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WHY IS HEALTH RELATED TO SOCIOECONOMIC STATUS? THE CASE OF PREGNANCY AND LOW BIRTH WEIGHT

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Working Paper 8231
http://www.nber.org/papers/w8231

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
April 2001

I am grateful to the National Institute on Aging and the National Bureau of Economic Research for financial support, to Stanley Henshaw of the Alan Guttmacher Institute for provision of data on abortion providers, and to Doug Staiger for kindly sharing data on abortion legislation. I would like to thank seminar participants around the country for helpful comments. I have greatly benefited from discussions with Dora Costa, David Cutler, Martin Feldstein, Sherry Glied, Larry Katz, Erzo Luttmer, Brigitte Madrian, David Meltzer, Katie Merrell, Elizabeth Richardson, Jeff Wurgler and Katia Zhuravskaya. All errors are my own. The views expressed herein are those of the author and not necessarily those of the National Bureau of Economic Research.

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NBER Working Paper No. 8231
April 2001
JEL No. I1

ABSTRACT

There are striking disparities in morbidity and mortality by socioeconomic status (SES) within the United States. I examine pregnancy and health at birth to investigate possible mechanisms linking SES and health. I find that a limited set of maternal health habits during pregnancy, particularly smoking habits, can explain about half (one third) of the correlation between SES and low birth weight among white (black) mothers. I show evidence on three hypotheses to explain why health habits vary by SES. First, differences in knowledge by SES create only modest differences in health behaviors by SES, explaining about 10 percent of differential smoking by education. Second, women respond to common knowledge differentially by SES, so that knowledge and its use combined explain up to one third of differential smoking by education. Third, the most important determinants of differential health behavior are "third variables," or variables that can simultaneously determine health habits and SES. Finally, I show evidence that network effects at the family level exacerbate differences in behavior regardless of the source.

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There are striking disparities in health by socioeconomic status (SES) in the United States and throughout the world. For example, in the United States, babies born to women over age 20 without a high school diploma are 90 percent more likely to die before their first birthday than babies born to women who graduated from college (National Center for Health Statistics 1998). Among adults aged 25-64, those with incomes below $10,000 are twice as likely to die from heart disease as those with incomes of $15,000 or more (National Center for Health Statistics, 1998). These disparities in health by SES are not unique.

Indeed, the relationship between health and SES has been documented for virtually all measures of health and SES, within many countries and over time. Blue-collar workers are more likely than white-collar workers to die from 80 percent of the 80 most common causes of death (Wilkinson 1996). Similar patterns hold true for other measures of SES such as income, wealth, and education.[1] The link between health and SES has been observed in myriad countries including Canada, the United Kingdom, The Netherlands, Sweden, France, and others.[2] In addition, the relationship between SES and health has been documented repeatedly over time. Adler et al. (1993) cite evidence that wealthy Rhode Island taxpayers in the 1860s had mortality rates less than half the population average. Preston (1996) describes how class differences in child mortality widened following intense public health campaigns to improve hygiene at the turn of the century. Although differences in health by SES narrowed in the first half of this century, recent evidence suggests these health disparities are widening (Marmot et al. 1991, Preston 1996).

Policy goals in the U.S. demonstrate a strong desire to eliminate such disparities. In its *Healthy People 2010*, the Public Health Service proclaims as one of two major goals, "to eliminate health disparities among different segments of the population." The U.S. spends over $180 billion annually on Medicaid to improve the health of the poor. But, is Medicaid spending

[1] See Menchik 1993, Marmot et al. 1984, 1987, and 1991, Townsend et al., 1988.

the most effective way to reduce disparities in health? Despite the pervasiveness of the disparities in health by SES, we know little about the channels linking SES and health.

I briefly review related literature in section I. The remainder of the paper does three things. First, it confirms the relationship between SES and infant health measured by low birth weight in section II. Low birth weight is highly correlated with measures of maternal education and household income. Second, section III compares the relative importance of four factors in creating disparities in infant health by SES: maternal health habits, demographics, access to medical care, and pre-pregnancy maternal health status. The results suggest that a limited set of maternal health habits during pregnancy, particularly smoking habits, explain much of the correlation between SES and infant health measures. Among white women, smoking alone can explain about 46 percent of the difference in low birth weight by maternal education. In contrast, controlling for differential access to medical care and differences in pre-pregnancy maternal health status has no impact on differential infant health outcomes by SES.

Because health habits drive much of the correlation between SES and health, section IV asks why health habits like smoking vary by education and income. Using a rich array of data sets I test the following hypotheses: 1) health knowledge drives health behavior and thus creates disparities by SES (Grossman 1972, and Kenkel 1991), 2) the use of common health knowledge varies by SES (Grossman 1972, and Kenkel 1991), and 3) "third variables" such as time preferences determine both health and SES (Fuchs 1982, Barsky et al. 1997). The paper also presents evidence that network effects, or the impact of information and stigma from those living and working near an individual, exacerbate any existing differences in smoking by SES regardless of the explanation.

Based on an analysis of trends in female smoking initiation and cessation following the 1964 Surgeon General's report on smoking, I conclude that "third variables," factors that cause individuals to make differential investments in both health and SES, contribute the most to

[2] See Wolfson et al. 1993, Marmot et al. 1991, Townsend et al. 1988, Kunst et al. 1990, and Feinstein 1993 for discussions of SES and health in other countries.

differential smoking during pregnancy by SES. In the years following public awareness regarding the health risks of smoking, the growth in differences in smoking initiation by age 16 between girls who would later attend college and others was large compared to differential rates of smoking during pregnancy. Differences in smoking initiation can explain 44 percent of smoking disparities by education in a cross-section.

The "third variables" hypothesis is important, but it does not explain all the variation in smoking behavior by education among childbearing women. Based on survey measures of health knowledge and differential rates of quitting smoking in response to the 1964 Surgeon General's Report, I estimate that differential knowledge and differential use of common knowledge can account for up to one third of the difference in smoking by education among pregnant women. Finally, by estimating the response of wives to their husbands' smoking behavior, using only variation from smoking bans at the husband's workplace, I estimate that networks at the family level exacerbate existing disparities in smoking by education level. Section V summarizes the main results and concludes.

I. Related Literature

The literature on health and SES is defined by the fact that the link between SES and health must arise from one or some combination of the following: poor health can lead to low SES, low SES can lead to adverse health outcomes, or a third variable determines both health and SES. The mechanism leading from health to SES is an important component of the correlation between health and SES, as demonstrated by Smith (1999) and Smith and Kington (1997). As Smith and others have noted, the path from health to SES cannot explain the entire relationship. The path from health to SES is not the focus of this paper. The idea that health and SES are related to some third variable has been explored by Farrell and Fuchs (1986) and separately by Fuchs (1982). These authors posit that a "third variable," such as time preferences, self-control, or stress, influences both SES and health investments. In a sample of several thousand men, differential smoking rates by post-high school education were as large at age 17

as at age 24. This implies that the realization of additional schooling did nothing to increase differences in smoking rates. Their results are consistent with the theory that rates of time preference influence health and SES, but Farrell and Fuchs point out that they cannot distinguish this from other "third variable" theories. In separate work, Fuchs (1982) attempted to measure discount rates through phone surveys to examine the link between schooling and health. The implied discount rates from survey responses were inconsistent measures one third of the time, and the relationship between discount rates and health status was often statistically insignificant and small in magnitude.

The bulk of the literature on health and SES discusses the pathway from SES to health. Ettner (1996) suggests that a pathway from SES to health is feasible by showing that differences in income, identified using variation in unemployment rates and parental income, can lead to differences in health outcomes. Ettner provides no evidence of how income affects health, but the most commonly mentioned mechanisms that might lead from SES to health include: access to medical care (Townsend et al. 1988, and Adler et al.1993), ability to collect and use health information to make wise investments (Grossman 1975, Townsend et al. 1988, Kenkel 1991, and Chomitz et al. 1995), unhealthy lifestyle choices of the poor (Adler et al. 1993, Marmot et al. 1984, Lantz et al. 1998), or that low status relative to others has direct and independent health effects (Wilkinson 1996, Marmot et al. 1991, Sapolsky 1993, Deaton and Paxson 1999). These studies focus primarily on older populations and have difficulty showing strong support for any of the above hypotheses, partially because contemporaneous health inputs may be only weakly correlated with health later in life.

Pregnancy and infant health at birth provide an opportunity to evaluate health inputs that have relatively immediate consequences. Many recent studies evaluate programs aimed at improving infant health outcomes among the poor.[3] This literature suggests that certain insurance expansions have successfully lowered adverse outcomes such as infant mortality.

[3] See, for example, Brien and Swann 1998, Joyce 1999, Currie and Gruber, 1996a, 1996b and 1997, Hanratty 1996, and Piper et al. 1990.

Gains do not appear to arise from producing healthier babies, as measured by birth weight, but instead through access to medical care at delivery and during the neonatal period (Piper et al. 1990, Hanratty 1996, Currie and Gruber 1997). Though successful at improving health, intensive medical care at birth is costly, so we have many incentives to understand how to prevent the prevalence of low birth weight and related conditions.

A related literature on the production of infant health emphasizes the relative importance of different health inputs such as access to family planning, access to abortion, prenatal care, and maternal health habits such as smoking in determining infant health.[4] This literature examines neither how these inputs relate to SES nor how they contribute to disparities in health by SES. One exception, a paper by Rosenzweig and Wolpin (1991), concludes that equalizing income would not significantly reduce health disparities by SES. Rosenzweig and Wolpin examine maternal behaviors and their effect on birth weight and gestation using fixed-effect models (within mother). In their model, any time-invariant factor specific to the mother is interpreted as a maternal health endowment. The authors conclude that 90 percent of the variance in birth weight is due to maternal endowments, and therefore other factors such as resource constraints necessarily play a small role in creating health disparities. One might interpret the endowment as reflecting a host of factors besides maternal health which are relatively stable among mothers such as: income, education, the ability to process health knowledge, or time preferences.

This paper differs from past literature in several ways. Unlike most literature on SES and health, it analyzes a situation where health inputs have relatively immediate impact on health, pregnancy and birth weight. It is distinct from work on infant health production functions because it focuses specifically on why health relates to SES using direct measures of income and education. Finally, while evaluations of programs for the poor usually focus on ways to compensate for health disparities by SES, this paper investigates why those disparities arise.

[4] See Joyce 1994, Rosenzweig and Wolpin 1991, Rosenzweig and Schultz 1982 and 1988, Corman, Grossman, and Joyce 1985.

II. Confirming the link between low birth weight and SES

The results reported in sections II and III use the 1988 *National Maternal and Infant Health Survey* (NMIH). The NMIH is a national survey conducted by the National Center for Health Statistics (1991). In the NMIH, women were retrospectively surveyed about pregnancy-related health habits, health care, insurance, living arrangements, and relevant demographic information. The survey includes detailed information from a sample of birth certificates, death certificates, and fetal death records. For comparison to official vital statistics data, I summarize data from the U.S. *Linked Birth/Infant Death Data Set: 1991 Birth Cohort* (National Center for Health Statistics 1995).

Due to dramatic differences in birth outcomes for babies of black mothers compared to babies of white mothers, calculations are done separately for these groups. These differences are explored in a large literature on racial differences in infant health and are beyond the scope of this paper (see for example Lieberman et al. 1987, or Frisbie et al. 1996). Both the NMIH and the vital statistics samples were restricted to singleton births to black or white mothers with complete smoking information. No births from the states of California, Indiana, New York, or South Dakota are included in the vital statistics sample because of the lack of information on the mother's smoking habits during pregnancy. The vital statistics sample includes 2,801,655 births, or about 68 percent of 1991 live births.

The 1988 NMIH sample includes 5,189 white women and 5,315 black women after dropping observations with missing data for the relevant health habits, access variables, or maternal health status. Because the NMIH survey over-sampled infant and fetal deaths and certain demographic groups, all calculations are weighted using the sample weights provided by the National Center for Health Statistics to form population-based estimates.[5]

[5] The weights on infant deaths are adjusted to avoid "double counting" of infant deaths

Tables 1a and 1b show detailed summary statistics for the 1988 NMIH and the 1991 vital statistics data for comparison. About 6 infants die for every 1,000 live births to white mothers and 12-15 infants die for every 1,000 live births to black mothers. Rates of low birth weight (<2500 grams, or 5.5 lbs.) are about 5 percent for whites, and 11-12 percent for blacks. In the NMIH, 19 percent of black mothers and 25 percent of white mothers smoked during pregnancy.

This paper focuses on maternal education and household income as measures of SES. These two measures are highly correlated, and they indicate the mother's access to physical and informational resources available to contribute towards her pregnancy. Because income in particular is likely to be measured with error in a sample of women who recently gave birth, I will consider education and income as measures of the same thing, SES, but I will not attempt to disentangle the effects of education and income. To measure infant health, I focus on low birth weight. Low birth weight babies are 20 times as likely to die as other infants and comprise half of infant deaths despite representing only 7 percent of live births. Surviving low birth weight infants are more likely to suffer long-term health difficulties (Hack et al. 1995). Changes in birth weight higher up in the birth weight distribution are not associated with similar adverse health outcomes.

Low birth weight results from one or a combination of the following factors: a short gestation, also called preterm birth, or retarded growth for a given gestational age, often referred to as intrauterine growth retardation (IUGR). The causes of IUGR are far better understood than causes of preterm birth, although preterm birth is also correlated with many of the same risk factors for IUGR such as maternal cigarette smoking (Kramer 1987, Alexander and Korenbrot 1995). Low birth weight then provides a summary measure of health representing both of these conditions. Although the analysis presented here combines the two causes of low birth weight for ease of exposition, the strongest correlates of low birth weight in my model are those that predict IUGR, consistent with literature suggesting that determinants of preterm birth are poorly

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because the weights in the infant death sample add up to the total infant deaths in 1988, but there are additional infant deaths in the live birth sample.

understood. Extensive research on causes of low birth weight (due to either IUGR or preterm birth) suggests that the most important risk factors for low birth weight in developed countries include cigarette smoking, poor nutrition during pregnancy, and low pre-pregnancy weight (Kramer 1987, Alexander and Korenbrot 1995). Other causal factors related to low birth weight include: birth order, female sex, short maternal stature, maternal low birth weight, alcohol consumption and general maternal morbidity (Kramer 1987). All of these factors fit into the four categories of potential mechanisms linking SES and low birth weight : health habits during pregnancy, demographics, access to medical care, and maternal health prior to pregnancy. Furthermore, the health effects of most of these risk factors are immediate, resulting in low birth weight births. These advantages of studying low birth weight contrast with studies that focus on the diseases of old age, where determinants of ill health accumulate over decades.

Figure 1 shows the strong relationship between my measures of SES and low birth weight. In figure 1a, infants born in households with income below $10,000 are about 3 percentage points more likely to be low birth weight than infants born into households with incomes between $20,000 and $30,000. Figure 1b displays the similarity in NMIH and vital statistics data. In both data sets, babies born to the most educated mothers are about half as likely to be low birth weight as those born to the least educated mothers. Although not shown in this graph, infant mortality is also twice as likely among babies born to the least educated women relative to the most educated.

III. Relating birth weight to SES, health habits, demographics, access and maternal health

This section estimates models of low birth weight to measure how SES affects infant health. I estimate equations of infant health outcomes as a function of SES controlling for four groups of variables: maternal health habits during pregnancy, demographics, access to health care, and maternal health status at the start of the pregnancy. Tables 2 and 3 report the implied

change in the probability of low birth weight based on the following probit model:

$$\Pr\left(Low\ Birth\ Weight\right)_i = F\left(\beta_1\left[Ln\ Hhd\ Income\right]_i + \beta_2{}'\left[Education\right]_i + \Gamma'\mathrm{X}_i\right) \quad (1)$$

In equation 1, F(.) is the cumulative normal distribution, and X_i is a vector of control variables. Income is measured as the natural log of household income in the year preceding delivery controlling for household size with 9 dummy variables.[6] In equation one, *Education* represents a vector of two dummy variables for the highest completed grade, a dummy for mothers with a high school degree, and a dummy for mothers with any education beyond 12 years. This specification was chosen because most mothers have 11-13 years education and additional years of education within the above categories is not associated with a significant marginal health benefit. Models using a linear specification for education (not shown) yield similar qualitative results. The first set of controls in X measure maternal health habits such as smoking, drinking, drug use, and timing of prenatal care.[7] Health habits of the mother during pregnancy are important for knowledge-based theories suggesting that better educated women know more about health and how to produce health efficiently through good habits (Grossman, 1972, Kenkel, 1991). Health habits also help us to learn about "third variables" that may drive investments both in education and good health habits, thus producing healthy babies (Fuchs, 1982, Farrell and Fuchs, 1986). The second set of controls, demographics, include dummy variables for marital status at delivery and infant sex. Male babies are known to be heavier than females at birth and there are important differences in birth outcomes for married and unmarried women. Third, access variables capturing insurance status during the prenatal period, reported

[6]The NMIH reports income in 20 bracketed groups. Thus, I code income as the midpoint of each group and median income for households above $60,000.

[7] Health habit variables include: dummy variables for whether the mother smoked, drank >5 drinks per week after learning of pregnancy, or used cocaine during pregnancy, a dummy variable for whether the mother obtained prenatal care in the first trimester, and a dummy equal to 1 if the mother took at least one of the following vitamins/minerals during pregnancy: multivitamins, vitamin A, vitamin C, folic acid, calcium, iron, and zinc.

barriers to prenatal care, and access to abortion providers are included in the model to address the hypothesis that SES influences health through differential access to medical care.[8] Finally, pre-pregnancy maternal health status variables reflect the fact that differences in infant health by SES may be the result of a health endowment passed on through generations (Rosenzweig and Wolpin 1991, Barker 1992). Maternal health is measured using controls for maternal age, maternal birth weight, interval between live births, infant's birth order and mother's body mass at the start of the pregnancy.[9]

The medical literature suggests substantial benefits from adequate weight gain during pregnancy (about 25-35 pounds for the average women); thus much of the literature on production of birth weight includes weight gain as a control variable (Kramer 1987 and Chomitz et al. 1995). Weight gain during pregnancy is divided among additional fat stores in the mother, the fetus itself (about 25 percent), increased blood volume, the enlarged uterus and breasts, the placenta and amniotic fluid. However, because fat stores provide the primary source of fetal energy the ideal measure of weight gain in a model would net out all other sources of weight gain. The inclusion of birth weight in the total measure of weight gain leads to a mechanical relationship between maternal weight gain and birth weight. For these reasons I choose to exclude weight gain from the model, but I have repeated the analyses in tables 2-4 and table 9

[8] Access variables include: the percent of people uninsured in the state of birth; dummies for whether prenatal care was hard to obtain due to cash problems, transportation/appointment problems, provider problems, or for other reasons; the number of minutes to the prenatal provider; whether prenatal care was paid for by Medicaid, private insurance, own income, or family income (these are not mutually exclusive); average distance to an abortion provider in the state, and dummy variables for whether a state had parental consent laws or restricted Medicaid abortion funding (See for example, Kane and Staiger 1996, Blank et al. 1996). State-level distance to abortion provider is a population-weighted average of county distance to provider using methods from Kane and Staiger (1996), but based on 1990 population centroid. Medicaid laws and parental consent laws were kindly provided by Doug Staiger and follow those used in Blank et al. (1996).

[9] The health status variables include: 10 dummies for maternal birth weight for each 500 gram increment up to 4999 grams, a dummy for over 4999 grams, an omitted category for unknown birth weight, a five-piece spline in maternal age with knots at 20,25,30 and 35, a quadratic term for birth order, a dummy indicating whether mother had a live birth less than 18 months before delivery, and 7 dummy variables for the mother's body mass index (BMI = the ratio of weight in kilograms to height^2 in meters). BMI has been shown to be correlated with mortality rates. Maternal age, birth order, and interval since last live birth can be modeled as choice variables, as in Rosenzweig and Schulz (1982, 1983, and 1988). However, they reflect underlying differences in health status, so it is reasonable to first assess how controlling for maternal health attenuates the correlation between SES and health.

using total weight gain, and found similar results to those presented here.[10]

Table 2 displays how the implied marginal effect of SES on low birth weight changes when I include the four groups of control variables. Each row shows the implied effects of a different model estimated separately for black and white mothers. The first row, *No Controls*, shows the basic correlation between low birth weight and the measures of SES, controlling only for household size. Among babies born to white women, a one standard deviation rise in household income of about $20,000 accompanies a seven percent drop in the probability of delivering a low birth weight baby. A rise in maternal education from 11 to 12 years accompanies a 1.37 percentage point decrease in the probability of low birth weight. Similarly, a move from less than a high school degree to some college accompanies a 2.7 percentage point decrease in the probability of low birth weight. For black mothers, a one standard deviation rise in household income of $16,000 accompanies a four percent drop in low birth weight. Among black mothers, the move from less than high school to a high school degree implies a 1.1 percentage point decrease in the probability of low birth weight while a move from less than high school to some college accompanies a 2.2 percentage point drop in low birth weight.

The second row shows the *Full Model* including SES, maternal health habits, demographics, access variables, and maternal health status. With all the controls, the marginal effect of household income on births to white(black) women becomes insignificant and falls by 60(28) percent compared with the *No Controls* model. The implied effect of maternal education falls by half(three quarters) for white(black) mothers. I thus conclude that income and education impact health mainly through other variables. About half of the effect works through the limited set of variables captured here, suggesting that the direct impact of education and income may be small.

[10] These alternative models yield highly similar results particularly with regard to the magnitude of the impact of smoking on disparities in health by SES presented below. Based on the alternative models with weight gain, it does not appear that smoking affects birth weight through an effect on maternal weight gain, consistent with literature comparing the maternal weight gain distribution of smokers and non-smokers (Meyer 1978). Tables including maternal weight gain in these models are available from the author.

The remaining rows of Table 2 help to determine which variables have the most power in diminishing the correlation between SES and health. Each row shows the impact of removing one set of control variables from the full model. We know that the implied effect of SES virtually disappears in the full model, so if the SES coefficients are significant and large when we omit one set of variables, we can conclude that the omitted variables are important for explaining the relationship between SES and infant health. The results illustrate the importance of health habits as a link between SES and low birth weight. Row 3 shows the full model omitting only the controls for maternal health habits. The marginal impact of college education is statistically significant and large compared with the full model. This is striking because this specification controls for access, maternal health status, and demographics, yet the correlation between education and low birth weight remains. This result suggests that maternal health habits play an important role in explaining differences in infant health by SES. The remaining models omit demographics, access variables, and health status, respectively. For black women, the SES variables are not significant in any of these remaining specifications, and the magnitudes of the implied effects of income and education are small for both the black and white samples, as in the full model.[11] Table 2 implies that understanding the link between health and SES requires one to understand how health habits relate to SES.

[11] The access variables may not adequately capture how abortion access affects socioeconomic disparities in infant health because information on abortion access by state may not be fully informative in a cross-section. As a test of how abortion access might affect SES disparities infant health, I provide evidence on the trend in infant mortality rates for the poorest and richest counties in the U.S. before and after the 1973 legalization of abortion. The advent of legalized abortion does not exacerbate disparities in infant mortality by county median income. Indeed, legalized abortion modestly diminishes the disparity between rich and poor counties, as shown by greater relative declines in mortality for poor counties after 1970.

Infant mortality rate before and after legalized abortion (1973 *Roe v. Wade*)

	Decile of median family income		Annual % change		
Years	Bottom decile	Top decile	Bottom decile	Top decile	Diff in Diff
'56-'60	36.8	22.6			
'66-'70	29.7	18.6	-2.1	-1.9	0.196
'74-'78	19.8	12.8	-5.1	-4.7	0.397
'88-'92	11.6	7.8	-3.8	-3.5	0.281

Source: 1995 Area Resource File. Unit of analysis is county infant mortality rate. Counties in sample include 117 (135) counties in the bottom (top) decile of median family income in county for all decades shown .

Table 3 displays the implied marginal effects of all variables in the full model of low birth weight, and it highlights the effects of maternal health habits by showing specifications with only health habits and SES. The effect of household income and education from the *No Controls* model from table 2 appear in column 1 for comparison. Columns 2 and 6 show the same model adding a dummy for whether the mother smoked during pregnancy. Smoking is strongly correlated with the probability of low birth weight, raising the chance of low birth weight by 4(10) percentage points for white(black) women, an enormous effect relative to the average rates of low birth weight of 5 and 12 percent. Furthermore, adding the smoking variable to the model substantially reduces the impact of income and education. Adding in other maternal health habits, shown in columns 3 and 7, further reduces the correlation between the SES variables and low birth weight. The implied effects from the full model are presented in columns 4 and 8. In these columns, the significant determinants of low birth weight have the expected sign and smoking still exhibits a large independent effect on low birth weight.

The interpretation of results on smoking behavior and health disparities

Tables 2 and 3 show that maternal education and income are strongly correlated with the probability of low birth weight. But, the correlation virtually disappears in a model including controls for health habits, demographics, access to health care, and maternal health status. Most of the relationship between SES and health seems to be driven by maternal health habits. Of these, smoking during pregnancy generates the largest gaps in low birth weight by SES. One may worry that smoking simply marks other traits not captured in the model. It is reassuring that the effect of smoking remains strong and unchanged when adding in other health habits. To offer additional evidence on why the variable, smoked during pregnancy, measures a true impact of smoking on infant health, table 4 compares results using the data presented here to those in the literature that have alternative exogenous measures of smoking behavior. Permutt and Hebel (1989) present evidence using a randomized smoking cessation program for pregnant women as an instrument for smoking during pregnancy. Evans and Ringel (1999) use changes in state cigarette taxes as instrumental variables for smoking during pregnancy in vital statistics data

from 1989 to 1992. Neither of these studies breaks out the impact of smoking by race, so table 4 displays pooled results using my data and coefficients on the smoking variable from OLS models of continuous birth weight to compare results to those shown in the other studies. I also show these results separately by maternal race. The magnitude of the impact of smoking does not differ significantly between my models and those previously estimated. Furthermore, table 4 resolves one puzzle in the results of tables 2 and 3, the differential impact of smoking between black and white mothers. One reason smoking might matter more for the probability of low birth weight among black women is that the black birth weight distribution is shifted slightly downward or to the left of the white birth weight distribution. In this case, a given average reduction in birth weight in grams will lead to a disproportionate increase in low birth weight among blacks. Table 4 confirms that the effect of smoking on continuous birth weight does not differ dramatically between the black and white samples.suggesting that these results measure true biological effects of smoking rather than a simple marker for other maternal characteristics. The findings in this section are important because they suggest that to understand the differences in infant health by SES, we need to understand the factors that link SES to maternal health habits, smoking habits in particular. The rest of the paper therefore focuses on the relationship between female smoking rates and SES.

IV. Hypotheses Explaining the Link Between Health Behaviors and SES

This section shows evidence on several hypotheses linking education to smoking behavior among childbearing age women. I focus on education rather than income for two reasons. First, most plausible explanations for the link between smoking and SES work through education rather than income. While income may be related to smoking indirectly, through a lack of alternative means of pleasure or stress relief, there is no sense in which financial constraints lead women to smoke. In fact, financial constraints reduce access to cigarettes among low-income women. Second, as seen in the models of low birth weight, income in the

year before delivery (the only available income measure) is a weaker predictor of low birth weight than education. This may reflect the fact that income in the 12 months before delivery does not pick up permanent income. Hence, it seems wise to focus on education, where the link between SES, behavior, and infant health is strongest. Evidence presented here on third variables and networks can apply to income as well as education.

I examine three hypotheses relating smoking behavior of childbearing women to education. First, I examine whether education leads to more complete health knowledge, and thus healthier habits. Second, I examine whether more education leads women to use a given level of health knowledge more efficiently. Finally, I examine whether other variables that determine one's education investments also determine investments in health such as smoking behavior. The end of this section presents evidence on how network effects, or the influence of those who live or work with an individual, can exacerbate differences in smoking behavior that occur for any of the above reasons. This is not a distinct hypothesis from the other three, but instead captures how small differences in health knowledge or other variables by SES may lead to dramatic differences in behavior and ultimately health outcomes.

The first two hypotheses on health knowledge and its use are closely related and will be tested in the same analysis.

1. Education leads to more complete health knowledge, and thus healthier habits

The first hypothesis posits that education alters an individual's perception of how health inputs affect health. This hypothesis offers a specific mechanism through which education might improve health as in Grossman (1972) and Kenkel (1991) where education allows individuals to produce health more efficiently. The hypothesis has the clear, testable implication that high SES individuals should have more complete health knowledge, and controlling for differences in health knowledge should reduce differential smoking by SES. A second test of the knowledge

theory is whether differences in health habits by SES group decline as knowledge becomes more widely disseminated.

2. Individuals with more education use knowledge more efficiently

A related hypothesis discussed by Kenkel (1991) and Grossman (1972) is that individuals with higher levels of schooling use knowledge more efficiently in the production of health than less educated individuals. Decisions regarding the tradeoffs between different pregnancy inputs are complex, and because pregnancy is infrequent, there are few opportunities for learning by doing. Those with higher levels of education may be able to synthesize health knowledge in a way that efficiently prioritizes those health investments with the largest benefit. One implication of this theory is that for a given level of health knowledge, more highly educated individuals should be more likely to respond to that knowledge. Furthermore, when new health knowledge emerges, better-educated women would be expected to respond more quickly to it.

Kenkel (1991) tests hypotheses one and two using the 1985 *Health Interview Survey* (HIS) and measures of knowledge on the consequences of smoking, drinking, and lack of exercise on general health. Kenkel finds that the coefficients on education in health behavior equations fall by 5 to 25 percent controlling for these measures of health knowledge. I test this in the case of pregnancy using a similar analysis of the HIS, because the NMIH contains no questions regarding respondents' health knowledge. Ideally one would estimate equations similar to those in tables 2 and 3, but including variables on smoking knowledge. Unfortunately, the HIS Supplements containing information on smoking during pregnancy do not include information on birth outcomes.

The HIS surveys the non-institutionalized U.S. population about general health status, specific health conditions, and demographics. In 1985 and 1990, the *Health Promotion and Disease Prevention Supplements* included questions for female respondents of childbearing age asking whether smoking increased the chances of: miscarriage, stillbirth, premature birth, and

low birth weight. All respondents in the 1985 and 1990 *Health Promotion and Disease Prevention Supplements* were asked whether smoking increases the chance of lung cancer, emphysema, bladder cancer, cancer of the larynx, cancer of the esophagus, or chronic bronchitis. The nature of these questions (all have an affirmative answer) may lead to an overstatement of health knowledge.

In the same years, the *Pregnancy and Smoking Supplements* asked female respondents who had given birth in the 5 years preceding the survey about smoking status during their last pregnancy. Combined with information on income and education, one can test hypotheses 1 and 2, whether health knowledge or one's response to health knowledge can explain differences in health habits by education. The variable "smoking knowledge" indicates the respondent's share of correct responses to the smoking questions described above. I also construct a variable, "drinking knowledge," described in table 5, to include in models of smoking behavior as a test of whether my smoking knowledge variable is an indicator of general health knowledge, or a specific knowledge of tobacco-related consequences.

Table 5 contains summary statistics on the smoking behavior of 6,832 women split into two groups: those who attended college and those with less education. Knowledge measures are significantly higher in the college-educated group compared with other women, but the magnitude of the difference is modest. In contrast, smoking habits vary widely across groups. Only 11 percent of college-educated women smoked while pregnant, compared with 29 percent among other women, an 18 percentage point difference. These rates of smoking mirror smoking rates by education in the NMIH which are 13 and 31 percent respectively.

One may worry that self-reported measures of smoking understate true rates of participation. This problem is likely to be small given that a recent study finds that rates of misclassification of regular female smokers as non-smokers are under 3 percent (Wells et al. 1998). The most serious problem for this type of analysis is one of cognitive dissonance. Individuals may know that smoking is unhealthy, but may smoke anyway. This could lead respondents who know the health risks but who smoke to either understate knowledge or

smoking behavior. One would expect respondents with low levels of knowledge to be more likely to correctly report smoking and knowledge. This systematic problem would bias estimates of the impact of smoking knowledge upward in absolute value, thus overstating the role of health knowledge in determining smoking behavior. In the absence of a powerful instrument for smoking knowledge, one must consider this estimate of the impact of knowledge on smoking as an upper bound.

Table 6 presents the implied marginal effects of each variable based on probit models of smoking behavior. I estimate the following model:

$$\Pr(smoke\ during\ \ pregnancy)_i = F(\alpha + \beta_1'[education]_i + \beta_2[\ln(household\ \ income)]_i + \beta_3[knowledge]_i + \beta_4'[knowledge * education]_i + \Gamma' X_i) \quad (2)$$

In equation two, *education* represents a vector of two dummy variables for the highest completed grade, a dummy for mothers with a high school degree, and a dummy for mothers with any education beyond 12 years. The variables *knowledge* and *household income* are defined as before in the text. To aid the interpretation of coefficients, knowledge and education variables are expressed as deviations from their means. All regressions include a full set of demographic and health-related covariates X.[12] In table 6, knowledge of smoking consequences strongly predicts smoking behavior during pregnancy and explains some of the relationship between SES and smoking. Specification 1 shows that a 10 percent rise in household income accompanies a .42 percentage point reduction in smoking during pregnancy. An increase in maternal education from less than a high school degree to a high school degree coincides with a 13.5 percentage point decrease in the probability of smoking during pregnancy, and a move to some college

[12] Covariates include dummies for: race (black, white, or other), 5-year age groups, year of delivery, Hispanic origin (Multiple Hispanic, Puerto Rican, Cuban, Mexican-Mexican, Mexican-American, Chicano, Other Latin American, other Spanish, Spanish of unknown type, Unknown if Spanish, Not Spanish origin), marital status (married spouse present, married spouse absent, widowed, divorced, separated, never married), respondent's major activity (working, keeping house, going to school, something else), respondent's activity limitations (unable to work, limited in kind/amount of work, limited in other activities, not limited), whether the respondent has a regular source of medical care, whether the respondent had a pap smear in last two years, whether self-reported health is fair/poor, four region dummies, and a dummy for whether stress has affected health in the past year.

accompanies a decrease of 26.8 percentage points. Based on the coefficients in column 2, a 10 percent increase in smoking knowledge coincides with a 3.0 percentage point reduction in smoking during pregnancy. Adding smoking knowledge to the model reduces the effect of household income and education by about 10 percent. Column 3 tests the importance of the hypothesis that people apply knowledge differently when they have more education. Consistent with Kenkel (1991), the impact of knowledge on smoking is significantly larger in magnitude for women as education rises. However, the interaction between knowledge and education does not explain differential smoking by SES because controlling for the differential response to smoking knowledge does not diminish the correlation between SES and smoking relative to column 2. Specifications 4 and 5 include both smoking and drinking knowledge to provide a more complete measure of health knowledge, and to distinguish knowledge about smoking from being generally better informed about health. The results are nearly identical to those in columns 2 and 3. Drinking knowledge does not significantly affect the probability of smoking nor does it explain the relationship between SES and smoking.

As mentioned before, a potential criticism of the knowledge measures for pregnant women is that they may be endogenous to behaviors due to cognitive dissonance. One way to circumvent the endogeneity problem is to examine situations where knowledge differentials by SES are expected to shrink, because we can observe the response to knowledge, and not self-reported knowledge. For example, one important channel of learning for all pregnant women is pregnancy itself. Women may learn the importance of health habits during a first pregnancy and they may use this knowledge during subsequent pregnancies. If one believes that knowledge disparities cause the differential smoking rates for high SES women versus other women, then the knowledge acquired during a first pregnancy offers a chance for low-SES women to "catch up." Consider a high-SES woman who prepares for pregnancy by reading pregnancy-related literature, consulting with her doctor about the timing and early pregnancy behaviors. Her pregnancy-related health knowledge may be more complete than for a less-educated woman as both enter their first pregnancy. While both women are likely to learn about appropriate prenatal

behavior during their first pregnancy, one might expect more of the information to be new for the less-educated woman. Thus, in a sample of women who have had exactly 2 births, one might expect differential smoking rates by education to be smaller for the second births than for first births, or the relative rise in smoking between births should increase with education.

Below, I show results from a test of whether less educated women "catch up" to the smoking behavior of more educated women in their second pregnancy. Using a sample of 2,064 women with exactly two births in the NMIH, I estimate the following model:

$$\Delta(\textit{Smoking status})_i = \underset{[0.046]}{-0.032}(HS)_i - \underset{[0.051]}{0.094}(\textit{College})_i - \underset{[0.014]}{0.022}(\textit{Ln hhd income})_i + \Psi' \mathbf{X}_i \quad (3)$$

where *i* indexes individuals, and $\Delta(\textit{Smoking status})$ reflects the change in each mother's smoking status during pregnancy between the first and second births. The variable $\Delta(\textit{Smoking status})$ takes on a value of 0 if there is no change, -1 if the mother quits smoking (smokes only in first pregnancy), and 1 if the mother initiates smoking (smokes only in her second pregnancy). The SES variables are as defined before in tables 2 and 3, and the matrix *X* includes the same covariates used in the *Full Model* in tables 2 and 3 with the following changes: variables on birth order and smoking are dropped; because the sample pools races, a dummy for black mother is added; and, dummies for year first pregnancy ended are added to X. The coefficient on *Education* indicates that differential smoking rates by education increased between births because more educated women decreased smoking during pregnancy relative to less-educated women. This evidence suggests that the response to health knowledge (hypothesis 2) is more important than the level of knowledge itself (hypothesis 1) in explaining differential smoking during pregnancy. The evidence shown above is consistent with an alternative hypothesis that could explain the differential response to smoking knowledge, the hypothesis that third variables determine both SES and investments in health.

3. *Unobservable variables such as time preferences determine both health habits and SES.*

Fuchs (1982 and 1993) suggests that time preferences determine both health and SES because individuals with low discount rates make larger investments in both education and health. This hypothesis can be distinguished from the previous hypothesis conceptually because it suggests that low SES individuals are making optimal decisions given their constraints. In contrast, hypothesis 2 suggests that individuals make sub-optimal decisions, and that one could raise welfare by improving an individual's ability to choose optimal health inputs, or alternatively by making the optimal decisions for them. Related "third variable" hypotheses that could drive both SES and the choice of health inputs include theories that stress, depression, and traits such as self-control determine SES and individual health habits (Thaler and Shefrin, 1981). If the "third variables" hypothesis can explain differences in health behavior by SES, one should observe differences in behavior that precede differences in knowledge or education among these groups. In other words, one should observe situations where knowledge and education cannot explain observed differences in smoking behavior across SES groups. I compare the relative importance of hypotheses 2 and 3 using a source of an exogenous health knowledge shock, the 1964 Surgeon General's Report.

Public information on the hazards of smoking began with articles in the popular press around 1953, and in 1964, the first highly publicized report of the Surgeon General on smoking was published. Public reports on smoking and health have been published annually since then. In 1966, cigarette-warning labels were first introduced with the message that smoking can be hazardous to one's health, and they were strengthened in 1983 with the *Comprehensive Smoking and Education Act*. The 1983 law changed warning labels to mention specific diseases related to smoking. But differentials in smoking by education were widening at this time.

To see the importance of smoking over time, compare the top and bottom panels in Figure 2. Based on various years of the Health Interview Survey, the top panel, 2a, plots smoking rates among adult women with college degrees compared to women with high school degrees or less. Smoking rates fall within both groups, but much more rapidly among college

graduates, particularly during the late 1970s and early 1980s. The bottom panel, 2b, shows median birth weights for women with college degrees and women with high school degrees. The birth weight distribution for college-educated women moves up more rapidly than it does for less-educated women. This divergence in birth weight is most rapid during the late 1970s and early 1980s, similar to the timing of the divergence in smoking rates. This suggests that the response to health knowledge across groups is very important for explaining differential smoking rates by education. To understand the trend better, I examine female smoking by cohort during the years 1966-1991. By splitting the samples into cohorts, one can distinguish changes in smoking rates that are attributable to quitting behavior and changes in smoking rates that occur because of changes in initiation among younger cohorts. This distinction is important because I want to use changes in quitting behavior following the 1964 Surgeon General's Report to estimate an upper bound on the impact of use of knowledge (hypothesis 2) on differential smoking behavior. As mentioned above, by 1975, about 90 percent of adults reported that smoking was hazardous to their health. This implies that differential quitting behavior across educational groups within a cohort of women is not likely explained by differences in the level of health knowledge. It may be the result of differences in how education helps women to use health knowledge. I call this estimate an upper bound because one could logically argue that factors besides education lead college-educated women to respond differently to health information than less educated women. In contrast, I want to use smoking initiation during high school to form a lower bound estimate of the contribution of other factors besides knowledge and education (hypothesis 3) to differential health habits by SES. As the hazards of smoking become known, any differential in teenage smoking initiation across education groups cannot be attributed to differences in college attendance, and given the nature of smoking information, it is not likely attributable to knowledge of health consequences. This implies that any remaining disparity among teenage girls reflects factors that determine their investments in health as well as investments that can impact SES.

In the top panel of Table 7, I use the 1966 and 1983 Health Interview Surveys to examine smoking cessation behavior by looking at the rate of smoking over time for the cohort of women born between 1917 and 1946. Current smokers in the table are women who report smoking "occasionally" or "regularly." Information on the hazards of smoking became public before 1966, my first year of data, but ideally we want to know how smoking behavior changed before 1966 as well. The 1966 survey asks respondents to categorize their smoking status as current regular, current occasional, former, and never smoked (smoked less than 100 cigarettes). I measure pre-1964 smoking rates as one minus the share of respondents who never smoked. This allows one to include in the estimate of pre-1964 current smokers those women who may have quit smoking in response to health information prior to the 1966 survey. For cohorts of women born between 1917 and 1946, pre-1964 rates of smoking do not vary by education; about half of women in these cohorts smoked prior to 1964. To get differential quitting rates by education, I compare the decline in smoking for college educated women to that for women with a high school degree or less between 1964 and 1983. Using this approach, the top panel of table 7 shows that there was a 6.0 percentage point difference in the relative decline in smoking between college-educated and other women between 1964 and 1983.[13] These differences are statistically significant and present evidence that highly educated women responded more rapidly than less-educated women to the new information provided by the media and the Surgeon General's report. One may interpret this estimate as an upper bound on the contribution of knowledge and its use to the cross-sectional difference in smoking during pregnancy. In other words, knowledge and its use potentially explain 6 percentage points, or about 33 percent of the observed 18-percentage point differential in smoking during pregnancy by education in the HIS and the NMIH. I consider the estimate as an upper bound because I cannot with these data rule out the

[13] This measure is imperfect because behavior may have changed as early as the 1950s when some evidence of negative health effects of smoking was published, but "ever smoked" rates probably do not reflect smoking rates in 1953 since many women in the sample would have been too young to smoke then. I obtain similar results if I limit the sample to women born from 1917 to 1936.

possibility that third variables drive the decision to quit smoking in response to the Surgeon General's Report.

I next examine how initiation of smoking changed following news of smoking hazards. The bottom panel of Table 7 focuses on differential smoking initiation over time, by looking at the share of women who started smoking by age 16. This exercise is similar in spirit to Farrel and Fuchs (1982) and provides information about the importance of "third variables" that may cause SES and health habits like smoking. Differences in smoking at age 16 by education cannot be caused by own college attendance. If one splits the sample into women who eventually will attend college and those who will not, a comparison of the share of women smoking at age 16 before the Surgeon General's report to smoking rates at 16 for those turning 16 in 1983, decades after the report, one can compare the difference in smoking rates by college attendance both before and after health consequences of smoking became known. We can therefore interpret the difference-in-difference estimate of smoking at age 16 as a lower bound on estimates of the share of differences in smoking due to "third variables," or factors besides education that cause girls to smoke. I use the difference-in-difference to purge from my estimates any pre-existing static differences in smoking at age 16 that are not attributable to differential investments in health.[14] For women turning 16 before 1953, the small 2.7-percentage point differential rate of smoking by future education level cannot be explained by a difference in the willingness to trade health benefits for the pleasure of smoking. As shown in Table 8, between 1953 and 1983, the disparity in smoking rates by education grew 7.9 percentage points. If we compare this estimate to the cross-sectional difference in smoking during pregnancy between college-educated and other women, "third variables" can explain at least 44 percent of the 18-percentage point difference.

[14] If trends in teenage smoking differ for reasons other than a response to new smoking knowledge, the difference in difference estimate cannot eliminate smoking disparities related to these non-information related reasons. One may indeed worry that this is the case given the steep rise in teenage smoking from 1953 to 1983 among girls who will not attend college, even in the face of considerable evidence regarding the health hazards of smoking. Even for college- bound girls, smoking at age 16 rises slightly during this period. Trends that have little to do with information such as differential peer effects may also drive differential smoking behavior. In any case, large disparities in smoking rates among 16 year olds relative to disparities among pregnant women indicate that something other than the impact of college education, some "third variable" drives differential smoking by

Taken together, the tests of knowledge and the response to knowledge "shocks" suggest the following. First, differential levels of health knowledge as measured here explain very little of the differences in health behaviors by SES, about 10 percent. Second, because the differences in behavior widen in situations where information diffuses, following a first birth and decades after the Surgeon General's Report, one can conclude that the response to knowledge plays a more important role than knowledge itself in creating differential behavior. If differential rates of quitting smoking following the Surgeon General's report could be attributed entirely to knowledge and its use, these factors would explain up to 33 percent of differential smoking rates between college and less-educated women. Third, although more highly educated women quit smoking faster following the Surgeon General's report, changes in the rates of initiation for women aged 16 or younger contributed more to differential rates of smoking by education, at least 44 percent. I thus conclude that "third variables" are the most important factors creating differential smoking rates by education among childbearing women.

The previous results suggest that "third variables" largely drive differences in health and SES. What are these third variables? One candidate is time preference, which is inherently difficult to measure. Some surveys have attempted to collect information on variables such as discount rates (Fuchs 1982, Barsky et al. 1997), but no reliable, consistent measure of the way people make inter-temporal tradeoffs exists. In other research, Meara (1999) uses proxies for discount rates, self-control, and measures of symptoms of depression to show that these variables are highly correlated with both parental SES and smoking and drug use of adolescent girls.

In particular, variables on depression and religious service attendance are highly correlated with behavior and SES. These results suggest that one should examine alternative hypotheses in more detail. High depression rates among teenage girls from low SES families may lead them to initiate smoking and perform poorly in school. Many of the measures of depression relate to stress. Provocative research on baboons by Sapolsky (1993) shows that

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education. Given this trend, one cannot interpret the difference-in-difference estimate as exclusively stemming from differences across groups of women in willingness to make health investments.

subordinate baboons exhibit measurable adverse physiological responses to exogenous declines in their rank among the group. Furthermore, the physiological responses impair the baboons' ability to handle stressful situations. One can imagine scenarios where stress among teenage girls makes it more difficult to resist tobacco or drugs. An alternative candidate for "third variables" that cannot be examined in these data would be the education of one's parents. Parental education likely does influence adolescent choices about both health and human capital investments, leading to results like those shown in this section. Further research should test more rigorously possible "third variables."

Network effects exacerbate existing differences in behavior by SES.

A large literature in the social sciences emphasizes the importance of networks and social interactions in influencing a variety of outcomes (See Wilson 1987, Jones 1994, Borjas 1995, Case and Katz 1991, Jencks and Mayer 1990, Bertrand et al. 2000). Networks are believed to be especially important for groups segregated along racial, ethnic, educational or economic dimensions. Given differences in the underlying propensity to invest in health inputs, networks may exacerbate cross-group differences either through information or stigma effects. For example, one's perceived health production function is altered by observing the health inputs (and outcomes) of others. Alternatively, stigma may exacerbate differences. One can model this by defining utility such that others' actions influence one's own utility of an action, as in Glaeser et al. (1996). Regardless of the mechanism, network effects can only contribute to differential health behavior by SES by exacerbating existing differences in behavior across groups. Rather than consider network effects as a separate hypothesis, I present empirical evidence on the magnitude of the impact that networks can have on behavior within a household. Empirically I cannot distinguish stigma from information effects, but I present evidence on the magnitude of networks by estimating how the smoking behavior of men can affect spousal smoking behavior.

If network effects explain an important part of differences in health behavior by SES, then exogenous changes in the smoking status of one family member should affect the smoking

status of other household members. Jones (1994) documents a significant relationship between the presence of other smokers in the household and smoking behavior of individuals. However, the presence of other smokers is probably correlated with one's own propensity to smoke, so his test likely suffers from omitted variable bias.

I test for the importance of network effects at the family level by analyzing the impact of spousal smoking status on a woman's propensity to smoke. To address the potential omitted variable bias, I use workplace-smoking bans as an instrument for spousal smoking status. Evans et al. (1999) conclude that workplace-smoking bans have a significant impact on workers' propensity to smoke. The existence of a smoking ban in the husband's workplace is correlated with his smoking status, but should affect the wife's smoking behavior only through the impact of him smoking. By using these bans to instrument for the husband's smoking status, one can estimate the importance of networks in determining differential smoking behavior.

In 1992 and 1993, the *Current Population Survey* (CPS) contains 3 supplemental surveys on tobacco use and habits. In the surveys, respondents were asked, "Does your place of work have an official policy that restricts smoking?" The survey also asks whether smoking is restricted in all work areas and all public areas. To determine the impact of spousal smoking status on smoking, I limit the CPS supplement samples to married couples with husbands who work indoors. A worker is considered to have a ban if he says that his work place has an official policy banning smoking in all work areas and all public areas. Table 8 shows the results of linear probability models of whether a woman currently smokes on her husband's smoking status, a dummy variable equal to one if husband currently smokes. Each specification includes the natural log of family income, both husband's and wife's education, specified as it has been throughout the paper, state fixed effects, dummies for race, ethnic origin, a dummy equal to one if the wife has a workplace smoking ban, a dummy for metropolitan area residence, and a quadratic in husband's and wife's age. I also include a full set of industry and occupation dummies for both the husband and wife to control for differences in the types of workers who face smoking bans.

Column 1 shows the results from a basic regression of wife's smoking on the SES variables. In this specification, a 10 percent rise in family income decreases the probability that the wife smokes by .26 percentage points. An increase in education from less than high school to some college or more reduces the probability the wife smokes by 15.6 percentage points. A similar increase in husband's education leads to a 3.5 percentage point decrease in the probability that a wife smokes. These effects are dwarfed by the size of the coefficient on husband's smoking status in column 2. The OLS specification suggests that women are 28 percent more likely to smoke if their husbands smoke. What is more interesting in specification 2 is that the impact of income and husband's education disappears. This implies two things: women are much more likely to smoke if their husbands smoke, and the husband's education has no impact on wives' smoking status independent of any effect on whether he smokes. In specification 3, using the smoking ban instrument, the coefficient on the husband's smoking status rises to .48. In other words, an exogenous 10 percent decrease in smoking rates of married men would be accompanied by a 5 percent decline in smoking by their wives simply due to the network effect. One would expect the IV estimate to be significantly higher than the OLS estimate in the case where there is measurement error in husband's smoking status. By using only the variation from the smoking ban, it is possible to obtain a cleaner estimate of the effect of spousal smoking status on wives' smoking patterns. Furthermore, this estimate refers to the small group of women whose smoking status is influenced by their husbands' workplace smoking policy. The estimates shown here represent a local average treatment effect for this group and may not generalize to the population. One should thus interpret the magnitude of the spousal effect with caution, but the results appear plausible.

I conclude, based on table 8, that within family networks exacerbate differences in smoking by SES. To the extent that there are inherent differences in the underlying propensity to smoke for low SES individuals versus high SES individuals, positive-assortive-mating may play a large role in exacerbating these differences. It is important to note that this differs from more traditional network effects because it is happening within the family and thus may not be

expected to work as a multiplier. Nonetheless, the network effect shown here is important because policies that affect the health investments of one family member can have substantial effects on other family members.

V. Conclusions

This paper used information on infant health at birth and health inputs during pregnancy to isolate mechanisms that can explain the large differences in health status by SES. Table 9 summarizes the results.[15] Among white women, health habits of the mother can explain about half of the differences in low birth weight. Smoking alone can account for 46 percent of the "Some College or more"- "High School or less" differential in low birth weight. For black women, smoking explains 28 percent of this birth weight differential by education while other health habits explain 9 percent of differential low birth weight. The remainder of the paper tested hypotheses for why health habits such as smoking differ by SES.

The bottom panel of table 9 summarizes my results based on smoking habits of childbearing age women. I find that "third variables" causing individuals to make different investments in both health and education play the largest role in creating differential smoking rates by SES. These "third variables" explain at least 44 percent of the difference in smoking behavior between college-educated and other women.

Another important explanation for differential behavior is that women with additional education and financial resources have more complete health knowledge and respond more to common health knowledge than women from low-SES groups. I estimate that knowledge and its use can explain a modest share of the differences in smoking during pregnancy by education, 10 to 33 percent, as shown in Table 9. Given disparities in health habits, I find evidence that

[15] The estimates in the top panel of Table 9 use the coefficients from a linear version of the Full Model in table 2 to predict the size of the differential holding a particular set of factors constant at their mean values. Due to non-linearities in the probit model, predicted shares of the differential low birth weight by education do not sum to one, so the linear model is more intuitively appealing for this exercise. The qualitative patterns in the probit decomposition are the same as those using the OLS model; smoking is most important, and other health habits are also important.

network effects at the family level augment these differences. Wives with husbands who smoke are nearly 50 percent more likely to smoke than women with nonsmoking husbands.

The results have several implications. First, the finding that infant health disparities by SES are largely determined by disparities in health habits and that these disparities exist early in life suggests that we may expect too much from prenatal programs for the poor. The disparities in smoking and low birth weight by SES have little to do with differences in smoking cessation during pregnancy. If much of health inputs are determined by investments chosen early in life, the prenatal period is too late in the process of health production to have a large impact on infant health. Indeed, much of the literature on prenatal care finds modest if any impact of prenatal care on infant health (Piper 1990, Alexander and Korenbrot 1995, Joyce 1999). Second, although income is correlated with infant health outcomes, programs that redistribute income without affecting "third variables" may not improve infant health.

What policies should be explored in light of these results? I show no direct evidence on policy effects in this paper, but my results suggest several factors that could guide policy design and research to reduce health disparities by income and education. First, the finding that behavior matters suggests efforts should target health behaviors with the most direct health impact, in this case smoking. Second, the finding that "third variables" determine much of the disparities in health behaviors early in the lives of women suggest that policies should target youths. Recent evidence supports the notion that taxation is the most effective policy instrument against youth smoking, although less so for the youngest teens (Gruber 2000). My results are consistent with the idea that cigarette taxes have positive benefits on health outcomes for the impacted youth and potentially their offspring. Taxation is one viable instrument, but not without costs to adult smokers who are disproportionately from low SES groups. Other policies to impact youth smoking have unclear effects, and deserve further scrutiny.

Finally, the results on network effects at the family level suggest that program design should consider how networks facilitate or impede program goals. Much of the effort to reduce teen smoking works in a top down manner, through public health campaigns, school-based

educational programs, or through the imposition of taxes. In some public health arenas, such as reproductive health, peer effects are acknowledged by enlisting peer counselors to educate young people regarding contraception or sexually transmitted diseases. These programs are intended to educate, but an equally important goal is to improve the image and acceptance of safe sexual practices. It would be worthwhile to investigate whether programs that use peers to educate and influence youth smoking behavior have any impact.

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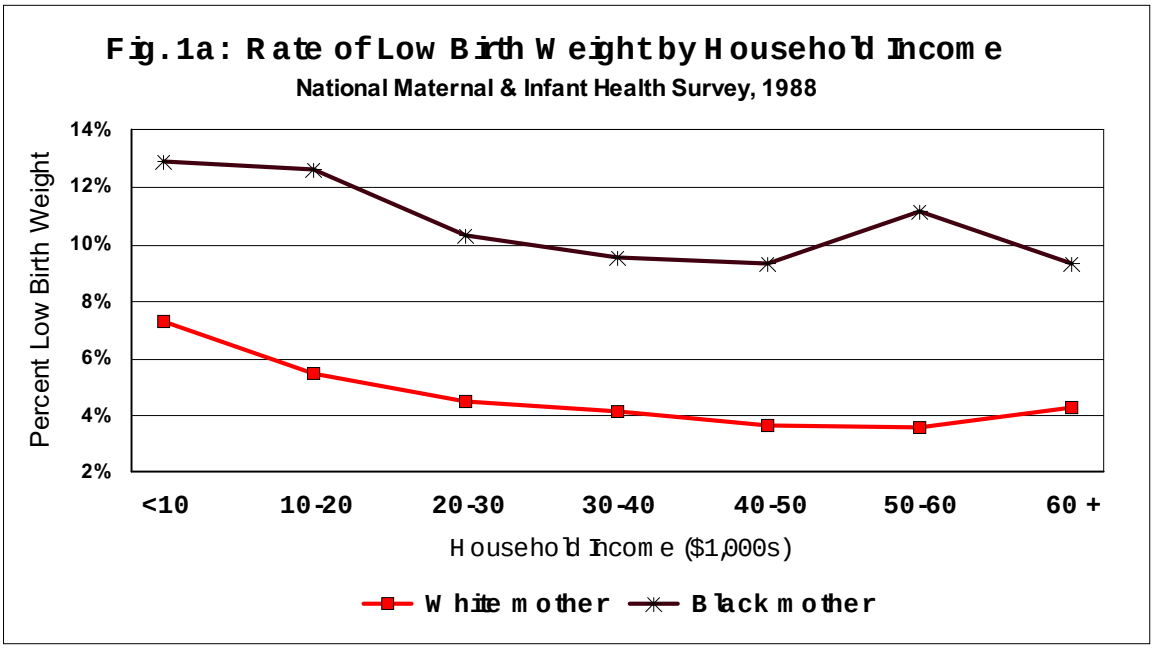
Fig. 1a: Rate of Low Birth Weight by Household Income
National Maternal & Infant Health Survey, 1988
Percent Low Birth Weight
14%
12%
10%
8%
6%
4%
2%
<10
10-20
20-30
30-40
40-50
50-60
60 +
Household Income ($1,000s)
White mother
Black mother

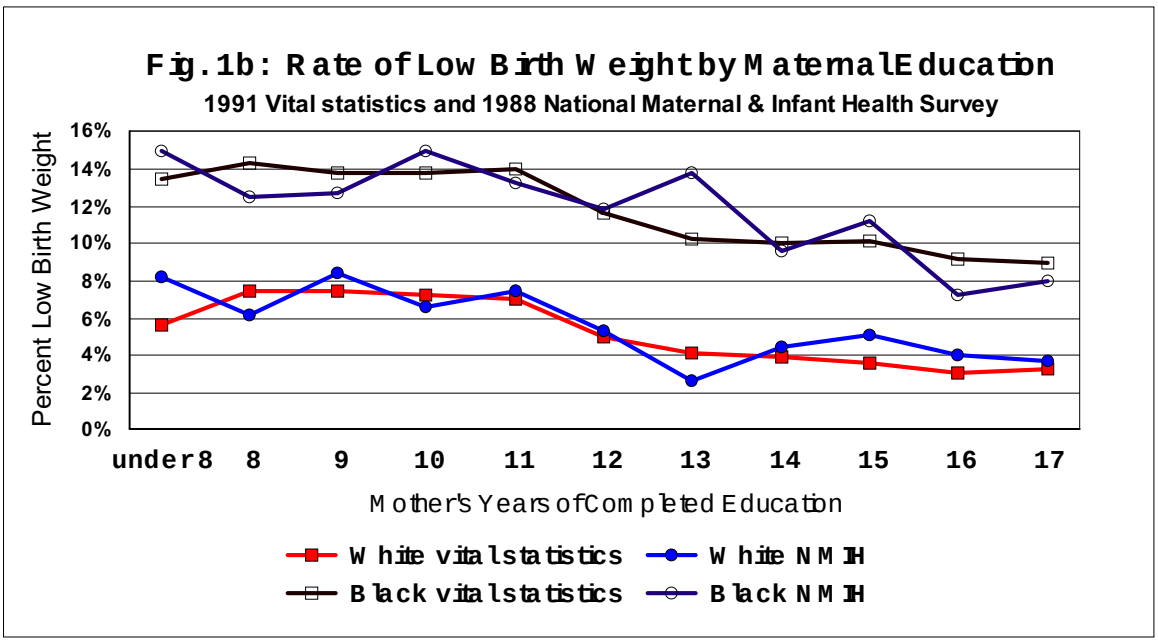
Fig. 1b: Rate of Low Birth Weight by Maternal Education
1991 Vital statistics and 1988 National Maternal & Infant Health Survey
Percent Low Birth Weight
16%
14%
12%
10%
8%
6%
4%
2%
0%
under 8
8
9
10
11
12
13
14
15
16
17
Mother's Years of Completed Education
White vital statistics
White NMIH
Black vital statistics
Black NMIH

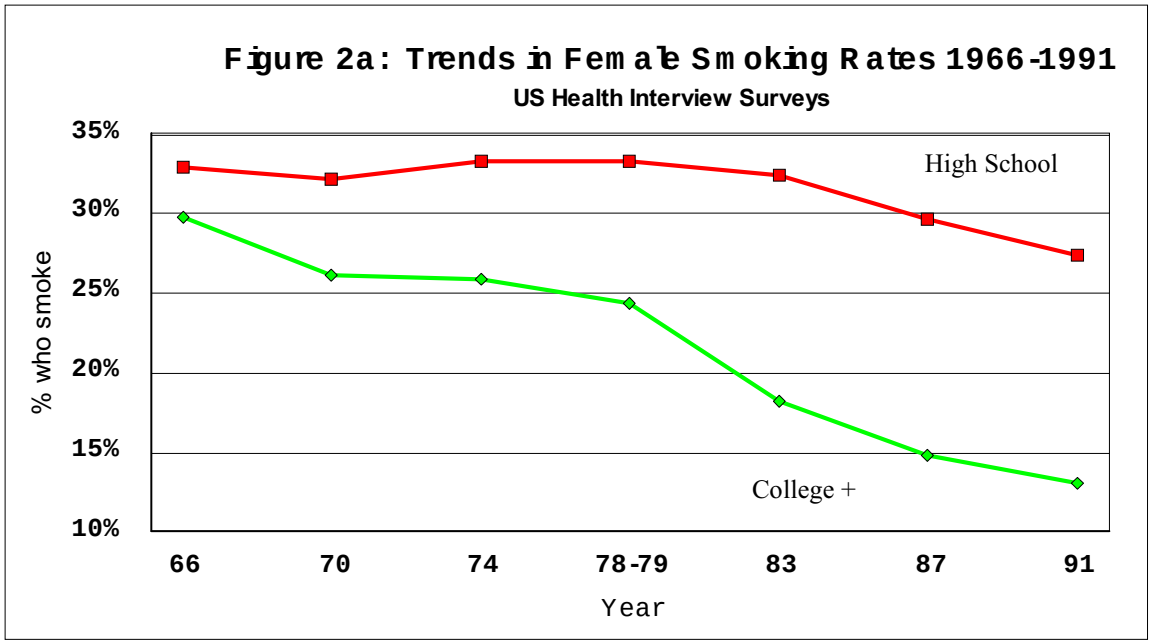
Figure 2a: Trends in Female Smoking Rates 1966-1991
US Health Interview Surveys
% who smoke
35%
30%
25%
20%
15%
10%
High School
College +
66
70
74
78-79
83
87
91
Year

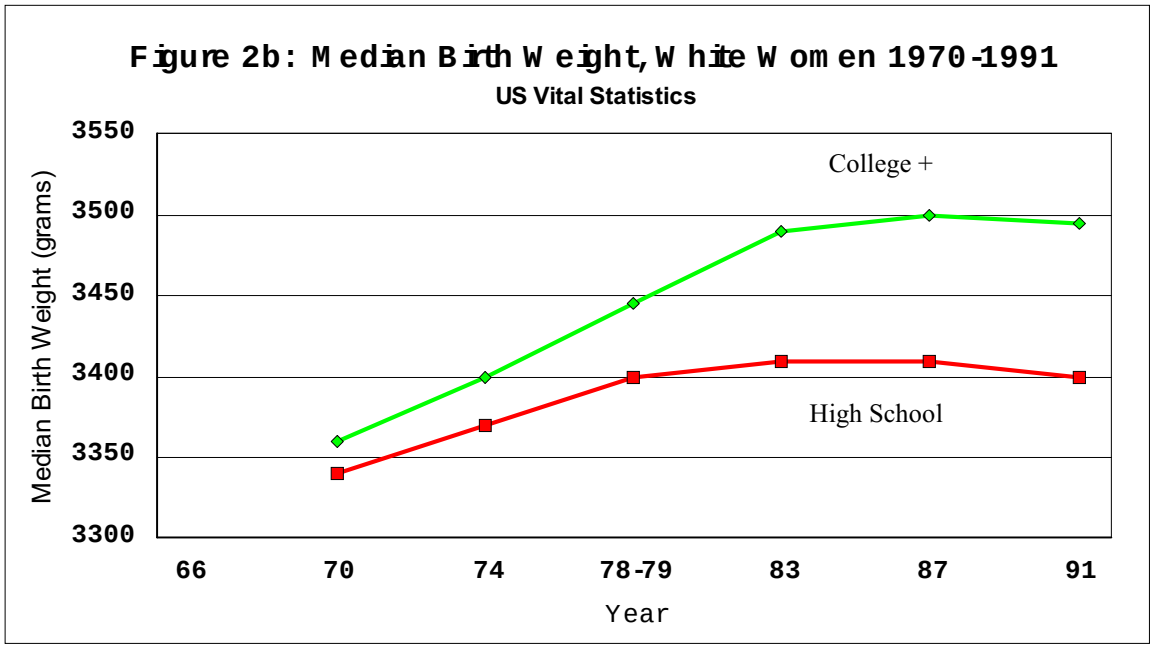
Figure 2b: Median Birth Weight, White Women 1970-1991
US Vital Statistics
Median Birth Weight (grams)
3550
3500
3450
3400
3350
3300
College +
High School
66
70
74
78-79
83
87
91
Year

Table 1a: Summary Statistics of 1988 National Maternal & Infant Health Survey

	White Mother			Black Mother		
	Mean	SD	Obs.	Mean	SD	Obs.
OUTCOME MEASURES						
Infant deaths per 1,000 live births	6.0		4,298	11.9		4,744
(Infant + Fetal deaths) per 1,000 births + fetal deaths	8.2		5,189	14.9		5,315
% Low Birth Weight (< 2500 g)	4.9		5,189	11.1		5,315
Birth Weight in grams	3,427	563	5,189	3,153	615	5,315
EDUCATION & INCOME						
Mother's highest year of school	12.8	2.2	5,189	12.1	1.9	5,315
Household Income ($1988)	29,949	20,148	5,189	16,102	15,946	5,315
DEMOGRAPHICS						
% of mothers married at delivery	83.9		5,189	35.6		5,315
% male infant	53.0		5,189	50.0		5,315
MATERNAL HEALTH HABITS						
% Who smoked during pregnancy[a]	25.4		5,189	18.8		5,315
% Moderate/heavy drinkers during pregnancy[b]	0.52		5,189	1.02		5,315
% Who used cocaine during pregnancy	0.36		5,189	1.20		5,315
% Prenatal Care in 1st trimester	83.4		5,189	62.7		5,315
% Who took vitamins while pregnant[c]:	90.2		5,189	86.4		5,315
MATERNAL HEALTH						
Mother's age in years	26.5	5.5	5,189	24.2	5.6	5,315
Birth order of infant	2.2	1.4	5,189	2.5	1.6	5,315
% with last live birth <18 months ago	5.5		5,189	12.2		5,315
Birth weight of mother in grams	3,217	580	4,092	3,088	631	3,252
Pre-pregnancy BMI=weight/height^2 (kg/m^2)	22.8	4.3	5,189	23.7	5.1	5,315
ACCESS/INSURANCE						
% Uninsured in state	12.5		5,189	14.3		5,315
% Who said prenatal care was hard due to:						
Cash problems?	6.1		5,189	5.3		5,315
Transportation/Appt. problems?	4.7		5,189	7.5		5,315
Problems with provider?	4.4		5,189	3.8		5,315
Other problems?	2.1		5,189	3.3		5,315
# Minutes to prenatal provider	20.4	15.0	5,189	21.4	16.1	5,315
% Who paid for prenatal care with the following:						
Own income?	38.3		5,189	16.0		5,315
Family contribution?	2.0		5,189	4.1		5,315
Private Insurance/HMO?	66.3		5,189	31.0		5,315
Medicaid?	13.1		5,189	47.4		5,315
Average distance to nearest abortion provider in mother's state measured in miles	17	16	5,189	15	13	5,315
% Living in state with parental consent law	28.4		5,189	74.5		5,315
% Living in state with restrictions on Medicaid funding for abortion	77.5		5,189	22.8		5,315

a - "Smoked During Pregnancy" equals 1 if mother smoked > 0 cigarettes /day after she found out she was pregnant
b - "Moderate/Heavy Drinker During Pregnancy" equals 1 if mother drank >=5 drinks /week after finding out she was pregnant
c – Vitamins include: multivitamins, vitamin A, vitamin C, calcium, folic acid, iron, zinc

Table 1b: Summary Statistics of 1991 Vital Statistics Sample

	White Mother			Black Mother		
	Mean	SD	Obs. (millions)	Mean	SD	Obs. (millions)
OUTCOME MEASURES						
Infant deaths per 1,000 live births	6.3		2.3	14.8		0.522
% Low Birth Weight (< 2500 g)	4.8		2.3	12.0		0.521
Birth Weight in grams	3,412	558	2.3	3,121	630	0.521
EDUCATION						
Mother's highest year of school	12.7	2.5	2.2	12.0	2.0	0.513
MATERNAL HEALTH HABITS						
% Who smoked during pregnancy[a]	18.0		2.3	13.9		0.522
% Moderate/heavy drinker during pregnancy[b]	0.16		2.3	0.59		0.516
% Prenatal care in 1st trimester	81.0		2.2	61.9		0.509
DEMOGRAPHICS						
% Mothers married at delivery	80.9		2.3	31.7		0.522
% Male infant	51.2		2.3	50.7		0.522
MATERNAL HEALTH						
Mother's age in years	26.7	5.6	2.3	24.1	5.9	0.522
Birth Order	2.3	1.5	2.3	2.6	1.7	0.520
% With last live birth <18 months ago	6.6		2.3	12.6		0.523

a- Smoked during pregnancy equals 1 if mother reported smoking > 0 cigarettes per day during pregnancy.

b - "Moderate/Heavy Drinker During Pregnancy" equals 1 if mother drank >=5 drinks /week during pregnancy

Table 2: Probit Models of Low Birth Weight

	White Mother (N=5,189)			Black Mother (N=5,315)		
Model	Ln Household Income	Maternal Education H.S. Degree	Some College+	Ln Household Income	Maternal Education H.S. Degree	Some College+
No Controls	-0.0053**	-0.0137**	-0.0271***	-0.0058*	-0.0111	-0.0223**
	(0.0024)	(0.0061)	(0.0065)	(0.0036)	(0.0098)	(0.0112)
Full Model	-0.0021	-0.0053	-0.0141**	-0.0042	-0.0013	-0.0053
	(0.0026)	(0.0063)	(0.0069)	(0.0038)	(0.0109)	(0.0137)
Full model excluding:						
Health Habits	-0.0032	-0.0086	-0.0220***	-0.0052	-0.0147	-0.0228*
	(0.0027)	(0.0064)	(0.0069)	(0.0039)	(0.0110)	(0.0130)
Demographics	-0.0031	-0.0050	-0.0135*	-0.0053	-0.0011	-0.0067
	(0.0026)	(0.0064)	(0.0069)	(0.0038)	(0.0110)	(0.0136)
Access	-0.0018	-0.0055	-0.0138**	-0.0031	-0.0003	-0.0045
	(0.0025)	(0.0063)	(0.0069)	(0.0036)	(0.0108)	(0.0135)
Maternal Health Status	-0.0008	-0.0046	-0.0113*	-0.0036	-0.0028	-0.0073
	(0.0027)	(0.0062)	(0.0067)	(0.0039)	(0.0099)	(0.0122)

Note: Sample includes only singleton births to black or white women. All calculations using NMIH '88 are weighted using the sample weights provided by NCHS. The numbers in this table show the implied change in the probability of being low birth weight (<2500 grams) with respect to the ln household income or mother's education when all other control variables are evaluated at their mean. Standard Errors are in parentheses. *- p<.10, **-p<.05, ***-p<.01

No Controls model includes: natural log of household income, mother's education, and 9 dummies for household size

Full Model includes: all variables in No Controls model, all Health Habits variables, Demographics, Access Variables, and Health Status variables.

Health Habits include: dummies for whether mother smoked, drank over 5 drinks/week, or used crack after learning of pregnancy, a dummy for receiving prenatal care in 1st trimester, a dummy equal to 1 if mother used multivitamins, vitamin A, vitamin C, folic acid, zinc, calcium, or iron.

Demographics include: dummy for marital status at delivery and for child sex.

Access includes: % uninsured in the state, distance to prenatal provider in minutes, dummies for whether prenatal care was paid for by own income, family income, private insurance, or Medicaid (women can have multiple payment sources), and dummies for whether mother had problems getting prenatal care due to: cash problems, transportation/appointment problems, problems finding provider, or other problems, the average distance in miles to the nearest abortion provider in the state, whether there were Medicaid restrictions on abortion funding in that state, and whether parental consent laws for abortion were in effect.

Health Status includes: 5-piece spline for maternal age, 12 dummies for mother's birth weight, 7 dummies for mother's body mass index, a dummy for whether mother's last live birth occurred < 18 months ago, birth order and its square.

Table 3: Detailed Results from Probit Models of Low Birth Weight

This table shows the implied change in the probability of being born Low Birth Weight (under 2500 g at birth)

	National Maternal and Infant Health Survey, 1988							
	White Mother				Black Mother			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of Household Income	-0.0053***	-0.0031	-0.0025	-0.0021	-0.0058*	-0.0044	-0.0041	-0.0042
	(0.0024)	(.0024)	(0.0024)	(0.0026)	(0.0036)	(0.0035)	(0.0035)	(0.0038)
Maternal Education								
High School Degree	-0.0137**	-0.0096	-0.0087	-0.0053	-0.0111	-0.0049	-0.0036	-0.0013
	(0.0061)	(0.0060)	(0.0060)	(0.0063)	(0.0098)	(0.0097)	(0.0097)	(0.0109)
Some College or More	-0.0271***	-0.0170**	-0.0156**	-0.0141**	-0.0223**	-0.0130	-0.0109	-0.0053
	(0.0065)	(0.0065)	(0.0066)	(0.0069)	(0.0112)	(0.0113)	(0.0115)	(0.0137)
MATERNAL HEALTH HABITS								
Smoked during pregnancy?		0.0427***	0.0421***	0.0358***		0.1000***	0.0917***	0.0939***
		(0.0066)	(0.0066)	(0.0062)		(0.0137)	(0.0137)	(0.0145)
Drank >5 drinks/week during pregnancy?			0.0307	0.0169			0.1095**	0.1084**
			(0.0389)	(0.0341)			(0.0594)	(0.0590)
Used cocaine during pregnancy?			-0.0074	-0.0169			0.0420	0.0441
			(0.0291)	(0.0190)			(0.0448)	(0.0443)
Prenatal care in 1st trimester?			-0.0096	-0.0015			-0.0063	-0.0039
			(0.0064)	(0.0059)			(0.0088)	(0.0090)
Took vitamins during pregnancy?			0.0024	0.0004			0.0027	0.0016
			(0.0069)	(0.0068)			(0.0119)	(0.0118)
DEMOGRAPHICS								
Sex of child (male=1, female=0)				-0.0087**				-0.0124
				(0.0040)				(0.0081)
Mother is married?				-0.0186***				-0.0188*
				(0.0085)				(0.0099)
MATERNAL HEALTH STATUS								
Includes 5-piece spline in mother's age?				Yes				Yes
Includes dummies for mother's birth weight?				Yes				Yes
Includes dummies for mother's BMI?				Yes				Yes
Birth order				-0.0077*				-0.0133**
				(0.0044)				(0.0066)
Birth order squared				0.0004				0.0013**
				(0.0005)				(0.0006)
Last live birth < 18 months ago?				0.0264**				-0.0021
				(0.0121)				(0.0132)

Table 3, continued: Probits of Low Birth Weight on Mother's Education and Household Income

	National Maternal and Infant Health Survey, 1988							
	White Mother				Black Mother			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
ACCESS								
% Uninsured in state				0.0010*				0.0004
				(0.0006)				(0.0013)
Prenatal care was hard due to:								
Cash problems?				-0.0018				0.0008
				(0.0089)				(0.0187)
Transportation / Appt. problems?				-0.0023				0.0080
				(0.0102)				(0.0169)
Problems with provider?				-0.0028				-0.0064
				(0.0102)				(0.0218)
Other problems?				0.0529***				0.0291
				(0.0273)				(0.0248)
How far to prenatal provider? (Minutes)				1.63×10^{-5}				0.0004
				(0.0001)				(0.0002)
Paid for prenatal care with:								
Own income?				0.0061				0.0062
				(0.0046)				(0.0126)
Family income?				0.0127				-0.0276
				(0.0176)				(0.0172)
Private insurance?				0.0012				0.0112
				(0.0052)				(0.0122)
Medicaid?				0.0044				-0.0024
				(0.0081)				(0.0114)
Average distance to abortion provider in mother's state (in miles)				-0.0001				-0.0005
				(0.0001)				(0.0004)
State restricts Medicaid funded abortions				-0.0005				0.0090
				(0.0054)				(0.0108)
State has parental consent law for abortion				0.0060				-0.0001
				(0.0047)				(0.0104)
Pseudo R-squared	0.0209	0.0375	0.0385	0.0757	0.0039	0.0234	0.0257	0.0449
Number of observations	5,189				5,315			

Notes: Sample includes only singleton births to black or white women for which information on the mother's smoking status during pregnancy is available. All calculations are weighted using the sample weights provided by NCHS. Standard Errors are in parentheses. All models include 9 dummy variables for household size. Each cell reports dF/dX, where F is the probability of being born low birth weight, and X is the independent variable shown in the first column, evaluated at the mean of all other independent variables. For discrete variables, dF/dX reflects the change in the probability of low birth weight for a 0 to 1 change in X.

*- p<.10, **-p<.05, ***-p<.01

Table 4: Comparison of impact of smoking on birth weight from different studies

	Controlled trial Permutt & Hebel '89	IV estimate (cigarette taxes) Evans & Ringel '99	Full Set of Covariates Author's calculation		
	Pooled	Pooled	Pooled	Black	White
Birth weight (grams)	-400	-369	-205	-200	-239
	(177)	(170)	(19)	(25)	(22)
Low birth weight (<2500 grams)	0.091	0.114	0.039	0.094	0.036
	(0.074)	(0.061)	(0.006)	(0.014)	(0.006)
Observations	867[a]	10,547,199	10,510	5,315	5,189

Standard errors are shown in parentheses. Table shows coefficient on smoking in models of birth weight and low birth weight.

a -This number was not reported in Permutt and Hebel (1989), but is based on sample sizes reported in Sexton and Hebel (1984)

Table 5: Summary of Knowledge & Health Behavior by Education

Childbearing Aged Women in the National Health Interview Survey, 1985 and 1990

	High School or Less		Some College or More		t-statistic for difference
	Mean	Obs	Mean	Obs	
Knowledge					
Smoking Knowledge	0.77 (0.34)	4,347	0.86 (0.28)	2,485	11.77
Drinking Knowledge	0.73 (0.23)	4,347	0.81 (0.20)	2,485	15.05
Smoking Behavior During pregnancy					
Smoked while pregnant?	0.29 (0.45)	4,347	0.11 (0.32)	2,485	19.21
Cut smoking while pregnant? (smoked in year before delivery)	0.53 (0.50)	1,818	0.56 (0.50)	518	1.21
Quit smoking while pregnant? (smoked in year before delivery)	0.18 (0.39)	1,818	0.28 (0.45)	518	4.59

Standard Deviations are in Parentheses. Sample includes women aged 18-44 who had live birth in past 5 years. All HIS calculations use sample weights Provided by NCHS.

Smoking knowledge = share of correct responses to questions on whether smoking increases chance of low birth weight, premature birth, miscarriage, stillbirth, lung cancer, emphysema, bladder cancer, cancer of the larynx, cancer of the esophagus, and chronic bronchitis.

Drinking knowledge = share of correct responses to questions on whether heavy drinking increases chance of: miscarriage, low birth weight, mental retardation, birth defects, cirrhosis of the liver, throat cancer, and cancer of the mouth, and 2 questions on the nature of fetal alcohol syndrome.

The rate of smoking during pregnancy in the NMIH'88 was .31 (.13) for women who completed High school or less (Some college or more).

Table 6: Models of How Knowledge and SES Impact Smoking During Pregnancy

Table shows implied marginal effect of each variable based on probit models of smoking behavior.
National Health Interview Survey, 1985 and 1990

	Dependent variable=Smoked while pregnant? (mean = .22)				
	(1)	(2)	(3)	(4)	(5)
Ln Household Income	-0.0424**	-0.0387**	-0.0382**	-0.0387**	-0.0380**
	(0.0085)	(0.0083)	(0.0083)	(0.0085)	(0.0084)
Maternal Education					
High School Degree	-0.1349**	-0.1221**	-0.1305**	-0.1224**	-0.1305**
	(0.0164)	(0.0164)	(0.0164)	(0.0164)	(0.0164)
Some College or More	-0.2680**	-0.2430**	-0.2469**	-0.2434**	-0.2471**
	(0.0196)	(0.0197)	(0.0196)	(0.0197)	(0.0197)
Smoking Knowledge		-0.3032**	-0.3356**	-0.3124**	-0.3464**
		(0.0280)	(0.0292)	(0.0336)	(0.0350)
High School*(Smoking Knowledge)			-0.1496*		-0.1685*
			(0.0609)		(0.0768)
College*(Smoking Knowledge)			-0.2649**		-0.2857**
			(0.0740)		(0.0919)
Drinking Knowledge				0.0184	0.0211
				(0.0355)	(0.0367)
High School*(Drinking Knowledge)					0.0375
					(0.0846)
College*(Drinking Knowledge)					0.0375
					(0.0996)
Number of Observations	6,083				

Control variables include dummies for each of the following: race (black, white, other), 5 year age categories, year of birth, hispanic background (Multiple Hispanic, Puerto Rican, Cuban, Mexican-Mexicano, Mexican-American, Chicano, Other latin American, other Spanish, Spanish of unknown type, Unknown, Not Spanish Origin),marital status, major activity last week, whether self reported health is fair/poor, region of residence, whether woman has a regular source of primary care, whether she has had a pap smear in the last 2 years, and whether stress has affected health in the past year. Mother's Education and knowledge variables are expressed as deviations from their means.

*- p<.05, **-p<.01

Smoking knowledge = share of correct responses to questions on whether smoking increases chance of low birth weight, premature birth, miscarriage, stillbirth, lung cancer, emphysema, bladder cancer, cancer of the larynx, cancer of the esophagus, and chronic bronchitis.

Drinking knowledge = share of correct responses to questions on whether heavy drinking increases chance of: miscarriage, low birth weight, mental retardation, birth defects, cirrhosis of the liver, throat cancer, and cancer of the mouth, and 2 questions on the nature of fetal alcohol syndrome.

Table 7: Differences-in-Differences Estimates of Smoking Behavior Following the 1964 Surgeon General's Report

Changes in Female smoking rates following Surgeon General's 1964 Report
Women born in 1917-1946, Health Interview Surveys 1966 & 1983

	Share of women who smoked in: Pre-1964[a]	1966	1983	Difference 1983-(Pre-1964)
High school or less	0.522	0.425	0.330	-0.192***
	(0.003)	(0.003)	(0.008)	(0.008)
Some college or more	0.510	0.376	0.258	-0.252***
	(0.007)	(0.007)	(0.011)	(0.013)
Difference	0.012	0.049***	0.072***	
	(0.008)	(0.008)	(0.014)	
			Difference-in Difference	**0.060*** (0.016)**

Share of all women who smoked by age 16
Health Interview Surveys,1978, 1979, 1987 & 1992

	Turned 16 before 1953	Turned 16 after 1983	Difference 1983-1953
High school or less	.098	0.181	0.0830***
	(0.003)	(0.008)	(0.0081)
Some college or more	0.071	0.075	0.0044**
	(0.005)	(0.005)	(0.0070)
Difference	-0.027***	-0.105***	
	(0.005)	(0.009)	
		Difference-in Difference	**-0.079*** (0.011)**

Standard Errors in parentheses.

a-Smoking rates for pre-1964 are approximated using share of women who had "ever smoked" in 1966.

The "Turned 16 before '53" sample includes women aged 17-36 in 1953

The "Turned 16 after '83" sample includes women aged 18-27 in 1992

*- p<.10, **-p<.05, ***-p<.01

Table 8: Models of the Impact of Spousal Smoking Status on Women's Smoking Status

Sample of 24,126 married couples from the Current Population Survey 9/92, 1/93, and 5/93 Tobacco Supplements

	Dependent Variable: Wife Smokes (mean=.174)		
	(1)	(2)	(3)
	OLS	OLS	IV Estimate
Husband Smokes		0.2849***	0.4848***
		(0.0056)	(0.1228)
Ln Family Income	-0.0262***	-0.0089*	0.0032
	(0.0056)	(0.0054)	(0.0092)
Wife's education			
High School Degree	-0.0827***	-0.0744***	-0.0687***
	(0.0102)	(0.0097)	(0.0106)
Some College+	-0.1562***	-0.1368***	-0.1232***
	(0.0109)	(0.0104)	(0.0106)
Husband's education			
High School Degree	-0.0087	0.0088	0.0210*
	(0.0103)	(0.0098)	(0.0126)
Some College+	-0.0350***	0.0037	0.0309
	(0.0109)	(0.0104)	(0.0198)
Industry & Occupation Dummies Included?	Yes	Yes	Yes
R^2	0.0737	0.1650	

Note: Standard Errors in parentheses. Controls include: dummy for whether wife has smoking ban in work place, state dummies, dummies for race (White, Black, American Indian and Aleut Eskimo, Asian & Pacific Islander, or Other), ethnic origin (Mexican American, Mexicano, Chicano, Puerto rican, Cuban, Central or South American, or other Spanish), whether residence is non-metro area or undefined area, a quadratic in husband and wife's age, and dummies for husband's and wife's industry (3 digit codes) & occupation (2 digit codes).

*-p<.1, **-p<.05, ***-p<.01

Regressions include only married couples where husband is an "indoor" worker. The variable "Workplace Ban" equals one if workplace forbids smoking in public and workplace areas.

Table 9: The Determinants of Differences in Low Birth Weight by Education

	White Mother		Black Mother	
	Percent difference in low birth weight	Share	Percent difference in low birth weight	Share
Difference in Rate of Low Birth Weight (HS or less – College educated)				
Actual Difference	39.6%	1.00	20.3%	1.00
Predicted Difference	39.6%		20.3%	
Share due to Smoking	18.1%	0.46	5.7%	0.28
Share due to Other Health Habits	1.0%	0.02	1.8%	0.09
Share due to All Other Factors**	20.5%	0.52	12.8%	0.63

Relative importance of hypotheses explaining the difference in female smoking behavior by education	
Part of smoking difference due to 3rd variables	44-73%
Part of smoking difference due to knowledge & its use	10-33%

The contributions of the share of birth weight differences due to Smoking, other Health Habits, and All Other Factors are based on predicted differences in birth weight from the linear version of the full model in Table 3. For example, the "Share due to Smoking" compares the predicted differences in low birth weight when smoking rates are replaced by mean rates to differences based on actual rates in the sample. For example, the actual difference in low birth weight among white mothers is 1.94 percentage points, or a 39.6 percent difference compared the white rate of low birth weight of 4.9 percent. If one replaces smoking rates in the model with mean smoking rates, there is a 20.9 percent difference in rates of low birth weight by college education. The "Share due to Smoking" is expressed as share of actual differences: (39.0-20.9)/39.6=18.1/39.6=.46.

The lower bound estimate of "Part of smoking due to 3rd variables" is based on the difference-in-difference estimate of differential rates of smoking at age 16 in table 7. From table 7, the 7.9 percentage point difference in early initiation of smoking is compared to the 18 percentage point difference in smoking during pregnancy between college-educated and less-educated women. The upper bound assumes that the 13.1 percentage point growth in differential smoking rates from 1966-91 resulted from third factors. See text for details.

The lower bound of "Part of smoking difference due to knowledge & its use" is based on a linear version of specification 5 in table 6, holding constant all knowledge variables at mean values. The upper bound of "Part of smoking due to knowledge & its use is based on the differential rates of quitting following the Surgeon General's report and assuming that quit rates did not vary by education prior to 1964. From Table 7, the 6.0 percentage point difference in the relative decline in smoking by education up until 1983 was divided by the 18 percentage point difference in smoking during pregnancy between college and less-educated women.

** All other factors include the remaining variables in the Full model in Table 3 and any remaining unexplained differences in smoking rates (error terms).