly adolescent substance use and dating violence, and includes both etiological and evaluation research.

Susan T. Ennett is an associate professor in the Department of Health Behavior and Health Education at the University of North Carolina at Chapel Hill's School of Public Health. Her research focuses on understanding interrelations between peer, family, school, and neighborhood contexts that influence adolescent substance use and other health risk behaviors.

Karl E. Bauman (PhD, sociology) is a professor of health behavior and health education in the School of Public Health at the University of North Carolina at Chapel Hill. He conducts etiological and evaluation research related to adolescent health risk behaviors.

Douglas A. Granger received his doctorate in psychology and social behavior from UC Irvine in 1990 and completed postdoctoral training in endocrinology and immunology at UCLA in 1994. He is a professor in the Departments of Biobehavioral Health and Human Development and Director of the Pennsylvania State University Behavioral Endocrinology Laboratory. The main themes guiding his research concern how developmental processes influence psychobiological events, how psychobiological events influence social developmental processes, and ultimately whether systematic individual differences in the reciprocal influences of these processes affect children's health and social development.

Thad Benefield is a biostatistician at Rho, Inc. He joined Rho after more than 5 years as a statistical analyst at the University of North Carolina at Chapel Hill, where he was the lead statistician for two National Institutes of Health–funded grants. There, he employed a vast array of statistical techniques to investigate dating violence in adolescents and has coauthored numerous publications. He has worked as a biostatistician for projects requiring development of methodology plans and advanced statistical analyses. He is experienced in statistical modeling, generation of statistical reports, and communicating statistical ideas. His proficiencies include mediation/moderation analyses, survival analysis, generalized estimating equations, interaction analysis, and mixed models.

Chirayath Suchindran received his PhD in biostatistics from the University of North Carolina at Chapel Hill in 1972. He is currently a professor of biostatistics and a fellow of the Carolina Population Center at the University of North Carolina at Chapel Hill. He is the director of the statistical demography training program in the Department of Biostatistics. His primary research interest is in the area of statistical demography.

Andrea M. Hussong received her PhD in clinical psychology from Arizona State University in 1996 and is currently an associate professor of psychology at the University of North Carolina at Chapel Hill. Her research interests include developmental pathways to alcohol and drug abuse and the risk and resilience shown by children of alcoholic parents in particular.

Katherine J. Karriker-Jaffe received her doctorate from the Department of Health Behavior and Health Education at the University of North Carolina at Chapel Hill. Her research focuses on understanding the role of neighborhoods, families, and individual factors on the development of youth health risk behaviors. She lives in San Mateo, California.

Robert H. DuRant, PhD, is a professor of pediatrics and professor of social science and health policy at Wake Forest University School of Medicine. His research has primarily focused on the epidemiology and prevention of health risk and problem behaviors among adolescents. His current National Institutes of Health funding includes a randomized group trial of the prevention of high-risk drinking behaviors and their consequences among college students, rapid response interventions on college campuses to address high-risk drinking behaviors, and a randomized control trial of brief interventions among middle school students in rural east Tennessee using tobacco, alcohol, and other drugs.