

Maternal prenatal substance use and behavior problems among children in the U.S.

Sen, Bisakha and Swaminathan, Shailender University of Alabama at Birmingham

31. October 2007

Online at http://mpra.ub.uni-muenchen.de/24307/ MPRA Paper No. 24307, posted 09. August 2010 / 21:41

Maternal Prenatal Substance Use and Behavior Problems Among Children in The U.S.

Bisakha Sen,^{1*} Shailender Swaminathan²

¹Associate Professor, Department of Healthcare Organization & Policy, University of Alabama at Birmingham, Birmingham, AL, USA ²Research Fellow, Center for Gerontology & Healthcare Research, Brown University, Providence, RI, USA

Abstract

Background: Prenatal exposure to smoking and alcohol consumption is associated with various adverse physical health consequences for children. Numerous studies find that prenatal substance use is associated with low birthweight, as well as subsequent developmental and cognitive problems. A growing body of literature has also begun to show associations between prenatal exposure to smoking or/and alcohol and behavior problems among children. However, it is not clear whether these latter associations arise from underlying confounding factors that can impact both the mother's decision to smoke or drink during pregnancy and subsequent child behavior.

Aims of Study: This study investigate the relationship between prenatal substance use and subsequent children's behavior problems in early childhood (4-6.5 years) and in later childhood (8-10.5 years). The datasets used are the Children of the National Longitudinal Survey (CNLSY), linked with the National Longitudinal Survey of Youth (NLSY79).

Methods: Prenatal substance use is measured by binary indicators of smoking during pregnancy and alcohol-use during pregnancy. The outcome of interest is the age and gender specific standardized Behavior Problem Index (BPI) scale that is constructed using 32 mother-reported items on the child's behavior, as well as six subscales of problem behavior. Initially OLS regressions are estimated to verify the positive association between prenatal substance use and higher-levels of behavior problems. Thereafter, maternal fixed effects, maternal household fixed effects, propensity score matching, and propensity score inclusive regressions are all employed to obtain estimates of the effects of prenatal smoking and alcohol-use after reducing bias from unobserved confounding factors.

Results: Initial OLS results find very strong associations between prenatal smoking and alcohol-use and higher levels of behavioral problems among both younger and older children. However, when we use fixed-effects, propensity-score matching and propensityscore inclusive regressions, prenatal alcohol use continues to be significant related with increases in behavior problems, but prenatal smoking by and large ceases to have any significant effects.

Source of Funding: This research was supported by an intramural grant from the Lister Hill Center, UAB.

Discussion: While prenatal smoking has many deleterious outcomes for children, mostly related to low birthweight, it appears that the association between prenatal smoking and behavioral problems among children is largely driven by other confounding factors. On the other hand, results of this study suggest that prenatal alcohol-use may have true physiological/biological effects on the fetus that eventually exacerbate behavior problems. However, it should be noted that none of the methods used can account for all potential confounding factors – especially time-variant ones – hence, there may still remain some estimation bias. It should also be noted that the study suffers from certain shortcomings – namely, behavioral problems as well as prenatal substance-use are all based on motherreported data, and thus there are concerns about the accuracy of these measures. Hence, there remains scope for further research into this topic using alternate datasets.

Implications for Policy: The 1999 United States Surgeon General's Report stated that almost one in five children and adolescents in the U.S. exhibit signs of mental and behavioral disorder. This study suggests that policies aimed at reducing alcohol-use among pregnant women might contribute to reducing the prevalence of such disorders. However, while reducing cigarette use among pregnant women has numerous other health benefits for their children, it may not help reduce the incidence of behavior problems.

Received 16 August 2006; accepted 15 October 2007

Introduction

The United States Surgeon General's Report, 1999, states that in the United States, approximately one in five children and adolescents in the U.S. exhibit the signs or symptoms of mental or behavioral disorders, and evidence suggests that children with these disorders stand at a higher risk of becoming school drop-outs and failing to become fully functional members of society in adulthood.¹ The background factors correlated with increased risk of behavioral disorders among children are many – including low birth weight, family history of mental and addictive disorders, poor socio-economic status, multigenerational poverty, and prenatal exposure to tobacco, alcohol, and other addictive substances.

Use of cigarettes and alcohol during pregnancy continues to be a substantial problem in the U.S. Data published by the Centers for disease control $(CDC)^2$ show that the overall prevalence of smoking among pregnant mothers fell from

^{*} **Correspondence to:** Bisakha Sen, Associate Professor, Dept of Healthcare Organization & Policy U. Alabama at Birmingham, RPHB 330, 1665 University Blvd, Birmingham, AL 35294, USA.

Tel.: +1-205-975 8960

Fax: +1-205-934 3347

E-mail: BSen@ms.soph.uab.edu

14.6 % in 1994 to just over 12 % in 1999. However, for teen mothers the prevalence of smoking during pregnancy increased 1 percentage point from 16.5 % to 17.5 % during the same time period. This fact, taken along with the relatively high birth rates (10 per 1000) in the age group 15-19, is cause for worry. In addition to smoking, alcohol prevalence during pregnancy is also a non-negligible problem. The National Pregnancy and Health Survey carried out in 1992/93 found that among the women in their sample, about 18.9 percent drank alcohol products during their pregnancies, 20.5 percent smoked cigarettes during their pregnancies, and there was a strong, positive correlation between cigarette consumption and alcohol consumption. In light of this, it seems likely that a substantial number of newborn children will continue to be exposed to alcohol and tobacco in the prenatal stage. Therefore, it is important to continue researching the various aspects of the costs imposed on children and on society from prenatal exposure to cigarettes and alcohol, including potential costs in form of exacerbated behavior problems in children.

The primary issue of interest in our study is the relationship between prenatal exposure to alcohol and tobacco and behavior problems among children. A growing literature (discussed further in the next section) finds associations between prenatal substance use and other problems among children - including exacerbated behavioral problems. However, while the existence of a correlation between prenatal substance use and children's behavior problems is convincingly established, it is not clear to what extent the relationship is 'real', and to what extent it is an artifact of underlying unobservable characteristics of the mother and/or of the home environment that lead both to substance use during pregnancy and to later behavior problems among children. It may be these characteristics, rather than the prenatal substance use per se, that exacerbate behavior problems in their children, and failure to account for this possibility can potentially bias the estimated effect of prenatal substance use on children's behavior. This can potentially lead to incorrect predictions about the true social costs of prenatal substance use, as well as incorrect predictions about the extent to which polices and programs that lower prenatal substance use will reduce behavior problems among children. Thus, we explore whether the relationship between prenatal substance use and children's behavior problems persist after the potential bias from confounding factors is minimized using appropriate empirical techniques.

Our study uses data from the Children of the National Longitudinal Survey of Youth (CNLSY). We consider the relationship between maternal smoking/drinking and behavior problems for children at two stages of childhood – when they are 'younger' (age 4-6 years) and when they are 'older' (age 8-10 years).* We start by estimating 'naïve' Ordinary Least Squares (OLS) regression equations to find the correlation between maternal prenatal use of cigarettes, alcohol, and a general Behavior Problem Index, as well as the sub-categories of behaviors that make up the main index. These models control for a conventional set of demographic and socio-economic characteristics. Thereafter, we re-estimate our models using maternal household level fixed-effects (which is made possible by the fact that some of the mothers in our dataset are siblings), and maternal level fixed-effects (made possible by the fact that some of the mothers in our dataset have multiple children). Finally, we use propensity score matching methods, and propensity scores in the regression context both with and without fixed-effects. Notably, we also attempted to use instrumental variables methods, but they performed poorly and produced unreliable results, hence we omit those results from our analyses.

Briefly, our findings are as follows: the naïve regressions yield strong associative relationship between prenatal substance use and increased behavior problems both among younger and older children. We continue to find significant relationships between maternal prenatal drinking and increases behavior problems using fixed-effects and propensity score methods. However, the relationship between prenatal smoking and behavior problems ceases to be significant in most of the cases, suggesting that those associations are largely driven by underlying confounders.

Background

Smoking during pregnancy is one of the most important risk factors for premature, low birth weight babies. An estimated 21-39% of LBW births are attributable to smoking.³ In turn, low birth weight babies have increased risk of morbidity, both in childhood and as adults,^{4,5} as well as several later life problems such as behavior problems and lower competence.⁶ Additionally, there is evidence that children exposed to prenatal smoking and alcohol may display behavior problems even apart from those arising from low birth weight.⁷⁻⁹ It has been posited that intra-uterine exposure to alcohol or nicotine is a biologic risk factor that can exert effects on brain structure and function and increases the likelihood of subsequently developing behavioral disorders.¹⁰

Clinical studies using convenience samples have established strong correlations between children's behavior problems and maternal prenatal smoking¹¹⁻¹³ and children's behavior problems and maternal prenatal drinking.^{14,9,15}* The association between children's behavior problems and maternal prenatal smoking is also confirmed by studies using large, secondary datasets. For example, Fergusson *et al.*⁷ use data on 1265 children in New Zealand and show that mothers that smoked in excess of 20 cigarettes daily had children with

190

^{*} We deliberately stop at the pre-adolescent stage, since onset of adolescence may in itself trigger fresh behavior problems.

^{*} For example, Wakschlag *et al.*¹² followed 177 boys aged 7-12 at time of first assessment who had been clinic-referred; Wakschlag *et al.*¹³ used the children of 60 mothers who agreed to participate from selected clinics in a Midwestern urban area. Roebuck *et al.*⁹ compared 32 children exposed to prenatal alcohol to a 'control group' of 32 children matched by age, gender and ethnicity. Sood *et al.*¹⁵ used a sample of children whose mothers had used an urban university-based maternity clinic in Detroit.

mean behavior problem scores 0.3 standard deviations more than children born to mothers that smoked less than 20 cigarettes daily; Weitzman et al.8 use data from first round of the Children of NLSY and find a positive association between maternal cigarette smoking and child behavior problems - though they are not able to distinguish whether its prenatal or postnatal smoking that primarily contribute to the problem. Maughan et al.¹⁶ use data from a 1970 British cohort study and find a strong dose-response relationship between extent of prenatal smoking and onset of childhood behavior problems, which are robust to social background factors and maternal characteristics, but not to maternal smoking after birth. Martin et al.¹⁷ use data from the Helsinki Longitudinal Project and find associations between prenatal smoking, temperament, behavior, and academic performance of children at age 5 and at age 12 - this is one of the few studies that investigate the association between prenatal smoking and child outcomes at different stages of childhood. In most of the above studies, the authors speculate that the results may be due to a biologic pathway from smoking/alcohol use to brain development to behavior, but add the caveat that there may be other confounding familial and maternal characteristics that give rise to the relationship, and that more research is required to explore whether that is the case.

Essentially, the issue is whether the behavior problems are a result of effects of intrauterine exposure to such substances on brain structure, or whether there are underlying confounding factors that are correlated both with mother's substance use during pregnancy and subsequent behavior problems in children. For example, smoking/drinking during pregnancy may be correlated with maternal cognitive, behavioral or mental health problems or low familial socioeconomic status, and these factors may contribute towards exacerbating children's behavior problems. Mothers who use substances while pregnant might also lack concern for the wellbeing of the child or otherwise have poor parenting skills, which can later lead to children's behavior problems. Under these circumstances, we might erroneously attribute a biological pathway from prenatal substance use to brain development to behavior, when in fact it is the other factors that play the larger role in affecting child behavior. In that case, smoking and drinking cessation policies that target pregnant women are unlikely to have the desired effect of reducing child behavior problems. Thus, in this study we consider ways to gauge the impact of prenatal substance use on children's behavior problems while trying to circumvent the problem of bias arising from such potentially confounding factors.

A few studies in the economics literature consider the effects of parental substance use on child health. For example, the seminal work by Rosenzweig and Wolpin¹⁸ treats maternal smoking as one of the inputs in the production function for child health. The study finds that prenatal smoking does have a detrimental effect on birthweight. Evans and Ringel¹⁹ consider the effect of excise cigarette taxes on prenatal smoking. Their estimates show that increased cigarette taxes reduce prenatal smoking and have beneficial effects on infant birthweight. These reduced

form relations are then used to do an instrumental variables estimation of the effects of prenatal maternal smoking on infant birthweight. The study concludes that prenatal smoking has causal effects on infant birthweight, and that increased cigarette taxes will be effective in reducing prenatal smoking and thus the number of low-birthweight babies.

More recent work by Chatterji and Markowitz²⁰ consider the effects of current maternal alcohol and illicit drug use on children's behavior after controlling for confounding familial or maternal characteristics by using 'fixed-effects' and instrumental variables techniques. They caution against relying on their instrumental variables results due to poor performance of the instruments. Results from their different fixed-effects specifications suggest that maternal drug use exacerbates children's behavior problems, but the effects of alcohol use are uncertain.

Data

The data for this study is drawn from the 'Children of the National Longitudinal Survey of Youth' (hereafter CNLSY). The National Longitudinal Survey of Youth (hereafter NLSY79) is a longitudinal national survey that was initiated in 1979 with 12,686 young people (6,283 of them female) aged 15-22, conducted annually up to 1994, and is being conducted biennially since then. Beginning in 1986, under the sponsorship of the National Institute of Child Health and Human Development (NICHD), the CNLSY was initiated as a supplemental biannual survey where the children of the female respondents of the NLSY79 were assessed on a range of developmental issues, including motor, social and cognitive development, and behavior problems. The 1986 survey included 5,255 children born to 2774 female NLSY79 respondents. We use data up to 1998, by which time the CNSLY included 8,395 children born to 3,533 NLSY79 female respondents. Family identification codes make it easy to link the information of a child from the CNLSY to any necessary information for the mother from the NLSY79.

The NLSY79 provides for all its respondents extensive longitudinal information on education, income and socioeconomic status, family background, labor market experiences, marital experiences, fertility and parenting experiences, religious upbringing, health, substance use at different stages in life, and other characteristics. A separate Geocode CD makes available the state and county of residence of each respondent in each survey year, making it possible to integrate state and county level variables of the researchers' choice with the main data.

For each child included in the CNLSY, the survey provides retrospective information about various prenatal and postnatal characteristics as reported by the mother, which include the mother's use of alcohol and cigarettes during pregnancy. Postnatal information includes gestation age, birthweight, breast-feeding practices, and the child's health history over the first year of life. Finally, in every survey year, the CNLSY provides for the children aged between 4-14 years a Behavior Problem Index (BPI) based on 32

	Youngest Age Group $(N = 6742)$			ge Group 4453)
	Mean	Std. Dev	Mean	Std. Dev
BPI standardized score	104.09	(14.95)	106.50	(14.87)
Antisocial standardized score	104.86	(14.41)	107.47	(14.37)
Anxious/Depressed standardized score	102.21	(13.20)	103.71	(13.30)
Headstrong standardized score	101.26	(12.87)	103.75	(13.26)
Hyperactive standardized score	105.47	(14.55)	103.97	(14.50)
Dependent standardized score	105.96	(13.87)	105.88	(13.53)
Peer conflict standardized score	104.78	(12.63)	104.58	(12.67)

mother-reported items on the children's behavior.* The BPI items, developed by Zill²¹ are modeled after the items from the Child Behavior Checklist (CBCL) of Achenbach and Edelbrook,²² and are chosen because of their reliability, high loading on the subscales of the CBCL, and adaptability to interview situations. The BPI contains within itself 6 subscales for specific categories of behavior problems. These are respectively: antisocial, anxious or depressed, headstrong, hyperactive, dependent and peer conflict. **Table A1** in the **Appendix** describes the specific items used to construct each of the subscales. Our empirical estimations use as dependent variables the standardized scores, adjusted for sex, age and school-enrollment status, for the aggregate BPI as well as for each of the separate sub-scales. In all cases, higher standardized scores reflect a higher degree of behavior problems.

We confine our sample to children born in 1981 or later,† for whom standardized BPI scores are available for at least one interview year. To address the general paucity of studies that look at the relationship between prenatal substance use and child outcomes at different stages of childhood, we focus on BPI scores at two separate stages of childhood – early childhood or 'younger group' (4-6.5 years), and later childhood or 'older group' but still pre-pubescent (age-group 8.5-10.5 years).* We deliberately omit observations for even older children, because puberty and entry into adolescence is a tumultuous stage of growth which may contribute to new or sudden changes in behavior problems, and may confound results.

For each child in each group, we use only the first year of data within that age-range for which the BPI scores are available for that child. Thus, there is only one observation per child within each age-group, and within each group, the majority of children are from the lower end of the agerange.* Due to the biennial nature of our data, we do not observe all children at all ages. Furthermore, children born in the earlier calendar years tend to be observed in both groups, whereas those born in later years are more likely to only be seen in the younger groups. Table 1 presents standardized scores for the BPI and the behavior subscales for each agegroup. Note that these scores should not be taken as representative of the population-wide scores for that agegroup due to the fact that children of younger mothers are overrepresented in the CNLSY, and due to the oversampling of blacks, Hispanics, and poor whites in the original NLSY79.

Empirical Model & Estimation Methods

The primary hypotheses of interest are whether prenatal alcohol use and cigarette use increase behavior problems in the children after accounting for potential endogeneity.[†]

The basic equation of interest is:

$$B_{jt} = PNS_j \lambda + X_{jt} \beta + e_{jt}$$
(1)

Where B_{jt} is a behavioral problem index (BPI) for child 'j' at time 't', PNS_j is a vector of binary indicators for prenatal cigarette and alcohol use (1 if substance use occurs, 0 otherwise), and X_{jt} is a vector of other observable characteristics that can affect the child's behavior problems. Higher values of B_{jt} correspond to more behavior problems

^{*} The reason for this is twofold. The CNLSY, by its design, over-represents children born to very young mothers, including mothers in their early and mid teens. By selecting children born in 1981 and later, we are able to ensure that we only pick up the children who are born to mothers aged 17 and higher, so that our results can be somewhat more readily generalized. Secondly, some of the state-policies for alcohol and tobacco that we used in the IV methods (though we later rejected that method) and also in the propensity score methods were only available to us for 1981 and later.

[†] This restriction is prompted by the availability of state policies that we use in our instrumental variables technique.

^{*} We measure age in months and convert it to years. In the first age-group, we have a small fraction of children who are a couple of months short of their 4th birthday. We include them in the data if information on their BPI is available.

^{*} In the younger group, 44.5 percent of the children are under age 5, 52 percent are between ages 5 and 6, and the remaining are older than 6. In the older group, 25 percent of respondents are between 8.5 and 9 years, 53.8 percent are between 9 and 10 years, and the remainder are older than 10 years.

⁺ Though it would have been interesting to do the analyses for other prenatal substance use as well, the numbers of mothers reporting prenatal use of marijuana/other illegal drugs seem too small to get meaningful estimation results.

Table 2. Descriptive Statistics by Age-Group

	Younger Children		Older	Children
	Mean	Std. Dev	Mean	Std. Dev
Prenatal smoke	0.27	(0.44)	0.27	(0.45)
Prenatal drink	0.30	(0.46)	0.32	(0.47)
Child was breast-fed	0.43	(0.49)	0.43	(0.49)
Breast fed information missing	0.07	(0.25)	0.04	(0.20)
# of Prenatal Visits	0.93	(0.25)	0.95	(0.22)
Missing prenatal info	0.06	(0.23)	0.04	(0.20)
Child's Age	4.99	(0.61)	9.46	(0.57)
Mother's Highest Grade Completed	12.18	(2.47)	12.14	(2.44)
Father in household	0.64	(0.48)	0.53	(0.50)
Household in poverty	0.24	(0.43)	0.21	(0.41)
Household poverty status unknown	0.15	(0.36)	0.18	(0.38)
Mother Currently Employed	0.51	(0.50)	0.59	(0.49)
Rural Residence	0.21	(0.41)	0.18	(0.39)
City Residence	0.15	(0.36)	0.14	(0.35)
Child is male	0.51	(0.50)	0.51	(0.50)
Child's mother black	0.30	(0.46)	0.33	(0.47)
Child's mother of Hispanic origin	0.21	(0.41)	0.23	(0.42)
Mother's age at birth	25.40	(3.96)	24.06	(3.13)
Worked in year 1 after child's birth	0.34	(0.47)	0.33	(0.47)
Smoked after child's birth	0.24	(0.43)	0.26	(0.44)
Binge drink after child's birth	0.23	(0.42)	0.26	(0.23)
Foreign language at mother's home	0.25	(0.43)	0.26	(0.44)
Mother lived in intact family	0.64	(0.48)	0.64	(0.48)
At least one foreign-born parent	0.13	(0.33)	0.13	(0.33)
Mother's mother worked for pay	0.50	(0.50)	0.50	(0.50)
Newspapers in (mother's) home	0.71	(0.46)	0.69	(0.46)
Library card in (mother's) home	0.69	(0.46)	0.68	(0.47)
Self-esteem score	32.89	(4.06)	32.66	(4.01)
CESD Score	10.96	(9.45)	11.03	(9.71)
First home environment score	970.69	(160.90)	966.82	(161.90)
Number of children considered ideal by mother	3.03	(1.36)	3.06	(1.39)
Mother has alcoholic relative	0.53	(0.50)	0.53	(0.50)
Information on alcoholic relative missing	0.03	(0.17)	0.04	(0.18)
Mother reports no church attendance in 1979	0.15	(0.35)	0.14	(0.35)
Mother reports infrequent church attendance in 1979	0.34	(0.47)	0.35	(0.47)
Mother reports church attendance more than once a week in 1979	0.11	(0.32)	0.11	(0.31)

in the child. The variables included in the X_{jt} include the number of prenatal care visits during the pregnancy; a binary indicator for whether the child was breastfed as an infant; the *child's age* (measured as "months since birth" and divided by 12 to convert to years); maternal highest grade completed; a binary indicator for whether the child's father is currently in the household; a binary indicator for 'missing' family income and poverty status; a binary indicator for whether the family is in poverty employed; binary indicators for whether the family resides in a rural area and whether the family resides in a city; binary indicators for the child's gender, race (1 if black) and Hispanic origin; the mother's age at time of the

child's birth; and a vector of survey year dummy variables. **Table 2** provides variable means. We confine our measures of substance-use to two binary indicators – one for whether the respondent reported smoking during pregnancy and one for whether she reported alcohol-use during pregnancy.

As noted earlier, we estimate the parameter λ using several variants of the model in equation 1. They differ in the source of variation used to identify λ . In the initial OLS specification, we take advantage of all variation in PNS within mothers and between mothers. The models are initially estimated under the assumption that e_{jt} is independent and identically distributed. They are then reestimated after clustering on "Primary Sampling Units"

(PSU) to adjust for possible heteroskedasticity for respondents living in the same PSU.* Both of these methods make the 'naïve' assumption that the error term is uncorrelated with PNS_i. However, due to the high likelihood of the presence of at least some unmeasured characteristics in the error term that are correlated both with the treatment (PNS_i), and the outcome (B_{it}), the naïve OLS estimate of λ are very likely to be biased and inconsistent. Hence, the second set of models that we estimate are fixed-effects models - where we first estimate models with "maternal household fixed-effects", and then models with 'maternal fixed effects'. The former model exploits the fact that many members of the original NLSY79 sample were drawn from the same households, hence some of the mothers in our sample are siblings or step-siblings, and it estimates λ using within-sibling (step-sibling) variation in PNS. This estimator should be closer to the true population estimate than the naïve OLS estimator because it removes the bias due to unmeasured factors that are natal-family specific or are otherwise shared by all siblings. However, it will not remove potential bias arising from those unmeasured characteristics that the mother does not share with her siblings. The next model, with "maternal fixed-effects", uses only the variation in PNS among children born to the same mother to identify the effect λ . This second method removes the bias due to all time-invariant mother-specific unobservables. However, the efficacy of this method depends on having sufficient mothers who have multiple children included in the sample and whose prenatal substance use behaviors vary across the children. Table A2 in the Appendix presents the distribution of the number of child-observations from the same mother and also from the same maternal household.

Both maternal-household fixed-effects and maternal fixedeffects models have been previously used in the extant literature to control for confounding family-background and behavioral factors. For example, Geronimus and Korenman²³ used variations in age of first child-bearing within siblings from the same household to see if teen childbearing truly affected the mother's future socio-economic status. A second study by Geronimus and Korenman²⁴ used this same framework to investigate whether teen motherhood truly led to infant health disadvantages. Currie and Cole²⁵ used both variations in AFDC enrollment among siblings (i.e. 'maternal household fixed-effects' models) and variations in AFDC enrollment for the same woman over time (i.e. 'maternal fixed-effects' models) to investigate whether maternal enrollment in AFDC during pregnancy affected the child's birthweight. Rosenzweig and Wolpin²⁶ used maternal fixed-effects models to utilize variations in the same mother's education level at time of birth of different children to see whether maternal education at time of birth affected the child's subsequent aptitude in math and language skills. Finally, the previously mentioned study by Chatterji and Markowitz²⁰ used both maternal household fixed-effects and maternal fixed-effects models to consider the effects of current maternal alcohol and illicit drug use on children's behavior.

For all fixed-effects models, we estimated corresponding random-effects models and conducted Hausman tests to see which model was preferred. In all cases, the Hausman tests rejected the null hypothesis that coefficient estimates from the random-effects models were equal to those from the fixed-effects models at 5% or better level of significance. This supports the existence of mother-specific or maternalfamily specific time-invariant unobservables in the error term that are correlated with PNS, and bias the estimated λ in absence of fixed-effects methods. However, as noted earlier, the fixed-effects models cannot remove bias due to confounding factors that are mother-specific but also timevariant. Hence, we also estimate the models using one more method - a propensity scores approach. This method, which is succinctly described by D'agostino,²⁷ essentially involves estimating first stage binary regressions for the 'treatment' in question using as control all available and relevant observable characteristics; obtaining the predicted probability of being subject to the treatment; and finally, including that predicted probability (i.e. the 'propensity score') as an additional control in the final outcomes regression which also includes the binary indicator of treatment. While propensity scores, by definition, only control for observable factors, if one is able to use a wide range of observables that directly measure or adequately proxy for the potential confounders to construct this scores, then one can argue that the coefficient estimate of the binary treatment now actually provides a minimally biased estimate of the 'true' effect of the treatment - in this case the 'real' physiological/biological effects of being exposed to prenatal smoking and drinking.

The advantages of including the propensity score in the final regression rather than attempting a more conventional propensity score 'matching' method as proposed by Rosenbaum & Ruben²⁸ is twofold – first, it prevents the loss of sample size since we do not have to omit observations from the non-treated group which fail to closely match members from the treated group; second, there is no intuitively obvious method to perform a conventional PSM analysis when two separate treatments - in this case prenatal smoking and prenatal drinking - are being simultaneously considered, but the Propensity-score inclusive regression method can accommodate two or more simultaneous treatments. However, to see how robust our results are to alternative methods, we also present results from a conventional propensity-score matching analysis, where we compare mean outcome levels for the 'treated' group and a matched 'control' group with the same number of observations, which is done separately for the 'treatments' of exposure to maternal prenatal smoking and maternal prenatal drinking.

BISAKHA SEN ET. AL.

^{*} The NLSY79 is a multi-stage clustered sample. The clusters were created by first dividing the entire U.S. into Primary Sampling Units, or PSUs. These PSUs were defined by NORC and were composed of Standard Metropolitan Statistical Areas (SMSAs), entire counties when the counties were small, parts of counties when the counties were large, and independent cities. NORC randomly selected two different sets of PSUs for inclusion in the study, each of which by itself randomly represents the U.S. This selection of two sets of PSUs means the NLSY79 study is comprised of two replicates or strata. Within each is a random selection of PSUs. The replicate or strata that a respondent belongs to is found in the NLSY Geocode data set only. Instructions for how to use use clustering on PSU to 'correct' for this sampling design can be found in ftp://www.nlsinfo.org/pub/ usersvc/NLSY79/NLSY79%202004%20User%20Guide/79text/cluster.htm.

We regress first stage probits of maternal prenatal smoking and drinking for each child on the following variables:

- A series of controls for the mother's background, including whether she lived in an intact parental family at age 14, whether either of her parents were foreign-born, whether there was a foreign language spoken at home, whether *her* mother worked when she was a child, whether she reports having a relative who suffered from problems of alcoholism, and proxies for the learning environment in her home in form of whether she had access to a library card when growing up and whether there were newspapers regularly delivered to her home when she was growing up.
- Her religiosity, measured by frequency of church attendance as reported in the 1979 survey, and her attitudes towards childbearing, proxied by how many children she reported considering 'ideal' for a family in the 1979 interview.
- Two measures of maternal mental health. The first is based on the 10-item Rosenberg Self-Esteem Scale that measures the self-evaluation that an individual makes and customarily maintains. Higher scores on this scale are indicative of greater self-esteem.* The NLSY79 administered these questions in the 1980 and 1987 interviews. We use the information from 1987. The second is based on the 20-item Center for Epidemiological Studies Depression Scale (CES-D). This scale measures symptoms of depression, discriminates between clinically depressed individuals and others, and is highly correlated with other depression rating scales. The full set of items was administered to all respondents in the 1992 survey. Subsequently, a sub-set has been administered only to respondents over 40 years of age. We use the information from 1992. Higher values on this scale indicate more depressive tendencies.

Note that the above variables are all time invariant for a particular mother. Hence, we interact them with her age at the time of the birth of the child, thus allowing the relationship of these variables with prenatal substance use to vary across each child for the same mother.

The probits also include the following controls that potentially vary across each child:

- A binary indicator for whether the mother was employed in the first year after the child's birth.
- The 'emotional support' score from the first 'Home Observation Measurement for Environment – short form (HOME-SF)' score available for each child.† Higher scores indicate a more supportive home environment.

- Two binary indicators, for whether the mother smoked *after* the child's birth, and whether she indulged in bingedrinking *after* the child's birth.* These variables are meant to proxy for her unobserved affinity towards smoking and drinking. We focus on binge-drinking rather than drinking per se, since occasional moderate and responsible drinking by adults is sometimes considered to have health benefits and thus be part of a 'positive' lifestyle.
- All the instruments used in the (later described) instrumental variables method.
- A vector of dummy variables for the birth year of the child.
- Finally, we included all the controls from **Table 3** (excluding the child's gender and age), with time-variant factors like presence of a father now being measured at the time of the child's birth.

In the section of our analysis that uses conventional propensity score matching, we match each 'treated' child with a member of the non-treated group using radius matching within the caliper distance of 0.0005. The statistical software STATA is used for all estimations.

Results

Table 3 presents results from the 'naïve' OLS model where the aggregate standardized BPI-score is regressed on prenatal smoking, drinking, and the other controls listed in X_{it}, both with and without correcting standard errors for clustering upon Primary Sampling Units. Unsurprisingly, strong statistical associations are found between both prenatal smoking and drinking and higher aggregate BPI scores. Prenatal smoking correlates to average increases of 3.178 and 2.746 units in the aggregate standardized BPI score for the younger and older group respectively, and prenatal drinking correlates to average increases in 1.446 and 1.984 units respectively. Among the other variables, maternal education and presence of a father in the household are correlated with lower BPI scores. Poverty is correlated with higher BPI scores and age correlated with lower BPI scores for the younger group only. Race and gender are largely insignificant. Higher maternal age at time of birth is correlated with lower BPI scores only among the older agegroup. Table 4 presents corresponding results for the standardized behavioral sub-scores, with only results pertaining to prenatal smoking and drinking being shown. Results for other control variables are available upon request. Again, both prenatal smoking and drinking are correlated with very significant increases in all the sub-scores for both groups, with the magnitudes of increases ranging from 1.35 units to 2.9 units for prenatal smoking, and 0.69 units to 1.74 units for prenatal drinking.

Before we move onto presenting results from the fixedeffects and propensity score methods, it seems appropriate to briefly mention the results from the instrumental variables

^{*} The scale is short, widely used, and has accumulated evidence of validity and reliability. It contains 10 statements of self-approval and disapproval with which respondents are asked to strongly agree, agree, disagree, or strongly disagree. Of these, on five items disagreeing is indicative of higher self-esteem, while on the remaining five disagreeing is indicative of lower self-esteem, and thus must be reversed when the items are added.

[†] The 'emotional support' score consists of multiple items, some of which are mother-reported and others are interviewer observations. While this score is obtained each year up to when the child is 14years old, the questions and items change with child's age. Details may be found in Appendix A of the CNSY User's Guide.

^{*} Questions about current smoking and binge drinking when the respondent is not pregnant are asked sporadically. We set the binary variables to 1 if she ever reports doing so after the child's birth and before the last year in the survey.

MATERNAL PRENATAL SUBSTANCE USE AND BEHAVIOR PROBLEMS AMONG CHILDREN IN THE U.S.

Table 3. Regression Results for BPI Scores, OLS

Variables	Unadj standarc		Standard errors clustered on PSU		
	Younger Children coefficient (t-statistic)	Older Children coefficient (t-statistic)	Younger Children coefficient (t-statistic)	Older Children coefficient (t-statistic)	
Prenatal Smoke	3.178***	2.746***	3.178***	2.746***	
	(7.31)	(4.89)	(4.33)	(3.20)	
Prenatal Drink	1.446***	1.984***	1.446***	1.984***	
	(3.55)	(3.78)	(2.97)	(3.59)	
Breast-Feed	-0.412	-0.141	-0.412	-0.141	
	(-1.04)	(-0.27)	(-0.89)	(-0.20)	
Aissing Breast-Feed Info	-0.522	-0.454	-0.522	-0.454	
	(-0.59)	(-0.33)	(-0.41)	(-0.22)	
of Prenatal Visits	0.233	-0.877	0.233	-0.877	
	(0.14)	(-0.39)	(0.12)	(-0.28)	
Aissing Prenatal Info	-0.125	-0.649	-0.125	-0.649	
	(-0.07)	(-0.25)	(-0.06)	(-0.18)	
Child's Age	-1.146***	0.188	-1.146***	0.188	
-	(-3.34)	(0.41)	(-2.75)	(0.33)	
Aother's Highest Grade Completed	-0.493 * * *	-0.256**	-0.493 * * *	-0.256*	
	(-5.92)	(-2.35)	(-4.84)	(-1.80)	
ather in Household	-1.930***	-2.742^{***}	-1.930***	-2.742^{***}	
	(-4.54)	(-5.22)	(-3.71)	(-3.85)	
n Poverty	1.192**	0.982	1.192*	0.982	
	(2.37)	(1.46)	(1.92)	(1.20)	
Iousehold Income Missing	0.034**	0.243	0.034	0.243	
-	(2.37)	(0.37)	(0.05)	(0.28)	
Aother Currently Employed	-0.153	-0.743	-0.153	-0.743	
	(-0.41)	(-1.51)	(-0.31)	(-1.23)	
Rural Residence	-0.995^{**}	0.653	-0.995*	0.653	
	(-2.19)	(1.08)	(-1.65)	(0.94)	
City Residence	0.173	-1.127*	0.173	-1.127	
	(0.33)	(-1.65)	(0.24)	(-1.17)	
Child is Male	-0.353	0.247	-0.353	0.247	
	(-1.00)	(0.53)	(-0.84)	(0.43)	
Aother is black	0.68	0.983	0.68	0.983	
	(1.45)	(1.62)	(0.81)	(1.11)	
Aother is Hispanic	0.57	0.933	0.57	0.933	
~	(1.14)	(1.42)	(0.93)	(1.24)	
Mother's Age at Child birth	-0.124	-0.207*	-0.124	-0.207*	
-	(-1.52)	(-1.92)	(-1.10)	(-1.66)	

* indicates significance at 90%

** indicates significance at 95%

*** indicates significance at 99%

Notes: All equations also include a vector of survey year dummies. Readers are referred to footnote 9 for more information on PSU (Primary Sampling Units).

methods. This proved to be particularly problematic. The first challenge consisted of finding feasible instruments – namely, variables that were correlated to the probability of prenatal smoking and drinking, but not directly correlated with children's behavior problems. We could not identify any individual maternal characteristics that could logically satisfy these conditions. Thus, we were forced to fall back on statelevel policies that could potentially affect the price and availability of alcohol and cigarettes, though there are 196 concerns about using instruments that vary only by state and year, and not by individuals, to estimate individual behavior.²⁹

We chose as our instruments the following variables: inflation adjusted state beer taxes, inflation adjusted state cigarette taxes, the 'retail outlet density' defined as the number of outlets licensed to sell liquor for on-premise or off-premise consumption per 1000 population, a binary indicators for whether the state has in place a BAC limit of

BISAKHA SEN ET. AL.

Table 4. Regression	Results	for Behavi	ior Sub-Score	s, OLS
---------------------	---------	------------	---------------	--------

	Unadj standarc		Standard errors clustered on PSU		
	Younger Children coefficient (t-statistic)	Older Children coefficient (t-statistic)	Younger Children coefficient (t-statistic)	Older Children coefficient (t-statistic)	
Antisocial Score					
Prenatal Smoke	2.361***	2.611***	2.361***	2.611***	
	(5.60)	(4.86)	(3.39)	(3.38)	
Prenatal Drink	1.313***	1.455***	1.313**	1.455***	
	(3.32)	(2.89)	(2.59)	(2.67)	
Anxious/Depressed Score			· /		
Prenatal Smoke	1.266***	1.819***	1.266**	1.819**	
	(3.27)	(3.62)	(2.15)	(2.36)	
Prenatal Drink	0.981***	1.533***	0.981**	1.533***	
	(2.70)	(3.25)	(2.27)	(2.72)	
Headstrong Score		. ,			
Prenatal Smoke	2.329***	2.047***	2.329***	2.047***	
	(6.16)	(4.11)	(4.15)	(2.99)	
Prenatal Drink	1.710***	1.234***	1.710***	1.234**	
	(4.82)	(2.63)	(4.45)	(2.37)	
Hyperactive Score					
Prenatal Smoke	2.903***	2.130***	2.903***	2.130***	
	(6.89)	(3.94)	(4.63)	(2.90)	
Prenatal Drink	0.696*	1.738***	0.696	1.738***	
	(1.76)	(3.43)	(1.53)	(3.01)	
Dependent Score					
Prenatal Smoke	1.787***	1.545***	1.787***	1.545**	
	(4.39)	(3.08)	(2.91)	(2.03)	
Prenatal Drink	0.596	1.023**	0.596	1.023*	
	(1.56)	(2.16)	(1.27)	(1.92)	
Peer Conflict Score					
Prenatal Smoke	1.591***	1.356***	1.591***	1.356**	
	(4.27)	(2.86)	(3.30)	(2.03)	
Prenatal Drink	1.024***	1.611***	1.024**	1.611***	
	(2.94)	(3.61)	(2.49)	(3.08)	

* indicates significance at 90%

** indicates significance at 95%

*** indicates significance at 99%

Notes: Each regression also includes the other control variables from Table 3 and survey year dummies. Those results are available upon request.

0.08, and finally, the total acres of land in the state devoted to tobacco farming divided by the state's population. The first stage results are shown in **Table A3**. In the first stage equation, for prenatal smoking the instruments failed to meet the Stock-Staiger condition of a joint F-statistic of 10 or more, though they did so in case of prenatal drinking. Finally, in our second stage results (**Table A4**), both prenatal smoking and drinking appeared to be statistically insignificant, but in both cases the magnitudes of the estimates were implausibly large and for prenatal drinking the estimate had a counter-intuitive negative sign.* Specifically, when overall BPI score was the dependent variable, for the younger group the coefficient estimates for prenatal smoking and drinking were respectively 6.75 and -2.72 (as compared to coefficient estimates of 3.18 and 1.44

respectively in the OLS equation in **Table 3**). For the older group, the coefficient estimates for prenatal smoking and drinking were respectively 4.30 and -5.51 (as compared to coefficient estimates of 2.74 and 1.98 respectively in the OLS equation in **Table 3**). We are highly skeptical about the

^{*} It has been posited that, for IV estimators to have a mean and a variance, the number of instruments should exceed the number of endogenous variables at least by two (Kinal & Ecta³⁰ Davidson and MacKinnon.³¹ In our case, this implies 4 valid and significant instruments in each first stage equation – a condition that we're unable to meet. Econometricians have argued that this is not a required condition when the sample is large. However, recent work by Deb *et al.*³² demonstrates using Monte Carlo simulations that insufficient instruments can lead to 2SLS estimates taking implausible values, even with a sample size of 10,000. This leads to further concerns about the validity of the IV results that we obtained.

	Mate Fixed-o		Maternal Family Fixed-effects		
	Younger Children coefficient (t-statistic)	Older Children coefficient (t-statistic)	Younger Children coefficient (t-statistic)	Older Children coefficient (t-statistic)	
Overall BPI Score [#]					
Prenatal Smoke	-0.461	-1.356	-0.604	0.347	
	(-0.55)	(-1.18)	(-0.79)	(0.33)	
Prenatal Drink	1.047*	2.017***	1.094**	2.241***	
	(1.88)	(2.69)	(2.07)	(3.11)	
Antisocial Score					
Prenatal Smoke	0.065	-1.585	0.366	-0.378	
	(0.08)	(-1.31)	(0.46)	(-0.35)	
Prenatal Drink	1.262**	0.497	1.048**	1.153	
	(2.17)			(1.58)	
Anxious/Depressed Score					
Prenatal Smoke	-1.185	-0.472	-1.494 **	0.338	
	(-1.5)	(-0.42)	(-2.07)	(0.33)	
Prenatal Drink	0.843	1.696**	0.756	2.126***	
	(1.61)	(2.27)	(1.52)	(3.04)	
Headstrong Score					
Prenatal Smoke	-0.92	-0.198	-1.472**	0.983	
	(-1.18)	(-0.18)	(-2.10)	(0.96)	
Prenatal Drink	0.794	0.929	1.247***	1.345*	
	(1.53)	(1.26)	(2.58)	(1.95)	
Hyperactive Score					
Prenatal Smoke	-1.650*	1.51	-1.375*	1.756	
	(-1.89)	(1.22)	(-1.75)	(1.57)	
Prenatal Drink	1.158**	1.703**	1.406***	2.222***	
	(1.99)	(2.10)	(2.59)	(2.94)	
Dependent Score					
Prenatal Smoke	1.264	-1.298	0.344	-0.264	
	(1.46)	(-1.13)	(0.44)	(-0.26)	
Prenatal Drink	0.07	1.732**	0.03	2.33***	
	(0.12)	(2.26)	(0.05)	(3.33)	
Peer Conflict Score		. *		. *	
Prenatal Smoke	-0.602	0.261	0.664	0.2	
	(-0.71)	(0.23)	(0.88)	(0.20)	
Prenatal Drink	1.041*	1.558**	0.859*	1.695**	
	(1.84)	(2.09)	(1.65)	(2.46)	

Table 5. Regression Results for BPI Score & Behavior Sub-Scores, Fixed-effects

* indicates significance at 90%

** indicates significance at 95%

*** indicates significance at 99%

Notes: Each regression also includes the other control variables from **Table 3** and survey year dummies. Those results are available upon request. #: Standard Hausman tests were conducted to test the hypothesis that random effects and fixed-effects specifications yield statistically equal coefficient estimates. For equations with overall BPI score as dependent variables, the resultant chi-square statistics were respectively 93.18 and 47.37 for the younger and older children in models with maternal-level effects, and respectively 78.45 and 69.68 for the younger and older children in models with maternal-level effects, and respectively 78.45 and 69.68 for the younger and older children in models with maternal-level effects. Hausman test statistics for the behavior sub-categories are available upon request. The hypothesis (and hence the random effects model specification) is rejected in favor of the fixed-effects model specification in all cases.

credibility of these results. Hence, though in theory, instrumental variables methods are an ideal solution for the problem of underlying confounders, in practice, given the lack of an adequate number of good instruments, we must rely on the other methods described earlier to minimize the bias from confounders. **Table 5** presents results from the fixed-effects models; again, only the estimates pertaining to prenatal smoking and drinking are presented for economy of space. We now see that, in models with maternal fixed-effects, prenatal smoking now ceases to have any significant effects on aggregate BPI scores altogether, and among the sub-scores, it only has a

198

BISAKHA SEN ET. AL.

weakly significant effect on the Hyperactive score for the younger. Prenatal drinking, in contrast, continues to have significant correlations with the aggregate BPI scores, with increases of 1.05 units and 2.02 units for the younger and older groups respectively. It also continues to be significantly correlated with all sub-scores except 'dependent' for the younger group, and all sub-scores except 'antisocial' and 'headstrong' for the older group. Similar patterns are detected when using maternal household fixed-effects, with prenatal smoking not correlated with, and prenatal drinking significantly correlated with, the aggregate BPI scores and most of the sub-scores for both groups. Recall that the models are now driven by mothers who have multiple children present in the sample and who change their substance-use behavior across pregnancies, or women from the same household who exhibit different prenatal substance use behavior during pregnancy. In case of maternal fixedeffects, only 10.5 percent and 6.5 percent of the child observations from the respective age-groups satisfy these criterion in case of prenatal smoking, 11.2 percent and 7.0 percent do so in case of prenatal drinking. In case of maternal household fixed-effects, 16.3 percent and 12.4 percent of the child observations from the respective age-groups satisfy these criterion in case of prenatal smoking, 31.2 percent and 13.4 percent do so in case of prenatal drinking. It is possible that the insignificant effects of prenatal smoking are driven by loss of statistical power, but the fact that prenatal drinking is still found to have significant effects on behavior problems lends credence to these results. Also, as a supplementary analyses, we tested the correlation of prenatal smoking with children's birthweight using ordinary least squares as well as maternal fixed-effects. There, we found that prenatal smoking is negatively correlated with birthweight, and the results continued to be statistically significant after using maternal fixed-effects. This lends considerable credence to the adequacy of statistical power of any estimations that depends on variations in prenatal smoking within-mother in this data. However, it should also be noted that, if there exists a modest amount of measurement error in the substance-use variables, then this measurement error can be responsible for part of the divergence between coefficient estimates obtained using fixed-effects methods instead of regular OLS.* Additionally, because the substance-use variables are dichotomous, measurement error cannot be 'classical', hence it is extremely difficult to sign the direction of bias on the substance abuse coefficients.³³ Unfortunately, there is no way to gauge the existence or the extent of measurement error in substance use variables in these dataset.

Table 6 presents the results from the first stage probits for smoking and drinking. Table 7 presents t-tests for the hypothesis of equality of mean BPI scores and sub-scores for the 'treated' groups and matched 'control' groups of children, with the treatments of maternal prenatal smoking and maternal prenatal drinking considered separately. In the cases when the treatment is exposure to prenatal smoking, the treated group exhibits significantly higher mean scores for aggregate BPI than the control group for younger children, though among the behavioral sub-scores, only in case of the 'hyperactive' score do we reject equality of means at better than 5 percent significance, and in case of 'headstrong' and 'dependent' scores, we reject equality of means at 10 percent but not 5 percent level of significance. In case of older children, there are no significant differences either in the mean aggregate BPI scores or any of the behavioral sub-scores between the treated and control groups. In contrast, when the treatment is exposure to prenatal alcohol use, the treated groups exhibit significantly higher mean aggregate BPI scores and behavioral sub-scores than the control group among both the younger and the older children.

Table 8 presents estimates from the regressions inclusive of propensity scores for both maternal prenatal smoking and maternal prenatal alcohol use. We also present models inclusive of both the propensity scores and maternal household level or maternal level fixed-effects. The rationale is that the propensity scores eliminate bias from confounders that are measured or proxied for with the observable variables used in the first stage probits, but there can remain other unobservable confounders, and the fixed-effects additionally control for unobservables that are invariant at the mother (or the mother's household) level. While we are aware that it is fairly unconventional to use propensity score regressions in conjunction with fixed-effects, we believe that it serves as a good validity test of the results.

In the first set of results, which are inclusive of propensity scores but exclusive of any fixed-effects, we find that prenatal drinking is significantly correlated with increases in aggregate BPI scores, by 1.58 units and 1.30 units for the younger and older group respectively. It is also significantly correlated with increases in almost all the behavior subscores. In contrast, prenatal smoking is not statistically correlated with behavior problems for the older group. For the younger group, it is weakly correlated with increases in the aggregate BPI score, and significantly correlated with three out of the six sub-scores. When the models are reestimated with maternal fixed-effects or with maternal household fixed-effects, prenatal drinking continues to be very significantly correlated with behavior problems, both in case of the aggregate BPI score and the sub-scores, for the younger and the older children. Prenatal smoking, on the other hand, has no significant associations with increases in either the aggregate BPI score or with the sub-scores in most cases. Indeed, in some of the models the coefficient estimate of prenatal smoking takes a counterintuitive negative sign, but they are so statistically imprecise that the negative sign

^{*} As Freeman³⁴ explains, the effects of a modest number of measurement error are accentuated in panel data analysis using fixed effects primarily because fixed effects methods rely on a relatively small number of 'changers' of the key explanatory variable (in this case, substance use) to obtain an estimated effect of that explanatory variable. Hence, the proportion of 'effective' observations that are erroneous will potentially be larger in a fixed-effects model than a cross-sectional analysis, and will produce a larger bias. On the other hand, if there are a very large number of measurement errors in the key explanatory variables, then the cross-sectional model will produce a larger bias than the fixed-effects model. Readers are also referred to the above paper for a simple but illuminating mathematical exposition of the effects of measurement error on estimation bias in cross-sectional versus longitudinal fixed effects models.

Table 6. First Stage Probits for Propensity For Maternal Prenatal Smoking & Maternal Prenatal Drinking

	Prenata	al drink	Prenata	l smoke
	Coefficient	(t-statistic)	Coefficient	(t-statistic)
Child was breast-fed	0.10***	(2.70)	-0.14***	(-3.16)
Breast fed information missing	-0.20**	(-2.00)	-0.25**	(-2.04)
# of Prenatal Visits	-0.14	(-0.87)	-0.14	(-0.76)
Missing prenatal info	-2.51***	(-6.63)	-3.03***	(-8.42)
Mother's Highest Grade Completed	0.02***	(2.64)	-0.07***	(-7.04)
Father in household	-0.15^{***}	(-3.63)	-0.18***	(-3.77)
Household in poverty	-0.10**	(-2.11)	0.05	(0.93)
Household poverty status unknown	-0.08*	(-1.67)	-0.07	(-1.18)
Mother Currently Employed	0.00	(-0.07)	-0.03	(-0.63)
Rural Residence	-0.22^{***}	(-5.07)	0.03	(0.56)
City Residence	-0.11^{**}	(-2.22)	0.10*	(1.70)
Child's mother black	-0.30***	(-6.26)	-0.46^{***}	(-8.12)
Child's mother of Hispanic origin	-0.37***	(-4.91)	-0.67***	(-7.34)
Mother's age at birth	0.06	(1.53)	0.05	(1.01)
Worked in year 1 after child's birth	0.03	(0.74)	-0.08*	(-1.71)
Child's first home environment score	0.00	(-1.57)	0.00**	(-2.19)
Foreign language at mother's home	0.55*	(1.73)	0.06	(0.15)
Mother lived in intact family	-0.17	(-0.73)	-0.24	(-0.89)
At least one foreign-born parent	-0.09	(-0.22)	0.57	(1.15)
Mother's mother worked for pay	0.07	(0.33)	-0.11	(-0.42)
Newspapers in (mother's) home	0.64**	(2.39)	-0.18	(-0.61)
Library card in (mother's) home	-0.13	(-0.48)	0.06	(0.22)
Self-esteem score	0.01	(0.50)	0.02	(0.58)
CESD Score	-0.01	(-1.23)	-0.03**	(-2.39)
Number of children considered ideal by mother	0.11	(1.26)	-0.03	(-0.27)
Mother has alcoholic relative	0.59**	(2.55)	0.65**	(2.47)
Information on alcoholic relative missing	0.39	(0.54)	0.34	(0.43)
Mother reports no church attendance in 1979	-0.25	(-0.74)	-0.16	(-0.43)
Mother reports infrequent church attendance in 1979	-0.43*	(-1.66)	0.33	(1.08)
Mother reports more than weekly church attendance in 1979	0.05	(0.14)	-0.62	(-1.35)
(Foreign language at mother's home) x mother's age	-0.02*	(-1.71)	0.00	(-0.23)
(Mother lived in intact family) x mother's age	0.00	(0.21)	0.00	(0.46)
(At least one foreign-born parent) x mother's age	0.00	(0.27)	-0.02	(-1.25)
(Mother's mother worked for pay) x mother's age	0.00	(-0.22)	0.02	(0.79)
(Newspapers in (mother's) home) x mother's age	-0.02*	(-1.76)	0.01	(0.94)
(Library card in (mother's) home) x mother's age	0.01	(0.82)	0.00	(-0.18)
(Self–esteem score) x mother's age	0.00	(-0.12)	0.00	(-0.96)
(CESD Score) x mother's age	0.00	(1.43)	0.00**	(2.54)
(Number of children considered ideal) x mother's age	0.00	(-1.31)	0.00	(0.45)
(Mother has alcoholic relative) x mother's age	-0.01	(-1.61)	-0.02**	(-2.05)
(Info on alcoholic relative missing) x mother's age	-0.01	(-0.58)	-0.02 -0.01	(-2.03) (-0.17)
(No church attendance) x mother's age	0.01	(-0.58) (0.58)	0.02	(-0.17) (1.13)
(Infrequent church attendance) x mother's age	0.01	(0.38)	-0.02	(-0.69)
(More than weekly church attendance) x mother's age	-0.01	(-0.74)	0.02	(-0.09) (1.07)
Smoked after child's birth	-0.01 0.14***	(-0.74) (3.33)	1.73***	(38.41)
Binge drink after child's birth	0.63***	(15.65)	0.22***	(4.84)

Notes: Also includes a vector of dummies for the child's birth-year.

Table 7. Comparison of Mean BPI Score & Behavioral Sub-Scores	s Using Propensity Score Matching
---	-----------------------------------

	Younger Children			Older Children		
	Treated Group	Matched Group	T-stat for equality of means	Treated Group	Matched Group	T-stat for equality of means
Prenatal Maternal Smoking						
BPI Standardized Score	107.35	105.45	2.56**	108.25	107.95	0.31
Antisocial Standardized Score	107.38	106.34	1.48	109.12	108.00	1.21
Anxious/Depressed Standardized Score	103.54	102.87	1.03	105.20	104.50	0.82
Headstrong Standardized Score	103.69	102.49	1.89*	105.27	104.71	0.66
Hyperactive Standardized Score	108.19	106.10	2.94 ***	105.71	105.71	0.01
Dependent Standardized Score	107.72	106.58	1.67*	106.32	107.44	-1.30
Peer Conflict Standardized Score	106.61	105.70	1.44	105.84	105.03	0.99
Prenatal Maternal Drinking						
BPI Standardized Score	105.51	103.89	3.23 ***	107.95	106.16	2.84 ***
Antisocial Standardized Score	106.08	104.50	3.24 ***	108.34	106.85	2.45 **
Anxious/Depressed Standardized Score	102.97	101.97	2.24 **	105.15	103.74	2.48 **
Headstrong Standardized Score	102.95	101.20	4.11 ***	105.01	103.83	2.15 **
Hyperactive Standardized Score	106.14	105.21	1.90 *	104.86	103.56	2.14 **
Dependent Standardized Score	106.60	106.12	1.04	106.57	105.81	1.34
Peer Conflict Standardized Score	105.40	104.38	2.38 **	105.60	104.09	2.78 ***

Notes: Propensity scores for exposure to prenatal smoking and drinking are obtained from first stage probits shown in **Table 6**. The 'control groups' are selected using radius matching within caliper distance of 0.0005.

probably does not warrant much concern.

Discussion & Conclusions

Our aim in this study has been to revisit the relationship between maternal prenatal cigarette-use and alcohol-use and children's behavior problems, and to investigate how the results change after attempts are made to control for underlying confounding factors. Because the instrumental variables method performs poorly in our study, we rely on fixed-effects and propensity score matching methods to minimize the bias from confounders. We acknowledge that neither of these methods fully addresses the problem of *timevariant* unobservable confounders. Thus, while we believe that we have substantially reduced potential bias, we probably have not eliminated it completely. Hence, caution should be exercised when interpreting the results from the point of view of establishing causality.

In summary, we find that strong associative results exists between both types of prenatal substance use and behavior problems in 'naïve' models that control for limited socioeconomic and demographic characteristics. When we use fixed-effects, propensity-score matching and propensityscore inclusive regressions, prenatal alcohol use continues to be significant related with increases in behavior problems, but prenatal smoking by and large ceases to have any significant effects. Thus, it appears that the associations that extant literature finds between maternal prenatal smoking and the subsequent behavior problems in children may be spurious, driven by underlying confounding factors like other maternal and familial characteristics. On the other hand, prenatal drinking may have true physiological/biological effects on the fetus that eventually exacerbate behavior problems.

We acknowledge certain potential shortcomings of the study that will be difficult to remedy. As in case of all selfreported survey data, there are concerns about the accuracy of reporting about prenatal substance use, and hence the possibility that there are measurement errors in our key explanatory variables. To the extent that this measurement error is random and present in a relatively small number of observations, it will induce a larger bias in the fixed-effects estimates compared to the OLS estimates, though the direction of the bias is uncertain. Another concern is that it is debatable whether mothers are necessarily the most objective reporters of children's behavior problems. There is some evidence that mothers with substance use problems are more punitive towards their children than non-substance-using contemporaries, therefore, there is the possibility that mothers who used substances while pregnant may be more dissatisfied with their children and more liable to exaggerate behavior problems. On the other hand, it may be that mothers who are prone to substance use may have lower standards of 'good' behavior in children, and may underestimate behavior problems. Finally, there is no information available in this dataset regarding the smoking habits of the mother's spouse/

	No Fixe	d-effects	Maternal Fixed-effects			Household effects
	Younger Children	Older Children	Younger Children	Older Children	Younger Children	Older Children
OVERALL BPI SCORE						
Prenatal Smoke	1.51*	0.70	-0.28	-1.03	-0.39	0.29
	(1.75)	(1.04)	(-0.24)	(-0.94)	(-0.50)	(0.28)
Prenatal Drink	1.58***	1.30**	1.19**	1.67**	1.33**	2.27**
	(3.56)	(2.54)	(2.12)	(2.35)	(2.49)	(2.38)
ANTISOCIAL SCORE						
Prenatal Smoke	1.21**	1.19*	-0.05	-2.02*	0.52	-0.68
	(2.33)	(1.86)	(-0.06)	(-1.76)	(0.64)	(-0.65)
Prenatal Drink	1.52***	0.83*	1.45**	0.05	1.26**	1.11
	(3.73)	(1.71)	(2.50)	(0.08)	(2.30)	(1.59)
ANXIOUS/DEPRESSED SCORE						
Prenatal Smoke	0.66	0.58	-1.25	-0.85	-1.41	-0.45
	(1.37)	(0.97)	(-1.56)	(-0.79)	(-1.08)	(-0.46)
Prenatal Drink	1.04***	1.36***	0.71*	1.54**	0.88*	2.37***
	(2.75)	(2.95)	(1.73)	(2.20)	(1.75)	(3.60)
HEADSTRONG SCORE						
Prenatal Smoke	0.71	0.48	-0.72	0.19	-1.27*	0.79
	(1.53)	(0.81)	(-0.91)	(0.19)	(-1.76)	(0.83)
Prenatal Drink	1.88***	0.95**	0.86*	0.55	1.32***	1.20*
	(5.12)	(2.07)	(1.65)	(0.88)	(2.71)	(1.89)
HYPERACTIVE SCORE						
Prenatal Smoke	1.80**	0.78	-1.34	1.70	-1.10	1.97*
	(2.05)	(1.20)	(-1.51)	(1.46)	(-1.36)	(1.85)
Prenatal Drink	0.73*	1.03**	1.31**	1.87**	1.67***	2.51***
	(1.78)	(2.09)	(2.24)	(2.49)	(3.02)	(3.51)
DEPENDENT SCORE	. ,					· · · ·
Prenatal Smoke	0.71	-0.64	1.51*	-1.79	0.40	-0.59
	(1.40)	(-1.06)	(1.71)	(-1.61)	(0.60)	(-0.60)
Prenatal Drink	0.48	0.73	0.08	1.64**	0.10	2.31***
	(1.21)	(1.59)	(0.04)	(2.31)	(0.20)	(3.50)
PEER CONFLICT SCORE						
Prenatal Smoke	0.97**	0.66	-0.65	1.21	0.61	0.77
	(1.89)	(1.17)	(-0.75)	(1.14)	(0.79)	(0.80)
Prenatal Drink	0.99***	1.11**	1.17**	1.52***	1.00*	1.87***
	(2.75)	(2.54)	(2.08)	(2.71)	(1.92)	(2.88)

Table 8. Regression Results for BPI Score & Behavior Sub-Scores with Propensity Score Included, With and Without Fixed-effects

* indicates significance at 90%

** indicates significance at 95%

*** indicates significance at 99%

Notes: All equations include propensity scores for exposure to prenatal smoking and drinking obtained from first stage probits (**Table 6**), the control variables from **Table 3**, and survey-year dummies.

partner during pregnancy, or of the smoking and drinking habits of the spouse/partner after the birth of the child, and this is potentially a source of omitted variable bias.

In conclusion, while there continues to be a need for further research into the relationship between prenatal substance use and children's future health, including mental health and behavior problems, we tentatively argue based on our findings that efforts to reduce prenatal drinking can yield 202 benefits in terms of reducing children's future behavior problems. On the other hand, while reducing prenatal smoking is an important public health endeavor that can reduce the incidence of low and very-low birthweight and all the developmental problems correlated with that, it may not necessarily yield benefits in terms of reducing children's behavior problems.

Acknowledgments

We are grateful to Sara Markowitz, to participants of the AEA/ASSA 2005 conference and IHEA 2005 conference for many helpful comments.

References

- Patterson GR, DeBaryshe BD, Ramsey E. A developmental perspective on antisocial behavior. *Am Psychol* 1989; 44: 329-335.
- 2. Centers for Disease Control (CDC). Surgeon general's reports related to tobacco. Various Years.
- 3. Oster G, Delea TE, Colditz GA. Maternal smoking during pregnancy and expenditures on neonatal health care. *Am J Prev Med* 1988; **4:** 216-219
- 4. Paneth NS. The problem of low birth weight. *Future Child* 1995; 5: 19-34.5. Barker DJP. *Mothers, Babies and Health in Later Life, Second Edition.*
- Edinburgh and London: Churchill Livingstone. 1998. 6. McCormick MC. The outcomes of very low birth weight infants: Are we
- asking the right questions? *Pediatrics* 1997; **99**: 869-76.
 7. Fergusson DM, Horwood LJ, Lynskey MT. Maternal smoking before and after pregnancy, effects on behavioral outcomes in middle childhood. *Pediatrics* 1993; **92**: 815-822.
- Weitzman M, Gortmaker S, Sobol A. Maternal smoking and behavior problems of children. *Pediatrics* 1992; 90: 342-349.
- Roebuck TM, Mattson SN, Riley EP. (1999). Behavioral and psychosocial profiles of alcohol-exposed children. *Alcohol Clin Exp Res* 1999; 23: 1070-1076.
- 10. Nichols PL, Chen TC. *Minimum Brain Dysfunction: A Prospective Study*. Hillsdale, NJ: Erlbaum. 1991
- Makin J, Fried PA, Watkinson B. A comparison of active and passive smoking during pregnancy: Long term effects. *Neurotoxicol Teratol* 1991; 13: 5-12.
- Wakschlag L, Lahey B, Loeber R, Green S, Gordon R, Leventhal B. Maternal smoking during pregnancy and the risk of conduct disorder in boys. *Arch Gen Psychiatry* 1997; 54: 670-676
- Wakschlag L, Leventhal B, Pine D, Pickett K, Carter A. Elucidating early mechanisms of developmental psychopathology: the case of prenatal smoking and disruptive behavior. *Child Dev* 2006; 77: 893-906
- Weinberg NZ. Cognitive and behavioral deficits correlated with parental alcohol use. J Am Acad Child Psy 1997; 36: 1177-1186.
- Sood B, Delaney-Black V, Covington C, Nordstrom-Klee B, Ager J, Templin T, Janisse J, Martier S, Sokol RJ. Prenatal alcohol exposure and childhood behavior at age 6 to 7 years: I. dose-response effect. *Pediatrics* 2001; **108** (electronic article: http://pediatrics.aappublications.org/cgi/ content/abstract/108/2/e34)
- 16. Maughan B, Taylor A, Caspi A, Moffitt T. Prenatal smoking and early

childhood conduct problems: testing genetic and environmental explanations of the association. *Arch Gen Psychiat* 2004; **61**: 836-843

- Martin RP, Dombrowski SC, Mullis C, Wisenbaker J, Huttunen MO. Smoking during pregnancy: Association with childhood temperament, behavior, and academic performance. *J Pediat Psychol* 2006; **31**: 490-500.
- Rosenzweig MR, Schultz TP. Estimating a Household Production Function: Heterogeneity, the demand for health inputs, and their effects on birth weight. *J Polit Econ* 1983; **91:** 723-746.
- 19. Evans NW, Ringel SJ. Can higher cigarette taxes improve birth outcomes? *J Public Econ* 1999; **72**: 135-154.
- Chatterji P, Markowitz S. The impact of maternal alcohol and illicit drug use on children's behavior problems: evidence from the children of the NLSY. *J Health Econ* 2001; 20: 703-731.
- Zill N, Peterson JL. Marital disruption, parent-child relationships, and behavior problems in children." J Marriage Fam 1986; 48: 295-307.
- Achenbach TM, Edelbrook C. Manual for Child Behavior Checklist and Revised Child Behavior Profile. Burlington, VT: Queen City Printers. 1983.
- 23. Geronimus AT, Korenman S. The socioeconomic consequences of teen childbearing reconsidered. *Q J Econ* 1992; **107**: 1187-1214.
- 24. Geronimus AT, Korenman S. Maternal youth or family background? On the health disadvantages of infants with teenage mothers. *Am J Epidemiol* 1993; **137**: 213-225.
- Currie J, Cole N. Welfare and Child Health: The link between AFDC participation and birth weight. *Am Econ Rev* 1993; 83: 971-985.
- 26. Rosenzweig MR, Wolpin KI. Are there increasing returns to the intergenerational production of human capital? Maternal schooling and child intellectual achievement. *J Hum Resour* 1994; **29:** 670-693.
- D'agostino RB. Tutorial in biostatistics: Propensity score methods for bias reduction in comparison of a treatment to a non-randomized control group. *Stat Med* 1998; 17: 2265-2281.
- Rosenbaum PR, Rubin DB. Reducing bias in observational studies using subclassification on propensity scores. *J Am Stat Assoc* 1984; 79: 516-524.
- Rashad I, Kaestner R. 2004. Teenage Sex, Drugs and Alcohol: Problems Identifying the Cause of Risky Behaviors? JHealth Econ 2004; 22: 225-248.
- 30. Kinal TW. The Existence of Moments of k-Class Estimators. *Econometrica* 1980; **48:** 241-49.
- Davidson R, MacKinnon JG. Estimation and Inference in Econometrics. New York and Oxford: University Press. 1993.
- 32. Deb P, Lakdawalla D, Sood N. Weak instruments and binary endogenous variables. Presentation at the 1st conference of the American Society of Health Economists, Madison, WI, June 2006.
- Bound J, Brown C, Mathiowetz N. 2001. Measurement error in survey data. In JJ Heckman, EE Learner (Eds) *Handbook of Econometrics*. Amsterdam, Netherlands: Elsevier Science B.V., 3705-3843.
- Freeman RB. Longitudinal analyses of the effects of trade unions. J Labor Econ 1984; 2: 1-26.

Appendix

Table A1. Specific Items Included in Each Behavior Problem Sub-Scale
--

<u>^</u>	
Antisocial	Cheats or lies Bullies/is mean and cruel to others Does not feel sorry for misbehaving Breaks things deliberately Disobedient in School Trouble with getting along with teachers
Anxious/Depressed	Sudden changes in moods and feelings Feels/complains about being unloved Overly fearful and anxious Feels worthless and inferior Unhappy, sad, depressed.
Dependent	Clings to adults Cries too much Demands a lot of attention Too dependent on others
Headstrong	High strung, tense, nervous Argues too much Disobedient at home Stubborn, sullen, irritable Loses temper easily
Hyperactive	Difficulty concentrating/paying attention Easily confused Impulsive – acts without thinking Trouble with obsessions Restless, overly active.
Withdrawn/ Peer conflicts*	Trouble getting along with others Not liked by other children Withdrawn, not involved

* In 1998, additional questions were added to the peer conflict scale, including 'feels suspicious of others', 'hangs around with kids who get into trouble', 'is secretive' and 'worries too much.'

Table A2. Distribution of Child Observation Frequencies for Mothers and Maternal-Households

	Number of Observations
Child Observations for Same Mother	
1	1624
2	2250
3	1119
4	312
5	65
6	18
>6	7
Child Observations for Same Mother's	
Original household	
1	1270
2	1804
3	1131
4	588
5	330
6	162
>6	110

Table A3. First Stage Results f	or IV regressions	(Effects of Instruments	s Shown Only)

	Younger Children		Older Children	
	Prenatal Smoke coefficient t-stat	Prenatal Drink coefficient t-stat	Prenatal Smoke coefficient t-stat	Prenatal Drink coefficient t-stat
Inflation Adjusted Beer Tax	-0.066***	-0.067***	-0.044	-0.05
	-2.88	-2.73	-1.48	-1.59
Per Capita Alcohol Retail Outlets	0.017*	0.061***	0.037***	0.058***
-	1.71	5.82	2.77	4.14
BAC 0.08	0.002	-0.024	0.05	0.167
	0.06	-0.73	0.39	1.23
Inflation Adjusted Cigarette Tax	-0.001	0.002***	-0.001	0.003***
	-1.28	3.03	-0.79	3.19
Per Capita Acres of Tobacco Farming	8.36E-06	5.14E-06	0.0001**	0.00001*
	1.13	0.65	2.38	1.74
F-stat for joint significance of instruments	3.31	18.62	2.52	13.64

MATERNAL PRENATAL SUBSTANCE USE AND BEHAVIOR PROBLEMS AMONG CHILDREN IN THE U.S.

205

Table A4. Instrumental Variables Regression Results

	Younger Children	Older Children
	coefficient (t-statistic)	coefficient (t-statistic)
Overall BPI Score		
Prenatal Smoke	6.754	0.298
	(0.63)	(0.03)
Prenatal Drink	-2.722	-5.513
	(-0.64)	(-0.99)
Antisocial Score		()
Prenatal Smoke	3.03	13.112
	(0.30)	(1.31)
Prenatal Drink	4.043	-6.712
	(0.99)	(-1.20)
Anxious/Depressed Score	× ,	
Prenatal Smoke	-10.193	-0.532
	(-1.00)	(-0.06)
Prenatal Drink	-0.391	0.905
	(-0.10)	(0.19)
Headstrong Score		
Prenatal Smoke	5.905	0.072
	(0.63)	(0.01)
Prenatal Drink	-1.954	-6.069
	(-0.53)	(-1.26)
Hyperactive Score		Ì,
Prenatal Smoke	10.583	-4.858
	(0.98)	(-0.51)
Prenatal Drink	-8.919**	0.51
	(-2.06)	(0.10)
Dependent Score		
Prenatal Smoke	-0.961	-1.724
	(-0.10)	(-0.19)
Prenatal Drink	-2.004	-0.800
	(-0.50)	(-1.11)
Peer Conflict Score		. ,
Prenatal Smoke	8.588	-11.945
	(0.94)	(-1.30)
Prenatal Drink	-2.934	-2.22
	(-0.79)	(-0.44)

* indicates significance at 90% ** indicates significance at 95% *** indicates significance at 99%

206