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Selection and the Effect of Prenatal Smoking

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Abstract

There is a debate about the extent to which the effect of prenatal smoking on infant health outcomes is causal. Poor outcomes could be attributable to mother characteristics which are correlated with smoking. I examine the importance of selection on the effect of prenatal smoking by using three British cohorts where the mothers' knowledge about the harms of prenatal smoking varied substantially. I find that the effect of smoking on the probability of a low birth weight birth conditional on gestation is slightly more than twice as large in 2000 compared to 1958, implying that selection could explain as much as 50 percent of the current association between smoking and birth outcomes.

Keywords

smoking; child health; selection

1 Introduction

Research has found that maternal smoking during pregnancy is associated with retarded fetal growth, prematurity, fetal and neonatal deaths, and Sudden Infant Death Syndrome. However, there is an on-going debate about the extent to which these associations are causal. It is difficult to determine whether these poor health outcomes are the result of prenatal smoking or are also attributable to characteristics of the mother which are correlated with prenatal smoking. One approach used by medical researchers to get around this difficulty is to provide evidence consistent with theories about the biological mechanism by which prenatal smoking affects birth outcomes. These studies use experiments with animals, examinations of human tissue, and controlled smoking cessation experiments. The theories argue that smoking causes cell damage and placental changes; reduces the levels of pregnancy estrogens and synaptic activity; and impairs the oxygen availability to the fetus (Slotkin, 1998; Walsh, 1994). Yet, the medical community has not come to a consensus about how this biological evidence fits together into a mechanism:

“...a considerable amount of biological evidence supports the view that smoking contributes to adverse pregnancy outcomes. However, mechanisms for the effects of smoking during pregnancy have not been clearly elucidated.” (Walsh, 1994)

Social scientists have applied a variety of other strategies to get at causality. First, researchers have attempted to control for selection using an elaborate set of observable characteristics. Brooke et al. (1989) include a total of 40 controls for alcohol, caffeine, socioeconomic factors, and measures of psychosocial stress and find that prenatal smoking

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is the most important determinant of low birth weight conditional on gestation. Reichman, Corman, Noonan, and Dave (2008) also include some typically unobserved variables like wantedness, taste for risky behavior, and the mother's health endowment, and find that these variables do not substantially alter the estimate of the effect of smoking on birth outcomes when these variables are excluded. Almond, Chay, and Lee (2005) adjust for bias due to observable confounders using a propensity score matching technique and find a higher incidence of low birth weight among smoking mothers consistent in size with estimates not using this technique.

Second, a few researchers have used an instrumental variable (IV) approach. Permutt and Hebel (1989) use randomized smoking cessation counseling as an instrument for smoking during pregnancy and find substantial increases in birth weight among those who received counseling. Evans and Ringel (1999) and Lien and Evans (2005) use state cigarette taxes as an instrument and in both studies find that the effect of smoking is similar to estimates where prenatal smoking is treated as exogenous. However, Brachet (2005) shows that when measurement error in self-reported smoking is taken into account, the instrumental variable estimate obtained using cigarette taxes as the instrument is substantially smaller than conventional estimates.

Third, Grossman and Joyce (1990) correct for selection using the Heckman correction technique. They use variation in the number of previous abortions to adjust for the biases resulting from the ability of women to choose to have a birth. Using this approach they find that the effect of prenatal smoking on birth weight is roughly the same whether or not the selection is corrected.

Finally, two studies have used a fixed effects approach. Rosenzweig and Wolpin (1991) use a within-mother fixed effects estimation procedure to purge unobservable mother characteristics to find that prenatal smoking significantly reduces birth weight, but the magnitude of the effect is smaller than when fixed effects are not included. Abrevaya (2006) follows Rosenzweig and Wolpin (1991) but with a much larger data set and also finds significantly smaller effect sizes on birth outcomes than found with other approaches.

Thus, while all studies find that prenatal smoking has a causal effect on birth outcomes, the degree to which this estimate differs from estimates which treat smoking as exogenous varies substantially. It is difficult to claim that one approach clearly dominates the others as each has a short-coming. The first approach requires that the important factors are all observable and measurable. The IV approach estimates an effect for only those women who change their behavior in response to the instrument. The paper using the Heckman selection correction only adjusts for selection into having a birth, not selection into smoking itself. Finally, fixed effects requires that some mothers change their smoking behavior across pregnancies, which is likely correlated with other changes in the mother's behavior.

In contrast to these approaches, in this paper, I examine the importance of selection on the estimated effect size of prenatal smoking on birth outcomes by comparing the effect sizes across different birth cohorts where the mothers' knowledge about the harms of prenatal smoking varied substantially. This approach assumes that mothers who smoked during pregnancies that occurred prior to widespread knowledge about the dangers of smoking were a less select group than mothers who smoked during pregnancies following this knowledge. This approach has the advantage that the exact type of selection need not be observable or measurable. Using three British birth cohorts – those children born in 1958, 1970, and 2000, I find that the effect of prenatal smoking on being born low birth weight conditional on gestation is approximately double for the last cohort compared to the earliest cohort, indicating that selection plays an important part in the measured effect.

2 Data and Sample

In this study, I use the 1958 National Child Development Study (NCDS), the 1970 British Cohort Study (BCS), and the Millennium Cohort Study (MCS). The 1958 NCDS involves a sample of all children born in Great Britain (Scotland, England and Wales) in the week of March 3, 1958. I restrict the sample to 16,549 children, excluding about 500 non-white children because of their small number and potential difference in birth weight and maternal smoking habits, and excluding multiple births because the production function of birth health is different in many ways. The parents of these children were interviewed at the time of birth and at several periodic follow-up interviews. The 1970 BCS mimics the design of the NCDS; it involves a sample of all children born during the week of April 5, 1970, and the first of several periodic interviews was conducted at birth. I restrict my sample to 15,750 white, singleton children from Great Britain, dropping about 500 non-white children and children from Northern Ireland. In contrast, the MCS employs a slightly different design; it involves a random sample of children born over 12 months starting September 2000 in England and Wales and December 2000 in Scotland, and includes over-samples of non-white children. I restrict my sample to 13,101 white, singleton children from Great Britain, dropping any children from Northern Ireland and over 3,000 children due to race/ethnicity. The first MCS interview occurs when the child is between 8 and 12 months old.

The observations from these three data sets are pooled creating a combined sample of 45,400 births. Because the MCS has a complicated sampling design, I use a sampling weight which takes into account the various over-sampled groups. The NCDS and BCS are representative birth samples in the year of the interview so all observations from these surveys are assigned a weight of 1. The results are unchanged whether the sampling weight for the MCS is used or not.

Questions about smoking during pregnancy were asked of mothers during the first interview of each study. I have coded mothers as smoking during pregnancy if the mother reported that she smoked after the fourth month of the pregnancy, except for the 1970 cohort where I cannot distinguish the month of cessation.¹ In this case, I categorize mothers as having smoked during pregnancy if they reported that they did not stop smoking during the pregnancy. Table 1 presents summary statistics. Nearly 34% of mothers smoked during pregnancy in the 1958 birth cohort. The prevalence of prenatal smoking in 1970 is 42%, consistent with statistics indicating that smoking rates among women in general were at their highest levels in the U.K. in the early 1970s (Peto et al., 2000).² In 2000, only 23% of mothers reported smoking past their fourth month of pregnancy.

¹The 1958 survey instrument asks 'Did the patient smoke as many as one cigarette a day during the 12 months before the start of this pregnancy?' and 'Did the patient change her smoking habits during this pregnancy?'. The response to the second question is open-ended and thus summarized in the publicly released data by a variable indicating whether the mother 1) never smoked, stopped pre-pregnancy, or stopped smoking before the fifth month of pregnancy; 2) smoked after the fourth month of pregnancy but smoked less than 10 cigarettes a day; 3) smoked after the fourth month of pregnancy and smoked 10 cigarettes a day or more; and 4) was a variable smoker after the fourth month of pregnancy. For this analysis, mothers were coded as smoking during pregnancy if she was in the latter three categories of this variable. The 2000 survey instrument asks 'Do you smoke tobacco products such as cigarettes, cigars or a pipe at all nowadays?', 'Have you smoked at all in the past two years?', 'Did you change the amount you smoked during your pregnancy?', and 'In what month of the pregnancy did you make this change?'. Mothers were coded as smoking during pregnancy if she 1) smoked before pregnancy and did not quit, 2) reduced her smoking during pregnancy but still smoked, or 3) quit smoking during pregnancy but not until after the fourth month. The mother was coded as a non-smoker if she 1) did not smoke in the 2 years prior to pregnancy, 2) quit smoking before the pregnancy, or 3) quit smoking before the fifth month of pregnancy. The 1970 survey instrument asks 'Does the mother smoke now?', 'Did she ever smoke?', 'How long ago did she stop?', and 'Has she smoked during this pregnancy?'. The responses to these questions are not available with the publicly released data. Instead, a composite variable created from these questions is provided which indicates whether the mother 1) never smoked, 2) stopped smoking pre-pregnancy, 3) stopped smoking during pregnancy, 4) smoked during pregnancy (<5 cigarettes per day), 5) smoked during pregnancy (5–14 cigarettes per day), or 6) smoked during pregnancy (15 or more cigarettes per day). As a result, the month of quitting during pregnancy cannot be determined. For the 1970 cohort, mothers were coded as smoking during pregnancy if she was in the latter three categories of this variable.

In this analysis, I concentrate on three related birth outcomes: low birth weight for gestation, prematurity, and (unconditional) low birth weight. Low birth weight for gestation is of interest because this measure captures intrauterine growth retardation and research suggests that prenatal smoking is its leading cause in developed countries (Kramer, 1987).³ Prenatal smoking is also a risk factor for prematurity (Kramer, 1987), defined as a gestation of less than 37 weeks. However, the cause of the vast majority of premature cases is unknown, thus, the relative importance of smoking on prematurity may be low. Low birth weight, defined as a birth weight of less than 2500 grams, or about 5.5 pounds, can be attributed to short gestational length or a slow rate of fetal growth in utero so I use this third measure to capture both of these mechanisms. The percent born low birth weight is between 5.5% and 6.8% across all cohorts, such that fewer births have low birth weight in 2000 than in the earlier cohorts. The rates of prematurity range between 5.2 and 7.0 percent, such that more pre-term deliveries take place in 2000 than in the earlier years, which probably reflects better survival rates due to technological advances (which I discuss further in a later section).

Controls include child's gender, marital status at birth, timing of first prenatal doctor's visit, parity, mother's age at birth, mother's school leaving age, father's social class at birth, and mother's social class at birth. Descriptive statistics for these variables are presented in Table 1 with the exception of the last four variables, which I describe in detail in the next section. In all cohorts, between 51 and 52 percent of the babies are boys. In the first two cohorts, more than 90 percent of mothers were married at birth where in the last cohort only 60 percent of mothers are married, consistent with the trend in non-marital births in the U.K. as well as in the U.S. The first prenatal doctor's visit occurred in the first trimester (<12 weeks of gestation) in 20 percent of births in 1958, but in over 40 percent of births in 2000, reflecting an increase in knowledge about the importance of early prenatal care. Very few mothers went without prenatal care in any cohort, but the percentage is substantially higher in the most recent cohort than in the earlier cohorts. Lastly, fewer births were of higher order in the most recent cohort compared to earlier cohorts, reflecting a decrease in family size over the cohorts.

3 Knowledge and Selection

While there were several early studies linking smoking and cancer (Adler, 1912; Lickint, 1929; Doll & Hill, 1950), Doll (1998) argues that the evidence was largely ignored until 1950 when five case-control studies (4 from the U.S. and 1 from the U.K.) were published. The harmful effects of smoking were reported in the popular press in the U.S. in the 1950s but very little was reported in the U.K. prior to the 1962 Royal College of Physicians of London Report and the 1964 report from the U.S. Surgeon General. However, these reports contained nothing specifically about women, nor the particular detrimental relationship between prenatal smoking and birth outcomes. Research connecting prenatal smoking and low birth weight was first published in 1957 (Simpson, 1957) but not widely reported until the 1969 U.S. Surgeon General's Report, and even then did not receive much attention in the U.K. Finally, a study published in 1972 using the 1958 British birth cohort argued a connection between prenatal smoking and low birth weight and received a lot of media attention in the U.K. (Butler, Goldstein, & Ross, 1972). Soon after, the Health Education Council mounted a television advertising campaign in Britain involving a naked, pregnant smoking woman asking 'Is it fair to force your baby to smoke cigarettes?' (Berridge, 2001).

²The 1970 statistic is likely an underestimate compared to the 1958 and 2000 numbers given that, for this cohort, I code mothers who quit smoking in the fifth through ninth months of pregnancy as non-smokers, where these mothers would be classified as smokers for the other two cohorts.

³Low birth weight for gestation is captured by using low birth weight as the dependent variable and gestation as an independent variable.

Given the timeline outlined above, I argue that knowledge about the connection between prenatal smoking and poor birth outcomes was not widespread until the early 1970s. Consistent with this, Peto et al. (2000) finds that smoking rates among women declined steadily beginning in the early 1970s. At its peak, about half of all British women under the age of 60 smoked cigarettes. This magnitude implies that women smokers must have come from many income and education groups prior to 1970. The decline in smoking beginning in the 1970s suggests that a more select group of women chose to continue or take-up smoking despite the public health campaigns. To demonstrate that adverse selection into prenatal smoking increases across the three cohorts, I provide the following four figures.

Figure 1 presents the percentage of mothers who smoked during pregnancy by the mother's age at birth. I divided mothers into three age categories – teenage mothers, older mothers, and a middle age group to whom the majority of births occur. The bars provide the distribution of mothers across these categories and the lines represent the percentage who smoked during pregnancy by age group. The distribution of births across mother's age has not changed in an important way across the cohorts. In contrast, the relationship between mother's age and smoking status has changed substantially over time; the percentage of smokers does not vary across age groups for the 1958 cohort while there is a 30 percentage point difference between the oldest and youngest mothers for the 2000 cohort. The smoking prevalence was higher for all age groups in the 1970 cohort and the difference across age groups is present but not as dramatic as for the 2000 cohort. This figure suggests that there was little selection into prenatal smoking by age group for the earliest cohort, but that as more information about the dangers of prenatal smoking disseminated, the more mature mothers in the later cohorts chose not to smoke.

The following three figures follow a similar pattern. Figure 2 presents the percentage who smoked during pregnancy by mother's education at birth. The indicator of mother's education is her age at leaving school. This question is asked soon after birth for both the 1970 and 2000 cohorts but is not asked of the 1958 cohort until several years later. Due to attrition, there is a large number of 1958 cohort mothers missing this variable. Note also that there is a large increase in maternal education between the 1970 cohort and the 2000 cohort due to an education law change in 1976. The fraction of mothers who smoked during pregnancy is higher among mothers who left school at younger ages than among more educated mothers, however, the contrast across education groups is greatest for the 2000 cohort and least striking for the 1958 cohort.

Figure 3 presents the percentage who smoked during pregnancy by the baby's father's social class at birth. In the U.K., social class is a popular indicator of economic status and is based solely on occupation. The six classes are professional (i), managerial (ii), non-manual skilled (iiim), manual skilled (iiim), manual semi-skilled (iv), and unskilled (v). Because information on fathers who do not reside in the child's household is not asked, I also include a separate column in the graph for missing observations. The distribution across the classes is roughly the same for the 1958 and 1970 cohort, however, there is some shuffling away from manual skilled to managerial in the 2000 cohort. The number of missing observations grows across the cohorts as this group is growing over time. The differences across groups in the percentage who smoked during pregnancy is smallest for the 1958 cohort, slightly larger for the 1970 cohort, and much larger for the 2000 cohort.

Finally, because father's social class has more missing observations in 2000 than in 1958, I also present the percentage of mothers who smoked during pregnancy by the mother's social class at birth in Figure 4. For mothers, there are five possible classes: professional or managerial (i or ii), skilled (iii), semi-skilled (iv), unskilled (v), and out of the labor force or unemployed (no job). There are large changes across the cohorts in the distribution of

occupational categories. In the more recent cohorts, fewer mothers do not have a job while more mothers are in the top two categories. The smoking rates among those without a job increases across the cohorts, probably reflecting a decrease in the social status among women associated with not working in the market. Still, among employed mothers, the difference in smoking rates across social classes is smallest for the 1958 cohort and largest for the 2000 cohort, suggesting again that selection into smoking changed as knowledge about smoking spread.⁴

It is reasonable to think that there is some adverse selection even in 1958 given that smoking has some undesirable side effects (like shortness of breath and coughing) which might deter healthier women. However, these four graphs illustrate worsening selection of easily observable characteristics across the cohorts. I argue that this pattern is highly suggestive of what we should expect among unobservable characteristics as well.

4 Empirical Strategy and Main Results

The strategy of this paper is to compare the effect of prenatal smoking on birth outcomes that occur in 2000, when adverse selection is great according to the suggestive evidence presented above, to the effect on birth outcomes that occur in 1958, when I argue there was much less adverse selection into smoking. I also use the 1970 sample as a comparison group to test whether there is a ‘dosage’ effect; that is, I assume that knowledge dispersion and thus adverse selection is greater in 2000 than in 1970, so a greater effect size in 2000 compared to 1970 is consistent with a higher ‘dose’ of adverse selection bias.

In essence, I employ a difference-in-differences approach to determine the extent to which selection biases the estimated effect of prenatal smoking on birth outcomes. Let B_i represent birth outcomes, and let S_i equal 1 if the mother smoked during pregnancy and 0 otherwise.

Let D_i^{70} and D_i^{00} be dummy variables indicating whether the pregnancy occurred in 1970 or 2000, respectively. Let X_i include child’s gender, mother’s age at the child’s birth, mother’s school leaving age, father’s social class at birth, mother’s social class at birth, mother’s marital status at birth, the timing of the first prenatal doctor’s visit and the mother’s parity. These variables are included to account for compositional changes in observed characteristics of smokers over time. These variables may be correlated with unobservable characteristics and thus may absorb some selection based on unobservables.

$$B_i = \beta_0 + \beta_1 S_i + \beta_2 S_i D_i^{70} + \beta_3 S_i D_i^{00} + \beta_4 D_i^{70} + \beta_5 D_i^{00} + \beta_6 X_i + \varepsilon_i \quad (1)$$

Then, in the above equation, β_1 represents the effect of prenatal smoking in 1958 when there is little selection. If there was no selection into smoking in 1958, this coefficient would reflect the true causal effect of prenatal smoking. However, given that there is likely some selection in 1958, it is an estimate of the effect of smoking with less selection bias than the effect in 1970 or 2000. The coefficients β_2 and β_3 represent the effect of selection into smoking based on those unobservable characteristics uncorrelated with the observable controls from the 1970 and the 2000 cohorts, respectively. If there is a dosage effect, β_3 should be larger than β_2 .

The first three columns of Table 2 present the results from applying the probit model to equation (1); the last three columns have as dependent variables the underlying continuous

⁴Tests of significance confirm that the difference across groups is significantly greater in 1970 than in 1958 and in 2000 compared to 1958 for all four characteristics.

variables which are used to create the dichotomous dependent variables in the first three columns. These are log birth weight (in grams) for gestation, gestation in weeks (ranging from 1, which represents 27 weeks or earlier, to 21, which represents 47 weeks), and unconditional log birth weight. Note that worse outcomes are smaller in value for the continuous variables, which implies that the signs of these coefficients will be the opposite of those in the first three columns. I use ordinary least squares (OLS) when the dependent variable is continuous.

The coefficients on ‘smoked’ are significant for all six outcomes. In the first column, the coefficients on the interactions indicate that the effects of smoking on the probability of being low birth weight conditional on gestational age for the 1970 and the 2000 cohorts are larger than for the 1958 cohort but only significantly so for the 2000 cohort. In addition, the coefficient on the 2000 cohort interaction is larger than the coefficient on the 1970 cohort interaction. In column (4), the effect of smoking on birth weight for gestation is significantly larger in both 1970 and 2000 than in 1958 and again the difference in magnitude is larger for the 2000 cohort than for the 1970 cohort.

The finding that the coefficient on the 1970 smoking interaction is smaller than the coefficient on the 2000 smoking interaction is consistent with the hypothesis that the effect size scales with the degree of selection. As discussed above, the public was just becoming aware of the dangers of prenatal smoking in 1970. However, there is another possibility that may explain why the effect is not significantly different between the 1958 and 1970 cohorts. It may be that the estimated effect is smaller for the 1970 cohort than for the 2000 cohort because the definition of a smoker is limited to those who did not quit during pregnancy, instead of including those who quit after the fourth month of pregnancy. This definition change means that those mothers who quit smoking after the fourth month of pregnancy are smokers in 1958 and 2000, but non-smokers in 1970. If this group has very poor infant outcomes, then the non-smokers will look similar to the smokers in 1970 (hence a small effect of smoking), but not in the other cohorts. I have no way of testing whether this is the reason for the 1970 coefficient size and thus focus most of the discussion on the difference between the 1958 and 2000 cohorts.

In the second and fifth columns, the coefficients on the interactions are not significantly different from zero suggesting that selection does not play an important role in the effect size of prenatal smoking on prematurity or gestation. If there are more factors involved in causing prematurity than there are in causing slow fetal growth, and adverse selection is only one of the possible factors, then we might expect a smaller effect of adverse selection on prematurity than on slow growth. Finally, the interaction coefficients in the third and last columns indicate that the effect of smoking is significantly greater for the 2000 cohort than for the 1958 cohort, but is not significantly different for the 1970 cohort.

At the bottom of each column, I report p-values from tests of joint significance of each set of the control variables. I report the coefficient on child’s gender and mother’s marital status at birth as well. For all controls, except mother’s marital status and mother’s social status (in a few instances), the controls are statistically significant predictors of the birth outcomes.

In sum, the coefficients in this table suggest that the effect of prenatal smoking in 2000 on low birth weight is over 50 percent greater than in 1958 and is approximately double with respect to the probability of a low birth weight birth conditional on gestation. Thus, approximately half of the estimated association between prenatal smoking and low birth weight for gestation using the most recent British cohort is attributable to unobserved characteristics of the mother which are correlated with prenatal smoking.⁵ This result is consistent with Rosenzweig and Wolpin (1991), Abrevaya (2006), and Brachet (2005), who

all find selection plays a significant role in the estimated effect of prenatal smoking on birth weight.

To provide a better sense of the magnitudes involved, I make comparisons to three sets of widely cited statistics reported in the 1983 U.S. Surgeon General's Report regarding the consequences of smoking during pregnancy (U.S. Office of the Surgeon General, 1983). First, the Report states that the risk of low birth weight is doubled for pregnant smokers compared to non-smokers. Consistent with this, in the first panel of Table 3, it appears that in 1970 and 2000, the percent of low birth weight babies born to smokers was double (97–149% greater than) the percentage born to non-smokers. By comparison, in 1958, a smoking mother faced only a 72% greater probability of having a low birth weight baby compared to a non-smoker.

Second, the Report states that infants born to mothers who smoked during pregnancy are on average 200 grams lighter than infants born to non-smokers. I found this to be roughly true for the 1970 sample, as shown in the middle panel of Table 3. The difference is even higher — 261 grams lighter — for the 2000 cohort, but only 161 grams lighter for the 1958 sample. Consistent with this value, the estimated effect of prenatal smoking on birth weight adjusted for selection found by Rosenzweig and Wolpin (1991), Abrevaya (2006), and Brachet (2005) is around 150 grams.

Finally, the Report states that about 20% of all low birth weight babies are attributable to smoking. To replicate this statistic, I predict the percent of low birth weight babies assuming that no mothers smoked during pregnancy, holding all else constant, and compare this number to the actual percent of low birth weight babies in the last panel of Table 3. Slightly higher than the Surgeon General's Report, in 1970 and 2000, the percent of low birth weight babies that would have been avoided if no mothers smoked would have been between 24.4 and 26.4 percent. In contrast, in 1958, the percent avoided would have been only 18.5 percent.

The statistics from the 1958 cohort indicate that while smoking during pregnancy has a large impact on birth weight, more current statistics exaggerate the impact of smoking. That is, if a pregnant woman today ceased smoking, it is unlikely the impact would be to cut in half the probability that she delivers a low birth weight baby or to increase the birth weight of the baby by a full 200 grams. Other characteristics of the mother which are correlated with smoking would not have changed and thus would still have an impact on the birth. Overall, these findings suggest that a portion of the effect is causal since there is a large and significant effect of smoking on outcomes in 1958, and suggests that selection contributes importantly to the measured effect of prenatal smoking in the more recent cohorts.

5 Other Possible Explanations

A lot has changed between 1958 and 2001. One important example is the price of cigarettes is substantially higher in 2000 than in 1958. This price increase reduces the demand for cigarettes such that only those individuals with a strong preference for smoking continue or take up the habit. Thus, this change across the cohorts is incorporated in this analysis as it contributes to increasing selection into smoking. However, there are four changes, unrelated to adverse selection into smoking, which could cause an increase in the effect size of smoking over time.⁶

⁵This result is consistent with Meera (2001), who finds that smoking explains half of the correlation between socioeconomic status and low birth weight for white mothers in the U.S.

The first two involve changes in the composition of births. First, the composition of babies born to smokers may have worsened because of improvements in medical technology which increase the chances that the sickest babies survive. Second, the composition of babies born to non-smokers may have improved because of the availability of birth control and abortions. In either case, the effect size of prenatal smoking on birth outcomes could have increased, holding selection into smoking constant.

Two other trends which may explain the increase in effect size across time are stigma and consumption. First, if mothers with higher education and incomes – mothers who ‘should know better’ in 2000 – are more likely to misreport smoking due to stigma, then the effect of smoking will appear larger merely because only less educated, poorer mothers admit to smoking. Second, the effects of smoking would be worse in 2000 if the consumption per smoker or the intensity of smoking increased. I evaluate the potential of each of these alternative explanations below.

5.1 Medical technology

If medical technology has increased the chances that the sickest babies survive, the estimated impact of selection could be biased upwards. Statistics on the percent of stillbirths is available for the 1958 and 1970 cohorts.⁷ There is nearly a 40% drop in the percent of deliveries that result in stillbirth between 1958 and 1970 – from 2.2% to 1.4%. Thus, prenatal smoking may be more likely to result in stillbirth in 1958 than in 1970, so it may look like smoking is worse for outcomes among a sample of live births in 1970 than in 1958. That is, the ‘true’ effect of prenatal smoking on live births may have actually increased across the cohorts as the composition of births includes lower and lower birth weight babies. This would imply that the 1958 estimated effect of smoking (β_1) is less than the ‘true’ effect in 2000 because only the heaviest low birth weight births actually occurred in 1958. Thus, the coefficients on the interactions between prenatal smoking and the later cohorts (β_2 and β_3) could be capturing some of the ‘true’ effect of prenatal smoking as the effect increases across cohorts, and not just the effects of adverse selection into smoking.

Only a small number of pregnancies end in stillbirth, so it may be the case that the compositional change is small enough to not have a noticeable effect on this analysis. However, I conduct two simple exercises to determine the impact of this compositional change in live births due to improvements in medical technology. First, I create an outcome measure which captures fetal death as well as birth outcomes for live births, and thus is not subject to this compositional change. Because data is not available on stillbirth for the MCS cohort, I can only conduct this analysis using the 1958 and 1970 cohorts. This new outcome measure takes a value of 1 if this was a stillbirth, a low birth weight birth, or a premature birth, and 0 otherwise. Consistent with the overall improvement in health due to technology, there are slightly fewer problematic births (stillbirths or poor living outcomes) in 1970 than in 1958 – 9.3% in 1970 compared to 9.6% in 1958.

Table 4 applies the probit model to equation (1) omitting the 2000 cohort, using the new outcome measure described above as the dependent variable. The effect of prenatal smoking in 1970 on having ‘any problem’ is positive and marginally significant. This suggests that

⁶Another possibility not investigated here is that the effect of smoking varies by mother. Adverse selection implies a specific group of mothers smokes; if the effect of smoking is greater for this group of mothers than for the average mother, then the causal effect of smoking will be different in 2000 than in 1958 because the group of mothers is different. The important concern is that we cannot infer that the 1958 effect is closer to the true causal effect than the 2000 estimate because if a pregnant woman in 2000 quit smoking, the benefit to her baby could be greater than the benefit to a baby born in 1958.

⁷The Millennium Cohort sample is designed to include only live births (the first interview occurs when the infant is around 9 months old).

the compositional change in live births due to medical technology is not the reason for the positive coefficients since this outcome is not subject to the compositional change.

The second test of the impact of medical improvements I conduct involves censoring the births that would have been on the margin of survival in 1958. In particular, I drop all births that were very low birth weight, defined as less than 1500 grams (or 3.3 pounds). In this case, the magnitudes and significance of the coefficients on the interaction terms are virtually the same as for the full sample. The results are also the same if I restrict the sample to all births that occurred after 34 weeks of pregnancy. In both sets of tests, the coefficients on *Smoked*1970* are close to zero and mostly not significant just as in Table 2. These tests suggest that compositional changes caused by improvements in medical technology do not have a large effect on this measurement of the impact of selection.

5.2 Birth control pill and abortion

Compositional changes among births to non-smokers could also cause this approach to overstate the importance of selection. If births to non-smokers are a smaller, more select group in 2000 than in 1958 because of the availability of birth control pills and abortion, then smoking could appear to be worse for babies merely because the comparison group has gotten better over time.

Birth control pills and abortions are particularly relevant for this time frame because the prevalence of both grew steadily after 1958. The 1967 Abortion Act allowed a woman in Great Britain to receive an abortion on the grounds of a greater number of considerations; previously abortions were only permitted in situations where the mother's health was at stake. As a result, between 1969 and 2000, the rate of abortions increased from approximately 5 abortions per 1000 women between the ages of 15 and 44 to 16 abortions per 1000 women (Watmore, 2001). In addition, the first birth control pill was introduced in the U.K. in 1961. Among ever-married women between the ages of 16 and 40, 13 percent reported using the pill in 1967 while 29 percent used the pill in 1983 (Murphy, 1993).

If these changes in the control that women have over pregnancy are more likely to affect non-smoking women than smoking women, then the difference between the birth outcomes of these two groups will widen. However, I argue that it is not obvious that the availability of abortion and birth control pills should have a greater effect on one group or the other. Women who have a higher cost of children are more likely to take advantage of the availability of abortion and birth control pills. The cost of children is higher for young, single women, who are more likely to be smokers; it is also higher for educated working women, who are more likely to be non-smokers.

Given that I have samples of women who chose to have a birth, I cannot examine the characteristics of the women who use birth control pills or abortion to prevent births using these cohorts. However, I can use another data source to determine whether the rates of abortion and birth control pill use are different by smoking status. The National Survey of Family Growth (NSFG) is a nationally representative, cross-sectional survey of American women between the ages of 15 and 44 which includes questions about contraception and fertility. There have been six rounds of the survey conducted between 1973 and 2002. I have chosen to use the 1995 survey to characterize the rates of abortion and pill use among women because it contains the largest sample of women among the more recent waves.

To make the sample comparable to the British Cohorts in terms of age and race, I restrict the sample to white women who had a pregnancy between the ages of 17 and 41. Thus, I observe 13,726 pregnancies of 4,935 women. Of the recorded pregnancies, 12 percent had ended in abortion. Thirty-eight percent of the pregnancies were to women who were

smokers at around the time they were pregnant (although they did not necessarily smoke while pregnant). Among those pregnancies where the woman was a smoker, 16 percent ended in an induced abortion compared to 9 percent if the woman was a non-smoker. Thus, smoking appears to be related to abortion. However, since smokers are more likely to avoid unwanted births through an abortion, this should, if anything, bias down the estimated effect of prenatal smoking in the later cohorts. That is, smokers may be trying to avoid adverse birth outcomes through selective abortion which would make the resulting births appear healthier in the later cohorts.

Turning now to birth control pill usage, 88 percent of women in the sample had used the pill at some point in time although only 12 percent were taking the pill at the time of the interview. Among those women who smoked at the time of the 1995 survey, 8 percent were on the pill compared to 13 percent of current non-smokers. Unlike in the case of abortion, this difference could bias up the estimated effect of prenatal smoking in the later cohorts as non-smokers are more likely to avoid unintended births which could result in a healthier comparison group.

To determine whether these differences in abortion and birth control pill usage by smoking status matter, I estimate the effects of mother characteristics on the probability of having an abortion and taking the pill using the NSFG. Then, I censor those mothers most likely to have an abortion and take the pill from the British Cohorts and re-estimate the effects of prenatal smoking on outcomes.

Table 5 shows the results from regressing age, marital status, and education of the mother on abortion and pill usage. In the first column, only age at pregnancy and year of pregnancy are included in the specification. Year of pregnancy is included as a control because the probability of abortion declines across time for this sample. This specification reveals that the youngest age group – those between the ages of 17 and 19 at the time of pregnancy, which is the omitted group – is the most likely to have an abortion, as is the common perception. However, age is highly correlated with education and marital status. When these variables are included in column (2), the effect of age disappears for the younger groups and reverses for the oldest age group – pregnant women between the ages of 35 and 41 are more likely to have an abortion holding education and marital status constant compared to younger women. Marital status is the strongest correlate of abortion, and the probability of abortion increases with mother's education at pregnancy.⁸ In the last two columns of Table 5, we see that education is the most important predictor of taking the pill – the probability of taking the pill at the time of the interview increases with education. Unlike for abortion, marital status has no significant effect on taking the pill and pill usage declines with age, holding all else constant.

Taking this information into account, I restrict the British cohorts sample in two ways. First, I omit all unmarried women, all women who left school after age 16, and all women above the age of 34 at pregnancy. The remaining sample should have a lower probability of abortion than the original sample. In the NSFG, the percent of pregnancies ending in abortion is only 5 percent for a sample with these characteristics, compared to 17 percent for mothers who do not meet these criteria. I do not show the results using this censored sample because they are similar to those shown in Table 2, which uses the full sample. In particular, the coefficients on the interaction between smoked and the 2000 cohort are the same magnitude (0.018 instead of 0.019 for the dichotomous dependent variable and -0.019 instead of -0.018 for the continuous variable, and significant when the dependent variable is

⁸Mother's education at pregnancy is an approximation based on questions about date of school completion, date of pregnancy, and completed education at the time of the interview.

continuous, for birth weight conditional on gestation, indicating that the effect of prenatal smoking in 2000 is larger than the effect in 1958.

The second way in which I restrict the sample is by omitting all women who left school after age 16 and all women under the age of 25 at pregnancy. In this case, the remaining sample should have a lower probability of taking birth control pills than the original sample. In the NSFG, the percent of women with these characteristics taking the pill is 9 percent, compared to 12 percent among those not meeting these criteria. I do not show the results using this censored sample because the coefficients on the interaction between smoking and the 2000 cohort are very close in sign, size, and significance to those in Table 2. Specifically, the coefficient on *Smoked*2000* is 0.022 instead of 0.019 with respect to low birth weight conditional on gestation and -0.014 instead of -0.18 with respect to log birth weight conditional on gestation.

In sum, the effect of prenatal smoking on gestation-adjusted low birth weight in 2000 is roughly twice the size of the effect in 1958, even for samples of mothers who are expected to be less affected by changes in the prevalence of abortion and birth control pills. These restricted samples are about half the size of the full sample and yet the results remained largely unchanged. These tests suggest that compositional changes in the births that occur because of the increased availability of abortions and birth control pills do not have an effect on this estimate of the impact of selection.

5.3 Stigma

With the spread of knowledge about the harms of smoking, social stigma attached to smoking while pregnant likely increased as well. Stigma may decrease the probability that mothers report smoking while pregnant when asked in a survey. The misreporting of smoking status has been studied by comparing self-reports to measured cotinine concentration levels (a marker for nicotine consumption levels) taken from saliva, urine, or blood samples. The findings from the studies with the largest sample sizes (above 400 mothers) indicate that misreporting is a large problem (Bardy et al., 1993; Boyd, Windsor, Perkins, & Lowe, 1998; Lindqvist, Lendahls, Tollbom, Aberg, & Hakansson, 2002). The one large study that finds little difference between self-reports and cotinine concentration is based on data from 1959–1966 (Klebanoff, Levine, Clemens, DerSimonian, & Wilkins, 1998). Since all of the other studies use data from the 1980s and 1990s, this is consistent with an increase in misreporting over time.

An increase in misreporting, if distributed evenly across all mothers, is just a case of increased classical measurement error, resulting in attenuation bias. Thus, if anything, this would bias down the effect of smoking in 2000. However, it is likely that misreporting is not evenly distributed across mothers. If it is the case that mothers with high social status and access to resources are more likely to underreport their smoking status in 2000, then the increased adverse selection observed in 2000 is exaggerated. That is, the effect of smoking in 2000 could be larger than the effect in 1958 because only the mothers with the lowest access to resources, and thus the worst outcomes, are admitting that they smoke in 2000.

The studies listed above do not do much by way of examining misreporting among subgroups of mothers. However, in a study of smoking in the general population, Adda and Cornaglia (2004) do examine the individual characteristics associated with misreporting. They find that for the U.K., using the Health Survey for England 1993–2001, misreporting is more common among individuals between the ages of 20 and 25 than older individuals, and among individuals with low education or in a manual occupation. If this pattern holds for pregnant women, then misreporting would not upward bias the effect of smoking in 2000.

5.4 Changes in Cigarettes and Smoking

Finally, the effect of smoking on birth outcomes could increase if either cigarettes became worse for smokers over time or if smokers increased their daily consumption of cigarettes or intensity of smoking over time. With the introduction of filters and low tar/nicotine cigarettes between 1950 and 1975, it seems unlikely, holding all else constant, that cigarettes became more harmful between 1958 and 2000. The U.S. Federal Trade Commission machine-measures the tar and nicotine output of cigarettes. Using these measurements, the average tar yields of U.S. cigarettes decreased by 44 percent and average nicotine yields decreased by 34 percent between 1968 and 2000 (U.S. Department of Health and Human Services, 2001).

However, it is argued that these lower yield cigarettes and higher prices affected the behavior of smokers, who cover the vent holes intended to reduce the intake of harmful substances or increase their consumption of cigarettes to compensate for the lower yields (U.S. Department of Health and Human Services, 2001; Adda & Cornaglia, 2006). Because the questions about the number of cigarettes smoked are not asked consistently across the British cohorts, I cannot make comparisons directly with the data. However, Wald and Nicolaides-Bouman (1991) report that the number of cigarettes smoked by women in the U.K. increased from the 1920s until the mid-1970s, where it peaked at 7 cigarettes per day. Adda and Cornaglia (2006) find that when cigarette taxes rise, the number of cigarettes fall but smoking intensity rises to compensate such that nicotine levels are the same. Thus, while it may be that smokers in 2000 consume fewer cigarettes than smokers in 1958, their intake of the harmful substances may be the same.

Adda and Cornaglia (2006) also find that individuals with lower economic status may be more likely to smoke intensively.⁹ Because of increasing adverse selection across the cohorts, this finding suggests that smokers in 2000 smoked more intensively than smokers in 1958 on average. Hence, the larger effect of smoking for the 2000 cohort may reflect a greater causal effect of smoking in 2000 derived from a larger amount of harmful substances consumed among smokers. Because smoking intensity differences cannot be observed across cohorts, the hypothesis that women in 2000 smoked more intensively, on average, than in 1958, which potentially caused the contrast in effect size across cohorts, cannot be tested directly. However, to test it indirectly, I restrict the sample to those mothers who have low economic status (defined by the baby's father having a social class of semi-skilled or unskilled worker at the time of birth or there was no father present at the time of birth). These mothers are expected to be more likely to smoke and to smoke intensively in any cohort. If smoking intensity was driving the results, then higher prices in 2000 should lead to a bigger than average effect-size difference among low economic status women who would be more sensitive to price. However, the results are virtually the same as those presented in Table 2. Specifically, with respect to low birth weight adjusted for gestation, the coefficient on the interaction between smoking and the 2000 cohort is 0.020 instead of 0.019. Similarly, with respect to the continuous dependent variable log birth weight adjusted for gestation, the coefficient is -0.014 instead of -0.018. The sub-sample coefficients are significant at the 10 percent level ($n=11,691$).

Conversely, I restrict the sample to those mothers who have high economic status (defined by the baby's father having a social class of professional, managerial, non-manual or manual skilled worker). These mothers are expected to be the least likely to smoke or to smoke intensively in any cohort. If smoking intensity was driving the results, then higher prices in 2000 should lead to a smaller than average effect-size difference among high economic

⁹They also find differences by race, but these results are not relevant to this study as the sample is restricted to white mothers only.

status women who would be less sensitive to price. However, again, the results are the same as those presented in Table 2. In particular, the coefficient on *Smoked*2000* is 0.015 instead of 0.019 when the dependent variable is low birth weight conditional on gestation, and -0.017 instead of -0.018 when the dependent variable is log birth weight conditional on gestation. The coefficients are significant at the 5 percent level ($n=28,193$). From these tests, I argue that it does not appear that the change in the average smoking intensity of smokers is driving the results since the results are unchanged even among those mothers whose smoking intensity should be high in any cohort and among those mothers whose smoking intensity should be low in any cohort.

Thus, I argue that there was probably little change in the average intake of tar and nicotine between 1958 and 2000 among smokers, and any changes in average smoking intensity are not likely to have doubled the estimated effect of prenatal smoking on birth outcomes over this period.

6 Conclusion

This paper uses variation in knowledge about the detrimental birth health effects of prenatal smoking to identify the causal effect of smoking during pregnancy on birth outcomes. The results indicate that the effect of prenatal smoking on low birth weight is significantly smaller in 1958 than in 2000. I argue that this is the result of rising adverse selection into smoking across the cohorts.

I consider several other potential explanations for this increase in effect size. First, an increase in effect size could result if improvements in medical technology reduced the average health of babies born to smokers by increasing the survival rates of high-risk births. Second, the effect of prenatal smoking would be greater if the increased availability of abortions and birth control pills improved the average health of babies born to non-smokers compared to smokers. I discuss evidence using censored samples in Section 5 which suggests that neither of these compositional changes explain the increase in the effect size of prenatal smoking. Finally, I discuss and dismiss the possibility that selective misreporting due to stigma or that increased smoking consumption or intensity over time caused the increase in effect size.

Thus, the results of this study are consistent with the theory that smoking during pregnancy has a causal effect on birth weight conditional on gestation. However, the magnitude of the effect is substantially lower than if smoking were assumed to be exogenous. The implication is that policy interventions to discourage smoking among pregnant women may not prevent as many unhealthy births as usually reported, which in turn affects cost-benefit calculations potentially used for policy decision-making.

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Figure 1. Prenatal Smoking by Cohort and Mother's Age

Figure 2. Prenatal Smoking by Cohort and Mother's Education

Note: The bars display the distribution of mothers across the categories and correspond to the left axis. The lines show the percent who smoked by category and correspond to the right axis.

Figure 3. Prenatal Smoking by Cohort and Father's Social Class

Figure 4. Prenatal Smoking by Cohort and Mother's Social Class

Note: The bars display the distribution of mothers across the categories and correspond to the left axis. The lines show the percent who smoked by category and correspond to the right axis.

Table 1

Descriptive Statistics

	1958	1970	2000
Percent who Smoked during Pregnancy	33.7%	42.1%	23.1%
Percent Low Birth Weight (<2500 g)	6.4%	6.8%	5.5%
Percent Premature (<37 weeks)	5.4%	5.2%	7.0%
Percent Male	51.6%	51.8%	51.4%
Percent Married at Birth	96.4%	92.8%	60.0%
First Prenatal Visit <12 Weeks	20.3%	25.5%	40.6%
First Prenatal Visit 12–23 Weeks	60.9%	56.8%	50.8%
First Prenatal Visit 23+ Weeks	15.7%	14.7%	1.3%
No Prenatal Visits	0.6%	0.9%	2.3%
First Born Child	37.0%	37.9%	43.4%
Second Born Child	30.9%	47.6%	37.0%
Third+ Born Child	32.2%	29.6%	19.7%
Observations	16,549	15,750	13,101

Table 2

The Effect of Prenatal Smoking on Birth Outcomes

Dependent Variable:	(1) Low Birth Weight	(2) Pre- mature	(3) Low Birth Weight	(4) Log Birth Weight	(5) Gestation	(6) Log Birth Weight
Gestation	-0.020** (0.001)			0.056** (0.001)		
Smoked	0.018** (0.003)	0.017** (0.004)	0.033** (0.004)	-0.044** (0.003)	-0.164** (0.036)	-0.053** (0.004)
Smoked*1970	0.005 (0.004)	0.002 (0.006)	0.006 (0.006)	-0.007+ (0.004)	0.004 (0.052)	-0.007 (0.005)
Smoked*2000	0.019** (0.006)	-0.008 (0.006)	0.017* (0.007)	-0.018** (0.005)	-0.026 (0.061)	-0.019** (0.006)
1970	-0.001 (0.003)	0.013** (0.005)	0.008+ (0.004)	-0.001 (0.003)	-0.115** (0.032)	-0.008* (0.003)
2000	-0.013** (0.003)	0.040** (0.006)	0.008 (0.005)	0.043** (0.003)	-0.513** (0.040)	0.014** (0.004)
Male	-0.011** (0.002)	0.010** (0.002)	-0.009** (0.002)	0.042** (0.002)	-0.091** (0.021)	0.037** (0.002)
Mother married at birth	-0.001 (0.003)	-0.004 (0.004)	-0.005 (0.005)	0.006* (0.003)	0.027 (0.041)	0.008* (0.004)
Constant				7.281** (0.017)	13.677** (0.181)	8.044** (0.017)
Observations	39884	39884	39884	39884	39884	39884
Joint Tests: (p-values)						
Smoked during pregnancy	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Mother's age at birth	0.0146	0.0000	0.0033	0.0268	0.0000	0.0105
Mother's school leaving age	0.0059	0.0000	0.0000	0.0000	0.0000	0.0000
Father's social class at birth	0.0302	0.0018	0.0013	0.0000	0.1211	0.0000
Mother's social class at birth	0.2681	0.4408	0.0166	0.1608	0.1833	0.0921
Timing of first prenatal visit	0.0598	0.0000	0.0015	0.0002	0.0000	0.0007
Parity	0.0000	0.0003	0.0000	0.0000	0.0038	0.0000

Note: The first three columns are probits with marginal effects reported. The last three columns report ordinary least squares coefficients. Standard errors in parentheses.

+ significant at 10%;
 * significant at 5%;
 ** significant at 1%

Table 3

Magnitudes

	1958	1970	2000
% LBW if non-smoker	5.0%	4.4%	4.1%
% LBW if smoker	8.6%	8.6%	10.2%
% greater if smoker	172%	197%	249%
Mean bwt if non-smoker	3380	3393	3464
Mean bwt if smoker	3219	3202	3203
Difference	161	191	261
% LBW	6.2%	6.1%	5.7%
% LBW if no smokers	5.1%	4.5%	4.3%
% LBW because of smoking	18.5%	26.4%	24.4%

Table 4

The effect of prenatal smoking on LBW, Prematurity, and Stillbirth

Dependent Variable:	Any Problem
Smoked	0.035** (0.005)
Smoked*1970	0.012+ (0.007)
1970	0.013* (0.005)
Male	-0.004 (0.003)
Mother married at birth	-0.000 (0.010)
Observations	29195
Joint Tests: (p-values)	
Smoked during pregnancy	0.0000
Mother's age at birth	0.0000
Mother's school leaving age	0.0000
Father's social class at birth	0.0000
Mother's social class at birth	0.0495
Timing of first prenatal visit	0.0000
Parity	0.0000

Note: Probits. Marginal effects reported. Standard errors in parentheses.

+ significant at 10%;

* significant at 5%;

** significant at 1%

Table 5

Characteristics associated with Abortion and Birth Control Pills, 1995 NSFG

Dependent Variable:	Pregnancy ended by induced abortion		Method of contraception is birth control pill	
Age 20–24	–0.067** (0.007)	–0.009 (0.007)	0.047 (0.044)	0.032 (0.042)
Age 25–29	–0.105** (0.007)	–0.006 (0.009)	0.018 (0.038)	–0.005 (0.035)
Age 30–34	–0.106** (0.006)	–0.006 (0.012)	–0.015 (0.033)	–0.036 (0.032)
Age 35–41	–0.079** (0.007)	0.044* (0.022)	–0.159** (0.036)	–0.186** (0.037)
Married		–0.327** (0.014)		–0.017 (0.011)
High school graduate		0.024** (0.009)		0.041** (0.015)
Some college		0.053** (0.014)		0.073** (0.018)
College graduate		0.046* (0.018)		0.105** (0.024)
College ⁺		0.079** (0.023)		0.117** (0.029)
Year of pregnancy	0.000 (0.001)	–0.003** (0.001)		
Observations	13726	12791	4935	4935

Note: Probits. Marginal effects reported. The independent variables are observed at the time of pregnancy in the first two columns and at the time of the 1995 interview in the last two columns. Standard errors in parentheses; standard errors are adjusted for intra-cluster correlations at the individual level in the first two columns.

⁺ significant at 10%;

* significant at 5%;

** significant at 1%.