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Prenatal Smoking Cessation and Infant Health: Evidence from Sibling Births

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This article uses a large data set of sibling births to examine when mothers must quit smoking in pregnancy to deliver healthy babies. It applies sibling fixed effects models to provide robust evidence that smoking cessation in the first trimester has a negligible effect on infant health, but cessation as late as second trimester or smoking throughout pregnancy is associated with substantially lower birth weights and higher risks of delivering low birth weight babies. In particular, about two thirds of the total detrimental smoking impact on birth outcomes occurs in the second trimester. Therefore, reallocating resources on prenatal smoking cessation towards the first trimester can lead to a significant efficiency gain. This study also shows when the timing information of prenatal smoking cessation is improperly used, it will introduce a new nontrivial downward bias in estimating the causality between the conventionally used group measure "prenatal smoker" and infant health.

JEL Classification: D13, I12, I18, J13

1. Introduction

It has been well established that smoking during pregnancy can substantially reduce infant birth weights, increase the risks of delivering low birth weight (LBW = infant birth weight less than 2500 g) babies, and cause a host of other adverse birth outcomes (Center for Chronic Disease Prevention and Health Promotion 2001). Adams et al. (2002) reports that the short-term neonatal costs attributable to prenatal smoking is over $700 per pregnant smoker (in 1996 dollars). More importantly, poor infant health due to prenatal smoking can result in childhood developmental problems, low educational attainment in adolescence (Lewit et al. 1995; Corman and Chaikind 1998), and subsequent adverse labor market outcomes in adulthood (Case, Fertig, and Paxson 2005). Because of the large intergenerational health and economic costs of prenatal smoking, promoting smoking cessation among pregnant women is a current focus of policy makers and medical practitioners. However, one important question on such cessation

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interventions remains unsettled: At which stage during pregnancy must mothers quit smoking such that fetal exposure to smoking only does negligible harm to infant health? The literature suggests prenatal smoking starts to have a significantly adverse impact on infant physical health either at the beginning of the second or the third trimester. Hence, the key question is whether smoking cessation as late as the second trimester makes the birth outcomes of late quitters much worse than those of nonsmokers.[1] If so, the first trimester is the critical time which any prenatal smoking cessation treatment must concentrate on (early cessation). If not, late cessation should be underscored, where an increasingly extensive cessation counseling throughout the second trimester can achieve a high quit rate. Such a prolonged intervention can be more cost effective, given that heavy smokers often need more time to quit in pregnancy.

Past studies on the timing of smoking cessation and infant health, especially related to birth weight and LBW,[2] nonetheless rarely take into account the endogeneity of smoking cessation at different pregnancy stages. An immediate concern is whether unobserved mother heterogeneity can drive the previous controversial findings. If late quitters, instead of early quitters, are on average more likely than nonsmokers to heavily engage in other unobserved risky health behaviors, then the estimated negative impact of late cessation can be substantially biased upward (Abrevaya 2006). This result makes the first trimester appear to be the critical period for prenatal smoking cessation. In contrast, disadvantaged smokers can compensate for the intergenerational transmission of their low health endowments (Rosenzweig and Wolpin 1991, 1995). They may quit early in pregnancy, or they may mitigate the adverse impact of delayed smoking cessation by investing in other health-augmenting inputs. Consequently, the negative effect of late cessation is understated towards zero. Then an opposite conclusion is reached: Mothers can quit smoking as late as the second trimester to nullify the negative smoking effect on newborn health. This article makes a first attempt to address these potential biases, controlling for the common maternal influence on both smoking cessation and infant health.

Understanding the relationship between prenatal smoking cessation and infant health is crucial for improving the cost effectiveness of any cessation program for pregnant smokers. However, the literature has provided little evidence to determine the optimal resource allocation strategies in this arena (Ruger and Emmons 2008). The present research fills in this gap by showing early rather than late cessation is absolutely necessary for smoking mothers to deliver infants as healthy as those of nonsmokers. This finding suggests a key component of the optimal strategies is reallocating prenatal smoking cessation resources towards the first trimester, which can lead to a significant efficiency gain. This article also highlights the importance of separating first- from second-trimester smoking cessation (and smoking throughout pregnancy) in defining a frequently used group measure of "prenatal smoker." Past studies on infant production function often consider prenatal smoking at any pregnancy stage as a key input, while ignoring the timing issue of prenatal smoking cessation (Rosenzweig

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[1] In this study, mothers who quit smoking prior to pregnancy, in the first trimester, in the second trimester, or who smoke throughout pregnancy are called "preconception quitters," "early quitters," "late quitters," and "continuous smokers," respectively.

[2] Birth weight and LBW are the two most widely studied measures, which this study also focuses on. Other infant health measures used in this literature include preterm birth (the birth of a baby of less than 37 weeks gestational age), perinatal mortality, head circumference, crown-heel length, and ponderal index (Rush and Cassano 1983; McDonald, Armstrong, and Sloan 1992; Lindley et al. 2000; McCowan et al. 2009). For future research, it is interesting to apply sibling models of this article to examine the relationship between smoking cessation and those alternative measures.

and Schultz 1983; Corman, Joyce, and Grossman 1987; Noonan et al. 2007; Reichman et al. 2009). However, this research shows early quitters give birth to babies as healthy as nonsmokers, yet late quitters do not. This then suggests that a well-defined group variable "prenatal smoker" should sensibly include only mothers who smoke beyond the first trimester.

This article uses a large data set of sibling births in the states of Pennsylvania and Washington to study the relationship between prenatal smoking cessation and infant health. While existing public health and epidemiological studies often use convenience samples that are selective or lack important socioeconomic variables, the sample used in this research is superior. Drawn from the universal births of the two states, it is very large with a rich set of infant, mother, and family level characteristics. Its panel structure can be used to handle the endogeneity of prenatal smoking cessation in sibling fixed effects models. This method is more feasible than an instrumental variable approach, since it is extremely hard to find multiple healthy policies as instruments for four endogenous smoking cessation statuses (cessation just before pregnancy, in the first/second trimester, and smoking throughout). Regressions using sibling fixed effects models also show that when the timing information of prenatal smoking cessation is improperly used, it will introduce a new nontrivial downward bias in estimating the causality between the conventionally used group measure "prenatal smoker" and infant health. In contrast, the existing studies concentrate on two other measurement error problems of prenatal smoking: change in the average intake of harmful substances per cigarette (Fertig 2010) and misclassification of smokers as nonsmokers (Brachet 2008).

2. Literature

The relationship between the timing of prenatal smoking cessation and infant health has been a key issue in many public health and epidemiological studies. However, so far, the evidence that they provide is quite mixed. The controversy centers upon whether prenatal smoking starts to significantly impair infant physical health at the beginning of the second or the third trimester. So the crucial question is whether late cessation in the second trimester is associated with substantially lower infant birth weights and higher risks of delivering LBW babies. Some studies report optimistic findings that the prenatal smoking cessation deadline can be so late as the first month of the third trimester (Rush and Cassano 1983; Lindley et al. 2000). Lieberman et al. (1994) shows that if mothers quit smoking by the end of the second trimester, the risks of delivering undersized infants are equal to those of nonsmokers. However, Macarthur and Knox (1988) finds smoking cessation after the 16th week (the beginning of the second trimester) only mitigates the adverse smoking impact on newborn babies. Quite a few recent studies further indicate it is necessary for smoking pregnant women to quit before the second trimester to make their infants as healthy as those of the nonsmokers (McDonald, Armstrong, and Sloan 1992; McCowan et al. 2009). There are two salient limitations in those studies. First, the samples they use are cross sectional, usually highly selective, and lack important characteristics of mothers and their families. Second, they only present a variety of associations between smoking cessation and infant health, without addressing any potential bias due to unmeasured mother heterogeneity. So far, no controlled epidemiological experiment has been reported on the timing of smoking cessation and infant health, mainly because intervening and tracking pregnant smokers at different pregnancy stages is very difficult in clinical trials (England et al. 2001). Randomized

trials are instead generally applied to evaluate the efficacy of a cessation counseling or compare different types of counselings (Floyd et al. 1993; Kelley, Bond, and Abraham 2001).

This study is also closely related to another body of literature which examines the causal effect of prenatal smoking on infant health with the following three empirical strategies. The first approach is ordinary least square (OLS) or matching, given that a very rich set of covariates is available such that the typically unobservable factors correlated with both prenatal smoking and infant health can be controlled for (Almond, Chay, and Lee 2005; Reichman et al. 2009). The second is to find an instrumental variable for prenatal smoking, such as state cigarette tax rates (Evans and Ringel 1999) and state cigarette tax hikes (Lien and Evans 2005).[3] Instrumental variable estimates are usually larger than those of OLS, because mothers who quit smoking due to these instruments are positively selected and thus more able to handle the adverse smoking effect on birth outcomes (Evans and Ringel 1999). The third approach is applying fixed effects models into panel data sets. Two studies by Rosenzweig and Wolpin (1991, 1995) address the relationship between prenatal smoking and infant health with the National Longitudinal Survey of Youth 1979. Abrevaya (2006) recently studies this issue using a large data set of matched sibling births.[4] In addition, Walker, Tekin, and Wallace (2009) applies fixed effects models to a sample of teenager mothers and their infants. Sibling fixed effects estimates of prenatal smoking are generally smaller than those of the other two strategies. Similar findings are shown in Abrevaya and Dahl (2008), which uses an alternative panel quantile estimation approach. The present study also uses sibling models, because the OLS method relies on a very strong identification assumption of selection on observables, and finding instruments for multiple smoking cessation statuses is extremely difficult. Finally, this literature pays no attention to the timing issue of smoking cessation in pregnancy, thereby often using an ill-defined group measure of "prenatal smoker." An important concern is then raised on whether the corresponding estimated impact of prenatal smoking is biased.

This article merges and advances the previous two lines of research. It presents new evidence of the critical period in pregnancy when mothers must quit smoking to deliver healthy babies, using a large data set of sibling births. It also carefully addresses what kinds of biases in the smoking cessation estimates are driven by the mother level unmeasured heterogeneity, when the sibling fixed effects are not controlled for. Moreover, it examines the biases in estimating the causality between the conventionally used group variable of "prenatal smoker" and infant health, when early quitters are incorrectly regarded as prenatal smokers or late quitters are regarded as prenatal nonsmokers.

3. Data and Empirical Methods

This study uses a Washington state longitudinal database of births and a Pennsylvania state matched panel of sibling births. Both data sets are constructed from the universal births in

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[3] In addition, this method can be readily applied with randomized cessation trials (Sexton and Hebel 1984; Floyd et al. 1993; Kelley, Bond, and Abraham 2001). As mentioned already, studies using controlled experiments focus primarily on the overall quit rate, leaving unaddressed the timing issue of smoking cessation.

[4] The sibling births in Abrevaya (2006) are not very precisely matched, lacking important matching identifiers such as mother's name and mother's date of birth. However, it uses a proxy to gauge the degree of correct matching and reports estimates with more precisely matched birth pairs. Such estimates are compared with the results of this article in the results section.

the two states, where sibling births are matched by mother's name, mother's date of birth, mother's race, parity, etc. The detailed information of every newborn in this data set is originally collected from the infant's birth certificate. A birth certificate filing form is usually jointly completed by each mother and her health care provider. The responsibility of each mother is to provide information on legal facts, family demographics, marital status, pregnancy history, prenatal smoking, and prenatal care utilization. Her health care provider uses hospital records to complete the newborn's statistical information (birth weight, gestation, plurality, etc.) as well as maternal medical and health information (medical risk conditions, maternal morbidity, method of delivery, etc.) in the filing form. In this study, the sample is restricted to mothers with singleton sibling births, where women having multiple-birth pregnancies (twins, triplets, etc.) are dropped. Since very few women delivered more than three sibling births in a short four-year sample period, they are also excluded.

The following baseline analysis focuses on a sample period between 2003 and 2006 for two reasons. First, Washington and Pennsylvania took the first initiative to code smoking cessation at different pregnancy stages in 2003, while a few other states with small population of pregnant women followed them later on. Second, when this research was launched in 2009, the latest data that can be released was collected in 2006 in each state. In some important robustness checks, an expanded sibling sample is used that contains the infants born in 2003–2006 in Washington and those in 2003–2010 in Pennsylvania. The restricted 2006–2010 Pennsylvania sibling birth data were released recently in 2012, but no such data were available for Washington. Overall, this data set of sibling births is suitable to address the present research question. Its panel structure can be used to handle unobserved mother heterogeneity. It also has a rich set of infant, mother, and family characteristics. Finally, there are many childbearing women in both states. Many of them smoked during pregnancy in the sample period.

The data set codes each mother's number of cigarettes smoked per day at three months before pregnancy and in each trimester. The response rates to all four questions on the timing of smoking in pregnancy are very high, about 98% in both states. Appendix 1 shows most smoking mothers tend to smoke continuously through different pregnancy stages. Therefore, the intermittent smokers, who account for less than 1% of all the mothers (or less than 5% of all the smokers), are excluded from the baseline analysis. Definition 1 of smokers only considers mothers who smoked continuously until a certain pregnancy stage, where the baseline group consists of those who did not report smoking in any stage prior to or during pregnancy. In this definition, four smoking categories, which correspond with the aforementioned four types of smokers, are "cessation before pregnancy" (SK_0), "cessation in the first trimester" (SK_1), "cessation in the second trimester" (SK_2), and "smoking throughout" (SK_3). Additional analysis is conducted on the four more broadly defined smokers, where mothers smoking intermittently or continuously until the same pregnancy stage are grouped together (Definition 2, Appendix 1). In addition, a small number of infants with gestational ages less than 30 weeks (0.7% of all the infants) are excluded from the baseline analysis for two reasons. First, this sample restriction makes late quitters' duration of fetal exposure to smoking at least three weeks shorter than continuous smokers. As a result, a clear distinction between such two types of smokers is drawn. Second, it also addresses a concern of reverse causality. This concern is raised because some late quitters may actually quit smoking at the end of the second trimester (28th to 29th week) right after they deliver unhealthy infants. The next section relaxes this restriction so that only the newborn babies with gestational ages less than 28 weeks are dropped, as well as discusses any potential bias due to gestational age restrictions.

Table 1. Descriptive Statistics of the Dependent Variables and Key Covariates

	Mothers in Washington				Mothers in Pennsylvania			
	Two births in 2003–2006		Three births in 2003–2006		Two births in 2003–2006		Three births in 2003–2006	
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Birth weight (g)	3448.021	510.252	3424.959	538.367	3393.420	523.556	3370.182	531.685
Low birth weight	0.034	0.182	0.043	0.203	0.046	0.209	0.049	0.215
Gestation (weeks)	38.903	1.508	38.775	1.650	38.854	1.577	38.840	1.651
Cessation before pregnancy	0.017	0.131	0.014	0.115	0.047	0.211	0.032	0.176
Cessation in the first trimester	0.010	0.100	0.011	0.105	0.021	0.144	0.017	0.129
Cessation in the second trimester	0.004	0.061	0.006	0.075	0.008	0.091	0.008	0.088
Smoke throughout	0.084	0.277	0.131	0.338	0.137	0.344	0.183	0.387
Infant male	0.515	0.500	0.512	0.500	0.513	0.500	0.511	0.500
Parity	1.265	1.356	2.084	1.934	1.290	1.427	2.051	1.786
Mother's age	27.492	5.690	25.655	5.428	27.403	5.576	25.607	5.183
Mother non-Hisp White	0.737	0.440	0.724	0.447	0.847	0.360	0.810	0.392
Mother non-Hisp Black	0.033	0.178	0.044	0.206	0.109	0.311	0.145	0.353
Mother Asian	0.068	0.251	0.044	0.205	0.011	0.104	0.007	0.085
Mother Hispanic	0.026	0.160	0.036	0.186	0.031	0.173	0.035	0.183
Mother edu = 12 yrs	0.233	0.423	0.308	0.462	0.266	0.442	0.270	0.444
Mother edu 13–15 yrs	0.297	0.457	0.279	0.449	0.244	0.429	0.176	0.381
Mother edu ≥ 16 yrs	0.286	0.452	0.144	0.351	0.312	0.463	0.179	0.384
Mother married	0.753	0.431	0.692	0.462	0.705	0.456	0.629	0.483
Father's age	30.250	6.127	29.029	6.064	30.094	6.014	28.878	6.008
Kessner index = 2	0.267	0.442	0.320	0.467	0.251	0.434	0.299	0.458
Kessner index = 3	0.057	0.232	0.116	0.320	0.076	0.265	0.179	0.384
Private insurance	0.519	0.500	0.379	0.485	0.592	0.492	0.388	0.487
Medicaid	0.347	0.476	0.481	0.500	0.225	0.418	0.320	0.467
WIC	0.349	0.477	0.482	0.500	0.319	0.466	0.397	0.489
Number of sibling births	61,892	61,892	3333	3333	95,268	95,268	6609	6609

The sample consists of all the mothers who gave birth to two or three babies with gestational ages ≥30 weeks between 2003 and 2006 in Pennsylvania and Washington state. Additional covariates include infant birth year and month, any risk factor, number of other pregnant outcomes, mother's education missing, father's race, father's age/education missing, delivery payment missing, WIC missing, and living in Pennsylvania.

Table 1 reports the descriptive statistics of the dependent variables and key covariates. The primary birth outcomes of interest are birth weight and LBW. Birth weight is the primary and most frequently used measure of infant health. LBW is a key indicator of poor health at birth, which has a lasting adverse impact on health, cognitive development, earnings, and other lifetime outcomes. Prenatal smoking can increase the risks of having LBW babies by either impairing fetal growth or shortening gestation. While fetal growth retardation due to smoking is well understood in the literature, the biological mechanisms through which prenatal smoking can reduce gestation remain unclear (Kramer 1987). Hence, fetal growth is used as an additional birth outcome measure in the next section. Meanwhile, many infant, parental, and family control variables are used in this analysis, with missing values coded as separate indicators. This table shows two-birth mothers have heavier infants than those with three

births. They are also more likely to be nonsmokers, better educated, and married. This pattern is not driven by the short sample period, because it is also presented in Royer (2004) which uses a sample of sibling births delivered within a longer period of 12 years.

Table 2 compares nonsmokers and four types of smokers in the sample of mothers having two sibling births whose gestational ages are at least 30 weeks. The first row indicates that infants of the preconception quitters and early quitters are almost as heavy as those born to the nonsmokers. However, newborn babies of the late quitters are much lighter than those of the early quitters by 100 to 140 grams (g). Smoking through the third trimester is further associated with a moderate decrease in infant birth weight by about 70 g. The second row presents a similar relationship between the timing of smoking cessation and LBW. The chances of giving birth to a LBW baby are close among the nonsmokers, preconception quitters, and early quitters. In contrast, mothers who smoke beyond the first trimester are 3.7 to 4 percentage points more likely to have a LBW baby. The other rows of this table show delayed smoking cessation in pregnancy is correlated with mothers' low socioeconomic status (SES). The late quitters and continuous smokers generally receive less education than the nonsmokers, preconception quitters, and early quitters. They are more likely to be unmarried and enrolled in Medicaid and the Women, Infants and Children program (WIC). It is well known that a poor in-utero environment due to prenatal smoking plays an important role in explaining why low SES mothers transmit their health and economic status to the next generation. The present study indicates that at the heart of this mechanism is failure to quit smoking in a timely manner during pregnancy. This is because, as shown below, late cessation leads to remarkably worse birth outcomes than those of the nonsmokers and earlier quitters, while poor infant health further has a negative influence on many lifetime outcomes (Case, Fertig, and Paxson 2005). Taking into account the timing of smoking cessation, this research sheds new light on the way in which fetal exposure to smoking affects intergenerational transfer of health and wealth among the poor (Currie 2009).

To assess the relationship between prenatal smoking cessation and infant health, the following reduced form model of an infant health production is first estimated:

$$Y_{ij} = \alpha + \sum_{m=0}^{3} \beta_m SK_{ijm} + \sum_{n=0}^{N_1} \gamma_n X_{ijn} + \varepsilon_{ij} \tag{1}$$

where Y_{ij} is a health measure such as birth weight or LBW of an infant delivered by mother i with birth order j. SK is a vector of four types of smokers as mentioned previously. X is a rich set of control variables, including birth characteristics (infant male, parity,[5] birth year, birth month), parental demographics (age, race, ethnicity, and education), mother socioeconomic background, prenatal care (Kessner index[6]), delivery payment types, participation in WIC, number of other pregnant outcomes, and any risk factor. The results are almost the same when the model includes parity fixed effects (or birth-order fixed effects) instead of parity. Besides, ε_{ij} is an infant-specific component of health.

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[5] As in Abrevaya (2006), parity is defined as the number of times that a woman has delivered a live birth. For example, the parity of the first child is 0, and the corresponding birth order is 1.

[6] Kessner index is a standard measure of the adequacy of prenatal care, which can be 1 (adequate), 2 (intermediate), and 3 (inadequate). In this study, two indicators of Kessner index are created to capture intermediate and inadequate prenatal care (the base is adequate prenatal care).

Table 2. Characteristics of Nonsmokers and Four Different Types of Smokers

Variable	Mothers in Washington					Mothers in Pennsylvania				
	Nonsmokers	$SK_0 = 1$	$SK_1 = 1$	$SK_2 = 1$	$SK_3 = 1$	Nonsmokers	$SK_0 = 1$	$SK_1 = 1$	$SK_2 = 1$	$SK_3 = 1$
Birth weight	3466.494	3451.365	3425.316	3321.802	3252.484	3438.950	3403.360	3357.463	3215.881	3140.767
Low birth weight	0.031	0.032	0.039	0.079	0.072	0.037	0.044	0.054	0.091	0.095
Parity	1.253	0.923	0.982	1.154	1.502	1.305	0.833	0.924	1.114	1.427
Mother's age	27.938	24.651	23.662	23.363	24.007	28.161	25.665	23.987	23.494	24.417
Mother edu = 12 yrs	0.215	0.320	0.352	0.410	0.377	0.222	0.360	0.414	0.442	0.450
Mother edu 13–15 yrs	0.303	0.397	0.334	0.261	0.210	0.247	0.337	0.295	0.207	0.188
Mother edu $\geq$ 16 yrs	0.320	0.093	0.043	0.017	0.017	0.379	0.179	0.083	0.041	0.029
Mother married	0.797	0.590	0.470	0.397	0.367	0.788	0.581	0.403	0.311	0.337
Father's age	30.558	27.694	27.333	26.990	28.025	30.592	28.553	27.676	27.492	28.298
Father's age missing	0.052	0.100	0.182	0.209	0.221	0.046	0.063	0.129	0.172	0.152
Private insurance	0.554	0.407	0.314	0.248	0.211	0.645	0.615	0.463	0.353	0.309
Medicaid	0.316	0.357	0.486	0.590	0.641	0.161	0.296	0.401	0.528	0.525
WIC	0.314	0.469	0.581	0.688	0.647	0.248	0.414	0.543	0.625	0.640
Number of sibling births	54,781	1075	628	234	5174	74,926	4442	2016	798	13,086

The sample consists of all the mothers who gave birth to two babies with gestational ages $\geq$30 weeks between 2003 and 2006 in both states. $SK_0 = 1$ represents the mothers who quit smoking three months prior to pregnancy, $SK_1 = 1$ represents those who quit smoking in the first trimester, $SK_2 = 1$ indicates those who quit smoking in the second trimester, and $SK_3 = 1$ indicates those who smoked throughout pregnancy.

However, the estimates of β in Equation 1 are biased when unobserved mother heterogeneity is correlated with smoking cessation and infant health. If such heterogeneity is birth invariant, then the following model gives unbiased estimates:

$$Y_{ij} = \alpha + \sum_{m=0}^{3} \beta_m SK_{ijm} + \sum_{n=0}^{N_2} \gamma_n X_{ijn} + \mu_i + \varepsilon_{ij} \tag{2}$$

where μ_i is an indicator of sibling fixed effects (or mother fixed effects) and $N_1 > N_2$. It can capture a mother's birth invariant unobserved characteristics, such as her taste for healthy behavior, health endowment (Wang et al. 2002), and earnings potential. The direction and magnitude of the bias in each β_i estimate by Equation 1 depend on the correlation between μ_i and SK_m. On one hand, it is possible that late quitters and continuous smokers are on average more likely to heavily use other harmful substances or have poor nutrition during pregnancy than nonsmokers and earlier quitters. If any of these risk behaviors are unobserved, SK_2 or SK_3 is strongly and negatively correlated with μ_i,[7] thus substantially biasing up the estimates of β_2 and β_3. Table 2 provides some evidence for this story. It shows late quitters and continuous smokers are remarkably negatively selected on education, marital status, unplanned pregnancy (father's age missing), and other observed characteristics. Hence, the same selection pattern may also hold for the unobserved variables. On the other hand, disadvantaged smokers can compensate for the transmission of their low health endowments to their offspring (Rosenzweig and Wolpin 1991, 1995). They might quit early in pregnancy, or they may mitigate the detrimental effect of delayed smoking cessation by using other health-augmenting inputs. In this case, SK_2 and SK_3 are strongly and positively correlated with μ_i,[8] so that the estimates of β_2 and β_3 are biased downward to zero. However, this compensatory story seems less plausible, since Table 2 shows low SES mothers tend to postpone rather than expedite smoking cessation.

A necessary condition for Equation 2 to provide unbiased estimates of β is the strict exogeneity condition, $Cov(SK_{ij+1} - SK_{ij}, \varepsilon_{ij+1} - \varepsilon_{ij}) = 0$ for two sibling births. If this condition fails, and the covariance between the two differences of SK and ε is large enough such that $\frac{Cov(SK_{ij+1} - SK_{ij}, \varepsilon_{ij+1} - \varepsilon_{ij})}{Var(SK_{ij+1} - SK_{ij})} > \frac{Cov(SK_{ij}, \mu_i)}{Var(SK_{ij})}$, then controlling for the sibling fixed effects in Equation 2 can bias the estimates of β even more than those associated with omitted variables in Equation 1. This circumstance can happen if a woman's birth outcome systematically affects her future smoking behavior. In particular, a newborn with very poor health might cause the mother to quit smoking promptly before the next pregnancy. Another important concern on the sibling fixed effects model of Equation 2 is measurement error in self-reported smoking. Griliches (1979) points out that even a modest level of measurement error may create a large attenuation bias in sibling models. Both caveats are carefully addressed herein.

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[7] The correlation between SK_0 (or SK_1) and μ_i is probably much smaller. Since preconception quitters and early quitters are less dependent on cigarettes than late quitters and continuous smokers, their tendency to adopt other unhealthy behaviors may be weak too.

[8] Mothers who want to smoke just in the first trimester may not quit earlier if they believe smoking for a short time at the beginning of pregnancy does little harm to their babies. So the correlation between SK_1 and μ_i is possibly weaker. A similar reasoning applies to SK_0.

4. Results

Table 3 presents the baseline results using a sample of mothers who had two sibling births with gestational ages ≥30 weeks between 2003 and 2006. Column 1 shows the coefficient estimates of β in Equation 1 with the Washington sample. The adverse effects of fetal exposure to smoking on birth weight (upper panel) and LBW (lower panel) are small and statistically insignificant, if mothers give up smoking prior to the second trimester. However, late cessation is associated with much lower infant birth weights by 100 g and higher risks of having a LBW baby by 4 percentage points. There is a further decrease in infant birth weights if mothers keep on smoking through the third trimester. Column 2 controls for the sibling fixed effects, shrinking the estimated impact of late cessation to a decrease of 64 g on birth weight and an insignificant increase of 3 percentage points on LBW. The parameter estimates on smoking throughout are also sizably reduced. According to Wooldridge (2002), the omitted variable μ_i biases the estimated impacts of late cessation and smoking throughout by $\frac{Cov(SK_{ij},\mu_i)}{Var(SK_{ij})}$ in Equation 1. A comparison of the estimate of β_2 on birth weight in column 1 with that in column 2 suggests such a bias is $-99.9-(-63.5)=-36.4<0$ for late cessation; thereby, $Cov(SK_2,\mu)<0$. Similarly, $Cov(SK_3,\mu)<0$ for smoking throughout. The two negative correlations indicate that mothers with low health endowments (small μ) tend to exacerbate rather than compensate for the transmission of their genetic disadvantages through smoking beyond the first trimester (large SK_2 and SK_3). This finding is consistent with Rosenzweig and Wolpin (1995), Abrevaya (2006), Abrevaya and Dahl (2008), and Walker, Tekin, and Wallace (2009).

Columns 3 and 4 in Table 3 also show late instead of early cessation is associated with noticeably worse birth outcomes than those of nonsmokers and preconception smokers in Pennsylvania. In contrast to column 1, early quitters in Pennsylvania are more negatively selected, such that column 3 demonstrates the estimated impact of early cessation on birth weight is significant yet small. However, it becomes much smaller and insignificant, once the sibling fixed effects are controlled in column 4. When the two-state pooled sample is considered, column 6 indicates late cessation reduces birth weight by about 67.5 g and increases LBW by 1.2 percentage points when the sibling fixed effects are added. It also suggests about two thirds of the total detrimental smoking impact on infant physical health occurs in the second trimester. The results of other control variables are in line with the literature (Abrevaya 2006; Abrevaya and Dahl 2008). For example, infants who are male and born to white and married mothers with adequate prenatal care are significantly heavier at birth. Finally, six Hausman specification tests are conducted to compare the estimates of sibling random effects models with those of sibling fixed effects models (Wooldridge 2002). They all reject the null hypothesis that random effects models are preferred to fixed effects models.

Columns 1 and 2 in Table 4 extend the benchmark analysis to a larger sample of mothers with two or three births. The results are very close to Table 3. Columns 3 and 4 relax the 30 gestation weeks restriction, such that the sample consists of mothers having two sibling births whose gestational ages are at least 28 weeks. The corresponding estimated impacts of late cessation are larger. This is because with a less stringent restriction on the newborn gestational ages, the average duration when fetuses of late quitters are free from exposure to smoking is reduced when those mothers stop smoking in the second trimester. In addition, the estimated effect of late cessation on LBW becomes statistically significant. The last two columns provide

Table 3. Timing of Smoking Cessation and Infant Health

	Dependent Variable: Birth Weight					
	WA		PA		Pooled	
	(1)	(2)	(3)	(4)	(5)	(6)
Sibling fixed effects?	N	Y	N	Y	N	Y
Cessation before pregnancy(SK_0)	5.204	2.241	−1.110	−5.650	−0.939	−2.089
	(16.075)	(18.583)	(8.360)	(9.912)	(7.407)	(8.692)
Cessation in the 1^{st} trimester(SK_1)	−9.330	−21.819	−24.382*	0.788	−22.859**	−2.360
	(20.893)	(23.650)	(12.544)	(14.429)	(10.744)	(12.225)
Cessation in the 2^{nd} trimester(SK_2)	−99.874***	−63.536*	−137.310***	−72.490***	−132.861***	−67.504***
	(38.832)	(38.099)	(21.255)	(21.622)	(18.669)	(18.699)
Smoking throughout(SK_3)	−189.605***	−69.036***	−235.737***	−103.666***	−226.527***	−89.841***
	(9.028)	(13.739)	(6.236)	(11.810)	(5.101)	(8.951)
R^2	0.07	0.75	0.10	0.76	0.09	0.76
Hausman test	F=18.86, P-value=0.000		F=31.27, P-value=0.000		F=57.80, P-value=0.000	
	Dependent Variable: Low Birth Weight					
Sibling fixed effects?	N	Y	N	Y	N	Y
Cessation before pregnancy(SK_0)	−0.005	−0.012	0.002	−0.001	0.001	−0.004
	(0.006)	(0.009)	(0.003)	(0.005)	(0.003)	(0.004)
Cessation in the 1^{st} trimester(SK_1)	0.003	0.012	0.007	0.003	0.007	0.006
	(0.008)	(0.010)	(0.005)	(0.007)	(0.004)	(0.006)
Cessation in the 2^{nd} trimester(SK_2)	0.040**	0.026	0.038**	0.007	0.040***	0.012
	(0.017)	(0.020)	(0.010)	(0.013)	(0.009)	(0.011)
Smoking throughout(SK_3)	0.035***	0.021***	0.044***	0.016**	0.043***	0.018***
	(0.004)	(0.008)	(0.003)	(0.007)	(0.002)	(0.005)
R^2	0.02	0.58	0.03	0.60	0.02	0.59
Hausman test	F=2.38, P-value=0.049		F=5.33, P-value=0.000		F=7.89, P-value=0.000	
Number of sibling births	61,892	61,892	95,268	95,268	157,160	157,160

The sample consists of all the mothers who gave birth to two babies with gestational ages ≥ 30 weeks between 2003 and 2006 in both states. Additional controls include infant male, parity, infant birth year and month effects, mother's age, mother's race, mother's education, marital status, father's age/missing, father's race/missing, father's education/missing, number of other pregnant outcomes, Kessner index/missing, delivery payment types/missing, WIC participation/missing, any risk factor, and living in Pennsylvania. Birth invariant variables are dropped in the sibling fixed effects specification. Robust standard errors clustered at the mother's level are in parentheses. * significant at 10% level; ** significant at 5% level; *** significant at 1% level.

Table 4. Timing of Smoking Cessation and Infant Health (Extension to 2–3 Sibling Births, Gestation ≥ 28 Weeks, and Fetal Growth)

	Dependent Variable: Birth Weight					
	Mothers with 2 or 3 Births		Gestation≥ 28 Weeks		Control for Gestation	
	(1)	(2)	(3)	(4)	(5)	(6)
Sibling fixed effects?	N	Y	N	Y	N	Y
Cessation before pregnancy(SK_0)	−1.562	3.521	−3.193	−3.917	−0.706	−9.348
	(7.253)	(8.439)	(7.535)	(8.880)	(6.304)	(7.394)
Cessation in the 1^{st} trimester(SK_1)	−23.856**	1.434	−25.052**	6.113	−16.606*	−4.121
	(10.440)	(11.736)	(10.937)	(12.516)	(9.117)	(10.303)
Cessation in the 2^{nd} trimester(SK_2)	−134.789***	−76.877***	−163.378***	−96.773***	−89.798***	−55.916***
	(17.942)	(18.140)	(20.058)	(20.244)	(13.756)	(15.251)
Smoking throughout(SK_3)	−226.725***	−86.579***	−228.058***	−89.530***	−186.924***	−79.391***
	(4.938)	(8.563)	(5.167)	(9.167)	(4.233)	(7.533)
R^2	0.09	0.75	0.09	0.75	0.35	0.82
	Dependent Variable: Low Birth Weight					
Sibling fixed effects?	N	Y	N	Y	N	Y
Cessation before pregnancy(SK_0)	0.002	−0.003	0.002	−0.003	0.001	−0.001
	(0.003)	(0.004)	(0.003)	(0.004)	(0.002)	(0.004)
Cessation in the 1^{st} trimester(SK_1)	0.007	0.005	0.008*	0.009	0.005	0.008
	(0.004)	(0.006)	(0.005)	(0.006)	(0.004)	(0.005)
Cessation in the 2^{nd} trimester(SK_2)	0.039***	0.014	0.052***	0.025**	0.027***	0.009
	(0.008)	(0.011)	(0.009)	(0.012)	(0.007)	(0.010)
Smoking throughout(SK_3)	0.043***	0.018***	0.044***	0.019**	0.030***	0.015***
	(0.002)	(0.005)	(0.003)	(0.006)	(0.002)	(0.005)
R^2	0.02	0.58	0.02	0.60	0.23	0.67
Number of sibling births	167,102	167,102	157,670	157,670	157,670	157,670

All three samples consist of mothers delivering sibling babies between 2003 and 2006 in both states. The sample in Columns 1 and 2 focuses on mothers with two or three sibling births whose gestational ages are at least 30 weeks. The one in Columns 2 to 6 is restricted to mothers with two sibling births whose gestational ages are at least 28 weeks. The same additional controls as Table 3 are added into all the regressions. Robust standard errors clustered at the mother's level are in parentheses. * significant at 10% level; ** significant at 5% level; *** significant at 1% level.

evidence for when the newborn gestational age is further controlled. The coefficient estimates of *SK* now capture the relationship between smoking cessation at different pregnancy stages and fetal growth. Column 6 shows that late cessation is associated with a decrease of 56 g on birth weight for gestational age and an increase of 1 percentage point on LBW for gestational age.

This study also examines four more broadly defined types of smokers and the role of smoking intensity. The results are not shown in any table to save space. When the intermittent smokers are also considered, as in definition 2 of Appendix 1, all the newly included late quitters smoked in the second trimester, while only 40% of the newly added mothers who are defined as continuous smokers actually smoked in the second trimester. If a large fraction of the total adverse smoking impact on infant health occurs in the second trimester, the new parameter estimate on late cessation should be closer to that of smoking throughout. The finding is consistent with this prediction. In the additional analysis on smoking intensity, the estimates show smoking more than 10 cigarettes per day in the first and second trimester further reduces infant birth weight by 20 g for mothers who smoke throughout pregnancy. Since there are more heavy prenatal smokers in the Pennsylvania sample, this finding can partly explain why the estimated impact of smoking throughout pregnancy on birth weight is larger in Pennsylvania than Washington.

Several robustness checks are then conducted using the two-state pooled sample. Table 5 examines the validity of the strict exogeneity condition. The upper panel focuses on postpartum maternal smoking (Sabia 2008). It shows mothers with previous poor birth outcomes, such as LBW or preterm birth, are not significantly less likely to smoke in the subsequent births. The lower panel focuses on two smaller samples of mothers with healthy first births. The results are very close to the full sample cases in Tables 3 and 4. The longevity of smoking on maternal health may also affect infant health. Since smoking earlier than three months before pregnancy is not coded in the data set, this concern is handled by controlling for the interaction terms of maternal age and the four smoker categories. The coefficient estimates of SK_0 to SK_3 are similar to Table 3, with all the interaction terms statistically insignificant. They are not reported for brevity.

The left panel of Table 6 considers additional prenatal conditions. Column 1 shows the results are quite close to Table 3 when an indicator of the first child and the number of other children are further added into the sibling fixed effects specification. Prenatal smoking may also be associated with alcohol consumption, drug use, and depression, etc. (Conway and Kennedy 2004; Reichman et al. 2009). None of these is coded in the data, admittedly a limitation of this study. If those unobserved risky behaviors and health problems are constant across births, they are controlled by the sibling fixed effects. If they are not birth invariant, the sibling fixed effects specification of Equation 2 still gives unbiased coefficient estimates on the four categories of smokers. Those estimates capture the total effects of smoking cessation at different pregnancy stages on infant health. Such a total effect consists of a direct impact of smoking on birth outcomes and an indirect effect where smoking affects newborn babies by changing other prenatal health behaviors. Hence, if researchers are only interested in the direct smoking impact, the estimates of smoking by Equation 2 are biased. For instance, the estimated direct impact of late cessation is biased upward (downward) if late quitters treat alcohol use as a complement to (a substitute for) tobacco. In order to gauge and deal with this bias, two commonly reported prenatal health problems, hypertension and diabetes, are sequentially added. The literature shows alcohol consumption, drug use, and depression are key contributors to these two health problems (Thadhani et al. 2002; Wannamethee et al. 2003;

Table 5. Validity of the Strict Exogeneity Condition

	Mothers with Two Births Postpartum Smoking		Mothers with Two or Three Births Postpartum Smoking	
	(1)	(2)	(3)	(4)
Sibling fixed effects?	Y	Y	Y	Y
The previous birth is LBW	−0.000		−0.001	
	(0.004)	—	(0.004)	—
The previous birth is		0.005		0.005
preterm	—	(0.004)	—	(0.004)
Number of sibling births	157,160	157,160	167,102	167,102
	Mothers with Two Births (Healthy First Birth)		Mothers with Two or Three Births (Healthy First Birth)	
	Birth Weight	Low Birth Weight	Birth Weight	Low Birth Weight
Sibling fixed effects?	Y	Y	Y	Y
Cessation before	−2.005	−0.002	4.520	−0.003
pregnancy (SK_0)	(8.620)	(0.003)	(8.357)	(0.003)
Cessation in the first	−1.975	0.001	2.861	−0.001
trimester (SK_1)	(12.014)	(0.005)	(11.528)	(0.005)
Cessation in the second	−66.715***	0.012	−75.387***	0.012
trimester (SK_2)	(18.066)	(0.010)	(17.535)	(0.009)
Smoking throughout	−89.408***	0.015***	−86.830***	0.016***
(SK_3)	(8.846)	(0.005)	(8.487)	(0.004)
Number of sibling births	146,160	146,160	155,130	155,130

All four samples consist of mothers who gave birth to sibling births with gestational ages ≥30 weeks between 2003 and 2006 in both states. Columns 1 and 2 focus on the mothers with two sibling births. Columns 3 and 4 study the mothers with two or three sibling births. In the the lower panel, the two samples are further restricted to those mothers whose first births between 2003 and 2006 are healthy. The same additional controls as Table 3 are added into all the regressions. Robust standard errors clustered at the mother's level in parentheses.

* Significant at the 10% level.

** Significant at the 5% level.

*** Significant at the 1% level.

Bonari et al. 2004; Martel et al. 2005; Kozhimannil, Pereira, and Harlow 2009). Therefore, controlling for hypertension and diabetes can reduce the aforementioned bias. Columns 2 and 3 show that it makes the estimated impacts of late cessation and smoking throughout on infant health slightly smaller.

As another concern, excluding infants with gestational ages less than 28 or 30 weeks can result in selective samples of mothers who can somewhat mitigate the adverse smoking effects on infant health. Appendix 2 addresses this by a series of mean-difference tests on birth weight and three key mother characteristics for the four categories of smokers. This table shows that, for each smoker type, the means of infant birth weights are not statistically different across three cases: no restriction on newborn gestational age, gestational age ≥28 weeks, and gestational age ≥30 weeks. Yet stronger restrictions, such as gestational age ≥34 weeks, make the mean infant birth weights significantly different from that of the sample with no gestational age restriction. The same findings are shown with one-sided mean tests (not reported). In particular, when the one-sided mean comparison is taken between the sample with gestational age ≥30 weeks and the one without any restriction, the p-value is still greater than but close to the 0.1 significance level. This suggests that 30 weeks gestational age for the newborns is an important cutoff, below which nonselective samples can be obtained. There is always no significant difference in the means of mother's age, education, and marital status across the

Table 6. Timing of Smoking Cessation and Infant Health: Sensitivity to Other Prenatal Conditions and Gestation Restriction

	Dependent Variable: Birth Weight					
	Other Prenatal Conditions Controlled for			Expanded Sample of the 1st and 2nd Births		
	"First Child" and "Other Children"	"Hypertension"	"Hypertension" and "Diabetes"	Any Gestation	Gestation ≥28 weeks	Gestation ≥30 weeks
	(1)	(2)	(3)	(4)	(5)	(6)
Sibling fixed effects?	Y	Y	Y	Y	Y	Y
Cessation before pregnancy (SK_0)	5.089	5.617	6.225	10.044	2.742	1.427
	(8.675)	(8.657)	(8.655)	(7.225)	(6.958)	(6.947)
Cessation in the 1st trimester(SK_1)	3.716	4.404	5.003	−3.423	−12.536	−10.743
	(12.166)	(12.164)	(12.157)	(10.968)	(11.046)	(10.981)
Cessation in the 2nd trimester(SK_2	−65.221***	−65.214***	−64.567***	−85.454***	−82.339***	−82.130***
	(18.658)	(18.595)	(18.583)	(18.079)	(18.174)	(17.714)
Smoking throughout (SK_3)	−87.250***	−87.027***	−86.903***	−112.901***	−111.805***	−109.819***
	(8.911)	(8.895)	(8.898)	(9.903)	(10.323)	(10.347)
	Dependent Variable: Low Birth Weight					
Sibling fixed effects?	Y	Y	Y	Y	Y	Y
Cessation before pregnancy (SK_0)	−0.005	−0.006	−0.006	−0.001	−0.002	−0.002
	(0.004)	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)
Cessation in the 1st trimester(SK_1)	0.005	0.004	0.004	0.009	0.006	0.003
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
Cessation in the 2nd trimester(SK_2)	0.013	0.011	0.011	0.025***	0.025**	0.024**
	(0.011)	(0.011)	(0.011)	(0.009)	(0.010)	(0.010)
Smoking throughout (SK_3)	0.018***	0.017***	0.017***	0.028***	0.027***	0.026***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)
Number of sibling births	157,160	157,160	157,160	164,450	163,132	161,896

Columns 1 to 3 use a sample of mothers who had two sibling births with gestational age ≥ 30 weeks between 2003 and 2006 in both states. Columns 4 to 6 use a larger sample of the 1st and 2nd sibling babies (with three different gestational age restrictions) born between 2003 and 2006 in Washington and between 2003 and 2010 in Pennsylvania. The same additional controls as Table 3 are added into all the regressions. Robust standard errors clustered at the mother's level are in parentheses. * significant at 10% level; ** significant at 5% level; *** significant at 1% level.

four samples with different gestational age restrictions. Next, additional sensitivity analysis is conducted with a more homogeneous sample of the first and second births (Abrevaya and Dahl 2008). It is a subset of the expanded sibling sample mentioned previously, which have many more sibling pairs than the one used for the benchmark analysis. Columns 4 to 6 in Table 6 present consistent results. The estimated impacts of late cessation and smoking throughout on birth weight and LBW are not only statistically significant, but they are also very similar in the three cases: no restriction on newborn gestational age, gestational age $\geq$28 weeks, and gestational age $\geq$30 weeks.

Identification using sibling fixed effects models comes from the mothers whose smoking cessation statuses and other characteristics change across births. Appendix 3 reports within-mother variations in the timing of smoking cessation, infant health, and four key covariates in the baseline sample of two-birth mothers. The upper panel shows 20% of the smoking mothers switch out from smoking throughout to smoking cessation before the third trimester, or switch in from earlier cessation into smoking throughout across births. Such a switching out/in is associated with moderate improvement/impairment in birth outcomes and about 10 to 20% changes in the means of other parental characteristics. The lower panel shows only 6% of the smoking mothers switch into or out from late cessation. The corresponding changes in birth outcomes are also small,[9] but the variations in other key parental characteristics are similar to the upper panel. Moreover, the fraction of the mothers who switch into or out from early cessation and preconception cessation is 13% and 22%, respectively. Put together, the within-mother variations of smoking, infant health, and other characteristics are at best moderate in the baseline sample, which spans a short period of 2003–2006. The small variation in SK_2 may explain why the estimated effect of late cessation on LBW is insignificant in Table 3. To deal with this limitation, the expanded sibling sample is used, which includes about 0.3 million sibling births, that is, twice as many as those in the baseline sample. Since this expanded sample spans a longer period in Pennsylvania, it codes many more mothers who changed smoking and other characteristics across births. Column 2 in Table 7 shows that, controlling for the sibling fixed effects, the estimated relationship between timing of smoking cessation and infant health is the same as Table 3. It is worth noting that the effect of late cessation on LBW is now precisely the same as that estimated in Tables 4 and 6. Overall, this research shows late cessation is associated with significantly higher risks of having a LBW baby than early or preconception smoking cessation.

Measurement error can be very problematic in sibling fixed effects models (Griliches 1979; Koch and Ribar 2007). Misclassification of smoking status in this study is more likely to be one-sided than classical. Consider one case where smokers can underreport themselves as nonsmokers with a probability P, but not vice versa. Then controlling for sibling fixed effects can mitigate the biases associated with omitted variables if $\frac{(1+P)Cov(SK_{ij},\mu_i)+\beta Var(SK_{ij})}{Var(SK_{ij})} > \frac{(1+P)\beta}{(1+3P)(Var(SK_{ij})-Cov(SK_{ij},SK_{ij+1}))}$ (Bound, Brown, and Mathiowetz 2001; Abrevaya 2006). Checking this condition requires a direct estimate of P in Pennsylvania and Washington states, which is not available. Moreover, another important complication is that smokers can misreport themselves as earlier quitters (not as nonsmokers).

[9] Mothers who just became late quitters had infants heavier than before because some of those mothers had smoked throughout and delivered very unhealthy babies in the previous pregnancy.

Table 7. Timing of Smoking Cessation and Infant Health: Measurement Error in Smoking Status

	Dependent Variable: Birth Weight						
	Pooled Sample(Expanded)		Introduce 20 to 30% Under- or Mis-reporting of SK_2 or SK_3				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Sibling fixed effects?	N	Y	Y	Y	Y	Y	Y
Cessation before pregnancy(SK_0)	15.100***	6.477	7.486	7.890	5.708	4.966	−1.342
	(4.338)	(5.319)	(5.305)	(5.305)	(5.299)	(5.307)	(5.135)
Cessation in the 1^{st} trimester(SK_1)	−15.858**	−7.411	−5.636	−4.919	−7.001	−7.442	−10.073
	(6.642)	(7.754)	(7.734)	(7.723)	(7.686)	(7.671)	(7.175)
Cessation in the 2^{nd} trimester(SK_2)	−96.129***	−61.607***	−58.268***	−56.253***	−57.181***	−56.735***	−58.120***
	(11.720)	(12.312)	(13.367)	(14.382)	(13.569)	(13.451)	(10.899)
Smoking throughout(SK_3)	−230.185***	−108.254***	−105.983***	−105.113***	−106.466***	−106.701***	−97.052***
	(3.541)	(6.250)	(6.199)	(6.184)	(6.230)	(6.234)	(5.904)
	Dependent Variable: Low Birth Weight						
Sibling fixed effects?	N	Y	Y	Y	Y	Y	Y
Cessation before pregnancy(SK_0)	−0.003*	−0.004	−0.004	−0.004	−0.003	−0.003	−0.001
	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Cessation in the 1^{st} trimester(SK_1)	0.005*	0.005	0.004	0.004	0.004	0.004	0.004
	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Cessation in the 2^{nd} trimester(SK_2)	0.025***	0.016**	0.015**	0.014*	0.015**	0.014*	0.014**
	(0.005)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)	(0.006)
Smoking throughout(SK_3)	0.045***	0.027***	0.026***	0.026***	0.026***	0.026***	0.024***
	(0.002)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Number of sibling births	317,674	317,674	317,674	317,674	317,674	317,674	317,674

The expanded sampel consists of all the mothers who gave bith to two sibling babies with gestational ages ≥ 30 weeks between 2003 and 2006 in Washington and between 2003 and 2010 in Pennsylvania. The same additional controls as Table 3 are added into all the regressions. Robust standard errors clustered at the mother's level are in parentheses. * significant at 10% level; ** significant at 5% level; *** significant at 1% level.

To examine how much these two types of measurement error bias the smoking effect estimates, this study follows Abrevaya and Dahl (2008) to artificially introduce a large one-sided underreporting or misreporting of smoking factor in Table 7. Column 3 randomly misclassifies 20% of the observed late quitters as nonsmokers. As expected, it results in an attenuation bias on the sibling fixed estimates of late cessation (column 2) by 3.3 g in birth weight and 0.1 percentage points in LBW. However, such biases only account for about 10% of the differences between the nonsibling (column 1) and sibling fixed effects estimates (column 2). So the smoking estimates become smaller in the sibling fixed effects specification, mainly because this specification reduces the omitted variable biases rather than exacerbates the attenuation biases from measurement error. Similar results hold for column 4, with a very large 30% of underreporting among the late quitters (Brachet 2008). In column 5, 20% of the late quitters are evenly misreported as early quitters, preconception quitters, and nonsmokers. Column 6 examines an unevenly misreporting case such that 10% of the late quitters are misclassified as early quitters, 5% of them as preconception quitters, and 5% as nonsmokers. Both columns give similar smoking estimates as column 3. Finally, column 7 further misclassifies 20% of the observed continuous smokers equally as early quitters, preconception quitters, and nonsmokers. Again, the corresponding biases on the parameter estimate of SK_3 (11.2 g in birth weight and 0.3 percentage points in LBW) are too small to explain the differences between the nonsibling and sibling fixed effects estimates.[10] To summarize, these sensitivity checks generally suggest that measurement error seems unlikely to generate a large attenuation bias on the impact estimates of smoking, when the sibling fixed effects are controlled for.

Table 8 addresses how regarding late quitters as prenatal nonsmokers or early quitters as prenatal smokers can bias downward the parameter estimate on the conventionally used group measure "prenatal smoker." It reports the estimated impacts of prenatal smoking for three different definitions of "prenatal smoker," using a sample of two sibling births without any restriction on gestation. In the benchmark definition, "prenatal smoker" only includes mothers who smoke beyond the first trimester (late quitters and continuous smokers), consistent with the findings above. Columns 1 and 3 show the estimates are similar across the three definitions, when the sibling fixed effects are not controlled for. However, columns 2 and 4 give quite different results, once the sibling fixed effects are added into regressions. On one hand, coding late quitters as prenatal nonsmokers such that only continuous smokers (smoking in every trimester) are defined as "prenatal smoker" biases downward the estimated impacts of prenatal smoking on birth weight and LBW, by a third and over a half, respectively. On the other hand, grouping early quitters together with late quitters and continuous smokers as "prenatal smoker" (smoking in any trimester) biases the coefficient estimate of prenatal smoking on birth weight downward by a quarter to −71 g. The estimated smoking impact on LBW is also slightly reduced to an increase of 2 percentage points. Such two estimates are close to the study by Abrevaya (2006), which uses the same "prenatal smoker" definition (smoking in any trimester) and a similar sample of matched sibling births in 1990–1998. That article shows that when the sample is restricted to the most precisely matched sibling births, prenatal smoking is associated with lower

[10] Moreover, if underreporting is positively correlated across births where a mother who misreports smoking in the previous birth is more likely to misreport during this birth, it will further reduce the one-sided misclassification bias (Abrevaya 2006).

Table 8. Timing of Smoking Cessation and Three Measures of "Prenatal Smoker"

	Dependent Variable: Birth Weight		Dependent Variable: LBW	
	(1)	(2)	(3)	(4)
Sibling fixed effects?	N	Y	N	Y
Prenatal Smoker (smoking through the second or third trimester)	−228.634*** (5.234)	−92.552*** (9.379)	0.046*** (0.002)	0.021*** (0.005)
R^2	0.09	0.74	0.03	0.60
Prenatal Smoker (smoking in every trimester)	−221.872*** (5.238)	−62.851*** (9.734)	0.042*** (0.003)	0.010** (0.005)
R^2	0.08	0.74	0.03	0.60
Prenatal Smoker (smoking in any trimester)	−204.776*** (5.011)	−70.976*** (12.226)	0.042*** (0.002)	0.020*** (0.007)
R^2	0.08	0.74	0.03	0.60
Number of sibling births	162,366	162,366	162,366	162,366

The sample consists of all the mothers who gave birth to two sibling babies between 2003 and 2006 in both states, without any restriction on newborn gestational ages. The same additional controls as Table 3 are added into all the regressions. Robust standard errors clustered at the mother's level are in parentheses.

* Significant at the 10% level.

** Significant at the 5% level.

*** Significant at the 1% level.

infant birth weights by 67–69 g and higher risks of having a LBW baby by 1–1.3 percentage points.

Table 8 also corroborates two points in Fertig (2010), which examines to what extent the association between prenatal smoking and infant health is driven by selection using three British cohorts (1958, 1970, and 2000). First, it defines mothers in the 1958 and 2000 cohorts who reported smoking after the fourth month of pregnancy as "prenatal smoker." This definition is appropriate since it is consistent with the benchmark case in Row 2 of Table 8. Second, because the timing of prenatal smoking cessation is not coded for the 1970 cohort, Fertig (2010) treats all the quitters in pregnancy (including late quitters) as "prenatal nonsmoker." The estimated impact of selection into smoking in 1970 is smaller than 2000, consistent with the original hypothesis that mothers became more aware of the smoking hazards. However, Fertig (2010) points out another potential contributor to this finding, which is grouping all the quitters (especially late quitters) with nonsmokers, if late cessation is associated with significantly worse birth outcomes. Indeed, Table 8 shows this possibility cannot be ruled out.

5. Conclusions

As a key modifiable risk factor for poor infant health, prenatal smoking generates substantial intergenerational costs on health care, education, and public assistance systems. This article uses a unique large data set of sibling births to examine when women must quit smoking during pregnancy to deliver healthy babies. The baseline sibling fixed effects estimation indicates fetal exposure to smoking has a negligible effect on infant health with early prenatal smoking cessation in the first trimester. However, late cessation in the second trimester

is associated with noticeably lower infant birth weights by 68 g and higher risks of having a LBW baby by 1.2 percentage points. This research also shows smoking throughout pregnancy leads to a decrease of 90 g in birth weight and an increase of 1.2 percentage points in LBW. About two thirds of the total adverse smoking impact on infant health occurs in the second trimester. This baseline relationship between smoking cessation and birth outcomes is robust when the following extensions are considered: a larger sample of two and three sibling births, controlling for additional prenatal conditions, alternative restrictions on newborn gestational ages, misreporting of smoking, and the more broadly defined four types of smokers. Moreover, this research demonstrates that when late quitters are coded as prenatal nonsmokers or early quitters as prenatal smokers, it will introduce a new nontrivial downward bias in estimating the causality between the conventionally used group variable "prenatal smoker" and infant health.

Four conclusions are reached. First, smoking cessation as late as the second trimester cannot nullify the negative smoking impact on birth outcomes. Therefore, the first trimester is the critical period for prenatal smoking cessation. If the timing information of prenatal smoking cessation is specific and available, a well-defined group measure "prenatal smoker" should sensibly include only mothers who smoke beyond the first trimester. Third, mothers with low health endowments tend to exacerbate rather than compensate for the transmission of their genetic disadvantages by late smoking cessation or smoking throughout pregnancy. Fourth, there is also suggestive evidence that failure to stop smoking promptly during pregnancy is important in explaining why low SES prenatal smokers transmit their health and economic status to the next generation.

The policy and practical implication of this article is straightforward. Prenatal smoking cessation intervention must concentrate on the first trimester (early cessation). In particular, reallocating resources on smoking cessation to the first trimester can significantly improve the efficacy of any smoking cessation counseling or health program (for example, Medicaid) that covers smoking cessation interventions for the insured mothers. In evaluating the cost-effectiveness of a prenatal smoking cessation intervention, such as 5 A's,[11] researchers should not only look at the quit rate but also examine how successfully it can achieve early cessation.

[11] From 2000 to 2005, the Robert Wood Johnson Foundation provided grant supports for the American College of Obstetricians and Gynecologists to promote a five-step smoking cessation guideline as a routine part of prenatal care visits. This intervention is known as 5 A's (Ask, Advise, Assess, Assist, Arrange).

Appendix 1. Patterns of Maternal Smoking prior to and during Pregnancy

Smoking prior to and during Pregnancy				Smoking Status		Mothers in WA		Mothers in PA	
Before Preg?	First Trimester?	Second Trimester?	Third Trimester?	Definition 1	Definition 2	2 Births	3 Births	2 Births	3 Births
Y	Y	Y	Y	SK_3	SK_3	5260	450	13,438	1278
Y	Y	Y	N	SK_2	SK_2	241	20	835	57
Y	Y	N	N	SK_1	SK_1	640	37	2087	119
Y	N	N	N	SK_0	SK_0	1092	45	4550	223
N	N	N	N	Nonsmokers	Nonsmokers	54,846	2808	75,114	5052
Y	Y	N	Y	Excluded	SK_3	42	4	196	14
Y	N	Y	Y	Excluded	SK_3	59	4	245	18
N	Y	Y	Y	Excluded	SK_3	16	1	38	4
Y	N	N	Y	Excluded	SK_3	28	4	210	14
N	N	N	Y	Excluded	SK_3	30	1	119	15
N	Y	N	Y	Excluded	SK_3	0	0	6	1
N	N	Y	Y	Excluded	SK_3	10	0	31	1
Y	N	Y	N	Excluded	SK_2	8	0	34	0
N	Y	Y	N	Excluded	SK_2	5	0	3	2
N	N	Y	N	Excluded	SK_2	7	1	18	2
N	Y	N	N	Excluded	SK_1	26	0	64	1

The sample consists of all the mothers who gave birth to two or three sibling babies between 2003 and 2006 in both states, without any restriction on newborn gestational ages. SK_0, SK_1, SK_2, and SK_3 are indicators of "cessation before pregnancy," "cessation in the first trimester," "cessation in the second trimester," and "smoking throughout," respectively.

Appendix 2. Tests of Mean Differences on Infant Birth Weight and Mother Characteristics by Gestational Ages

	SK_0	Mean Test	SK_1	Mean Test	SK_2	Mean Test	SK_3	Mean Test
Birth weight (g)								
No restriction	3394.057		3357.426		3179.725		3175.459	
(G_1)	(558.418)		(572.488)		(739.714)		(532.404)	
Gestation	3404.744	$G_2 = G_1$:	3364.822	$G_2 = G_1$:	3195.565	$G_2 = G_1$:	3178.501	$G_2 = G_1$:
≥28 wks (G_2)	(526.415)	$P = 0.295$	(543.112)	$P = 0.623$	(627.561)	$P = 0.634$	(513.288)	$P = 0.592$
Gestation	3407.547	$G_3 = G_1$:	3367.412	$G_3 = G_1$:	3219.491	$G_3 = G_1$:	3182.245	$G_3 = G_1$:
≥30 wks (G_3)	(510.556)	$P = 0.209$	(527.844)	$P = 0.539$	(589.734)	$P = 0.228$	(504.686)	$P = 0.232$
Gestation	3429.493	$G_4 = G_1$:	3390.189	$G_4 = G_1$:	3299.252	$G_4 = G_1$:	3209.054	$G_4 = G_1$:
≥34 wks (G_4)	(488.012)	$P = 0.000$	(503.333)	$P = 0.025$	(509.580)	$P = 0.000$	(483.248)	$P = 0.000$
Mother's age								
No restriction	25.452		23.848		23.780		24.387	
(G_1)	(5.032)		(4.809)		(4.936)		(4.955)	
Gestation	25.405	$G_2 = G_1$:	23.857	$G_2 = G_1$:	23.545	$G_2 = G_1$:	24.396	$G_2 = G_1$:
≥28 wks (G_2)	(5.038)	$P = 0.619$	(4.806)	$P = 0.945$	(4.937)	$P = 0.321$	(4.937)	$P = 0.873$
Gestation	25.404	$G_3 = G_1$:	23.851	$G_3 = G_1$:	23.549	$G_3 = G_1$:	24.396	$G_3 = G_1$:
≥30 wks (G_3)	(5.035)	$P = 0.612$	(4.799)	$P = 0.982$	(4.849)	$P = 0.339$	(4.929)	$P = 0.874$
Gestation	25.425	$G_4 = G_1$:	23.844	$G_4 = G_1$:	23.502	$G_4 = G_1$:	24.376	$G_4 = G_1$:
≥34 wks (G_4)	(5.033)	$P = 0.776$	(4.786)	$P = 0.975$	(4.811)	$P = 0.207$	(4.954)	$P = 0.837$
Mother edu ≥12 yrs								
No restriction	0.510		0.369		0.259		0.234	
(G_1)	(0.500)		(0.483)		(0.438)		(0.423)	
Gestation	0.504	$G_2 = G_1$:	0.367	$G_2 = G_1$:	0.260	$G_2 = G_1$:	0.231	$G_2 = G_1$:
≥28 wks (G_2)	(0.500)	$P = 0.523$	(0.482)	$P = 0.878$	(0.439)	$P = 0.959$	(0.422)	$P = 0.513$
Gestation	0.504	$G_3 = G_1$:	0.368	$G_3 = G_1$:	0.262	$G_3 = G_1$:	0.231	$G_3 = G_1$:
≥30 wks (G_3)	(0.500)	$P = 0.523$	(0.482)	$P = 0.939$	(0.440)	$P = 0.879$	(0.421)	$P = 0.516$
Gestation	0.506	$G_4 = G_1$:	0.369	$G_4 = G_1$:	0.259	$G_4 = G_1$:	0.235	$G_4 = G_1$:
≥34 wks (G_4)	(0.500)	$P = 0.671$	(0.483)	$P = 1.000$	(0.438)	$P = 1.000$	(0.424)	$P = 0.833$
Mother married								
No restriction	0.588		0.419		0.334		0.344	
(G_1)	(0.492)		(0.493)		(0.472)		(0.475)	
Gestation	0.580	$G_2 = G_1$:	0.411	$G_2 = G_1$:	0.321	$G_2 = G_1$:	0.344	$G_2 = G_1$:
≥28 wks (G_2)	(0.494)	$P = 0.388$	(0.492)	$P = 0.547$	(0.467)	$P = 0.535$	(0.475)	$P = 1.000$
Gestation	0.581	$G_3 = G_1$:	0.412	$G_3 = G_1$:	0.322	$G_3 = G_1$:	0.345	$G_3 = G_1$:
≥30 wks (G_3)	(0.493)	$P = 0.450$	(0.492)	$P = 0.597$	(0.467)	$P = 0.569$	(0.475)	$P = 0.826$
Gestation	0.583	$G_4 = G_1$:	0.413	$G_4 = G_1$:	0.330	$G_4 = G_1$:	0.346	$G_4 = G_1$:
≥34 wks (G_4)	(0.493)	$P = 0.590$	(0.492)	$P = 0.653$	(0.470)	$P = 0.836$	(0.476)	$P = 0.662$

All four samples consist of mothers who gave birth to two sibling babies between 2003 and 2006 in both states. However, the samples differ in their restrictions of newborn gestational ages. Standard errors are reported in parentheses.

Appendix 3. Within-Mother Changes in the Timing of Smoking Cessation, Infant Health, and Key Covariates

	20% of Smoking Mothers Switch from $SK_3=1$ to $SK_3=0$ ("Switch Out"), or from $SK_3=0$ to $SK_3=1$ ("Switch In")				6% of Smoking Mothers Switch from $SK_2=1$ to $SK_2=0$ ("Switch Out"), or from $SK_2=0$ to $SK_2=1$ (called "Switch In")			
	Switch Out (SK_3)		Switch In (SK_3)		Switch Out (SK_2)		Switch In (SK_2)	
	Previous Birth	This Birth	Previous Birth	This Birth	Previous Birth	This Birth	Previous Birth	This Birth
Birth weight (g)	3206.198	3315.927	3277.618	3210.455	3265.260	3335.884	3205.568	3243.003
	(511.247)	(525.059)	(560.317)	(514.384)	(570.187)	(523.085)	(554.666)	(593.799)
Low birth weight	0.088	0.063	0.081	0.079	0.066	0.053	0.114	0.091
	(0.284)	(0.242)	(0.273)	(0.270)	(0.249)	(0.225)	(0.318)	(0.289)
Mother's age	23.599	25.495	22.270	24.222	24.475	25.495	22.912	24.760
	(4.784)	(4.823)	(4.628)	(4.626)	(4.618)	(4.823)	(4.721)	(4.777)
Mother edu >12 yr	0.255	0.292	0.237	0.250	0.254	0.280	0.252	0.265
	(0.436)	(0.455)	(0.425)	(0.433)	(0.436)	(0.449)	(0.435)	(0.442)
Mother married	0.339	0.435	0.289	0.354	0.315	0.389	0.268	0.360
	(0.474)	(0.496)	(0.454)	(0.478)	(0.465)	(0.488)	(0.444)	(0.481)
Father's age	27.627	29.169	26.435	28.337	26.512	28.413	27.140	28.671
	(5.957)	(5.718)	(5.984)	(5.774)	(5.836)	(5.768)	(6.036)	(5.765)

The sample consists of all the mothers who gave birth to two sibling babies with gestational ages ≥30 weeks between 2003 and 2006 in both states. Standard errors are reported in parentheses.

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