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Author manuscript

*Alcohol Clin Exp Res*. Author manuscript; available in PMC 2015 June 02.

Published in final edited form as:

*Alcohol Clin Exp Res*. 2010 October ; 34(10): 1813–1821. doi:10.1111/j.1530-0277.2010.01269.x.

## The Impact of Maternal Age on the Effects of Prenatal Alcohol Exposure on Attention

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### Abstract

**Background**—Prenatal exposure to alcohol has a variety of morphologic and neurobehavioral consequences, yet more than 10% of women continue to drink during pregnancy, placing their offspring at risk for fetal alcohol spectrum disorders (FASD). Identification of at-risk pregnancies has been difficult, in part, because the presence and severity of FASD are influenced by factors beyond the pattern of alcohol consumption. Establishing maternal characteristics, such as maternal age, that increase the risk of FASD is critical for targeted pregnancy intervention.

**Methods**—We examined the moderating effect of maternal age on measures of attention in 462 children from a longitudinal cohort born to women with known alcohol consumption levels (absolute ounces of alcohol per day at conception) who were recruited during pregnancy. Analyses examined the impact of binge drinking, as average ounces of absolute alcohol per drinking day. Smoking and use of cocaine, marijuana, and opiates were also assessed. At 7 years of age, the children completed the Continuous Performance Test, and their teachers completed the Achenbach Teacher Report Form.

**Results**—After controlling for covariates, stepwise multiple regression analyses revealed a negative relation between levels of prenatal binge drinking and several measures of attention. The interaction between alcohol consumption and maternal age was also significant, indicating that the

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impact of maternal binge drinking during pregnancy on attention was greater among children born to older drinking mothers.

**Conclusion**—These findings are consistent with previous findings that children born to older alcohol-using women have more deleterious effects of prenatal alcohol exposure on other neurobehavioral outcomes.

### Keywords

Attention; Prenatal Alcohol; Prenatal Exposure; Maternal Age

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THE FETAL ALCOHOL spectrum disorders (FASD; Bertrand et al., 2004; Hoyme et al., 2005; Sokol et al., 2003), including fetal alcohol syndrome (FAS), include specific neural and craniofacial malformations, growth restriction, and neurobehavioral and cognitive deficits (Hoyme et al., 2005; Kodituwakku, 2007; Nash et al., 2006; Sokol et al., 2003; Spadoni et al., 2007). The estimated incidence of FAS ranges from 0.3 to 2.0 per 1,000 live births in the general population (CDC, 2002a, 2002b; May and Gossage, 2001), with a higher incidence among certain groups depending upon socio-demographic, behavioral, clinical, and other risk factors (Abel, 1995; CDC, 2002a, 2002b; May et al., 2007, 2008). The combined incidence of all FASD is higher and estimated at about 10 per 1,000 live births (Manning and Hoyme, 2007; O’Leary, 2004; Sampson et al., 1997).

While awareness of the risk of alcohol use during pregnancy has increased substantially since 1989 when warning labels began to appear on alcohol beverage containers, more than half of women of child-bearing age continue to consume alcoholic beverages, and over 11% report continued drinking during pregnancy (CDC, 2009; Stratton et al., 1996). The rates of certain patterns of consumption that put fetuses at greater risk for FASD, especially self-reported binge drinking—defined as 5 or more drinks per occasion (CDC, 2002a, 2002b)—have remained essentially unchanged at around 2% since 1991 (CDC, 2009). However, levels of drinking also vary with maternal age. Older pregnant women (>34 years old) are 37% more likely to report drinking during pregnancy than younger women (CDC, 2009). However, self-reported rates of binge drinking were not significantly different between younger pregnant women (18 to 24 years old), at 2.5%, and older pregnant women (>34 years old), at 1.8% (CDC, 2009).

In this paper, we are examining attention problems because they are among the more frequently reported outcomes associated with FASD (Brown et al., 1991; Carmichael Olson et al., 1998; Coles et al., 1997; Fryer et al., 2007; Kodituwakku, 2007). Deficits have been identified in sustained attention (Coles et al., 2002; Lee et al., 2004), as well as executive function, memory, IQ, fine motor skills, and other neurodevelopmental domains (Coles et al., 1997; Kodituwakku, 2007; Kodituwakku et al., 1995; Mattson et al., 1998). Coles and colleagues (1997) reported other alcohol-related attention deficits in visual / spatial skills, encoding of information, and flexibility in problem solving. Yet there are inconsistencies in the occurrence of attention problems and hyperactivity in prenatal alcohol-exposed children. Nanson and Hiscock (1990) reported increased hyperactivity and slower reaction times, while Roebuck and colleagues (1999) and Coles and colleagues (1997) did not. Carmichael Olson and colleagues (1992) found deficits in focused attention but not as severe as those

found in children diagnosed with attention deficit hyperactivity disorder (ADHD). These variations in reported attention deficits may be related to multiple factors including which prenatal alcohol exposure variable was assessed, differential difficulty of the task assessing attention (Kodituwakku, 2007), sample size, or additional moderating variables, such as maternal age. In addition to maternal age and how risk drinking is assessed, several other factors are influential in the variable expression of attention effects in FASD and ADHD, such as how attention is measured and cohort characteristics (e.g., age, demographics, etc.). These are considered further in the Discussion.

Alcohol-related neurobehavioral deficits are certainly also influenced by differences in vulnerability and individual susceptibility, even at comparable levels and rates of maternal drinking. The variable expression of prenatal alcohol-related effects may be because of differential genetic susceptibility, exposure periods, or drinking patterns (Abel, 1995; American College of Obstetrics & Gynecology, 2006; Chiodo et al., 2009; Ernhart et al., 1987; Henderson et al., 2007; Jacobson and Jacobson, 1994, 1999; Maier and West, 2001; Martínez-Frías et al., 2004; NIAAA, 2005a; Olney, 2004; Sokol et al., 1986; Stratton et al., 1996; West et al., 1994), as well as nutritional or other risk factors. One factor that may influence the impact of prenatal alcohol on neurobehavioral outcome is the pattern of alcohol consumption. Detection of patterns of maternal drinking that place fetuses at risk for FASD is critical to diagnosis, treatment, and prevention but is challenging and often insufficient during pregnancy. We recently showed that outcomes depend on which measure of prenatal alcohol exposure is used and that a metric that accounts for several different measures of risk-level drinking during pregnancy is a better predictor of neurobehavioral effects of prenatal alcohol, including focus and divided attention, than the individual alcohol consumption measures alone (Chiodo et al., 2010).

Older age appears to be another maternal characteristic that is related to differential susceptibility to the effects of prenatal alcohol exposure. Jacobson and colleagues (1998, 2004) and Burden and colleagues (2005) showed greater adverse performance in attention and working memory tasks for infants and children born to older drinking mothers (30 years old) compared to younger mothers (Burden et al., 2005). Prenatal alcohol exposure-related deficits in working memory were greater among children born to women 30 years of age. Despite a small sample, children born to older mothers performed significantly-worse in 4 of 7 measures of attention, memory and general cognitive ability. For example, there were increased errors of omission in a Digit Cancellation task, working memory deficits in the digit span test, and lower scores on the Wechsler Intelligence Scale for Children–Revised (WISC-R) Arithmetic subscale, and others, while only one outcome—executive function measured by the Tower of London task—was significantly impaired for children whose mothers were <30 years of age.

In this study, we examined the impact of maternal age at the time of the first prenatal clinic visit on measures of attention in early school-age children. We hypothesized that the deficits in attention outcomes related to prenatal alcohol exposure would be greater among children born to older mothers, that is, 30 years of age. Understanding the influence of maternal age on the relation between prenatal alcohol and neurobehavioral outcome might assist in the development of focused primary care interventions for older drinking mothers.

## METHOD

### Sample

The sample consisted of children born to prospectively identified inner city African American women participating in a longitudinal pregnancy study which recruited women receiving prenatal care at a university antenatal clinic. Study inclusion criteria were singleton birth between September 1989 and August 1991 and continued residence in the Detroit area. Exclusion criteria included multiple-gestation, children born to women known to be HIV positive, or born with congenital malformations. Offspring from repeat pregnancies to the same participating mother were also excluded. As African American women constituted more than 90% of the antenatal clinic population, participation was limited to this group. After an exhaustive search, the 656 eligible children located in the Detroit area at 7 years of age comprised the potential study sample; 94% of these families agreed to participate and 85% completed laboratory testing ( $N = 556$ ; 49.1% female). At the 7-year follow-up, 6 children were deceased, and 4 children were recognized to have nonalcohol-related congenital malformations and excluded. Within the Detroit area, families were not geographically stable. Most of the families (86.1%) had moved at least once since the child was born, and there was an average of 3.1 home address changes.

The final sample consisted of 462 of the 556 children for whom teacher data were also available. Analyses were performed comparing those 462 children included in these analyses and the 94 who were not included on several demographic and child variables [e.g., maternal IQ, socioeconomic status (SES), child gender and age] as well as prenatal alcohol exposure and attentional outcomes. Among the 15 variables examined, the 2 groups differed on child IQ, total Home Observation for Measurement of the Environment (HOME) score, and child age. Children not included in the analyses had higher IQ scores ( $t = 5.5$ ,  $p < 0.001$ ), higher total HOME scores ( $t = 2.5$ ,  $p = 0.012$ ), and were slightly younger ( $t = 3.4$ ,  $p = 0.001$ ). There were no differences between the groups on either the predictor or outcome variables.

### Prenatal Alcohol Exposure

As detailed in Nordstrom-Bailey and colleagues (2004) and Hannigan and colleagues (2010), pregnant women were queried extensively by trained researchers at each prenatal visit to estimate pattern, quantity and frequency of current and peri-conceptual alcohol consumption using a structured interview developed specifically to assess alcohol use during pregnancy (Sokol et al., 1985). A 2-week recall by beverage type was obtained with questions linked to specific drinking habits and particular times of the day and days of week and included queries about binge drinking. From these data, several alcohol exposure variables were calculated as ounces of absolute alcohol per day (AAD) or per drinking day (AADD) at around conception, at the first antenatal visit, and averaged across pregnancy. The current analyses focused on ounces of absolute alcohol per drinking day across pregnancy (AADDXP). In addition, at the first prenatal visit, the 25-item Michigan Alcoholism Screening Test was administered (MAST; Selzer, 1971). At each visit, the adverse effects of alcohol consumption during pregnancy on the fetus were explained and women advised to stop or at least reduce their alcohol intake.

**Other Prenatal Drug Use**—At each prenatal visit, the use of cocaine, heroin, marijuana, and nonmedical opiates was also ascertained by maternal self-report and women were classified as users or nonusers. Prenatal tobacco exposure was quantified as the typical number of cigarettes the mothers reported they smoked each day.

## Procedures

At 7 years of age, following the date of the child's expected entry into first grade and after informed parental consent, the child and primary caregiver (usually the biologic mother) were tested in our laboratory. Female research assistants, blind to the child's prenatal exposure status, interviewed each child and mother independently. Permission was given to obtain teacher assessments of child behavior. A third research assistant, also unaware of the child's exposure status, collected data from the child's teacher. These procedures and the many measures used in these analyses are detailed in Nordstrom- Bailey and colleagues (2004).

## Child Outcomes

The computer-administered version of the Continuous Performance Test (CPT), developed to evaluate sustained attention (Conners, 1995), was used to generate distinct measures of inattention and impulsivity. In this 15-minute task, a series of letters are presented sequentially to the child who presses a keyboard spacebar for every letter presented with the exception of "X." The child is instructed not to press the spacebar when the letter "X" is presented. Commission errors (pressing the bar when an "X" appeared) were considered a measure of impulsivity, and omission errors (failing to press the bar when a letter other than "X" appeared) were considered a measure of inattention. Table 1 presents the CPT outcomes. In addition, mean hit reaction time to correct responses (processing speed),  $d'$  ("d prime"), and  $\beta$  score were obtained. Slower reaction time with an increased error rate also indicates inattention. The  $d'$  statistic is an assessment of attention measured as how consistently the child is correct in discriminating a "target" from a "nontarget" in this task. D-Prime ( $d'$ )  $T$ -scores at or above 60 indicate poor ability to discriminate and attend to the target stimulus (Conners, 1995). Individuals with high response rates (yielding low  $\beta$  scores;  $T$ -scores  $\leq 40$ ) are more impulsive and greater risk-takers, whereas those with low response rates (high  $\beta$  scores;  $T$ -scores  $\geq 60$ ) are considered more cautious (Conners, 1995). Finally, for each child the number of  $T$ -scores for the various indicators (i.e.,  $T$ -scores for errors of omission, errors of commission,  $d'$ ,  $\beta$  score, hit reaction time, response variability, and standard error block change) that were  $\geq 2$  SDs above the mean was summed to obtain an overall attention score ("ADHD Score"—an approximation of clinical severity). Thus, a child could have a score of 0, indicating none of their scores were indicative of an attention deficit, or a score of 7, suggesting that all attention problem indicators were elevated. Although this is not a standard CPT variable, it is an approximation of an ADHD profile. In general, one or more  $T$ -score  $\geq 60$  is an indication that the child had difficulty on this task and attention problems are probable. Increased numbers of elevated  $T$ -scores are indicative of greater attention deficiency (Conners, 1995).

The Conners version of the CPT was used in this study because it allows speed of presentation variability, which is often lacking in other CPT paradigms (Conners, 1995).

Thus, the Conners CPT is able to assess deficiencies in both the slow and fast presentations of the stimuli, which is very important for assessment of hyperactivity. In addition, the Conners CPT employs a larger number of target responses to avoid a “floor effect” (Conners, 1995).

The widely utilized Teacher Report Form (TRF) was used to assess teacher-reported child behavior problems in the classroom (Achenbach, 1991). In addition to a total behavioral problem score, 8 syndrome scales were examined: aggressive behavior, delinquent behavior, anxious / depressed, withdrawn, somatic complaints, attention problems, social problems, and thought problems. Aggressive and delinquent behavior syndrome scales were summed for a total externalizing behavior score, while anxious / depressed, somatic complaints, and withdrawn comprised the internalizing behavior problem scale. Among the TRF scales, the attention problem syndrome scale was the only outcome of interest in this study.

### Control Variables

At the 7-year follow-up visit, a structured interview with the caregiver assessed postnatal family drug, alcohol, and cigarette use, demographic information, including education, SES (Hollingshead, 1975), quality of the home environment (a laboratory-adapted version of the HOME; Caldwell and Bradley, 1984), as well as self-reported caregiver psychopathology (Derogatis et al., 1973) and an assessment of maternal verbal IQ (Wechsler, 1981). The children’s whole-blood lead levels were also assessed.

### Data Analyses

Checks were performed for missing and out-of-range data, and for deviations from normality. To examine the impact of maternal age on the relations between prenatal alcohol exposure and measures of child attention, a series of multiple regression analyses were performed, controlling for confounding variables listed above. Because a control variable cannot be a confounder unless it is related to both exposure and outcome, association with either exposure or outcome can be used as the criterion for statistical adjustment (Schlesselman, 1982). In this study, control variables were selected for inclusion based on their relation to the outcome, which has the additional advantage of increasing precision by also including covariates unrelated to exposure and maternal age (Kleinbaum et al., 1988). Pearson’s correlation coefficients (“*r*”) were used to examine the relation of each control variable to each outcome. All control variables that were even modestly related to each outcome ( $p < 0.10$ ) were adjusted statistically by regressing the attention outcome and the control variables related to that outcome. All covariates (related to an attention outcome  $p < 0.10$ ) were entered together in the first step along with maternal age and the alcohol exposure measure (AADDXP). The maternal age  $\times$  alcohol interaction term was entered in the second step. In the regression analyses, continuous measures of both levels of alcohol drunk per drinking day (AADDXP) and maternal age variables were used. The relations of alcohol consumption measures, maternal age, and attention were considered significant at  $\alpha < 0.05$ , after controlling for the potential confounders. In the tables presenting the regression analyses, the bivariate correlations are shown as Pearson’s “*r*.” The relation of attention and the interaction term is shown as the “ $\beta$ ” statistic, the standardized regression coefficient (Table 3).

## RESULTS

Data from the TRF were not available for 56 (10.1%) of the 556 children assessed on the CPT in the laboratory at 7 years of age. Data from children with Performance IQs <65 (28 children; an additional 7.6%) were removed from the analyses to minimize attention problems that might be secondary to profound general cognitive delays or dysfunction. The final sample included 462 children. Within this sample, just over half of the caregivers had the equivalent of a high school diploma for both the younger and older women (57.7 and 57.5% high school graduate or General Education Degree (GED) respectively). In addition, 75% of the women were unmarried and the majority of caregivers were the biologic mothers (81.6%). Most families (58%) were in the lowest SES group based on income and employment (Hollingshead, 1975). Mean child whole-blood lead level was 5.0  $\mu\text{g}/\text{dl}$ ; however, 25 children had values >10  $\mu\text{g}/\text{dl}$  and 2 had values >15  $\mu\text{g}/\text{dl}$  (see Chiodo et al., 2007). Table 2 presents these maternal, family and child characteristics for both maternal age groups, <30 years of age vs.  $\geq$  30 years of age at the time of the first prenatal assessment. Older mothers were significantly more likely to use alcohol, cocaine, and nicotine during the prenatal period. There were no differences between older and younger mothers in marijuana usage or in any family or child characteristic. Average child total TRF *T*-scores as reported by the teacher were within the normal range. However, 20.1% of all children had clinical or borderline behavior problem scores. In addition, 11.5% of the sample was identified by the teacher as having clinical levels of attention behavior problems (TRF attention problems *T*-score  $\geq$  70), while an additional 7.1% had borderline levels of attention problems (*T*-scores  $\geq$  67 and  $\leq$  69). There was no significant relation between maternal drinking and the TRF attention problems syndrome scale (Table 3).

Regression analyses controlling for covariates identified a significant maternal age by across-pregnancy alcohol consumption levels per drinking day (AADDXP) interaction on the number of errors of omission (i.e., not responding to a “target”) and the  $\beta$  score (Table 3). In addition, there was a marginally significant effect ( $0.05 < p < 0.10$ ) for  $d'$  and the summary ADHD score, providing further support for the presence of a maternal age  $\times$  prenatal alcohol exposure interaction on attention.

Further depiction of this relation is provided in Fig. 1 which provides a closer examination of each attention measure. For each figure, alcohol consumption level per drinking day (AADDXP) was dichotomized at 5 drinks per occasion. Those who drank <5 drinks per drinking day were coded as a 1, while those who drank  $\geq$  5 drinks per drinking day were coded as a 2 (see Fig. 1). Children born to older mothers (i.e.,  $\geq$  30 years old) who binge drank have the highest  $\beta$  score, suggesting they performed very slowly and cautiously. In addition, these children have the highest numbers of errors of omission. On the other hand, there was no significant interaction between maternal age and AADDXP for errors of commission. Thus, these alcohol-exposed children were not behaving impulsively in this task, pressing the keys cautiously (high  $\beta$  score) but still making more mistakes. In addition, children of older women who binge drank had the lowest  $d'$  scores, indicating they had more difficulty sustaining attention across the task. The presence of an interaction between maternal age and AADDXP on the summary ADHD score suggests that children of older mothers performed more poorly on these attention measures in comparison to children born

to mothers <30 years of age. Children of older women (≥ 30 years old) have poorer attention scores overall than children born to younger mothers when exposed prenatally to higher levels of alcohol.

Although previous examination of the CPT and IQ have not been significant (Hoerig, 1999; Pepin and Loranger, 1996), these 2 domains are significantly related in this sample ( $r$ 's range =  $-0.04$  to  $-0.20$ ). Owing to the significant relation between the CPT and IQ, regression analyses were redone including Wechsler Preschool and Primary Scale of Intelligence (WPPSI) Full IQ, also assessed at the age 7- year assessment, as a covariate. In both analyses, the maternal age  $\times$  alcohol interaction term remained significant, suggesting that these alcohol-related attention deficits are specific to attentional abilities and were unrelated to the impact of prenatal alcohol exposure on general intelligence.

In addition to a significant maternal age  $\times$  alcohol exposure interaction, there was a significant main effect of maternal age for 2 of the attention variables ( $d'$  and  $\beta$ ;  $\beta = 0.13$ ,  $p = 0.05$  and  $\beta = -0.18$ ,  $p = 0.007$ , respectively). These results indicate that although children of older women performed more poorly than children of younger women, regardless of drinking status (the main effect), the children of older drinking women also performed more poorly than children of younger drinking women on both attention measures (the interaction).

## DISCUSSION

Children born to drinking mothers 30 years of age or older but not children born to younger drinking mothers (<30 years old), showed statistically significant deficits in attention measures on the CPT at age 7 years. Regression analyses identified a significant interaction between maternal age and a measure of levels of alcohol consumed per drinking day across pregnancy (AADDXP) on several indicators of attention, but not impulsivity. This occurred over and above significant decreases in CPT performance in children of older women, regardless of drinking during pregnancy.

This finding is consistent with research identifying differential susceptibility to prenatal alcohol exposure on other cognitive outcomes in young children because of older maternal age. Jacobson and colleagues (1998) identified this interaction for behavioral maturation in infants assessed by the Bayley Scales of Development and Elicited Play. In infants born to women drinking alcohol in binge-like patterns of 5 or more drinks per occasion, there was no increased incidence of functionally significant deficits in infants born to younger women (<30 years old). However, infants born to older women (≥ 30 years of age) drinking at these levels were 2- to 5-times more likely to be functionally impaired on the Bayley Scales and Elicited Play (Jacobson et al., 1998). These same investigators later showed that maternal age also moderated the cognitive effect of prenatal alcohol exposure on IQ at 7.5 years of age (Jacobson et al., 2004). Among children born to older mothers (i.e., ≥ 30 years of age), there were statistically significant decreases in Full Scale IQ related to the amount of alcohol consumed per day during pregnancy, as well as a decrease in the Freedom of Distractibility IQ scale (FD). To ensure that this reduction in attention abilities was not actually because of general IQ, Jacobson and colleagues (1994) examined an additional composite Freedom



from Distractibility- Verbal Comprehension score (FD-VC). Analyses revealed a significant alcohol-related effect for children born to older women only, further supporting the idea that specific attention effects related to alcohol exposure are moderated by maternal age. Burden and colleagues (2005) showed that working memory was also differentially susceptible to prenatal alcohol exposure as a function of maternal age. Our current results are consistent with this and demonstrate that children of older women appear to be more vulnerable to the effects of prenatal alcohol on attention as well.

A brief overview of previous studies of attention deficits after prenatal alcohol exposure suggests that several factors besides maternal age may also influence the likelihood of demonstrating prenatal alcohol-related attention problems. These include, for example, diagnosis (esp., ADHD) of comparison groups (e.g., Mick et al., 2002; Nanson and Hiscock, 1990), presence of dysmorphia in the alcohol-exposed children (Coles et al., 1997), prenatal cigarette exposure, parental ADHD, “social adversity” (Mick et al., 2002), the age of the children, and the specific outcome measure (Boyd et al., 1991; Coles et al., 1997, 2002; Mattson et al., 2006; Mick et al., 2002; Nanson and Hiscock, 1990). Others have discussed in more detail how important a careful definition of the components of attention is to understanding the apparently variable responses to prenatal alcohol exposure (e.g., Burden et al., 2005; Coles et al., 2002; Lee et al., 2004). Finally, child IQ may not influence attention deficits in FASD. This seems to be the case as attention problems in children with FAS/FAE and low IQ (i.e., 78) or with ADHD and normal IQ (i.e., 104 to 110) were similar (Nanson and Hiscock, 1990). Also Jacobson and colleagues (2004) demonstrated an independence of attention and IQ effects following prenatal alcohol exposure. On the other hand, specific deficits in cognitive or executive functioning have been related to the patterns of attention problems in children exposed to “heavy” prenatal alcohol (e.g., Vaurio et al., 2008).

The reason for increased susceptibility of children of older mothers is not clear but various reasonable hypotheses have been considered. Older mothers are likely to have been drinking longer than younger mothers and so are more likely to have greater tolerance to alcohol and more alcohol-related health problems, including liver dysfunction, all of which can lead to higher levels of alcohol in their fetuses. Chronic drinking in older mothers is also more likely than for younger mothers to be related to primary and secondary under-nutrition and so may lead to a greater reduction in the availability, absorption and activity of nutrients critical for fetal development (Fisher, 1988; Lieber, 1988). In addition, the ratio of body fat to water increases with advancing maternal age, thereby exposing both mother and fetus to relatively higher peak blood alcohol concentrations per unit of alcohol consumed over a longer period (Vestal et al., 1977). However, the relation between increased maternal body fat and fetal alcohol levels needs further investigation. Finally, others have suggested that parity, a variable that increases significantly in relation to maternal age may also play a role in the incidence of FAS (Abel, 1998; Abel and Hannigan, 1995).

Prenatal alcohol is known to cause a variety of adverse effects on the developing fetus and at this time there is no clearly defined minimum amount of alcohol known to be harmful to embryos or fetuses, nor any defined “safe” level of drinking during pregnancy that may be promulgated as a reasonable public health message (Abate et al., 2008; ACOG, 2006;

Henderson et al., 2007; Roebuck et al., 1999; Sampson et al., 2000; Sokol et al., 2003). What is becoming clearer, however, is that the pattern of alcohol consumption—higher amount, faster rate, and/or greater frequency of drinking—has important and proximate teratogenic consequences (Abel and Hannigan, 1995; Elliott and Bower, 2004; Olney, 2004; West et al., 1994). Jacobson and colleagues (1994, 1998) showed virtually no developmental effects on birth weight, the Bayley Mental and Psychomotor indices, and cognitive processing speed, with drinking <7 drinks per week on average, but substantial deficits were found in children exposed to more than this. Several researchers have reported that binge drinking and chronic alcoholism both are associated with neurobehavioral effects and decreases in IQ in the children born to these mothers (Ikonomidou et al., 2000; Sampson et al., 1989; Streissguth et al., 1989). Yet not all fetuses exhibit effects of prenatal alcohol exposure even at comparable rates of maternal drinking. The cause of these differential susceptibilities has not been elucidated but has been attributed to influences ranging from genetic predisposition, nutritional inadequacy and variation in the vulnerability of different brain regions (Maier and West, 2001).

As previously recommended, women ought to be warned that with increasing maternal age, fetuses may be more severely affected by alcohol exposure, even when the mother's alcohol intake during pregnancy has not increased from previous pregnancy, and even if prior pregnancies and older children may appear to have been unaffected. Health care professionals need to be aware that increased maternal age among their pregnant patients (i.e., >30 years) increases the susceptibility of the fetus to effects of alcohol. In addition, physicians need to be able to appropriately tailor their interventions to patients during standard clinical visits about the relative risks of maternal drinking to fetuses when the mothers are older. Additional studies to evaluate how maternal age moderates prenatal alcohol-induced deficits in attention and other behavior or cognitive outcomes are needed to more fully understand the risks and consequences of maternal alcohol use during pregnancy.

One study limitation needs to be addressed, potentially poor generalizability. The current cohort included only low SES urban African American women and their children. We should also note that although significant, the effect size is small (Cohen's  $F^2 = 0.03$  and  $0.02$  for  $\beta$  and errors of commission respectively). For these attention measures, the alcohol  $\times$  maternal age interaction term accounts for 3% ( $\beta$ ) and 2% of the variance (errors of commission). In addition, the cohort in which the initial maternal age effects were obtained (Jacobson et al., 1994) had similar homogenous demographics. Thus, these findings need to be reassessed in more heterogeneous populations. We also note the lack of significance in teacher-reported attention findings (i.e., the TRF). This lack of concordance is not too surprising as other researchers have found nonsignificant relations between the CPT and the TRF in children with ADHD (DuPaul et al., 1992; Halperin et al., 1991; Lovejoy and Rasmussen, 1990), suggesting that they are in part assessing different constructs.

In conclusion, this study demonstrates poorer attention performance in 7-year-old children exposed to alcohol prenatally born to older but not to younger drinking mothers. These findings may justify targeting older drinking mothers for particular attention in primary care settings because their fetuses are at greater risk than those of younger drinking mothers for alcohol-related deficits in attention.

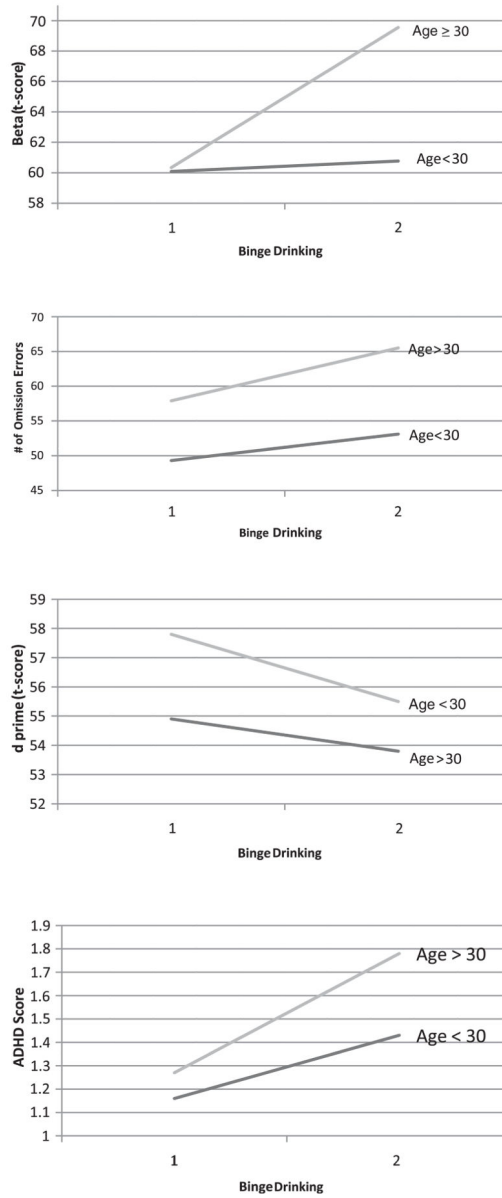
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**Fig. 1.** Attention outcomes across age and binge drinking groups (1 = AADDXP  $< 2.5$ ; 2 = AADDXP  $\geq 2.5$ ). AADDXP, absolute alcohol per drinking day across pregnancy.

**Table 1**

## Conners Continuous Performance Test (CPT) Variables Assessed

Variable	Domain assessed	Measurement
Mean hit reaction time	Processing speed	Time to correct response
Errors of omission	Inattention	Number of missed target responses
Errors of commission	Impulsivity	Number of nontarget responses
d' (d-prime)	Attention	Consistent discrimination of target / nontarget responses
$\beta$ score	Impulsivity	Rate of responding
ADHD score	Overall attention	Number of <i>T</i> -scores <sup>a</sup> 2 SD above the mean

<sup>a</sup>*T*-scores: errors of omission, errors of commission, d',  $\beta$  score, hit reaction time, response variability, and standard error block change.



Table 2

## Sample Characteristics by Maternal Age Status

	Maternal age <30	Maternal age 30	<i>t</i> or $\chi^2$
Maternal characteristics	<i>N</i> = 334	<i>N</i> = 128	
Maternal age at the first prenatal visit (years)	22.4 ± 4.2	33.7 ± 3.0	-27.7***
Alcohol during pregnancy (oz AADXP)	0.11 ± 0.3	0.2 ± 0.6	-2.9**
Alcohol per drinking occasion during pregnancy (oz AADDXP)	1.1 ± 1.4	1.4 ± 1.2	-1.9 <sup>†</sup>
Cigarettes during pregnancy (# / day)	6.5 ± 8.3	12.8 ± 11.0	-6.6***
Cocaine during pregnancy (% used)	33%	52%	16.0***
Marijuana during pregnancy (% used)	35.6	35.9	0.0
Caregiver characteristics (7-year assessment)			
Primary caregiver (% biological mother)	82.9%	85.2%	0.3
Marital status (% married)	26.5%	26.6%	0.0
SES <sup>a</sup>	29.7 ± 10.0	28.7 ± 10.0	1.0
Home observation for measurement of the environment	31.8 ± 5.9	32.8 ± 5.9	-1.6
Education <sup>b</sup>	4.6 ± 1.4	4.7 ± 1.4	-0.5
Current caregiver alcohol use (oz AAD)	0.30 ± 0.6	0.31 ± 0.6	0.0
Current caregiver cocaine use (yes / no)	0.3%	4.0%	9.9*
Current caregiver marijuana use (yes / no)	14.8%	12.7%	3.8
Child characteristics (7-year assessment)			
Age (years)	6.9 ± 0.2	6.9 ± 0.3	1.5
Gender (% male)	49.4%	51.6%	0.1
TRF total behavior problems <i>T</i> -score	55.8 ± 11.4	57.0 ± 12.1	1.1
TRF attention problems syndrome <i>T</i> -score	57.9 ± 9.5	59.6 ± 9.6	3.5 <sup>†</sup>
CPT			
Total hits	273.7 ± 35.9	269.2 ± 33.6	1.3
Mean hit reaction time	555.1 ± 100.8	579.0 ± 103.6	4.6*
Errors of commission	21.3 ± 3.4	20.4 ± 6.2	1.4
Errors of omission	50.3 ± 35.9	54.8 ± 33.6	1.3
ADHD score (# <i>t</i> 's 2SD+)	1.2 ± 1.5	1.5 ± 1.6	3.2 <sup>†</sup>
Mean blood lead (Pb) levels	4.8 ± 2.8	5.3 ± 3.5	2.2

AAD, absolute alcohol per day; AADXP, absolute alcohol per day across pregnancy; AADDXP, absolute alcohol per drinking day across pregnancy; SES, socioeconomic status; TRF, Teacher Report Form; CPT, continuous performance test.

<sup>†</sup>  $p < 0.10$ .

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

\*\*\*  $p < 0.001$ .

<sup>a</sup>Based on Hollingshead, 1975.

<sup>b</sup> 1 = <7th grade; 2 = junior high; 3 = some high school; 4 = GED; 5 = high school grad; 6 = some college; 7 = college grad.

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**Table 3**

Relation Between Prenatal Alcohol Exposure and Measures of Attention

Attention measures	Pregnancy drinking per drinking day			Across pregnancy average		
	Maternal age		oz Alc / drinking day	Interaction term		oz Alc / day
	r	$\beta$	r	$\beta$	r	$\beta$
CPT						
Errors of omission <sup>a</sup>	0.03	-0.06	0.13**	-0.37	0.15***	0.52*
Errors of commission <sup>b</sup>	-0.06	-0.12	0.03	-0.03	0.03	0.08
ADHD T-score (# >2 SD's above mean) <sup>c</sup>	0.10 <sup>†</sup>	0.02	0.02	-0.36 <sup>†</sup>	0.06	0.38 <sup>†</sup>
d' (d prime) <sup>d</sup>	0.04	0.13*	-0.12**	0.27	-0.13**	-0.42 <sup>†</sup>
$\beta$ score <sup>e</sup>	-0.01	-0.18**	0.10 <sup>†</sup>	-0.63**	0.12	0.78***
Mean hit reaction time <sup>f</sup>	0.16***	0.15*	0.01	-0.13	0.03	0.10
TRF						
Attention problems <sup>g</sup>	0.09	0.05	0.03	-0.20	0.06	0.21

CPT, Continuous Performance Test; HOME, Home Observation for Measurement of the Environment; SES, socioeconomic status; TRF,

Teacher Report Form.

<sup>†</sup>  $p < 0.10$ .

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

\*\*\*  $p < 0.001$ .

Covariates assessed by outcome:

<sup>a</sup> Prenatal cocaine, 7-year blood lead levels, caregiver education, HOME score, Peabody Picture Vocabulary Test (PPVT).

<sup>b</sup> Prenatal cocaine, 7-year blood lead levels, child gender, caregiver marital status, prenatal marijuana, 7-year caregiver marijuana.

<sup>c</sup> Prenatal cocaine, 7-year blood lead levels, caregiver education, prenatal nicotine, age of child.

<sup>d</sup> Prenatal cocaine, 7-year blood lead levels, HOME score, child gender, PPVT.

<sup>e</sup> Prenatal cocaine, 7-year blood lead levels, caregiver education, SES, PPVT.

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$f_{7\text{-year}}$  blood lead levels, prenatal nicotine, child gender.

<sup>8</sup>Prenatal cocaine, 7-year blood lead levels, caregiver education, prenatal nicotine, HOME score, SES, age of child.