

A 14-year retrospective maternal report of alcohol consumption in pregnancy predicts pregnancy and teen outcomes

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Abstract

Detecting patterns of maternal drinking that place fetuses at risk for fetal alcohol spectrum disorders (FASDs) is critical to diagnosis, treatment, and prevention but is challenging because information on antenatal drinking collected during pregnancy is often insufficient or lacking. Although retrospective assessments have been considered less favored by many researchers due to presumed poor reliability, this perception may be inaccurate because of reduced maternal denial and/or distortion. The present study hypothesized that fetal alcohol exposure, as assessed retrospectively during child adolescence, would be related significantly to prior measures of maternal drinking and would predict alcohol-related behavioral problems in teens better than antenatal measures of maternal alcohol consumption. Drinking was assessed during pregnancy, and retrospectively about the same pregnancy, at a 14-year follow-up in 288 African-American women using well-validated semistructured interviews. Regression analysis examined the predictive validity of both drinking assessments on pregnancy outcomes and on teacher-reported teen behavior outcomes. Retrospective maternal self-reported drinking assessed 14 years postpartum was significantly higher than antenatal reports of consumption. Retrospective report identified 10.8 times more women as risk drinkers (\geq one drink per day) than the antenatal report. Antenatal and retrospective reports were moderately correlated and both were correlated with the Michigan Alcoholism Screening Test. Self-reported alcohol consumption during pregnancy based on retrospective report identified significantly more teens exposed prenatally to at-risk alcohol levels than antenatal, in-pregnancy reports. Retrospective report predicted more teen behavior problems (e.g., attention problems and externalizing behaviors) than the antenatal report. Antenatal report predicted younger gestational age at birth and retrospective report predicted smaller birth size; neither predicted teen IQ. These results suggest that if only antenatal, in-pregnancy maternal report is used, then a substantial proportion of children exposed prenatally to risk levels of alcohol might be misclassified. The validity of retrospective assessment of prior drinking during pregnancy as a more effective indicator of prenatal exposure was established by predicting more behavioral problems in teens than antenatal report. Retrospective report can provide valid information about drinking during a prior pregnancy and may facilitate diagnosis and subsequent interventions by educators, social service personnel, and health-care providers, thereby reducing the life-long impact of FASDs. © 2010 Elsevier Inc. All rights reserved.

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Introduction

Fetal alcohol spectrum disorders (FASDs) involve wide-ranging deficits in growth, anatomy, behavior, and cognition (Kodituwakku, 2007; Nash et al., 2006; Sokol et al., 2003; Spadoni et al., 2007). Fetal alcohol syndrome (FAS), the most severe of the FASDs, includes prenatal and/or postnatal growth retardation, central nervous system dysfunction with or without obvious brain malformation—including various

learning disabilities, hyperactivity, mental retardation, and behavioral problems—and a defining pattern of craniofacial malformations (Bertrand et al., 2005; Hoyme et al., 2005; Sokol et al., 2003). The estimated incidence of FAS ranges from 0.3 to 2.0 per 1,000 live births in the general population (CDC, 2002; May and Gossage, 2001), with a higher incidence among certain groups depending upon sociodemographic, behavioral, clinical, and other risk factors (Abel, 1995; CDC, 2002; May et al., 2007, 2008). The combined incidence of all the FASDs (FAS, alcohol-related neurodevelopmental disorders [ARNDs], etc.) ranges up to 10 per 1,000 live births (Manning and Hoyme, 2007; O'Leary, 2004; Sampson et al., 1997) yet identification and diagnosis of children, adolescents, and adults with non-FAS FASDs is challenging (Hoyme et al., 2005) in part because information on maternal risk drinking during pregnancy may be insufficient (Astley, 2006; Ernhart et al., 1989; Stratton et al., 1996).

When compared with antenatal report—sometimes called “prospective” or “concurrent” report—retrospective report of maternal alcohol consumption about a prior pregnancy has been considered less precise or less accurate or less valid by some investigators (e.g., Jacobson et al., 1991, 2002; Little, 1976; Little et al., 1977). It has been argued that altered motivation after birth leads to greater accuracy or validity of retrospective report (Rosett and Weiner, 1984). Differences between one antenatal report and another collected anywhere from 1 week to 4 months later within the same pregnancy (test–retest design) have been interpreted to mean retrospective report is either less accurate due to forgetting or altered motive to deny use (e.g., Robles and Day, 1990; Streissguth, 1976) or “more accurate” (e.g., Alvik et al., 2006a) although there is no independent standard with which to judge accuracy. Differences in the ability of various reports about drinking during pregnancy to predict child outcomes, however, can indicate the relative validity of each report.

In a study focusing on under-reporting, Ernhart et al. (1988) tested the predictive validity of maternal self-report for child outcomes. Both antenatal report and retrospective report almost 5 years later predicted number of craniofacial anomalies, whereas only the retrospective report significantly predicted other anomalies (Ernhart et al., 1998). In a later study, retrospective report at 13 months postpartum predicted significantly delayed psychomotor development assessed with the Bayley Scales of Infant Development at 13 months of age, and slower cognitive processing speed on a cross-modal transfer task on the Fagan Tests of Infant Intelligence averaged across tests at 6.5 and 12 months of age (Jacobson et al., 2002). Yet the authors conclude that retrospective report at 13 months postpartum was less accurate and less valid in predicting cognitive outcome than antenatal report (Jacobson et al., 1991, 2002). Curiously, mothers' retrospective report provided during a pregnancy about periconceptional drinking, that is, before a woman knows she is pregnant, is often considered more reliable

and to have greater predictive power for outcomes than in-pregnancy, antenatal report alone (e.g., Jacobson et al., 1991, 2002; Sokol et al., 1985). The perceived greater reliability of this particular retrospective report is possibly due to reduced maternal denial regarding her drinking “out of pregnancy” (cf, Ernhart et al., 1988; Morrow-Tlucak et al., 1989).

Clinicians diagnosing FAS and other FASDs who lack antenatal report of in-pregnancy drinking because, for example, of incomplete medical records, must often assess gestational exposure years after delivery. The accuracy and validity of retrospective reports of maternal alcohol consumption in current practice are critically important to the diagnosis of FASDs (Hoyme et al., 2005), and to making services available for affected children. In the absence of the facial features that distinguish FAS from other FASDs, valid information on maternal drinking is necessary to correctly diagnose FASDs (Astley, 2006; Bertrand et al., 2005; Hoyme et al., 2005; Stratton et al., 1996). In practice, this information about drinking during pregnancy is more likely to be from retrospective report than periconceptional or in-pregnancy antenatal report. Similarly, epidemiological studies of the population incidence of FASDs typically rely on retrospective report from school-based and active case ascertainment studies (e.g., Aragon et al., 2008a; Kodituwakku et al., 2006; May et al., 2000, 2005, 2006, 2007). Finally, because neurobehavioral outcomes are also necessary to diagnose FASDs, information on how retrospective reports of maternal drinking during pregnancy relate to child behavioral outcomes is also critical to validating those retrospective reports.

For all these reasons, therefore, it is worth examining how maternal postnatal retrospective self-report, even years after delivery, compares to antenatal report in predicting prenatal alcohol-related outcomes. We hypothesized that retrospective self-report of maternal alcohol consumption during a prior pregnancy may be at least as, if not a more valid indicator of the fetal “at-risk” drinking that produces alcohol-related effects on pregnancy outcomes, including the child's neurobehavioral and cognitive sequelae, than antenatal report during that same pregnancy.

Methods

All the procedures had prior approval of the Wayne State University Institutional Review Board and all the participants gave appropriate informed consent.

Sample

Adolescent participants and their mothers ($N = 288$) were identified originally through a larger prospective pregnancy study that recruited women receiving prenatal care at our University maternity hospital. Mothers were screened extensively at each prenatal visit for use of tobacco, alcohol, and

illicit drugs using a structured interview. Inclusion criteria for the longitudinal child study were singleton birth between September 1989 and August 1991 and continued residence within the Detroit area for assessments at 7 and 14 years of age. Exclusion criteria for the longitudinal child study included multiple gestation (e.g., twins or triplet), children born to women known to be HIV positive, and those with multiple congenital malformations. Offspring from repeat pregnancies to the same participating mother were excluded. Because African-American women constituted more than 90% of our prenatal clinic population, participation was limited to this group. At the initial follow-up visit at age 7 years, six children were deceased and four others were recognized to have congenital malformations, and were thus excluded. Families were geographically stable in that they remained in the area but moved frequently within Detroit. The average number of home address changes was three. Of 656 eligible children at 7 years of age, 94% agreed to participate, and 85% completed lab testing ($N = 556$; 49.1% female). Families and children participating at age 7 years were contacted again at age 14 years for an additional follow-up study. The cohort size at age 14 years was reduced to 530 by out-of-state moves (15 families), closed adoptions (2), child/teen deaths (3), and teen incarcerations (6). Twenty-six families (4.7% of the age 7 years cohort) could not be located and 39 (7.0%) refused to participate. Completed teen assessments ($N = 432$; 50.5% female) represent 81.5% of the available age 7 years cohort. Of these, retrospective reports about prior drinking in pregnancy were available for 288 mothers, 266 (92.4%) from the biological mothers themselves, and an additional 22 retrospective assessments from kin collaborator reports. The final cohort of 288 represent 43.9% of the original recruitment cohort of 656, 51.8% of the 556 subjects completing initial testing at age 7 years, and 66.7% of the 432 subjects completing follow-up testing at age 14 years. In addition to the previously noted child deaths, 18 mothers were deceased by the age 14 years exam. Maternal and kin reports were included in the analyses. There were five biological mothers who, although not the current primary caregiver, provided 14-year retrospective data.

Prenatal drug and alcohol exposure

As detailed in Nordstrom-Bailey et al. (2004), mothers were screened extensively at each prenatal visit to estimate pattern, quantity and frequency of current and periconceptional alcohol consumption using a semistructured interview developed specifically to assess alcohol use during pregnancy (Sokol et al., 1985) and conducted by trained researchers. At each visit to our prenatal clinic (mean visits = 5.9; SD = 3.2; range = 1–14), a previous 2-week recall by beverage type was obtained; questions linked to specific drinking habits, alcohol use, defined as the number of standard drinks, at particular times of the day and days of week, and binge drinking. From these data, alcohol exposure variables were calculated as *average ounces of*

absolute alcohol per day (AAD) and *average ounces of absolute alcohol per drinking day* (AADD). Because a standard drink contains approximately one half ounce (or ~15 mL) of absolute alcohol, an AAD of one is equivalent to two drinks. Both average AAD and AADD were calculated without including an estimate of periconceptional drinking. In addition, at the first prenatal visit, the 25-item Michigan Alcoholism Screening Test (MAST) was administered (Selzer, 1971). At each visit, the adverse effects of alcohol consumption during pregnancy on the fetus were explained and women were advised to stop or at least reduce their alcohol intake.

Risk drinking in pregnancy was defined for this study as an average of one standard North American drink (i.e., 12 oz beer; 4–5 oz wine, or 1.5 oz spirits) per day or an AAD ≥ 0.5 or more than five drinks per drinking day (AADD > 2.5) based on Jacobson et al. (1993) (see also ACOG, 2006; Jacobson and Jacobson, 1994). At each prenatal visit, the use of cocaine, heroin, marijuana, and nonmedical opiates were 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.

Retrospective alcohol report

At the 14-year follow-up visit, following their report of current alcohol use, mothers were asked if they drank alcohol during pregnancy. If a mother reported prenatal alcohol use she was asked to think back to a typical week during pregnancy and describe what she drank at each day during the week. Mothers were also asked if there were periods of time they drank more or less. Different drinking patterns were averaged across pregnancy. For example, a woman may have drunk heavily during the first 12 weeks, drank lightly or moderately for the next 18 weeks, and then abstained from alcohol for the last 10 weeks. An AAD measure was constructed for each time period and then prorated for pattern duration and averaged. In certain instances ($N = 22$), the child's current caregiver was with the biologic mother frequently during pregnancy and reported being able to describe the biological mother's alcohol drinking patterns during pregnancy.

Outcomes

Pregnancy outcomes included measures of growth (i.e., infant birth weight, length, and head circumference) and gestational age at birth. When the children were 14 years old, the Teacher Report Form (Achenbach and Rescorla, 2001) was used to assess teacher-reported child behavior problems. In addition to total scores, problem behaviors are described along several syndrome scales (Achenbach and Rescorla, 2001): Aggressive and delinquent behavior (externalizing); anxious/depressed, somatic complaints, and withdrawn scales (internalizing); attention, social, and

thought problems; and Diagnostic and Statistical Manual-IV (DSM-IV)-based scales: conduct disorder (CD), oppositional defiant disorder (ODD), affective, anxiety, somatic, and attention deficit hyperactivity disorder (ADHD) (including inattention and hyperactivity/impulsivity). The DSM-IV oriented scales consist of items that clinicians have judged consistent with DSM-IV diagnostic categories (Achenbach and Rescorla, 2001). In addition to behavior problems, IQ was assessed via the Wechsler Intelligence Scale for Children—3rd Edition (WISC-III). Both Verbal IQ and Performance IQ have good concurrent and construct validity with other IQ assessments (Wechsler, 1991). Maternal reports of teen behavior were collected but not included in this analysis because maternal perceptions of teen behavior problems might have influenced maternal self-report of prior drinking, thereby exaggerating relations between drinking and outcomes.

Additional variables

Additional pregnancy, child, and caregiver data were also collected to control for potential confounding variables in analyses. Information about the use of alcohol, cigarettes, and other drugs during pregnancy was collected antenatally by maternal interview at each prenatal visit. Self-report of alcohol use during the 2 weeks preceding the pregnancy visit was converted to AAD and averaged across pregnancy. The number of cigarettes smoked per day during each 2-week period was also recorded and averaged across pregnancy. Data for other drug use (including marijuana and opiates) was also collected antenatally. Maternal drug use during pregnancy was also assessed retrospectively at the 7-year follow-up. Neonatal and maternal information was obtained from medical records at birth. At the 7-year follow-up, caregivers were queried about drug, alcohol and cigarette use in the home, completed demographic forms, and a quality of home environment assessment (based on the HOME; Caldwell and Bradley, 1984) was performed in the laboratory. Whole blood lead levels were assayed by the institutional laboratory and recorded in $\mu\text{g/dL}$.

Analyses comparing those who have already participated in the teen follow-up and those not tested identified no group differences in maternal age at the time of conception, caregiver education, IQ, or socioeconomic status (SES), child gender, or prenatal drug or alcohol exposures. The only difference was found in marital status: caregivers who were married at the age 7-year assessment were less likely to participate at the age 14-year follow-up than caregivers who were not married at the age 7-year assessment ($F = 5.5$, $df = 1$, $P = .019$).

Data analyses

Prior to analyses, data were checked for missing and out-of-range values and for deviations from normality. To

examine change in alcohol report group status across time, chi-square analyses examined group membership in the following categories for AAD: abstainers/light drinkers <0.50 ; moderate drinkers $= 0.50\text{--}0.99$; heavy drinkers $= 1.00\text{--}1.99$; very heavy drinkers ≥ 2.00 ; and for AADD: abstainers/light drinkers <1.00 ; moderate drinkers $= 1.00\text{--}2.49$; heavy drinkers $= 2.50\text{--}4.99$; very heavy drinkers ≥ 5.00 . Paired t -tests and Wilcoxon signed-rank tests next examined differences in the antenatal and retrospective AAD group alcohol distributions, as well as differences in maternal and kin reports. Finally, linear regression analyses examined the ability of both the antenatal and retrospective assessments of prenatal alcohol exposure to predict negative teen behavior. (For clarity, we note that both antenatal and retrospective reports of mothers' drinking during pregnancy properly "predict" later outcomes of that pregnancy, such as birth weight or child behavior, etc. In contrast, the mothers' current drinking, that is at the 14-year assessment, does not properly "predict" outcomes, but is tested for significant "relations" to outcome.)

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 a criterion for statistical adjustment (Schlesselman, 1982). In this study, control variables were selected for inclusion in the regression analyses based on their relations to outcome measures (Kleinbaum et al., 1988). All the control variables that were even modestly related to each outcome ($P < .10$) were adjusted statistically by regressing the outcome on prenatal alcohol exposure level and the control variables related to that outcome. In the results of the regression analyses (Table 6), relations to the endpoints after adjustment for confounders are the standardized regression coefficient (" β "). Note that the predictors in the regression equations were log transformed due to skewed distributions.

Results

Sample characteristics

Mean gestational age at the mothers' initial antenatal clinic visit was 22.7 weeks ($SD = 7.6$); only 11.5% of the mothers obtained prenatal care during their first trimester. An additional 58.2% of mothers obtained care in the second trimester, whereas 30.3% did not receive prenatal care until the third trimester. The average maternal age at the first prenatal visit was 26.0 years ($SD = 6.6$); 8.3% were <18 years of age at the time of delivery. Demographic data from the age 14-year visit revealed that mothers were poorly educated (see Table 1): $>32\%$ had not graduated from high school, 21% attended some college, and 5% had a college degree. Similar to age 7-year assessment, most mothers were low SES (56%) with an average total household annual income $<\$20,000$. Fewer than 25% of the caregivers were married.

Table 1
Sample characteristics

	N	Mean or %	SD	Range
Maternal/pregnancy data				
Age at first prenatal visit	288	26.0	6.6	12–45
GA at first prenatal visit	287	22.7	7.6	6–38
Maternal education (years)	288	11.5	1.6	7–17
Drug use (antenatal report)				
Cigarettes per day	288	8.3	10.1	0–40
Marijuana use (%)	288	35.1	—	—
Heroin use (%)	288	5.2	—	—
Cocaine use (%)	288	31.9	—	—
14-year follow-up visit data				
Mother's education (years)	288	12.1	1.8	6–19
Marital status (% married)	288	22.6	—	—
SES (mother) ^a	288	27.9	11.1	8–61
IQ (WAIS—performance)	286	83.4	9.9	53–112
Caregiver's age at teen visit ^b	287	42.4	8.0	28.4–79.6
Teen's age	288	14.7	0.9	13.3–17.8
Teen IQ (full)	282	77.4	13.8	40–143
Teen gender (% male)	288	50.7	—	—

GA = gestational age.

^aBased on Hollingshead, (1975), Four Factor Index of Social Status.

^bFive biological mothers provided 14-year retrospective data but were not the primary caregiver.

The mean teen age at testing was 14.7 years (SD = 0.9); 50.7% were male. At testing, 90.6% of teens were in the custody of their biological mother. The teens in this study performed on average > 1 SDs lower than national norms on the IQ assessment (mean full-scale IQ = 77.4; SD = 13.8), consistent with results from other studies evaluating similar urban cohorts of African-American children (e.g., Howell et al., 2006; Jacobson et al., 2004; Nordstrom-Bailey et al., 2004).

Among the 288 retrospective assessments, 22 were non-maternal kin report, including 17 reports from very close kin who were now the teen's primary caregiver (six were spouses of the biologic mother, three siblings, and eight mothers). The remaining five reports were from extended family or nonfamily members. There were no differences found between maternal and kin report in any demographic data with the obvious exception that a kin and not the mother was the primary caregiver. Unless specified otherwise, analyses of retrospective reports described below include both maternal and kin reports.

Comparisons between antenatal and retrospective reports of prenatal alcohol exposure

During pregnancy, mothers reported drinking an average of 0.03 (SD = 0.01) ounces of absolute AAD, well under the average of one standard drink per day (AAD = 0.5) that defines risk drinking (ACOG, 2006; Table 2). In contrast, 14 years later, mean retrospectively reported drinking was an average AAD of 0.47 (SD = 1.3), significantly more than the antenatal report ($t = 5.68$, $df = 287$, $P < .001$).

Table 2
Antenatal, retrospective, and current alcohol consumption

	Maternal report (N = 266)		"Kin" report ^{a,b} (N = 22)	
	Mean	SD	Mean	SD
Antenatal report ^c				
AAD	0.03	0.12	0.06	0.16
AADD	0.12	0.27	0.19	0.41
Retrospective report				
AAD	0.39	1.18	1.48	2.31
AADD	0.89	2.26	1.59	2.28
Current 14-year report				
AAD	0.64	1.65	0.28	0.57
AADD	1.68	2.81	1.66	3.05

AAD = Average across-pregnancy absolute ounces of alcohol drank; AADD = Average across-pregnancy absolute ounces of alcohol drank per drinking day.

^a"Kin" report was given by the caregivers of these children, that is the custodial grandmothers or aunts, etc., of the teens.

^bMaternal retrospective AAD report was significantly lower than retrospective kin AAD reports ($t = -3.81$, $df = 286$, $P < .001$).

^cAntenatal report significantly lower than retrospective report (AAD: $t = 5.68$, $df = 287$, $P < .001$; AADD: $t = 6.25$, $df = 287$, $P < .001$).

Kin retrospective report of in-pregnancy alcohol consumption by the mothers was significantly higher than both maternal antenatal self-report ($t = 2.91$, $df = 21$, $P = .008$) and maternal retrospective self-report ($t = 3.81$, $df = 286$, $P < .001$). Similarly, maternal retrospective report of how much the women drank per drinking day (AADD) was significantly higher than the initial antenatal assessment ($t = 6.25$, $df = 287$, $P < .001$). Retrospective kin report of AADD was higher than antenatal AADD ($t = 2.95$, $df = 21$, $P = .008$) but not significantly different from maternal retrospective AADD report ($P = .16$).

Additional analyses of the magnitude of the relation between antenatal and retrospective reports indicated moderate correlations (AAD: $r = 0.29$; AADD: $r = 0.37$; P s < .001; see Table 3a). Reports of AAD and AADD from both antenatal and retrospective assessments were significantly related to antenatal and retrospective scores on the MAST (see Table 3b).

The women were categorized based on the levels of self-reported alcohol consumption and changes in category membership between the antenatal and retrospective categories were also assessed. Comparison of categorical antenatal ("prospective") report of maternal alcohol consumption, for both AAD and AADD, to categorical retrospective report found that antenatal report was significantly lower than retrospective report (AAD: $\chi^2 = 39.95$, $df = 6$, $P < .001$; see Table 4). Among the 288 women, 43 (14.9%) reported more average drinking retrospectively (AAD) than they had previously reported antenatally. Also, 7.0% of women who reported only light or no drinking antenatally reported "very heavy drinking" retrospectively. Only four women antenatally reported more than light drinking, and three of the four retrospectively reported even more drinking

Table 3a
Correlations among antenatal and retrospective reports

	Antenatal AADD	Retrospective AAD	Retrospective AADD
(N = 288)			
Antenatal AAD	0.79***	0.29***	0.34***
Antenatal AADD	—	0.31***	0.37***
Retrospective AAD	—	—	0.87***

AAD = Average across-pregnancy absolute ounces of alcohol drank;
AADD = Average across-pregnancy absolute ounces of alcohol drank
per drinking day.

than in their antenatal report. Retrospective report of AAD identified 10.8 times more women as risk drinkers (operationalized as \geq one drink per day [$AAD \geq 0.5$]) than the antenatal report (Table 4).

Categorical analyses of AADD revealed a very similar pattern: Antenatal report was significantly lower than retrospective report (AADD: $\chi^2 = 23.16$, $df = 3$, $P < .001$; see Table 5). Only three women reported fewer drinks per occasion retrospectively than antenatally. In addition, no women antenatally reported risk levels of daily drinking ($AADD \geq 2.5$), whereas 21 women retrospectively reported risk levels of alcohol consumption per drinking day (AADD). With an additional 14 women who reported $AADD \geq 2$, a total of 35 women (12%) retrospectively reported drinking significant amounts of alcohol on days when they did drink during pregnancy. In contrast, no woman reported this level of alcohol consumption when asked during pregnancy.

Reported alcohol consumption and pregnancy outcomes

Earlier gestational age at birth but not birth weight or birth length was significantly predicted by antenatal report of AAD and AADD (Table 6). Retrospective report of maternal drinking during pregnancy, on the other hand, predicted birth weight (AADD) and birth length (AAD and AADD) but not gestational age. After controlling for gestational age and other potential confounders, head circumference was not significantly related to either antenatal or retrospective report of drinking in pregnancy (Table 6).

Reported alcohol consumption and teen outcomes

After controlling for potential confounders, regression analyses identified significant problem behaviors by teacher ratings, with about 26% of teens in the borderline or clinical range. Only somatic problems (somatic disorder and somatic complaints) were predicted significantly by the maternal antenatal AAD or AADD report (Table 6). In contrast, several indicators of teen problem behavior were related significantly to retrospectively reported maternal alcohol consumption (Table 6). As for antenatal report, retrospective AAD and AADD were also related significantly to somatic disorder and somatic complaints. Unlike antenatal report, retrospective AAD and AADD both also

Table 3b
Correlations of antenatal and retrospective reports with MAST scores

	MAST scores	
	Antenatal	14 years
(N = 288)		
Antenatal AAD	0.55***	0.35***
Antenatal AADD	0.40***	0.37***
Retrospective AAD	0.29***	0.35***
Retrospective AADD	0.40***	0.44***

AAD = Average across-pregnancy absolute ounces of alcohol drank;
AADD = Average across-pregnancy absolute ounces of alcohol drank
per drinking day; MAST = Michigan Alcohol Screening
Test. *** $P < .001$.

predicted ADHD (including hyperactivity/impulsivity), ODD, CD, anxious/depressed, rule breaking, aggressive problems, and total externalizing behavior. Retrospective AADD, but not AAD, was also related significantly to attention problems (Table 6). Finally, there were no significant relations between teen IQ scores and any report of maternal drinking during pregnancy—antenatal or retrospective, AAD or AADD.

We also examined relations between current caregiver alcohol use (i.e., when the children were 14 years old) and all outcomes (Table 6). Current caregiver drinking was unrelated to any birth measure or to teen IQ. In contrast, after controlling for potential confounders, current caregiver AAD and AADD were related to some behavior problems that were predicted by retrospective AAD and AADD, specifically increased teen CD, anxiety/depression, and rule breaking. Current caregiver AAD was also related significantly to teen total externalizing behavior. However, current caregiver AAD and/or AADD were also related to several outcomes *not* predicted by either retrospective or antenatal report, including affective disorder; anxiety disorder; inattention; and social, thought, and other problems (Table 6).

Based on the results with current caregiver drinking, additional regression analyses were performed for outcomes relative to retrospective AAD and AADD controlling, respectively, for current caregiver AAD or AADD (Table 6). When controlling for current caregiver alcohol consumption, retrospective AAD and AADD remained significantly related to most outcomes, including birth length, somatic disorders, ADHD, hyperactivity/impulsivity, ODD, and somatic complaints. Additionally, retrospective AADD remained significantly related to anxious/depressed, rule breaking, aggressive problems, and total externalizing behaviors after controlling for significant current caregiver drinking. However, CD and rule breaking were no longer significantly related to retrospective AAD, whereas the relations of anxious/depressed, aggressive problems, and total externalizing behaviors to retrospective AAD became marginal ($P < .10$). CD was no longer predicted by retrospective AADD, whereas the relation of attention problems to retrospective AADD became marginal ($P < .10$) after controlling for current drinking.

Table 4

Comparison of antenatal and retrospective across pregnancy drinking^a

Antenatal (“Prospective”) report (AAD)	N	14-year retrospective report (absolute alcohol per day [AAD])			
		<0.50	0.5–0.99	1.0–1.99	≥2.0 Very heavy drinkers
<0.50	284	244 (85.9%)	9 (3.2%)	11 (3.9%)	20 (7.0%)
Abstainers-Light Drinkers					
0.5–0.99	3	1 (33.3%)	0	2 (66.6%)	0
Moderate drinkers					
1.0–1.99	1	0	0	0	1 (100%)
Heavy drinkers					
Totals	288	245 (85.1%)	9 (3.1%)	13 (4.5%)	21 (7.3%)

^aNumber of women reporting various levels of antenatal and retrospective consumption antenatally and retrospectively. Percents are relative to antenatal report (or total).

Discussion

Retrospective report was related to significantly more birth and teen outcomes than antenatal report. The present study compared maternal antenatal self-reports of alcohol consumption obtained during pregnancy with retrospective self-reports obtained 14 years after the same pregnancy to evaluate how well each report predicted birth and behavior outcomes in a sample of 14-year-old inner-city African-American adolescents. In addition, postpartum measures of current maternal drinking were also correlated to important outcomes associated with the FASDs. Confirming our a priori hypotheses, retrospective maternal report of gestational alcohol consumption was higher, and relative to the antenatal report, was a more sensitive measure.

Accurate identification of teens with “at-risk” prenatal alcohol exposure is vital for diagnosis and treatment of teens with behavioral problems, and for determining the true level of risk for FASDs in a population. The present results help fill gaps in the evidence that may lead to a preference for using “prospective” (antenatal or concurrent) reports in diagnosis, and in studies assessing relations of maternal drinking to child neurobehavioral outcomes. Present and prior evidence demonstrates that retrospective report is at least as valid as antenatal report. Based on this greater predictive validity and utility, it may be inferred that retrospective report may be a more accurate measure of in-pregnancy drinking than antenatal report. Antenatal reports have been viewed as being more susceptible to denial and/or distortion motivated by guilt and fear of discovery (cf.,

ACOG, 2006; Ernhart et al., 1988; Morrow-Tlucak et al., 1989), although the opposite interpretation has also been made (Rosett and Weiner, 1984).

Maternal retrospective self-report was related significantly to more negative pregnancy outcomes (i.e., low birth weight and length) and to more teacher-reported behavior problems than the maternal antenatal report given 14 years earlier. The significant behavioral outcomes related to the retrospective report—attention problems, hyperactivity/impulsivity, ODD, rule breaking, and aggressive behavior problems—are well documented problems associated with FASDs (e.g., Burden et al., 2005; Jacobson, 1998; Kodituwakku, 2007; Nash et al., 2006; Riley et al., 2003; Spadoni et al., 2007; Streissguth et al., 1998; Vaurio et al., 2008). Consistent with previous studies, 13.2% of women reported more drinking retrospectively than antenatally; 7.2% of the women who while pregnant had reported only light drinking or less, retrospectively reported “very heavy drinking” during the index pregnancy. Although antenatal and retrospective reports were moderately correlated with each other, retrospective report was a more sensitive measure of risk drinking. Antenatal report identified only 1.1% of the women as “risk drinkers” (i.e., ≥one drink/day during pregnancy, or ≥0.5 AAD), and the 14-year retrospective report identified 10.8 times more “risk drinkers.” Only four women antenatally reported risk drinking, and all but one of them retrospectively reported even more drinking. In addition, no women antenatally reported risk levels of daily drinking (AADD ≥ 2.5), or even

Table 5

Comparison of antenatal and retrospective drinks per drinking day across pregnancy^a

Antenatal (“Prospective”) Report (AADD)	N	14-year retrospective report (absolute alcohol per drinking day [AADD])			
		0.0 Abstainers	0.1–0.99	1.0–2.49	≥2.5
0.0 Abstainers	278	222 (98.7%)	25 (89.3%)	14 (100.0%)	17 (81.0%)
0.1–0.99	10	3 (1.3%)	3 (10.7%)	0 (0.0%)	4 (19.0%)
Totals	288	225 (85.1%)	28 (3.1%)	14 (4.5%)	21 (7.3%)

^aNumber of women reporting various levels of antenatal and retrospective consumption antenatally and retrospectively. Percents are relative to antenatal report (or total). No women reported antenatal AADD > 1.0.

Table 6

Regression analyses (β) examining relations between alcohol reports and teen outcome^a

	Antenatal report		Retrospective report		14-year current drinking		Retrospective corrected for current drinking	
	AAD	AADD	AAD	AADD	AAD	AADD	AAD	AADD
Pregnancy outcomes								
Birth weight ^{1–5}	–0.07	–0.07	–0.07	–0.10*	–0.02	0.02	–0.07	–0.11*
Birth length ^{1–5}	–0.08	–0.09 [†]	–0.10*	–0.11*	–0.03	0.02	–0.10*	–0.13**
Head circumference ^{1–5}	–0.03	–0.02	–0.04	–0.05	–0.08 [†]	0.00	–0.04	–0.06
Gestational age ^{2–5}	–0.15**	–0.22***	–0.08	–0.09	0.02	0.01	–0.08	–0.09
WISC-III								
Full IQ ^{3,6–9,11,12,14,15}	–0.06	–0.10 [†]	–0.01	0.01	0.10 [†]	0.05	–0.01	0.02
Performance IQ ^{6,8,9,11–13,15}	–0.04	–.07	0.05	0.05	0.08	0.05	0.03	0.06
Verbal IQ ^{3,6–12}	–0.06	–0.10 [†]	–0.05	–0.03	0.11*	0.05	–0.05	–0.03
TRF^b								
DSM-IV oriented								
Affective disorder ^{6,8,9,11,14}	–0.01	–0.01	–0.06	0.00	0.07	0.16*	0.07	0.04
Anxiety disorder ^{3,6,8,9,12}	–0.01	–0.03	0.11	0.11 [†]	0.15*	0.10	0.07	0.10
Somatic disorder ^{6,8}	0.34***	0.23***	0.25***	0.27***	0.03	0.10	0.25***	0.27***
ADHD ^{13,14}	0.09	0.09	0.15*	0.15*	0.07	0.10	0.15*	0.13*
Inattention ^{6,14}	0.00	–0.01	0.02	0.01	0.05	0.17**	0.02	0.00
Hyperactivity/Impulsivity ^{3,13,14}	0.10	0.10	0.19**	0.19**	0.06	0.05	0.19**	0.17**
Oppositional defiant disorder ^{3,6,12}	0.10	0.11	0.15*	0.18**	0.10	0.05	0.15*	0.16**
Conduct disorder ^{6,9,13,14}	0.05	0.10	0.13*	0.17**	0.21**	0.19**	0.08	0.12 [†]
Syndrome scales								
Anxious/depressed ^{6,8}	0.02	0.05	0.14*	0.18**	0.15*	0.14*	0.11 [†]	0.16**
Withdrawn ^{6,8,10}	–0.06	–0.09	–0.11 [†]	–0.09	–0.01	0.09	–0.11 [†]	–0.09
Somatic complaints ^{6,8}	0.34***	0.24***	0.20**	0.22***	0.01	0.12 [†]	0.20**	0.22***
Social problems ^{6,8,14,15}	0.01	0.00	0.02	0.07	0.19**	0.14*	–0.03	0.03
Thought problems ^{6,8,12,14}	0.02	–0.05	0.06	0.09	0.21**	0.17*	0.02	0.07
Attention problems ^{6,13,14}	0.09	0.09	0.12 [†]	0.13*	0.08	0.13 [†]	0.12 [†]	0.09 [†]
Rule breaking behavior ^{3,6,8,9,11,14}	0.04	0.05	0.13*	0.16**	0.17**	0.15*	0.09	0.13*
Aggressive behavior ^{3,6,12,13}	0.12 [†]	0.10	0.15*	0.18**	0.12 [†]	0.09	0.12 [†]	0.17*
Other behavior problems	0.08	0.02	0.07	0.10	0.09	0.18**	0.05	0.08
Total internalizing ^{6,8}	0.04	0.02	0.04	0.06	0.06	0.13 [†]	0.04	0.05
Total externalizing ^{3,6,8,13,14}	0.10	0.10	0.16**	0.19**	0.14*	0.11	0.13 [†]	0.16*

[†] $P < .10$; * $P < .05$; ** $P < .01$; *** $P < .001$. Significant β 's are in bold font. Covariates: ¹Gestational age; ²Pregnancy heroin use; ³Pregnancy marijuana use; ⁴Pregnancy cocaine use; ⁵Pregnancy cigarette use; ⁶Mother/caregiver education; ⁷Mother/caregiver marital status; ⁸SES; ⁹HOME Total Score; ¹⁰Number of children in the home; ¹¹Maternal IQ; ¹²7-year blood lead levels; ¹³Maternal age at conception; ¹⁴Teen gender; ¹⁵Teen age at testing.

^aValues are β 's after controlling for covariates related $P < .10$.

^bTRF = Teacher Report Form (Achenbach and Rescorla, 2001).

AADD ≥ 2 , and all of the per-drinking-day risk drinking (AADD) was detected solely with retrospective report. Both reports were also moderately correlated with scores on the MAST, an instrument-assessing problem drinking, suggesting that both reports are related to the negative consequences of alcohol consumption.

As far as we are aware, Ernhart et al. (1988) were the first to demonstrate better predictive validity of retrospective than antenatal self-report of maternal alcohol consumption during a prior pregnancy for child outcomes (i.e., morphological anomalies). The only prior study to compare relations of antenatal and postnatal retrospective reports of alcohol intake to child neurobehavioral outcome was by Jacobson et al. (1991, 2002). In that research, 13-month retrospective report was higher than antenatal reports of alcohol use collected during pregnancy. Both antenatal and retrospective reports were significantly and negatively

related to infant birth weight and birth length (Jacobson et al., 1991, 2002), which in the present study were significant only for retrospective report. That prior study is similar to the present results in that antenatal but not retrospective maternal report predicted earlier gestational age at birth. However, those findings differ from the present results in that their retrospective report predicted smaller head circumference (Jacobson et al., 1991, 2002) and the current data, after controlling for potential confounders, do not. Also in contrast to the current behavioral results at 14 years, relations of antenatal report with infant behavioral and cognitive outcomes at 13 months (e.g., Bayley scales and slower processing speed and reaction time) were as strong or stronger than for the retrospective report (Jacobson et al., 2002). The authors concluded that “the antenatal data are more accurate” (p. 822) for these measures in infants and with this interval between the maternal self-reports. Similar to our

findings, antenatal report identified 10.6% of women as “at-risk” drinkers, whereas the 13-month retrospective report identified 32.3% as “risk drinkers,” an increase of more than 300% (Jacobson et al., 1991). Although concluding that retrospective report may be “a better indication” of maternal drinking, consistent with Ernhart et al. (1988), the authors also concluded that retrospective report may be “less precise” in predicting outcomes. Nevertheless, as detailed above, that retrospective report was related significantly to delayed psychomotor development and slower cognitive processing speed in infants (Jacobson et al., 2002). The present results cannot address accuracy per se, but do suggest that retrospective report 14 years later may be more sensitive and more valid than antenatal report in predicting teen behavioral outcomes. Ernhart et al. (1988) also concluded that, compared with antenatal report, retrospective report ~5 years later was as valid, or more valid, when predicting craniofacial and other anomalies associated with prenatal alcohol exposure.

The greater levels of alcohol consumption in the retrospective maternal self-report in the present study are consistent with prior research in pregnant women with inter-report intervals ranging from 1 to 20 weeks within the same pregnancy (e.g., Alvik et al., 2006a, 2006b; Little, 1976; Little et al., 1977; Robles and Day, 1990; Streissguth et al., 1976), or from 3 months to almost 5 years postpartum (Alvik, 2006a, 2006b; Ernhart et al., 1988; Jacobson et al., 1991, 2002). Another study assessing 5-year recall in a sample of nonpregnant gynecology outpatients also found that retrospective report of alcohol consumption was higher than the current report (Czarnecki et al., 1990). The work by Jacobson et al. (1991, 2002) in a cohort very similar to ours found retrospectively reported drinking was 4.5 times higher than their average antenatal report, which included periconceptional (i.e., pre-pregnancy) drinking, and identified > 3 times more women as “at-risk” drinkers than the antenatal report. Although the categories of drinking levels varied between that study and the present analyses, the pattern and extent of differences between antenatal and retrospective report were quite similar. As with the present study, MAST scores at each report were similar, and related to both reports (Jacobson et al., 2002; see also Ernhart et al., 1988). Similar results were reported by Alvik et al. (2006a, 2006b) in a Norwegian cohort where postnatal retrospective maternal reports of drinking levels collected 6 months postpartum were substantially higher than antenatal reports at either 17 or 30 weeks of gestation. The fact that retrospectively reported intake was greater even during the pregnancy is important because the presumed social pressures to under-report would still be operative.

One possible explanation for greater retrospectively reported levels of drinking is that current drinking may cue recall, although retrospective report was also greater after very brief intervals (e.g., Streissguth et al., 1976), within the same pregnancy (e.g., Alvik et al., 2006a, 2006b; Robles and Day, 1990; Streissguth et al., 1976), and in nonpregnant women (Czarnecki et al., 1990). The present results show that

caregiver drinking assessed at the 14-year assessment predicted some of the same outcomes as retrospective report, and that controlling for current caregiver drinking eliminated some, but not all, significant relations between retrospective AAD and/or AADD and outcomes. Although these analyses suggest that the retrospective report may have been “cued” by the current report collected at the same time (cf., Alvik et al., 2006a, 2006b), it is at least as likely that measures of current drinking and retrospective report are closely related because they reflect consistent levels of drinking across time. In addition, at least six outcomes are predicted by current drinking but not by either antenatal or retrospective report, suggesting that current drinking is also assessing something quite separate from effects of prenatal alcohol exposure. Taken together, these results indicate that statistically controlling for current drinking would overcorrect for its potential influence on retrospective report and that retrospective report is a valid measure of prenatal maternal alcohol consumption.

Another possible explanation for greater retrospective than antenatal report is that mothers may be trying to explain current cognitive or behavioral problems in the child and so unintentionally exaggerate prior use. However, Alvik et al. (2006b) reported that the mothers’ levels of anxiety concerning their perceptions about “abnormality” in their children at 6 months of age did not influence the differences in retrospective versus concurrent antenatal report, and greater retrospective report was also found in gynecological patients with no children being involved (Czarnecki et al., 1990). Czarnecki et al. (1990) is relevant because with no children to potentially bias report, a mother’s knowledge of her child’s status alone may not account for greater retrospective report, although of course, such a bias is still possible. Further, in the present study and in Ernhart et al. (1988), the retrospective maternal self-report of drinking was significantly related to pregnancy or later child outcomes that would not be directly influenced by maternal perceptions or expectations (i.e., birth measures, facial anomalies, and teacher reports) so these outcomes minimize or avoid the impact of this potential maternal response bias. It does not seem, therefore, that differences in maternal motivation either to deny or exaggerate levels of in-pregnancy drinking after delivery explain higher retrospective report.

One limitation of this study is potentially poor generalizability. The current findings were from a cohort that included only low SES urban African-American women and their children. Reliability of assessments of maternal alcohol use or abuse may differ across populations (Bradley et al., 1998; Chan et al., 1993; Ewing, 1984; Nardini et al., 2006; Sokol et al., 1989). The effectiveness of antenatal versus retrospective report should be replicated on a larger sample and in other populations. It is valuable to note, however, that epidemiological and clinical studies in other populations including Native American, South African, and Italian children have also found significant relations between retrospective report and child outcome (e.g.,

Aragon et al., 2008a, 2008b; Kodituwakku et al., 2006; May et al., 2006, 2007, 2008). Another possible limitation is the use of kin report substituting for maternal retrospective report for 22 of the cases (7.6%). Reports by witnesses can be qualitatively different from self-report and might be susceptible to increased error (e.g., exaggeration or fabrication) or underestimation (e.g., compare results in Chang et al., 1999). On the other hand, adding these 22 subjects to the analyses may have increased predictive power by retaining some of the more affected children. These children were in kin care for a variety of reasons, including maternal drug and alcohol abuse. It was not possible to assess relations with outcomes in 22 cases alone for lack of power. If collaborative kin retrospective reports did underestimate mothers' use, as results from Chang et al. (1999) might suggest, this would have made it more difficult to identify significant relations between prenatal alcohol exposure and outcomes. However, the fact that the predictive validity for several outcomes increased when the 22 cases were added, compared with the analyses of biological mothers alone, suggested that kin report is valid and did not introduce significant error.

Identifying the level of maternal alcohol consumption that places a fetus at risk for FASDs remains both an important clinical and public health issue and a difficult research question (Abel, 2006; Sokol et al., 2003). Although many factors can increase the risk for FASDs (Abel and Hannigan, 1995; Coles et al., 2000; Elliott and Bower, 2004; May et al., 2008), the pattern of alcohol consumption during pregnancy—higher amounts, faster rates, and/or greater frequencies of drinking—is the most direct and influential factor. The present results are consistent with the hypothesis that a retrospective assessment of that pattern of drinking may be at least as effective as an antenatal report, and perhaps more so, in identifying risk drinking related to child outcomes, and may also avoid some factors that contribute to denial and maternal under-reporting during pregnancy (Ernhart et al., 1987, 1988; Jacobson et al., 2002; Morrow-Tlucak et al., 1989). In the absence of reliable biomarkers of long-term drinking (Cook, 2003; Kulaga et al., 2006; Ostrea et al., 2006), retrospective report may also be the only alternative available when seeking to identify fetal risk drinking when making diagnoses later in life. The present results suggest that retrospective report may be a viable option given the likelihood of maternal under-reporting of alcohol consumption during pregnancy, or when faced with alcohol drinking assessments missing from normal prenatal care or incomplete medical records, despite the possibility of a response bias. Further, retrospective report may be and in practice usually is the only information available on prenatal alcohol exposure. The present results validate the current diagnostic practices by showing that retrospective report is effective in predicting outcomes associated with FASDs. Because initiating interventions for exposed and affected children is usually predicated upon a diagnosis that, in the absence

of the defining facial characteristics of FAS, requires identifying risk levels of prenatal alcohol exposure, there is a compelling need to determine the effectiveness of retrospective assessment of at-risk drinking in a prior pregnancy. The present results support the hypothesis that retrospective reports, even 14 years after the pregnancy, may be valid indicators of fetal risk drinking and valid predictors of pregnancy and teen outcome. Antenatal report remains important, however, because the prenatal clinical contact where antenatal drinking information is solicited is a critical time for intervention and prevention of risk drinking in pregnancy (cf, ACOG, 2006).

Uncertainties in assessing prenatal alcohol exposure via maternal self-report present difficulties in diagnosis of FASDs and may explain some of the diverse findings in the literature examining the impact of prenatal alcohol exposure on the neurobehavioral outcomes in infants, children, and teens (Chiodo et al., 2009; Kodituwakku, 2007). Decreased cognitive ability and increased behavior problems in childhood have been associated with varying levels of prenatal alcohol exposure in many (Coles et al., 2002; Kable and Coles, 2004; Mattson and Riley, 1999; Nordstrom-Bailey et al., 2004; Russell et al., 1991; Uecker and Nadel, 1996), but not all studies or outcomes (Brown et al., 1991; Fried et al., 1992; Greene et al., 1990; Richardson et al., 1995; Russell et al., 1991), when assessed by antenatal report.

Improving the detection of fetal risk maternal alcohol consumption levels with improved and expanded retrospective methods could aid in the proper diagnosis of—and subsequent intervention with—affected children. All current diagnostic systems require knowledge of “significant” or “heavy” or “substantial” maternal alcohol consumption during pregnancy for a diagnosis of ARNDs, or alcohol-related birth defects (Astley, 2006; Bertrand et al., 2005; Hoyme et al., 2005; Manning and Hoyme, 2007; Stratton et al., 1996). The current results support the hypothesis that retrospective report can be at least as effective as antenatal estimates in defining maternal risk drinking. It remains possible that retrospective report after many years could be compromised by poor or altered recall, influenced by current or intervening maternal drinking, or biased by maternal perceptions of child outcomes. Nonetheless, using valid retrospective report may also reduce the likelihood of denying or distorting alcohol use during pregnancy, can substantiate antenatal assessments, and effectively aid in later diagnoses. In conclusion, valid retrospective assessment of prior in-pregnancy drinking can be an effective indicator of prenatal exposure, predict common prenatal alcohol-related behavioral problems, and may facilitate diagnosis and subsequent intervention by educators, social service personnel, and health-care providers, thereby reducing the life-long impact of FASD.

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References

- Abel, E. L. (1995). An update on incidence of FAS: FAS is not an equal opportunity birth defect. *Neurotoxicol. Teratol.* 17, 437–443.
- Abel, E. L. (2006). Fetal alcohol syndrome: a cautionary note. *Curr. Pharm. Des.* 12, 1521–1529.
- Abel, E. L., and Hannigan, J. H. (1995). Maternal risk factors in fetal alcohol syndrome: provocative and permissive influences. *Neurotoxicol. Teratol.* 17, 445–462.
- Achenbach, T. M., and Rescorla, L. A. (2001). *Manual for the ASEBA School-Age Forms and Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth & Families.
- Alvik, A., Haldorsen, T., Groholt, B., and Lindemann, R. (2006a). Alcohol consumption before and during pregnancy comparing concurrent and retrospective reports. *Alcohol. Clin. Exp. Res.* 30, 510–515.
- Alvik, A., Heyerdahl, S., Haldorsen, T., and Lindemann, R. (2006b). Alcohol use before and during pregnancy: a population-based study. *Acta Obstet. Gynecol.* 85, 1292–1298.
- ACOG—American College of Obstetrics & Gynecology (2006). Drinking and reproductive health: a fetal alcohol spectrum disorders prevention tool kit. [online]. Available at: www.acog.org/departments/healthIssues/FASDOrderForm.pdf. Accessed October 12, 2009.
- Aragon, A., Coriale, G., Fiorentino, D., Kalberg, W. O., Buckley, D., Gossage, J. P., et al. (2008a). Neuropsychological characteristics of Italian children with fetal alcohol spectrum disorders. *Alcohol. Clin. Exp. Res.* 32, 1909–1919.
- Aragon, A., Kalberg, W. O., Buckley, D., Barela-Scott, L. M., Tabachnick, B. G., and May, P. A. (2008b). Neuropsychological study of FASD in a sample of American Indian children: processing simple versus complex information. *Alcohol. Clin. Exp. Res.* 32, 2136–2148.
- Astley, S. J. (2006). Comparison of the 4-digit diagnostic code and the Hoyme diagnostic guidelines for fetal alcohol spectrum disorders. *Pediatrics* 118, 1532–1545.
- Bertrand, J., Floyd, R. L., and Weber, M. L. (2005). Guidelines for identifying and referring persons with fetal alcohol syndrome. *MMWR Morb. Mortal. Wkly. Rep.* 54, 1–14.
- Bradley, K. A., Boyd-Wickizer, J., Powell, S. H., and Burman, M. L. (1998). Alcohol screening questionnaires in women: a critical review. *JAMA* 280, 166–171.
- Brown, R. T., Coles, C. D., Smith, I. E., Platzman, K. A., Silverstein, J., Erickson, S., et al. (1991). Effects of prenatal alcohol exposure at school age: II. Attention and behavior. *Neurotoxicol. Teratol.* 13, 369–376.
- Burden, M. J., Jacobson, S. W., Sokol, R. J., and Jacobson, J. L. (2005). Effects of prenatal alcohol exposure on attention and working memory at 7.5 years of age. *Alcohol. Clin. Exp. Res.* 29, 443–452.
- Caldwell, B. M., and Bradley, R. H. (1984). *Administration Manual, Revised Edition: Home Observation for Measurement of the Environment*. Little Rock, AK: University of Arkansas at Little Rock.
- CDC (2002). Fetal alcohol syndrome—Alaska, Arizona, Colorado and New York, 1995–1997. *MMWR Morb. Mortal. Wkly. Rep.* 51, 433–435.
- Chan, A. W., Pristach, E. A., Welte, J. W., and Russell, M. (1993). Use of the TWEAK test in screening for alcoholism/heavy drinking in three populations. *Alcohol. Clin. Exp. Res.* 17, 1188–1192.
- Chang, G., Goetz, M. A., Wilkins-Haug, L., and Berman, S. (1999). Prenatal alcohol consumption: self versus collateral report. *J. Subst. Abuse Treat.* 17, 85–89.
- Chiodo, L. M., Janisse, J., Delaney-Black, V., Sokol, R. J., & Hannigan, J. H. (2009). A metric of maternal prenatal risk drinking predicts neurobehavioral outcomes in preschool children. *Alcohol. Clin. Exp. Res.* 33, 634–644.
- Coles, C. D., Kable, J. A., Drews-Botsch, C., and Falek, A. (2000). Early identification of risk for effects of prenatal alcohol exposure. *J. Stud. Alcohol* 61, 607–616.
- Coles, C. D., Platzman, K. A., Lynch, M. E., and Freides, D. (2002). Auditory and visual sustained attention in adolescents prenatally exposed to alcohol. *Alcohol. Clin. Exp. Res.* 26, 263–271.
- Cook, J. D. (2003). Biochemical markers of alcohol use in pregnant women. *Clin. Biochem.* 36, 9–19.
- Czarnecki, D. M., Russell, M., Cooper, M. L., and Salter, D. (1990). Five-year reliability of self-reported alcohol consumption. *J. Stud. Alcohol* 51, 68–76.
- Elliott, E. J., and Bower, C. (2004). FAS in Australia: fact or fiction? *J. Paediatr. Child Health* 40, 8–10.
- Ernhart, C. B., Morrow-Tlucak, M., Sokol, R. J., and Martier, S. (1988). Underreporting of alcohol use in pregnancy. *Alcohol. Clin. Exp. Res.* 12, 506–511.
- Ernhart, C. B., Sokol, R. J., Ager, J. W., Morrow-Tlucak, M., and Martier, S. (1989). Alcohol-related birth defects: assessing the risk. *Ann. N. Y. Acad. Sci.* 562, 159–172.
- Ernhart, C. B., Sokol, R. J., Martier, S., Moron, P., Nadler, D., Ager, J., et al. (1987). Alcohol teratogenicity in the human: a detailed assessment of specificity, critical period, and threshold. *Am. J. Obstet. Gynecol.* 156, 33–39.
- Ewing, J. A. (1984). Detecting alcoholism: the CAGE questionnaire. *JAMA* 252, 1905–1907.
- Fried, P. A., O'Connell, C. M., and Watkinson, B. (1992). 60- and 72-month follow-up of children prenatally exposed to marijuana, cigarettes, and alcohol: cognitive and language assessment. *J. Dev. Behav. Pediatr.* 13, 383–391.
- Greene, T., Ernhart, C. B., Martier, S., Sokol, R. J., and Ager, J. (1990). Prenatal alcohol exposure and language development. *Alcohol. Clin. Exp. Res.* 14, 937–945.
- Hollingshead, A. B. (1975). *Four-factor index of social status*. Unpublished paper. New Haven, CT: Department of Social Work. Yale University.
- Howell, K. K., Lynch, M. E., Platzman, K. A., Smith, G. H., and Coles, C. D. (2006). Prenatal alcohol exposure and ability, academic achievement, and school functioning in adolescence: a longitudinal follow-up. *J. Pediatr. Psychol.* 31, 116–126.
- Hoyme, H. E., May, P. A., Kalberg, W. O., Kodituwakku, P., Gossage, J. P., Trujillo, P. M., et al. (2005). A practical clinical approach to diagnosis of fetal alcohol spectrum disorders: clarification of the 1996 Institute of Medicine criteria. *Pediatrics* 115, 39–47.
- Jacobson, S. W. (1998). Specificity of neurobehavioral outcomes associated with prenatal alcohol exposure. *Alcohol. Clin. Exp. Res.* 22, 313–320.
- Jacobson, S. W., Chiodo, L. M., Sokol, R. J., and Jacobson, J. L. (2002). Validity of maternal report of prenatal alcohol, cocaine, and smoking in relation to neurobehavioral outcome. *Pediatrics* 109, 815–825.
- Jacobson, J. L., and Jacobson, S. W. (1994). Prenatal alcohol exposure and neurodevelopmental development: where is the threshold? *Alcohol Health Res. World* 18, 30–36.
- Jacobson, J. L., Jacobson, S. W., Sokol, R. J., Martier, S. S., Ager, J. W., and Kaplan, M. G. (1991). Maternal recall of alcohol, cocaine, and marijuana use during pregnancy. *Neurotoxicol. Teratol.* 13, 535–540.
- Jacobson, S. W., Jacobson, J. L., Sokol, R. J., Chiodo, L. M., and Corobana, R. (2004). Maternal age, alcohol abuse history, and quality of parenting as moderators of the effects of prenatal alcohol exposure on 7.5-year intellectual function. *Alcohol. Clin. Exp. Res.* 28, 1732–1745.
- Jacobson, J. L., Jacobson, S. W., Sokol, R. J., Martier, S. S., Ager, J. W., and Kaplan-Estrin, M. G. (1993). Teratogenic effects of alcohol on infant development. *Alcohol. Clin. Exp. Res.* 17, 174–183.
- Kable, J. A., and Coles, C. D. (2004). The impact of prenatal alcohol exposure on neurophysiological encoding of environmental events at six months. *Alcohol. Clin. Exp. Res.* 28, 489–496.
- Kleinbaum, D. G., Kupper, L. L., and Muller, K. E. (1988). *Applied Regression Analysis and Other Multivariable Methods*, 2nd ed., Boston, MA: PWS-Kent.
- Kodituwakku, P. W. (2007). Defining the behavioral phenotype in children with fetal alcohol spectrum disorders: a review. *Neurosci. Biobehav. Rev.* 31, 192–201.
- Kodituwakku, P. W., Coriale, G., Fiorentino, D., Aragon, A. S., Kalberg, W. O., Buckley, D., et al. (2006). Neurobehavioral characteristics of children with fetal alcohol spectrum disorders in communities from Italy: preliminary results. *Alcohol. Clin. Exp. Res.* 30, 1551–1561.

- Kulaga, V., Caprara, D., Iqbal, U., Kapur, B., Klein, J., Reynolds, J., et al. (2006). Fatty acid ethyl esters (FAEE): comparative accumulation in human and guinea pig hair as a biomarker for prenatal alcohol exposure. *Alcohol Alcohol.* 41, 534–539.
- Little, R. E. (1976). Alcohol consumption during pregnancy as reported to the obstetrician and to an independent interviewer. *Ann. N. Y. Acad. Sci.* 273, 588–592.
- Little, R. E., Mandell, W., and Schultz, F. A. (1977). Consequences of retrospective measurement of alcohol consumption. *J. Stud. Alcohol* 38, 1777–1780.
- Manning, M. A., and Hoyme, E. H. (2007). Fetal alcohol spectrum disorders: a practical clinical approach to diagnosis. *Neurosci. Biobehav. Rev.* 31, 230–238.
- Mattson, S. N., and Riley, E. P. (1999). Implicit and explicit memory functioning in children with heavy prenatal alcohol exposure. *J. Int. Neuropsychol. Soc.* 5, 462–471.
- May, P. A., Brooke, L., Gossage, J. P., Croxford, J., Adnams, C., Jones, K. L., et al. (2000). Epidemiology of fetal alcohol syndrome in a South African community in the Western Cape Province. *Am. J. Public Health* 90, 1905–1912.
- May, P. A., Brooke, L., Gossage, J. P., Snell, C., Hendricks, L., Croxford, J., et al. (2005). Maternal risk factors for fetal alcohol syndrome in the Western Cape Province of South Africa: a population-based study. *Am. J. Public Health* 95, 1190–1199.
- May, P. A., Fiorentino, D., Gossage, J. P., Kalberg, W. O., Hoyme, H. E., Robinson, L. K., et al. (2006). Epidemiology of FASD in a province in Italy: Prevalence and characteristics of children in a random sample of schools. *Alcohol. Clin. Exp. Res.* 30, 1562–1575.
- May, P. A., and Gossage, J. P. (2001). Estimating the prevalence of fetal alcohol syndrome: a summary. *Alcohol Res. Health* 25, 159–167.
- May, P. A., Gossage, J. P., Marais, A. S., Adnams, C. M., Hoyme, H. E., Jones, K. L., et al. (2007). The epidemiology of fetal alcohol syndrome and partial FAS in a South African community. *Drug Alcohol Depend.* 88, 259–271.
- May, P. A., Gossage, J. P., Marais, A. S., Hendricks, L. S., Snell, C., Tabachnick, B. G., et al. (2008). Maternal risk factors for fetal alcohol syndrome and partial fetal alcohol syndrome in South Africa: a third study. *Alcohol. Clin. Exp. Res.* 32, 738–753.
- Morrow-Tlucak, M., Ernhart, C. B., Sokol, R. J., Martier, S., and Ager, J. (1989). Underreporting of alcohol use in pregnancy: relationship to alcohol problem history. *Alcohol. Clin. Exp. Res.* 13, 399–401. <http://firstsearch.oclc.org/WebZ/FSQUERY?searchtype=hotauthors:format=BI:numrecs=10:dbname=MEDLINE::termh1=Sokol+RJ:indexh1=au%3D:sessionid=fsapp5-38227-fci9c0bz-sy0v8w:entitypagenum=42:0:next=html/records.html:bad=error/badsearch.html>.
- Nardini, K., Anderson, R., and the National Association of State Alcohol and Drug Abuse Directors (NARSADAD). (2006). Alcohol research on prenatal alcohol exposure, prevention, and implications for state AOD. Systems State Issue Brief No. 2 [online]. Available at: <http://pubs.niaaa.nih.gov/publications/NASADAD/PrenatalBrief2.htm>. Accessed February 14, 2007.
- Nash, K., Rovet, J., Greenbaum, R., Fantus, E., Nulman, I., and Koren, G. (2006). Identifying the behavioural phenotype in fetal alcohol spectrum disorder: sensitivity, specificity and screening potential. *Arch. Womens Ment. Health* 9, 181–186.
- Nordstrom-Bailey, B., Delaney-Black, V., Covington, C. Y., Ager, J., Janisse, J., Hannigan, J. H., et al. (2004). Prenatal exposure to binge drinking and cognitive and behavioral outcomes at age 7 years. *Am. J. Obstet. Gynecol.* 191, 1037–1043.
- O'Leary, C. M. (2004). Foetal alcohol syndrome: diagnosis, epidemiology and developmental outcomes. *J. Paediatr. Child Health* 40, 2–7.
- Ostrea, E. M. Jr., Hernandez, J. D., Bielawski, D. M., Kan, J. M., Leonardo, G. M., Buda-Abela, M., et al. (2006). Fatty acid ethyl esters in meconium: are they biomarkers of fetal alcohol exposure and effect? *Alcohol. Clin. Exp. Res.* 30, 1152–1159.
- Richardson, G. A., Day, N. L., and Goldschmidt, L. (1995). Prenatal alcohol, marijuana, and tobacco use: infant mental and motor development. *Neurotoxicol. Teratol.* 17, 479–487.
- Riley, E. P., Mattson, S. N., Li, T. K., Jacobson, S. W., Coles, C. D., Koditwakku, P. W., et al. (2003). Neurobehavioral consequences of prenatal alcohol exposure: an international perspective. *Alcohol. Clin. Exp. Res.* 27, 362–373.
- Robles, N., and Day, N. L. (1990). Recall of alcohol consumption in pregnancy. *J. Stud. Alcohol* 51, 403–407.
- Rosett, H. L., and Weiner, L. (1984). *Alcohol and the Fetus*. New York: Oxford Press. pp. 105–112.
- Russell, M., Czamecki, D. M., Cowan, R., McPherson, E., and Mudar, P. J. (1991). Measures of maternal alcohol use as predictors of development in early childhood. *Alcohol. Clin. Exp. Res.* 15, 991–1000.
- Sampson, P. D., Streissguth, A. P., Bookstein, F. L., Little, R. E., Claren, S. K., Dehaene, P., et al. (1997). Incidence of fetal alcohol syndrome and prevalence of alcohol-related neurodevelopmental disorder. *Teratology* 56, 317–326.
- Schlesselman, J. J. (1982). *Case-Control Studies, Design, Conduct and Analysis*. New York, NY: Oxford University Press.
- Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: the quest for a new diagnostic instrument. *Am. J. Psychiatry* 127, 1653–1658.
- Sokol, R. J., Delaney-Black, V., and Nordstrom, B. (2003). Fetal alcohol spectrum disorders. *JAMA* 290, 2996–2999.
- Sokol, R. J., Martier, S., and Ager, J. W. (1989). The T-ACE questions: practical prenatal detection of risk-drinking. *Am. J. Obstet. Gynecol.* 160, 863–870.
- Sokol, R., Martier, S., and Ernhart, C. (1985). Identification of alcohol abuse in the prenatal clinic. In N. C. Chang, & H. M. Chao (Eds.), *NIAAA Research Monograph 17: Early Identification of Alcohol Abuse* (pp. 85–128). Washington, DC: DHHS Publication, U.S. Department of Health and Human Resources.
- Spadoni, A. D., McGee, C. L., Fryer, S. L., and Riley, E. P. (2007). Neuroimaging and fetal alcohol spectrum disorders. *Neurosci. Biobehav. Rev.* 31, 239–245.
- Stratton, K., Howe, C., and Battaglia, F. (1996). *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment*. Washington, DC: National Academy Press.
- Streissguth, A. P., Bookstein, F. L., Barr, H. M., Press, S., and Sampson, P. D. (1998). A fetal alcohol behavior scale. *Alcohol. Clin. Exp. Res.* 22, 325–333.
- Streissguth, A. P., Martin, D. C., and Buffington, V. E. (1976). Test-retest reliability of three scales derived from a quantity-frequency-variability assessment of self-reported alcohol consumption. *Ann. NY Acad. Sci.* 273, 458–466.
- Uecker, A., and Nadel, L. (1996). Spatial locations gone awry: object and spatial memory deficits in children with fetal alcohol syndrome. *Neuropsychologia* 34, 209–222.
- Vaurio, L., Riley, E. P., and Mattson, S. N. (2008). Differences in executive functioning in children with heavy prenatal alcohol exposure or attention-deficit/hyperactivity disorder. *J. Int. Neuropsychol. Soc.* 14, 119–129.
- Wechsler, D. (1991). *Wechsler Intelligence Scale for Children*, 3rd ed., New York, NY: Psychological Corporation.