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Adverse environmental exposures during gestation and childhood: Predictors of adolescent drinking

Marie D. Cornelius[a], **Natacha De Genna**[a], **Lidush Goldschmidt**[b], **Cynthia Larkby**[a], and **Nancy Day**[a]

[a]University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania, USA

[b]University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

Abstract

Adverse conditions, including exposures to drugs and other environmental influences during early development, may affect behaviors later in life. This study examined the role of environmental influences from the gestation and childhood on adolescent drinking behavior. 917 mother/offspring dyads were followed prospectively from pregnancy to a 16-year follow-up assessment. Interim assessments occurred at delivery, 6, 10, and 14 years. Prenatal exposures to alcohol, tobacco, and marijuana were measured during gestation. Data were collected at each phase on childhood environment, including parenting practices, quality of the home environment, maternal depression and hostility, and lifetime exposure to child maltreatment and community violence. Alcohol outcomes were offspring age of drinking initiation and level of drinking at age 16 years. Cox Proportional Hazards ratios were used to model offspring age of drinking initiation. Logistic regression analyses were used to evaluate significant predictors of drinking level. Childhood environment, including less parental strictness, greater exposure to violence and childhood maltreatment, significantly predicted earlier age of alcohol initiation. Level of drinking among the adolescent offspring was significantly predicted by prenatal exposure to alcohol, less parental strictness, and exposures to maltreatment and violence during childhood. Whites and offspring with older mothers were more likely to initiate alcohol use early and drink at higher levels. Early and heavier alcohol use was associated with early exposures to adversity such as prenatal alcohol exposure, and child exposures to maltreatment and violence. These results highlight the importance of environmental adversity and less effective parenting practices on the development of adolescent drinking behavior.

Keywords

adolescence; alcohol use; prenatal exposure; child maltreatment; violence; parenting

CORRESPONDENCE AND REPRINT REQUESTS: Marie D. Cornelius, Ph.D., Program in Epidemiology, Western Psychiatric Institute and Clinic, 3811 O'Hara Street, University of Pittsburgh, Pittsburgh, PA 15213, 412-681-3482; 412-681-1261(fax), mdc1@pitt.edu.

Introduction

Adolescence is a critical developmental period when alcohol use is initiated and patterns of drinking behavior are formed (Masten et al., 2008). Earlier age of alcohol onset and problem use are associated with risky behaviors including other drug use, driving while under the influence, sexual risk taking, and school failure (Zucker et al., 2008). Although many studies have demonstrated that earlier age of alcohol onset is one of the strongest predictors for the development of alcohol-related problems, which early developmental factors that contribute to early drinking remain unclear.

From conception through adolescence, the pace of development is rapid as the human brain undergoes significant growth, and maturation. Adverse conditions, including exposures to drugs and other environmental factors during critical developmental periods, may affect the direction toward more risky behavioral pathways. Conversely, children with protective resources tend to make the most successful transitions to the developmental challenges of adolescence (Masten et al., 2004). It is important to identify targets early in the developmental period and across multiple domains for prevention and intervention efforts. Several studies have identified more proximal risk factors that place children at higher risk for early alcohol use. These environmental, personality, and sociodemographic factors have been studied extensively but usually separately, in cross-sectional analyses, or with study time frames that span a few years. In addition, few of the previous studies had data on early development, and even fewer had data on gestational exposures. Our analysis utilizes prospective data from gestation through 16 years, and considers influences from multiple environmental factors.

Gestational Substance Exposure

During gestation there is robust growth and adaptation to adverse exposures in the central nervous system (CNS). Toxic exposures during gestation may have significant and long-lasting effects on development (Fox & Rutter, 2010; Volkow, 2013). The effects of prenatal alcohol exposure on offspring development have been identified in both the human and animal literature (Jacobson & Jacobson, 2003; Riley, 1990). Reported effects of prenatal alcohol exposure include growth reduction (Cornelius et al., 2002; Day et al., 1994), cognitive deficits (Richardson et al., 1995; Willford et al., 2004; 2006), and increased rates of activity and attention deficits (Leech et al., 1999). Adolescents with prenatal alcohol exposure have higher rates of psychological symptoms and psychiatric diagnoses (Larkby et al., 2006; 2011) and more behavior problems in young adulthood (Day et al., 2013). Two studies reported a significant association between maternal drinking during pregnancy and increased risk of drinking in their adolescent daughters (Griesler & Kandel, 1998; Pfinder et al., 2014). However, in both of these studies, prenatal alcohol exposure was collected retrospectively. Two previous prospective studies examined the potential link between prenatal alcohol exposure and offspring drinking; both found an association (Alati et al., 2008; Baer et al., 2003). However, these studies did not consider other gestational or childhood environmental exposures.

Childhood Environmental Exposures

Adverse environmental factors during childhood including the home environment, child exposure to maternal distress, parenting behavior, maltreatment, and violence are also associated with poorer outcomes. Quality of the home is a well-established predictor of child cognitive and behavioral outcomes (Bradley et all, 2001; Evans, 2004; Wasik et al., 1990). In turn, satisfactory cognitive development is protective against earlier risky behavior, including drinking (Englund & Siebenbruner, 2012; Weiland et al., 2012).

Maternal psychological characteristics and parenting behaviors may be associated with adolescent drinking by way of difficult child temperament and cognitive and emotional delays (Muzik & Borovska, 2010) and depression in adolescence (Pawlby et al., 2009). Maternal depression has also been linked to early and heavy episodic drinking in adolescent offspring (Lamis et al., 2012).

Parental involvement may play an important role in the risk for early and heavier alcohol use in youth. Strict alcohol-specific parenting can be effective in reducing or delaying adolescent alcohol use (Janssen et al., 2014). Although general discipline may not affect alcohol initiation, it may have an impact on reducing levels of drinking in later adolescence (Siobhan et al., 2010). Jackson and colleagues (2014) reported that parents who had a low level of knowledge of their children's daily activities were more likely to have children with more alcohol-related outcomes.

Adverse childhood experiences including emotional, physical and sexual abuse, emotional and physical neglect, have been associated with alcohol initiation in early and mid-adolescence (Dube et al., 2006). Significant associations between childhood maltreatment and alcohol use, including earlier age of alcohol onset, have also been reported (Hamburger et al., 2008), as has problem use among adolescents (Shin et al., 2012; Shin et al., 2013). Exposure to violence is common among U.S. children. In a nationally representative sample, 53% of children experienced assault in the past year (Finkelhor, 2005). Both indirect (heard about or witnessed) and direct (experienced) violence have been linked to alcohol use (Pinchevsky et al., 2013; Schwab-Stone et al., 1995) and problem alcohol use (Jester et al., 2015).

Sociodemographic Characteristics

Several sociodemographic characteristics are also related to alcohol use. In the National Survey on Drug Use and Health (SAMSHA, 2011), White adolescents were more likely to report current alcohol use, binge drinking, and earlier age of alcohol onset. However, socially disadvantaged Black youth are at higher risk for alcohol problems (Mulia et al., 2008), and are more likely to die from alcohol-related causes (Polednak, 2007) than Whites, which may in part be due to different consumption patterns between races. The majority of studies do not find sex differences in age onset of drinking (Donovan, 2004), although peer influence may more strongly affect drinking for girls than boys (Donovan, 2004).

Adolescents with lower socioeconomic status have a higher rate of alcohol use (Hardaway & Cornelius, 2014), and lower maternal education predicts alcohol use disorders in young adults (Hayatbakhsh et al., 2008). However, in a report from the National Longitudinal

Survey of Adolescent Health, higher parental income was associated with higher rates of binge drinking in adolescent offspring among non-Hispanic Whites, but not among non-Whites and Hispanic Whites (Humensky, 2010). Finally, the potential effects of maternal age have not been previously examined as a possible contributing factor for earlier or heavier alcohol use in offspring. Children of young mothers are at greater risk than children of older mothers for cognitive impairment, social and behavioral deficits, child abuse and neglect, and school failure (Cornelius et al., 2006; Furstenberg et al., 1990; Jaffe et al., 2001; Sommer et al., 2000). By adolescence, these offspring are more likely to use substances (Fergusson & Woodward, 1999).

Though multiple studies have examined one or some of these factors, none has considered all of them using prospective data. We hypothesized that early adversity risk factors would predict earlier ages of onset of alcohol use and drinking at heavier levels.

Materials and Methods

Procedures

This report is from the Maternal Health Practices and Child Development Project. Study participants were recruited from the public prenatal clinic at the Magee-Womens Hospital in Pittsburgh Pennsylvania. This is an inner-city health care facility with a catchment area that captures primarily lower socio-economic women. Mothers were interviewed prenatally, and with their offspring at delivery, 6, 10, 14, and 16 years. Standardized protocols were used to assess the psychological, environmental, and alcohol use characteristics of the mothers and their offspring. The data from gestation and the 6-, 10, 14- and 16-year follow-up phases were used for this analysis.

Data were drawn from two cohorts from the Maternal Health Practices and Child Development Project that had comparable measures of maternal and child development, psychological status, and environmental characteristics. The combined cohort had 1176 mother/offspring dyads and was comprised of three studies: two were from studies of prenatal alcohol and marijuana use among adult mothers (Adult Mothers Cohort, n=713), and one was from a study of gestational substance use among teenage mothers (Teen Mothers Cohort, n=413). Adult Cohort mothers were 18–42 years old at recruitment during the mid 1980’s and Teen Cohort mothers were 12–18 years old at recruitment during the early 1990’s. We were able to combine the Adult Mother and the Teen Mother Cohort samples because 1) women were drawn from the same prenatal clinic, 2) we used the same instruments, assessments, and personnel to evaluate all cohorts, and 3) we had the same follow-up time periods. The primary differences between the two cohorts were maternal age and race: The younger cohort had a higher proportion of Black mothers. The Institutional Review Boards of the Magee-Womens Hospital and the University of Pittsburgh approved each of these studies. In addition, Certificates of Confidentiality were obtained from the National Institutes on Health for all phases of the studies.

Participants—The median age of the women in the combined cohorts during their fourth month of pregnancy was 20 years (range: 12–42) and 79% were unmarried. Fifty-eight percent drank alcohol during the first trimester, 50% smoked cigarettes, and 32% used

marijuana. The average daily number of drinks among first trimester drinkers was 0.84 (range: 0.002–19.6). Descriptions of the alcohol and other substance use and the measures of substance use of the mothers have been published (Cornelius et al., 1994; 1995; Day et al., 1989; 1991) in earlier publications.

At birth, the combined sample size was 1176 live singleton infants. By the 16-year follow-up, 103 offspring were lost to follow up, 67 refused participation, 13 children had died, 15 were adopted or in foster care, and 52 had moved out of the area. Nine offspring did not complete the drug and alcohol assessment, which, resulted in a sample of 917, or 78% of the original birth cohort (Adult Mothers n=588; Teen Mothers n=329). At the 16-year phase, 43% of the adolescents were White, 57% were Black, and 49% were female. We compared offspring who were lost to attrition (N=259) compared to those who were retained in the sample (N=917). There were no differences in prenatal alcohol exposure, maternal age, prenatal marijuana, or prenatal tobacco exposure.

Measures

Gestational Exposure—Prenatal alcohol exposure was assessed for each trimester of pregnancy using the usual, maximum, and minimum frequency and quantity of each alcoholic beverage (wine, beer, liquor, and beer and wine coolers), and the average daily number of drinks was calculated from these data. Questions for alcohol were: “During the first trimester, on the days that you drank (specific beverage), how many (specific containers) did you usually drink?” Next, they were asked: “How often did you drink this amount?” Responses included: every day; almost every day; 3–4 times a week; 1–2 times a week, 2–3 times a month; once a month; 1–2 times in 3 months. Questions were repeated for beer, wine liquor, wine coolers and beer coolers. All of the alcohol data, including typical, higher, and lower levels of drinking were combined to create the average daily number of drinks variable. The distribution of average daily number of drinks was positively skewed, so log linear transformations were used to reduce skewness. Marijuana use was also assessed as the quantity and frequency of the usual, maximum, and minimum use, parallel to alcohol. Marijuana, hashish, and sinsemilla use were transformed into average daily joints: a blunt of marijuana was converted to four joints, and a hashish cigarette or bowl was counted as three joints based on the relative amount of delta-9-THC in each (Gold, 1989). Other illicit drug use was rare during pregnancy and at the follow-up phases, and was not considered in our analyses. Cigarette smoking was measured as number of cigarettes/day. Average daily cigarettes, a continuous measure, was used in our analyses. All interviews took place in a private setting by interviewers who were comfortable discussing alcohol and drug use and who were trained to use the instrument reliably. Because substance use declined beyond the first trimester, thus limiting statistical power to detect any effects, we used first trimester exposures in our analyses.

Childhood Environmental Exposures—*Home environment* was measured at 6 years with the Home Observation for Measure of the Environment-Short Form (HOME-SF) (Caldwell & Bradley, 1984) (Teen cohort) and the Home Screening Questionnaire (HSQ) (Frankenburg & Coons, 1986) (Adult cohort). The HSQ correlates well with the HOME (Frankenburg & Coons, 1986). Both instruments measure the quality and quantity of support

available to the child for cognitive, social, and emotional development. The HSQ and HOME-SF scores were transformed to z-scores and combined for the analyses. The age 6 measures of home environment were used for this analysis, because this age precedes the normative age when children begin to drink.

Maternal Depression and *Hostility* were assessed at all study phases. For this study, measures from the 6-year assessment were used. Maternal depressive symptoms were assessed using the CES-D (Radloff, 1977). Maternal hostility during pregnancy was measured using the Spielberger State-Trait Anxiety Inventory (Spielberger et al., 1983). The 6-year post-partum measures of maternal depression and hostility were used in this analysis because this age precedes the normative age when children begin to drink.

Parenting practices were measured by the My Parents instrument (Steinberg, Lamborn, Dornbusch, & Darlin, 1992), an assessment of parenting practices as reported by adolescents. This instrument, completed by the offspring at age 14, has three scales: acceptance/involvement, strictness/supervision, and psychological autonomy granting scale.

*The Childhood Trauma Questionnaire (CTQ) (*Bernstein & Fink, 1998), a well-validated self-report measure that assesses the spectrum of lifetime abuse and neglect experiences covering the past 16 years, measured childhood maltreatment. The data from the 16-year follow-up were examined as a continuous variable to reflect the range of adverse experiences and as an ordinal variable to assess the number of subscales above the recommended cutpoints for moderate/severe maltreatment (cumulative maltreatment exposure).

The Screen for Adolescent Violence Exposure (SAVE; Hastings & Kelley, 1997), a self-report scale, assessed offspring's exposure to violence covering the past 16 years. We adapted the SAVE for our study, changing the Likert-scale ratings into dichotomous (yes/no) responses. For these analyses, violence exposure was defined as the sum of personal victimization incidents such as having been shot or shot at, beaten, and hurt/stabbed by a knife. This measure was assessed at the 16-year follow-up phase.

The *sociodemographic* covariates included race (dichotomous), child sex (dichotomous), maternal education (highest number of years of education), and economic hardship. *Maternal age* at recruitment (4th gestational month) was used as a continuous variable. *Economic hardship* was constructed as a latent variable from three measures: monthly family income, ability to handle bills, and financial strain. Financial strain was constructed from three questions in the maternal interview that inquired how often mothers were short of money at the end of the month, could not buy essential things for their child, and could not do extra things for their child.

The Pubertal Development Scale (PDS) (Petersen et al., 1988) is a self-report measure of pubertal status. The PDS collects data in a non-intrusive manner with good reliability and validity, and reflects the sequence of pubertal development described by Tanner (1962) by including questions about the development of secondary sex characteristics and was collected at the 14-year follow-up.

Drinking Outcome Measures

Adolescent Alcohol Use—Questions were developed by Donovan (1994). Measures included quantity, frequency of beer, liquor, wine, and wine and beer coolers. Offspring drinking was measured at ages 10, 14, and 16 years, and included self-reported age at first full drink. Key outcomes for these analyses included: 1) offspring age at first full drink (earliest age reported at the 10-, 14-, or 16-year assessment was used); and 2) past year frequency and quantity of each beverage as reported at the 16-year follow-up assessment. For the analyses, level of alcohol use was based on average daily drinks and was categorized into 0, < 1 drink/week, and 1+ drinks per week.

Statistical Analysis

The outcome variables included age of onset and alcohol use over the past year. The risk/protective factors were selected from demographic, gestational, and childhood domains. Demographic variables included race, maternal age, child age, economic hardship, and number of people in the household. Gestational variables included prenatal alcohol, marijuana, and tobacco exposures. Variables from childhood included home environment, maternal depression, maternal hostility, parental involvement, parental strictness, exposure to maltreatment, and exposure to violence.

Bivariate relations between risk/protective factors and the three categories of alcohol use were tested using analyses of variance (ANOVA) for the continuous variables and χ^2 for the dichotomized variables. Ordinal polytomous logistic regression was used for the multivariate analysis that included all significant covariates from the bivariate analyses and used forward stepwise regression to avoid multicollinearity. Polytomous logistic regression is an extension of logistic regression suitable for categorized ordinal outcome variables, and the resulting odds ratios indicate the probability of being in a higher category of use. Cox proportional hazards models were applied for the analyses of age of initiation. For subjects that had not initiated alcohol use, age at assessment was used to represent censored time in the survival analysis. The proportionality assumption of the Cox hazards model was tested for prenatal alcohol exposure and each significant covariate (Kalbfleisch & Prentice, 1980). The proportionality test was not rejected, suggesting that the Cox model was appropriate.

Results

Adolescent Drinking

Forty-three percent of the offspring had begun to drink between ages 13 and 16.9 years, 22% had begun to drink before 14 years, and 10% had begun drinking prior to 13 years. Forty-seven percent reported alcohol use over the past year and 20% were drinking at least weekly (Table 1). Thirty-nine percent had ever smoked cigarettes and 51% had used marijuana at the 16-year follow-up phase (Table 1).

Demographic and Environmental relations with Adolescent Drinking

Age of onset of drinking—In the bivariate analyses, White race, less economic hardship, and older maternal age significantly predicted earlier age of alcohol onset (Table 2). Significant gestational and childhood predictors of earlier age of alcohol onset were prenatal

alcohol exposure, prenatal tobacco exposure, less parental strictness and involvement, and greater exposure to childhood maltreatment and violence (Table 2). Pubertal development was marginally related to earlier age of alcohol onset. Offspring sex and maternal psychological status were not related to earlier age of alcohol onset.

A stepwise Cox proportional hazards regression analysis was used to identify the associations between variables that were significant in the bivariate analyses and earlier age of alcohol onset (Table 4). White race, lower parental strictness, exposure to violence and childhood maltreatment, and older maternal age significantly predicted earlier age of alcohol onset (Table 4). After considering these variables, prenatal exposures to alcohol and tobacco and economic hardship were no longer significantly related to age of initiation. A hazard ratio of 2.6 for race indicates that the hazard rate of earlier age of alcohol onset among White adolescents was 2.6 times higher than the hazard rate of early initiation among Black adolescents. For continuous variables such as exposure to violence, for each additional exposure to violence, the hazard rate of initiation of alcohol use was increased by 1.08.

Level of drinking—In the bivariate analyses, White race, less economic hardship, older maternal age and offspring age were associated with a higher level of drinking. Environmental variables from the gestational and childhood periods that significantly predicted a higher level of drinking were more exposure to prenatal alcohol and cigarette smoke, less parental strictness, less parental involvement, greater maternal hostility during childhood, and greater exposure to child maltreatment and violence (Table 3). Variables that were significant at the bivariate level were added to the multivariate analyses.

In the multivariate analyses for the outcome alcohol drinking level, White race, older maternal age, prenatal alcohol exposure, parental strictness, and exposures to maltreatment and violence during childhood remained significantly associated with level of use (Table 5). Prenatal exposure to tobacco, maternal hostility, economic hardship, and parental involvement were not significant after controlling for the other covariates. Each drink per day increase in maternal drinking during pregnancy increased the odds of being in a higher level of drinking group by 1.7 times.

For each unit of difference in parental strictness, the odds of being in a higher level of drinking were decreased by 0.86 times.

Discussion

We examined data from two combined birth cohorts in which mothers' ages spanned 12 to 42 years. Subjects were assessed at comparable time points from the fourth month of gestation to age 16. Measures from these cohorts included early adversity in the gestational period, childhood environment, and socio-demographic factors. We identified factors that significantly predicted earlier age of alcohol onset and level of drinking from both the gestational and childhood periods. Importantly, we also identified those that did not.

Earlier age of onset of alcohol use was predicted by White race, parental strictness (less), mother's age (older), and more lifetime exposure to maltreatment and violence. When other early adversity risk factors (significant at the bivariate level) such as maternal hostility,

parenting involvement and pubertal timing were considered into the multivariate models, they were no longer statistically significant. These findings are in general agreement with other reports that have assessed the predictors of earlier age of alcohol onset.

Similarly, White race, parental strictness (less), maternal age (older), childhood exposures to maltreatment and violence, and prenatal alcohol exposure were significantly related to level of drinking. Prenatal alcohol exposure did not predict age of onset of alcohol use, but it did predict higher levels of drinking. This is consistent with the literature on alcohol use in adolescents with prenatal alcohol exposure (Alati et al., 2008; Baer et al., 1998). Children with prenatal alcohol exposure may not be more susceptible to earlier use, but once they try alcohol, they may be more vulnerable to its effects. Animal studies suggest that in utero ethanol exposure causes embryological changes resulting in oxytocin system changes (McMurray et al., 2008) neurobehavioral deficits (Shea et al., 2012), and basal neural activity (Fabio et al., 2013). These changes may result in increased preferential intake of alcohol among exposed offspring. In addition, our findings could reflect a common familial pattern between mothers who drink during pregnancy and offspring use. Alternatively, prenatal alcohol exposure could have a direct association with adolescent drinking or the effect of prenatal alcohol exposure could be mediated by some of the consequences of prenatal alcohol exposure.

Mother’s age has not been previously evaluated in terms of predicting adolescent drinking. We found that the older mothers had offspring who were more likely to have an earlier age of onset and drink at a higher level. These results may be an artifact of a higher proportion of prenatal drinkers among the mothers in the adult cohort (64%) as compared to the mothers in the teen cohort (48%). It is possible that women who drink during pregnancy are also more likely to be drinkers across their offspring’s lifespan, and this may influence adolescent use due to parental modeling, access to alcohol in the home, and perceived parental acceptability of alcohol use. Our findings parallel the results of recent studies indicating that older mothers are more likely to drink while pregnant (Kitsantas et al., 2014; Meschke et al., 2013). These results are consistent with a study of mothers from Detroit - the effects of prenatal alcohol exposure were more pronounced in the offspring of older mothers (Jacobson et al., 2004).

Children of less strict parents and who provided less supervision were also more likely to drink at an earlier age and to drink at greater levels. This finding is consistent with other reports (Alati et al., 2010; Janssen et al., 2014). Although there is evidence that indicates that overly harsh parenting is linked to greater alcohol use in adolescents (Alati et al., 2014; Brody & Ge, 2001), our measure of parental strictness represented facets of appropriate parental monitoring and setting limits on the adolescent’s behavior. Less parental involvement and acceptance were significantly related to earlier initiation of alcohol and greater drinking level in the bivariate analyses, but were no longer significant when multiple factors were considered together. Thus, parental supervision and strictness play a more important role in adolescent drinking behavior than parental involvement and acceptance.

Finally, childhood exposures to child maltreatment and violence significantly predicted both earlier onset of alcohol use and higher levels of drinking among the offspring. These

findings have been reported previously in both cross-sectional (Hamburger et al., 2008; Shin et al., 2012; 2013; Vermeiren et al., 2003) and prospective (Taylor & Kliewer, 2006) studies. These results have important implications for the risk of alcohol use and abuse among young victims of maltreatment and violence.

This study utilizes two rich datasets with excellent retention rates that include a wide range of maternal ages and extensive information on adversity, maternal and child substance use measured at multiple time points across a 17-year span. One limitation of the study is that the sample represents a low SES group, 43% White and 57% Black. Therefore, these results may not extend to families from middle and higher socio-economic brackets or families from other racial ethnic groups. Another potential limitation is that is possible that some women might misreport their alcohol use when asked during their pregnancy. The prenatal data, however, were collected in the early 1980s and 1990's when alcohol use during pregnancy was not as stigmatized as it is now. We did not use biological measures of alcohol use, as they do not allow an accurate assessment of alcohol use over a longer period, such as a trimester. To increase the accuracy of the data that were reported, we constructed detailed questions, carefully selected interviewers, and extensively trained our staff in interviewing techniques.

Our findings have several public health implications. Early exposures to adversity such as prenatal alcohol exposure, maltreatment and violence, and less parental strictness are markers of risk for adolescents who are vulnerable to earlier and/or heavier alcohol use. These results highlight the importance of reducing alcohol use during pregnancy and reaching young women in the prenatal clinic. Pregnancy is an excellent time to reach vulnerable families, because women are more likely to seek health care at this period in their lives. Our findings also underscore the importance of parenting as an important protective factor for adolescent alcohol initiation and use. Thus, youth with these identified characteristics should be targeted for intervention.

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Table 1

Description of Offspring at the 16-Year Follow-up Assessment

DEMOGRAPHIC	
Average Age (years)	16.8 range 15.9–19.5
Race % Black	60%
Sex % Male	49%
Not in Maternal Custody	17%
Total number of people in household	4.3 (2–11)
Total number of siblings	1.5 (0–7)
SUBSTANCE USE	
Alcohol	
Ever full drink (%)	55.5
Past year full drink (%)	**47%**
1+ drink/week (%)	20.3
Mean age alcohol initiation	14 (7–18)
% Initiation before age 13 years	**10%**
% Initiation before age 14 years	22.2
% Initiation between 13 and 16.9 years	**43%**
Other Substances	
Ever smoke cigarettes (%)	38.7
Ever used marijuana (%)	51.0

Table 2

Bivariate Relations between Demographic/Environmental Factors and Earlier Age of Onset of Drinking.

Variable	Hazard Ratio[a]	P Value
Demographic		
Race (1=White, 0=Black)	2.14	<0.001
Offspring Sex (1=male, 0=female)	0.90	NS
Economic Hardship	0.97	<0.05
Maternal Age (at recruitment)	1.05	<0.001
Gestational and Childhood Factors		
Prenatal Alcohol Exposure[b]	1.32	<0.01
Prenatal Tobacco Exposure[c]	1.02	<0.001
Prenatal Marijuana Exposure[d]	0.99	NS
Home Environment	1.09	NS
Maternal Depression	1.00	NS
Maternal Hostility	0.92	NS
Parental Strictness	0.93	<0.001
Parental Involvement	0.95	<0.001
Pubertal Status[e]	0.91	<0.10
Childhood Maltreatment	1.31	<0.001
Exposure to Violence	1.06	<0.001

[a] Hazard Ratio per unit of measurement

[b] Average daily drink, log transformed to reduce skewness

[c] Average cigarettes/day

[d] Average joints/day

[e] 1=very early relative to peers, 5=much later than peers

Table 3

Bivariate Relations between Demographic/Environmental Factors and Offspring Level of Drinking

Variable	Offspring Drinking Level			P Value
	None N=484	≤1 Drink per week n=247	>1 Drink per week n=186	
Demographic				
Race (% White)	28.1	45.7	61.8	<0.001
Offspring Sex (% Male)	49.8	44.9	52.2	NS
Economic Hardship	12.8	12.3	12.1	<0.01
Maternal Age (at recruitment)	19.9	21.2	21.3	<0.001
Offspring Age	16.7	16.8	17.0	<0.001
Gestational and Childhood Factors				
Prenatal Alcohol Exposure	0.22	0.29	0.36	<0.001
Prenatal Tobacco Exposure	5.2	7.2	9.9	<0.001
Prenatal Marijuana Exposure	0.32	0.29	0.30	NS
Home Environment	−0.05	−0.03	0.09	NS
Maternal Depression	37.5	38.3	38.1	NS
Maternal Hostility	16.2	16.4	17.3	0.030
Parental Strictness	20.1	18.3	17.1	<0.001
Parental Involvement	30.8	29.6	29.0	<0.001
Pubertal Status	2.9	2.9	2.7	<0.10
Childhood Maltreatment Exposure	2.0	2.27	2.4	<0.001
Exposure to Violence	8.0	8.8	9.7	<0.001

Table 4

Demographic/Environmental Predictors of Earlier Age of Onset of Drinking: Multivariate Analyses

Significant Variables	Coefficient	Hazard Ratio*	P Value
Race	0.95	2.59	<0.001
Maternal Age	0.04	1.04	<0.001
Parental Strictness	−0.08	0.92	<0.01
Childhood Maltreatment Exposure	0.12	1.13	<0.05
Exposure to Violence	0.07	1.08	<0.001
Non-Significant Variables			
Economic Hardship	**−0.01**	**0.99**	**NS**
Prenatal Alcohol Exposure	**0.16**	**1.17**	**NS**
Prenatal Tobacco Exposure	**0.008**	**1.01**	**NS**
Parental Involvement	**−0.007**	**0.99**	**NS**

*Hazard ratio is the ratio of the hazard rates corresponding to the conditions described by two levels of an explanatory variable. The instantaneous hazard rate is the limit of the number of adolescents who begin to drink per unit time divided by the number at risk (those who have not yet initiated drinking) as the time interval approaches zero.

Table 5

Demographic/Environmental Predictors of Drinking Level: Multivariate Analyses

Significant Variables	**Coefficient**	**Cumulative OR**	**P Value**
Race	1.21	3.30	<0.001
Maternal Age	0.06	1.10	<0.001
Prenatal Alcohol Exposure	0.52	1.70	0.003
Parental Strictness	−0.15	0.86	<0.001
Childhood Maltreatment Exposure	0.25	1.40	< 0.01
Exposure to Violence	0.09	1.10	<0.001
Non-Significant Variables			
Economic Hardship	**−0.02**	**0.98**	**NS**
Prenatal Tobacco Exposure	**0.008**	**1.0**	**NS**
Maternal Hostility	**−0.001**	**1.0**	**NS**
Parental Involvement	**−0.02**	**0.98**	**NS**