DEMOGRAPHIC ISSUES IN INFANT HEALTH IN THE 1990'S AND MEASUREMENT ISSUES IN COSTING MEDICAID EXPANSIONS

by

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Abstract

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National measures of infant health in the 1990s were flat, but rates of low birth weight and preterm birth among blacks, especially in center cities, improved. Health gains were especially marked in Washington, DC. Analysis at the metropolitan area level reveals that center city-suburban gaps in black infant health declined. The first two chapters of this dissertation use the 1990-2001 National Center for Health Statistics (NCHS) Natality Files to examine improvements in infant health among African-Americans, first, in Washington, DC, and second, in 37 metropolitan areas with large black populations.

Although Washington, DC also experienced substantial, above-average reductions in its non-marital and teen birth ratios, changes in the sociodemographic profile (age, marital status, education, parity) of mothers in the District of Columbia contributed little, if anything, to black infant health gains in the 1990s. Instead, a steep decline in prenatal smoking is the most important, identifiable cause of improved infant health, though we cannot distinguish between the effects of declines in measured tobacco use and unmeasured crack use. These findings are applicable to black trends in center city and suburban infant health and spatial health disparities in a broad sample of metropolitan areas, as well. Decomposition analysis using 1990 and 2000 Census data reveals that

changes in age-specific fertility rates and within-age rates of low birth weight and preterm birth explain more of the change in spatial inequality than changes in age-related population composition.

Chapter 3 departs from the area of infant health, focusing instead on methodological issues related to estimating the costs of expanding Medicaid through increased eligibility or simplification of enrollment and recertification procedures. Many estimates extrapolate from the per-enrollee costs of current Medicaid beneficiaries. We use month-to-month health insurance transitions, expenditures, and service utilization patterns for adults in the 1996-2003 Medical Expenditure Panel Survey (MEPS) to show that individuals who enroll in Medicaid and maintain coverage today have greater health needs than those with unstable or no Medicaid coverage. These results suggest that ignoring the adverse selection of current Medicaid enrollees will lead to overestimates of the per-enrollee costs of expanding eligibility or increasing take-up.

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Chapter 1 Why Did Reproductive Health in Washington, DC Improve in the 1990s? The Role of Demographic and Socioeconomic Change

Introduction

Infants born to unwed or teenage mothers are disproportionately likely to be born at low birth weight and/or to die within their first year of life (Hofferth and Hayes 1987; National Center for Health Statistics 1995; Maynard 1997). This association has been and continues to be used to justify many policy initiatives aimed at reducing births to unmarried women and teens. A 1994 article in an influential policy journal argued that the scale of welfare benefits in the 1980s had statistically significant, though small, positive effects on extra-marital childbearing (Murray 1994). A companion piece in the same issue boldly claimed that high rates of out-of-wedlock and teenage childbearing were responsible for high rates of infant mortality and low birth weight among Washington, DC's predominantly African American population:

> Impersonal "social forces" – material deprivation, joblessness, economic insecurity – cannot explain why one of the very richest black populations in America suffers from black America's very worst infant mortality rates. This perverse situation can, however, be explained in terms of dysfunctional or even pathological behavior by parents and adults – including parents and adults who happen to be neither poor nor poorly educated. Illegitimacy, welfare dependence, and the environment of violent crime mark out a vector of deadly risks to infants in Washington, DC (Eberstadt 1994).

The joint implication of these articles is that reducing welfare dependency would reduce illegitimacy and consequently improve infant health.[1]

Two years later, in 1996, Congress passed major legislation overhauling welfare, many elements of which aimed to reduce welfare dependency and nonmarital

[1] See, however, the exchange in a later issue between Korenman and Eberstadt (Eberstadt 1994; Korenman 1994).

childbearing. For example, welfare reform introduced lifetime limits on receipt of benefits and minimum work requirements. Many states also imposed caps on family size-related increases in benefits. In some cases, attempts to reach the goals of welfare reform involved large federal outlays. The Out-of-Wedlock Birth Reduction Bonus, for example, awarded a total of $100 million per year to those states with the greatest reductions in their nonmarital birth ratios in each of fiscal years 1999 through 2004.

Ten years after implementation of the Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA), it is instructive to consider whether welfare reform had its intended effects. There is little evidence that welfare reform impacted nonmarital or teenage birth rates (Acs and Koball 2003; Joyce, Kaestner et al. 2003), and few studies have explored the link between illegitimacy and infant health. Nevertheless, the belief that this relationship is causal continues to motivate and shape public policy. For example, welfare reauthorization in the Deficit Reduction Act of 2005 (S.1932) repealed the Out-of-Wedlock Bonus, but introduced block grants for programs emphasizing "healthy marriage promotion" and "responsible fatherhood". Evaluations of the efficiency and cost-effectiveness of programs such as these depend in part on understanding the extent to which illegitimate childbearing actually influences adverse infant health outcomes.

The experience of Washington, DC in the 1990s presents an especially compelling opportunity to study this. During the second half of the 1990s, Washington, DC experienced substantial reductions in its nonmarital birth ratio, and, as a result, "won" the Out-of-Wedlock Birth Reduction Bonus for six consecutive years. Over the same period, infant health in the District of Columbia improved dramatically. For example,

between 1991 and 2000, infant mortality fell from 20 to 12 per 1,000 live births, the lowest rate ever recorded for the District (Figure 1), and low birth weight fell by 23 percent.[2] As a result, the longstanding disparity between DC and national rates of infant mortality fell in half, to 5 per 1,000 live births, and nearly disappeared among blacks (District of Columbia State Center for Health Statistics Administration 2003) (Figure 1). The low birth weight gap between DC and the US also fell by nearly one-half, to 4 percentage points (Martin, Hamilton et al. 2002). Although these trends are consistent with the hypothesis that reducing welfare dependency and curbing nonmarital childbearing improves infant health, this study shows that changes in the sociodemographic profile (age, marital status, education, parity) of mothers in the District of Columbia contributed little, if anything, to black infant health gains in the 1990s.

At the time of Eberstadt's 1994 article in *The Public Interest*, only aggregate census tract-level data for 1990 were available to study the relationship between illegitimacy and infant health, and this data lacked information on race. We use individual-level birth certificate data for the period 1990 to 2001 to examine whether reductions in nonmarital or teenage childbearing in DC during the 1990s can explain coincident declines in adverse birth outcomes. We find that they cannot, for three main reasons. First, infant health improvements in Washington, DC during this time were nearly exclusive to blacks (Figure 2), and though the decline in nonmarital and teenage births were large for the District as a whole, among black women these demographic changes were simply too small to account for the large gains in infant health. Second,

[2] Unless otherwise stated, birth data are derived from authors' tabulations of the NCHS Natality files. We include singleton births occurring in DC from 1990 through 2001 to DC residents ages 15 to 39.

infant health improved primarily among infants born to *unmarried* black women, and not as a result of an increase in the proportion of births to married black women. Third, the decline in the proportion of black births to teens actually dampened improvements in infant health in the 1990s because among black women in the District, the risk of adverse birth outcomes rises with maternal age. These findings suggest that if welfare reform were responsible for improvements in reproductive health, the primary causal pathway could not have been reduced nonmarital and teenage childbearing.

Our analysis focuses on reproductive health improvements among the black population of Washington, DC. This population is notable not only because African-American infants are at elevated risk of adverse health outcomes relative to those of other racial backgrounds but also because infants in DC are at elevated health risk relative to those in the rest of the US (National Center for Health Statistics 1981; Geronimus and Bound 1990; Geronimus 1996; National Center for Health Statistics 2004). Additionally, births to black women comprise three-quarters of all births in Washington, DC during this period and account for most of the health gains.

We note, however, that the share of infants born to black mothers in DC declined by 12 percentage points over the 1990s. Therefore, some of the aggregate improvement in infant health must be due to changes in the racial composition of Washington, DC's population. In results not shown, we find that the reduction in the share of births to black women account for 27 and 20 percent of the unadjusted declines in District-wide low birth weight and preterm birth rates, respectively. From the standpoint of evaluating public policy, the importance of this finding is mitigated by another study's conclusion that the reduction in the proportion of births to black women resulted primarily from a

decline in the black population of the District, and secondarily, from a decline in black fertility rates (Korenman, Joyce et al. 2004).

Though we consider several other explanations for the improvements in infant health among African-Americans in Washington, DC, the decline in prenatal smoking, or unmeasured correlates of smoking, emerges as the most important. Trends in prenatal alcohol use or in the utilization of prenatal care and improvements in socioeconomic status (SES) are relatively unimportant after we control for smoking, though measurement error may reduce the explanatory power of these variables. In an appendix, we consider and are unable to reject the possibility that the effect we attribute to prenatal tobacco exposure reflects an unmeasured decline in crack use.

In the next section of the paper we describe our data. We then discuss changes in sociodemographic characteristics, lifestyles, and medical care that are relevant to infant health and that took place in Washington, DC during the 1990s. We describe our estimation approach, present regression results, and show that our findings are robust to many alternative specifications. Finally, we discuss of some implications of our findingsfor social policy.

Data Definitions and Hypotheses

This study analyzes the National Center for Health Statistics (NCHS) public-use Natality data files for the calendar years 1990-2001. The Natality files are the national compilation of all non-identifying information recorded on birth certificates for births occurring in the US and its territories. Since state laws mandate the completion of birth certificates, and federal law mandates the national compilation of this data, the Natality files can reasonably be expected to represent every birth occurring in the US and are

therefore free from many of the shortcomings of survey data. Though each state uses its own standardized birth certificate, these are based on recommendations from the NCHS, and so the information they collect is fairly comprehensive and consistent across time.

We include only those births occurring in Washington, DC to mothers who are DC residents. We further restrict the sample to singleton births to mothers ages 15 to 39, eliminating approximately 7 percent of births. We address possible bias introduced by our sample restrictions and other data choices later in the paper and show that our results are qualitatively robust to these considerations.

Race/ethnicity

We divide race/ethnicity into non-Hispanic black and everyone else, which we label, "nonblack". Throughout the paper we shorten the term, "non-Hispanic black", to "black". For brevity, at times we refer to infant race/ethnicity, though racial/ethnic identity is that of the mother. We focus on African-Americans, but repeat all of our analyses for nonblacks and for all races combined. Since we are interested in understanding changes in the black/nonblack health gap, we include results for nonblacks in Appendix 1A. There were a total of 10,164 births to all DC women in 1990; this number declined steadily each year, falling to 6,363 in 2001. The number of births to black women ranged from a maximum of 8,073 in 1991 to a minimum of 4,193 in 2001.

Prenatal alcohol and tobacco use

Prenatal alcohol and tobacco use are self-reported in terms of usage and frequency of use. Other studies have stressed that the health effects of these behaviors depend critically on dosage as well as usage (Sprauve, Lindsay et al. 1999). When the number of cigarettes smoked per day is unknown for smokers (5 percent of cases), we assign the

average among known cases. We then obtain a measure of frequency of use conditional on smoking by multiplying the number of cigarettes with the usage dummy. We are unable to control for the number of drinks consumed conditional on alcohol usage because frequency of alcohol use is not reported as a continuous variable in all years.

Self-reporting of behaviors that carry stigma may result in measurement error, and in fact, underreporting of smoking during pregnancy in birth certificate data is well-known. In many cases underreporting may be unintentional. Lack of specificity in questions about alcohol or tobacco use may lead women who quit smoking or reduce alcohol consumption when they discover they are pregnant to underreport, for example. The wording of survey questions can also greatly affect reporting accuracy (Kharrazi, Epstein et al. 1999). However, trends in the rate of smoking derived from the Natality Files are consistent with those from other data sources (Buescher, Taylor et al. 1993; Ventura, Hamilton et al. 2003). Though we are not aware of studies that estimate the accuracy of reported alcohol use, we also have no evidence to suggest it is problematic for analyses of trends in infant and maternal health.

Underreporting will result in misclassification and is likely (though not certain) to bias downwards the estimated effects of tobacco or alcohol use on birth outcomes in a cross-section.[3] It is unclear, however, what effect this bias would have on trends in birth outcomes. We explore the possibility of changes in reporting bias by regressing birth outcomes on prenatal alcohol or tobacco use in each year, separately, and find that the estimated effects of both are relatively steady over time (see Tables 1A-3 and 1A-4 in

[3] One study of birth certificate data in six states found a correlation between underreporting and birth weight, suggesting that analyses of birth certificate data would overestimate the association between low birth weight and prenatal smoking (Dietz, Adams et al. 1998).

Appendix 1A). This suggests that reporting bias did not change over time even as reported alcohol or tobacco use during pregnancy declined.[4,5] Additionally, the share of women for whom this information is missing does not trend over time, and we have no reason to believe that underreporting varies systematically with other maternal characteristics of interest. Therefore, we think it unlikely that measurement error in prenatal smoking or alcohol accounts for our main findings.

Adequacy of prenatal care

We measure adequacy of prenatal care using the Kotelchuck Index, which encompasses two concepts – adequacy of initiation of prenatal care and adequacy of received services (Kotelchuck 1994). The first corresponds to the month in which prenatal care began. The second compares the actual number of prenatal visits to the number of visits, adjusted for gestational age at initiation, recommended by the American Academy of Obstetricians and Gynecologists. Combining these two components results in four classifications of prenatal care: inadequate, intermediate, adequate, or adequate plus. We add a separate category for records for which classification cannot be determined. Though neither characterizes the content of prenatal care, the Kotelchuck Index is more comprehensive than the Kessner Index, which has been criticized on a number of dimensions: overemphasis on the timing of initiation of prenatal care; failure to distinguish between inadequacy of care due to late initiation from that due to an insufficient number of visits; a number of visits requirement that is too lenient at

[4] If underreporting increased over time, then we would expect the estimated effect of alcohol or tobacco to decline with time.

[5] Note that among nonblacks the effects of prenatal alcohol and tobacco use are more often than not statistically insignificant and sometimes negative.

gestational ages 36 weeks or greater; and lack of guidance on dealing with missing data (Kotelchuck 1994).

However, missing data severely compromise our ability to estimate reliably the contribution of changes in the adequacy of prenatal care to infant health improvements (Alexander, Tompkins et al. 1991). On average, 15 percent of birth records for black infants lack the information necessary to classify adequacy of prenatal care. Furthermore, this figure rises markedly over time, from 4 percent in 1990 to 21 percent in 2001.[6] We include adequacy of prenatal care in some of our analyses (including a dummy for missing) because measures of prenatal care are widely studied determinants of infant health and, prenatal care is potentially, a key policy instrument, but we note that our failure to detect an effect may be the result of poor data quality.

Medical conditions

Birth certificate data classify and record seventeen "medical risk factors" (MRF): anemia, cardiac disease, diabetes, genital herpes, hydramnios/oligohyramnios, hemoglobinopathy, chronic and pregnancy-associated hypertension, eclampsia, an incompetent cervix, renal disease, RH sensitization, uterine bleeding, a previous infant born weighing 4,000 or more grams, a previous infant born preterm or small-for-gestational age, or "other". There are also sixteen indicators for complications of labor and/or delivery (CLB): meconium, premature rupture of the membrane, abruptio placenta, placenta previa, other excessive bleeding, seizures during labor, prolonged labor, dysfunctional labor, breech, cephalopelvic disproportion, cord prolapse, anesthetic

[6] The share of nonblack birth records with missing prenatal care data is also high and also increases sharply over time.

complications, fetal distress, or "other" (for a brief description of selected MRF and CLB, see Table 1A-7 in Appendix 1A). Several, such as an incompetent cervix and abruptio placenta, can be linked directly to adverse birth outcomes.[7] Others, such as anemia, may reflect underlying, unmeasured conditions that contribute to adverse birth outcomes.[8]

Although the incidence of most medical risk factors and complications of labor are quite low, we include in our regressions separate dummy variables for each, because they may represent different underlying causes.[9] However, with one exception, we enter all of the risk factors or all of the complications into the regressions simultaneously rather than additively. We treat separately the widely emphasized risk associated with having had a previous infant born preterm or small-for-gestational age (Kramer 1987; Goldenberg, Iams et al. 1998; Meis, Goldenberg et al. 1998).

Outcomes

Low birth weight and preterm birth (combined) are the second most important factors in infant mortality among all races, and the leading causes of death among infants born to black mothers (MacDorman and Atkinson 1999). We construct these measures using birth weight and length of gestation.[10] We define low birth weight (LBW) as birth

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[7] An incompetent cervix is a weakened cervix that predisposes a woman to mid-term miscarriage or preterm delivery. Abruptio placenta occurs when the placenta detaches prematurely from the uterus wall and can lead to preterm birth.

[8] Anemia may reflect poor diet and nutrition.

[9] Fourteen of the named medical risk factors and ten of the named complications of labor have a prevalence among blacks of less than 2 percent.

[10] Two other commonly used measures of infant health are very low birth weight (VLBW – birth weight less than 1500 grams) and intrauterine growth retardation, or small-for-gestational age (IUGR – low birth weight among full-term births). Though we repeat all of our analyses for these outcomes, we do not present these findings here since they add

weight less than 2500 grams. Preterm births are those with length of gestation less than 37 weeks.[11] We lose 174 observations due to missing birth weight and 360 observations due to missing gestational age.

While measurement of birth weight is both transparent and reliable, measurement of gestational age is subject to some controversy (Alexander and Allen 1996). Despite a lack of consensus on the optimal method of estimating gestational age, several studies have concluded that measuring length of gestation using the date of last menses (LMP) produces overestimates of preterm birth rates relative to other methods, including clinical estimates (Zhang and Bowes Jr 1995; Mustafa and David 2001). Therefore, estimating rates of preterm birth using birth certificate data is potentially problematic. Gestational age reported in the Natality Files is first computed using month, day, and year of birth and date of LMP. In cases where only day of LMP is missing, the NCHS imputes length of gestation (Taffel, Johnson et al. 1982). When the computed gestational age falls outside the NCHS's acceptable range of 17-49, the clinical estimate is used. Exploration of our sample reveals that the use of clinical estimates rose during the study period overall and among births we classify as preterm.[12] However, rates of preterm birth are

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no new information to our main conclusions. Also, very low rates of VLBW and IUGR (3 and 5 percent, on average) make it difficult to detect trends.

[11] We use preterm birth as both an outcome variable and as a control variable in the low birth weight regressions. When we treat preterm birth as an outcome, we use a bivariate indicator, but when we treat it as a control, we employ separate dummies for spontaneous and non-spontaneous preterm delivery, where non-spontaneous preterm births include induced labors and C-sections.

[12] The share of records for which the clinical estimate of gestational age is used rose from 0 percent in 1990 to 8 percent in 1991 and to 12.9 percent in 2001.

actually slightly higher among infants for which the clinical estimate of gestational age is used than among those for which date of LMP is used.[13]

Table 1-1 presents summary information for these and other variables included in our regression models, for all births and for black and nonblack births, separately, for the years 1990 and 2001. The health disadvantages of blacks are stark. On average over the twelve years we study, black infants are three times as likely as nonblack infants to be born low birth weight and twice as likely to born preterm. These disparities are correlated with substantial socioeconomic differences. Black mothers are younger and less well-educated than nonblack mothers. They are also more likely to have had a previous child and to have used tobacco or alcohol during pregnancy. Nearly three-quarters of black mothers were born in Washington, DC, compared with an average of just 8 percent of nonblack mothers. This difference is vast even when including women born in Maryland, Virginia, and West Virginia. Among nonblack mothers, one-third were born in states other than these three or the District of Columbia, and more than half were born outside the US.

As noted earlier, adequacy of prenatal care suffers from appreciable missing data. Education is missing/unknown for approximately 6 percent of women, on average. No other variables suffer from missing data.

The Times, They are a Changin'…

As evident in Table 1-1, Washington, DC underwent significant changes during the 1990s, many of which are relevant to infant health. In this section we discuss some of

[13] This difference – 1.3 percentage points – is statistically significant.

these changes and how they may have affected rates of low birth weight and preterm birth.

Illegitimacy and maternal age

As evidenced by its receipt of the Out-of-Wedlock Birth Reduction Bonus, the District of Columbia experienced substantial declines in nonmarital and teenage childbearing. The share of births to single mothers fell 7 percentage points between 1990 and 2001, and the share of births to teens fell 4 percentage points. Unmarried and teenage mothers have higher than average rates of low birth weight and preterm birth, and this could be due to several factors.

Unwed mothers are, on average, poorer than married mothers, and may be unable to depend on the logistical and psychological support of the baby's father. Financial hardship or lack of social support can, in turn, cause stress and anxiety, which some studies have linked to low birth weight and preterm birth (Rutter and Quine 1990; Goldenberg and Rouse 1998; Misra, O'Campo et al. 2001).

Because they are unmarried, single mothers may be less likely than married women to have employer-sponsored health coverage. To the extent that they work disproportionately in part-time and/or low-paying jobs, they may be unable to afford purchasing health insurance in the individual nongroup market (Currie 2003). Though many qualify for Medicaid, complicated enrollment and recertification requirements often present significant barriers to Medicaid participation (Glied and Remler 2003; Gardner, Lew et al. 2004; Klein, Glied et al. 2005). Lack of health insurance is likely to impact infant health through its association with restricted access to care (Schoen and DesRoches 2000; Glied and Little 2003). A few studies have shown that health insurance

expansions lead to greater utilization of prenatal care and improved birth outcomes (Currie and Gruber 1996; Long and Marquis 1998).

Like unwed mothers, teenage mothers tend to have grown up in economically disadvantaged circumstances and tend to have low education at the time of their first birth. The vast majority of teenage mothers are also unmarried.[14] Thus, risks associated with low education and single motherhood are also relevant to teenage mothers. However, there is little evidence that teen age *per se* is a causal factor in the health disadvantages of their infants. Once socioeconomic status is controlled for, the birth weight of infants to teens is comparable to that of older women (Strobino, Ensminger et al. 1995).

There are even several reasons to believe that among blacks, at least, a decline in teenage childbearing may not be advantageous for infant health outcomes. Several studies have shown that among women of low socioeconomic status, particularly among African-Americans, infants born to teens are healthier than those born to women in their 20s and 30s (Geronimus 1991; Geronimus 1992; Geronimus and Korenman 1992; Geronimus and Korenman 1993). Moreover, maternal age above age 30 is associated with increased risk of adverse pregnancy outcomes, either due to biological aging or increased lifestyle risks such as a higher incidence of prenatal smoking. Among poor black women in particular, heightened risk of adverse birth outcomes after the teen years may reflect the cumulative effects of stress and social and economic disadvantage (Geronimus, Hicken et al. 2006). Therefore, reduced teenage childbearing may not be beneficial for infant health because,

[14] Among teen mothers in our sample, 98 percent were unmarried at the time of birth.

first, the late teen ages may be ages of low risk, even relative to the 20s for some women, and, second, delaying pregnancy may result in shifts of fertility into ages of elevated risk.

Table 1-1 highlights that although nonmarital and teenage birth ratios declined significantly overall, they fell only modestly among the black population. The proportion of black infants born to unwed mothers rose slightly prior to 1994 and then declined (not shown), resulting in a net increase of 1 percentage point. Similarly, the proportion of black infants born to teens (ages 15-19) fell a modest 2 percentage points over the period. By contrast, aggregate declines in nonmarital and teenage birth ratios were 7 and 4 percentage points, respectively.

Thus, nonmarital and teenage childbearing did not trend strongly enough among blacks – the group that accounts for most of the District's health gains – to be a major factor in their infants' substantial health improvements. Moreover, the reductions in low birth weight and preterm birth occurred within marital status and maternal age groups, and especially among higher-risk unmarried and teenage women (not shown). Evidence also suggests that any improvements attributable to reduced teenage childbearing may have been at least partially offset by increased childbearing among women ages 35-39, whose share of births rose, and who are at higher risk of adverse birth outcomes.

Socioeconomic change: Education, migration, and other proxies

As in many other large US cities, the 1990s were a period of substantial economic growth and urban rebirth for Washington, DC (Brookings Institution Center on Urban and Metropolitan Policy 1999). Increases in the socioeconomic status of DC residents are reflected in part in higher educational attainment among new mothers. Table 1-1 shows

that the share of black mothers with less than a college education fell more than 12 percentage points, to 67 percent in 2001.[15]

Since education is negatively correlated with adverse birth outcomes (not shown), this change may have contributed to black infant health improvements. The disadvantages of low educational attainment may operate indirectly, through access to resources and medical care, or directly, through a limited ability to understand or follow medical advice. In economic terms, poorly educated women may be "inefficient producers" of prenatal and infant health (Grossman and Kaestner 1997). To the extent that less education is associated with substantial discounting of the future, women with low education may discount heavily the future health risks to their infants of current risky behaviors.[16] Improvements in education seem unlikely to explain black infant health gains in DC, however, since the decline in preterm birth occurred within education strata and the decline in low birth weight is largely limited to higher-risk, low education women (not shown).

Since changes in the educational attainment of DC mothers may not fully capture the health effects of socioeconomic changes in the District, we use mother's place of birth to proxy unobserved differences between low-risk mothers born in DC and those who migrated to DC during the 1990s. Socioeconomic effects might also be reflected in an increased willingness of mothers born outside DC to move to the District or to raise children there. We find, though, that the share of black women born in Washington, DC

[15] The population of the District may have become more affluent either because incomes improved among longer-term residents, migration altered favorably the socioeconomic composition of the District, or some combination of these factors.

[16] However, see Cutler and Glaeser (2005) for a new interpretation and evidence (Cutler and Glaeser 2005).

remained fairly constant over time, and also that health improvements occurred within group (not shown).

Not fully satisfied with using educational attainment and migration status to proxy socioeconomic status, we note also that low socioeconomic status or lack of health insurance – which is highly correlated with income and minority status – may be expressed through observable medical conditions and complications of labor and/or delivery (Monheit and Vistnes 2000; Ferry, Glied et al. 2004). Poverty may affect infant health, specifically, through the availability (or lack) of affordable, nutritious food in poor neighborhoods or through stress, which may trigger preterm labor (Grossman and Rashad 2004). However, socioeconomic disparities are also linked to poor health, in general (National Center for Health Statistics 1998). Poverty often implies financial, spatial, and qualitative lack of access to health care, and these can manifest themselves in health conditions of all kinds (Fossett, Perloff et al. 1991; Perloff, Kletke et al. 1995; Berk and Schur 1998; Currie 2003). Thus, reductions in medical risk factors or complications of labor may reflect unmeasured improvements in socioeconomic status. In fact, the share of black births to women with any medical risk factor fell from 35 percent to 29 percent between 1990 and 2001, and the share to women with any complication of labor declined 7.9 percentage points, to 33.6 percent.

Risky behaviors: Alcohol and tobacco

Cigarette smoking during pregnancy is a key determinant of infant health, and among black women in the District of Columbia, its incidence plummeted (Lightwood, Phibbs et al. 1999; England 2001). Prenatal smoking is more strongly associated with low birth weight than with preterm birth, operating on birth weight primarily through fetal

growth restriction (Kramer 1987; U.S. Department of Health and Human Resources 2001; U.S. Department of Health and Human Resources 2004). It is also associated with premature rupture of the membrane (PROM), infections, placenta previa, and placental abruption, which are themselves associated with preterm delivery (U.S. Department of Health and Human Resources 2004).[17]

Prenatal alcohol exposure is an additional, related risk factor for adverse birth outcomes (Day, D et al. 1989). Alcohol exposure during pregnancy can cause fetal alcohol syndrome, which is characterized by fetal growth retardation and other defects (Chomitz, Cheung et al. 1995).

Table 1-1 shows that the rate of prenatal smoking among blacks fell steeply by 15 percentage points, or 74 percent, during the 1990s. This decline greatly outpaced the national decline. Between 1990 and 1999, the national share of black women who smoked during pregnancy fell 43 percent (Matthews 2001). The number of cigarettes per day among smokers also fell. Prenatal alcohol use declined just as dramatically, from 6.4 percentage points in 1990 to 1.6 in 2001. The decline in racial disparity in smoking rates is of notice, as it suggests that trends in prenatal smoking may account for some of the narrowing of black/nonblack gaps in infant health. By contrast, the presentation of medical risk factors and complications of labor and the incidence of prenatal alcohol use trend similarly in sub-samples of blacks and nonblacks, indicating that they probably cannot account for relative reductions in adverse birth outcomes among blacks.

[17] For a description of the biological pathways through which smoking may affect birth weight and gestation, see the 2004 Surgeon General's Report and Cnattingius (1996) (Cnattingius 1996; U.S. Department of Health and Human Resources 2004).

Prenatal care

Prenatal care may be important for both infant and maternal health, particularly as it influences diet and nutrition, but evidence on the beneficial effects of prenatal care is limited. There is a positive correlation between weight gain during pregnancy and the infant's birth weight, and a wholesome diet can influence birth weight, fetal development, and general maternal health (Kleinman 1990). Additionally, early and regular monitoring of fetal development and the mother's physical health may reduce the chance of complications. Most studies of the effects of Medicaid expansions to pregnant women, however, find that greater utilization of prenatal care leads to modest or no improvement in infant health (Buescher, Roth et al. 1991; Fiscella 1995; Levinson and Ullman 1998; Joyce 1999; Dubay, Joyce et al. 2001). Evidence from the Women, Infants, and Children (WIC) program, which combines nutritional awareness with vouchers for baby formula, milk, eggs, and other healthy foods, is similarly discouraging (U.S. General Accounting Office 1992; Joyce, Gibson et al. 2004).

Unfortunately, we are unable to reliably examine trends in the adequacy of prenatal care in our data. The share of black women receiving inadequate prenatal care fell during the 1990s, but our regression results suggest that births with unknown adequacy should be more properly regarded as inadequate. The share of women receiving inadequate *or* unknown prenatal care was flat during the period.

DC Healthy Start

During the 1990s, Washington, DC launched Healthy Start, a set of federally funded, community-based programs intended to reduce infant mortality in high-risk areas. However, the timing and scale of this program do not correspond well to the trends

we observe District-wide in our study period. Prior to 1997, the program operated in only two of the District of Columbia's eight wards and was implemented gradually, between 1992 and 1994. Additionally, there is little statistical evidence that DC Healthy Start achieved its goals. A comprehensive evaluation of the program through 1996 concluded that Healthy Start had a positive effect on just one of ten outcomes examined, and that effect amounted to a reduction in low birth weight of less than 10 percent, or 1.7 percentage points (Moreno, Devaney et al. 2000). For these reasons, we do not attempt to estimate the effect of DC Healthy Start in our regression analyses.

Regression Approach

The trends we have described thus far suggest that sociodemographic changes cannot fully account for the District of Columbia's gains in black infant health and consequent reductions in racial health disparities. Declines in nonmarital and teenage childbearing among African-Americans were modest, and improvements in health occurred within high-risk sociodemographic groups. In this section, we use regression analysis to estimate formally the contributions of demographic characteristics and other factors to changes in black infant health.

We estimate the following equation for each of our four birth outcomes using linear probability models with robust standard errors to correct for heteroscedasticty:[18]

$$outcome = \alpha + \beta * year + \gamma * sex + \delta' X \qquad (1)$$

[18] We estimate linear probability models (rather than logits or probits) because they are easy to interpret and directly yield the parameters of greatest interest (probability derivatives). Furthermore, Norton and colleagues have demonstrated that uncovering the probability derivatives from logit and probit models is not straightforward, especially in models with interaction terms, and the results of these models are nearly universally misinterpreted by researchers (Ai and Norton 2003).

The variable, *year*, is a linear time trend chosen on the basis of graphical analysis (Figure 2). β, or the coefficient on the trend term, is the parameter of most interest. We build the model incrementally, observing how $\hat{\beta}$ changes as we vary the covariate set contained in the vector *X*. We show estimated β's for each of our models in the first column of Tables 1-2 and 1-3. Coefficients reported in all tables are inflated by a factor of 100, and so the coefficient of the trend term represents the average yearly percentage point change in the outcome. Multiplying this by twelve yields the percentage point change over the entire period studied. We present analogous tables for singleton births to nonblack women in Tables 1A-1 and 1A-2 in Appendix 1A.[19]

Results

Model 1 of Tables 1-2 and 1-3 documents the tremendous declines in low birth weight and preterm birth, adjusted only for the infant's sex. The rate of low birth weight declined 0.38 percentage points per year, or 4.6 percentage points over 12 years. The rate of preterm birth declined 0.51 percentage points per year, or 6.1 percentage points over 12 years.

The role of demographic changes

Since we are especially interested in whether reductions in out-of-wedlock or teenage childbearing contributed to health improvements, Models 2 and 3 add controls for marital status and maternal age, separately, to the base specification (Model 1). Comparing the estimated β in Models 1 and 2, for example, reveals how much of the

[19] Regressions on the sample of nonblacks include an additional regressor for race/ethnicity not included in the regressions for blacks.

trend in the outcome is due to changes in marital status. Models 5-6 test for socioeconomic effects by adding controls for maternal education and mother's place of birth. Model 6 also adds a control for parity in order to improve the precision of the coefficients contained in $\hat{\delta}$.[20]

Models 2-5 reveal that among blacks no more than 8 percent of the estimated reduction in either the rate of low birth weight or the rate of preterm birth between 1990 and 2001 is due to declines in the shares of births to unmarried, teenage, or less-educated mothers, whether these characteristics are entered into the regression alone or in combination. When all races are included, black racial identification accounts for about a quarter of the downward trend in low birth weight and one-fifth of the decline in preterm birth (not shown), but marital status, maternal age, and maternal education still explain little, if any, of the decline in either outcome above and beyond that captured by race. Models 5 and 6 suggest little role for changes in socioeconomic status. Adjustments for maternal education and mother's place of birth also have minimal, if any, effects on the estimated declines in birth outcomes.[21]

Additional hypotheses

Model 6 then becomes the base specification for the remaining models. Models 7 and 8 test the hypothesis that reductions in prenatal tobacco or alcohol use contributed to

[20] First pregnancies may be more likely to result in an adverse outcome if they are more stressful or if higher order births reflect selection for good maternal health. On the other hand, first-time mothers may be less likely to deliver a low birth weight infant since first pregnancies are less likely to deliver prematurely.

[21] Mother's place of birth may not have any effect in the sample of blacks because the shares of black mothers born in DC or in nearby states changed little. Controlling for place of birth has a modest effect on the trend for some outcomes in the sample of nonblacks, however.

reproductive health improvements. Model 9 examines whether changes in the adequacy of prenatal care influenced birth outcomes, and Model 10 includes a control for previous preterm or small-for-gestational age birth. We then add controls for spontaneous and non-spontaneous preterm delivery to the low birth weight regressions, treating it as a proximate cause. This allows us to identify the role of gestational age in the incidence of low birth weight. Our final two models add controls for medical risk factors, and complications of labor (Models 11-13). Since we are interested in preterm birth and maternal health as mediating factors reflecting unmeasured true causes, we do not interpret the coefficients of these variables, but only consider how their inclusion in the models affects the trend coefficient.

The role of prenatal smoking

Models 7-10 demonstrate the importance of tobacco use. When prenatal smoking is added into the model, the trend coefficient in the low birth weight regression falls from –0.42 to –0.26. Thus, the reduction in smoking accounts for 38 percent of the decline in the rate of low birth weight and 20 percent of the decline in preterm birth not already attributed to changes in demographics, birth order, and maternal place of birth (Model 7). Moreover, neither alcohol use during pregnancy nor adequacy of prenatal care contributes appreciably to the estimated reductions in low birth weight or preterm birth beyond those captured by prenatal tobacco use (Models 8-9). A similar finding applies to changes in the share of women with a previous preterm or small-for-gestational-age birth (Model 10). Controlling for prenatal care actually increases the incidence of adverse birth outcomes, suggesting that changes in prenatal care utilization contributed to rather than

improved the black infant health (Model 9). However, significant measurement error in the adequacy of prenatal care may bias any estimates of its effects on birth outcomes.

Preterm birth as a proximate cause

Not surprisingly, preterm birth is an important predictor of low birth weight. The effect of the decline in preterm birth on the estimated trend in low birth weight is both large and distinct from that of prenatal smoking (Model 11). The trend coefficient in the low birth weight regression falls 0.17 percentage points when we control for preterm birth (Model 10 vs. 11). In other words, the decline in preterm birth accounts for 74 percent of the decline in low birth weight not already attributed to changes in demographics, birth order, maternal place of birth, prenatal alcohol and tobacco use, and prenatal care utilization. The regression-adjusted reduction in low birth weight in Model 11 is only 0.06 percentage points per year, and is not significantly different from zero at the 5 percent level. As evidenced by the reduction in the smoking coefficient when we add a control for preterm birth (not shown), some of the association between the reduction in low birth weight and decreased prenatal smoking rates operates through preterm birth. However, the dramatic change in the trend coefficient between models 10 and 11 shows that preterm birth, or the unmeasured risks associated with it, have an effect on low birth weight distinct from that of prenatal tobacco exposure.

Medical conditions

Models 12-13 suggest that poverty, improved medical technology, and/or some other unmeasured, underlying cause associated with the presentation of medical conditions may also be important. Controlling for medical risk factors reduces the trend in low birth weight an additional 0.02 percentage points, indicating that changes in the

prevalence of medical risk factors contributed to the decline in low birth weight (Model 12). Changes in the share of women with complications of labor may have also lowered the rate of low birth weight (Model 13). In neither of these models is the trend coefficient statistically significant.

Complications of labor have a particularly strong (beneficial) effect on the estimated trend in preterm birth. Controlling for medical risk factors reduces the trend in preterm birth to 0.45 percentage points, and controlling for complications of labor reduces that trend even further, to 0.35 percentage points, or by 22 percent (Models 11-12). The trend coefficient in the final model is still significantly different from zero and only 17 percent lower than in the model adjusted only for infant's sex. Declines in the prevalence of medical risk factors and complications of labor, perhaps representing changes in unmeasured causes of poor maternal health, are partially responsible for reductions in preterm birth, but there remains a substantial portion that we cannot account for either directly, or indirectly.

Cross-sectional effects

In order to lend confidence to our models and data, we show the full set of coefficients for Model 6 in Table 1A-5 of Appendix 1A. These results confirm the importance of sociodemographic risk factors for adverse birth outcomes. Black women who are married or have at least some college education are significantly less likely to bear low birth weight or preterm infants. First pregnancies are less likely to result in preterm delivery, but more likely to result in a low birth weight infant. Women born outside the US or in states other than Maryland, Delaware Virginia, or West Virginia are at lower risk of adverse birth outcomes relative to those born in DC. The insignificance of

the coefficient for mothers born in MD, DE, VA, or WV relative to those born in DC suggests either that any relevant differences between women born in DC and migrants from nearby states are already captured by other controls or that maternal place of birth is an inadequate measure for these differences. This may explain, in part, our finding that controlling for maternal place of birth does not affect estimated trends in birth outcomes.

Contrary to the conventional hypothesis that young age is disadvantageous for infant health, black teenagers are at lower risk of adverse pregnancy outcomes than any other age group. Furthermore, risk rises monotonically with age after the teen years, for low birth weight, and after age 24, for preterm birth. Though we control for sociodemographic characteristics in the model shown in the table, this finding is robust to model specification, including the simple model that adjusts only for infant sex and maternal age. This pattern is unique to blacks, implying that racial disparities in infant health also rise with age, and is consistent with the weathering hypothesis proposed by Geronimus, in which the impact of socioeconomic disadvantage accumulates and produces a positive age-risk profile among low SES black mothers (Geronimus 1986).

Taken literally, these results suggest that improvements in black infant health would have been even greater had maternal age not risen in the 1990s. However, a study of birth outcomes in the 1980s and 1990s suggests that age effects reflect better health among younger cohorts rather than weathering (Almond and Chay 2003). We show in Appendix 1B that including cohort dummies (and dropping the time trend) eliminates the weathering effect only in models that do not also include controls for education.

Table 1A-6 of Appendix 1A presents coefficients from Model 10 for tobacco and alcohol use during pregnancy, adequacy of prenatal care, and pregnancy history. As

expected, prenatal smoking has large, detrimental, and significant effects on infant health. Tobacco use during pregnancy is associated with an increase in the likelihood of low birth weight among blacks of 5.3 percentage points, and this probability increases by 0.3 percentage points for every additional cigarette smoked. The estimated effect of prenatal smoking on low birth weight is greater here than in the model in which we also control for preterm delivery (not shown). This suggests that some of the effects of smoking operate through preterm birth.

Though changes in alcohol use and prenatal care do not contribute to trends in infant health beyond that already attributed to smoking, they do affect the probability of adverse outcomes. Alcohol use is associated with a 10.5 percentage point increase in the likelihood of low birth weight and has a comparable relationship to preterm birth.[22] Like smoking, prenatal alcohol use appears to have both direct and indirect (through preterm birth) effects on low birth weight. Intermediate and adequate prenatal care are associated with better infant health and have larger effects on the probability of preterm birth than low birth weight.

Perhaps counter-intuitively, "adequate plus" prenatal care is associated with worsened infant health. This may reflect endogeneity bias. Women at the highest risk seek prenatal care earlier and require more frequent visits. When we control for preterm birth, "adequate plus" prenatal care is negatively associated with low birth weight, and its beneficial effect is greater than that of adequate or intermediate care.

In summary, infant health improvements among African-Americans in Washington, DC over the period 1990-2001 cannot be attributed to declines in nonmarital

[22] Oddly, the coefficient on prenatal alcohol use is negative in regressions on the sample of nonblacks. However, it is never statistically significant.

and teenage childbearing. We identify two key factors in the decline in low birth weight – a reduction in prenatal smoking and a decline in preterm deliveries. However, we view preterm birth primarily as a proxy for unmeasured risk factors rather than a direct cause. Also, the effects of prenatal smoking may reflect unmeasured risky behaviors such as crack cocaine use. In exploratory analyses in Appendix 1C, we are unable to reject this possibility. We find evidence that prenatal smoking (and/or unmeasured correlates) influences low birth weight indirectly through preterm birth as well as directly. We also find evidence that changes in unmeasured risk, perhaps associated with decreased poverty and which we proxy using the presentation of medical risk factors and complications of labor, contributed to the decline in low birth weight both directly and indirectly, through preterm birth. However, the unmeasured causes of preterm birth and medical conditions contribute independently to the trend in low birth weight, suggesting that at least some of these unobserved factors that influence preterm birth differ from those that influence medical conditions.

Robustness checks

In this section, we examine the sensitivity of our results to select sample restriction, measurement, and methodological decisions. Our main conclusions are unaltered by all of these alternative considerations.

Sample restrictions

Our primary sample consists of singleton births to African-American women ages 15-39, who were residents of DC and gave birth in DC. Since multiple gestations have elevated risk for adverse birth outcomes, excluding them reduces rates of low birth

weight and preterm birth.[23] Multiple gestations as a share of all births rose during the 1990s. This implies that the degree of under-measurement of adverse birth outcomes resulting from their exclusion is greater in 2001 than in 1990. In other words, restricting the sample to singleton births overstates improvements in infant health. An analogous concern arises from excluding births to women ages 40 or older.[24] However, since multiple gestations and births to women in their 40s and 50s represent only a tiny share of all births, rates of low birth weight and preterm birth declined significantly even when we include them.[25]

Nonetheless, we alter our main sample by including either multiple gestations or women of all ages and re-estimate all of our regression models. These results are shown in the fourth and fifth columns of Tables 1-2 and 1-3. Changes in sociodemographic characteristics still cannot explain reductions in low birth weight and preterm birth rates, and the decline in prenatal smoking remains the most important identifiable factor.

The same is true when we expand the sample to births occurring in Washington, DC to non-DC residents or to births occurring outside the District to DC residents (Columns 2 and 3 of Tables 1-2 and 1-3). Our main sample requires that the District of Columbia be both the state of occurrence and state of residence. Excluding nonresidential

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[23] The average rate of low birth weight in our primary sample is 15 per 100 live births compared with 17 when we include multiple gestations.

[24] Increased risk for adverse birth outcomes among women ages 40 or older relative even to women in their 30s is especially pronounced among black women. Black women over age 39 have a rate of low birth weight nearly 10 percentage points higher than all younger women of color and 4 percentage points higher than women in their 30s.

[25] Births to women ages 40 and older represent less than 2 percent of black singleton births, and multiple gestations represent less than 3 percent of black births to women ages 15-39.

births that occur in DC raises the concern that infant health improved because lower-risk women from the surrounding suburbs became more likely to deliver in DC hospitals. This trend is evident among blacks, but not among nonblacks (not shown). One explanation for this may be that urban revitalization spread to private hospitals in the District. Excluding resident births that occur outside the District raises concerns that infant health improved because high-risk women became more likely to deliver in neighboring suburbs. Budget cuts led to downsizing of a major public hospital, DC General, towards the end of the 1990s, and its eventual closing in 2001, and this may have shifted receipt of health care to surrounding counties. However, the share of black women living in DC that gave birth in DC increased slightly between 1990 and 2001 (not shown). Moreover, as the results in Tables 1-2 and 1-3 demonstrate, neither restriction impacts our main conclusions.[26]

Measurement issues

We also re-estimate our models using alternative measures of prenatal smoking and adequacy of prenatal care. Though the Kessner Index of adequacy of prenatal care is considered inferior to the Kotelchuck Index and evidence suggests that failure to control for smoking dosage would result in specification bias, we examine whether our conclusions are sensitive to these considerations. These results are reported in Columns 6 and 7 of Tables 1-2 and 1-3. The trend coefficients from these models are virtually identical to those in our main analysis. One exception is that changes in prenatal care as measured by the Kessner Index have a very modest effect on the change in preterm birth.

[26] One exception occurs among nonblacks. In the sample that includes DC births to non-DC residents, the trend in smoking has no impact on the change in either low birth weight or preterm birth (Tables 1A-1 and 1A-2 in Appendix 1A).

As expected, the estimated effect of prenatal tobacco use on infant health is greater in models that do not also control for frequency of use. Like the sample restrictions, however, using alternative measures of smoking and adequacy of prenatal care do not alter our conclusions about the lack of explanatory power of sociodemographic changes or the substantial role of reduced smoking in the health gains of black infants in Washington, DC.

Linear probability vs. logit models

We estimate linear probability models because their coefficients are easy to interpret, but they are often criticized because predictions from linear probability models are not constrained to lie between 0 and 1. Logit models solve this problem, but their coefficients (or odds ratios) are difficult to translate into probabilities. The last columns of Tables 1-2 and 1-3 show that estimating logit models does not impact our primary findings.

Discussion

The 1996 PRWORA represented a significant shift in the federal government's approach to welfare, and its perceived effects continue to inform policy ten years after the fact. One scholar concluded prior to welfare reform that with respect to Washington, DC, high rates of illegitimacy among blacks were responsible for high rates of black infant mortality (Eberstadt 1994). During the 1990s, Washington, DC experienced exceptional and contemporaneous declines in nonmarital fertility and improvements in infant health. Some may view this as evidence of a link between illegitimacy and adverse birth

outcomes. Welfare reauthorization signed into law recently followed the original reform law's precedent of allocating federal funds for reducing out-of-wedlock childbearing.

This study finds, however, that sociodemographic changes account for little of the dramatic infant health improvements in Washington, DC during the 1990s. Based on these findings, it would be inappropriate to attribute improvements in infant health outcomes to welfare reform-driven reductions in out-of-wedlock or teenage childbearing or other demographic shifts. Despite a substantial decline in the proportions of births to unmarried women and teens of all races, improvements in infant health took place largely among black mothers, and among blacks the share of births to unwed mothers declined only modestly. Additionally, most health gains resulted from improvements within the populations at highest risk of adverse birth outcomes, especially less-educated and unmarried black women. Delayed childbearing among blacks in this period may even have worsened birth outcomes since teen age was found to be protective for infant health. There is also very little evidence that welfare reform was responsible for changes in marriage or fertility behavior in the 1990s. Therefore, if welfare reform were responsible for reducing adverse birth outcomes, it would have to have been through channels other than marriage or fertility behavior (e.g., perhaps through increased employment or decreased poverty).

Instead, a steep decline in prenatal tobacco exposure is the most powerful explanation for infant health gains, though a sharp reduction in preterm birth, or the true underlying causes it reflects, also comprise a substantial portion of the reduction in low birth weight. It is tempting to interpret these results to mean that interventions or improved knowledge about the health hazards of smoking were successful in improving

the health of black mothers and infants, but we there are at least two reasons to approach this interpretation with caution. First, the late 1980s and early 1990s were characterized by elevated crack use, which may be correlated with smoking, and so the infant health improvements we observe in the 1990s may reflect a regression to the mean following the health deteriorating effects of increased crack use in the 1980s. Since we lack a reliable measure of crack use and do not have a measure of tobacco use spanning the 1980s and 1990s, it is difficult to study the effects of crack and tobacco simultaneously, but our analyses in Appendix 1C do not allow us to reject this hypothesis. Second, assuming crack use is not an important factor, the aggregate effect of further reductions in prenatal smoking on birth outcomes may be small given that rate of prenatal exposure to tobacco is now very low – only 5 percent of black pregnant women smoked in 2001. The nonblack experience of a decline in tobacco use from an already low rate and a relatively flat trend in infant health is suggestive of the limitations of future reductions in smoking for improving the health of black infants.

Table 1 - 1. Summary Statistics for Singleton Births to Women in Washington, DC, 1990 & 2001

Percent, unless indicated otherwise

	1990			2001		
	All	*Blacks*	*Nonblacks*	*All*	*Blacks*	*Nonblacks*
Number of births	*10,164*	*7,934*	*2,230*	*6,363*	*4,193*	*2,170*
Outcomes						
Low birth weight (LBW)	14.9	17.1	7.0	10.2	12.9	5.0
Preterm (PRETERM)	21.1	23.6	12.1	14.9	18.3	8.4
Very low birth weight (VLBW)	3.6	4.2	1.8	2.4	3.2	0.9
Intrauterine growth retardation (IUGR)	6.0	7.1	2.7	4.0	5.1	2.0
Covariates						
Female infant (FEMALE)	49.5	49.2	50.5	49.4	49.6	49.0
Non-Hispanic black (BLACK)	78.1	100.0	0.0	65.9	100.0	0.0
Married (MARRIED)	31.5	21.5	67.4	38.6	20.4	73.7
Maternal age (AGE)						
15-19	18.6	21.6	8.1	14.4	19.2	5.2
20-24	28.0	30.0	20.9	25.3	30.8	14.6
25-29	25.9	26.2	25.0	22.6	23.7	20.6
30-34	18.9	16.4	27.7	23.5	17.3	35.5
35-39	8.5	5.7	18.3	14.2	9.1	24.1
Maternal educational attainment (EDUC)						
< High school	33.1	33.4	32.1	23.4	23.1	24.1
High school	39.9	46.3	16.9	33.2	44.4	11.7
College +	26.1	19.6	49.4	35.8	24.2	58.4
Unknown	0.9	0.7	1.7	7.5	8.4	5.9

Table continues on the next page. See notes at the end of the table.

Table 1 - 1, continued

	1990			2001		
	All	*Blacks*	*Nonblacks*	*All*	*Blacks*	*Nonblacks*
Parity (PARITY)						
0	39.4	36.6	49.6	42.1	37.3	51.5
1+	60.5	63.4	50.4	57.9	62.7	48.5
Unknown	0.0	0.0	0.0	0.0	0.0	0.0
Mother's place of birth (MPLB)						
DC	60.4	73.5	13.7	51.0	73.6	7.4
DE/MD/VA/WV	5.6	6.1	3.7	5.5	6.4	3.8
Other state	18.1	14.2	32.3	18.4	10.4	33.8
Foreign	15.9	6.2	50.3	25.1	9.6	55.0
Prenatal tobacco use (TOBACCO)	17.0	20.2	5.8	3.6	5.2	0.6
No. cigarettes/day conditional on use (TOBXCIGNUM)	10.0	10.1	9.1	7.0	7.1	5.1
Alcohol use during pregnancy (ALCOHOL)	6.4	6.4	6.6	1.4	1.6	1.0
Prenatal care (PNC)						
Inadequate	40.6	44.6	26.4	23.5	28.5	13.6
Intermediate	18.3	18.9	16.3	21.6	20.2	24.1
Adequate	25.3	21.6	38.5	25.9	20.9	35.5
Adequate Plus	12.0	10.9	15.7	12.9	13.1	12.5
Unknown	3.8	4.0	3.1	16.2	17.2	14.1

Table continues on the next page. See notes at the end of the table.

Table 1 - 1, continued

	1990			2001		
	All	*Blacks*	*Nonblacks*	*All*	*Blacks*	*Nonblacks*
Pregnancy term (TERM)						
Full-term birth	78.9	76.4	87.9	85.1	81.7	91.6
Spontaneous preterm birth	15.1	16.9	9.0	8.5	10.5	4.7
Nonspontaneous preterm birth	6.0	6.8	3.1	6.4	7.8	3.8
Previous preterm or IUGR birth (PREV)	1.0	1.2	0.3	0.4	0.4	0.4
Any medical risk factor (ANYMRF)	33.6	34.8	29.4	26.4	28.5	22.2
Any complications of labor (ANYCLB)	40.5	41.5	37.0	32.6	33.6	30.8

Notes:

1 Variable names are in parentheses.

2 Each MRF and each CLB is entered into the regressions as a separate dummy.

Table 1 - 2. Regression-adjusted Trends in Low Birth Weight Among Blacks in Washington, DC, 1990-2001

			Robustness checks						
			Sample				**Data**		**Estimation**
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
1	FEMALE	-0.38*	-0.30*	-0.33*	-0.36*	-0.33*			-2.98* [0.971*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.31 [0.003])
2	FEMALE, MARRIED	-0.38*	-0.29*	-0.33*	-0.36*	-0.33*			-3.03* [0.970*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.31 [0.003])
3	FEMALE, AGE	-0.41*	-0.33*	-0.35*	-0.40*	-0.36*			-3.26* [0.968*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.31 [0.003])
4	FEMALE, MARRIED, AGE	-0.43*	-0.34*	-0.37*	-0.42*	-0.38*			-3.40* [0.967*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.31 [0.003])
5	FEMALE, MARRIED, AGE, EDUC	-0.43*	-0.33*	-0.37*	-0.42*	-0.38*			-3.47* [0.966*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.32 [0.003])

Table continues on the next page. See notes at the end of the table.

Table 1 - 2, continued

			Robustness checks						
			Sample			Data			Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
6	FEMALE, MARRIED, AGE, EDUC, PARITY, MPLB	-0.42*	-0.32*	-0.36*	-0.42*	-0.38*			-3.43* [0.966*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.32 [0.003])
7	6 + TOBACCO, TOBXCIGNUM	-0.26*	-0.19*	-0.21*	-0.25*	-0.21*	-0.26*	-0.26*	-2.14* [0.979*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.33 [0.003])
8	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	-0.23*	-0.17*	-0.19*	-0.23*	-0.19*	-0.23*	-0.24*	-2.01* [0.980*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.33 [0.003])
9	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	-0.27*	-0.22*	-0.21*	-0.27*	-0.24*	-0.24*	-0.28*	-2.38* [0.977*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.34 [0.003])

Table continues on the next page. See notes at the end of the table.

Table 1 - 2, continued

			Robustness checks						
			Sample			Data			Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	-0.23*	-0.18*	-0.19*	-0.23*	-0.21*	-0.20*	-0.24*	-2.07* [0.979*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.34 [0.003])
11	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV, TERM	-0.06	-0.04	-0.03	-0.05	-0.05	-0.07*	-0.06	-0.75* [0.993*]
		(0.04)	(0.03)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.38 [0.004])
12	11 + MRF(16)	-0.04	-0.02	-0.02	-0.04	-0.03	-0.06	-0.05	-0.60 [0.994]
		(0.04)	(0.03)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.38 [0.004])
13	11 + MRF(16), CLB(16)	0.03	0.04	0.04	0.03	0.04	0.02	0.02	0.20 [1.002]
		(0.04)	(0.03)	(0.03)	(0.03)	(0.04)	(0.04)	(0.04)	(0.39 [0.004])
	Approximate N	71,000	116,000	77,000	73,000	73,000	71,000	71,000	71,000

See notes on the next page.

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 1-1 for variable definitions and categories.

3 The Kessner Index is coded as: Inadequate (reference category), Intermediate/Adequate, Unknown.

4 Rather than catchall "any" dummies for MRF and CLB, which are shown in Table 1-1, regressions use 16 separate dummies, each, for MRF and CLB.

5 For the logit regressions, we report both coefficients and odds ratios. The latter (and their SEs) are in brackets.

Table 1 - 3. Regression-adjusted Trends in Preterm Birth Among Blacks in Washington, DC, 1990-2001

			Robustness checks						
			Sample				**Data**		**Estimation**
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
1	FEMALE	-0.51*	-0.41*	-0.45*	-0.49*	-0.44*			-3.10* [0.969*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.27 [0.003])
2	FEMALE, MARRIED	-0.51*	-0.40*	-0.45*	-0.50*	-0.45*			-3.15* [0.969*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.27 [0.003])
3	FEMALE, AGE	-0.53*	-0.44*	-0.47*	-0.53*	-0.47*			-3.27* [0.968*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.27 [0.003])
4	FEMALE, MARRIED, AGE	-0.55*	-0.44*	-0.49*	-0.54*	-0.49*			-3.39* [0.967*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.28 [0.003])
5	FEMALE, MARRIED, AGE, EDUC	-0.52*	-0.41*	-0.46*	-0.52*	-0.46*			-3.27* [0.968*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.28 [0.003])

Table continues on the next page. See notes at the end of the table.

Table 1 - 3, continued

			Robustness checks						
			Sample			Data			Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
6	FEMALE, MARRIED, AGE, EDUC, PARITY, MPLB	-0.52*	-0.41*	-0.46*	-0.52*	-0.46*			-3.28* [0.968*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)			(0.28 [0.003])
7	6 + TOBACCO, TOBXCIGNUM	-0.42*	-0.33*	-0.37*	-0.41*	-0.36*	-0.42*	-0.42*	-2.69* [0.973*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.05)	(0.04)	(0.04)	(0.29 [0.003])
8	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	-0.41*	-0.32*	-0.35*	-0.40*	-0.35*	-0.41*	-0.41*	-2.61* [0.974*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.05)	(0.04)	(0.04)	(0.29 [0.003])
9	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	-0.51*	-0.42*	-0.43*	-0.50*	-0.47*	-0.39*	-0.51*	-3.43* [0.966*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.05)	(0.04)	(0.30 [0.003])

Table continues on the next page. See notes at the end of the table.

Table 1 - 3, continued

			Robustness checks						
			Sample				Data		Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	-0.47*	-0.39*	-0.43*	-0.47*	-0.44*	-0.35*	-0.47*	-3.22* [0.968*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.05)	(0.04)	(0.30 [0.003])
11	10 + MRF(16)	-0.45*	-0.36*	-0.40*	-0.44*	-0.41*	-0.32*	-0.45*	-3.05* [0.970*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.05)	(0.04)	(0.30 [0.003])
12	10 + MRF(16), CLB(16)	-0.35*	-0.26*	-0.31*	-0.34*	-0.31*	-0.23*	-0.35*	-2.52* [0.975*]
		(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.32 [0.003])
	Approximate N	71,000	115,000	77,000	72,000	73,000	71,000	71,000	71,000

See notes on the next page.

* p<0.05

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

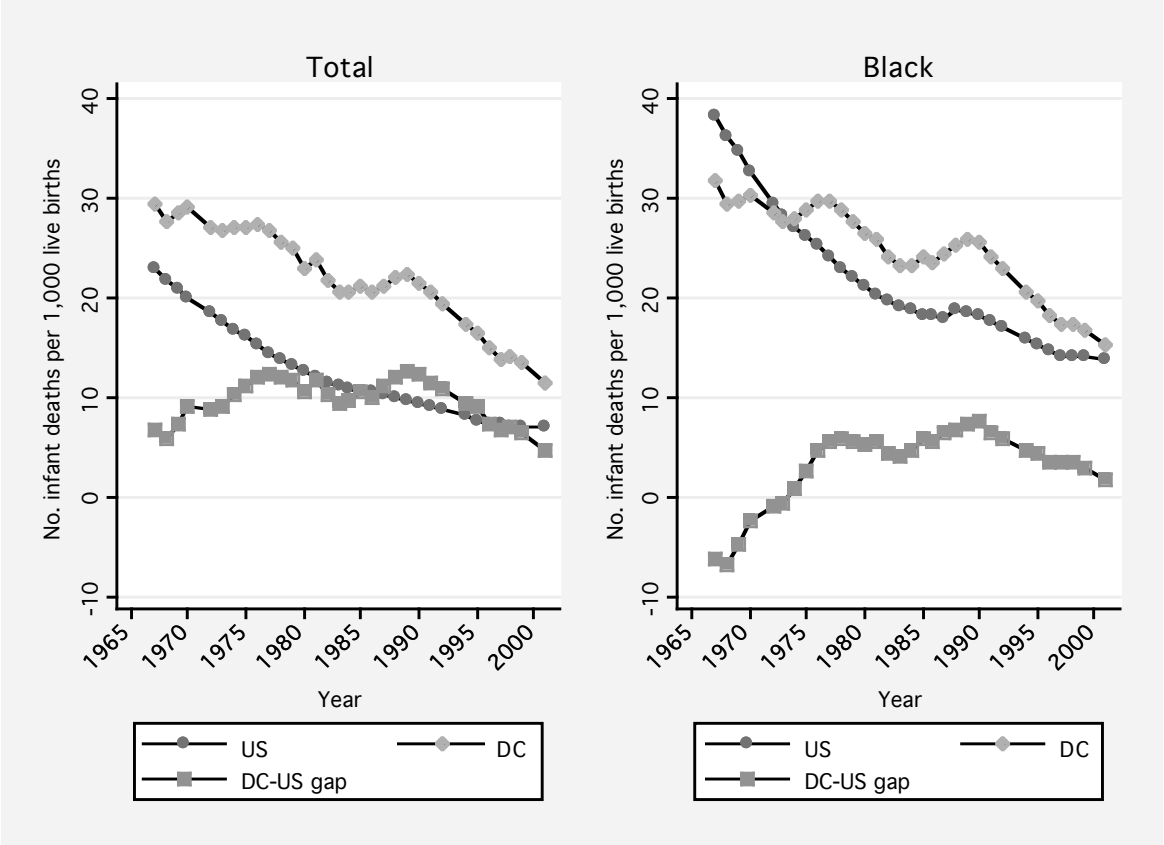
2 See Table 1-1 for variable definitions and categories.

3 The Kessner Index is coded as: Inadequate (reference category), Intermediate/Adequate, Unknown.

6 Rather than catchall "any" dummies for MRF and CLB, which are shown in Table 1-1, regressions use 16 separate dummies, each, for MRF and CLB.

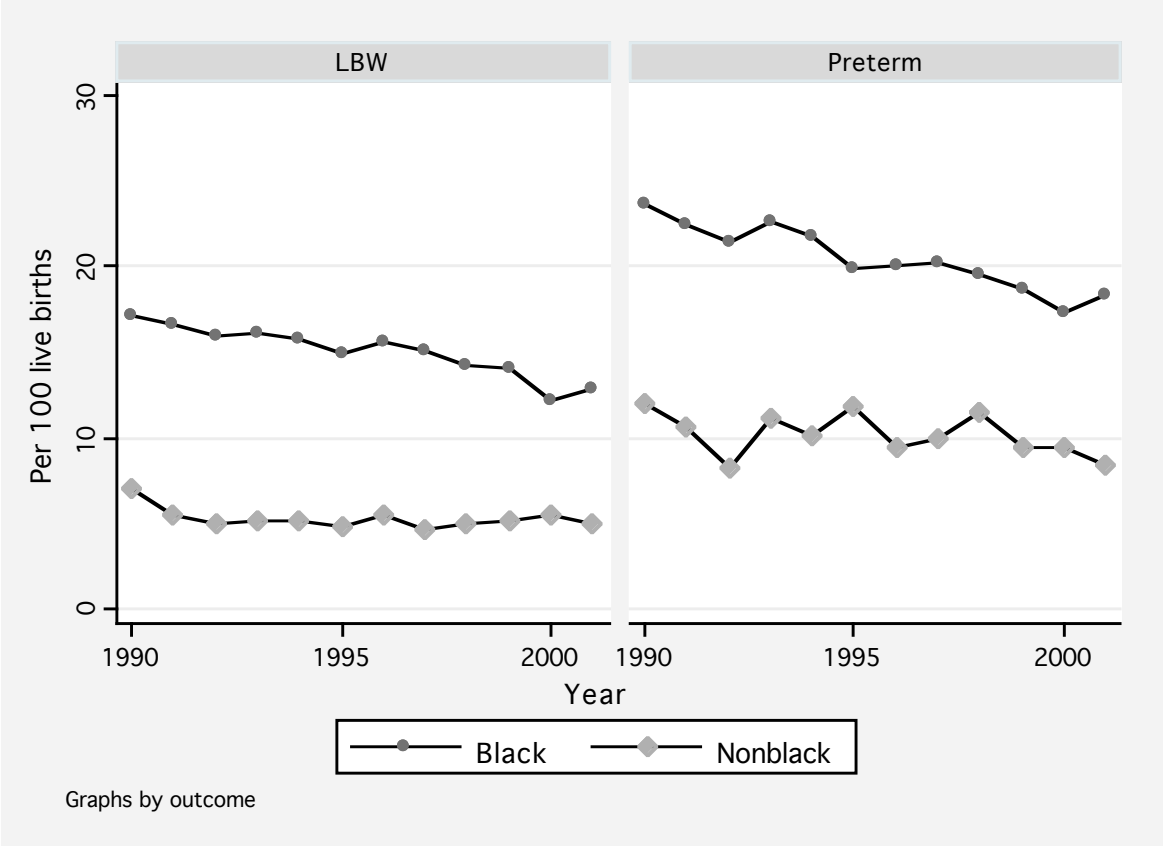
5 For the logit regressions, we report both coefficients and odds ratios. The latter (and their SEs) are in brackets.

Figure 1 - 1. Historical Trends in Infant Mortality Rates by Race, 1967-2001



Source: National Center for Health Statistics, "Health, United States", 1981, 2004.
Notes: 3-year moving averages. Prior to 1995-97, figures for blacks include Hispanic blacks.

Figure 1 - 2. Birth Outcomes by Race, 1990-2001



Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births in Washington, DC to women aged 15-39.

Appendix 1A: Supplemental Tables for Primary Analyses

Table 1A - 1. Regression-adjusted Trends in Low Birth Weight Among Nonblacks in Washington, DC, 1990-2001

			Robustness checks						
			Sample				Data		Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
1	FEMALE	-0.08	-0.13*	-0.06	-0.06	-0.02			-1.66 [0.984]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.85 [0.008])
2	FEMALE, RACE	-0.08	-0.14*	-0.05	-0.05	-0.01			-1.51 [0.985]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.86 [0.008])
3	FEMALE, MARRIED	-0.07	-0.14*	-0.05	-0.05	-0.01			-1.40 [0.986]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.86 [0.008])
4	FEMALE, AGE	-0.07	-0.12*	-0.05	-0.05	-0.02			-1.40 [0.986]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.85 [0.008])
5	FEMALE, RACE, MARRIED	-0.06	-0.14*	-0.04	-0.04	0.00			-1.25 [0.988]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.85 [0.008])

Table continues on the next page. See notes at the end of the table.

Table 1A – 1, continued

			Robustness checks						
			Sample				Data		Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
6	FEMALE, RACE, MARRIED, AGE	-0.06	-0.13*	-0.04	-0.04	-0.01			-1.28 [0.987]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.86 [0.008])
7	FEMALE, RACE, MARRIED, AGE, EDUC	-0.06	-0.12*	-0.04	-0.04	-0.01			-1.25 [0.988]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.86 [0.009])
8	FEMALE, RACE, MARRIED, AGE, EDUC, PARITY, MPLB	-0.06	-0.12*	-0.04	-0.04	0.00			-1.22 [0.988]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)			(0.86 [0.008])
9	6 + TOBACCO, TOBXCIGNUM	-0.04	-0.11*	-0.01	-0.02	0.02	-0.04	-0.04	-0.76 [0.992]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.05)	(0.04)	(0.04)	(0.86 [0.009])

Table continues on the next page. See notes at the end of the table.

Table 1A – 1, continued

			Robustness checks						
			Sample			Data			Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	-0.05 (0.04)	-0.11* (0.02)	-0.02 (0.04)	-0.03 (0.04)	0.01 (0.05)	-0.05 (0.04)	-0.05 (0.04)	-0.89 [0.991] (0.87 [0.009])
11	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	-0.04 (0.04)	-0.12* (0.02)	-0.01 (0.04)	-0.02 (0.04)	0.02 (0.05)	-0.06 (0.04)	-0.04 (0.04)	-0.84 [0.992] (0.90 [0.009])
12	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	-0.04 (0.04)	-0.12* (0.02)	-0.01 (0.04)	-0.02 (0.04)	0.02 (0.05)	-0.05 (0.04)	-0.04 (0.04)	-0.80 [0.992] (0.90 [0.009])
13	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV, TERM	-0.04 (0.04)	-0.09* (0.02)	-0.02 (0.04)	-0.02 (0.04)	0.00 (0.04)	-0.05 (0.04)	-0.04 (0.04)	-0.68 [0.993] (0.99 [0.010])

Table continues on the next page. See notes at the end of the table.

Table 1A – 1, continued

Model	Covariates	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
14	13 + MRF(16)	-0.03	-0.08*	-0.02	-0.01	0.00	-0.04	-0.03	-0.53 [0.995]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(1.01 [0.010])
15	13 + MRF(16), CLB(16)	-0.03	-0.07*	-0.02	-0.01	0.02	-0.03	-0.03	0.10 [1.001]
		(0.04)	(0.02)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(1.04 [0.010])
	Approximate N	22,000	75,000	27,000	24,000	23,000	22,000	22,000	22,000

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 1-1 for variable definitions and categories.

3 The Kessner Index is coded as: Inadequate (reference category), Intermediate/Adequate, Unknown.

4 Race is coded as: White (reference category), Black, Hispanic, Other.

5 Rather than catchall "any" dummies for MRF and CLB, which are shown in Table 1-1, regressions use 16 separate dummies, each, for MRF and CLB.

6 For the logit regressions, we report both coefficients and odds ratios. The latter (and their SEs) are in brackets.

Table 1A - 2. Regression-adjusted Trends in Preterm Birth Among Nonblacks in Washington, DC, 1990-2001

			Robustness checks						
			Sample				Data		Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
1	FEMALE	-0.17*	-0.14*	-0.11*	-0.16*	-0.10			-1.82* [0.982*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
2	FEMALE, RACE	-0.15*	-0.16*	-0.09	-0.14*	-0.09			-1.66* [0.984*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
3	FEMALE, MARRIED	-0.14*	-0.15*	-0.08	-0.13*	-0.08			-1.55* [0.985*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
4	FEMALE, AGE	-0.13*	-0.12*	-0.08	-0.12*	-0.08			-1.42* [0.986*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
5	FEMALE, RACE, MARRIED	-0.13*	-0.16*	-0.07	-0.12*	-0.07			-1.48* [0.985*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])

Table continues on the next page. See notes at the end of the table.

Table 1A - 2, continued

			Robustness checks						
			Sample				Data		Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
6	FEMALE, RACE, MARRIED, AGE	-0.13*	-0.15*	-0.07	-0.12*	-0.08			-1.47* [0.985*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
7	FEMALE, RACE, MARRIED, AGE, EDUC	-0.12*	-0.13*	-0.06	-0.11*	-0.07			-1.32* [0.987*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
8	FEMALE, RACE, MARRIED, AGE, EDUC, PARITY, MPLB	-0.11*	-0.13*	-0.06	-0.11*	-0.06			-1.23* [0.988*]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)			(0.62 [0.006])
9	6 + TOBACCO, TOBXCIGNUM	-0.09	-0.13*	-0.05	-0.09	-0.04	-0.09	-0.09	-1.02 [0.990]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)	(0.06)	(0.06)	(0.63 [0.006])

Table continues on the next page. See notes at the end of the table.

Table 1A - 2, continued

			Robustness checks						
			Sample			Data			Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	-0.10 (0.06)	-0.14* (0.03)	-0.05 (0.05)	-0.09 (0.05)	-0.05 (0.06)	-0.10 (0.06)	-0.10 (0.06)	-1.09 [0.989] (0.63 [0.006])
11	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	-0.06 (0.06)	-0.13* (0.03)	-0.02 (0.05)	-0.06 (0.05)	-0.02 (0.06)	-0.09 (0.06)	-0.06 (0.06)	-0.77 [0.992] (0.67 [0.007])
12	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	-0.06 (0.06)	-0.13* (0.03)	-0.02 (0.05)	-0.06 (0.05)	-0.01 (0.06)	-0.09 (0.06)	-0.06 (0.06)	-0.72 [0.993] (0.67 [0.007])
13	12 + MRF(16)	-0.04 (0.06)	-0.11* (0.03)	-0.01 (0.05)	-0.04 (0.05)	0.01 (0.06)	-0.06 (0.06)	-0.04 (0.06)	-0.46 [0.995] (0.68 [0.007])

Table continues on the next page. See notes at the end of the table.

Table 1A - 2, continued.

			Robustness checks						
			Sample			Data			Estimation
Model	**Covariates**	**Main results**	*Inc. births to non-DC residents*	*Incl. non-DC births to DC residents*	*Incl. ages 40+*	*Incl. multiple gestations*	*PNC: Kessner Index*	*Tobacco: Usage dummies only*	*Logit models*
14	12 + MRF(16), CLB(16)	-0.03	-0.10*	0.00	-0.02	0.03	-0.05	-0.03	-0.07 [0.999]
		(0.06)	(0.03)	(0.05)	(0.05)	(0.06)	(0.06)	(0.06)	(0.70 [0.007])
	Approximate N	22,000	74,000	27,000	24,000	23,000	22,000	22,000	22,000

* p<0.05

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 1-1 for variable definitions and categories.

3 The Kessner Index is coded as: Inadequate (reference category), Intermediate/Adequate, Unknown.

4 Race is coded as: White (reference category), Black, Hispanic, Other.

5 Rather than catchall "any" dummies for MRF and CLB, which are shown in Table 1-1, regressions use 16 separate dummies, each, for MRF and CLB.

6 For the logit regressions, we report both coefficients and odds ratios. The latter (and their SEs) are in brackets.

Table 1A - 3. Yearly Regression Estimates of the Effects of Prenatal Smoking on Adverse Birth Outcomes, Births to Women in Washington, DC, 1990-2001

	Outcome			
Year	*Blacks: LBW*	*Blacks: Preterm*	*Nonblacks: LBW*	*Nonblacks: Preterm*
1990	14.06*	11.73*	10.66*	10.24*
	(1.21)	(1.28)	(3.36)	(3.70)
1991	15.77*	11.09*	15.37*	10.48
	(1.33)	(1.37)	(5.78)	(5.91)
1992	18.03*	12.49*	3.71	2.12
	(1.40)	(1.43)	(3.72)	(4.05)
1993	14.14*	12.50*	4.89	-1.10
	(1.55)	(1.64)	(4.77)	(4.80)
1994	14.21*	6.88*	3.07	6.19
	(1.65)	(1.66)	(4.52)	(6.11)
1995	13.67*	9.29*	1.97	-5.26
	(1.85)	(1.89)	(4.59)	(4.62)
1996	9.56*	8.28*	11.27	8.32
	(2.04)	(2.14)	(8.81)	(9.28)
1997	15.64*	10.40*	1.18	1.82
	(2.44)	(2.47)	(5.73)	(7.85)
1998	8.69*	5.62*	-5.07*	3.93
	(2.44)	(2.55)	(0.54)	(10.04)
1999	12.17*	7.78*	14.92	4.00
	(2.99)	(3.02)	(10.35)	(8.81)
2000	13.09*	9.77*	16.86	12.76
	(3.53)	(3.63)	(13.87)	(13.88)
2001	14.14*	8.42*	23.75*	27.47*
	(3.03)	(3.05)	(12.09)	(12.83)
N	8,048	8,044	2,224	2,223

See notes on the next page.

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Models also include an intercept term.

3 Prenatal smoking is measured by a single dummy for usage.

Table 1A - 4. Yearly Regression Estimates of the Effects of Prenatal Alcohol Use on Adverse Birth Outcomes, Births to Women in Washington, DC, 1990-2001

	Outcome			
Year	*Blacks: LBW*	*Blacks: Preterm*	*Nonblacks: LBW*	*Nonblacks: Preterm*
1990	19.11*	12.75*	-2.45	-3.51
	(2.16)	(2.18)	(1.84)	(2.44)
1991	21.78*	16.14*	-0.47	-1.62
	(2.13)	(2.15)	(2.06)	(2.72)
1992	23.14*	15.25*	-4.13*	-4.10
	(2.16)	(2.15)	(1.21)	(2.25)
1993	18.35*	17.70*	2.52	-0.22
	(2.35)	(2.44)	(2.81)	(3.33)
1994	20.18*	14.06*	-0.65	-5.90*
	(2.75)	(2.77)	(2.58)	(2.63)
1995	23.43*	17.67*	-3.20	-7.17*
	(3.28)	(3.28)	(1.71)	(2.88)
1996	17.08*	13.43*	4.56	-2.31
	(3.94)	(3.99)	(5.51)	(4.92)
1997	22.69*	16.98*	-4.77*	3.72
	(4.12)	(4.13)	(0.53)	(7.36)
1998	15.74*	17.42*	-5.08*	-4.86
	(4.82)	(5.11)	(0.54)	(6.49)
1999	18.09*	4.16	-0.66	-4.87
	(5.76)	(5.19)	(4.47)	(4.50)
2000	27.93*	13.73*	-5.47*	-9.57*
	(5.95)	(5.63)	(0.52)	(0.68)
2001	18.19*	15.81*	-0.44	-3.92
	(5.75)	(5.90)	(4.47)	(4.48)
N	8,048	8,044	2,224	2,223

See notes on the next page.

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Models also include an intercept term.

3 Prenatal alcohol use is measured by a single dummy for usage.

Table 1A - 5. Regression Estimates of Sociodemographic Effects on Adverse Birth Outcomes, Births to Women in Washington, DC, 1990-2001

	Outcome			
Covariate	*Blacks: LBW*	*Blacks: Preterm*	*Nonblacks: LBW*	*Nonblacks: Preterm*
Year	-0.42*	-0.52*	-0.06	-0.11*
	(0.04)	(0.04)	(0.04)	(0.06)
Female	2.71*	-0.54	1.35*	-0.73
	(0.27)	(0.30)	(0.30)	(0.40)
Race/ethnicity = Hispanic			0.26	1.57*
			(0.60)	(0.78)
Race/ethnicity = Other			2.23*	4.78*
			(0.76)	(0.98)
Married	-6.09*	-6.25*	-2.34*	-3.15*
	(0.37)	(0.41)	(0.45)	(0.61)
Age = 20-24	2.77*	0.55	0.01	-1.76
	(0.39)	(0.46)	(0.79)	(1.10)
Age = 25-29	7.87*	4.59*	0.38	-2.36*
	(0.46)	(0.53)	(0.80)	(1.10)
Age = 30-34	12.44*	7.34*	1.03	-1.50
	(0.53)	(0.59)	(0.83)	(0.00)
Age = 34-39	15.56*	11.20*	1.74*	-0.63
	(0.67)	(0.74)	(0.88)	(1.19)
Education = High School	-2.24*	-2.53*	0.99	0.34
	(0.35)	(0.39)	(0.58)	(0.78)
Education = College+	-5.86*	-6.58*	-2.18*	-3.69*
	(0.44)	(0.49)	(0.64)	(0.84)
Parity = 1+	-0.69*	3.20*	-1.90*	0.40
	(0.31)	(0.35)	(0.32)	(0.43)

Table continues on the next page. See notes at the end of the table.

Table 1A - 5, continued

	Outcome			
Covariate	*Blacks: LBW*	*Blacks: Preterm*	*Nonblacks: LBW*	*Nonblacks: Preterm*
Maternal place of birth = DE/MD/VA/WV	-0.04	-0.10	-1.82	-1.84
	(0.58)	(0.65)	(1.00)	(1.22)
Maternal place of birth = Other State	-1.45*	-1.79*	-2.37*	-1.61*
	(0.42)	(0.47)	(0.66)	(0.81)
Maternal place of birth = Foreign	-3.89*	-2.35*	-2.93*	-2.04*
	(0.47)	(0.55)	(0.71)	(0.88)
N	71,250	71,149	22,455	22,370

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Reference categories are: Race/ethnicity = White, Age = 15-19, Education = < High School, Parity = 0, Maternal place of birth = DC.

3 Models also include dummies for unknown education and unknown parity as well as an intercept term.

Table 1A - 6. Regression Estimates of the Effects of Smoking, Alcohol, Adequacy of Prenatal Care, and Pregnancy History on Adverse Birth Outcomes, Births to Women in Washington, DC, 1990-2001

	Outcome			
Covariate	*Blacks: LBW*	*Blacks: Preterm*	*Nonblacks: LBW*	*Nonblacks: Preterm*
Prenatal smoking	5.32*	4.45*	7.55*	2.13
	(0.88)	(0.90)	(2.09)	(2.38)
Cigarettes per day, if smoked	0.29*	0.04	-0.13	0.40
	(0.08)	(0.08)	(0.17)	(0.23)
Prenatal alcohol use	10.49*	7.69*	-0.84	-1.37
	(0.94)	(0.95)	(0.72)	(1.01)
PNC = Intermediate	-5.90*	-11.04*	-0.83	-8.78*
	(0.36)	(0.38)	(0.44)	(0.58)
PNC = Adequate	-6.06*	-6.76*	-1.37*	-5.36*
	(0.34)	(0.39)	(0.41)	(0.59)
PNC = Adequate Plus	4.42*	20.37*	-0.95	14.34*
	(0.52)	(0.61)	(0.60)	(0.94)
Previous preterm or IUGR birth	23.00*	19.60*	3.67	18.98*
	(1.44)	(1.46)	(3.21)	(4.79)
N	71,249	71,148	22,357	22,370

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Reference categories are: PNC = Inadequate.

3 Models also include controls for year, infant sex, marital status, maternal age, maternal education, parity, and maternal place of birth as well as an intercept term.

Table 1A - 7. Description of Selected Medical Risk Factors and Complications of Labor

Medical Risk Factors	
Eclampsia	Convulsions or coma late in pregnancy[1]
Hemoglobinopathy	Group of rare, inherited disorders involving abnormal structure of the hemoglobin molecule[1]
Hydramnios	Excessive accumulation of the amniotic fluid[1]
Oligohydramnios	Deficiency of amniotic fluid[1]
Incompetent Cervix	Weakened cervix that predisposes a women to mid-term miscarriage or premature delivery[2]
RH Sensitization	Disease in which the Rh-negative system of a mother's immune system produces antibodies that destroy the fetus's Rh-positive red blood cells[1]
Complications of Labor	
Abruptio Placenta	Premature detachment of the placenta from the wall of the uterus[1]
Celphalopelvic Disproportion	Small maternal pelvis relative to size of the fetal head[1]
Cord Prolapse	Baby's umbilical cord falls into the birth canal ahead of the baby's head or other parts of the baby's body[3]
Fetal Distress	Slow heart rate from lack of oxygen[4]
Meconium	Dark greenish mass of desquamated cells, mucus, and bile that accumulates in the bowel of the fetus and is typically discharged shortly after birth[1]
Placenta Previa	Abnormal implantation of the placenta at or near the internal opening of the uterine cervix so that it tends to precede the child at birth[1]
Premature Rupture of the Membrane	Membranes that hold amniotic fluid break before labor in full-term pregnancies[2]

Sources:
1 Medline Plus/Merriam-Webster
2 Pennhealth.com
3 Medline Plus Medical Encyclopedia
4 MedicineNet.com

Appendix 1B: Age, Cohort, and Period Effects

This study finds that among blacks, teen age is relatively advantageous for infant health and that risk rises monotonically with age after the teen years, for low birth weight, and after age 24, for preterm birth. This is consistent with several studies of weathering conducted by Geronimus and others (Geronimus 1986). However, in a study of birth outcomes in the 1980s and 1990s, Almond and Chay conclude that weathering effects may be an artifact of better health among younger cohorts (Almond and Chay 2003). Evidence of weathering disappears in their analyses when they control for birth cohort of mother. One difficulty with this analysis, however, is their inability to estimate convincingly age, period, and cohort effects simultaneously.[27]

In this appendix we examine the sensitivity of our low birth weight age effects to cohort controls. We estimate four models, for blacks and whites separately, in which the dependent variable is low birth weight, and we vary the control variables (Table 1B-1). In the first model, we regress low birth weight on maternal age and child's birth year. In the second model, we regress low birth weight on maternal age and mother's birth cohort. Models 3 and 4 repeat Models 1 and 2, but also include controls for maternal education. We eliminate younger cohorts - women younger than age 25 in 2001 – because among these women, low education may simply reflect their age, and women younger than age 22 may not have completed their education. We also limit the sample to first births, thereby comparing the effects of the timing of first birth.

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[27] Almond and Chay also use NCHS Natality data and control for education, but their study differs from ours in a number of ways that could explain the different results. They include births in all 50 states and the District of Columbia, as well as births in the 1980s. Their sample is limited to births to mothers born during the 1960s. Mothers in our sample were born over a longer time horizon – from the mid-1950s to the early 1980s. Finally, their estimation approach differs from ours in that they control simultaneously for mother's age, birth cohort, and survey year.

When we control for child's birth year, black teens are at lower risk of low birth weight relative to women ages 25 and older, but not relative to women ages 20-24 (Panel A). Controlling for mother's cohort reduces the magnitude of age effects and eliminates weathering among women aged 25-29. When we also control for education, however, we find strong evidence of weathering at all age levels above the teen years. This is true whether we include controls for birth year or cohort, though age effects are larger in the model that controls for child's birth year. Not only are black teens less likely to bear unhealthy infants than those ages 20-24, but also the health disadvantage of older age rises with age.

It is notable that these models find that teens have reduced risk for low birth weight since they do not include controls for socioeconomic status. Unmeasured heterogeneity in socioeconomic background factors is known to be substantial and to bias estimates of effects of teen childbearing on birth outcomes towards a finding of disadvantage (Geronimus and Korenman 1993; Rosenzweig and Wolpin 1995). However, we make some adjustment for socioeconomic heterogeneity by limiting the sample to first births. When we eliminate this restriction (Table 1B-2), age effects in the models that control for year of birth are somewhat larger. Among all parity births, teen age is disadvantageous relative to ages 20-24 in the model that controls for only for maternal age and cohort, but this disadvantage is not statistically significant and disappears when we also control for education (Table 1B-2).

One interpretation of these findings is that if delayed child bearing increases education, then it may improve infant health. On the other hand, if delaying childbearing merely delays age at birth but does not result in increased education, it may harm infant

and maternal health. Which cohort model is correct, then – the model that includes or excludes education controls? If we wish to estimate the effects of age at birth, and if delayed childbearing is responsible for increasing education across cohorts, then it is inappropriate to control for education. However, if education is increasing across cohorts for reasons other than fertility delay (which seems likely), then we should control for education. Bias from unmeasured heterogeneity aside, the true age effect within cohort probably lies between the results with and those without education controls. This means that the truth lies between a substantially protective effect of teen age (15-19), and no effect (relative to 20-24); whereas nearly all estimates suggest a substantially adverse effect of delaying beyond age 24.

Table 1B - 1. Sensitivity of Age Effects to Cohort Controls in Regressions on Low Birth Weight, First Births to Women Ages 25 and Older in 2001 in Washington, DC, 1990-2001

	Panel A: Blacks			
Covariates	1	2	3	4
Age = 20-24	0.78	0.02	2.97*	2.19*
	(0.67)	(0.70)	(0.76)	(0.78)
Age = 25-29	2.75*	1.03	5.89*	4.04*
	(0.80)	(0.94)	(0.90)	(1.04)
Age = 30-34	7.21*	4.41*	10.65*	7.55*
	(1.00)	(1.37)	(1.11)	(1.45)
Age = 34-39	9.39*	5.59*	13.09*	8.84*
	(1.43)	(1.97)	(1.50)	(2.03)
N	17,342			
	Panel B: Whites			
Covariates	1	2	3	4
Age = 20-24	0.16	0.06	2.15	2.06
	(3.10)	(3.10)	(3.42)	(3.42)
Age = 25-29	-2.41	-2.66	1.29	1.06
	(2.83)	(2.89)	(3.29)	(3.34)
Age = 30-34	-2.53	-2.92	1.45	1.08
	(2.80)	(2.93)	(3.28)	(3.38)
Age = 34-39	-1.90	-2.47	2.08	1.52
	(2.82)	(3.07)	(3.30)	(3.50)
N	6,921			
Also includes controls for:				
Child's birth year	X		X	X
Mother's birth cohort		X		
Maternal education			X	X

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Reference category is Age = 15-19.

3 Models also include an intercept term.

Table 1B - 2. Sensitivity of Age Effects to Cohort Controls in Regressions on Low Birth Weight, All Parity Births to Women Ages 25 and Older in 2001 in Washington, DC, 1990-2001

	Panel A: Blacks			
Covariates	1	2	3	4
Age = 20-24	1.50*	-0.66	3.18*	1.01*
	(0.48)	(0.48)	(0.50)	(0.50)
Age = 25-29	5.26*	0.62	7.54*	2.81*
	(0.51)	(0.56)	(0.53)	(0.59)
Age = 30-34	8.75*	1.55*	11.46*	4.06*
	(0.56)	(0.73)	(0.59)	(0.76)
Age = 34-39	11.37*	1.71	14.38*	4.42*
	(0.69)	(0.98)	(0.72)	(1.01)
N	59,450			
	Panel B: Whites			
Covariates	1	2	3	4
Age = 20-24	-2.11	-2.52	-0.56	-0.92
	(2.98)	(2.99)	(3.05)	(3.05)
Age = 25-29	-5.33	-6.22*	-1.91	-2.72
	(2.81)	(2.84)	(2.94)	(2.96)
Age = 30-34	-6.29*	-7.58*	-2.26	-3.46
	(2.78)	(2.86)	(2.92)	(2.98)
Age = 34-39	-6.05*	-7.75*	-1.92	-3.52
	(2.79)	(2.93)	(2.93)	(3.04)
N	11,901			
Also includes controls for:				
Child's birth year	X		X	X
Mother's birth cohort		X		
Maternal education			X	X

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Reference category is Age = 15-19.

3 Models also include an intercept term.

Appendix 1C: Further Exploration of Unmeasured Crack Use

In this appendix, we consider whether our estimated effects of the decline in smoking on infant health reflect a reduction in other, unmeasured risky behaviors. A recent study attributed the decline in low birth weight in New York City during the 1990s to the waning of the crack epidemic in that city (Joyce, Gibson et al. 2005). If tobacco (and alcohol) and cocaine use are correlated, then the infant health improvements that we attribute to reductions in prenatal smoking may at least partially reflect unmeasured reductions in illegal drug use. The trends shown in Figure 1C-1 suggest this concern should be considered carefully. Like crack use, rates of low birth weight and preterm birth among blacks rose in the 1980s and fell in the 1990s. Data limitations preclude us from establishing, definitively, whether improvements in infant health in the 1990s attributed to the decline in prenatal smoking are in fact a regression to the mean resulting from a contemporaneous peak and decline of crack use. However, exploratory analyses do not allow us to reject this possibility.

Our first approach is to employ a District- and year-specific index of crack use constructed from cocaine arrest, death, and "bust" rates from Federal Bureau of Investigation (FBI), NCHS, and Drug Enforcement Agency (DEA) databases, respectively, as well as crack-related emergency room visits from the Drug Abuse Warning Network (DAWN) and the number of newspaper citations referencing cocaine (Fryer, Heaton et al. 2005). As shown in Figure 1C-2, the index follows an inverse-U pattern, peaking in 1989.

We add this index to regression models 6 and 7 and report these results in Table 1C-1. Standard errors in models that include the crack index are clustered on birth year. For ease of comparison, in Columns 1 and 2, we reproduce our results from the original

models that control only for sociodemographic characteristics (Column 1) and that control for both sociodemographic characteristics and prenatal smoking (Column 2).[28] The model in Column 3 adjusts for sociodemographic characteristics and crack use. Comparing the trend coefficient between Columns 1 and 3 shows that the decline in crack use had very little impact on low birth weight and preterm birth after adjusting for infant sex, marital status, maternal age and education, parity, and maternal place of birth. The model in Column 4 adjusts for sociodemographic characteristics, smoking, and crack use. Comparing Columns 2 and 4 reveals that the trend in crack use contributed little, if anything, to the reduction in the preterm birth rate beyond that captured by changes in sociodemographic characteristics and prenatal smoking. And, if anything, the decline in crack use is associated with a *increase* in the rate of low birth weight. Moreover, adding a control for crack use does not affect the coefficient of the prenatal smoking variables, indicating that crack use does not impact low birth weight and preterm birth indirectly through smoking.

This latter finding is consistent with other studies, which have found that prenatal cocaine use does not affect gestational age or birth weight after controlling for marijuana, alcohol, and tobacco use (Chasnoff, Burns et al. 1985; Eyler, Behnke et al. 1998). However, this does not explain why the decline in the crack index does not affect the trends in infant health when we do not control for prenatal smoking.

One reason may be that the index is a poor measure of crack use, especially among pregnant women. Horowitz has criticized the use of DEA drug bust data for

[28] Since the crack index is unavailable for the year 2001, these models are estimated on a data sample covering the years 1990-2000. Therefore, the coefficients in these columns may not match exactly those reported in Tables 1-2 and 1-3.

economic analyses on the grounds that they are not a random sample since the data acquisition process reflects local law enforcement priorities (Horowitz 2001). Arrest data suffer the same bias. Also, non-age and non-gender specific drug use statistics may not accurately reflect use among pregnant women, which has been shown to be more common among women in their late 20s and early 30s (Hollinshead, Griffin et al. 1990; Nalty 1991; Buchi, Varner et al. 1993; Vega, Kolody et al. 1997).

Therefore, we repeat this analysis, separately, for women less than 25 and age 25 or older. If the waning of the crack epidemic is responsible for declines in adverse birth outcomes in the 1990s and this is being measured by tobacco use, then we would expect the declines in low birth weight and preterm birth and smoking's estimated contribution to those declines to be greater among the older women. Tables 1C-2 and 1C-3 show just this. When we adjust only for sociodemographic characteristics (Columns 1), the estimated yearly decline in low birth weight among blacks younger than 25 is 0.34 percentage points compared with 0.53 percentage points among those ages 25 and older. Though smaller, this differential is also evident with respect to preterm birth. Additionally, the absolute value of the trend coefficients decline much more, in percentages, in the low birth weight and preterm birth regressions for older than younger women when we add controls for tobacco use (Columns 1 vs. 2). On the other hand, the coefficients on the crack index are the wrong sign in the regressions for women at least age 25 and statistically insignificant in all regressions.

Our second approach is to extend the sample backwards through 1980. This allows us to capture the periods prior to as well as after the crack epidemic. One

limitation with this approach, however, is that birth certificate data do not include information on prenatal tobacco use prior to 1989.

Since crack use among pregnant women is more common among older and less educated women, we first plot low birth weight and preterm birth trends for these subpopulations. We would expect to see adverse birth outcomes increase more steeply in the 1980s and decline more sharply in the 1990s among these groups than among younger, more educated women, if the crack epidemic has any import. Figure 1C-3 shows that rates of low birth weight and preterm birth did rise more steeply in the 1980s among older than younger women, but they subsequently declined from their peaks at similar rates across age groups.[29] Low birth weight and preterm birth rates among women with less than a high school education fell more quickly in the 1990s but rose more slowly in the 1980s than those with higher schooling (Figure 1C-4).

We next estimate a series of regression models on a data sample spanning the years 1980-2000. These models are identical to Models 1-7 of our main analysis, except that we include both linear and quadratic time trend terms and substitute the crack index for tobacco use in Model 7.[30] We report the coefficients on the trend terms and the average yearly change in each outcome implied by those coefficients in Tables 1C-4 and 1C-5. As before, standard errors in models that include the crack index are clustered on birth year. In order to facilitate interpretation of the results from these regressions, we plot the low birth weight and preterm birth trends implied by our estimated linear and quadratic trends for models 1, 6, and 7 in Figures 1C-5 and 1C-6. The lines labeled

[29] The rapid rise in births to women over age 34 during the 1990s, however, may have mitigated health improvements among women aged at least 25.

[30] The crack index is unavailable in 2001.

"Model 1" correspond to the trends adjusted only for infant sex. While adjusting for sociodemographic characteristics dampens the rate of increase in the low birth weight rate that occurred during the 1980s (Table 1C-5, Model 6), also adjusting for crack use erases this increase altogether (Model 7). This suggests that the rise in crack use was largely responsible for the increase in low birth weight during the 1980s. However, the rate of low birth weight declines in the 1990s even when we adjust for crack use, with the decline beginning as early as 1986. This suggests that the waning of the crack epidemic is not fully responsible for the health improvements of the 1990s not already attributed to changes in sociodemographic characteristics. In contrast, adjusting for sociodemographic characteristics and crack use has a much more modest impact on the trend in preterm birth (Table 1C-6). Thus, the trend in crack use appears to have contributed to the trend in low birth weight, if not preterm birth.

The sum of these findings is inconclusive. In the 1990-2000 regression analyses, the trend in crack use does not appear to have contributed to improvements in black infant health even when we do not control for smoking, but our measure of crack use may inappropriate for analyses of pregnancy outcomes. On the other hand, health improvements in the 1990s were greater among women ages 25 and older, who are thought to be more likely to have used crack, and the estimated role of reduced smoking (which may be proxying for falling crack use) in these gains is also greater among these women. Additionally, the contribution of reduced smoking (and possibly crack use) to infant health improvements are greater with respect to low birth weight than preterm birth, which is consistent with the theory that crack use should affect the former more than the latter, since cocaine use has been shown to retard fetal growth (Eyler, Behnke et

al. 1998).[31] Smoking also affects fetal growth, though, and smoking is also more common among older women.

Results from the 1980-2000 analysis are similarly mixed. The regression analyses suggest that the trend in crack use contributed to changes in the rate of black low birth weight but not preterm birth. However, since we are unable to control for prenatal smoking in these models, it is impossible to know whether this effect is independent of the unmeasured effect of smoking. Some of the graphical analyses support a role for crack use in infant health trends, but some do not, and these graphs also do not adjust for smoking trends, which track closely with the crack index between 1989 and 2000 (Figure 1C-2). Moreover, we are unable to reconcile the fact that the crack index has explanatory power in regression analyses for the 1980-2000 period but not the 1990-2000 period. Therefore, while we cannot reject the crack hypothesis based on these results, neither can we definitively accept it.

[31] To the extent that crack use represents a generally unhealthy lifestyle, it may not be expected to affect preterm birth less than low birth weight. Under this assumption, our finding that tobacco and crack use affect low birth weight substantially more than preterm birth is not supportive of the crack hypothesis.

Table 1C - 1. Regression Estimates of the Effects of Crack Use on Adverse Birth Outcomes Among Births to Black Women in Washington, DC, 1990-2000

	Panel A: Outcome = LBW			
Covariate	1	2	3	4
Year	-0.44*	-0.26*	-0.48*	-0.32*
	(0.04)	(0.05)	(0.10)	(0.11)
Prenatal smoking		7.70*		7.70*
		(0.79)		(0.79)
Cigarettes per day, if smoked		0.38*		0.38*
		(0.06)		(0.06)
Crack Index			-0.24	-0.35
			(0.47)	(0.51)
N		67,060		
	Panel B: Outcome = Preterm			
Covariate	1	2	3	4
Year	-0.57*	-0.45*	-0.53*	-0.43*
	(0.05)	(0.05)	(0.09)	(0.08)
Prenatal smoking		6.13*		6.13*
		(1.41)		(1.41)
Cigarettes per day, if smoked		0.09		0.09
		(0.13)		(0.13)
Crack Index			0.19	0.13
			(0.42)	(0.40)
N		66,971		

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Models also include controls for infant sex, marital status, maternal age, maternal education, parity, and maternal place of birth as well as an intercept term.

3 Standard errors in Models 2 and 4 are clustered on year of birth.

Table 1C - 2. Regression Estimates of the Effects of Crack Use on Low Birth Weight Among Births to Black Women Age<25 or Age 25+ in Washington, DC, 1990-2000

	Panel A: Age<25			
Covariate	1	2	3	4
Year	-0.34*	-0.26*	-0.29*	-0.23*
	(0.06)	(0.05)	(0.11)	(0.10)
Prenatal smoking		6.04*		6.04*
		(1.72)		(1.72)
Cigarettes per day, if smoked		0.46*		0.46*
		(0.15)		(0.15)
Crack Index			0.29	0.18
			(0.44)	(0.42)
N		33,981		
	Panel B: Age=25+			
Covariate	1	2	3	4
Year	-0.53*	-0.25*	-0.67*	-0.40*
	(0.07)	(0.06)	(0.11)	(0.13)
Prenatal smoking		8.29*		8.28*
		(0.72)		(0.72)
Cigarettes per day, if smoked		0.33*		0.33*
		(0.07)		(0.07)
Crack Index			-0.81	-0.90
			(0.56)	(0.66)
N		33,079		

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Models also include controls for infant sex, marital status, maternal age, maternal education, parity, and maternal place of birth as well as an intercept term.

3 Standard errors in Models 2 and 4 are clustered on year of birth.

Table 1C - 3. Regression Estimates of the Effects of Crack Use on Preterm Birth Among Births to Blacks Age<25 or Age 25+ in Washington, DC, 1990-2000

	Panel A: Age<25			
Covariate	1	2	3	4
Year	-0.52*	-0.49*	-0.35	-0.32
	(0.07)	(0.09)	(0.17)	(0.16)
Prenatal smoking		4.44		4.43
		(2.04)		(2.04)
Cigarettes per day, if smoked		0.07		0.07
		(0.13)		(0.13)
Crack Index			1.02	0.97
			(0.79)	(0.78)
N		33,941		
	Panel B: Age=25+			
Covariate	1	2	3	4
Year	-0.60*	-0.41*	-0.71*	-0.53*
	(0.07)	(0.06)	(0.13)	(0.13)
Prenatal smoking		7.03*		7.03*
		(1.30)		(1.29)
Cigarettes per day, if smoked		0.07		0.07
		(0.14)		(0.14)
Crack Index			-0.64	-0.68
			(0.57)	(0.55)
N		33,030		

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Models also include controls for infant sex, marital status, maternal age, maternal education, parity, and maternal place of birth as well as an intercept term.

3 Standard errors in Models 2 and 4 are clustered on year of birth.

Table 1C - 4. Regression-adjusted Trends in Low Birth Weight Among Blacks in Washington, DC, 1980-2000

		Quadradic in time		
Model	**Covariates**	Linear term	Quadratic term	**Implied yearly change**
1	FEMALE	141.64*	-0.04*	0.039
		(13.48)	(0.00)	(0.38)
2	FEMALE, MARRIED	131.55*	-0.03*	-0.001
		(13.16)	(0.00)	(0.35)
3	FEMALE, AGE	136.70*	-0.03*	0.003
		(13.58)	(0.00)	(0.37)
4	FEMALE, MARRIED, AGE	118.03*	-0.03*	-0.075
		(13.16)	(0.00)	(0.32)
5	FEMALE, MARRIED, AGE, EDUC	126.61*	-0.03*	-0.082
		(13.46)	(0.00)	(0.34)
6	FEMALE, MARRIED, AGE, EDUC, PARITY, MPLB	123.26*	-0.03*	-0.081
		(13.43)	(0.00)	(0.33)
7	FEMALE, MARRIED, AGE, EDUC, PARITY, MPLB, CRKINDEX	33.83	-0.01	-0.104
		(33.48)	(0.01)	(0.09)
	N		122,720	

*
p<0.05

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 1-1 for variable definitions and categories.

3 Standard errors in Model 7 are clustered on year of birth.

Table 1C - 5. Regression-adjusted Trends in Preterm Birth Among Blacks in Washington, DC, 1980-2000

		Quadradic in time		
Model	**Covariates**	Linear term	Quadratic term	**Implied yearly change**
1	FEMALE	212.73*	-0.05*	0.132
		(15.53)	(0.00)	(0.57)
2	FEMALE, MARRIED	198.87*	-0.05*	0.080
		(15.42)	(0.00)	(0.53)
3	FEMALE, AGE	211.01*	-0.05*	0.109
		(15.24)	(0.00)	(0.56)
4	FEMALE, MARRIED, AGE	190.46*	-0.05*	0.026
		(15.09)	(0.00)	(0.51)
5	FEMALE, MARRIED, AGE, EDUC	189.98*	-0.05*	0.029
		(15.26)	(0.00)	(0.51)
6	FEMALE, MARRIED, AGE, EDUC, PARITY, MPLB	181.12*	-0.05*	-0.003
		(15.33)	(0.00)	(0.48)
7	FEMALE, MARRIED, AGE, EDUC, PARITY, MPLB, CRKINDEX	141.48*	-0.04*	-0.013
		(28.84)	(0.01)	(0.38)
	N		120,366	

*
p<0.05

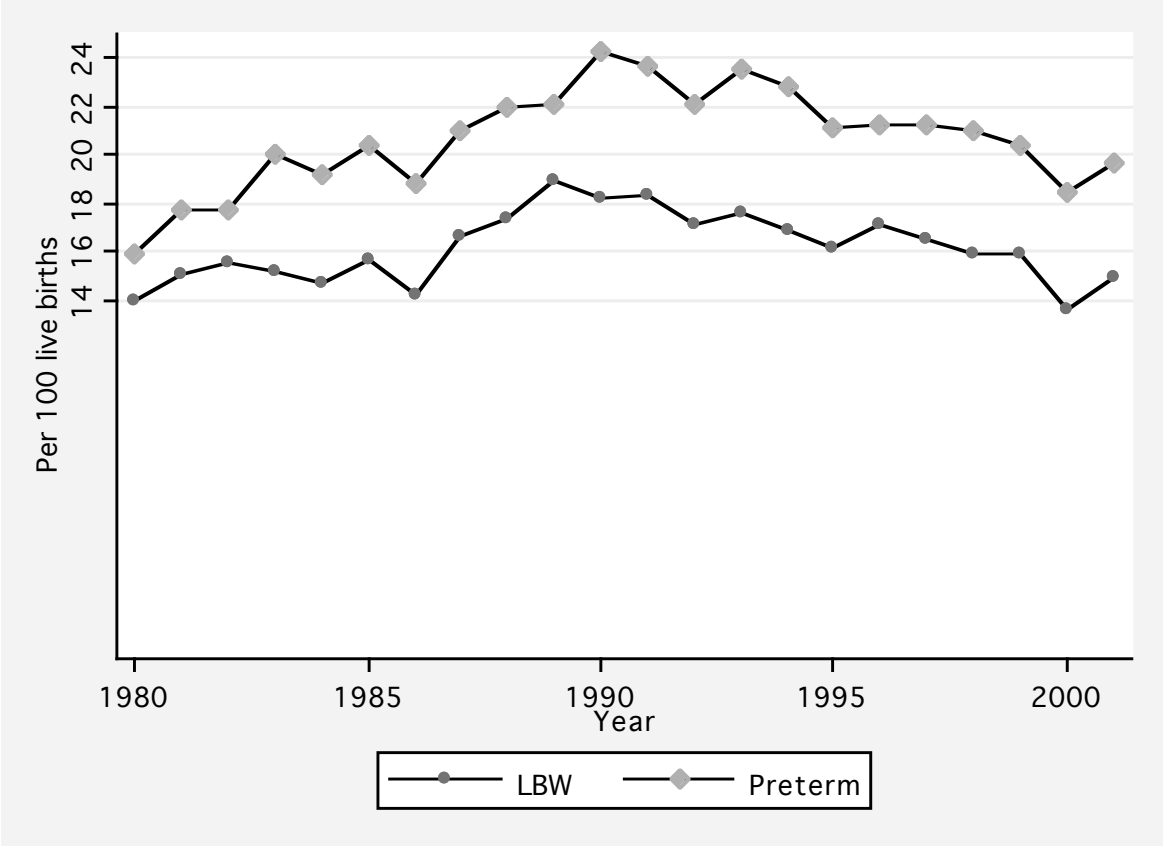
Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 1-1 for variable definitions and categories.

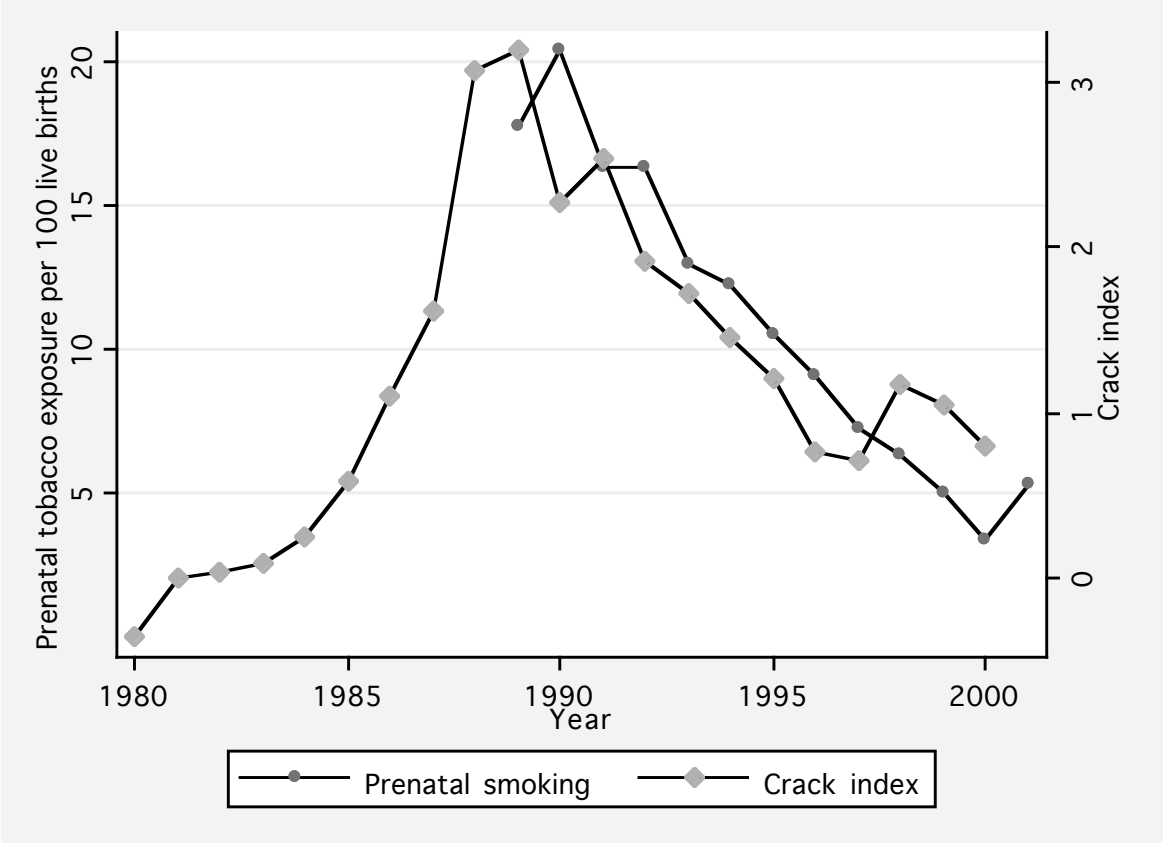
3 Standard errors in Model 7 are clustered on year of birth.

Figure 1C - 1. Birth Outcomes Among Blacks in Washington, DC, 1980-2001



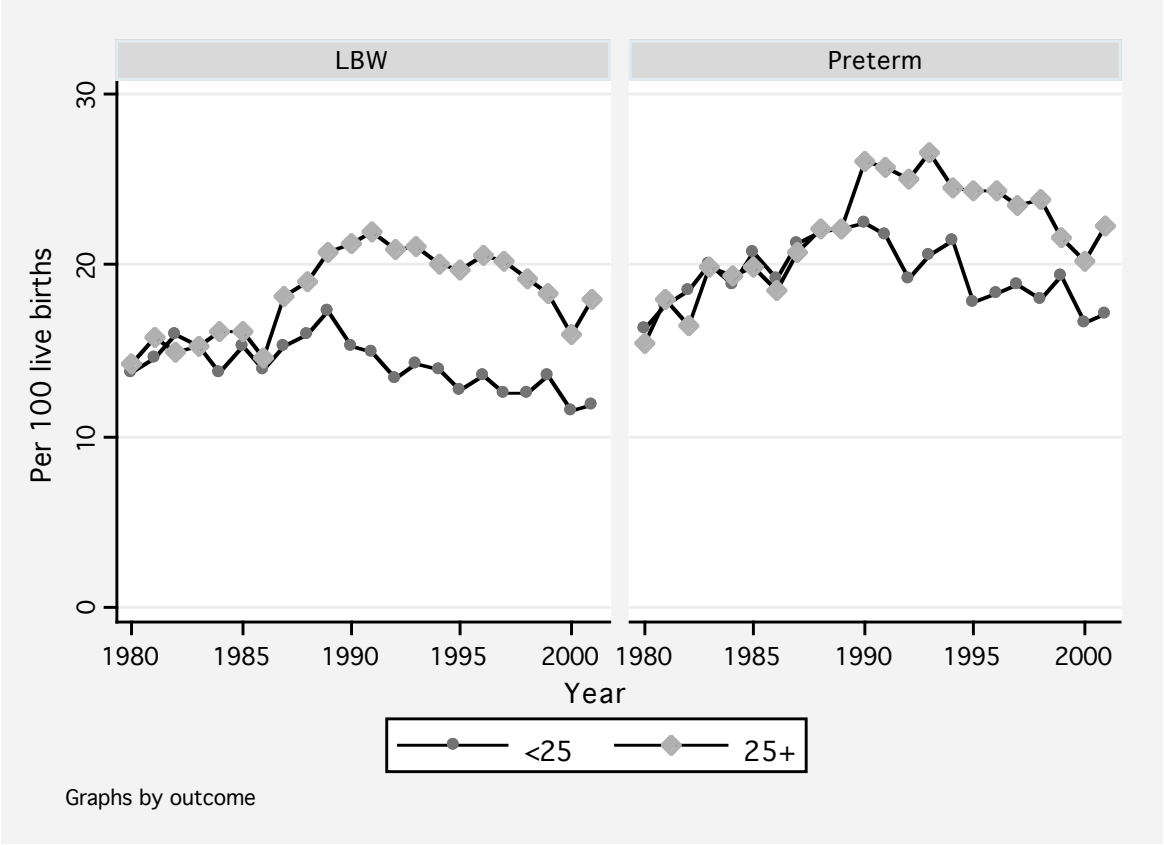
Source: Authors' tabulations of 1980-2001 NCHS Natality Files.
Note: Singleton births in Washington, DC to women aged 15-39.

Figure 1C - 2. Tobacco and Crack Use Among Blacks in Washington, DC, 1980-2001



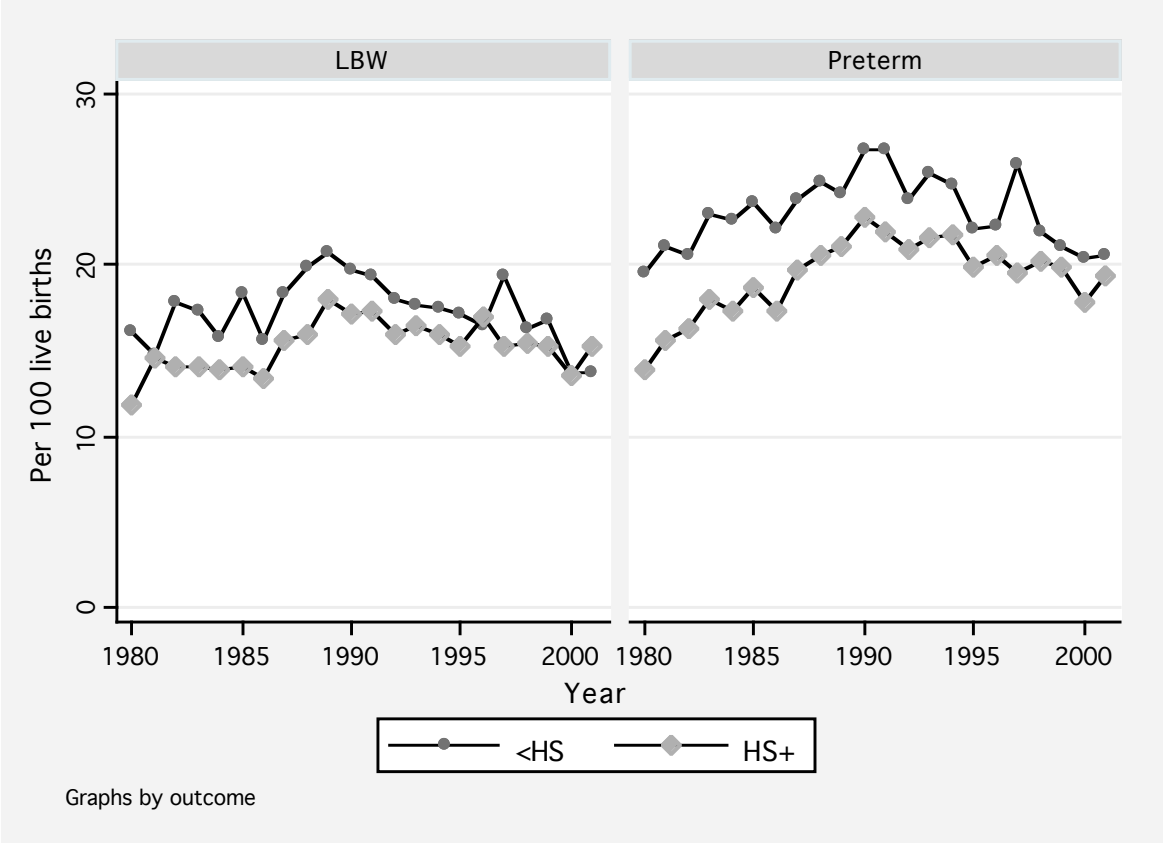
Sources: Tobacco use - Authors' tabulations of 1980-2001 NCHS Natality Files; Crack use – Fryer, et al. (2005).
Note: Singleton births in Washington, DC to women aged 15-39.

Figure 1C - 3. Birth Outcomes Among Blacks in Washington, DC by Maternal Age, 1980-2001



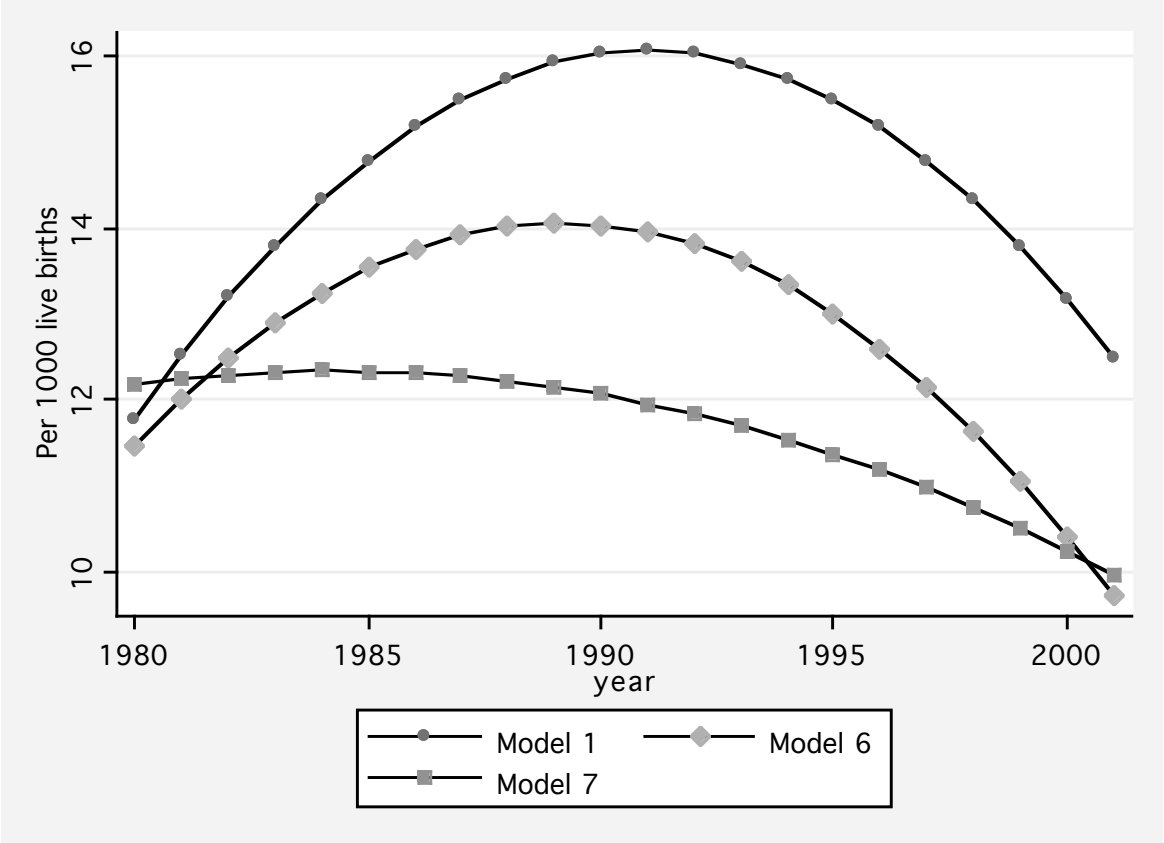
Source: Authors' tabulations of 1980-2001 NCHS Natality Files.
Note: Singleton births in Washington, DC to women aged 15-39.

Figure 1C - 4. Birth Outcomes Among Blacks in Washington, DC by Maternal Education, 1980-2001



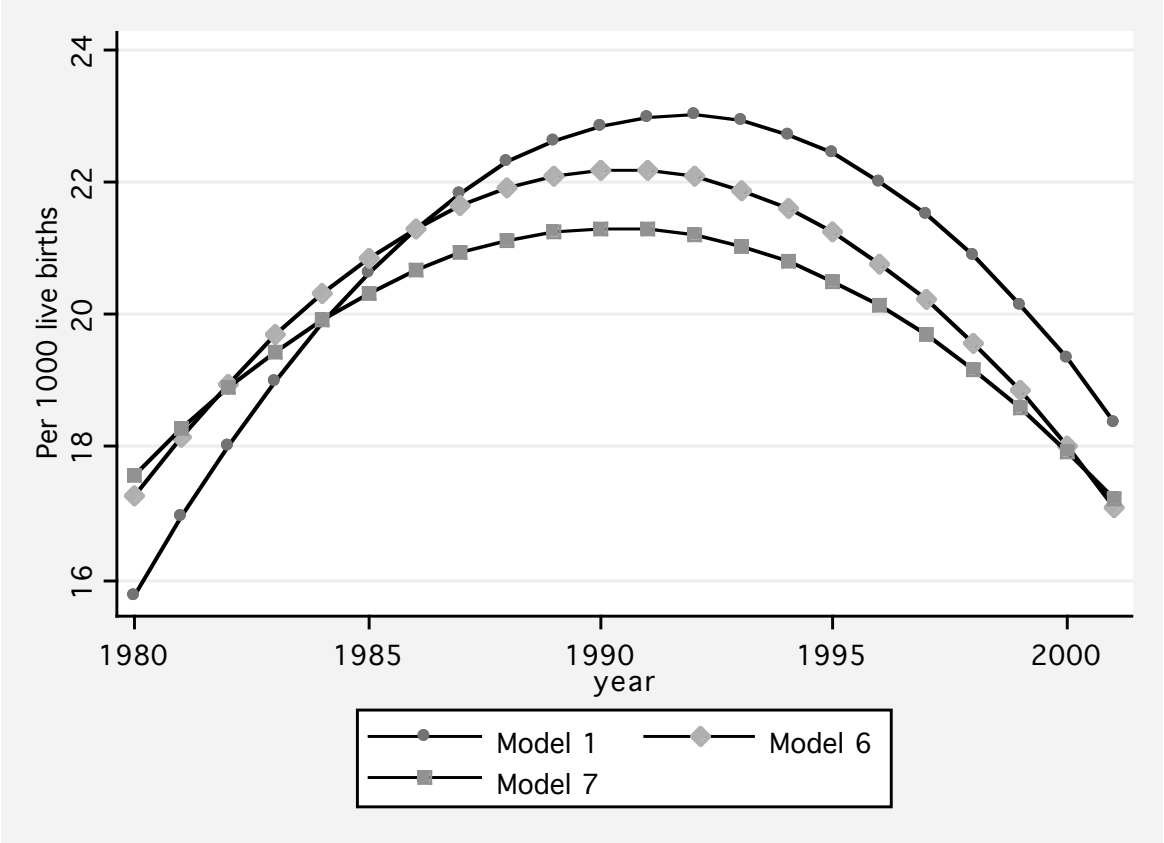
Source: Authors' tabulations of 1980-2001 NCHS Natality Files.
Note: Singleton births in Washington, DC to women aged 15-39.

Figure 1C - 5. Regression-implied Trend in Low Birth Weight Among Blacks in Washington, DC, Adjusted for Varying Characteristics, 1980-2001



Source: Computed using regression results shown in Table 1C-4.
Note: Singleton births in Washington, DC to women aged 15-39.

Figure 1C - 6. Regression-implied Trend in Preterm Birth Among Blacks in Washington, DC, Adjusted for Varying Characteristics, 1980-2001



Source: Computed using regression results shown in Table 1C-5.
Note: Singleton births in Washington, DC to women aged 15-39.

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Chapter 2 Changes in the Spatial Distribution of Black Infant Health in the 1990s

Introduction

In sharp contrast to social and health gains on many other fronts, rates of low birth weight and preterm birth suggest unchanging or even deteriorating infant health during the 1990s (Martin, Hamilton et al. 2002). The 1990s marked a period of sustained economic expansion, increases in education, advancements in medical care and technology, and substantial expansions in eligibility for public health insurance (National Bureau of Economic Research; U.S. Department of Education 2003). The US also experienced declines in crime, teen childbearing, the number of high-poverty neighborhoods, and concentrated poverty (Martin, Hamilton et al. 2002; Travis and Waul 2002; Jargowsky 2003). And yet, aggregate measures suggest these gains did not extend to infant health.

In Chapter 1 we revealed that the experience of African-Americans in Washington, DC represents an exception to flat aggregate rates of low birth weight and preterm birth during the 1990s. While Washington, DC represents an extreme case, however, it is nonetheless but one example of a health improving trend specific to center city black infants (Figure 2-1).[32] Among suburban infants, black adverse birth outcomes either changed little (low birth weight) or declined more slowly than in center cities (preterm birth). Infants born to women living in center cities – especially black infants – are at elevated risk of adverse health outcomes relative to those of nearby suburbs. And

[32] Though Figure 1 includes only births to women living in 37 metropolitan areas with large populations, the trends are similar when drawn for all US metropolitan areas and for the entire US. The 37 metropolitan areas shown represent 29 percent of the entire US and 47 percent of the non-Hispanic black population.

so, it is notable that the 1990s were characterized by decreasing spatial inequality in black infant health (Figure 2-2).

This chapter explores the decline in center city-suburban inequality in black infant health in 37 metropolitan areas with large black populations. The analyses of Chapter 1 debunked a likely misperception that Washington, DC's exceptional infant health improvements in the 1990s were due to equally dramatic declines in nonmarital and teenage childbearing. Thus, any link between welfare reform and infant health gains could not have occurred through reductions in births to unmarried women and teenagers. Instead, a decline in prenatal smoking, or other, unmeasured risk factors associated with smoking, accounted for the largest identifiable share of Washington, DC's decline in low birth weight and preterm birth rates.

The first goal of this chapter, then, is to ascertain whether these findings are applicable to a broad sample of metropolitan areas with large black populations. Our findings are consistent with those for Washington, DC – measured changes in sociodemographic characteristics (age, marital status, education, parity) affected neither center city or suburban trends in black infant health nor spatial disparities, but the decline in prenatal tobacco exposure (or unmeasured correlates) did. We then test whether the rise in cigarette prices (and taxes) can account for the decline in spatial inequality in black infant health, but our data do not support this hypothesis. Finally, we use decomposition analysis to highlight the differing roles of changes in population composition, age-specific fertility rates, and within-age adverse birth outcomes. In general, changes in age-specific fertility rates and within-age rates of low birth weight and preterm birth explain more of the change in spatial inequality than changes in age-

related population composition. The existence of health gains within groups, especially higher-risk groups, is indicative of changes in unmeasured risk.

After we describe our data, we examine trends in sociodemographic and other risk factors for adverse birth outcomes in center cities and suburbs. We consider not only what changes may have contributed to center city and suburban trends in infant health, but also what (center city vs. suburban) *relative* changes may have affected (center city vs. suburban) *relative* birth outcomes. This is addressed both by examining simple trends, and more formally, by estimating multiple regression models. Our regression results are insensitive to choice of regression method (linear probability vs. logit models) and the inclusion or exclusion of metropolitan area fixed effects. We next extend the regression analysis to explore the role of increased cigarette prices (and taxes) in reduced spatial disparities in infant health. Finally, we decompose the changes in the gaps in low birth weight and preterm birth between center cities and suburbs into those due to changes in age-specific population composition, age-specific fertility rates, and within-age birth outcomes.

Data

Like those of Chapter 1, these analyses are based on birth certificate records contained in the 1990-2001 National Center for Health Statistics (NCHS) Natality Files. Thus, most of the variables we use have already been described in the previous chapter. Our outcomes of interest remain low birth weight and preterm birth. We continue to focus on singleton births to women ages 15 to 39, and include non-Hispanic black women living in the fifty metropolitan statistical areas (MSAs) or primary metropolitan

statistical areas (PMSAs) with the largest non-Hispanic black populations in the 1990 Census Summary File 1.[33] The latter restriction is designed to improve estimate precision.

Our prior analyses found that the singleton birth and maternal age restrictions did not substantively alter our main conclusions. Our results were also robust to alternative measures of adequacy of prenatal care and prenatal exposure to tobacco.[34] Since we have no reason to believe these restrictions or measurement issues would affect a broader sample differently than our Washington, DC sample, we do not explore their effects here.

In the previous chapter, our ability to measure adequacy of prenatal care was compromised by a large and increasing share of incomplete records. In 2001, 17 percent of Washington, DC birth records lacked the information necessary to classify adequacy of prenatal care. In this sample, adequacy of prenatal care cannot be determined in only 6 percent of both center city and suburban records, on average, and this number is relatively stable over time. Since Washington, DC is included in our expanded sample, however, we note that measuring adequacy of prenatal care may be problematic within some metropolitan areas.

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[33] A MSA is defined by the Federal Office of Management and Budget as a geographic entity with a high degree of social and economic integration between a large population core and surrounding areas. A PMSA meets the qualifications for a MSA, but is larger and has the additional restriction that areas within it have substantial commuting interchange. See http://factfinder.census.gov/home/en/epss/glossary_a.html for more details.

[34] Our main analysis measured adequacy of prenatal care using the Kotelchuck Index and prenatal smoking using both a dummy for usage and an interaction term of usage and dosage. In sensitivity analyses, we replaced the Kotelchuck Index with the Kessner Index, and measured prenatal smoking using only a usage dummy.

In this analysis, we must classify place of residence by center city or suburban status.[35] The Natality files include codes for city of residence for all places with population greater than 100,000 in 1990, but do not include an indicator for center city. We therefore use the correspondence between city of residence and center city indicator in the 2000 Census Summary File 1 to determine center city status in birth records. Four metropolitan areas have no births in a center city, and so are eliminated from the sample.[36] Nine are eliminated due to state- or city-wide non-reporting of prenatal tobacco and alcohol use for all or part of the study period.[37] A list of the remaining 37 metropolitan areas and their 1990 and 2000 population ranks is included in Appendix Table 2A-1. We consider any place within a MSA/PMSA not classified as center city to be suburban.[38] There are an average of 296,000 births per year in our sample, 64 percent of which occurred to residents of center cities.

The decomposition analysis requires population data. We employ the 1990 and 2000 Census Summary Files 1. Public use data availability limits the level of

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[35] The previous chapter explores the sensitivity of our results to selecting our sample on state of residence or state of occurrence. We have no choice but to use place of residence, here, because data on place of occurrence is available only at the state level.

[36] These are: Augusta, GA-SC; Charleston, SC; Nassau-Suffolk, NY; Columbia, SC.

[37] California did not collect information on either tobacco or alcohol use for the full duration of our study period. Indiana and New York City did not report smoking before 1999 and 1994, respectively. New York State began reporting alcohol use in 1994 and smoking in 1999. We lose the following MSAs/PMSAs as a result: Cincinnati, OH-KY-IN; Gary, IN; Indianapolis, IN; Los Angeles-Long Beach, CA; Louisville, KY-IN; New York, NY; Oakland, CA; Riverside-San Bernardino, CA; San Diego, CA; San Francisco, CA.

[38] Defining suburbs can be problematic. The methodology here follows that of Jargowsky (2001), among others. One problem lies in the diversity of suburbs. Lumping them together masks these differences, which may be important (Lucy and Phillips 2001).

disaggregation possible – for black females, we are able to disaggregate population counts by age only. In order to match population figures to birth data, we first obtained population data for black females by five year age categories living in each "place". We obtained identical population data measured at the MSA/PMSA level, as well. Since place of residence is identifiable in the Natality files only for places with population greater than 100,000 in 1990, we discard any places in the population data not meeting this criteria. Mappings of place to center city and place to MSA/PMSA are available in the 2000 Census, but not the 1990 Census. Therefore, we determine center city status and assign each place to a MSA/PMSA in both the 1990 and 2000 population data using the 2000 mappings. This may result in some error, though center city status seems unlikely to have changed for places in the large MSAs/PMSAs we study. Given this, and the fact that center cities seem less likely to have changed MSA/PMSA than non-center cities, we discard the latter. We then compute suburban populations in each MSA/PMSA as the difference between total MSA/PMSA population and center city population.

One additional problem is noteworthy. Race categories changed between the 1990 and 2000 Censuses. Respondents in 1990 were required to identify themselves as a single race, but those in 2000 were permitted to specify up to five different races. We use population data in 2000 from the "black only" category, which will likely undercount blacks somewhat relative to population data for blacks in 1990. Also, since age- and sex-specific population data are not available for non-Hispanic blacks in either year, for the purposes of the decomposition analysis, we re-draw birth data to include Hispanic blacks.

Hypotheses: Economic and Policy Context

This study address two key issues: 1) Why did center city and suburban infant health among blacks improve?, and 2) What explains the reduction in inequality between center city and suburban black infant health?

The rate of low birth weight at time, *t*, may be thought of as the average of the rate of low birth weight among *i* subpopulations, weighted by each subpopulation's share of all births:

$$LBW_t = \Sigma_i (B_{it} / B_t) * LBW_{it} \quad (1)$$

where *LBW* is the rate of low birth weight, and *B* is number of births. Therefore, the center city-suburban gap in low birth weight is:

$$LBW^{cc}{}_t - LBW^{sub}{}_t = \Sigma_i \{[(B^{cc}{}_{it} / B^{cc}{}_t) * LBW^{cc}{}_{it}] - [(B^{sub}{}_{it} / B^{sub}{}_t) * LBW^{sub}{}_{it}]\} \quad (2)$$

and the center city-suburban ratio in low birth weight is:

$$LBW^{cc}{}_t / LBW^{sub}{}_t = \Sigma_i (B^{cc}{}_{it} / B^{cc}{}_t) * (B^{sub}{}_t / B^{sub}{}_{it}) * (LBW^{cc}{}_{it} / LBW^{sub}{}_{it}) \quad (3)$$

All else equal, then, a decline in the share of center city births to a subpopulation with elevated risk for low birth weight relative to that subpopulation's share of suburban births, or a relative (center city vs. suburban) within-group decline in low birth weight will tend to reduce spatial inequality in the rate of low birth weight. An analogous argument applies to changes in the spatial distribution of preterm birth. Put another way, the decline in center city-suburban disparities in black infant health could be due to a shift in the profile of risk factors for adverse birth outcomes favoring center cities or improved health among high-risk groups relative to low-risk groups.

Several economic and policy changes are likely to have affected center cities more than suburbs and could account for the reduction in spatial disparities in infant health among blacks. First, the goals of the 1996 Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA) included decreasing teenage and nonmarital childbearing, and the welfare population disproportionately resides in center cities. Although there is little evidence that welfare reform affected out-of-wedlock or teenage fertility (Acs and Koball 2003; Joyce, Kaestner et al. 2003), we nonetheless test whether relative (center city vs. suburban) changes in the shares of births to unmarried women and teenagers contributed to declining spatial inequality.

PRWORA also introduced work requirements designed to increase employment among low-income populations. These (and the strong economy of the 1990s) may have raised the socioeconomic status (SES) of low-income city dwellers relative to their suburban counterparts. There is evidence that overall relative socioeconomic status improved – the ratio of center city-suburban per capita income grew by 3 percentage points between 1990 and 1999 (Dreier, Mollenkopf et al. 2001).[39]

While the excess risk of center city women for adverse birth outcomes may have declined because SES-related risk changed among fixed populations, residential mobility could also have changed center city vs. suburban SES-related risk profiles. During the 1990s, robust GDP growth of 3 percent per year, on average, led to urban revival in many large metropolitan areas (U.S. Department of Commerce; Grogan and Proscio 2000).

[39] However, this figure does not indicate whether relative socioeconomic status improved within metropolitan areas. While this could be indicative of region-wide increases in income in areas with large cities combined with stagnating or lesser increases in regions with large suburban populations, the fact that poverty declined in center cities and rose in many inner ring suburbs is at least suggestive of within-MSA changes in relative socioeconomic status (Jargowsky 2003).

Jargowsky's finding that the spatial distribution of poverty in many metropolitan areas shifted away from center cities over the 1990s and towards many inner-ring suburbs is consistent with the hypothesis that low SES, low-risk women from the suburbs sought gentrified urban neighborhoods and/or high SES, high-risk women fled them (Jargowsky 2003).

Whichever the underlying reason for center city improvements in SES relative to suburbs, this hypothesis is difficult to test directly because our data lack measures of family income and employment status. Instead, we examine whether changes in educational attainment affected trends in center city and suburban birth outcomes and thus spatial inequality. We caution, however, that even though income and employment are correlated with educational attainment, schooling may not adequately measure socioeconomic status.

Another possibility is that increased health insurance coverage among the poor, who are concentrated in center cities, improved access to prenatal care, and greater use of prenatal care among this population reduced the urban-suburban infant health gap among blacks. Federal legislation in 1990 raised the maximum income allowed for Medicaid eligibility of pregnant women from 100 to 133 percent of the Federal Poverty Level, and many states implemented additional expansions in the subsequent years. Therefore, we also consider whether relative (center city vs. suburban) changes in the use of prenatal care contributed to declining spatial disparities. Most other research has found, however, that although Medicaid expansions raised the use of prenatal care, increased prenatal care

has only minimal impact on adverse birth outcomes (Buescher, Roth et al. 1991; Fiscella 1995; Levinson and Ullman 1998; Joyce 1999; Dubay, Joyce et al. 2001).[40]

Our final hypothesis stems from Chapter 1's finding that in Washington, DC, only the decline in smoking had a substantial, identifiable impact on reductions in the rates of low birth weight and preterm birth. Although we were unable to rule out the possibility that the effect of reduced tobacco use reflects unmeasured crack use, we test in this chapter whether increases in cigarette prices during the 1990s helped reduce spatial inequalities in black infant health. Cities have high smoking rates relative to suburbs, and cigarette prices rose by an average of $1.46 per pack between 1990 and 2001 in the states in our sample. In some states, cigarette tax increases account for one-third of price increases (The Tobacco Institute 2003). Several studies have found cigarette taxes to affect rates of prenatal smoking and birth outcomes (Evans and Ringel 1999; Colman, Grossman et al. 2003).

Regression Methods

In order to examine formally trends in center city and suburban black infant health and the related decline in spatial disparities, we estimate a series of linear probability regression models of the form:

$$outcome = \alpha + \beta * time + \chi * cc + \delta * time * cc + \varepsilon' MSA + \phi * female + \gamma' X \qquad (4)$$

where *time* is measured in years, *cc* is a dummy for center city, *MSA* is a vector of dummies representing 36 MSAs/PMSAs, *female* is a dummy for infant sex, and *X* is a vector of additional explanatory variables. We build the model incrementally, varying the covariate set contained in *X*, and observe how β, and δ change. Coefficients reported in

[40] An exception is Medicaid expansions in Florida (Long and Marquis 1998).

all tables are inflated by a factor of 100. The coefficient, β, is the regression-adjusted yearly percentage point change in the outcome in suburbs, and δ, the additional change in center cities, reflects changes in the urban-suburban gap. Thus, β + δ is the regression-adjusted yearly percentage point change in the outcome in center cities. All standard errors are robust standard errors and are corrected for heteroscedasticity. We use metropolitan area fixed effects to compare center cities to suburbs within the same MSA/PMSA. Later, we show that our results are not sensitive to estimating linear probability models and the fixed effects specification.

Descriptive Statistics

Table 2-1 presents means for our full set of explanatory variables and outcomes, overall and disaggregated by center city-suburban status.[41] Rates of low birth weight and preterm birth are higher in center cities than in suburbs. Unsurprisingly, then, positive correlates of adverse birth outcomes characterize a larger share of births in center cities. The nonmarital share of births is 1.25 times higher in center cities than in suburbs, and infants in center cities are more likely to be born to a teenager and/or to a woman without a high school diploma.[42] Smoking and alcohol use are 1.5-1.6 times more common among center city mothers. Given that they smoke, however, there is no difference in

[41] These means are effectively weighted by the number of births in each MSA/PMSA. Computing them with each metropolitan area contributing equally does not generally affect the trends evident in this table.

[42] This latter point has partially to do with the fact that the teen share is higher in center cities. However, even among women ages 25-39, the gap between center cities and suburbs in the share of births to women who have not completed high school is 8.6 percentage points.

frequency of use. Women in center cities are also more likely to have received inadequate prenatal care.

The suburban advantage is less evident in the presentation of medical risk factors and complications of labor and/or delivery. Center city women are only slightly more likely to have any medical risk factors and equally likely to have had a previous preterm or intrauterine growth retarded (IUGR) birth. As we do in Chapter 1, we treat history of preterm or IUGR birth separately from other medical risk factors in our regression models because it is a strongly emphasized risk factor for adverse birth outcomes in the medical literature (Kramer 1987; Goldenberg, Iams et al. 1998; Meis, Goldenberg et al. 1998).[43] Center city women are less likely to have at least one of 16 complications of labor and/or delivery reported on birth certificates.[44,45]

Results

Trends

Like Washington, DC, the 37 metropolitan areas we study here experienced declines in adverse birth outcomes as well as declines in births to many of the highest risk

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[43] Excepting preterm/IUGR history, there are 16 medical risk factors on birth certificates: anemia, cardiac disease, diabetes, genital herpes, hydramnios/oligohyramnios, hemoglobinopathy, chronic and pregnancy-associated hypertension, eclampsia, an incompetent cervix, renal disease, RH sensitization, uterine bleeding, a previous infant born weighing 4,000 or more grams, and "other".

[44] These are: meconium, premature rupture of the membrane, abruptio placenta, placenta previa, other excessive bleeding, seizures during labor, prolonged labor, dysfunctional labor, breech, cephalopelvic disproportion, cord prolapse, anesthetic complications, fetal distress, and "other".

[45] Unlike Chapter 1, we enter medical risk factors and complications of labor as single dummies representing "any" medical risk factors or "any" complications of labor. Although we argue in Chapter 1 that these conditions may represent different risks, computing limitations preclude their disaggregation in this analysis.

groups. These changes are summarized in Table 2-1, but we also show the 1990 to 2001 change in low birth weight and preterm birth in each individual metropolitan area in Figures 2A-1 and 2A-2 of the Appendix. Rates of low birth weight and preterm birth declined 9 and 11 percent, respectively, in center cities. The trend in suburban preterm birth was comparable to that of cities, but the reduction in suburban low birth weight was quite modest. These changes led to 32 to 21 percent declines in the low birth weight and preterm birth center city-suburban gaps.[46]

Many types of risk for adverse birth outcomes fell, too. The shares of births to women ages 15-19 and with less than a college education declined steeply, though the nonmarital birth ratio did not. The rate of prenatal smoking halved in both center cities and suburbs, and the incidence of prenatal alcohol use declined even more markedly. The share of women receiving inadequate prenatal care fell, while the share receiving intermediate or adequate care rose. The incidence of medical risk factors and complications of labor and/or delivery rose, however.

Sociodemographic changes are unlikely to have contributed to declining spatial inequality in infant health because the profile of these risk factors did not change in center cities relative to in suburbs. Only trends in prenatal tobacco and alcohol use and the share of women receiving inadequate prenatal care shifted favorably for center cities. Women in center cities were 4.5 percentage points more likely to smoke during pregnancy than suburban mothers in 1990, and this gap fell to 2.9 percentage points in 2001, or by 36 percent. The urban-suburban gap in shares of births exposed to prenatal alcohol use declined an even more dramatic 57 percent. By contrast, the center city-

[46] The ratios of center city to suburban low birth weight and preterm birth rates also declined, by 5 and 2 percent, respectively.

suburban gap in the share of women with low educational attainment rose, and the corresponding gaps in the shares of births to umarried and teenage mothers changed little.

One exception stands out. The suburban share of births to women ages 35-39, who are at elevated risk for adverse birth outcomes, exceeds that in center cities, and this gap more than doubled during the 1990s. However, births to women ages 35-39 comprise only a small share of all births in either location – 7 percent, in 2001, compared with 21 percent for teens.

Appendix Figures 2A-3 – 2A-6 show that there were also within-group health improvements, especially among high-risk births to unmarried and poorly educated women, those exposed to tobacco in utero, and women receiving inadequate prenatal care. Since high-risk births comprise a larger share of center city than suburban births, greater improvements among high-risk births may have contributed to improved infant health in center cities relative to suburbs. On the other hand, infant health was stagnant among lower risk married women and those with at least a college education (not shown). Teens had lower rates of preterm birth (but not low birth weight) in 2001 than in 1990, but women ages 20-34 account for most of the improvement as well as the closing disparity between center city and suburban infant health (not shown).

Main regression models

Table 2-2 presents the results of our regression analyses. These models test whether changes in sociodemographic characteristics – age, marital status, education, and parity – can explain trends in center city and suburban infant health and the associated decline in spatial inequality. They also explore the roles of risky behaviors such as prenatal smoking and alcohol use and adequacy of prenatal care.

Model 1, which adjusts only for time, infant sex, and metropolitan area of residence, estimates that suburban low birth weight and preterm birth declined 0.05 and 0.15 percentage points per year (Columns 1a and 1b), respectively, while these measures declined 0.12 and 0.23 percentage points per year in center cities (Columns 3a and 3b). These figures amount to 1.4 and 2.8 percentage point declines in center city low birth weight and preterm birth between 1990 and 2001. The "extra" decline in low birth weight and preterm birth rates in center cities relative to suburbs within the same metropolitan area is 0.07-0.08 percentage points per year, or 0.8-1.0 percentage points over 12 years (Columns 2a and 2b).

Why did center city and suburban infant health improve?

We first discuss center city and suburban regression-adjusted trends in infant health. These can be found in the Columns 1a/1b and 1c/2c of Table 2-2. Models 2-6 gradually add controls for demographic characteristics and socioeconomic status. Since the estimated trends in suburban low birth weight and preterm birth are identical in Models 1 and 2, changes in marital status had no impact on the decline in suburban infant health. We reach a similar conclusion with respect to the much larger improvement in health among center city infants.[47] In fact, none of the sociodemographic controls included in Models 2-6 reduce the estimated trends in birth outcomes in either residential setting. Controlling for maternal age actually increases estimated declines in low birth weight and preterm birth, suggesting that if not for changes in the age distribution of mothers (away from teens and towards women ages 35-39), infant health improvements

[47] This is consistent with the small decline in the share of births to unmarried women in both center cities and suburbs.

would have been even greater. This is not surprising when one realizes that women ages 35 and older have elevated risk for adverse birth outcomes, and among African-Americans, teens have the lowest risk. Thus, our finding for Washington, DC – that changes in sociodemographic characteristics are not responsible for infant health improvements among blacks – is also applicable to a broad national sample of center cities and suburbs.

Models 7-13 include all of the regressors in Model 6 and add controls, one by one, for risky behaviors, adequacy of prenatal care, pregnancy history, and other, mediating factors that may reflect otherwise unmeasured risk. Between Models 6 and 7, the regression-adjusted trends in infant health outcomes in both center cities and suburbs shrink markedly. The decline in prenatal smoking had a large effect on health gains among black infants, even when we control for infant sex, marital status, maternal age and education, and birth order. The estimated decline in center city low birth weight, for example, falls from 0.13 percentage points per year in the model that controls only for sociodemographic characteristics to 0.04 in the model that also controls for prenatal tobacco exposure. The suburban trend in low birth weight becomes positive, but statistically insignificant, though the decline is negligible, initially. Smoking plays a lesser role in the decline in the preterm birth rate, but still accounts for 26 percent of that decline in center cities and 27 percent in suburbs.

These findings diverge somewhat from those for our analysis of Washington, DC with respect to prenatal alcohol use. Here, a reduction in alcohol use during pregnancy has an impact on the low birth weight trend independent of prenatal smoking (Model 8). The estimated center city decline in low birth weight falls by half when we control for

prenatal alcohol exposure. The trend in the rate of suburban low birth weight, which is zero in Model 7, becomes statistically significantly positive when we control for prenatal alcohol use. The trend in alcohol use had little impact on the change in the rate of preterm birth in either center cities or suburbs, on the other hand. Like the effect of the decline in prenatal alcohol use on the reduction in suburban low birth weight, changes in the adequacy of prenatal care appear to also have worsened infant health.

As in the previous chapter, we interpret the effects of covariates that we add to models 10-13 as effects of mediating factors rather than root causes of poor infant health. The decline in the share of women with a history of preterm or IUGR infant(s) had very little effect on trends in adverse birth outcomes (Model 10). Model 11 suggests, predictably, that changes in pregnancy term are associated with a decrease in the rate of low birth weight in both center cities and suburbs.[48] Changes in the share of women with any medical risk factors had health-improving effects on trends in center city and suburban rates of low birth weight, but health-worsening effects on center city and suburban trends in preterm birth (Models 12).[49] Trends in the center city and suburban shares of women with complications of labor and/or delivery did not contribute to improvements in infant health (Model 13).

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[48] In the regressions, pregnancy term is classified as full-term, spontaneous preterm, or non-spontaneous preterm, where non-spontaneous indicates premature induction of labor or c-section. This covariate is not included in regressions in which preterm birth is the outcome.

[49] In the Washington, DC analysis, regression models include dummies for each of the 16 medical risk factors or complications of labor and/or delivery. Due to a much larger sample size in this analysis and constraints on computing capacity, we use single dummies here for "any" medical risk factors or "any" complications of labor.

What explains the decline in spatial inequality in infant health?

Columns 2a and 2b of Table 2-2 show the differential between the center city and suburban trends, adjusted for a varying set of factors. Changes in this coefficient reflect changes in the spatial disparity of health among infants born to African-Americans living in the 37 metropolitan areas in our sample. That this coefficient is statistically significant regardless of specification and outcome indicates that none of our models fully explain the decline in the center city-suburban gap in infant health.

Still, changes in some risk factors affected the gap. While changes in marital status, maternal age or education, and birth order explain little if any of the reduction in spatial inequality in rates of low birth weight or preterm birth, the greater decline in smoking in center cities had a substantial effect on the center city-suburban gap in low birth weight and a modest effect on the preterm birth gap. The 0.02 percentage point difference in the time-center city interaction coefficient between low birth weight regression models 6 and 7 implies that about 30 percent of the decline in the low birth weight gap not due to changes in sociodemographic risk factors can be attributed to changes in smoking rates. This is twice the effect of reduced prenatal smoking on the gap in preterm birth rates. Model 8 reveals, however, that the trend in prenatal alcohol use had no impact on the center city-suburban gap in either outcome beyond that already captured by smoking. Since changes in the adequacy of prenatal care were associated with worsened infant health in center cities, they also raised the degree of inequality between center cities and suburbs (Model 9).

The final four models suggest varying roles for the proximate causes we are able to measure. The trend in pregnancy history did not affect changes in urban-suburban disparities in low birth weight and preterm birth (Model 10). Unsurprisingly, however,

the decline in the center city-suburban gap in preterm birth is linked strongly to a reduced center city-suburban differential in low birth weight (Model 11). Controlling for pregnancy term changes the coefficient on the interaction term in the low birth weight regression from -0.07 to -0.03. In both the low birth weight and preterm birth regressions, changes in medical risk factors and complications of labor have modest to no impact on the urban-surburban gaps (Models 12-13).

In summary, sociodemographic changes affected neither the trends in center city and suburban rates of low birth weight or preterm birth nor the decline in urban-suburban spatial health disparities in the 1990s. Declines in prenatal exposure to tobacco contributed substantially to both center city and suburban trends in adverse birth outcomes as well as declining spatial inequalities. The same conclusions hold with respect to pregnancy term (in the low birth weight regressions). If not for reductions in prenatal alcohol use, center city and suburban rates of low birth weight would have risen, but the beneficial effects of less exposure to alcohol did not affect the urban-suburban gap. Trends in the adequacy of prenatal care, on the other hand, had the opposite effects. Rates of center city and suburban low birth weight and preterm birth as well as the gap in these rates between center cities and suburbs would have been lower if not for these trends. Changes in the share of women with medical risk factors contributed to the declines in center city and suburban low birth weight, but dampened improvements in rates of preterm birth.

Robustness checks

In this section, we examine whether these conclusions are sensitive to estimation method or metropolitan area fixed effects specification. We estimate linear probability

models because they easier to interpret than logit or probit models, especially in the presence of interaction terms, but they are often criticized because predicted outcomes are not constrained to lie between 0 and 1 (Ai and Norton 2003). Therefore, we repeat the analysis, estimating logit rather than linear probability models. We report these results in Table 2A-3 in the Appendix. We show both estimated coefficients and odds ratios (and the robust standard errors of each). Though decomposition effects are difficult to derive from logit model results, this table verifies that the sign and significance of key covariates are not sensitive to the choice of logit or linear probability model specification.

We include fixed effects in our main analysis because we are interested in center city-suburban spatial inequality within metropolitan areas. Excluding fixed effects compares all center cities to all suburbs. Therefore, metropolitan area-specific conditions could give the impression, for example, that the teen share of births in center cities fell relative to that in suburbs, when in fact the teen share of births fell in metropolitan areas with large center cities relative to that in metropolitan areas with large suburbs. Our results suggest this need not be of concern, however. When we re-estimate our models without metropolitan area fixed effects, our conclusions remain unchanged (Table 2A-2).

The role of cigarette prices

Since prenatal smoking is the only identifiable risk factor to have substantially contributed to the reduction in spatial infant health disparities, we explore in this section whether increases in cigarette prices contributed to declining inequalities, using price data from The Tobacco Institute (2003). In the 24 states covered by our sample, federal and state cigarette taxes rose 26 cents per pack during the 1990s and the total cost of cigarettes rose by $1.46. Other research has found that cigarette taxes can impact both

rates of prenatal smoking and birth outcomes (Evans, Farrelly et al. 1999; Colman, Grossman et al. 2003; The Tobacco Institute 2003). Therefore, we estimate two variants of Model 7. First, we replace prenatal smoking with cigarette prices. Second, we estimate Model 7 with controls for both tobacco use and cigarette prices. We present these results in Table 2-3. For ease of comparison, we display our original results from Models 6 and 7 in Columns 1 and 2 of the table.

These analyses do not generally support a role for rising cigarette prices in explaining either the trends in center city or suburban infant health or the decline in spatial health disparities among black infants. Comparing Columns 1 and 3 suggests that the increase in cigarette prices is associated with infant health deterioration – not improvement – in both center cities and suburbs and not related to the trend in spatial inequality. When we add cigarette prices to the models that control for prenatal smoking, the estimated decline in center city and suburban low birth weight as well as the low birth weight gap between residential settings declines (Panel A; Columns 2 vs. 4), but this is not true for preterm birth rates (Panel B). Thus, the increase in cigarette prices (and taxes) may have contributed to the decline in spatial inequality in low birth weight, but it is strange that this finding only holds in the model that controls for prenatal smoking. Also discouraging is the fact that the coefficients on cigarette prices are always of the wrong sign. Adding cigarette prices to the models that control for smoking does not impact the tobacco coefficients in either the low birth weight or preterm birth regressions. This suggests any effects of cigarette prices on adverse birth outcomes do not operate through the effects of prenatal smoking and frequency of tobacco use.

Prior research may help explain these non-findings. Ebrahim, et al found that decline in smoking among pregnant women between 1987 and 1996 was due primarily to a decline in smoking initiation rates rather than pregnancy-related quit rates (Ebrahim, Floyd et al. 2000). Therefore, changes in prenatal smoking may be more appropriately attributed to changing cigarette prices occurring earlier in life – before pregnancy – at least for women giving birth out of their teen years. Another reason for our non-result may be lack of statistical power. One study concluded that 10 million births were needed before the reduced form effect of taxes on birth weight could be detected (Evans and Ringel 1999). Or, if the effect of declining smoking rates largely reflects a reduction in unmeasured crack use, then our finding that increases in cigarette prices did not contribute to the decline in spatial inequality in infant health is perfectly reasonable.

Decomposition analysis

In this section we decompose the change in spatial health disparities among black infants in center cities and suburbs into those due to group-specific changes in population composition, group-specific fertility rates, and within-group adverse birth outcome rates. Equation (1) can be rewritten as:

$$LBW_t = (POP_t / B_t) * \Sigma_i (POP_{it} / POP_t) (B_{it} / POP_{it}) * LBW_{it} \qquad (5)$$

by multiplying both sides of the equation by $(POP_t / POP_t) *(POP_{it} / POP_{it})$. The first term in the summation sign is group *i*'s population share, the second term is group *i*'s fertility rate, and the last term, as in equation (1), is group *i*'s rate of low birth weight. Expressing the rate of low birth weight as a function of all rates avoids bias in the decomposition introduced by population growth. The center city-suburban gap in low birth weight is then:

$$LBW^{cc}_{t} - LBW^{sub}_{t} = [(POP^{cc}_{t} / B^{cc}_{t}) * \Sigma_i (POP^{cc}_{it} / POP^{cc}_{t}) (B^{cc}_{it} / POP^{cc}_{it}) * LBW^{cc}_{it}] - [(POP^{sub}_{t} / B^{sub}_{t}) * \Sigma_i (POP^{sub}_{it} / POP^{sub}_{t}) (B^{sub}_{it} / POP^{sub}_{it}) * LBW^{sub}_{it}] \quad (6)$$

Analogous relationships can be constructed for preterm birth rates. As mentioned earlier, we are restricted in the level of disaggregation possible by population data availability. Therefore, we disaggregate by five age categories (15-19, 20-24, 25-29, 30-34, 35-39) and MSA/PMSA, although a finer disaggregation involving at least marital status and education would be preferred. Since we must rely on the decennial Censuses for population data, this analysis compares changes between 1990 and 2000 and includes Hispanic blacks.

In order to decompose the change in the rate of low birth weight (or preterm birth) gap between center cities and suburbs, we allow to change, one-by-one, group specific population shares, fertility rates, and low birth weight (or preterm birth) rates. For example, to see the effect of changes in population composition, we compute the center city and suburban rates of low birth weight (or preterm birth) in 2000 using group-specific fertility rates and low birth weight (or preterm birth) rates in 1990, and group-specific population shares in 2000. We then take the difference between center cities and suburbs and compare this to the true gap in 1990. We similarly estimate the contribution of changes in group-specific fertility rates or birth outcome rates by allowing only fertility rates or only birth outcome rates to change to their 2000 levels.

These results are presented in Tables 2-4 and 2-5. We estimate the 1990 to 2000 change in low birth weight (Table 2-4) and preterm birth (Table 2-5) gaps for all

MSAs/PMSAs combined and for each individually. We show the true change in the first column of each table. Looking at the first row of Table 2-4, we see that the true change in the center city-suburban low birth weight gap between 1990 and 2000 among all 37 MSAs/PMSAs combined was -1.05. If only group-specific population shares had changed, this would have been -0.74. Therefore, changes in population composition contributed to about 70 percent of the decline in spatial inequality in low birth weight among black infants. However, this analysis does not indicate whether these changes are due to changes among a fixed population or residential mobility. Changes in group specific fertility and low birth weight rates – individually – accounted for all (and more) of the reduction in spatial disparities in low birth weight. A similar conclusion applies to the change in spatial inequality in preterm birth rates among all 37 MSAs/PMSAs combined, and generally, within individual metropolitan areas.

The tables highlight some interesting differences within metropolitan areas, though. A select few MSAs/PMSAs, such as Milwaukee, WI and Jacksonville, FL experienced increasing spatial inequality during the 1990s. Some, like Baltimore, MD and Washington, DC experienced vast reductions in their urban-suburban gaps in infant health. In Fort Lauderdale, FL and Columbus, OH, the declines in spatial inequality in preterm birth would have been 3 and 14 and times as great if only group-specific fertility rates had changed. In Orlando, FL, spatial inequality in preterm birth would have increased 5 times more than it actually did if only group-specific fertility rates had changed.

Discussion

Although the scale of Washington, DC's health gains are largely unmatched by other cities with substantial black populations (see Tables 2A-2 and 2A-3 in the Appendix), improvements in black infant health were not limited to the District of Columbia. Adverse birth outcomes were stagnant or increased for the US as a whole and for whites, but declined among black infants living in all metropolitan areas and in the 37 metropolitan areas this chapter studies. Furthermore, there was a significant shift in the spatial distribution of black infant health in US metropolitan areas in the 1990s. African-American infants in center cities have higher rates of low birth weight and preterm birth than black infants in nearby suburbs, but the center city-suburban gap in black infant health declined between 1990 and 2001.

Chapter 1 concludes that infant health gains among blacks in Washington, DC cannot not be attributed to changes in sociodemographic risk, but rather (in part) to a steep decline in either prenatal smoking or unmeasured risk associated with tobacco use, such as the use of crack cocaine. The findings of this chapter, which analyze a broader, national sample of metropolitan areas with large black populations, are consistent with those for Washington, DC. Changes in age, marital status, education, and birth order did not affect center city or suburban trends in infant health or the decline in spatial health disparities. The decline in smoking emerged, again, as the single identifiable contributing factor, although this analysis is also vulnerable to the possibility that the effect of tobacco use reflects the effect of unmeasured crack use. This may be one reason why we find that increased cigarette prices (and taxes) did not contribute to the reduction in spatial

inequality in infant health, though this may be due instead to mismeasurement or lack of statistical power.

We find evidence of both changes in relative risk for adverse birth outcomes and changes in the probability of adverse outcomes conditional on risk, and these changes contributed to declines in spatial disparities in infant health. While risk associated with sociodemograhic characteristics in center cities did not decline relative to that in the suburbs, lifestyle-related risk did. For example, the gaps in rates of prenatal smoking and alcohol use between center cities and suburbs narrowed substantially during the 1990s despite decreased smoking and drinking in both residential settings. Within-group declines in adverse birth outcomes occurred primarily among high-risk demographic groups such as teens, single women, women with low education, those receiving inadequate prenatal care, and infants exposed in utero to tobacco. The contribution of within-group declines in low birth weight and preterm birth is evident in both our regression and decomposition analyses.

Distinguishing further between the effects of behavioral changes, migration, or fertility changes could help improve health and social policy. For example, our research suggests that anti-smoking interventions in the 1990s benefited center city African-American infants, although the influence of reduced smoking may reflect, at least in part, the waning of the crack epidemic. It also suggests that, although reductions in births to teens and women with little education were substantial, these reductions explained neither improvements in relative infant health between center cities and suburbs nor absolute infant health in center cities.

Many urban centers experienced substantial economic revival and gentrification during the 1990s. Evidence for the migration hypothesis would raise concerns about transplanted high-risk women living in suburbs, where access to quality health care may be more limited. It would also suggest caution in celebrating relative health gains in center cities. Decomposition analysis supports a role for changes in population composition in the decline in spatial inequality, but does not allow us to distinguish between changes in risk for adverse birth outcomes among a fixed population or changes in risk due to residential mobility. Even if migration is responsible for just a share of the relative center city-suburban improvement in infant health, then this improvement would overstate the true relative health gains achieved by a constant urban population.

Table 2 - 1. Summary Statistics for Singleton Births to Black Women in 37 Metropolitan Areas, 1990 & 2001

Percents, unless indicated otherwise

	1990			2001			CC-Sub Gap: 2001-1990
	Center cities	*Suburbs*	*CC-Sub Gap*	*Center cities*	*Suburbs*	*CC-Sub Gap*	
Number of births	223,680	97,357		167,766	115,300		
Outcomes							
Low birth weight (LBW)	12.6	10.7	1.9	11.8	10.5	1.3	-0.6
Preterm birth (PRETERM)	18.7	16.8	1.9	16.8	15.4	1.5	-0.4
Covariates							
Female infant (FEMALE)	49.3	49.5	-0.2	49.3	49.6	-0.3	-0.1
Married (MARRIED)	28.9	40.7	-11.8	27.0	38.6	-11.6	0.2
Maternal age (AGE)							
15-19	25.1	20.5	4.6	20.8	16.5	4.3	-0.3
20-24	32.6	30.5	2.2	35.0	30.5	4.5	2.4
25-29	23.9	26.4	-2.4	23.1	24.2	-1.1	1.3
30-34	13.6	16.6	-3.1	14.3	18.8	-4.6	-1.5
35-39	4.7	6.0	-1.3	6.8	10.0	-3.1	-1.9
Maternal educational attainment (EDUC)							
< High school	32.3	24.1	8.2	27.7	19.4	8.4	0.1
High school	41.5	41.5	0.0	38.9	36.5	2.4	2.4
College +	25.0	32.9	-7.9	31.7	42.7	-11.0	-3.1
Unknown	1.2	1.4	-0.3	1.7	1.4	0.3	0.6

Table continues on the next page. See notes at the end of the table.

Table 2 - 1, continued

	1990			2001			CC-Sub Gap:
	Center cities	*Suburbs*	*CC-Sub Gap*	*Center cities*	*Suburbs*	*CC-Sub Gap*	**2001-1990**
Parity (PARITY)							
0	37.0	39.6	-2.6	38.0	39.6	-1.6	1.0
1+	62.7	59.9	2.8	61.7	60.2	1.5	-1.3
Unknown	0.2	0.5	-0.2	0.3	0.2	0.0	0.3
Prenatal tobacco use (TOBACCO)							
Yes	16.5	12.8	3.6	9.0	7.2	1.8	-1.8
Unknown	6.1	6.3	-0.1	2.3	2.6	-0.3	-0.2
No. cigaretts/day conditional on use (TOBXCIGNUM)	10.1	9.9	0.2	7.3	7.7	-0.3	-0.5
Prenatal alcohol use (ALCOHOL)							
Yes	3.7	3.0	0.7	0.9	0.8	0.1	-0.5
Unknown	6.2	6.4	-0.1	2.3	2.6	-0.3	-0.2
Prenatal care (PNC)							
Inadequate	31.3	27.1	4.2	19.8	17.5	2.4	-1.8
Intermediate	21.5	20.7	0.8	18.2	19.8	-1.6	-2.4
Adequate	28.7	31.6	-2.8	35.5	38.3	-2.8	0.0
Adequate plus	14.2	16.1	-1.8	20.8	20.5	0.3	2.1
Unknown	4.2	4.5	-0.3	5.6	3.9	1.7	2.0

Table continues on the next page. See notes at the end of the table.

Table 2 - 1, continued

	1990			2001			CC-Sub Gap: 2001-1990
	Center cities	*Suburbs*	*CC-Sub Gap*	*Center cities*	*Suburbs*	*CC-Sub Gap*	
Pregnancy term (TERM)							
Full-term birth	81.3	83.2	-1.9	83.2	84.6	-1.5	0.4
Spontaneous preterm birth	14.4	12.3	2.0	10.6	9.2	1.4	-0.7
Nonspontaneous preterm birth	4.3	4.5	-0.2	6.3	6.2	0.1	0.3
Previous preterm or IUGR birth (PREV)							
Yes	1.3	1.2	0.0	1.3	1.2	0.1	0.1
Unknown	3.4	3.0	0.4	0.2	0.2	0.0	-0.4
Any medical risk factors (ANYMR)							
Yes	23.2	21.2	2.0	34.1	31.8	2.2	0.3
Unknown	3.4	3.0	0.4	0.2	0.2	0.0	-0.4
Any complications of labor and/or delivery (ANYLB)							
Yes	33.1	34.3	-1.2	32.5	33.4	-0.8	0.4
Unknown	3.1	2.6	0.5	0.1	0.1	0.0	-0.5

Notes:

1 Variable names in parentheses.

2 See Table 2A-1 for a list of MSAs included in the sample.

Table 2 - 2. Selected Regression Coefficients From Linear Probability Models Explaining Adverse Birth Outcomes Among Black Women in 37 Metropolitan Areas, 1990-2001

		Outcome = LBW			Outcome = PRETERM		
Model	**Covariates**	***β* = *Sub Trend* (1a)**	***δ* = *CC/Sub trend differential* (2a)**	***β* + *δ* = *CC Trend* (3a)**	***β* = *Sub Trend* (1b)**	***δ* = *CC/Sub trend differential* (2b)**	***β* + *δ* = *CC Trend* (3b)**
1	FEMALE	-0.05*	-0.07*	-0.12*	-0.15*	-0.08*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
2	FEMALE, MARRIED	-0.05*	-0.08*	-0.12*	-0.15*	-0.08*	-0.24*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
3	FEMALE, AGE	-0.06*	-0.06*	-0.13*	-0.16*	-0.07*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
4	FEMALE, MARRIED, AGE	-0.08*	-0.06*	-0.14*	-0.18*	-0.07*	-0.25*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
5	FEMALE, MARRIED, AGE, EDUC	-0.06*	-0.07*	-0.12*	-0.15*	-0.08*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
6	FEMALE, MARRIED, AGE, EDUC, PARITY	-0.05*	-0.07*	-0.13*	-0.15*	-0.07*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)

Table continues on the next page. See notes at the end of the table.

Table 2 - 2, continued

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
7	6 + TOBACCO, TOBXCIGNUM	0.01	-0.05*	-0.04*	-0.11*	-0.06*	-0.17*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
8	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	0.03*	-0.05*	-0.02*	-0.10*	-0.06*	-0.16*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
9	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	0.02*	-0.07*	-0.05*	-0.15*	-0.11*	-0.25*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	0.01	-0.07*	-0.06*	-0.15*	-0.11*	-0.26*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
11	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV, TERM	0.05*	-0.03*	0.03*			
		(0.01)	(0.01)	(0.01)			

Table continues on the next page. See notes at the end of the table.

Table 2 - 2, continued

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
12	11 + MRF(16)	0.02*	-0.03*	-0.01	-0.20*	-0.11*	-0.31*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
13	11 + MRF(16), CLB(16)	0.02*	-0.02*	0.00	-0.20*	-0.10*	-0.30*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
	Approximate N		3,500,000			3,500,000	

* p<0.05

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 2-1 for variable definitions and categories.

3 All models include controls for MSA-level fixed effects.

Table 2 - 3. Regression Estimates of the Effects of Cigarette Prices on Adverse Birth Outcomes Among Black Women in 37 Metropolitan Areas, 1990-2001

	Panel A: Outcome = LBW			
Covariate	1	2	3	4
Suburban trend	-0.05*	0.013	-0.08*	0.001
	(0.01)	(0.01)	(0.01)	(0.01)
Center city-suburban trend differential	-0.07*	-0.05	-0.07*	-0.05*
	(0.01)	(0.01)	(0.01)	(0.01)
Center city trend	-0.13*	-0.04*	-0.15*	-0.05*
	(0.01)	(0.01)	(0.01)	(0.01)
Prenatal smoking		5.95*		5.95*
		(0.11)		(0.11)
Cigarettes per day, if smoked		0.37*		0.37*
		(0.01)		(0.01)
Cigarette price (in cents)			0.002*	0.001*
			(0.00)	(0.00)
N		3,544,292		

Table continues on the next page. See notes at the end of the table.

Table 2 - 3, continued

	Panel B: Outcome = Preterm			
Covariate	1	2	3	4
Suburban trend	-0.15*	-0.11*	-0.22*	-0.17*
	(0.01)	(0.01)	(0.01)	(0.01)
Center city-suburban trend differential	-0.07*	-0.06*	-0.07*	-0.05*
	(0.01)	(0.01)	(0.01)	(0.01)
Center city trend	-0.23*	-0.17*	-0.28*	-0.22*
	(0.01)	(0.01)	(0.01)	(0.01)
Prenatal smoking		3.66*		3.66*
		(0.12)		(0.12)
Cigarettes per day, if smoked		0.21*		0.21*
		(0.01)		(0.01)
Cigarette price (in cents)			0.006*	0.005*
			(0.00)	(0.00)
N	3,519,837			

* $p<0.05$

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 Models also include controls for infant sex, marital status, maternal age, maternal education, parity, and maternal place of birth as well as an intercept term.

Table 2 - 4. Estimated 1990 to 2000 Change in the Center City-Suburban Gap in Low Birth Weight if Only Age-specific Population Shares, Fertility Rates, or Low Birth Weight Rates Changed

MSA	Actual	If only group-specific population shares changed	If only group-specific fertility rates changed	If only group-specific LBW rates changed
Combined	-1.05	-0.74	-1.34	-1.15
ATL	-0.56	0.64	-0.48	-0.59
BLT	-0.67	0.53	-2.17	-0.67
BTR	-0.76	-0.04	0.18	-0.83
BIR	0.32	0.26	1.87	0.44
BOS	-1.67	0.05	2.92	-1.64
CLT	-3.44	0.25	1.17	-3.78
CHI	-0.88	-0.07	-2.09	-0.93
CLE	-1.52	-0.17	-1.47	-1.56
CMH	2.60	0.25	-13.65	1.92
DAL	-0.98	0.16	-0.79	-1.04
DAY	-2.06	0.15	0.71	-2.42
DET	-0.34	-0.07	-2.02	-0.33
FLL	-1.81	0.81	-2.91	-1.68
FTW	-0.10	-0.15	-1.05	0.31
GWS	0.63	0.54	-1.02	1.43
HOU	-1.05	0.05	0.46	-0.86
JAC	-1.15	0.36	0.08	-1.33
JAX	3.10	0.25	0.69	3.22
KSC	-1.91	0.24	-1.72	-2.23
LAX	-2.16	0.10	-2.66	-2.18
MEM	-1.56	0.55	-0.56	-1.44
MIA	-2.48	0.34	-1.80	-2.64
MIL	2.58	0.12	-5.24	2.32
MOB	-4.78	0.26	0.33	-4.48
NSH	1.00	0.03	-0.41	0.52
NOL	-1.87	0.09	-0.49	-1.83
NWK	1.46	-0.04	0.11	1.21
NVB	-0.22	0.11	-0.73	-0.32
ORL	-2.14	-0.33	4.53	-2.03
PHL	-2.69	0.16	-2.38	-2.79
PIT	-0.36	-0.52	-1.09	-0.51
RDU	1.29	0.32	0.81	0.83
RIC	-0.55	0.54	-1.42	-0.06

Table continues on the next page. See notes at the end of the table.

Table 2 - 4, continued

MSA	Actual	If only group-specific population shares changed	If only group-specific fertility rates changed	If only group-specific LBW rates changed
STL	-0.77	0.19	-1.98	-0.73
SHV	1.79	0.62	-0.69	1.72
TPA	-0.64	-0.27	2.37	-0.54
WAS	-4.04	0.18	-3.78	-4.33

Note:

1 See Table 2A-1 for full MSA names.

Table 2 - 5. Estimated 1990 to 2000 Change in the Center City-Suburban Gap in Preterm Birth if Only Age-specific Population Shares, Fertility Rates, or Preterm Birth Rates Changed

MSA	Actual	If only group-specific population shares changed	If only group-specific fertility rates changed	If only group-specific preterm rates changed
Combined	-0.97	-0.87	-1.81	-1.145
ATL	-2.27	1.03	-0.99	-2.21
BLT	-3.96	0.93	-3.11	-3.90
BTR	-1.23	-0.27	0.35	-1.04
BIR	-0.24	0.22	3.18	-0.12
BOS	-1.40	0.08	3.83	-1.60
CLT	-1.27	0.62	1.92	-1.37
CHI	-1.40	-0.03	-2.76	-1.35
CLE	-0.01	-0.15	-1.90	-0.09
CMH	-0.90	-0.16	-12.93	-1.24
DAL	0.02	0.42	-0.66	0.10
DAY	-2.27	0.02	0.89	-2.44
DET	1.20	-0.24	-2.63	1.09
FLL	-1.59	1.08	-4.40	-1.67
FTW	0.64	-0.39	-1.14	0.75
GWS	0.00	1.12	-0.61	0.37
HOU	-1.68	0.09	0.74	-1.91
JAC	-3.82	0.44	0.30	-4.07
JAX	1.82	0.45	0.58	1.88
KSC	-2.80	0.19	-1.81	-2.84
LAX	-2.47	0.13	-3.55	-2.40
MEM	-0.79	1.15	-0.56	-0.71
MIA	-2.24	0.57	-3.07	-2.17
MIL	6.03	-0.22	-5.47	4.88
MOB	-3.63	0.38	0.43	-3.47
NSH	2.54	0.04	-1.20	2.54
NOL	-1.01	0.20	-0.85	-1.00
NWK	1.26	0.07	0.79	0.97
NVB	-2.85	0.16	-1.38	-2.85
ORL	1.21	-0.54	6.66	1.34
PHL	-3.54	0.42	-3.28	-3.77
PIT	-0.78	-0.57	-1.14	-0.90
RDU	0.97	0.57	1.67	0.73
RIC	-2.07	0.68	-2.17	-1.68

Table continues on the next page. See notes at the end of the table.

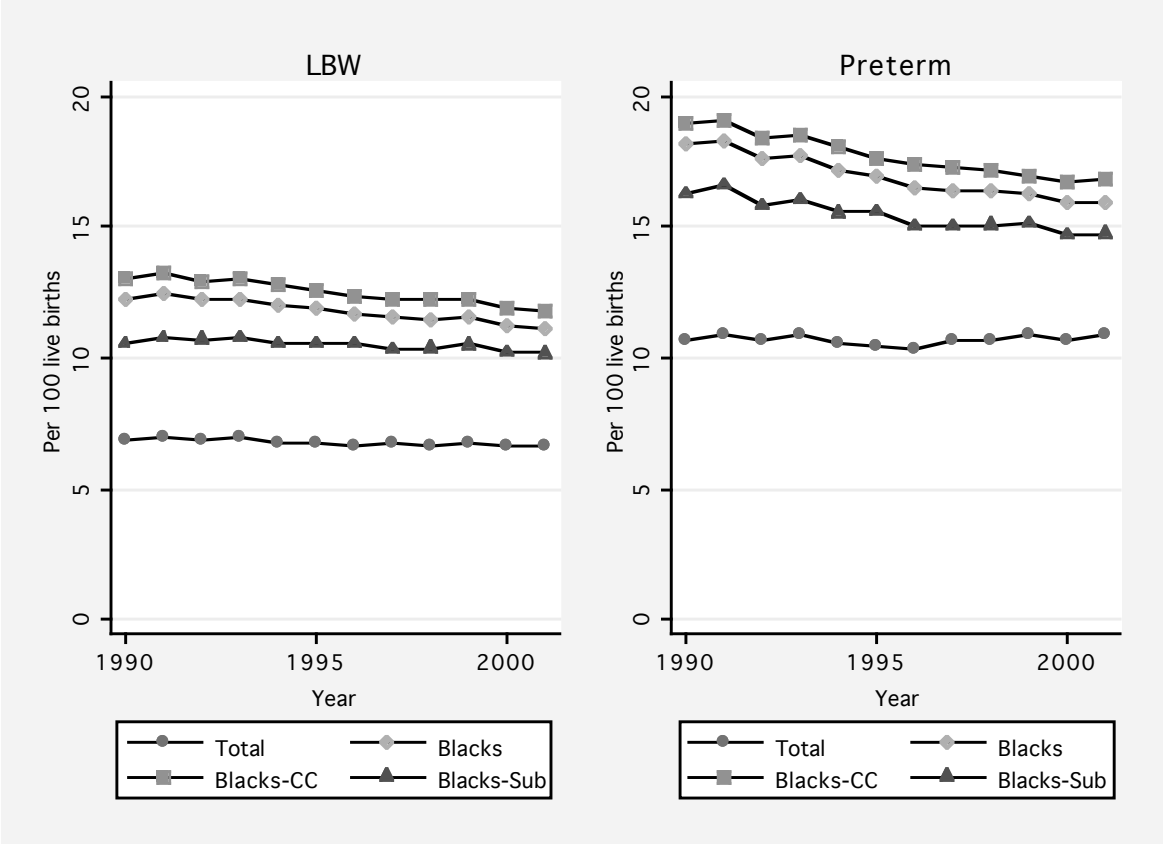
Table 2 - 5, continued

MSA	Actual	If only group-specific population shares changed	If only group-specific fertility rates changed	If only group-specific preterm rates changed
STL	-1.01	0.51	-2.96	-1.09
SHV	1.07	0.99	-1.20	1.02
TPA	2.99	-0.29	3.51	3.12
WAS	-4.21	0.42	-5.42	-4.16

Note:

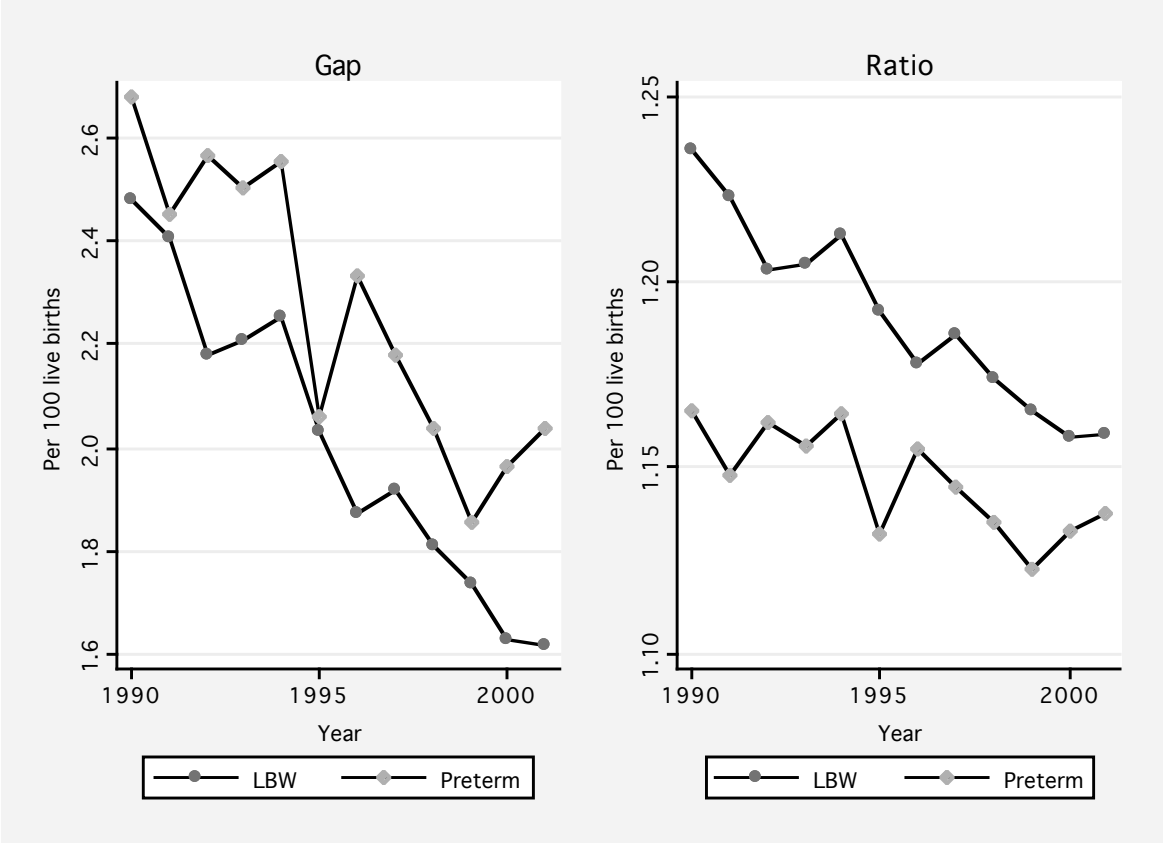
1 See Table 2A-1 for full MSA names.

Figure 2 - 1. Birth Outcomes Among Blacks in 37 Metropolitan Areas, 1990-2001



Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births to women ages 15-39.

Figure 2 - 2. Center City-Suburban Birth Outcome Gap and Ratio Among Blacks in 37 Metropolitan Areas, 1990-2001



Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births to women ages 15-39.

Appendix 2A: Supplemental Tables and Figures

Table 2A - 1. List of 37 Metropolitan Areas Included in the Primary Analysis Sample

Population* rank in 1990 Census	Population* rank in 2000 Census	Abbreviation	Name
2	2	CHI	Chicago, IL PMSA
3	3	WAS	Washington, DC--MD--VA--WV PMSA
4	6	DET	Detroit, MI PMSA
6	5	PHL	Philadelphia, PA--NJ PMSA
7	4	ATL	Atlanta, GA MSA
8	9	BLT	Baltimore, MD PMSA
9	8	HOU	Houston, TX PMSA
10	11	NOL	New Orleans, LA MSA
11	14	STL	St. Louis, MO--IL MSA
12	15	NWK	Newark, NJ PMSA
13	10	DAL	Dallas, TX PMSA
14	12	MEM	Memphis, TN--AR--MS MSA
15	13	NVB	Norfolk--Virginia Beach--Newport News, VA--NC MSA
16	16	MIA	Miami, FL PMSA
17	17	CLE	Cleveland--Lorain--Elyria, OH PMSA
19	21	RIC	Richmond--Petersburg, VA MSA
20	22	BIR	Birmingham, AL MSA
21	20	CLT	Charlotte--Gastonia--Rock Hill, NC--SC MSA
22	31	KSC	Kansas City, MO--KS MSA
23	27	BOS	Boston, MA--NH PMSA
24	29	MIL	Milwaukee--Waukesha, WI PMSA
25	34	CIN	Cincinnati, OH--KY--IN PMSA
26	18	FLL	Fort Lauderdale, FL PMSA
28	23	RDU	Raleigh--Durham--Chapel Hill, NC MSA
29	25	GWS	Greensboro--Winston-Salem--High Point, NC MSA
30	26	TPA	Tampa--St. Petersburg--Clearwater, FL MSA
31	28	JAX	Jacksonville, FL MSA
34	36	JAC	Jackson, MS MSA
35	37	PIT	Pittsburgh, PA MSA
36	35	CMH	Columbus, OH MSA
37	40	BTR	Baton Rouge, LA MSA
39	38	NSH	Nashville, TN MSA

Table continues on the next page. See notes at the end of the table.

Population* rank in 1990 Census	Population* rank in 2000 Census	Abbreviation	Name
41	39	FTW	Fort Worth--Arlington, TX PMSA
43	48	MOB	Mobile, AL MSA
44	32	ORL	Orlando, FL MSA
45	51	DAY	Dayton--Springfield, OH MSA
49	49	SHV	Shreveport--Bossier City, LA MSA

* Non-Hispanic Black population

Table 2A - 2. Selected Regression Coefficients From Linear Probability Models Without Fixed Effects Explaining Adverse Birth Outcomes Among Black Women in 37 Metropolitan Areas, 1990-2001

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
1	FEMALE	-0.05*	-0.07*	-0.12*	-0.16*	-0.07*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
2	FEMALE, MARRIED	-0.05*	-0.08*	-0.13*	-0.16*	-0.07*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
3	FEMALE, AGE	-0.06*	-0.06*	-0.13*	-0.17*	-0.06*	-0.23*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
4	FEMALE, MARRIED, AGE	-0.08*	-0.06*	-0.14*	-0.18*	-0.06*	-0.24*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
5	FEMALE, MARRIED, AGE, EDUC	-0.06*	-0.07*	-0.12*	-0.16*	-0.06*	-0.22*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
6	FEMALE, MARRIED, AGE, EDUC, PARITY	-0.05*	-0.07*	-0.13*	-0.16*	-0.06*	-0.22*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)

Table continues on the next page. See notes at the end of the table.

Table 2A - 2, continued

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
7	6 + TOBACCO, TOBXCIGNUM	0.00	-0.05*	-0.05*	-0.13*	-0.05*	-0.18*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
8	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	0.02*	-0.05*	-0.03*	-0.12*	-0.05*	-0.17*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
9	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	0.01	-0.07*	-0.06*	-0.17*	-0.09*	-0.26*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	0.01	-0.07*	-0.06*	-0.16*	-0.10*	-0.26*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
11	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV, TERM	0.06*	-0.04*	0.02*			
		(0.01)	(0.01)	(0.01)			

Table continues on the next page. See notes at the end of the table.

Table 2A - 2, continued

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
12	11 + MRF(16)	0.02*	-0.04*	-0.01*	-0.21*	-0.10*	-0.31*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
13	11 + MRF(16), CLB(16)	0.03*	-0.04*	-0.01	-0.20*	-0.10*	-0.30*
		(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
	Approximate N		3,500,000			3,500,000	

* p<0.05

Notes:

1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 2-1 for variable definitions and categories.

3 Models do not include controls for MSA-level fixed effects.

Table 2A - 3. Selected Regression Coefficients From Logit Models Explaining Adverse Birth Outcomes Among Black Women in 37 Metropolitan Areas, 1990-2001

		Outcome = LBW			Outcome = PRETERM		
Model	**Covariates**	***β*** **=** *Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** **=** *Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
1	FEMALE	-0.49* [0.995*] (0.08 [0.001])	-0.60* [0.994*] (0.10 [0.001])	-1.08* [0.989*] (0.06 [0.001])	-1.15* [0.99*] (0.07 [0.001])	-0.44* [1.00*] (0.09 [0.001])	-1.58* [0.98*] (0.05 [0.000])
2	FEMALE, MARRIED	-0.52* [0.995*] (0.08 [0.001])	-0.62* [0.994*] (0.10 [0.001])	-1.14* [0.989*] (0.06 [0.001])	-1.18* [0.99*] (0.07 [0.001])	-0.46* [1.00*] (0.09 [0.001])	-1.63* [0.98*] (0.05 [0.000])
3	FEMALE, AGE	-0.65* [0.993*] (0.08 [0.001])	-0.49* [0.995*] (0.10 [0.001])	-1.15* [0.989*] (0.06 [0.001])	-1.24* [0.99*] (0.07 [0.001])	-0.37* [1.00*] (0.09 [0.001])	-1.61* [0.98*] (0.05 [0.000])
4	FEMALE, MARRIED, AGE	-0.77* [0.992*] (0.08 [0.001])	-0.50* [0.995*] (0.10 [0.001])	-1.27* [0.987*] (0.06 [0.001])	-1.35* [0.99*] (0.07 [0.001])	-0.37* [1.00*] (0.09 [0.001])	-1.71* [0.98*] (0.05 [0.000])
5	FEMALE, MARRIED, AGE, EDUC	-0.56* [0.994*] (0.08 [0.001])	-0.57* [0.994*] (0.10 [0.001])	-1.13* [0.989*] (0.06 [0.001])	-1.15* [0.99*] (0.07 [0.001])	-0.42* [1.00*] (0.09 [0.001])	-1.57* [0.98*] (0.05 [0.000])

Table continues on the next page. See notes at the end of the table.

Table 2A - 3, continued

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
6	FEMALE, MARRIED, AGE, EDUC, PARITY	-0.54* [0.995*] (0.08 [0.001])	-0.62* [0.994*] (0.10 [0.001])	-1.16* [0.988*] (0.06 [0.001])	-1.15* [0.99*] (0.07 [0.001])	-0.40* [1.00*] (0.09 [0.001])	-1.55* [0.98*] (0.05 [0.000])
7	6 + TOBACCO, TOBXCIGNUM	0.08 [1.001] (0.08 [0.001])	-0.46* [0.995*] (0.10 [0.001])	-0.37* [0.996*] (0.06 [0.001])	-0.87* [0.99*] (0.07 [0.001])	-0.32* [1.00*] (0.09 [0.001])	-1.18* [0.99*] (0.05 [0.001])
8	6 + TOBACCO, TOBXCIGNUM, ALCOHOL	0.22* [1.002*] (0.08 [0.001])	-0.43* [0.996*] (0.10 [0.001])	-0.21* [0.998*] (0.06 [0.001])	-0.79* [0.99*] (0.07 [0.001])	-0.30* [1.00*] (0.09 [0.001])	-1.10* [0.99*] (0.05 [0.001])
9	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC	0.10 [1.001] (0.09 [0.001])	-0.66* [0.993*] (0.10 [0.001])	-0.56* [0.994*] (0.06 [0.001])	-1.25* [0.99*] (0.08 [0.001])	-0.66* [0.99*] (0.09 [0.001])	-1.92* [0.98*] (0.05 [0.001])

Table continues on the next page. See notes at the end of the table.

Table 2A - 3, continued

Model	Covariates	Outcome = LBW			Outcome = PRETERM		
		β *= Sub Trend (1a)*	*δ = CC/Sub trend differential (2a)*	*β + δ = CC Trend (3a)*	***β*** *= Sub Trend (1b)*	*δ = CC/Sub trend differential (2b)*	*β + δ = CC Trend (3b)*
10	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV	0.08 [1.001]	-0.67* [0.993*]	-0.59* [0.994*]	-1.27* [0.99*]	-0.67* [0.99*]	-1.94* [0.98*]
		(0.09 [0.001])	(0.10 [0.001])	(0.06 [0.001])	(0.08 [0.001])	(0.09 [0.001])	(0.05 [0.001])
11	6 + TOBACCO, TOBXCIGNUM, ALCOHOL, PNC, PREV, TERM	0.61* [1.006*]	-0.36* [0.996*]	0.25* [1.002*]			
		(0.10 [0.001])	(0.12 [0.001])	(0.07 [0.001])			
12	11 + MRF(16)	0.15 [1.002]	-0.35* [0.997*]	-0.20* [0.998*]	-1.70* [0.98*]	-0.64* [0.99*]	-2.35* [0.98*]
		(0.10 [0.001])	(0.12 [0.001])	(0.07 [0.001])	(0.08 [0.001])	(0.09 [0.001])	(0.05 [0.001])
13	11 + MRF(16), CLB(16)	0.25* [1.002*]	-0.33* [0.997*]	-0.08 [0.999*]	-1.68* [0.98*]	-0.62* [0.99*]	-2.30* [0.98*]
		(0.10 [0.001])	(0.12 [0.001])	(0.07 [0.001])	(0.08 [0.001])	(0.09 [0.001])	(0.05 [0.001])
	Approximate N		3,500,000			3,500,000	

See notes on the next page.

* $p<0.05$

Notes:

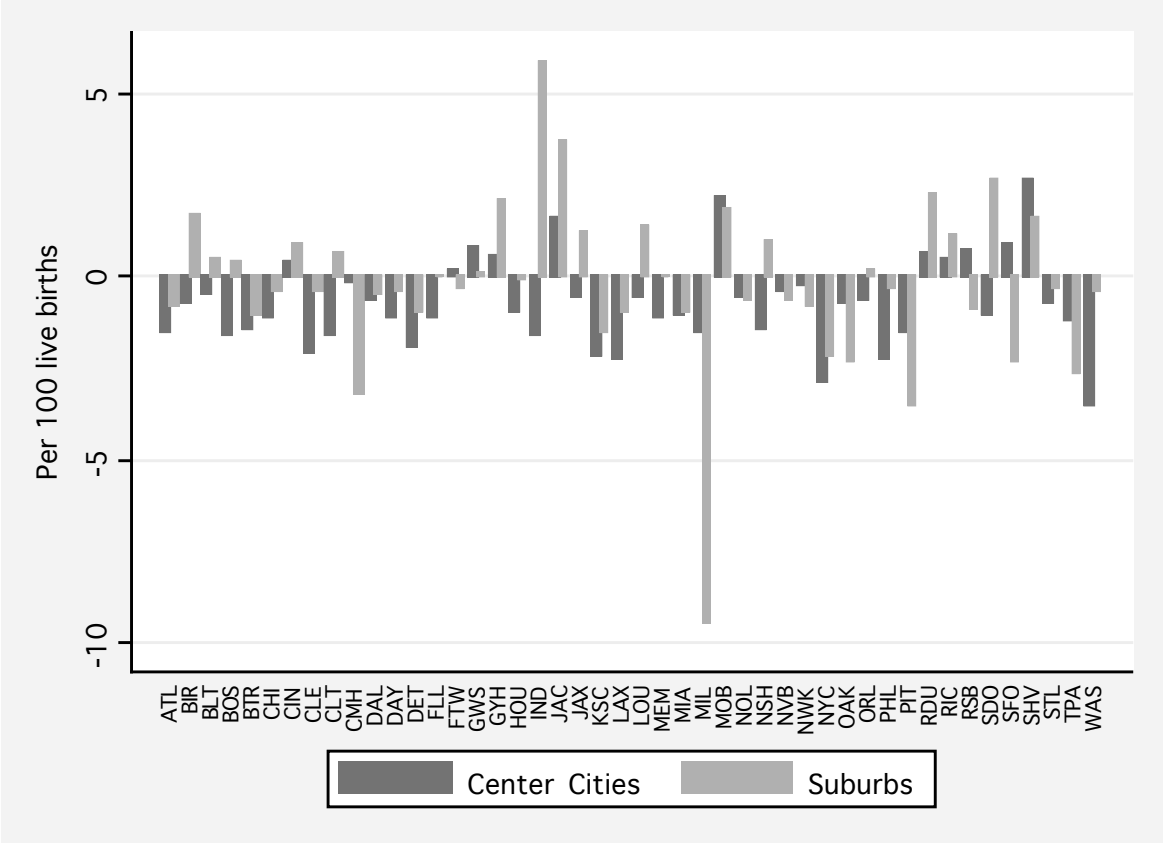
1 Robust standard errors in parentheses. Coefficients are inflated by 100.

2 See Table 2-1 for variable definitions and categories.

3 All models include controls for MSA-level fixed effects.

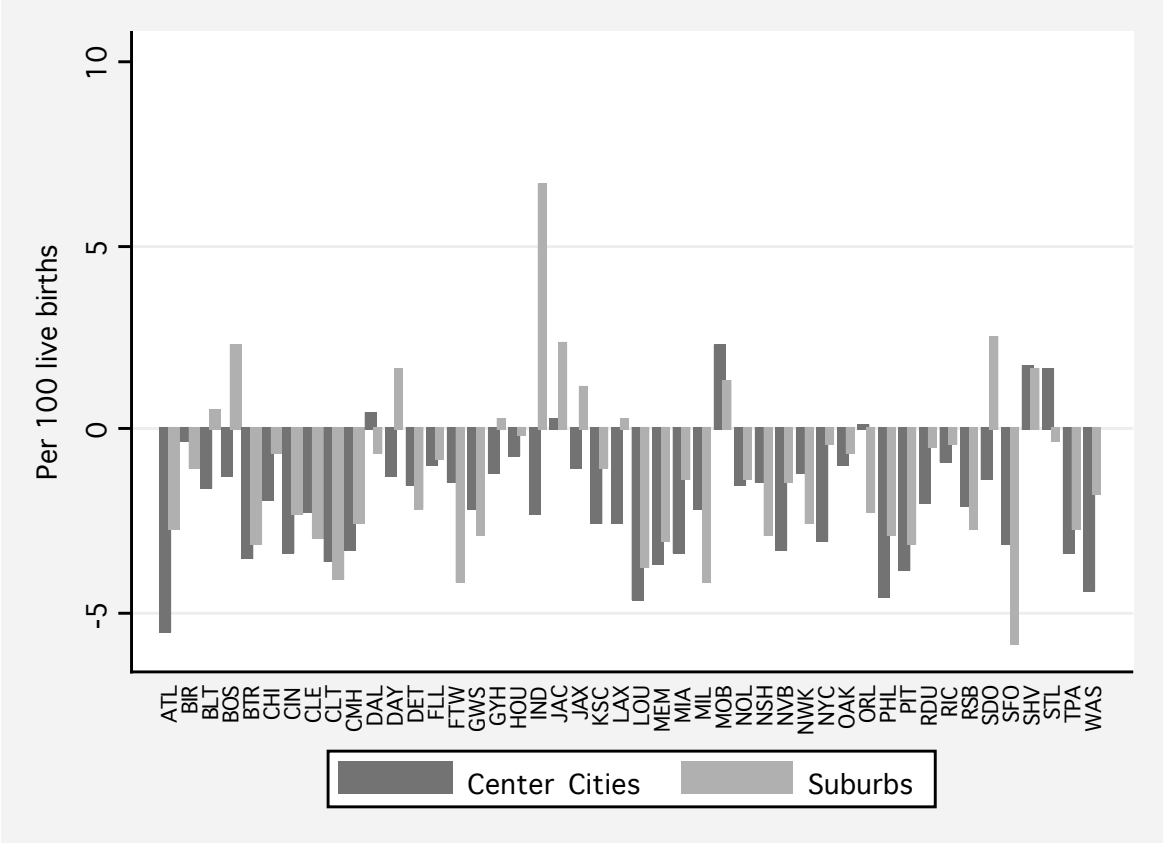
4 Odds ratios (and robust SEs) in brackets.

Figure 2A - 1. 1990 to 2001 Change in Low Birth Weight Among Blacks in 37 Metropolitan Areas by Center City/Suburban Status



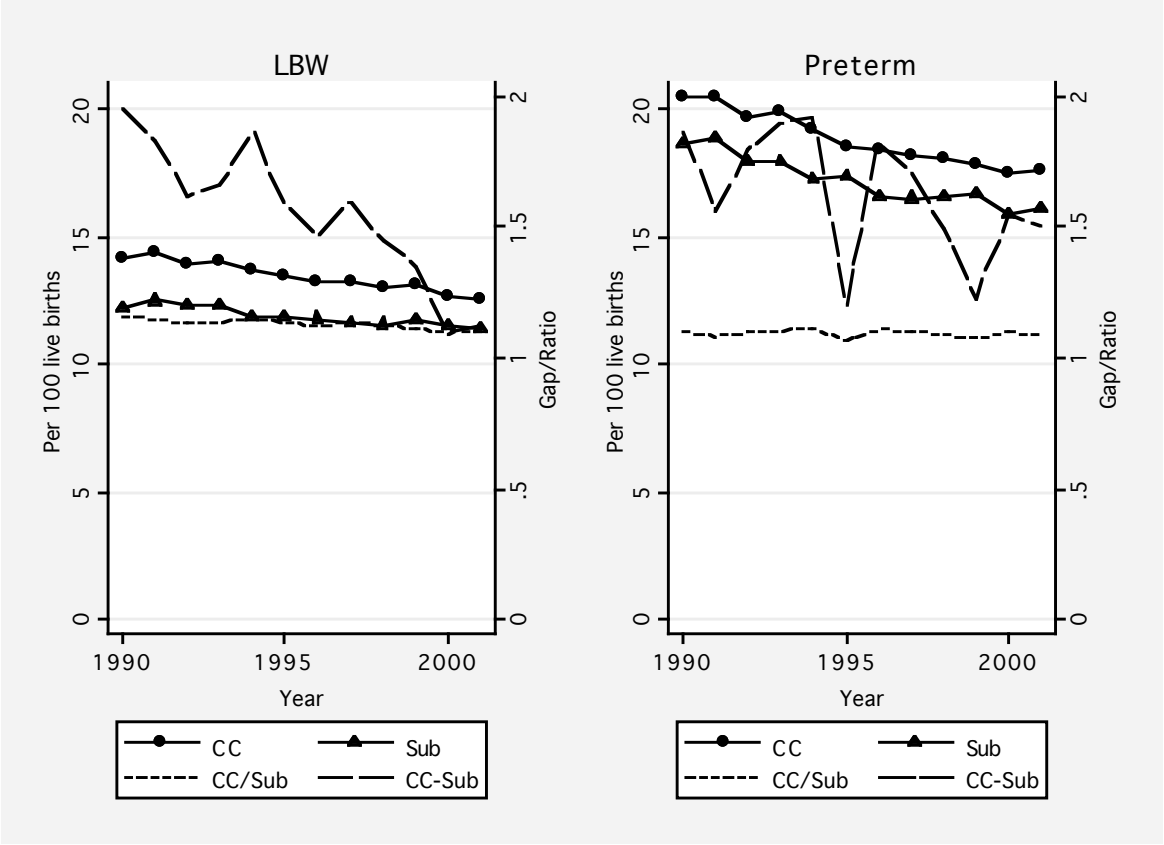
Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Notes: Singleton births to women ages 15-39. See Table 2A-1 for full MSA names.

Figure 2A - 2. 1990 to 2001 Change in Preterm Birth Among Blacks in 37 Metropolitan Areas by Center City/Suburban Status



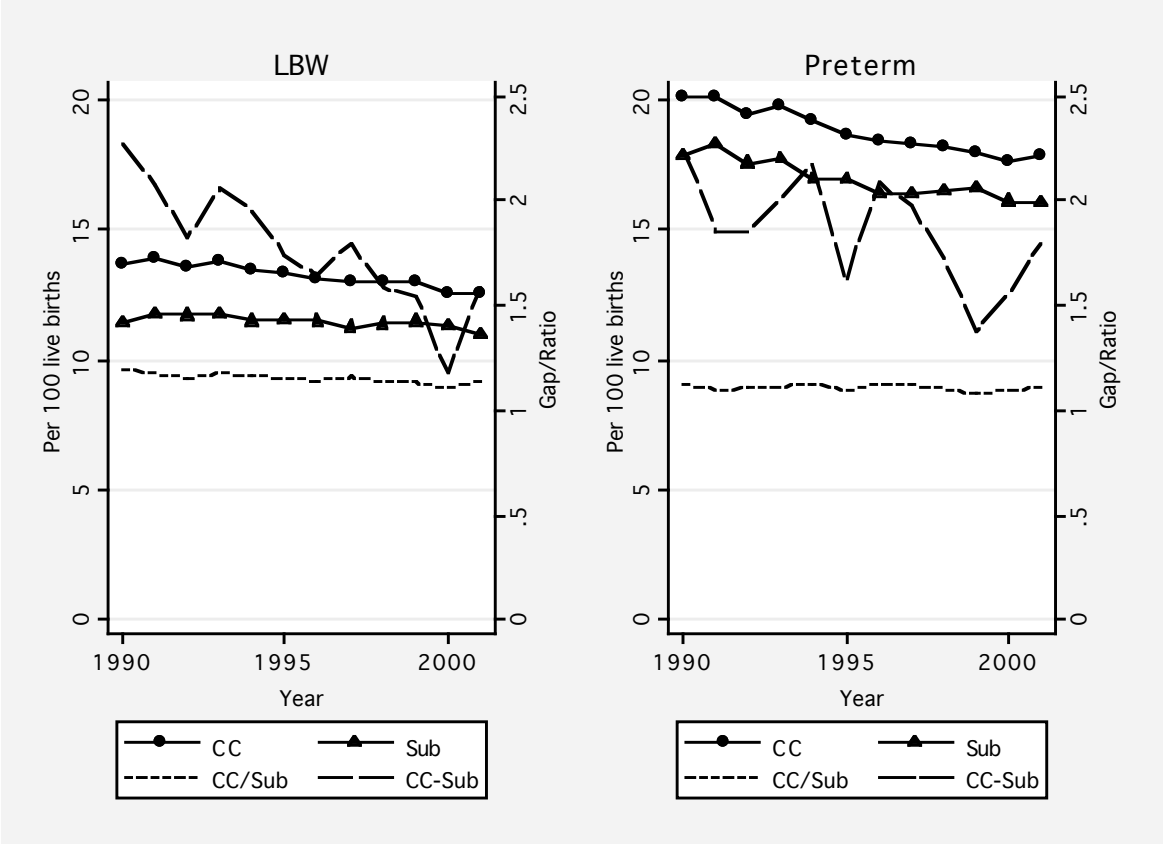
Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Notes: Singleton births to women ages 15-39. See Table 2A-1 for full MSA names.

Figure 2A - 3. Center Cities vs. Suburbs: Birth Outcomes Among Black, Unmarried Women in 37 Metropolitan Areas, 1990-2001



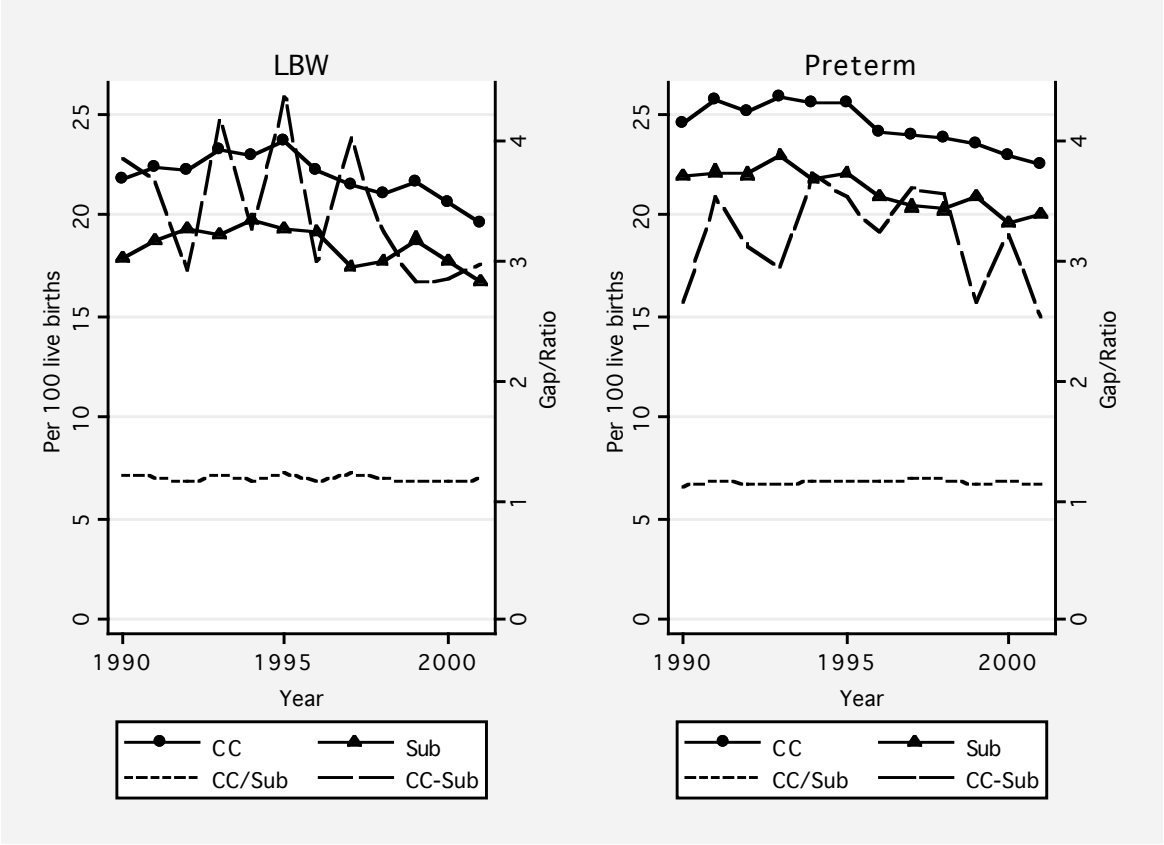
Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births to women ages 15-39.

Figure 2A - 4. Center Cities vs. Suburbs: Birth Outcomes Among Black Women with Less Than a College Education in 37 Metropolitan Areas, 1990-2001



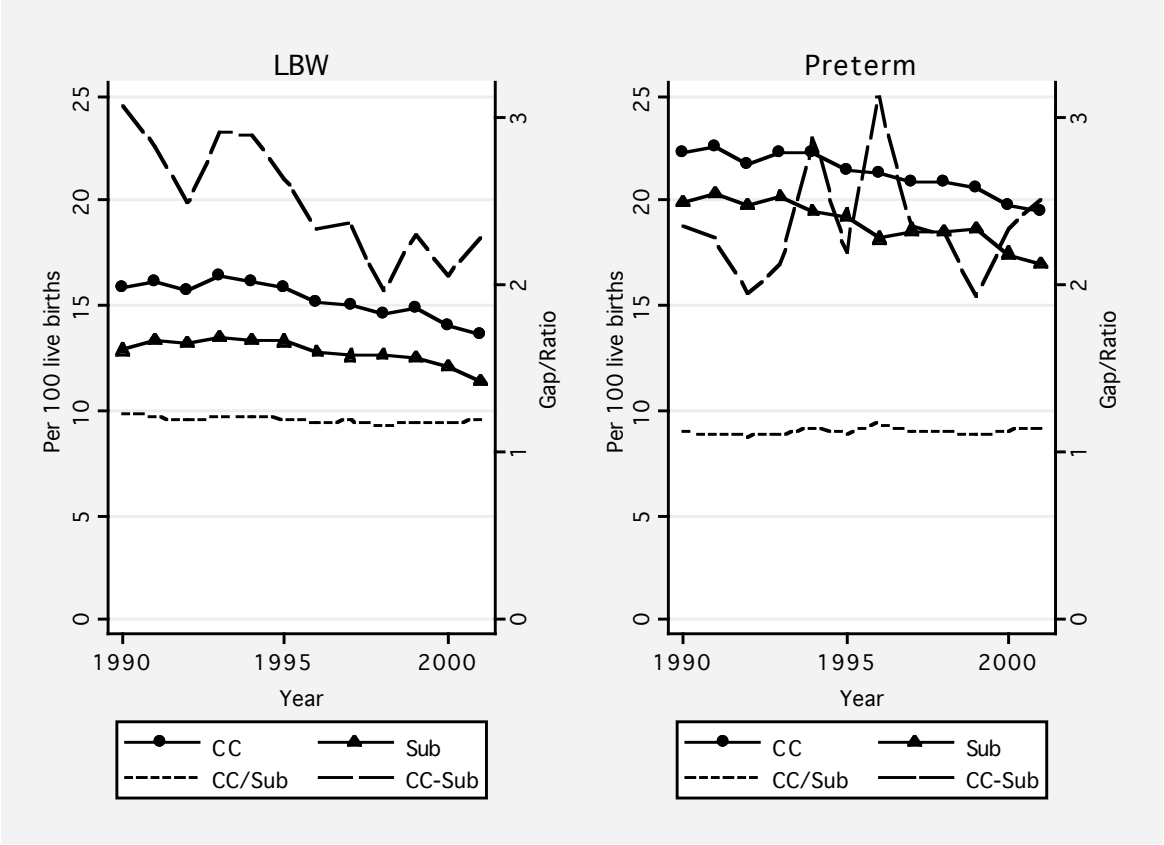
Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births to women ages 15-39.

Figure 2A - 5. Center Cities vs. Suburbs: Birth Outcomes Among Black Women Who Smoked During Pregnancy in 37 Metropolitan Areas, 1990-2001



Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births to women ages 15-39.

Figure 2A - 6. Center Cities vs. Suburbs: Birth Outcomes Among Black Women Who Received Inadequate Prenatal Care in 37 Metropolitan Areas, 1990-2001



Source: Authors' tabulations of 1990-2001 NCHS Natality Files.
Note: Singleton births to women ages 15-39.

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Chapter 3 Expanding Medicaid May Cost Less Than You Think: Evidence from Health Insurance Churning in the 1996-2003 MEPS

Introduction

In 2001, over 46 million people received health care services financed through Medicaid (Centers for Medicare and Medicaid Services 2004). Still, many low-income individuals lack health insurance coverage. In a survey of the healthcare safety net system in 30 states, the Agency for Healthcare Research and Quality found that between 19 and 42 percent of Americans with incomes below 200 percent of the federal poverty level are uninsured (Billings and Weinick 2003). Due to state variations in income eligibility thresholds, many uninsured individuals with low income are not eligible for Medicaid (Glied and Gould 2005). Others are eligible but not enrolled (Remler and Glied 2003). In most states, enrollment for eligible individuals is administratively cumbersome.

Maintaining coverage presents additional difficulties. Medicaid enrollees must fulfill regular recertification requirements that may be both burdensome and confusing, especially for poorly educated and/or non-English speaking applicants. In the face of fiscal crises, many states have sought to reduce Medicaid costs by cutting their Medicaid rolls via increasing the frequency of required recertification for those already enrolled (Ross and Ku 2002). In a study conducted by the Health Policy Analysis Program at the University of Washington, researchers found that most people who failed to renew eligibility did so because of procedural barriers rather than substantive ineligibility (Gardner, Lew et al. 2004). Two states currently require recertification every three months or less, eleven states require recertification every four to eleven months, and the rest require recertification only annually or if financial circumstances change (Ross and

Ku 2002).[50] Even among those who require annual recertification, involuntary disenrollment can be high, and more frequent recertification only exacerbates the problem (Lipson, Fishman et al. 2003).

This complexity contributes to widespread instability of insurance coverage among those eligible for Medicaid. Thirty percent of low-income individuals covered by Medicaid in January of 1998 or 1999 experienced at least one spell of uninsurance in the subsequent two years (Klein, Glied et al. 2005). Moreover, those who transfer into and out of Medicaid coverage represent a large portion of the uninsured (Lipson, Fishman et al. 2003).

Several studies have found that lack of and instability in health coverage are associated with unmet health needs and lower health care utilization. Medicaid-eligible adults are more likely than Medicaid enrollees to report unmet need due to cost (Davidoff, Garrett et al. 2001). Therefore, the gap between eligibility and enrollment has prompted some policy analysts to suggest that it would be desirable to simplify Medicaid's enrollment and recertification procedures. Evidence from states that have simpler procedures suggests that such administrative changes would in fact lead to increased enrollment in the program. A broader, related proposal would extend Medicaid eligibility to anyone without health coverage.

Cost is obviously a concern, however. Greater enrollment implies greater total costs to state and federal governments. In 2003, Medicaid accounted for about 15 percent

[50] As a check on misreporting of insurance coverage, we looked for people with three or fewer consecutive months of Medicaid coverage. Since most states require recertification with a frequency of at least four months, a large share of individuals with very short spells of Medicaid coverage would be indicative of misreporting. Among individuals that had Medicaid in at least once month on the panel, 3, 4, and 5 percent experienced Medicaid spells of 1, 2, or 3 months, respectively.

of state general fund spending, and Medicaid expenditures grew at a significantly faster rate than total state spending (Holahan, Weiner et al. 2003). High per enrollee costs of current Medicaid beneficiaries relative to per enrollee costs in other health plans may suggest that raising coverage through either increased eligibility or simplification of enrollment and recertification procedures would be less cost-efficient than alternative strategies. Average monthly expenditures of adult Medicaid beneficiaries are two and a half times greater than those of privately insured adults, for example.

To the extent that those enrolled in Medicaid are less healthy than those who remain uninsured, however, these costs may be overstated. Gaining and maintaining Medicaid benefits are likely to be more of a priority for individuals who expect to need care in the future (or, in the case of retroactive eligibility, have needed care in the recent past) than for those in good health. Thus, complex enrollment and recertification requirements may represent key barriers to Medicaid coverage among those with fewer health needs. If so, simplifying administrative procedures could increase coverage, giving low-income individuals stable access to preventive and chronic illness care – at lower per enrollee cost than anticipated.

We examine this theory by analyzing health care expenditure and utilization patterns using a large national sample of adults drawn from the Medical Expenditure Survey (MEPS), who were followed for two full years between 1996 and 2003. We exploit the MEPS's monthly survey design to examine the costs associated with *maintaining* Medicaid coverage for those eligible adults who currently allow their coverage to expire and increasing Medicaid take-up among the currently uninsured who are already eligible or who might be newly eligible under a Medicaid expansion. We use

month-to-month health insurance transitions to identify and characterize the expenditure and utilization profiles of populations with unstable Medicaid coverage and compare them with the expenditure and utilization patterns of those with stable Medicaid coverage and the persistently uninsured. The richness of MEPS data allow us to adjust mean health expenditures for individual covariates other than insurance status that may also impact spending patterns.

Prior Research

Several studies support our hypothesis that Medicaid coverage is associated with elevated health care needs. Holahan, examining self-reported health status in the 1996 Medical Expenditure Panel Survey (MEPS), found that Medicaid beneficiaries were, on average, less healthy than uninsured or privately insured individuals (Holahan 2001). He concluded that current Medicaid enrollees are more expensive than average to insure, and that policymakers should not rely solely on their per enrollee costs when considering proposals to expand Medicaid to the uninsured. Similarly, a recent evaluation of the cost of enrolling eligible but uninsured adults in New York's Medicaid program estimated that the cost of covering an uninsured, Medicaid-eligible adult in New York would be only about 60 percent that of covering the average non-disabled, non-elderly adult currently enrolled (Birnbaum, Haslanger et al. 2004). Another study not only found evidence of adverse selection into Medicaid, but also showed that after controlling for insurance selection, access to and use of health care among mothers on Medicaid was comparable to those of low income privately insured mothers (Long, Coughlin et al. 2005). The analysis presented here extends upon these prior studies, analyzing a nationally representative sample of individuals spanning eight years.

Data and Methods

Data for these analyses are drawn from Panels 1-7 of the MEPS, a comprehensive national probability survey exploring the utilization and financing of medical care in the US. The MEPS is ideal for studying health insurance transitions because it includes monthly data pertaining to individuals' health status, medical conditions, health expenditures, and utilization of medical services spanning two years, as well as demographic information for each respondent.

All analyses pertain to adults. Since we wish to eliminate health insurance transitions resulting from aging into Medicare, respondents in our sample were between the ages of 19 and 62 in their first year on the panel. We require that individuals in our sample remained in the study for the full two years and that information on health insurance status is complete in all 24 months. Thus, our 55,509 respondents participated in the survey over the years 1996-1997 (Panel 1), 1997-1998 (Panel 2), 1998-1999 (Panel 3), 1999-2000 (Panel 4), 2000-2001 (Panel 5), 2001-2002 (Panel 6), or 2002-2003 (Panel 7).

Respondents may report multiple sources of health insurance each month. Therefore, we assign a primary source of health coverage according to an algorithm that ranks coverage types, in descending order of importance, as follows: Medicaid, private group, "other" private, Medicare, "other" public. Individuals with none of these types of insurance are deemed uninsured. Table 3A-1 in the Appendix shows the average monthly distribution of primary insurance coverage as well as the share of people in each category who also reported other types of insurance. Twenty percent of individuals are uninsured in a given month, on average, and 6 percent have Medicaid.

Our primary analysis compares expenditures among groups with varying health insurance coverage in the first or second months prior to and after transition.[51] In the latter case, we require that individuals have constant coverage over those two months. In one set of sensitivity analyses, we require constant coverage over the four months prior to and after transition. These restrictions reduce sample size, but eliminate, for example, many people who repeatedly cycle on and off of Medicaid or experience a single month off of Medicaid. The health insurance transitions of these individuals are less likely to reflect changing health needs than simply disorganization on the part of the individual or Medicaid office. Table 3A-2 in the Appendix shows sample sizes for each type of transition, given the one, two, or four month restrictions. Though we show expenditures for individuals who transitioned from Medicaid to private group insurance or private group insurance to Medicaid, we caution that these estimates are less stable than others because sample sizes are often small.

We employ monthly data on expenditures for and numbers of office-based medical visits, emergency room visits, inpatient hospital discharges, outpatient hospital visits, and prescription drugs. Total health care expenditures include expenditures resulting from these five types of health care. Physician expenditures pertain to both office-based and inpatient physician services. Physician visits are defined similarly. Hospital expenditures are the sum of emergency room, inpatient, and outpatient expenditures. All expenditures are adjusted for inflation, using January 1996 as the reference period.

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[51] We look only at the months prior to and after the transition. Months in which the transition took place are excluded from our analyses.

Study Design

Our first research question considers the per-enrollee cost of increasing Medicaid coverage – by raising take-up or expanding eligibility to the uninsured – relative to the per-enrollee cost of current Medicaid recipients. In this case, we compare the average monthly health expenditures of: 1) individuals who were without insurance for two full years and 2) those who transitioned from uninsurance to Medicaid, in the first or second months prior to transition. If the decision to enroll in Medicaid is correlated with health needs, then we expect the pre-transition expenditures of those who switched from uninsurance to Medicaid (i.e., spending while still uninsured) to exceed those of individuals who were persistently uninsured. We also examine the expenditures of the stable Medicaid population – those who were uninsured for the full two years – but note that their expenditures are not strictly comparable to those of the persistently uninsured since the latter pay for all health care costs out of pocket. For the same reason, comparison of pre- and post-transition expenditures is not straightforward. We further explore the health care needs of these populations by substituting for expenditures the share of individuals with physician visits (office-based and inpatient), hospital admissions, emergency room visits, or prescription drugs.

The second question explores the cost of simplifying Medicaid recertification procedures so as to eliminate temporary or permanent transitions off of Medicaid among those who remain eligible. Here we compare the average monthly expenditures of: 1) individuals enrolled in Medicaid for 24 months; and 2) those who transitioned off of Medicaid to either no coverage or private group insurance, in the first or second month prior to transition. In this case, we expect the expenditures of those persistently on

Medicaid to exceed the pre-transition expenditures of individuals that switched from Medicaid to either no coverage or private group coverage, on the theory that the latter allowed their Medicaid coverage to lapse due to more limited health needs.

We also consider the effect of crowd-out. Medicaid expansions and simpler enrollment procedures are likely to induce some individuals with private group insurance to drop that coverage and enroll in Medicaid. This will be especially likely among those with disadvantageous cost-sharing health plans and elevated health care needs. Therefore we also compare the average monthly expenditures of: 1) individuals enrolled in private group insurance for the full panel period; and 2) those who transitioned off of private group insurance to Medicaid, in the first or second month prior to transition.

We report both "unadjusted" and "adjusted" mean expenditures. Unadjusted mean expenditures are simply arithmetic means of expenditures within a particular subpopulation. Adjusted mean expenditures are computed in a regression framework, in which we predict mean expenditures while controlling for age, sex, race/ethnicity, educational attainment, poverty status, health status, and geographic region of residence. We summarize these characteristics in Table 3A-3 in the Appendix. This analysis allows us to ascertain whether variations in observable characteristics account for differences in expenditure patterns between those who enrolled in Medicaid and those who remained uninsured (or those who left Medicaid and those who maintained coverage), or whether these differences are due to characteristics that cannot be readily observed by policymakers or analysts. It also helps mitigate possible bias resulting from the fact that we cannot distinguish between individuals that take up or leave Medicaid because of

changes in eligibility and those that transition on or off of Medicaid because of changes in health needs.

We predict adjusted mean expenditures using a two-part model that estimates, first, the probability of having positive expenditures, and second, mean expenditures among those with positive expenditures. We estimate part one using a logit specification and part two using a Generalized Linear Model (GLM) with Gamma distribution and log link. Bias and precision are sensitive to choice of estimator in the presence of skewed outcomes, as in the case of mean positive expenditures (Manning and Mullahy 2001). Application of Manning's and Mullahy's (2001) algorithm for identifying an optimal estimator in the presence of skewed outcomes to our data suggests the use of a GLM Gamma regression in part two (Manning and Mullahy 2001).

Standard errors of both unadjusted and adjusted mean expenditures are adjusted for the single-stage random sampling design of the MEPS as well as the possibility of an individual having multiple transitions using the survey commands of Stata 9.2 and/or the robust standard errors with clustering option of Stata's regression commands (StataCorp 2005). All tables report the results of t-tests performed on each comparison.

Results

Medicaid enrollees vs. the uninsured

Table 3-1 shows mean total expenditures for individuals with stable Medicaid coverage, the persistently uninsured, and those who switched from no coverage to Medicaid. Uninsured individuals who switched to Medicaid had substantially and statistically significantly higher health expenditures than those who did not transition out of uninsurance. This is true even when we adjust for demographic characteristics and

health status. The average adjusted expenditures of those uninsured individuals who subsequently transitioned to Medicaid were more than nine times those of the persistently uninsured in the month prior to enrollment in Medicaid and six times greater in the second month prior to transition. Also, individuals who were persistently uninsured had lower monthly total health care expenditures – both unadjusted and adjusted – than did those who were on Medicaid for the full panel period. The average adjusted monthly healthcare expenditures of those who were always uninsured were just 6 percent as high as those of long-term Medicaid enrollees. However, some of this difference may be due to the fact that people without insurance coverage generally bear the full cost of health care services received. As such, the difference, though statistically significant at the 5 percent level, does not necessarily indicate greater health needs among those with long-term Medicaid coverage.

Table 3-2 shows that these patterns of greater expenditures among the stable Medicaid population and the uninsured who switched to Medicaid persist when we disaggregate total spending into physician, hospital, and prescription drug expenditures. The long-term uninsured spent about 40 percent as much on physician visits, 16 percent as much on hospital visits, and 25 percent as much on prescriptions as did those who subsequently switched to Medicaid. When we adjust for demographic characteristics and health status, these differences widen. Moreover, all of the one month pre-transition versus persistently uninsured comparisons yield statistically significant differences.

Not surprisingly, health care utilization patterns mirror expenditure patterns (Table 3-3). While 20 percent of uninsured individuals who subsequently enrolled in Medicaid saw a doctor in the month prior to enrollment, only 7 percent of individuals

who remained uninsured did so in any single month. In the month prior to switching to Medicaid, uninsured individuals were 2.7 times more likely to see a physician, 7 times more likely to be admitted to a hospital, and 2.9 times more likely to fill a prescription than those who remained uninsured for two years. Those who switched to Medicaid were also more likely to visit the emergency room in their last month without health coverage than individuals who were persistently uninsured (4 percent vs. 1 percent). All of these comparisons as well as comparisons between those always uninsured and those always on Medicaid were statistically significant at the 5 percent level.

These findings also hold among individuals with greater than average health care needs. Repeating the analysis in Table 3-3 among only adults with chronic conditions (diabetes, asthma, arthritis, hypertension, or heart disease) shows that even in this group, the persistently uninsured had lower monthly health care utilization rates than did the uninsured who subsequently switched to Medicaid and those with Medicaid in all 24 months (Table 3-4).

Though often smaller in magnitude, the excess in monthly expenditures or utilization rates of those who switched to Medicaid over the persistently uninsured remain when we use the second month prior to enrollment as the comparison month for those who transitioned.[52] That the magnitude of the differences often declines as the pre-

[52] Statistical significance of differences in means tends to decline as the pre-transition reference month retreats from the month of transition. While some is due to a smaller difference in means, it is also due to smaller sample sizes. This is because when we look backward (or forward) two or more months, we require that the individual have a constant insurance status in those two or more months prior to and after the transition. For example, among the 2,179 adults who transitioned from no coverage to Medicaid and had Medicaid in the month following transition, only 1,828 were also uninsured in the second month prior to transition and had Medicaid in the second month after transition (Table 3A-2).

transition reference month retreats from the month of transition may be indicative of increasing health care need among uninsured, Medicaid-eligible individuals leading to enrollment once need reaches some crucial threshold.

It may also reflect the effects of retroactive eligibility, however. Medicaid coverage may be applied retroactively for up to three months, provided the beneficiary was eligible during that period. If health care spending and utilization decisions among the uninsured in the three months prior to transition are made as if Medicaid coverage were already in place, they may overstate true differences between switchers and non-switchers. Therefore, we also compare in Tables 3-1 – 3-4 spending or utilization in the fourth month prior to switching to Medicaid with that among the persistently uninsured. Although the statistical significance of differences tends to decline, individuals who transitioned from no coverage to Medicaid still spent more and used more services four months prior to enrollment than did those who remained uninsured for two full years.[53]

In many comparisons, own health care expenditures or utilization rates increase after individuals transition from no health coverage to Medicaid. This may reflect improved access to care and lower cost-sharing under Medicaid than among the uninsured, further deterioration in health status, or pent-up demand for health care. The statistical significance of these differences varies, however, and in some cases, post-transition Medicaid expenditures and utilization rates are comparable to or less than those for long-term Medicaid enrollees.

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[53] While retroactive eligibility is relevant only to the comparisons in Tables 3-1 – 3-4, we show health expenditures in the fourth months prior to and after other types of transitions in Tables 3-5 and 3-6 as well. Tracking spending in the four months leading up or following to a transition can enhance our understanding of the factors causing that transition.

Medicaid disenrollees vs. long-term Medicaid enrollees

In order to assess the cost of reducing temporary or permanent transitions from Medicaid to uninsurance or private group coverage, we compare in Table 3-5 the average monthly total expenditures of long-term Medicaid beneficiaries with the pre-transition Medicaid expenditures of those individuals who switched from Medicaid to either no coverage or private group insurance. The unadjusted monthly per-enrollee cost of Medicaid coverage for those left Medicaid and subsequently became uninsured, in the first month prior to switching, was 73 percent of that of individuals who maintained their enrollment, and the difference is statistically significant. When we adjust for demographic characteristics and health status, this ratio declines slightly. Although the Medicaid expenditures of switchers in the second and fourth months prior to transition exceed those of long-term Medicaid enrollees, these differences are not statistically significant. We observe falling expenditures as time approaches the month of transition which is consistent with declining health needs and thus a lessening incentive to become recertified.

This pattern of declining Medicaid expenditures as time nears the transition month is less evident among Medicaid participants who switch to private group insurance, but pre-transition expenditures are lower than those of stable Medicaid enrollees as far back as four months prior to transition. And, only the adjusted expenditures in the fourth month prior to transition are significantly different than those of the long-term Medicaid population.

Crowd-out

Medicaid expansions and simpler enrollment and recertification procedures are likely to induce some eligible individuals with private group health coverage to switch to Medicaid. This represents "crowd-out". Table 6 shows that the pre-transition expenditures of individuals that switched from private group insurance to Medicaid rose in the months leading up to transition and greatly exceed the expenditures of those with private group coverage for 24 months. In the first month prior to transition, the unadjusted and adjusted expenditures of switchers are only 4 and 3 percent as large as those of individuals with long-term private group insurance. However, in the first and second months after transition, their expenditures are comparable to those of long-term Medicaid enrollees. This suggests that crowd-out is unlikely to raise per-enrollee Medicaid costs.

Results from regression analyses

From the preceding discussion, it is evident that adjusting for age, sex, race/ethnicity, education, poverty status, health status, and region of residence generally increases the spending and utilization differences between our comparison groups as well as the statistical significance of those differences. Thus, even the unadjusted monthly expenditures of individuals transitioning on and off of Medicaid likely overstate the per-enrollee costs of expanding Medicaid coverage. This implies that the pattern of results we find is due to choices that cannot easily be captured by observable characteristics, and therefore cannot be readily adjusted for in standard simulation analyses.

Robustness Checks

We have already shown that individuals who switch from no health coverage to Medicaid spend more on health care and use more services than those who are persistently uninsured, even when we limit the analysis to adults with chronic health conditions or eliminate bias resulting from retroactive Medicaid eligibility. In this section, we show that our findings, with respect to Medicaid enrollees, Medicaid disenrollees, and crowd-out, are also robust to including in expenditure estimates the cost of uncompensated care and limiting the sample to individuals with income less than 200 percent of the federal poverty level (FPL).

Columns 2-4 of Tables 3-1, 3-5, and 3-6 show unadjusted and adjusted expenditures that include the cost of uncompensated care. We estimate the cost of uncompensated care for all individuals who spent at least part of a year uninsured. The cost of uncompensated care is defined as the larger of actual expenditures or average cost, which we compute by multiplying the mean of the ratio of expenditures to charges, among full-year, privately insured respondents in the MEPS with each individual's actual charges (Hadley and Holahan 2003). In Table 3-1, including the cost of uncompensated care increases the excess pre-transition expenditures of individuals who switched from uninsurance to Medicaid over long-term Medicaid enrollees. In Table 3-5, it raises the unadjusted and adjusted Medicaid spending of those who left Medicaid and became uninsured above that of individuals who were uninsured for 2 full years, but the difference is not statistically significant when the reference period is the first month prior to the transition. It has little effect on the comparison between Medicaid spending among long-term enrollees with disenrollees who depart for private group insurance. Similarly,

the inclusion of the cost of uncompensated care has little impact on our analysis of crowd-out in Table 3-6.

We also repeat our main analyses in Tables 3-1, 3-5, and 3-6 on a sample of individuals with income less than 200 percent of FPL in at least one year on the panel, and show these results in the last two columns of these tables. One limitation with this analysis is our inability to distinguish between individuals that take up or leave Medicaid because of changes in eligibility or changing health needs. By adjusting for some correlates of eligibility, the adjusted expenditures partially account for these differences, but another approach is to eliminate (or limit) health insurance transitions resulting from changes in eligibility by restricting the sample to low-income individuals. Thus, these analyses are especially relevant to policies that would expand Medicaid coverage among the currently eligible population by simplifying enrollment and recertification procedures, but not necessarily relevant to proposals to expand Medicaid coverage to all uninsured individuals.[54]

This sample restriction reduces the difference between the one month pre-transition expenditures of those people who gained Medicaid coverage following a spell of uninsurance and the persistently uninsured, but that difference remains statistically significant (Table 3-1). It has less effect on the pre-transition Medicaid spending in Table 3-5, especially the unadjusted expenditures, and again, does not alter our primary findings. The expenditure patterns shown in the last two columns of Table 3-6 suggest that crowd-out may be less costly among the low-income population than among the

[54] As Table 3A-3 shows, nearly half of the persistently uninsured have income above 200 percent of the FPL.

general population of adults, at least in the first and second months after individuals switch to Medicaid from private group insurance.

Discussion

This chapter attempts to inform cost estimates of proposals to expand Medicaid coverage. Coverage expansion might occur by encouraging take-up and coverage maintenance among the currently eligible population or by expanding the eligible population to include anyone without health insurance. The evidence presented here suggests that complicated enrollment and recertification requirements may present substantial barriers to Medicaid coverage among individuals with modest health needs.

The pre-transition health care spending and utilization of individuals who switched from no insurance to Medicaid exceeds that of the persistently uninsured. This, and our finding that expenditures and utilization rates rise prior to transition, is consistent with the notion of increasing health needs culminating in the acquisition of Medicaid coverage. Of course, some of these individuals may have taken up Medicaid because they became newly eligible, and we cannot identify this situation in our data. It is not clear a priori whether this would bias our estimates of pre-Medicaid spending and utilization rates downwards or upwards or at all, since individuals who take up Medicaid immediately upon gaining eligibility are likely to have elevated health needs, according to our hypothesis. However, limiting the sample to low income individuals partially mitigates this bias (if it exists) and does not substantively alter our conclusion that new Medicaid enrollees spend more and use more services while still uninsured than the persistently uninsured. This conclusion also holds among individuals with greater than

average health needs (those with chronic conditions) and when we look backwards four months.

Similarly, we find that the Medicaid disenrollees spend less in the month prior to transition, than do long-term Medicaid enrollees in any given month, suggesting that declining health needs reduce the incentive to recertify. While we cannot differentiate between those who lose coverage voluntarily or involuntarily because of loss of eligibility, our results are robust to the low-income sample restriction, and this lack of information is likely to bias upwards our estimates of pre-transition, Medicaid expenditures. Given our theory that individuals who maintain Medicaid enrollment do so because of elevated health needs, those that leave Medicaid due to loss of eligibility are likely to have greater health needs than those that leave Medicaid because recertification is complicated and time-consuming.

This analysis suggests, therefore, that the costs associated with expanding Medicaid coverage are likely to be substantially less than what an extrapolation from the current Medicaid population would suggest. States should consider both expanding Medicaid eligibility and working to improve retention among those beneficiaries who are currently eligible as a viable approach to the problem of the uninsured. Further research could perhaps identify ways to mitigate crowd-out.

Table 3 - 1. Medicaid Enrollees vs. the Uninsured: Average Total Monthly Health Care Expenditures, by Insurance Status/Transition

			Robustness checks			
	Main results		*Incl. cost of uncompensated care*		*Sample: <200% FPL*	
Insurance Status/Transition	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
Always uninsured	44~	13~	78~	21~	47~	12~
	(3)	(2)	(5)	(2)	(5)	(1)
Uninsured --> Medicaid						
Uninsured_{m-4}	62#	34#	237*	144*#	71#	35**#
	(11)	(5)	(26)	(5)	(14)	(2)
Uninsured_{m-2}	157*	85*#	340*	190*#	152**	46*#
	(54)	(4)	(60)	(4)	(62)	(2)
Uninsured_{m-1}	192*	122~#	384*	250*#	136*#	71*#
	(45)	(4)	(53)	(4)	(34)	(2)
Medicaid_{m-1}	225	248~#	411~	396~#	229#	220~#
	(38)	(4)	(47)	(4)	(44)	(2)
Medicaid_{m-2}	240	279~#	415~	420~#	259	235~#
	(43)	(5)	(48)	(5)	(53)	(3)
Medicaid_{m-4}	245#	240#	387	352~#	144~#	125~#
	(92)	(5)	(94)	(5)	(24)	(2)
Always Medicaid	243*	206*	243*	210*	250*	182*
	(20)	(4)	(19)	(4)	(22)	(2)

See notes on the next page.

~ Statistically different from "Always Medicaid" at the 5% level (~~ 10% level)

* Statistically different from "Always Uninsured" at the 5% level (** 10% level)

Statistically different from own expenditures prior to/post switch at the 5% level (## 10% level)

Notes:

1 Standard errors in parentheses.

2 Adjusted expenditures are estimated using a two-part regression model that controls for age, age squared, sex, race/ethnicity, education, poverty status, health status, and region of residence.

3 Adjusted expenditures shown for the "Always uninsured" and "Always Medicaid" categories are drawn from the sample with transition window of +/- 1 month. These figures are nearly identical when estimated for samples with transition windows of +/- 2 or 4 months.

Table 3 - 2. Medicaid Enrollees vs. the Uninsured: Average Monthly Health Care Expenditures, by Type and Insurance Status/Transition

	Physician		Hospital		Rx	
Insurance Status/Transition	*Unadjusted*	*Adjusted*	*Unadjusted*	*Adjusted*	*Unadjusted*	*Adjusted*
Always uninsured	22~	5~	20~	2	3~	0~
	(1)	(1)	(3)	(0)	(0)	(0)
Uninsured --> Medicaid						
$Uninsured_{m-4}$	45*	20#	4*#	1#	7**	2
	(10)	(2)	(2)	(1)	(2)	(0)
$Uninsured_{m-2}$	31*#	14#	105	35*#	8*	2#
	(4)	(2)	(53)	(1)	(2)	(0)
$Uninsured_{m-1}$	55*#	29*#	125*	36*#	12*	3*##
	(8)	(2)	(44)	(0)	(2)	(0)
$Medicaid_{m-1}$	89#	59#	136	62~#	11*	3
	(13)	(2)	(37)	(1)	(2)	(0)
$Medicaid_{m-2}$	108#	95~#	139	51~#	12	3
	(14)	(2)	(41)	(1)	(3)	(0)
$Medicaid_{m-4}$	181	116~#	76~#	13~#	11	2
	(105)	(3)	(21)	(1)	(3)	(0)
Always Medicaid	103*	66*	148*	29	14*	4*
	(7)	(2)	(18)	(0)	(1)	(0)

See notes on the next page.

~ Statistically different from "Always Medicaid" at the 5% level (~~ 10% level)

* Statistically different from "Always Uninsured" at the 5% level (** 10% level)

Statistically different from own expenditures prior to/post switch at the 5% level (## 10% level)

Notes:

1 Standard errors in parentheses.

2 Adjusted expenditures are estimated using a two-part regression model that controls for age, age squared, sex, race/ethnicity, education, poverty status, health status, and region of residence.

3 Adjusted expenditures shown for the "Always uninsured" and "Always Medicaid" categories are drawn from the sample with transition window of +/- 1 month. These figures are nearly identical when estimated for samples with transition windows of +/- 2 or 4 months.

Table 3 - 3. Medicaid Enrollees vs. the Uninsured: Unadjusted Average Monthly Health Care Utilization, by Type and Insurance Status/Transition

	Physician	Hospital	Emergency room	Rx
Insurance Status/Transition	*Unadjusted*	*Unadjusted*	*Unadjusted*	*Unadjusted*
Always uninsured	7.2~	0.3~	1.0~	3.4~
	(0.2)	(0.0)	(0.0)	(0.1)
Uninsured --> Medicaid				
$Uninsured_{m-4}$	13.5*#	0.3#	2.5*	6.3*##
	(1.7)	(0.1)	(0.6)	(0.9)
$Uninsured_{m-2}$	16.1#	1.2*	3.4*	6.0*#
	(1.3)	(0.3)	(0.6)	(0.8)
$Uninsured_{m-1}$	19.5*#	2.1*	4.0*	9.7*
	(1.2)	(0.5)	(0.6)	(0.9)
$Medicaid_{m-1}$	28.1#	2.3	3.2	9.7
	(1.3)	(0.4)	(0.5)	(0.8)
$Medicaid_{m-2}$	29.2~#	2.0	3.1	9.3#
	(1.4)	(0.4)	(0.6)	(0.9)
$Medicaid_{m-4}$	33.5~#	2.2#	2.9	9.1##
	(1.8)	(0.6)	(0.6)	(1.2)
Always Medicaid	27.2*	1.7*	2.7*	9.0*
	(0.2)	(0.1)	(0.1)	(0.2)

~ Statistically different from "Always Medicaid" at the 5% level (~~ 10% level)

* Statistically different from "Always Uninsured" at the 5% level (** 10% level)

Statistically different from own expenditures prior to/post switch at the 5% level (## 10% level)

Note:

1 Standard errors in parentheses.

Table 3 - 4. **Medicaid Enrollees vs. the Uninsured: Unadjusted Average Monthly Health Care Utilization Among Adults with Chronic Conditions, by Type and Insurance Status/Transition**

	Physician	Hospital	Emergency room	Rx
Insurance Status/Transition	*Unadjusted*	*Unadjusted*	*Unadjusted*	*Unadjusted*
Always uninsured	18.0~	0.9~	2.0~	7.5~
	(0.7)	(0.1)	(0.1)	(0.2)
Uninsured --> Medicaid				
$Uninsured_{m-4}$	21.8#	0.4	4.8**#	12.0**
	(3.2)	(0.3)	(1.6)	(2.4)
$Uninsured_{m-2}$	26.8*#	2.0	5.5*	10.4**
	(2.6)	(0.7)	(1.3)	(1.6)
$Uninsured_{m-1}$	27.4*##	2.5**	5.6*	12.5*
	(2.4)	(1.0)	(1.3)	(1.9)
$Medicaid_{m-1}$	33.1~##	2.4	3.6	12.2
	(2.5)	(0.8)	(0.9)	(1.6)
$Medicaid_{m-2}$	35.4#	3.1	3.3	12.4
	(2.8)	(1.2)	(0.9)	(1.8)
$Medicaid_{m-4}$	40.5#	2.3	1.2~#	13.4
	(3.7)	(1.6)	(1.7)	(2.6)
Always Medicaid	38.8*	2.3*	4.0*	13.2*
	(0.9)	(0.2)	(0.2)	(0.4)

~ Statistically different from "Always Medicaid" at the 5% level (~~ 10% level)

* Statistically different from "Always Uninsured" at the 5% level (** 10% level)

Statistically different from own expenditures prior to/post switch at the 5% level (## 10% level)

Note:

1 Standard errors in parentheses.

Table 3 - 5. Medicaid Disenrollees vs. Long-Term Medicaid Enrollees: Average Monthly Health Care Expenditures, by Type and Insurance Status/Transition

			Robustness checks			
	Main results		*Incl. cost of uncompensated care*		*Sample: <200% FPL*	
Insurance Status/Transition	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
Always Medicaid	243	206	243	209	250	182
	(19)	(4)	(78)	(4)	(22)	(2)
Medicaid --> Uninsured						
$Medicaid_{m-4}$	252#	256#	348~#	319~#	240#	199#
	(44)	(5)	(48)	(5)	(41)	(3)
$Medicaid_{m-2}$	254#	213#	372~#	300~#	226#	162~~#
	(42)	(5)	(45)	(4)	(40)	(2)
$Medicaid_{m-1}$	178~~	145~#	294	223#	146~	102~#
	(27)	(4)	(31)	(4)	(22)	(2)
$Uninsured_{m-1}$	124**	38#	230	89#	53#	21#
	(46)	(3)	(30)	(4)	(15)	(2)
$Uninsured_{m-2}$	52#	18#	156#	59#	51#	15#
	(13)	(4)	(21)	(4)	(16)	(2)
$Uninsured_{m-4}$	55#	14#	136#	35#	50#	12#
	(18)	(4)	(24)	(4)	(18)	(2)

Table continues on next page. See notes at the end of the table.

Table 3 - 5, continued

	Main results		Robustness checks: *Incl. cost of uncompensated care*		Robustness checks: *Sample: <200% FPL*	
Insurance Status/Transition	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
Medicaid --> Private group						
$Medicaid_{m-4}$	220	103~~#	222	98~~	76~	35~#
	(139)	(6)	(139)	(6)	(19)	(3)
$Medicaid_{m-2}$	255	219#	259	196#	386#	225~#
	(59)	(6)	(60)	(6)	(112)	(3)
$Medicaid_{m-1}$	222	157#	226	157	205	141#
	(52)	(6)	(52)	(6)	(52)	(3)
Private $group_{m-1}$	150	192#	153	156	218	165#
	(47)	(5)	(47)	(6)	(87)	(3)
Private $group_{m-2}$	161	68#	164	67#	94#	25#
	(48)	(5)	(48)	(6)	(39)	(3)
Private $group_{m-4}$	288	84#	289	85	575	118#
	(213)	(5)	(213)	(6)	(506)	(3)

See notes on the next page.

~ Statistically different from "Always Medicaid" at the 5% level (~~ 10% level)

Statistically different from own expenditures prior to/post switch at the 5% level (## 10% level)

Notes:

1 Standard errors in parentheses.

2 Adjusted expenditures are estimated using a two-part regression model that controls for age, age squared, sex, race/ethnicity, education, poverty status, health status, and region of residence.

3 Adjusted expenditures shown for the "Always Medicaid" category are drawn from the sample with transition window of +/- 1 month. This figure is nearly identical when estimated for samples with transition windows of +/- 2 or 4 months.

Table 3 - 6. Crowd-out: Average Monthly Health Care Expenditures, by Type and Insurance Status/Transition

			Robustness checks			
	Main results		*Incl. cost of uncompensated care*		*Sample: <200% FPL*	
Insurance Status/Transition	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
Always private group	119~	54~	119~	54~	139~	57~
	(3)	(4)	(3)	(2)	(8)	(1)
Private group --> Medicaid						
Private group$_{m-4}$	158#	87#	171#	91#	232#	101#
	(59)	(6)	(60)	(7)	(104)	(4)
Private group$_{m-2}$	395^^	239^#	409^	245^#	570^^	320^#
	(143)	(6)	(145)	(6)	(239)	(3)
Private group$_{m-1}$	2790	1557^#	2805	1458^#	582^^##	343^#
	(2216)	(6)	(2216)	(6)	(259)	(3)
Medicaid$_{m-1}$	284	183#	298	191#	152~##	121~#
	(73)	(6)	(74)	(6)	(31)	(3)
Medicaid$_{m-2}$	208	200#	222	177#	254	154#
	(47)	(6)	(188)	(7)	(75)	(3)
Medicaid$_{m-4}$	619#	514~#	627~#	494~#	773~#	676~#
	(160)	(7)	(161)	(7)	(213)	(4)
Always Medicaid	243^	206^	243^	211^	250^	182^
	(19)	(4)	(19)	(4)	(22)	(2)

See notes on the next page.

~ Statistically different from "Always Medicaid" at the 5% level (~~ 10% level)

^ Statistically different from "Always private group" at the 5% level (^^ 10% level)

Statistically different from own expenditures prior to/post switch at the 5% level (## 10% level)

Notes:

1 Standard errors in parentheses.

2 Adjusted expenditures are estimated using a two-part regression model that controls for age, age squared, sex, race/ethnicity, education, poverty status, health status, and region of residence.

3 Adjusted expenditures shown for the "Always private group" and "Always Medicaid" categories are drawn from the sample with transition window of +/- 1 month. These figures are nearly identical when estimated for samples with transition windows of +/- 2 or 4 months.

Appendix 3A: Supplemental Tables

Table 3A - 1. Reporting of Multiple Sources of Health Insurance

		Percent that also reported…				
Primary insurance	**Percent**	*Medicare*	*Medicaid*	*Private group*	*Other private*	*Other public*
Uninsured	20.2	0.0	0.0	0.0	0.0	0.0
Medicare	8.7	100.0	0.0	0.0	0.0	0.0
Medicaid	6.0	13.1	100.0	5.5	1.3	0.0
Private group	67.6	0.6	0.0	100.0	0.0	1.4
Other private	4.5	1.8	0.0	0.0	100.0	0.9
Other public	0.9	0.0	0.0	0.0	0.0	100.0
Total	*100.0*	*2.1*	*6.0*	*67.9*	*4.5*	*1.9*

Table 3A - 2. Sample Sizes Given Constant Health Coverage for 1, 2, or 4 Months Pre- and Post-Transition

	Panel A: All adults		
Insurance Status/Transition	1 month	2 months	4 months
Always uninsured	195,072	195,072	195,072
Always Medicaid	62,976	62,976	62,976
Always private group	664,992	664,992	664,992
Uninsured --> Medicaid	2,179	1,828	1,159
Medicaid --> Uninsured	1,891	1,634	1,006
Medicaid --> Private group	429	372	264
Private group --> Medicaid	390	340	212
	Panel B: Adults with chronic conditions		
Insurance Status/Transition	1 month	2 months	4 months
Always uninsured	34,968	34,968	34,968
Always Medicaid	28,212	28,212	28,212
Always private group	197,664	197,664	197,664
Uninsured --> Medicaid	559	488	299
Medicaid --> Uninsured	468	390	229
Medicaid --> Private group	114	100	63
Private group --> Medicaid	110	98	54
	Panel C: All poor adults (< 200% FPL)		
Insurance Status/Transition	1 month	2 months	4 months
Always uninsured	118,296	118,296	118,296
Always Medicaid	55,092	55,092	55,092
Always private group	77,244	77,244	77,244
Uninsured --> Medicaid	1,815	1,507	901
Medicaid --> Uninsured	1,587	1,347	791
Medicaid --> Private group	255	215	135
Private group --> Medicaid	256	217	128

Table 3A - 3. Descriptive Statistics for Demographic Adjustors Used in Regression Models

		Insurance Status/Transition						
	Total	*Always uninsured*	*Always Medicaid*	*Always private group*	*Uninsured --> Medicaid*	*Medicaid --> Uninsured*	*Medicaid --> Private group*	*Private group --> Medicaid*
Mean age (years)	39.4	36.7	38.4	41.8	33.6	32.4	33.0	33.3
Female (%)	51.0	41.8	66.1	51.1	71.0	71.7	73.6	72.3
Race/ethnicity (%)								
White	71.3	50.8	46.4	78.6	51.9	48.9	63.5	59.3
Black	8.6	10.6	20.8	7.1	14.0	12.1	14.9	16.7
Hispanic	12.0	29.6	17.2	7.4	22.9	25.0	13.2	13.5
Other	8.0	9.0	15.6	6.8	11.2	14.0	8.4	10.5
Education (%)								
< HS	6.8	19.1	23.6	3.0	17.4	16.7	8.2	10.3
HS	32.1	34.9	35.8	30.8	34.6	36.3	39.0	40.3
Some college	24.5	17.6	12.4	25.8	17.2	16.5	27.5	25.9
College+	26.2	9.5	3.2	34.2	3.9	3.8	9.6	7.9
Unknown	10.3	18.9	25.1	6.1	27.0	26.7	15.8	15.5
Poverty status (%)								
< 100% FPL	9.7	21.5	56.8	1.7	46.6	48.1	17.0	18.2
100-125% FPL	3.5	7.7	10.5	1.1	13.0	12.5	9.6	9.7
125-200% FPL	11.8	23.5	16.9	6.2	21.6	21.3	28.5	33.3
200-400% FPL	32.0	30.8	11.9	32.9	14.7	15.4	25.5	23.2
400%+ FPL	43.1	16.5	3.9	58.2	4.1	2.6	19.5	15.6

Table continues on the next page.

Table 3A - 3, continued

		Insurance Status/Transition						
	Total	*Always uninsured*	*Always Medicaid*	*Always private group*	*Uninsured --> Medicaid*	*Medicaid --> Uninsured*	*Medicaid --> Private group*	*Private group --> Medicaid*
Health								
Excellent	28.7	25.4	12.7	30.5	16.9	17.3	21.6	19.6
Very good	35.1	30.6	19.8	37.9	25.2	26.9	33.0	32.0
Good	26.4	31.4	30.4	25.0	35.1	38.3	31.7	29.4
Fair	7.4	9.9	23.1	5.3	16.3	13.6	11.0	14.5
Poor	2.5	2.6	13.9	1.3	6.5	3.9	2.7	4.5
Region (%)								
Northeast	19.2	14.9	28.4	20.6	19.2	18.9	21.1	21.8
Midwest	22.9	15.3	18.0	25.9	19.0	17.4	23.1	26.6
South	35.1	43.8	28.2	33.0	31.6	32.5	27.8	30.5
West	22.8	26.0	25.4	20.4	30.3	31.2	27.9	21.1

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