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THE ECONOMICS OF CHILDBEARING: THREE ESSAYS

by

ANDREA KUTINOVA

B.A., Charles University in Prague, 2001

M.A., University of New Hampshire, 2003

DISSERTATION

Submitted to the University of New Hampshire

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ABSTRACT

THE ECONOMICS OF CHILDBEARING: THREE ESSAYS

by

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University of New Hampshire, May, 2006

Expenditure programs, business cycles, and government interventions can affect many decisions surrounding the birth of a child. For example, public insurance programs such as Medicaid have the potential to increase the utilization of prenatal care. This, in turn, may lead to better infant and maternal health outcomes. Given the high and increasing number of pregnant women covered by Medicaid, the effectiveness of the program in promoting prenatal care use and improving health needs to be evaluated. Also, the impacts of business cycles on childbearing are of interest to policymakers. For example, does unemployment substantially affect the decision to conceive a child or the ability to obtain appropriate medical services? And, if so, are infant and maternal health outcomes compromised during economic downturns? A well-informed government can design policies to help deal with issues such as these. Government interventions, however, can have unintended (and potentially harmful) consequences as well. For example, several recent economics papers have demonstrated that fiscal policies may affect fertility and the timing of delivery. Understanding the incentives embedded in government programs and assessing the responsiveness of individual behavior to these incentives is therefore key. My dissertation consists of three essays in which I investigate an important understudied aspect of the Medicaid program, inform policymakers about the impacts of unemployment on prenatal care use, infant and maternal health, and add to

our understanding of the unintended effects of government interventions on the timing of births.

PREFACE

The first essay of my dissertation (coauthored with Karen S. Conway) contributes to evidence regarding the effectiveness of the Medicaid expansions by focusing on a key beneficiary - the mother - who has previously been overlooked. Using the Natality Detail Files for 1989-96, we estimate the relationship between Medicaid eligibility and potentially avoidable maternal morbidities (placental abruption, anemia, and pregnancy-related hypertension) for several treatment groups and a control group. The validity of our cohorts is verified in an analysis of the Current Population Survey data. Potential biases caused by improved reporting are addressed by using a 'straw man' maternal complication (diabetes) not preventable with prenatal care. Our results suggest that increased Medicaid eligibility leads to fewer preventable maternal complications among women most likely to have benefited from the Medicaid expansions.

As the first essay demonstrates, a well-informed government can design policies promoting prenatal care use and health outcomes. This may be especially important during temporary economic downturns. Interestingly, infant health and prenatal care use reportedly improve when the unemployment rate increases. In my second essay, I test the hypothesis that one avenue for these beneficial effects is an improved access to health insurance via the Medicaid program. I focus on local labor markets and add maternal health to the outcomes studied. Using the Natality Detail Files for 1989-99 aggregated to county/year/race cells, I find that higher unemployment at the county level is associated with improved infant health especially among whites and increased prenatal care use and potentially improved maternal health among blacks. In some cases, both unemployment per se and Medicaid contribute to these benefits. In others, Medicaid mitigates, offsets, or

outweighs detrimental effects of unemployment. At least some of these aggregate results are due to changes in the selection into pregnancy. Consistent with the role of Medicaid as a safety net for credit-constrained populations, unemployment increases - and Medicaid eligibility decreases - the proportion of high-SES mothers.

While the first two essays show beneficial effects of a targeted public program, government interventions can have unintended (and potentially harmful) consequences as well. During the conflict in Vietnam, married men with dependents could obtain a deferment from the draft. In 1965, following President Johnson's Executive Order 11241 and a Selective Service System announcement, this policy changed substantially in a way that provided strong incentives for childless American couples to conceive a first-born child. Since the changes were both unexpected and widely publicized, this is an ideal opportunity to study the effects of policy on fertility. Information about the fecundity of the U.S. population in the 1960's and anecdotal evidence (e.g., conception of the current Vice President's first daughter) suggest that young couples were ready to react quickly. In my third essay, I extract time series data from the Vital Statistics for 1963-68 and employ a difference-in-differences methodology. My analysis suggests that the number of first births increased by 15,532 in June and August 1966 in response to the policy changes. Such an increase represents over 7% of the total number of first deliveries and about 28% of the Selective Service System calls for inductees in those months.

All in all, in my dissertation, I contribute to our knowledge about how the government and the economy interact to influence the birth of a child. In particular, I investigate an important understudied aspect of the Medicaid program, inform policymakers about the impacts of unemployment on prenatal care use, infant and

maternal health, and add to our understanding of the unintended effects of government interventions on the timing of births. By complementing previous studies in the economics of childbearing, my research helps policymakers design programs that benefit both the mother and the child.

1. THE FORGOTTEN BENEFICIARY OF THE MEDICAID EXPANSIONS

1.1. Introduction

In the late 1980's and early 1990's, the Medicaid eligibility rules changed substantially. The income thresholds increased and individuals in two-parent families started to qualify. By providing health insurance coverage to all low-income pregnant women and their children, the policymakers hoped to achieve their ultimate goal: improve health outcomes. Have they succeeded? Trying to answer this question, several studies have investigated the effects of the expansions on infant health (Currie and Gruber 1996a, 1997; Dubay et al. 2001; Currie and Grogger 2002) and a few studies have focused on the effects on child health (Currie and Gruber 1996b; Kaestner et al. 2001). So far, the results have been mixed, leading to a general skepticism about the effectiveness of the Medicaid eligibility expansions in improving health.

We argue, however, that an important potential beneficiary of the expansions – the mother – has been left completely out of the analysis. To our knowledge, no economic study has investigated the effects of the policy changes of the 1980's and 1990's on maternal health. However, pregnant women have always been a key target population of the Medicaid program. Therefore, without estimating the impacts of the expansions on maternal health (in addition to infant health and child health), any evaluation of the effectiveness of the policy is incomplete. In this paper, we attempt to close the gap. In particular, using the *Nativity Detail Files* for 1989-96, we estimate the relationship between Medicaid eligibility and maternal health outcomes for several treatment groups and a control group. Our results suggest that increased Medicaid

eligibility indeed lead to fewer preventable maternal complications among women most likely to have benefited from the expansions.

1.2. Background

It is surprising to find that the direct health effects of policies targeted at disadvantaged women in the US have largely been overlooked in the economics literature. After ten years, an observation made by Jennifer Haas and her coauthors (Haas et al. 1993) remains valid: “Although there has been substantial policy interest in interventions to improve the neonatal outcomes of disadvantaged women, little attention has been paid to the health status of pregnant women themselves.” (p.61) As our previous research suggests, this is an important oversight. In Conway and Kutinova (2005), we demonstrate that timely and adequate prenatal care (to which Medicaid can improve access) may increase the probability of maintaining a healthy weight after the birth.

We are aware of only one recent economic policy-oriented study dealing with the health status of disadvantaged women in the US: Kaestner and Tarlov (2003) investigate the effects of the welfare contractions of the 1990’s on women’s health (overall health status and mental health) and health behaviors (smoking, drinking, and exercise). In particular, the authors hypothesize that the welfare changes were likely to affect the “employment stress”, “organizational stress”, and “financial stress” faced by low-income women and thus might have indirectly affected the health status of these women. While the Kaestner and Tarlov (2003) study certainly represents an important contribution to the health economics literature, it does not fill the gap identified above. First, the authors focus on the general health of a disadvantaged population rather than studying the

particular health complications women may encounter due to pregnancy and/or maternity. Second, the study deals with an indirect impact of a general welfare program on health outcomes and behaviors rather than estimating the effects of a policy - such as Medicaid - designed primarily to improve the health status of its target population.

To our knowledge, there are only two economic studies of health policies in the US that include the mother - Currie and Gruber (1997, 2001). However, these studies focus on the effects public insurance has on the medical *treatments and procedures* provided to the mother (i.e., cesarean section delivery, use of a fetal monitor, receipt of ultrasound and induction/stimulation of labor). They do not estimate any impact on maternal *health outcomes*.

Is maternal health an issue in a developed country such as the US? We believe that it is. As Haas et al. (1993) note in their study: "Although only 10 per 100,000 women die from a complication of pregnancy or childbirth, 60% of women receive medical care for some complication of pregnancy, and 30% suffer complications that result in serious morbidity."(p.61) An interest in the issues surrounding maternity in the US is finally awakening among applied economists; for instance, Chatterji and Markowitz (2004) estimate the impacts of the length of maternity leave on maternal depression and women's "overall health" (number of outpatient visits) postpartum. The importance of maternal health has also repeatedly been recognized in national health guidelines – most recently the Healthy People 2010¹ (Public Health Service 2000). Also, the Medicaid

¹ The Healthy People 2010 include an explicit objective to "reduce maternal illness and complications due to pregnancy" (Objective 16-5) which involves reduction in "prenatal illness and complications" as well as "complications during labor and delivery."

program itself has been designed to help disadvantaged pregnant *women* and their infants and children.

Unfortunately, due to the lack of research in the area, there is not a generally recognized measure of maternal health (an analog to birth weight in infant health studies). Facing this problem in our current study, we have decided to focus on the incidence of three complications to maternal health identified in the medical literature as potentially preventable by prenatal care: placental abruption, pregnancy-associated hypertension, and anemia. In addition, due to the rarity of these events, we have also employed a summary indicator of maternal health capturing the presence of any of the three complications mentioned above. All of our measures of maternal health can be derived using the information available in vital statistics.

Placental abruption² and pregnancy-associated hypertension³ are identified in Haas et al. (1993) as important causes of maternal morbidity that can be prevented by interventions during the prenatal period. The Healthy People 2010 stress the need for timely and high-quality prenatal care which would “improve maternal health by identifying women who are at particularly high risk and taking steps to mitigate risks, such as the risk of high blood pressure [...]” (Public Health Service 2000, p.16-8) In the public health literature, the role of comprehensive prenatal care in managing hypertension has long been recognized (Scholl et al. 1994, Sachs et al. 1988). As for anemia,⁴ several

² “Premature separation of a normally implanted placenta from the uterus.” (CDC 2003)

³ “An increase of blood pressure of at least 30mm Hg systolic or 15mm Hg diastolic on two measurements taken 6 hours apart after the 20th week of gestation.” (CDC 2003)

recent medical papers have investigated the options for preventing the occurrence of this complication in pregnant women and have concluded that adequate iron therapy during prenatal period can be very effective (Bashiri et al. 2003, Makrides et al. 2003, Villar et al. 2003). The Healthy People 2010 recommendations urge to “reduce anemia among low-income pregnant females in their third trimester” and to “reduce iron deficiency among pregnant females.” (Public Health Service 2000, Objectives 19-13 and 19-14, respectively) Laditka et al. (2005a) who have constructed an index of avoidable maternity complications (AMCs) stress the role of prenatal care in preventing and treating anemia. Therefore, if the Medicaid expansions increased the health insurance coverage of low-income pregnant women and improved their access to prenatal care, our four measures (including the summary indicator) should be able to capture the potential positive impact of the expansions on maternal health.

As a CDC report notes, anemia and hypertension were among the most common complications of pregnancy in the 1990’s (CDC 2001). In the year 1999, for example, 2.32 and 3.82 percent of pregnant women suffered from anemia and pregnancy-associated hypertension, respectively. Placental abruption occurs less frequently (0.6 percent of pregnant women had it in the year 1999) but its consequences are more severe.

Preventing maternal complications such as anemia, hypertension, and placental abruption can lead to improvements in the quality of life as well as to substantial cost savings. In the year 1997, for example, pregnancy-related hypertension and anemia were among the top 100 primary diagnoses associated with the highest national expenditures for hospital stays; the costs of “hypertension complicating pregnancy, childbirth and the

⁴ “Hemoglobin level of less than 10.0 g/dL during pregnancy or a hematocrit of less than 30 percent during pregnancy.” (CDC 2003)

puerperium” were over \$1,237,239,000 and the costs of anemia (pregnancy-related or other) over \$962,102,000. For purposes of comparison, the national charges for hospital stays due to “short gestation, low birth weight, and fetal growth retardation” were about \$1,102,761,000 in the year 1997 (Geocities 2004). Estimates of the overall annual costs of “hypertensive disorders of pregnancy” for the year 2003 exceed \$3 billion (Preeclampsia Foundation 2004). Placental abruption is a rarer - but still costly - morbidity. In the year 1996, for example, the annual national costs of hospitalizations due to placental abruption were \$156 million (AHRQ 1996). These numbers further highlight the fact that maternal health is an important issue.

1.3. Empirical Strategy and Data

The two major changes to the Medicaid policy in the late 1980's and early 1990's were a dramatic increase in the income cutoff below which women qualified for Medicaid and an extension of Medicaid eligibility to married women. The federal government has played a key role in initiating these changes. By April 1990, all states were required to offer Medicaid coverage to pregnant women with incomes below 133% of the federal poverty line. However, the states were given some freedom in designing their Medicaid programs. For example, states could increase the eligibility threshold for pregnant women up to 185% of the poverty line and still qualify for subsidies from the federal government. It is also important to note that the states started from very different positions with initial eligibility ranging from 34% (Louisiana) to 185% (Massachusetts, Minnesota, Mississippi, and Vermont) of the federal poverty line in 1988. As a result, while the increase in Medicaid eligibility in the early 1990's was a nation-wide

phenomenon, the states differed with respect to the *magnitude* of the increase. Also, the *timing* of the changes varied widely across states. Figure 1 shows how the minimum, maximum, and average eligibility cutoffs changed over time and Figure 2 shows how the eligibility rules differed across the five largest US states. This variability allows us to study the effects of the Medicaid eligibility increases on utilization of prenatal care and the associated maternal health improvements while controlling for state heterogeneity in unobservable characteristics as well as a national time trend.

1.3.1. Empirical Strategy

Mothers with low socioeconomic status (SES) were most likely to be affected by the Medicaid policy changes. Many of these women did not qualify for Medicaid before the reforms (either because they had incomes above the cutoff or because they were married) but gained their eligibility in the early 1990's. High SES women, on the other hand, are unlikely to benefit from the reforms because their incomes are too high. This variability in the likely effects of the Medicaid eligibility expansions across individuals allows us to further identify the causality of the relationship between the expansions and prenatal care utilization and health outcomes. In particular, we adopt a difference-in-differences type of approach and compare the effects of the policy changes among members of several treatment groups – low SES married and single mothers – and a control group – high SES married women.⁵ If Medicaid did help its target population, we

⁵ For the sake of comparability, we follow the general spirit of the infant health literature (Currie and Grogger 2002) and treat marital status as exogenous. As some have suggested, however, the decision to marry might have itself been affected by the changes in the Medicaid eligibility rules. (Yelowitz 1998)

would expect to find a significant effect of the expansions on women in the treatment groups but an insignificant effect on women in the control group.

Furthermore, we hypothesize that very-low SES pregnant women benefited from the expansions the most. As previous studies have found, the eligibility expansions were most likely to lead to insurance coverage increases (high take-up rate, low crowd-out of private insurance), increases in the utilization of a variety of obstetric procedures, and infant health improvements among the lowest SES women (Currie and Gruber 1996a, 1997, 2001). Therefore, any improvements in maternal health attributable to the Medicaid eligibility expansions would likely be concentrated in the very-low SES cohort. Since married women might be more strongly affected by the eligibility changes than single mothers (many single women already qualified for Medicaid before the reforms) and since the two groups of women could also be differentially affected by the welfare declines of the early 1990's (only single women generally qualified for AFDC at that time⁶) we have decided to stratify our treatment population by marital status. The control group is selected to represent mothers least likely to benefit from the expansions (with high SES married women typically ineligible for means-tested public programs).

Unfortunately, our data (to be discussed shortly) do not include information on individual-level income. Therefore, we follow earlier studies (Dubay et al. 2001; Currie and Grogger 2002; Kaestner and Kaushal 2002; Kaestner and Tarlov 2003; Joyce et al. 2003) and proxy for socioeconomic status with educational achievement. In particular, we assign women with less than 12 years of education ("less than high school"), 12 years

⁶ Married women could only qualify for the AFDC-UP (Unemployed Parent) program which provided transitional cash assistance to families in which both parents were living in the household and the principal earner, whether the father or the mother, was unemployed.

of education (“high school completed”), and between 13 and 15 years of education (“some college”) into three separate less educated/low SES cohorts and women with 16 or more years of education (“college completed”) into the highly educated/high SES cohort.

We explore the validity of this stratification with an analysis of the Current Population Survey (CPS) data. In particular, we use 1989 and 1996 data from the CPS to calculate cohort-specific “treatment probabilities” according to the following formula:

$$\begin{aligned} \text{Prob}(\text{treatment}) &= \text{prob}(\text{covered by Medicaid in 1996} | \text{not covered by Medicaid in 1989}) \\ &= \text{prob}(\text{covered by Medicaid in 1996 and not in 1989}) / \text{prob}(\text{not covered} \\ &\quad \text{by Medicaid in 1989}), \end{aligned}$$

which we approximate with the following:

$$= 100 * (\% \text{ covered by Medicaid in 1996} - \% \text{ covered by Medicaid in 1989}) / (100 - \% \text{ covered by Medicaid in 1989}).$$

The treatment probabilities for each of our race/education/marital status cohorts are reported in the first column of Table 3, and they clearly demonstrate that less educated women were most affected by the expansions. Among blacks, married women with “less than high school” education had the highest treatment probability ($\text{prob}(\text{treatment})=17.51$) followed by single low-educated women ($\text{prob}(\text{treatment})=4.32$). While the treatment probability in the latter group seems relatively low, it is important to note that unmarried black women with “less than high school” education experienced a decline in the AFDC caseload of about 11 percentage points in the studied period (in contrast to a modest *increase* in most other groups). Thus, Medicaid

expansions above and beyond welfare had to be especially strong in this cohort in order to offset the negative effect of the AFDC contraction.

Among the whites, the treatment probability was the highest among single mothers with “less than high school” education ($\text{prob}(\text{treatment})=13.24$), followed by single mothers with “high school completed” ($\text{prob}(\text{treatment})=8.17$), and married women with “less than high school” education ($\text{prob}(\text{treatment})=7.73$). As hypothesized, treatment probabilities were close to zero among highly educated women from both racial groups ($\text{prob}(\text{treat})=-1.36$ and 0.79 among blacks and whites, respectively). These findings strongly support our selection of the treatment and control groups. Furthermore, as a recent paper (Lewbel 2003) demonstrates, any misclassification into the treatment and/or control cohorts will bias the estimated treatment effects towards zero making our results conservative.

In addition to treatment misclassification, however, there is still a possible confounding factor in that improved prenatal care access (and by extension Medicaid) could lead to increased reporting of maternal health complications. This reporting bias would cause further underestimation of the impact Medicaid has on maternal health. To deal with this issue, we use a “straw man” complication against which we compare the results for our key measures of maternal health (placental abruption, anemia, and pregnancy-related hypertension). Unlike our central measures, this complication should not be preventable by prenatal care and thus should not be affected by the Medicaid eligibility changes. Based on our reading of the medical literature, diabetes fulfills these requirements (Buchanan and Xiang 2005, Ecker 2004, Farrell 2003, Gabbe and Graves 2003, Simmons 1996). Also, the high prevalence of diabetes makes this disease a suitable

candidate for the “straw man”.⁷ If reporting bias is present, we would expect Medicaid to have a positive, if anything, effect on the incidence of diabetes and we could use this estimated effect to make adjustments to the predicted effects of Medicaid on our other - preventable - complications.

1.3.2. Description of Data and Models Estimated

Our data comes from the Natality Detail Files for the years 1989 to 1996 (US Dept. of Health and Human Services 1990-1997). This data (for the period 1990 to 1996) is used by Currie and Grogger (2002) who estimate the impacts of the Medicaid eligibility increases on prenatal care utilization and infant health. Since our approach here is close to that followed by Currie and Grogger (with the important exception that we focus on maternal health instead of infant health), the use of the same dataset has the advantage of making comparisons between the two studies possible.

Since 1985, the Natality Detail Files have included information on all US births and so have contained more than 3 million observations annually. The large sample size is especially useful given our goal which is to study the determinants of relatively rare outcomes – particular complications of pregnancy and delivery. Furthermore, since we estimate all of our models separately for the treatment and control groups and also want to stratify our sample by race, a large number of observations is a necessity.

The Natality Detail Files include information on maternal and infant characteristics (such as mother’s age, marital status, race, Hispanic origin, education, and

⁷ In the 1990s, diabetes was – together with anemia and pregnancy-related hypertension – among the three most common complications of pregnancy (CDC 2001).

state of residence; and infant's sex and birth weight) as well as the characteristics of pregnancy (such as gestation and birth order) typically employed in infant health studies. In addition, since 1989, variables describing maternal morbidity during pregnancy and delivery have also been present. For example, and importantly for our purposes in this paper, the files contain information on the incidences of placental abruption, pregnancy-associated hypertension, anemia, and diabetes in pregnant women.⁸

The biggest disadvantage of using the Natality Detail Files is that the dataset does not include any information on individual-level income or insurance status. Therefore, as discussed above, we rely on educational achievement as a proxy for socioeconomic status. While certainly imprecise, we believe this measure allows us to identify low SES and high SES women as reliably as possible. Moreover, stratification of the sample based on education seems appropriate in that educational achievement is unlikely to be affected by the Medicaid policy changes. Note also that the absence of individual-level income data does not cause a problem in constructing our Medicaid eligibility variable. To capture the exogenous effect of the Medicaid policy on pregnant women, we follow Cutler and Gruber (1996), Currie and Grogger (2002) and others and use a state-wide eligibility measure (to be described later) rather than the eligibility status of each particular individual.

⁸ Sixteen "medical risk factors" (anemia, cardiac disease, lung disease, diabetes, genital herpes, hydramnios/oligohydramnios, hemoglobinopathy, hypertension chronic and pregnancy-associated, eclampsia, incompetent cervix, previous infant 4000+ grams, previous preterm or small-for-gestational age infant, renal disease, Rh sensitization, and uterine bleeding) and fifteen "complications of labor and/or delivery" (febrile, meconium, premature rupture of membrane, abruption placenta, placenta previa, other excessive bleeding, seizures during labor, precipitous labor, prolonged labor, dysfunctional labor, breech/malpresentation, cephalopelvic disproportion, cord prolapse, anesthetic complication, and fetal distress) are separately identified in the natality files. Out of these, we focus on conditions that significantly affect maternal health and are known to be preventable by timely and adequate prenatal care. Diabetes serves as a "straw man".

In this study, we limit our sample to non-Hispanic black and white women. Since previous studies of the effects of Medicaid on prenatal care use and pregnancy outcomes document large racial differences (Dubay et al. 2001 and Currie and Grogger 2002), we stratify all of our models by race. This strategy is also supported by the fact that the “treatment probabilities” calculated from the CPS vary greatly between blacks and whites within each education/marital status group. We only look at women 19 to 50 years of age who had a singleton birth in the period from 1989 to 1996. As Joyce et al. (2003) note, the variability of educational achievement and marital status among teenage mothers is not sufficient to reliably assign these women into the treatment and control groups. Furthermore, in the case of teenage pregnancies, it is not clear whether the mother herself is the ultimate decision maker. In the baseline models, we include women with no or “unknown” prenatal care utilization. As a robustness check, however, we also exclude these women from the analysis, and the qualitative results do not change. Foreign residents are excluded. Further, women from Louisiana and Nebraska in the year 1989, Oklahoma in years 1989-1990, and New York in years 1989-1991 are excluded due to missing information on maternal health. Mothers from Washington State in years 1989-1991 are excluded due to missing information on marital status and those from New Hampshire in years 1989-1992 due to missing information on ethnicity. Finally, it is important to note again that we only focus on selected treatment and control groups in the current study. These data cuts leave us with 10,855,048 observations in our final sample.

To investigate the impacts of the Medicaid eligibility increases of the 1990’s on prenatal care utilization and maternal health, we estimate several reduced-form models. First, following Currie and Grogger (2002), we regress different measures of prenatal

care utilization (PNC) on a measure of Medicaid eligibility (ELIG), welfare caseload (CASELOAD), unemployment rate (UNEMPL), a full set of state and year dummies (u and v , respectively), and individual characteristics (X). Our model has the following general form:

$$PNC_{ist} = \alpha + \beta * ELIG_{st} + \gamma * CASELOAD_{st} + \delta * UNEMPL_{st} + u_s + v_t + \theta * X_{ist} + \epsilon_{ist}$$

where i represents individuals, s states and t time periods.

While the ultimate outcome of interest is maternal health, we find it useful to focus on prenatal care utilization (an input into health production in the household production framework) first. This is done in order to explore the most likely channel through which Medicaid eligibility can indirectly benefit pregnant women. We focus on two measures of prenatal care (based on Currie and Grogger 2002): “timely prenatal care” as determined by whether the women received prenatal care in the first trimester of her pregnancy and “adequate prenatal care” as defined by “adequate” or “intermediate” care on the APNCU (Adequacy of Prenatal Care Utilization) scale.

After estimating the prenatal care equations, we turn our attention to models of maternal health outcomes (MHEALTH) in the following general form:

$$MHEALTH_{ist} = \alpha + \beta * ELIG_{st} + \gamma * CASELOAD_{st} + \delta * UNEMPL_{st} + u_s + v_t + \theta * X_{ist} + \epsilon_{ist}$$

The right-hand-side variables are defined as above and the subscripts i , s , and t index individuals, states, and time periods, respectively.

As mentioned above, we focus on four measures of maternal health (MHEALTH): the incidence of placental abruption as a complication of delivery, the

incidences of pregnancy-related hypertension and anemia as complications of pregnancy, and the incidence of at least one of the three complications listed (a “summary variable”). In addition, we explore the impacts of the Medicaid expansions on a “straw man” maternal complication: diabetes. The maternal health models include all of the explanatory variables from the prenatal care equations.

Our measure of eligibility is the state-level time-specific Medicaid eligibility cutoff (as a percent of the federal poverty line) below which pregnant women qualified for Medicaid (Hill 1992, National Governors’ Association 2003). Following Currie and Grogger (2002), we merge the eligibility measure to the vital statistics by half-years. This is done to account for the fact that the eligibility rules often change twice in a year.

As discussed in Currie and Grogger (2002), the declines in welfare caseloads throughout the 1990’s might have affected prenatal care utilization by pregnant women by making the access to Medicaid more difficult for them. While the link between Medicaid and welfare has formally been eliminated, the authors make the point (and support it by empirical evidence) that an “administrative link” between Medicaid and AFDC has persisted making the application process for Medicaid more burdensome for women not enrolled in welfare. Therefore, we include a welfare caseload variable in our models. The measure (same as in Currie and Grogger (2002)) is constructed as the percentage of each state’s population enrolled in the welfare program in each year (Administration for Children and Families 2003a, 2003b; US Census Bureau 2003a, 2003b). While the variable clearly does not capture all the institutional changes to the welfare program in the 1990’s, it is used here as a simple proxy for the program’s overall generosity.

Like Currie and Grogger (2002), we include the unemployment rate (Bureau of Labor Statistics 2005) in our analyses to proxy for the general economic conditions facing women in the different state/year cells. In addition, full sets of state and year dummies are employed in order to account for state-specific, time-invariant effects and general time trends, respectively. As in Currie and Grogger (2002) and Joyce et al. (2003), we lag our policy variables (Medicaid eligibility threshold, welfare caseloads, and unemployment rate) by six months to allow them to impact pregnant women at a crucial stage of their pregnancies.

As for individual characteristics, we use education and marital status dummies to define our treatment and control groups. We also stratify our sample by race (focusing on non-Hispanic black and white mothers only). In addition, mother's age, age squared, parity, and infant gender are included in all of our models.

In our econometric analyses, for reasons of computational convenience and to ensure that the observed differences in the significance of the Medicaid eligibility coefficients are not driven by vast differences in sample sizes among our treatment and control cohorts, we use a 1/3 random sub-sample of the largest highly educated/white population. We check the robustness of our findings to re-sampling. All models are estimated by both logit and OLS, with standard errors adjusted for clustering by state and year.

1.4. Descriptive Statistics

Table 1 shows the descriptive statistics for the full samples of less educated married and single and highly educated married mothers. As can be seen, black women

are substantially more likely to suffer from anemia than white women. Note also that this racial difference exists at all education levels. Furthermore, the gap seems to be proportionally the largest among highly educated women. The incidences of placental abruption and hypertension, on the other hand, are similar across the races.⁹ Interestingly, among blacks, hypertension occurs much more frequently among highly educated mothers than among women from the less educated cohorts. This may perhaps be attributable to the significantly higher mean age in the highly educated sample. According to the Centers for Disease Control and Prevention, the incidence of pregnancy-associated hypertension is elevated at the extreme tails of the maternal age distribution (CDC 2003). Anemia and placental abruption are more prevalent in the less educated groups. Due to the offsetting effects of education on hypertension versus placental abruption and anemia, the incidence of “any complication” appears fairly stable across the education cohorts. The incidence of diabetes is higher among married than among single women and, among single women, it increases with education. This pattern likely reflects the variation in maternal age.

The other patterns in Table 1 corroborate findings of previous studies. Namely, black women (in all cohorts) tend to start prenatal care later and are also less likely than white women to receive adequate care. Highly educated women have higher utilization of prenatal care than less educated mothers. Married low-educated women receive earlier and more adequate care than single low-educated women. Highly educated mothers are substantially older than less educated mothers and have fewer children on average. Black women are disproportionately represented in the “unmarried” cohorts and, irrespective of

⁹ In a recent report, the CDC also discovers and notes these racial patterns (CDC 2003).

marital status, have higher parity than white women. As expected, there are no big differences across the racial and education groups with respect to the state-level variables. On average, all cohorts face similar Medicaid eligibility thresholds, welfare caseload levels, and unemployment rates.

Table 2 shows the trends in prenatal care utilization (represented by receipt of prenatal care in the first trimester) and maternal health in the period under study. As is apparent, in years 1989-1996, the utilization of prenatal care increased substantially. The percentage of women receiving early prenatal care rose for all education categories and the change was especially remarkable for the less educated cohorts. For example, for black single low-educated (“less than high school”) women - the most “disadvantaged” group - the percentage of those receiving prenatal care in the first trimester of their pregnancy increased from about 48% in the year 1989 to approximately 60% in the year 1996.

As also evident from Table 2, the incidence of anemia initially slightly decreased (reaching minimum in years 1990-1992) and then kept increasing (for most cohorts) during the mid-1990’s. This pattern was even stronger for hypertension. The incidence of placental abruption, on the other hand, did not change or declined modestly. As a CDC report notes, anemia and hypertension were (in addition to diabetes) among the three most common complications of pregnancy in the 1990-1999 period and “their rates have risen steadily” (27% increase in anemia and 40% increase in hypertension; CDC 2001, p.11). Unfortunately, a simple descriptive analysis does not enable us to study the underlying causes of these observed trends. Most likely, several factors affected the incidence of maternal complications simultaneously. For example, average maternal age

first modestly decreased and then increased in the mid-1990's. If maternal age is an important determinant of women's health, this could explain some of the observed patterns. Similarly, an initial acceleration and a later slowdown of the Medicaid eligibility expansions would be consistent with the observed trends. To account for all of these concomitant changes, a multivariate approach is needed.

Another problem with the reported numbers is that they do not enable us to distinguish between a "true" increase in maternal complications and a better monitoring of already existing morbidities. As the CDC report acknowledges: "Some of the apparent increases since 1990 may be an artifact of improved reporting." (CDC 2001, p.11) As long as the improvements in reporting have been universal (independent of the Medicaid expansions), their effects should be captured by the year dummies and should not bias our policy coefficients. It is highly probable, however, that the Medicaid expansions did contribute to better reporting. If women targeted by the Medicaid program had traditionally been those most likely to go without prenatal care and if Medicaid succeeded in providing these women with such care (of which pregnancy monitoring is a key component), we could observe a positive correlation between the Medicaid expansions and the reported maternal complications.

We take some comfort in the fact that - based on the descriptive statistics - the "bad" trends seem to have been similar across the education cohorts. Moreover, even if our estimates of the "true" beneficial impacts of the expansions suffer from this reporting bias, the direction of the bias will be downward, making our results conservative. And, finally, our analysis of diabetes provides a "straw man" against which to compare the central results. Specifically, as argued above, diabetes does not seem to be preventable by

prenatal care. Thus, any effect of the Medicaid expansions on the incidence of diabetes would only reflect improvements in reporting. The steady increase in the incidence of diabetes in the 1989-1996 period (Table 2) is at least consistent with this hypothesis.

1.5. Empirical Results

1.5.1. Medicaid Eligibility

Table 3 shows the effects - expressed as both odds ratios and marginal effects evaluated at the population means - of our Medicaid policy variable on the utilization of prenatal care and maternal health.¹⁰ Table 4 reports the same results from the models estimated with OLS. Note that the marginal effects from the logit and the OLS coefficients are similar in magnitude. The full set of logit results is reported in Table 5 for the summary measure. The general finding in Tables 3 and 4 is that the Medicaid expansions of the 1990's benefited less educated mothers, especially whites. First, as apparent, increases in Medicaid eligibility significantly increase the probability of receiving prenatal care in the first trimester and of receiving adequate or intermediate prenatal care among less educated single women (both black and white; second and third columns of Tables 3 and 4). On the other hand, as hypothesized, the eligibility coefficients are at best marginally significant (blacks) or have the opposite sign (whites) among highly educated mothers. This finding corroborates the results in Currie and Grogger (2002) where women with low socioeconomic status benefited from the

¹⁰ A negative (positive) coefficient suggests the variable decreases (increases) the probability of the outcome, thus yielding an odds ratio of less than (greater than) one. As mentioned earlier, we have used 100% of observations in all but the biggest "white" control cohorts. Recall also that the first column of Tables 3 and 4 shows the cohort-specific "treatment probability" calculated from the CPS.

Medicaid expansions but women with high socioeconomic status did not. When testing for the significance of the differences, we strongly reject the null hypothesis that white “treatment” and “control” women are affected equally. In the linear probability model (Table 4) but not the logit model (Table 3), this is also the case for blacks. Surprisingly, however, the beneficial effect of Medicaid in promoting prenatal care use did not reach statistical significance among low-educated (“less than high school”) married women of either race.

The fourth column of Tables 3 and 4 presents our results for placental abruption. As can be seen, there is no strong evidence of a beneficial effect of the Medicaid eligibility expansions on the incidence of this complication. This is not too surprising given the rarity of the event: in the 1989-1996 period, no more than 1% of women suffered from placental abruption in any of our sub-samples (Table 1).

The results for the other three measures of preventable complications are more supportive of an effect of Medicaid, as the estimated coefficients are universally of the correct sign and in general are the largest in magnitude for the most disadvantaged women. Column five reports encouraging results for anemia: while not significant at conventional levels, all of the Medicaid eligibility coefficients are negative among women in the treatment groups and the sizes of the effects are sometimes substantial (odds ratios between 0.89 and 0.97). The causality of the relationship between anemia and Medicaid is further supported by the fact that neither black nor white highly educated married mothers seem to have been affected by the policy changes (mostly positive coefficients but differences from treatment groups statistically insignificant). These are potentially important findings given that anemia has been a relatively common

complication of pregnancy (over 3% of less educated blacks and close to 2% of less educated whites suffered from anemia in the 1989-1996 period; Table 1).

The results for hypertension in the sixth column of Tables 3 and 4 are even stronger. Again, the eligibility coefficients always have the correct sign among women in the treatment groups. Furthermore, the beneficial effects of Medicaid are highly significant among white women with “less than high school” education (both married and single) and, in the logit model, marginally significant among single black and white mothers with “high school completed”. Among married whites, the effect is significantly different between women in the treatment and control groups.

The results for the summary measure (experiencing “any complication”) are reported in the seventh column of Tables 3 and 4. The Medicaid expansions appear to have reduced the incidence of any of the three maternal complications studied among low-educated (“less than high school”) whites (both married and single; coefficients highly significant) and married blacks (coefficients marginally significant). The effects are sizeable (odds ratios of about 0.88) and among whites (but not blacks) significantly different between women in the treatment and control groups. This is an important result given that 5-7% of women in our sample suffered from at least one of the preventable morbidities in the 1989-1996 period (Table 1).

Finally, the last column of Tables 3 and 4 shows the estimates for diabetes. As hypothesized, Medicaid eligibility has no beneficial effect on the incidence of this “straw man” complication. In fact, the coefficients are positive and statistically significant among white women with at least high school education. This result is consistent with the concept of improved reporting discussed earlier. In particular, since diabetes is not

believed to be preventable by prenatal care and since the Medicaid expansions likely improved monitoring of maternal morbidities, we would expect the effect of Medicaid on the *observed* incidence of diabetes to be positive, if anything.

To get more insight into the reporting bias present in our maternal health estimates, we compare the effects of Medicaid eligibility on preventable maternal complications with the effects on diabetes. Specifically, we test for whether there is a statistically significant difference between the coefficients on each of the preventable complications and the coefficients on diabetes. If all complications share the same reporting bias, our exercise is essentially purging the preventable complications' estimates of this bias. Of course, this is a very strong assumption and so we view our calculations as illustrative only. Also, we caution that our exercise compares *percentage point* changes in the outcomes of interest which limits its applicability in situations where the incidence of maternal complications vastly differs.

Comparison to diabetes leads to statistically significant effects of Medicaid on all of the white treatment groups and for almost every maternal health measure. For hypertension and the summary measure, the 'bias-purged' effects are significantly negative for all four white treatment groups (Tables 3 and 4). For anemia, they are significantly negative for two (Table 3) or three (Table 4) of the treatment cohorts; only less educated married mothers did not significantly benefit, mostly because diabetes did not increase in this group. Among white women with at least "high school completed", even placental abruption now seems to have significantly lower incidence (especially in Table 4).

For blacks, however, the “straw man” exercise has little impact on our conclusions. Since the incidence of diabetes seems generally unaffected by Medicaid, the beneficial effects of Medicaid on preventable complications remain mostly insignificant. Only the strongest (unadjusted) effect we find – the reduction in the incidence of “any complication” among married less educated blacks – maintains its marginal significance after the adjustment (Table 4).

These apparent racial differences in the effects of Medicaid on prenatal care use and maternal health merit further discussion. As our findings suggest, both black and white mothers obtained more adequate prenatal care as a result of the Medicaid expansions. Indeed, in the OLS model, the same treatment groups for both races (i.e., single mothers) experienced improvements that are significantly different from the effects on the corresponding control groups (Table 4). Among treatment whites, these increases in access translated into improved maternal outcomes even among those who did not experience improvements in our measures of prenatal care (i.e., married women). Among blacks, on the other hand, few improvements in maternal health are observed and none are statistically different from the effects on the control group. A further puzzle is that the black cohort most affected in terms of maternal health - less educated married mothers - did *not* experience an increase in our measures of prenatal care.

While the observed racial disparities in the effects of Medicaid on maternal health mentioned above could partly be attributable to lower sample sizes among blacks, they seem inconsistent with the highly statistically significant (and substantial) improvements in the utilization of prenatal care experienced across the races.¹¹ A possible explanation

for this phenomenon is that black women receive lower quality of prenatal care than whites. The role of prenatal care quality has been mentioned in Currie and Grogger (2002), who found strong racial differences in the effects of Medicaid on prenatal care use and infant health. Unfortunately, the quality of prenatal care cannot be investigated using data from the vital statistics. Suggestive evidence, however, can be found in other studies. For example, Kogan et al. (1994) find that pregnant blacks are less likely than pregnant whites to receive advice on cessation of alcohol consumption and smoking cessation even when the timing of prenatal care initiation is controlled for. In a recent paper, Chandra and Skinner (2003) argue that blacks tend to seek care in areas where quality levels for all patients (black and white) are lower.

A different explanation for the observed racial disparities can be found in Geronimus and Bound (1990). In particular, the authors' main argument is that the health of black women deteriorates with age more rapidly than the health of white women and that this can be attributed to a cumulative effect of poor medical care among blacks. If so, black women may have more pre-existing morbidities when they reach their childbearing age which can make it more difficult for prenatal care providers to intervene. Using hospital discharge data from South Carolina, Laditka et al. (2005b) find disparities in the incidence of potentially preventable maternal complications between black and white mothers enrolled in Medicaid. Interestingly, these racial disparities are eliminated once socio-economic characteristics and comorbidities are controlled for. Future research should reconcile the above findings.

¹¹ Racial disparities of a similar sort have been observed elsewhere. For example, Decker and Rapaport (2002) show that becoming eligible for Medicare at the age of 65 increases the chances of receiving mammography among low-educated blacks and whites but is associated with improvements in the stage of breast cancer diagnosis only among whites.

1.5.2. Sensitivity Checks

In order to test the robustness of our results, we conduct several sensitivity checks. First, we re-estimate our logit models excluding 1989 from the analysis (Table A1 in the Appendix). 1989 is the first year when maternal complications were recorded in the Natality Detail Files and we want to verify that the adoption of new birth certificates did not somehow contaminate our findings. In addition, information on maternal health outcomes is missing for three states – Louisiana, Nebraska, and Oklahoma – in the year 1989. Limiting the period studied to 1990-1996 leaves the results qualitatively unchanged.

Second, we redefine prenatal care adequacy as receiving “adequate” (as opposed to “adequate” or “intermediate”) prenatal care (Table A2 in the Appendix). The results remain qualitatively the same. This is also true if we exclude women with “no” or “unknown” prenatal care utilization from the analysis (Table A3 in the Appendix). Finally, drawing different random samples (1/3 of all births) from the control white population has little influence on our estimates.

1.5.3. Other Results

What factors besides Medicaid affect maternal health? In Table 5, we report the full set of results (except for state and year dummy coefficients) for the incidence of “any complication” estimated with logit. (The full set of results from the other models are available upon request.) Among white treatment women, welfare surprisingly seems to modestly increase the incidence of pregnancy complications. This result probably reflects

the positive association between poverty and health care need. The effects of unemployment are insignificant.

As expected, the incidence of “any complication” first decreases (until the mid to late 20s) and then increases with maternal age.¹² Controlling for age, parity generally decreases the probability of complications. And, finally, having a male infant is associated with more complications among whites but with fewer complications among blacks. This finding may be attributable to a differential effect of infant gender on the incidence of specific morbidities. In particular, our unreported results suggest that male infants are associated with a higher incidence of hypertension (at least among whites) and with a lower incidence of anemia (among both blacks and whites).

1.6. Concluding Remarks

Overall, our results suggest that there was an additional beneficiary of the Medicaid expansions of the 1990’s – the mother. Specifically, the eligibility changes lead to a higher utilization of prenatal care and fewer preventable maternal health complications among those women (i.e., economically disadvantaged) most likely to have benefited from the expansions. While the increases in prenatal care access seem similar across the races, however, our findings indicate that the improvements in maternal health were mostly concentrated among whites.

To get a better idea about the magnitude of the estimated health effects, consider an example of California in the year 1989. In the early 1990’s, California experienced an increase in the Medicaid eligibility threshold from 109 to 185 percent of the federal

¹² Recall the U-shape relationship between maternal age and hypertension noted in the 2003 CDC report.

poverty line (this was one of the largest percentage point increases nationally). Based on our (statistically significant) results, such an increase would be associated with a decline in the odds of hypertension of:

12.9% among married whites with “less than high school” education,

10.6% among single whites with “less than high school” education,

5.3% among single whites with “high school completed”, and

7.6% among single blacks with “high school completed”.

Similarly, the Californian expansion would cause a decline in the odds of “any complication” of:

9.1% among married whites with “less than high school” education,

9.9% among married blacks with “less than high school” education, and

8.4% among single whites with “less than high school” education.

Given the costs of pregnancy complications to the mother and society, these are not negligible improvements.

To better understand the link between Medicaid eligibility and maternal health, future research could consider a wider range of maternal health outcomes, such as other types of maternal complications of pregnancy/delivery and more “subtle” measures of maternal health (mothers’ weight status postpartum, for example) and women’s general health behaviors (such as maternal smoking). Another promising extension is to investigate the possible endogeneity of both marital status (Yelowitz 1998) and fertility (Bitler and Zavodny 2004).

Finally, the results of our research reveal that maternal health improved among some disadvantaged mothers who did not experience an apparent change in the timing

and/or adequacy of prenatal care. Conversely, other women (such as single blacks) experienced an increase in prenatal care access but failed to experience improved maternal health. These findings beg the question of what other channels exist through which Medicaid eligibility actually affects maternal health. For example, Currie and Gruber (1997, 2001) demonstrate that the eligibility expansions affect the treatments and procedures received by women during their pregnancy and delivery. The question remains, however, whether and how these changes translate into improvements in maternal outcomes. According to our findings, the potential of public policies to improve the health status of disadvantaged pregnant women may be large.

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Figure 1.

Medicaid Eligibility Threshold, 1988-1996
Minimum, Maximum, and Average

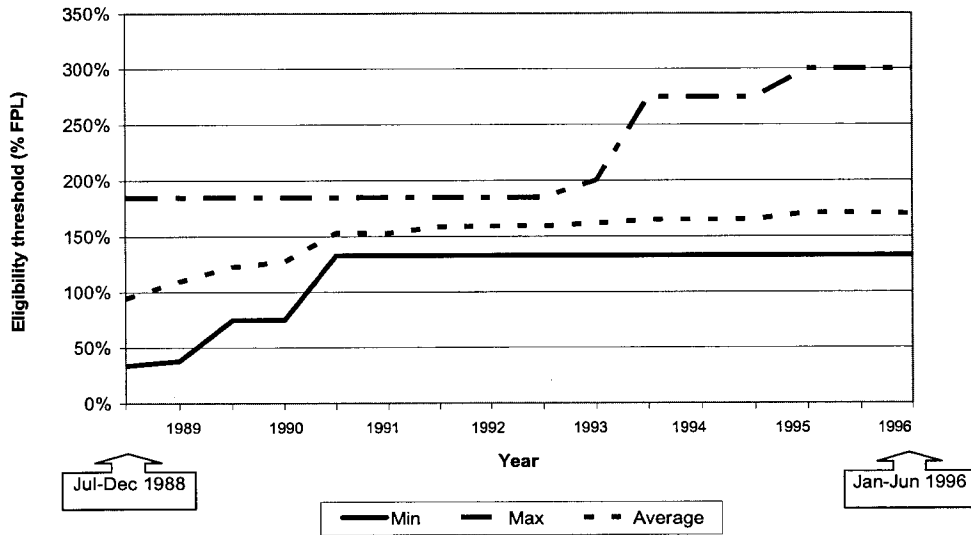
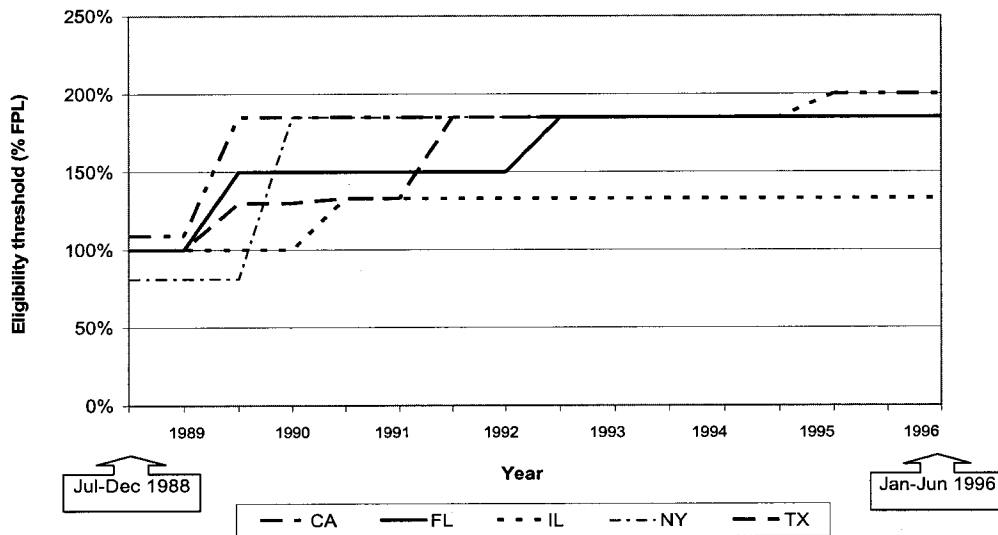


Figure 2.

Medicaid Eligibility Threshold, 1988-1996
Five Largest States



**Table 1. Descriptive Statistics
1989-1996 Births**

	Blacks					Whites				
	Treatment group #1 (less than high school, married)	Treatment group #2 (less than high school, single)	Treatment group #3 (high school completed, single)	Treatment group #4 (some college, single)	Control group (college completed, married)	Treatment group #1 (less than high school, married)	Treatment group #2 (less than high school, single)	Treatment group #3 (high school completed, single)	Treatment group #4 (some college, single)	Control group (college completed, married)
# of observations	134,196	619,946	1,204,987	495,896	278,471	1,110,384	723,699	1,341,567	533,435	4,412,467
Placental abruption (%)	0.81	0.86	0.74	0.68	0.55	0.79	0.89	0.77	0.70	0.48
Anemia (%)	3.49	3.86	3.41	3.21	2.48	1.93	2.23	1.86	1.80	1.14
Hypertension (%)	2.45	2.04	2.80	3.33	3.62	2.47	2.29	3.35	3.70	2.95
Any complication (%)	6.56	6.56	6.73	7.02	6.48	5.08	5.27	5.84	6.06	4.49
Diabetes (%)	3.21	1.54	1.83	2.23	3.51	2.57	1.92	2.15	2.33	2.34
PNC in first trimester (%)	62.51	53.11	61.70	69.07	90.18	70.86	63.46	70.37	74.35	95.15
Adequate/intermed. PNC (%)	84.21	75.47	84.50	89.55	97.59	90.22	86.94	91.40	92.67	99.04
Age (years)	27.00	24.04	24.50	25.57	30.82	24.90	23.45	24.46	25.67	31.00
Parity (# of live births)	3.44	3.07	2.31	1.96	1.90	2.58	2.25	1.78	1.66	1.83
Male infant	50.75	50.61	50.75	50.89	50.65	51.25	51.11	51.35	51.26	51.43
Medicaid eligibility* (% of FPL/100)	1.55	1.54	1.55	1.57	1.58	1.50	1.54	1.57	1.60	1.58
Welfare caseload* (% on welfare)	4.90	5.10	5.05	5.16	4.96	4.69	4.85	4.89	4.93	4.83
Unemployment rate*	6.35	6.30	6.25	6.30	6.20	6.13	6.13	6.09	6.09	6.07

* Medicaid eligibility, welfare caseload, and unemployment rate are state-level explanatory variables.

Table 2. Trends in Prenatal Care Use and Maternal Health

	1989	1990	1991	1992	1993	1994	1995	1996
Treatment group #1 (less than high school, married) – Black								
PNC in first trimester	57.92	58.26	59.90	60.89	63.52	65.95	68.60	69.63
Placental abruption	1.06	0.73	0.73	0.77	0.74	0.82	0.78	0.89
Anemia	3.65	3.51	3.39	3.51	3.27	3.69	3.59	3.29
Hypertension	2.55	2.36	2.32	2.12	2.27	2.57	2.72	2.97
Diabetes	2.81	2.75	2.79	3.28	3.37	3.46	3.65	3.88
Treatment group #2 (less than high school, single) – Black								
PNC in first trimester	48.18	48.98	50.39	51.11	53.37	56.06	57.93	59.78
Placental abruption	0.93	0.87	0.86	0.85	0.83	0.87	0.81	0.85
Anemia	4.16	3.91	3.61	3.47	3.61	4.05	4.06	4.13
Hypertension	2.02	1.85	1.72	1.71	1.87	2.25	2.33	2.69
Diabetes	1.34	1.23	1.34	1.46	1.76	1.69	1.68	1.84
Treatment group #3 (high school completed, single) – Black								
PNC in first trimester	56.10	56.74	58.35	60.05	62.30	65.01	67.16	68.24
Placental abruption	0.82	0.74	0.69	0.72	0.73	0.71	0.75	0.74
Anemia	3.76	3.41	3.34	3.11	3.23	3.55	3.58	3.39
Hypertension	2.67	2.51	2.47	2.49	2.61	2.91	3.27	3.53
Diabetes	1.41	1.40	1.60	1.87	1.93	2.08	2.08	2.18
Treatment group #4 (some college, single) – Black								
PNC in first trimester	62.96	63.95	65.42	67.43	69.44	71.77	73.42	74.48
Placental abruption	0.74	0.77	0.73	0.69	0.67	0.64	0.63	0.65
Anemia	3.38	3.13	3.19	2.99	3.07	3.27	3.44	3.23
Hypertension	3.17	2.93	2.91	3.03	3.18	3.51	3.67	3.96
Diabetes	1.78	1.71	2.02	2.21	2.43	2.30	2.45	2.61
Control group (college completed, married) – Black								
PNC in first trimester	88.40	89.09	89.71	89.73	90.33	90.59	91.26	91.64
Placental abruption	0.67	0.70	0.51	0.50	0.55	0.51	0.50	0.54
Anemia	2.67	2.51	2.35	2.35	2.50	2.36	2.66	2.49
Hypertension	3.43	3.62	3.32	3.30	3.52	3.65	3.99	3.98
Diabetes	3.15	3.20	3.42	3.65	3.73	3.59	3.52	3.66
Treatment group #1 (less than high school, married) – White								
PNC in first trimester	67.30	68.24	68.89	71.07	72.21	73.46	74.29	74.49
Placental abruption	0.84	0.80	0.77	0.80	0.75	0.77	0.78	0.79
Anemia	2.01	1.82	1.85	1.87	1.96	2.03	2.05	1.94
Hypertension	2.28	2.23	2.24	2.33	2.51	2.58	2.87	3.07
Diabetes	2.14	2.21	2.47	2.77	2.87	2.72	2.76	2.86
Treatment group #2 (less than high school, single) – White								
PNC in first trimester	56.22	57.53	60.28	63.12	65.30	67.09	68.09	69.27
Placental abruption	0.94	0.94	0.87	0.87	0.89	0.83	0.88	0.89
Anemia	2.39	2.14	2.13	2.11	2.11	2.33	2.36	2.28
Hypertension	2.17	1.92	1.95	2.12	2.19	2.55	2.64	2.76
Diabetes	1.53	1.57	1.77	2.12	2.08	2.08	2.04	2.08
Treatment group #3 (high school completed, single) – White								
PNC in first trimester	63.36	64.78	66.86	69.60	71.58	73.26	74.50	75.44
Placental abruption	0.90	0.77	0.78	0.75	0.76	0.77	0.72	0.72
Anemia	1.81	1.70	1.75	1.83	1.78	1.97	2.02	1.98

Hypertension	3.03	2.92	2.92	3.14	3.30	3.55	3.74	3.92
Diabetes	1.73	1.78	2.09	2.26	2.32	2.28	2.23	2.29
Treatment group #4 (some college, single) – White								
PNC in first trimester	67.29	68.89	71.00	73.21	74.92	76.50	77.93	78.56
Placental abruption	0.74	0.77	0.65	0.72	0.74	0.71	0.68	0.66
Anemia	1.78	1.58	1.61	1.68	1.76	1.94	1.92	1.93
Hypertension	3.33	3.22	3.15	3.52	3.54	3.92	4.06	4.29
Diabetes	1.92	1.92	2.35	2.52	2.45	2.40	2.35	2.46
Control group (college completed, married) – White								
PNC in first trimester	94.44	94.83	94.93	95.18	95.24	95.41	95.50	95.39
Placental abruption	0.55	0.52	0.48	0.47	0.47	0.47	0.46	0.46
Anemia	0.95	0.99	1.08	1.15	1.11	1.22	1.29	1.24
Hypertension	2.71	2.59	2.64	2.85	2.96	3.10	3.16	3.37
Diabetes	2.19	2.19	2.36	2.63	2.46	2.34	2.23	2.31

**Table 3. The Effects of Medicaid Eligibility Rules on PNC Use and Maternal Health
Odds Ratios and Marginal Effects from a Logit; 1989-1996**

Cohort	Treatment probability	PNC in first trimester	Adequate/Intermed. PNC	Placental abruption	Anemia	Hypertension	Any complication	Diabetes
Treatment group #1 (less than high school, married) – Black	17.51	1.00 [-0.001] (-0.05)	1.04 [0.005] (0.77)	1.09 [0.001] (0.49)	0.89 [-0.003] (-1.04)	0.84 [-0.004] (-1.58)	0.87* [-0.008] (-1.92)	1.06 [0.001] (0.55)
Treatment group #2 (less than high school, single) – Black	4.32^	1.18*** [0.041] (4.16)	1.16*** [0.024] (3.50)	0.95 [-0.000] (-0.48)	0.89 [-0.004] (-1.24)	0.92 [-0.002] (-0.86)	0.90 [-0.006] (-1.37)	0.90 [-0.002] (-1.17)
Treatment group #3 (high school completed, single) – Black	1.74	1.16*** [0.034] (4.09)	1.19*** [0.021] (4.29)	0.99 [-0.000] (-0.15)	0.93 [-0.002] (-1.01)	0.90* [-0.002] (-1.69)	0.93 [-0.004] (-1.34)	0.96 [-0.001] (-0.68)
Treatment group #4 (some college, single) – Black	3.14	1.15*** [0.031] (2.92)	1.20*** [0.019] (3.52)	0.91 [-0.001] (-0.82)	0.97 [-0.001] (-0.40)	0.90 [-0.003] (-1.25)	0.94 [-0.004] (-0.96)	0.93 [-0.001] (-1.00)
Control group (college completed, married) – Black	-1.36	1.06 [0.008] (0.87)	1.22* [0.008] (1.95)	0.85 [-0.001] (-0.93)	1.07 [0.002] (0.64)	0.94 [-0.001] (-0.75)	0.98 [-0.001] (-0.30)	0.98 [-0.000] (-0.20)
Treatment group #1 (less than high school, married) – White	7.73	1.00 [0.000] (0.06)	1.01 [0.000] (0.09)	1.02 [0.000] (0.30)	0.90 [-0.001] (-1.45)	0.83*** ^b [-0.005] (-4.28)	0.88*** ^a [-0.006] (-3.72)	0.97 [-0.001] (-0.62)
Treatment group #2 (less than high school, single) – White	13.24	1.14*** [0.029] (3.11)	1.17*** [0.014] (2.97)	0.92 [-0.001] (-0.95)	0.91 [-0.002] (-1.28)	0.86** ^b [-0.004] (-2.36)	0.89** ^b [-0.006] (-2.46)	1.07 [0.002] (1.00)
Treatment group #3 (high school completed, single) – White	8.17	1.11*** [0.020] (3.33)	1.16*** [0.011] (3.59)	1.02 ^b [0.000] (0.37)	0.93 ^c [-0.001] (-1.10)	0.93* ^c [-0.002] (-1.93)	0.95 ^c [-0.003] (-1.59)	1.20*** [0.005] (3.94)
Treatment group #4 (some college, single) – White	4.65	1.12*** [0.021] (3.15)	1.11** [0.007] (2.12)	0.99 [-0.000] (-0.10)	0.92 ^b [-0.001] (-0.99)	0.95 ^b [-0.001] (-0.85)	0.96 ^b [-0.002] (-0.98)	1.16** [0.004] (2.20)
Control group (college completed, married) – White	0.79	0.97 [-0.001] (-0.56)	0.87* [-0.002] (-1.74)	0.98 [-0.000] (-0.27)	1.08 [0.001] (0.83)	0.99 ^a [-0.000] (-0.25)	1.01 ^a [0.000] (0.17)	1.11** [0.002] (2.35)

Notes to Table 3:

The first row in each cell reports the odds ratio. Marginal effects evaluated at population means are given in square brackets.

*, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively.

Standard errors have been adjusted for clustering at the state/year level. T-statistics are given in parentheses.

“Treatment probability” has been calculated from CPS according to the following formula: $\text{treatment probability} = 100 * (\% \text{ covered by Medicaid in 1996} - \% \text{ covered by Medicaid in 1989}) / (100 - \% \text{ covered by Medicaid in 1989})$. Most substantial increases are bolded (see note ^ below).

^ Unmarried black women with less than high school education experienced a decline in the % on AFDC of about 11 percentage points (in contrast to a modest *increase* in most other groups). Thus, Medicaid expansions above and beyond welfare had to be especially strong in this cohort in order to offset the negative effect of the AFDC contraction.

All coefficients have been compared between treatment and control cohorts. Cells with a difference significant at the 95% or 99% confidence level are shaded.

Coefficients on placental abruption, anemia, hypertension, and ‘any complication’ have been compared to coefficients on diabetes within cohorts. ^a, ^b, and ^c indicate differences statistically significance at the 90%, 95%, and 99% confidence levels, respectively.

Table 4. The Effects of Medicaid Eligibility Rules on PNC Use and Maternal Health
Coefficients from a Linear Probability Model; 1989-1996

Cohort	Treatment probability	PNC in first trimester	Adequate/ Intermed. PNC	Placental abruption	Anemia	Hypertension	Any complication	Diabetes
Treatment group #1 (less than high school, married) – Black	17.51	0.000 (0.04)	0.011 (1.43)	0.001 (0.74)	-0.004 (-1.15)	-0.004 (-1.48)	-0.009* ^a (-1.87)	0.002 (0.72)
Treatment group #2 (less than high school, single) – Black	4.32[^]	0.040*** (4.21)	0.033*** (4.01)	-0.000 (-0.45)	-0.005 (-1.28)	-0.001 (-0.52)	-0.006 (-1.30)	-0.001 (-0.88)
Treatment group #3 (high school completed, single) – Black	1.74	0.036*** (4.45)	0.029*** (4.97)	-0.000 (-0.04)	-0.003 (-1.08)	-0.002 (-1.17)	-0.004 (-1.25)	-0.000 (-0.13)
Treatment group #4 (some college, single) – Black	3.14	0.033*** (3.26)	0.023*** (4.00)	-0.001 (-0.90)	-0.001 (-0.40)	-0.003 (-0.97)	-0.004 (-0.94)	-0.001 (-0.72)
Control group (college completed, married) – Black	-1.36	0.004 (0.74)	0.005** (1.99)	-0.001 (-0.95)	0.001 (0.52)	-0.002 (-0.76)	-0.002 (-0.42)	-0.000 (-0.11)
Treatment group #1 (less than high school, married) – White	7.73	0.004 (0.46)	0.005 (0.95)	0.000 (0.43)	-0.002 (-1.25)	-0.004***^b (-3.81)	-0.005***^b (-3.27)	-0.001 (-0.81)
Treatment group #2 (less than high school, single) – White	13.24	0.032*** (3.23)	0.022*** (3.36)	-0.001 (-0.96)	-0.002 ^a (-1.33)	-0.003* ^b (-1.88)	-0.005**^b (-2.19)	0.001 (1.02)
Treatment group #3 (high school completed, single) – White	8.17	0.023*** (3.58)	0.015*** (4.07)	0.000 ^c (0.59)	-0.001 ^c (-1.10)	-0.002 ^c (-1.42)	-0.002 ^c (-1.25)	0.004*** (3.82)
Treatment group #4 (some college, single) – White	4.65	0.024*** (3.44)	0.012*** (2.92)	-0.000 ^b (-0.12)	-0.001 ^b (-0.98)	-0.001 ^a (-0.54)	-0.002 ^a (-0.76)	0.003** (2.27)
Control group (college completed, married) – White	0.79	-0.001 (-0.50)	-0.001 (-1.52)	-0.000 ^b (-0.18)	0.000 (0.46)	0.000 (0.10)	0.000 (0.29)	0.002** (2.41)

*, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively.

Standard errors have been adjusted for clustering at the state/year level. T-statistics are given in parentheses.

“Treatment probability” has been calculated from CPS according to the following formula: treatment probability = 100*(% covered by Medicaid in 1996-% covered by Medicaid in 1989)/(100-% covered by Medicaid in 1989). Most substantial increases are bolded (see note [^] below).

[^] Unmarried black women with less than high school education experienced a decline in the % on AFDC of about 11 percentage points (in contrast to a modest *increase* in most other groups). Thus, Medicaid expansions above and beyond welfare had to be especially strong in this cohort in order to offset the negative effect of the AFDC contraction.

All coefficients have been compared between treatment and control cohorts. Cells with a difference significant at the 95% or 99% confidence level are shaded.

Coefficients on placental abruption, anemia, hypertension, and ‘any complication’ have been compared to coefficients on diabetes within cohorts. ^a, ^b, and ^c indicate differences statistically significance at the 90%, 95%, and 99% confidence levels, respectively.

Table 5. Any Complication
Odds Ratios from a Logit; 1989-1996

	Blacks					Whites				
	Treatment group #1 (less than high school, married)	Treatment group #2 (less than high school, single)	Treatment group #3 (high school completed, single)	Treatment group #4 (some college, single)	Control group (college completed, married)	Treatment group #1 (less than high school, married)	Treatment group #2 (less than high school, single)	Treatment group #3 (high school completed, single)	Treatment group #4 (some college, single)	Control group (college completed, married)
Medicaid eligibility	0.87* (-1.92)	0.90 (-1.37)	0.93 (-1.34)	0.94 (-0.96)	0.98 (-0.30)	0.88*** (-3.72)	0.89** (-2.46)	0.95 (-1.59)	0.96 (-0.98)	1.01 (0.17)
Welfare caseload	1.00 (0.03)	0.98 (-0.82)	0.97 (-1.12)	0.95* (-1.78)	0.96 (-1.20)	1.05*** (2.71)	0.99 (-0.46)	1.03** (2.12)	1.06*** (3.58)	1.02 (1.06)
Unemployment rate	1.00 (-0.03)	1.00 (-0.08)	1.00 (0.01)	1.01 (0.52)	1.01 (0.47)	1.00 (0.11)	1.01 (0.63)	1.01 (0.80)	0.99 (-0.83)	1.00 (0.35)
Age	0.87*** (-8.67)	0.91*** (-10.02)	0.91*** (-14.77)	0.95** (-4.73)	0.87*** (-7.62)	0.88*** (-17.09)	0.88*** (-12.36)	0.89*** (-15.90)	0.91*** (-9.62)	0.79*** (-20.19)
Age squared	1.00*** (8.97)	1.00*** (9.60)	1.00*** (15.03)	1.00*** (5.62)	1.00*** (8.58)	1.00*** (19.55)	1.00*** (13.64)	1.00*** (17.10)	1.00*** (10.87)	1.00*** (21.47)
Parity	1.01 (1.57)	1.01 (1.59)	0.97*** (-5.34)	0.95*** (-6.47)	0.89*** (-11.32)	0.91*** (-19.44)	0.93*** (-10.60)	0.86*** (-22.83)	0.87*** (-14.67)	0.75*** (-35.80)
Male infant	0.97 (-1.28)	0.96*** (-3.47)	0.98*** (-2.82)	0.96*** (-3.19)	0.96*** (-2.69)	1.03*** (3.82)	1.04*** (3.87)	1.06*** (7.08)	1.03** (2.43)	1.02** (2.19)

*, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively.
Standard errors have been adjusted for clustering at the state/year level. T-statistics are given in parentheses.

Appendix

Table A1. The Effects of Medicaid Eligibility Rules on PNC Use and Maternal Health Odds Ratios from a Logit; 1990-1996

Cohort	PNC in first trimester	Adequate/ Intermed. PNC	Placental abruption	Anemia	Hypertension	Any complication	Diabetes
Treatment group #1 (less than high school, married) – Black	0.99 (-0.15)	1.01 (0.14)	0.89 (-0.53)	0.89 (-0.92)	0.78* (-1.85)	0.82** (-2.51)	1.04 (0.29)
Treatment group #2 (less than high school, single) – Black	1.19*** (3.56)	1.11** (2.20)	0.86 (-1.41)	0.95 (-0.48)	0.93 (-0.67)	0.93 (-0.91)	0.97 (-0.28)
Treatment group #3 (high school completed, single) – Black	1.17*** (3.66)	1.15*** (3.11)	0.91 (-1.41)	0.92 (-0.91)	0.94 (-0.98)	0.93 (-1.17)	0.97 (-0.41)
Treatment group #4 (some college, single) – Black	1.19*** (2.96)	1.19*** (2.63)	0.87 (-0.94)	0.91 (-0.98)	0.89 (-1.25)	0.91 (-1.45)	0.97 (-0.29)
Control group (college completed, married) – Black	1.11 (1.40)	1.39*** (2.98)	0.77 (-1.16)	1.05 (0.42)	0.88 (-1.38)	0.92 (-0.96)	0.98 (-0.22)
Treatment group #1 (less than high school, married) – White	1.05 (0.69)	1.03 (0.33)	0.99 (-0.12)	0.93 (-0.87)	0.81*** (-4.17)	0.87*** (-3.36)	1.00 (-0.03)
Treatment group #2 (less than high school, single) – White	1.21*** (3.87)	1.20*** (2.72)	0.82* (-1.96)	0.86* (-1.82)	0.88* (-1.87)	0.85*** (-3.10)	1.15* (1.73)
Treatment group #3 (high school completed, single) – White	1.12*** (3.02)	1.17*** (3.05)	1.03 (0.48)	0.89 (-1.60)	0.95 (-1.13)	0.95 (-1.49)	1.23*** (3.82)
Treatment group #4 (some college, single) – White	1.15*** (3.40)	1.12** (2.01)	0.99 (-0.09)	0.92 (-0.85)	0.98 (-0.43)	0.97 (-0.74)	1.17** (2.08)
Control group (college completed, married) – White	0.98 (-0.42)	0.83* (-1.88)	0.96 (-0.49)	1.17 (1.51)	1.01 (0.14)	1.03 (0.75)	1.11* (1.94)

*, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. Standard errors have been adjusted for clustering at the state/year level. T-statistics are given in parentheses.

**Table A2. The Effects of Medicaid Eligibility Rules on PNC Adequacy
Odds Ratios from a Logit; 1989-1996**

Cohort	Adequate/ Intermediate PNC	Adequate PNC
Treatment group #1 (less than high school, married) – Black	1.04 (0.77)	1.00 (-0.07)
Treatment group #2 (less than high school, single) – Black	1.16*** (3.50)	1.17*** (4.12)
Treatment group #3 (high school completed, single) – Black	1.19*** (4.29)	1.12*** (3.23)
Treatment group #4 (some college, single) – Black	1.20*** (3.52)	1.09** (2.00)
Control group (college completed, married) – Black	1.22* (1.95)	0.95 (-0.98)
Treatment group #1 (less than high school, married) – White	1.01 (0.09)	0.99 (-0.21)
Treatment group #2 (less than high school, single) – White	1.17*** (2.97)	1.11*** (2.73)
Treatment group #3 (high school completed, single) – White	1.16*** (3.59)	1.06** (2.05)
Treatment group #4 (some college, single) – White	1.11** (2.12)	1.03 (0.86)
Control group (college completed, married) – White	0.87* (-1.74)	0.84*** (-3.56)

*, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. Standard errors have been adjusted for clustering at the state/year level. T-statistics are given in parentheses.

**Table A3. The Effects of Medicaid Eligibility Rules on PNC Use and Maternal Health
Odds Ratios from a Logit; 1989-1996; PNC Users**

Cohort	PNC in first trimester	Adequate/ Intermed. PNC	Placental abruption	Anemia	Hypertension	Any complication	Diabetes
Treatment group #1 (less than high school, married) – Black	0.95 (-1.02)	0.94 (-1.00)	1.17 (0.84)	0.89 (-1.11)	0.84 (-1.56)	0.87* (-1.84)	1.04 (0.36)
Treatment group #2 (less than high school, single) – Black	1.17*** (4.35)	1.15*** (3.41)	N/A	0.89 (-1.30)	0.92 (-0.85)	0.90 (-1.44)	0.90 (-1.16)
Treatment group #3 (high school completed, single) – Black	1.14*** (3.80)	1.16*** (3.65)	0.95 (-0.65)	0.94 (-0.93)	0.90* (-1.78)	0.93 (-1.39)	0.95 (-0.79)
Treatment group #4 (some college, single) – Black	1.13** (2.58)	1.16*** (2.72)	0.93 (-0.65)	0.96 (-0.43)	0.90 (-1.29)	0.94 (-0.99)	0.93 (-0.94)
Control group (college completed, married) – Black	1.02 (0.28)	1.06 (0.48)	0.87 (-0.79)	1.07 (0.65)	0.95 (-0.73)	0.98 (-0.24)	0.98 (-0.24)
Treatment group #1 (less than high school, married) – White	0.99 (-0.21)	0.97 (-0.49)	1.00 (0.06)	0.90 (-1.49)	0.83*** (-4.20)	0.88*** (-3.83)	0.97 (-0.70)
Treatment group #2 (less than high school, single) – White	1.13*** (3.01)	1.15*** (2.77)	0.94 (-0.64)	0.89 (-1.45)	0.88** (-2.12)	0.89** (-2.43)	1.07 (1.02)
Treatment group #3 (high school completed, single) – White	1.10*** (3.15)	1.14*** (3.25)	1.03 (0.50)	0.93 (-1.03)	0.93* (-1.90)	0.95 (-1.47)	1.20*** (3.89)
Treatment group #4 (some college, single) – White	1.12*** (3.15)	1.11** (2.04)	1.02 (0.16)	0.94 (-0.66)	0.96 (-0.73)	0.96 (-0.78)	1.15** (2.06)
Control group (college completed, married) – White	1.01 (0.31)	0.86* (-1.80)	1.00 (-0.00)	1.10 (0.94)	1.00 (0.13)	1.03 (0.96)	1.08* (1.69)

*, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively.
Standard errors have been adjusted for clustering at the state/year level. T-statistics are given in parentheses.

2. THE EFFECTS OF UNEMPLOYMENT ON CHILDBEARING

2.1. Background

Are recessions good for your health? At the end of the 20th century, the simple answer for an average American seems to be: Yes (Ruhm 2000). But what about the traditionally vulnerable groups? In this paper, my goal is to contribute to our understanding of the relationship between economic fluctuations and health by asking a more targeted question: Are recessions good for your pregnancy? Focusing on prenatal care utilization, infant and maternal health, I find the answer to be: Yes, overall, but... In particular, analyzing the US Natality Detail Files data for the 1990's aggregated by county, year, and race, I conclude that at least some of the overall apparent benefits of unemployment may be attributable to the Medicaid 'safety net'.

The relationship between macro-level unemployment fluctuations and health has recently received increased attention in the economics literature. Ruhm (2000, 2003) finds that the general health status - as measured either by cause-specific mortality rates (including infant and neonatal mortality) or by more subtle measures (such as activity limitations and the use of medical care) - improves during temporary economic downturns. Ruhm (2000, 2005) observes that health-related behavior improves during recessions. The author explains his findings by the cyclical fluctuations in non-market time and the related time costs of health-producing activities.

Recently, Dehejia and Lleras-Muney (2004) report reduced incidence of low birth weight and very low birth weight and lower neonatal and post-neonatal mortality rates in times of higher unemployment. In the same paper, they also find a positive impact of unemployment on prenatal care use. In line with Ruhm's reasoning (Ruhm 2000, 2003, 2005), a possible explanation for this phenomenon is that in times of higher

unemployment women face looser time constraints, which increases their ability to obtain appropriate prenatal care. In the case of behaviors and health outcomes related to pregnancy, however, the observed aggregate effects of unemployment may also mask compositional changes. Specifically, as stressed in Dehejia and Lleras-Muney (2004), selection through fertility decisions may play an important role. If, for example, only the most affluent women decide to conceive during temporary economic downturns, the average prenatal care utilization and health outcomes will likely improve.

Importantly, another reason why women in depressed labor markets may get more appropriate medical care is that unemployment makes many of them eligible for Medicaid. For women in low-skilled jobs (which often do not offer employer-sponsored health insurance coverage), eligibility for Medicaid can significantly improve access to prenatal care. While the effects of increased Medicaid eligibility on prenatal care utilization and health outcomes have been studied, the interplay of unemployment and Medicaid in the production of health has not systematically been investigated. Therefore, in the current paper, my goal is to study how the interaction between unemployment changes and Medicaid eligibility affects childbearing.

I use the Natality Detail Files data for years 1989-1999 aggregated to county/year/race cells and estimate the effects of unemployment – overall, direct (‘unemployment per se’), and indirect (through increased Medicaid eligibility) – on prenatal care utilization and health outcomes. By conducting the analysis at the county level rather than by state (as previous research has done), I am able to construct better proxies for the actual economic conditions facing pregnant women. Since unemployment varies greatly within states and local labor market conditions are important for Medicaid

eligibility (to be discussed shortly), a county-level analysis seems superior. In some of my sensitivity analyses, however, I use state-level cells and unemployment rates for comparison purposes.

As in Dehejia and Lleras-Muney (2004), I employ several measures of prenatal care use and infant health. In addition, I study the effects of unemployment on a key but overlooked outcome -- *maternal* complications of pregnancy and delivery. My econometric analysis indicates that, overall, higher unemployment at the county level is associated with improved infant outcomes especially among whites. Among blacks, unemployment increases prenatal care utilization (and potentially improves maternal health). In some cases, both unemployment per se and unemployment interacted with Medicaid eligibility seem to contribute to the beneficial effects. In others, the Medicaid 'safety net' acts to mitigate, completely offset, or outweigh detrimental effects of unemployment.

2.2. Framework

Changes in unemployment lead to changes in resources available to women of reproductive age. This, in turn, has consequences for selection into pregnancy as well as for behavior and health while pregnant. Figure 1 lays out these two paths (columns) and the various ways these two paths may be affected by unemployment.

First, as unemployment increases, the average wage income and fringe benefits (such as private health insurance) decrease. According to the standard economic theory of fertility (Becker 1960), this will lead to a reduction in birth rates (upper left cell of Figure 1). In perfect markets, the negative income effect will only demonstrate itself in

the presence of long-run unemployment changes (because permanent rather than transitory income determines fertility). As Dehejia and Lleras-Muney (2004) argue, however, the existence of credit constraints may lead to significant fertility reactions even in the short run. Further, in response to the negative income shock, prenatal care utilization (a normal good) will fall among those pregnant (upper right cell of Figure 1). The impacts on health outcomes will depend on the relative magnitudes of the income effects for healthy versus unhealthy behaviors (Ruhm 2000, Dehejia and Lleras-Muney 2004). Ruhm (2005) estimates that the pure income effect of unemployment on health-related behavior is weak and Ruhm (2003) associates the negative income shock with an increase in medical problems and activity limitations.

Second, higher unemployment leads to more leisure (or non-work) time. As the opportunity costs fall, the demand for children (and thus fertility) will increase (Becker 1965; middle left cell of Figure 1). As Dehejia and Lleras-Muney (2004) point out, the substitution effect – moving from labor market to childbearing – will be especially strong among women whose human capital depreciates slowly. For women who have decided to become pregnant, the demand for time-intensive activities (such as regular prenatal care visits and exercise) will increase (Becker 1965, Ruhm 2000; middle right cell of Figure 1). In an empirical analysis, Ruhm (2005) finds support for this hypothesis and associates the non-market time available during times of higher unemployment with improvements in health-related behavior. Once again, however, the effects on health (conditional on becoming pregnant) will theoretically be ambiguous.

I contribute to this conceptual framework by adding a third row to Figure 1 -- the possible role that Medicaid plays in enforcing or mitigating the effects of unemployment.

Theoretically, the effect of unemployment on Medicaid enrollment is ambiguous. On the one hand, lower incomes in times of higher unemployment will qualify additional women for the receipt of Medicaid. On the other, higher unemployment may lead to fiscal pressures and budget cuts (Cawley and Simon 2005). In empirical studies, Holahan and Garrett (2001) calculate that a 1 percentage point increase in unemployment will lead to an increase in Medicaid enrollment by 1.5 million¹³ and Cawley and Simon (2005) also find Medicaid enrollment to be counter-cyclical. These two studies reveal the potential for a Medicaid 'safety net' effect.

Changes in Medicaid eligibility may affect both selection into pregnancy and circumstances during pregnancy (conditional on becoming pregnant). In a recent study, Bitler and Zavodny (2004) show that the effects of Medicaid on fertility are theoretically ambiguous since Medicaid gives pro-natalist incentives by lowering the costs of prenatal and infant medical care but also potentially anti-natalist incentives by funding abortions in some states (lower left cell of Figure 1). In an empirical analysis, the authors find the pro-natalist effects to dominate (especially among low-educated, single, and white women). In theory, higher Medicaid eligibility should also lead to increased prenatal care utilization and improved health conditional on being pregnant (lower right cell of Figure 1). Empirical studies generally find supportive evidence for these hypotheses (see, for example, Currie and Grogger (2002) for evidence on prenatal care use and infant health and Kutinova and Conway (2005) for evidence on prenatal care use and maternal health).

¹³ This number includes non-disabled adults, children, and the disabled.

Given all of these effects of unemployment on fertility, prenatal care use, and health, the overall impact cannot easily be determined. Moreover, previous studies suggest that the effects will likely differ by women's characteristics such as race, marital status, and education (Bitler and Zavodny 2004, Dehejia and Lleras-Muney 2004). The evidence to date suggests that, in times of higher unemployment, fertility decreases especially among single blacks (Dehejia and Lleras-Muney 2004) and among low-educated women (Bitler and Zavodny 2004). Prenatal care use increases in aggregate data (Dehejia and Lleras-Muney 2004) and general health-related behaviors improve in both aggregate and individual-level data (Ruhm 2000, 2005). Finally, infant health improves with unemployment in aggregate data (Dehejia and Lleras-Muney 2004), overall health (including infant health) improves in aggregate data (Ruhm 2000), and medical problems and activity limitations decrease with unemployment in individual-level data (Ruhm 2003). So far, no systematic study explicitly investigating the interplay of unemployment and Medicaid, focusing on local labor markets, and considering a broader range of outcomes (including a key outcome of pregnancy -- maternal health) has been conducted. The primary goal of this paper is to fill this gap.

2.3. Data and Methods

2.3.1. Data

The main source of data are the Natality Detail Files for years 1989-1999 (US Dept. of Health and Human Services 1991-2000). Since 1985, the files have included information on all US births and so have contained more than 3 million observations

annually. Traditionally, infant health measures (such as birth weight and the Apgar score) and various maternal characteristics (such as age, marital status, race, ethnic origin, education, and place of residence) have been reported. In addition, since 1989, variables describing maternal morbidity during pregnancy and delivery have also been included.¹⁴

In this study, foreign residents are excluded from the analysis. Further, women from Louisiana and Nebraska in the year 1989, Oklahoma in years 1989-1990, and New York in years 1989-1991 are excluded due to missing information on maternal health. Mothers from Washington State in years 1989-1991 are excluded due to missing information on marital status and those from New Hampshire in years 1989-1992 due to missing information on ethnicity. Only non-Hispanic black and white women are included in the analysis (as in Currie and Grogger 2002 and Kutinova and Conway 2005). Also, the sample is restricted to women between 19 and 50 years of age who had a singleton birth and resided in a county with population of at least 100,000 people (FIPS codes for smaller counties are not available in the natality files making it impossible to match county-level unemployment rates to these observations).

In the main model specification, the Natality Detail Files data are aggregated to county/year/race cells and are merged with county-level annual unemployment rates from the 2002 Area Resource File. Six-month lags of unemployment are used in order to

¹⁴ Sixteen “medical risk factors” (anemia, cardiac disease, lung disease, diabetes, genital herpes, hydramnios/oligohydramnios, hemoglobinopathy, hypertension chronic and pregnancy-associated, eclampsia, incompetent cervix, previous infant 4000+ grams, previous preterm or small-for-gestational age infant, renal disease, Rh sensitization, and uterine bleeding) and fifteen “complications of labor and/or delivery” (febrile, meconium, premature rupture of membrane, abruption placenta, placenta previa, other excessive bleeding, seizures during labor, precipitous labor, prolonged labor, dysfunctional labor, breech/malpresentation, cephalopelvic disproportion, cord prolapse, anesthetic complication, and fetal distress) are separately identified in the natality files.

allow the economic conditions to impact women at a crucial stage of their pregnancies rather than just before delivery.¹⁵ In my data, there is substantial variation in unemployment within states which would not be captured by simple across-state comparisons (Figure 2). In particular, the standard deviation of the state-level unemployment rates is 1.23 and the average standard deviation of county-level unemployment rates *within state* is 1.48. In this case, the state unemployment rates mask significant county-level differences. Performing the analysis on the county level rather than by state therefore allows me to construct more precise proxies for the actual economic conditions facing pregnant women. And, as Baughman (2005) shows, local labor market conditions play an important role in determining Medicaid coverage.

To mitigate the problem of influential observations in very small samples, cells with less than 100 pregnancies are excluded from the estimations.¹⁶ The omission of small counties is common in infant health studies (Corman and Grossman 1985, Corman et al. 1987). The above restrictions leave me with 3,426 county/year observations in the black cohort and 5,125 observations in the white cohort.¹⁷

¹⁵ Since annual unemployment rates are used, all deliveries in the last 6 months of ‘year 1’ or in the first 6 months of ‘year 2’ are assigned unemployment rates for ‘year 1’. The same algorithm is later employed to assign Medicaid eligibility rules. In principle, different lags should be used to study selection into pregnancy and behavior conditional on becoming pregnant. The use of annual unemployment rates and Medicaid eligibility rules, however, makes a precise distinction impossible.

¹⁶ Because of the large within-state variation in unemployment and the importance of local labor markets for the effectiveness of government policies, a county-level analysis has many advantages over a state-level analysis. Admittedly, however, there are drawbacks to this approach as well. Most importantly, by excluding small counties, rural areas may be left out from the analysis. As descriptive statistics suggest, women included in my baseline sample have slightly higher utilization of prenatal care and better infant and maternal health outcomes than the state-level average (Tables 1 and 3). As a sensitivity check, I have therefore reestimated the models in this paper using state/year/race cells. The results remain qualitatively the same (see ‘State-Level Analyses’ below).

First, I estimate the effects of the unemployment rate on prenatal care utilization. Two measures of prenatal care use are employed: the percentage of women receiving prenatal care in the first trimester of pregnancy and the percentage of women with ‘adequate’ or ‘intermediate’ prenatal care (on the Kessner adequacy scale). Next, I turn to two measures of infant health: the incidences of low birth weight (birth weight between 1,500 and 2,500 grams) and very low birth weight (birth weight below 1,500 grams). Finally, I explore four measures of maternal health: the incidences of placental abruption¹⁸, anemia¹⁹, pregnancy-associated hypertension²⁰, and ‘any maternal complication’ (i.e., any of the above) in pregnant women. As Haas et al. (1993) note in their study of maternal complications in the US: “Although only 10 per 100,000 women die from a complication of pregnancy or childbirth, 60% of women receive medical care for some complication of pregnancy, and 30% suffer complications that result in serious morbidity.” (p.61) Therefore, adding maternal health measures to infant health measures in a study of pregnancy outcomes seems highly relevant. Placental abruption, anemia, and pregnancy-related hypertension have all been identified in the medical and public health literatures as important causes of maternal morbidity that are sensitive to interventions during the prenatal period (Laditka et al. 2005, Bashiri et al. 2003,

¹⁷ In order to verify that potential racial differences in my results are not driven primarily by different sample sizes, I have also estimated all the models for white women including only counties used in the estimations for blacks. The results remained qualitatively the same.

¹⁸ “Premature separation of a normally implanted placenta from the uterus.” (CDC)

¹⁹ “Hemoglobin level of less than 10.0 g/dL during pregnancy or a hematocrit of less than 30 percent during pregnancy.” (CDC)

²⁰ “An increase of blood pressure of at least 30mm Hg systolic or 15mm Hg diastolic on two measurements taken 6 hours apart after the 20th week of gestation.” (CDC)

Makrides et al. 2003, Villar et al. 2003, Scholl et al. 1994, Haas et al. 1993, Sachs et al. 1988). As such, these morbidities are among those most likely to be affected by the economic environment women face.

2.3.2. Empirical Strategy

Following earlier studies on the effects of unemployment changes on health-related behavior and health, I first estimate prenatal care use, infant and maternal health models in the following general form:

$$y_{ijt} = \beta_0 + \beta_1 * \text{unemployment rate}_{jt} + \beta_2 * J + \beta_3 * T + \varepsilon_{ijt},$$

where y is a measure of prenatal care use, infant or maternal health, i indexes race cohorts, j counties, and t years. J and T denote vectors of county and year dummies, respectively. In the above equation, β_1 measures the overall effects of unemployment on the outcomes of interest. In order to capture the *overall* (i.e., reduced form) effects of unemployment, cohort characteristics which change as ‘selection into pregnancy’ changes are not included on the right hand side.

Next, in order to investigate the interaction between unemployment and Medicaid in their effects on childbearing, I turn to models in the following form:

$$y_{ijst} = \gamma_0 + \gamma_1 * \text{unemployment rate}_{jt} + \gamma_2 * \text{Medicaid eligibility}_{st} + \gamma_3 * \text{unemployment rate}_{jt} * \text{Medicaid eligibility}_{st} + \gamma_4 * J + \gamma_5 * T + \mu_{ijst},$$

where i indexes race cohorts, j counties, s states, t years, and y , J , and T are defined as above. Medicaid eligibility is measured as the income cutoff (as a percent of the

federal poverty line) below which pregnant women qualified for Medicaid in a given state and year. This data comes from Hill (1992) and the National Governors' Association (2003) and six-month lags are used. In the interacted models, γ_1 measures the effects of 'unemployment per se' and γ_3 the effects of the unemployment and Medicaid eligibility interaction. As discussed above, the signs of these coefficients are theoretically ambiguous but it seems reasonable to assume that both 'unemployment per se' (γ_1) and the Medicaid 'safety net' (γ_3) will have a significant and independent impact on the outcomes of interest.

All models in this paper are estimated with OLS and robust standard errors correcting for heteroskedasticity and clustering at the county level are calculated.²¹ Observations are weighted by the number of individuals in each county/year/race cell.

2.4. Results

2.4.1. Descriptive Statistics

Following Dehejia and Lleras-Muney (2004), I first estimate all models stratified by mother's race. As has previously been demonstrated, black and white women have different fertility and pregnancy-related behavior and there are also large 'unexplained' racial disparities in infant and maternal health (e.g., Dubay et al. 2001, Currie and Grogger 2002, Kutinova and Conway 2005, Conway and Kutinova 2005). Therefore, it

²¹ As a sensitivity check, all models have also been estimated with tobit (to account for the fact that the values of prenatal care and infant health measures are bounded between 0 and 100 and those of maternal health measures between 0 and 1,000; i.e. the variables are censored). The coefficients of interest are robust to the choice of the estimation method. As suggested in Bertrand et al. (2004), clustering at the county level has been performed in order to account for a possible serial correlation within counties.

seems important to let the effects of unemployment (and Medicaid eligibility) differ by race.

The racial disparities documented elsewhere can also be observed in my data (Table 1). In particular, black mothers are less likely than white mothers to obtain prenatal care in the first trimester and to receive 'adequate' or 'intermediate' care. The incidence of low birth weight among black infants is more than twice that among white infants (9.06% vs. 3.73%, $p < 0.01$). The racial gap in the incidence of very-low birth weight is even bigger (2.64% vs. 0.72%, $p < 0.01$). Turning to maternal health, black women again have poorer outcomes than whites. In particular, 6.64% of black women and 4.90% of white women suffer from at least one of the morbidities studied ($p < 0.01$) and the racial gap is the largest for the incidence of anemia (3.19% vs. 1.43%, $p < 0.01$). Some of these differences may be attributable to the fact that black women are, on average, younger, less educated, and less likely to be married than whites (Table 1).

As to the economic environment facing the two groups of women, it can be seen that black mothers live in *states* with slightly higher average unemployment rates (5.90 vs. 5.80, $p < 0.05$; Table 1). The racial disparity in the average *county*-level unemployment rate is even higher (5.92 vs. 5.34, $p < 0.01$). The finding that the variation in the economic conditions (proxied by the unemployment rate) across counties is larger than the variation across states adds justification for the choice of the county-level variable in the econometric analysis. Finally, the descriptive statistics demonstrate that black and white women live in counties with similar per-capita income levels and Medicaid eligibility rates.

2.4.2. Baseline Results

In the baseline model similar to that in Dehejia and Lleras-Muney (2004), the county-level unemployment rate is the sole determinant (along with county and year dummies) of prenatal care use, infant and maternal health (upper panel of Table 2; combined effects from all cells of Figure 1). In this specification, the reduced-form effect of unemployment on prenatal care use is positive and statistically significant among blacks and insignificant (negative when measured by prenatal care initiation in the first trimester and positive when measured by ‘adequate/intermediate’ prenatal care) among whites. While Dehejia and Lleras-Muney (2004) find positive effects for both races, a negative (or zero) effect of unemployment among whites is consistent with their more general finding of ‘negative selection’ among women in this cohort. As in Dehejia and Lleras-Muney (2004), unemployment has a beneficial effect on infant health across the races (statistically significant only among whites). An equally strong beneficial effect is not observed for maternal health.²²

Models that add Medicaid eligibility and an interaction between unemployment and Medicaid eligibility to the unemployment rate on the right hand side (bottom panel of Table 2) help disentangle some of the overall effects observed above. It seems that unemployment per se (first and second rows of Figure 1) contributes to the higher prenatal care use among blacks and that Medicaid (third row of Figure 1) strengthens this effect (coefficients statistically insignificant). Among whites, on the other hand, unemployment per se significantly decreases prenatal care use but Medicaid eligibility

²² Note that *positive* signs on prenatal care measures indicate an increased use of prenatal care and *negative* signs on adverse infant and maternal health outcomes indicate improvements and infant and maternal health.

partly offsets this effect. Using the estimated coefficients, I have calculated the threshold level of Medicaid eligibility (M^{\wedge}), i.e., the level of Medicaid eligibility needed to completely offset the detrimental effects of unemployment on the outcomes of interest. Note that when M^{\wedge} is low, the overall effects of unemployment in the simple models (with unemployment as the sole explanatory variable) are driven by the unemployment*Medicaid eligibility interaction. When M^{\wedge} is high, the effects of unemployment per se dominate. Similarly, U^{\wedge} is used to indicate the level of unemployment at which the effects of Medicaid on prenatal care use (and health outcomes) switch from those driven by Medicaid per se to those driven by the unemployment*Medicaid interaction. As can be seen (Table 2), the noninteracted Medicaid variable sometimes has the counter-intuitive sign (decreasing prenatal care use) while the unemployment*Medicaid interaction acts in the expected direction. Fortunately, the level of U^{\wedge} is mostly very low (reaching 'out-of-sample' values), indicating that the effects of Medicaid per se are not relevant for the range of unemployment rates actually observed.

Turning to infant health, Medicaid seems to be playing a role in the reduction of low birth weight among both races (significant for whites; bottom panel of Table 2). Similarly, Medicaid in times of higher unemployment has a weak beneficial effect on maternal health. While mostly insignificant, the coefficients on the unemployment*Medicaid eligibility variable are consistently negative. Furthermore, among white women, the Medicaid 'safety net' significantly reduces the incidence of placental abruption (bottom panel of Table 2).

Thus, overall, the uninteracted results suggest that black women experience increases in prenatal care use (and potentially small improvements in health outcomes) when unemployment temporarily increases. As the interacted results show, Medicaid may be playing a beneficial role. The statistical significance of these results is weak, however. Among whites, infant health improves and prenatal care utilization and maternal health do not change significantly when unemployment (overall) increases. While these aggregate findings may seem puzzling, the interacted models shed more light on the mechanisms behind the observed reduced-form results. In particular, as expected, Medicaid eligibility in times of higher unemployment has a beneficial impact on all three sets of outcomes - increasing prenatal care utilization and improving infant and maternal health. Since unemployment per se mostly worsens outcomes, the resulting reduced-form impacts of unemployment reflect the relative magnitudes of the counteracting effects.

There are several reasons why the effects of unemployment per se may differ across the races (as well as across the outcomes studied). As mentioned above, unemployment induces income and substitution effects which influence 'selection into pregnancy' as well as women's behavior while pregnant (first and second rows of Figure 1). And, it seems likely that the relative sizes of the substitution and income effects as well as their impacts on selection and behavior will differ. For example, Dehejia and Lleras-Muney (2004) argue that the income effect is relatively stronger among black women who tend to be more credit constrained and that this effect demonstrates itself in a strong positive selection among blacks. Infant health improvements among whites, on the other hand, seem to be attributable to healthier behavior of white women during

pregnancy and these behavioral changes, in turn, are likely a consequence of significant substitution effects (Dehejia and Lleras-Muney 2004). No matter what the impacts of unemployment per se, however, the effects of the Medicaid ‘safety net’ (third row of Figure 1) seem to be -- at least weakly -- beneficial for both racial cohorts and across all sets of outcomes studied.

2.4.3. State-Level Analyses

As discussed above, a county-level analysis is superior in many respects to a state-level analysis. There are, however, limitations to using counties as well. Most importantly, the Natality Detail Files do not report geographic codes for counties with population of less than 100,000. In addition, to mitigate the problem of influential observations in very small samples, I have excluded county/year/race cells with less than 100 births from the estimations. Thus, my analysis omits the most sparsely populated (rural) areas. To investigate the possibility of a bias, I have reestimated the baseline models using *state/year/race* cells. This comparison is also useful in judging the potential limitations of using state level unemployment rates, as previous studies have done.

As descriptive statistics suggest, women included in my baseline sample have slightly higher utilization of prenatal care and better infant and maternal health outcomes than the state-level average (Tables 1 and 3). They are also more likely to be older, highly educated and married. To test the sensitivity of my results to the level of aggregation, I therefore consider two alternatives to the baseline county-level models: 1. a county-level analysis with state unemployment rates (Table 4) and 2. a state-level analysis with state unemployment rates (Table 5).

In the reduced form models with unemployment as the sole explanatory variable of interest, both state-level analyses confirm the county-level results (upper panels of Tables 2, 4, and 5). In particular, in all three specifications, unemployment significantly increases prenatal care use among black mothers and improves infant health among whites. Thus, the main findings are robust to the level of aggregation. This result also suggests that using state unemployment rates (as previous research has done) may be a reasonable practice.

The unit of analysis and the unemployment measure seem more important in the models that add Medicaid eligibility and the unemployment*Medicaid interaction on the right hand side (lower panels of Tables 2, 4, and 5). This is an appealing finding since the benefits of using county-level data should be the largest in models which already have a state-level variable – Medicaid eligibility – on the right hand side. It is noteworthy, however, that the differences in results across the three model specifications are again not large. In particular, among black women, the interacted results do not reach statistical significance in any of the model specifications but the coefficients mostly have the same sign. Among white women, unemployment per se decreases prenatal care use and the Medicaid ‘safety net’ increases it across all three specifications. Not surprisingly, the results are most significant when county-level cells are used (lower panels of Tables 2 and 4). In fact, the Medicaid ‘safety net’ is associated with significant benefits to infant and maternal health only when county-level cells are used (lower panels of Tables 2 and 4) and the results are most consistent across outcomes studied when county-level unemployment is also employed (lower panel of Tables 2).

Overall, the above sensitivity checks suggest that using county-level data is more important in the interacted models than in the simplest reduced form but, in both cases, the main findings seem reasonably robust to the level of aggregation.

2.4.4. Stratification by Socioeconomic Status

As previous research suggests, individuals with low socioeconomic status are the most vulnerable to the cyclical unemployment changes. For example, Hines et al. (2001) conduct a systematic review of the literature and corroborate the finding that “[...] labor market outcomes are procyclical, with greater sensitivity among lower skilled groups.” (p.5) Furthermore, since Medicaid is designed as a program for credit constrained populations, the ‘safety net’ should play the largest role among economically disadvantaged women. In order to test this hypothesis, I stratify the baseline sample by two measures of socioeconomic status: marriage and education.

2.4.4.1. Marital Status

Within both racial cohorts, there are large differences in the outcomes and characteristics of married and single women (Table 6). For example, among blacks, 64.36% of single women and 81.72% of married women receive prenatal care in the first trimester ($p < 0.01$). Among whites, the corresponding figures are 72.72% and 91.31% ($p < 0.01$). As to infant and maternal health, single women are, on average, more likely to deliver a low birth weight infant (10.28% vs. 6.94% among blacks and 6.00% vs. 3.29% among whites, $p < 0.01$) and to suffer from at least one of the maternal morbidities studied (6.71% vs. 6.40% among blacks and 5.55% vs. 4.77% among whites, $p < 0.01$). Of course, many of these disparities are probably partly driven by differences in socio-

economic status. Single women of both races are younger and less educated, on average. Differences in the macroeconomic conditions facing the different cohorts of mothers are remarkable as well. Most interestingly, the county-level unemployment rate (much more than the state-level measure) varies substantially with single pregnant women of both races facing higher unemployment than their married counterparts (Table 6).

When the samples are stratified by marital status, single women of both races seem to be more strongly affected by unemployment changes than married women (upper panel of Table 7). Namely, unemployment significantly increases prenatal care utilization among single black women and decreases it among single whites; infant health improves with unemployment among both single blacks and single whites; and, there is some evidence suggesting that maternal health improves with unemployment among single blacks. Married women are only weakly affected.

The models which explicitly take the interaction between unemployment and Medicaid eligibility into account yield additional interesting insights (bottom panel of Table 7). Among blacks (both single and married), the results generally do not reach statistical significance. There is one exception, however: Medicaid in times of higher unemployment significantly increases 'adequate/intermediate' prenatal care use among married blacks. The results for single whites are very informative. In this cohort, unemployment per se decreases prenatal care use and potentially leads to a deterioration of infant and maternal health. In all these cases, however, the unemployment*Medicaid eligibility interaction acts to (partially) offset unemployment's detrimental effects. The Medicaid 'safety net' therefore seems particularly operative in this group of women.

Among married whites, prenatal care use again decreases with unemployment per se and increases with Medicaid but the results for infant and maternal health are more mixed.

2.4.4.2. Education

Among both black and white women, education is a significant correlate of prenatal care use and infant and maternal health (Table 8). For example, among blacks, 56.58% of women with 'less than 12 years' of education, 71.82% of women with '12 to 15 years' of education, and 88.40% of women with '16 or more years' of education initiate prenatal care in the first trimester ($p < 0.01$). The corresponding numbers are 69.18%, 87.31%, and 95.33% among whites ($p < 0.01$). Less educated women of both races also deliver more low birth weight and very-low birth weight infants and have higher incidences of placental abruption and anemia. Pregnancy-associated hypertension, on the other hand, does not seem to fall monotonically with education. As expected, less educated black and white mothers are younger and less likely to be married. Notably, the county-level unemployment rate (more than the state-level variable) facing pregnant women consistently decreases with education. The county-level per-capita income increases.

In the models stratified by education, the prenatal care increases in times of higher unemployment previously observed among black women occur only among those with 'less than 12 years' or '12 to 15 years' of schooling (upper panel of Table 9). Among highly-educated blacks ('16 or more years' of education) and among whites of all education levels, the unemployment rate is associated with prenatal care decreases or with no significant change. With respect to infant health, there is some evidence of general improvements in times of higher unemployment across all racial and education

cohorts (with the exception of highly-educated blacks). White women with '12 to 15 years' or '16 or more years' of education seem most strongly affected. The effects of unemployment on maternal health exhibit no clear pattern. (If anything, blacks with '12 to 15 years' and whites with 'less than 12 years' of education seem to benefit and more educated whites seem to be hurt.)

Once again, the interacted models with unemployment and Medicaid eligibility reveal additional interesting relationships (bottom panel of Table 9). Among blacks with 'less than 12 years' and '12 to 15 years' of education, both unemployment per se and unemployment interacted with Medicaid eligibility potentially increase prenatal care use. Among highly-educated blacks ('16 or more years' of education; coefficients insignificant) and among whites of all education levels (significant effects), prenatal care decreases with unemployment per se but Medicaid has a protective effect.

The results for infant health are suggestive. Namely, unemployment per se seems deleterious and unemployment*Medicaid eligibility beneficial among black women with '12 to 15 years' of education and among whites with 'less than 12 years' of schooling. Women from these groups are likely the most vulnerable to the cyclical unemployment changes but they are also those most likely to be enrolled in Medicaid when the economy temporarily deteriorates. Similarly, unemployment per se increases maternal complications and unemployment*Medicaid eligibility decreases them among black women with 'less than 12 years' of education and among whites with 'less than 12 years' or '12 to 15 years' of schooling. The results for other cohorts are mixed. Overall, the above findings are consistent with the hypothesis that unemployment per se may be

harmful for women with low socioeconomic status and that Medicaid eligibility acts to mitigate the unemployment's detrimental effects.

2.4.5. The Effects of Unemployment on Cohort Characteristics

As discussed above, the effects of unemployment (and Medicaid) on aggregate-level behaviors and health outcomes may arise both from differential changes in fertility across different groups of women (first column of Figure 1) and from changes in behaviors and outcomes conditional on becoming pregnant (second column of Figure 1). In order to investigate how much of the effects of unemployment observed in this paper are due to 'selection into pregnancy', I regress the county/year-specific mean characteristics of the two racial cohorts on the same set of explanatory variables employed above (Table 10).

The reduced-form effects of unemployment on maternal characteristics are highly significant (upper panel of Table 10; combined effects from the first column of Figure 1). In particular, in times of higher unemployment, the percentages of married and older women significantly increase among both blacks and whites. This finding is intuitively appealing because married and older women are less likely to be credit constrained (and thus subjected to a large negative income shock caused by unemployment) than single and young mothers.

The interacted results add further insights (bottom panel of Table 10). Specifically, among blacks, unemployment per se (upper and middle left cells of Figure 1) increases the percentage of mothers with '16 or more years' of education as well as the percentages of older mothers. Interestingly, the unemployment*Medicaid interaction

(lower left cell of Figure 1) acts in the opposite direction, significantly decreasing the percentages of more educated and older blacks. Similarly, among whites, unemployment per se has a significant positive effect on both maternal education (as measured by the percentage with '16 or more years' of schooling) and age. In addition, the percentage of married white women increases when the economy temporarily deteriorates. In all these cases, Medicaid acts to partially offset the unemployment's effects; i.e., it significantly decreases maternal education, age, and the percentage of married moms. These findings are consistent with the hypothesis that unemployment per se leads to a 'positive selection into pregnancy' (only more affluent women becoming pregnant) and that the Medicaid 'safety net' mitigates this effect by financially supporting less affluent women.

There are, however, seemingly puzzling patterns in the interacted results as well. First, while unemployment per se significantly increases (and Medicaid decreases) the percentage of white mothers with '16 or more years' of schooling, the effect of unemployment (unemployment*Medicaid) on the percentage of white women with 'less than 12 years' of education is positive (negative) and significant as well. Notably, however, the statistical significance of the latter effect is lower. Also, the results appear less counter-intuitive when keeping in mind that the percentages of mothers with 'less than 12 years', '12 to 15 years', and 'at least 16 years' of education have to sum up to 100%. Viewed from this perspective, my results suggest that unemployment decreases (and Medicaid increases) the percentage of mothers with '12 to 15 years' of schooling – an economically vulnerable group subject to large business cycle fluctuation in employment and health insurance coverage.

Another puzzling finding is that ‘Medicaid per se’ significantly increases the percentages of married, older, and most educated whites. Since the threshold levels of unemployment (U^*) at which the effects of Medicaid switch sign are relatively high (above the national average), these effects may be ‘truly operative’ for at least some of the counties in my sample. In these cases, Medicaid eligibility expansions far beyond the poverty line may be causing ‘positive selection’ among whites. Overall, however, my results strongly suggest that unemployment per se increases - and the Medicaid ‘safety net’ decreases - maternal education, age, and the percentage of married moms.

2.5. Conclusion

The goal of this paper is to contribute to our understanding of the effects of unemployment on health-related behaviors and health by studying the specific impacts of unemployment on pregnant women. Explicitly recognizing that, in this population, Medicaid eligibility in times of higher unemployment may serve as a ‘safety net’, I investigate the interplay of unemployment and Medicaid in affecting prenatal care utilization, infant and maternal health. These relationships have not systematically been investigated in research to date. Also, by including an important overlooked pregnancy outcome -- maternal health -- and by using a more refined measure of unemployment, I add new value to previous work.

My empirical analysis of US data for the 1990’s indicates that, overall, higher unemployment at the county level is associated with improved infant health especially among whites and increased prenatal care utilization (and potentially improved maternal health) among blacks. In some cases, both unemployment per se and unemployment

interacted with Medicaid eligibility seem to be contributing to the beneficial effects. In others, the Medicaid 'safety net' acts to mitigate, completely offset, or outweigh detrimental effects of unemployment. Interestingly, at least some of these aggregate-level effects are apparently due to changes in the selection of women into pregnancy. Specifically, unemployment per se increases - and unemployment*Medicaid eligibility decreases - the percentages of highly educated, older, and married mothers. These results are consistent with the role of Medicaid as a 'safety net' for vulnerable, credit constrained populations. In analyses stratified by marital status and education, Medicaid plays the largest role among economically disadvantaged (single and less educated) women. Thus, unemployment may be good for your pregnancy -- provided Medicaid steps in.

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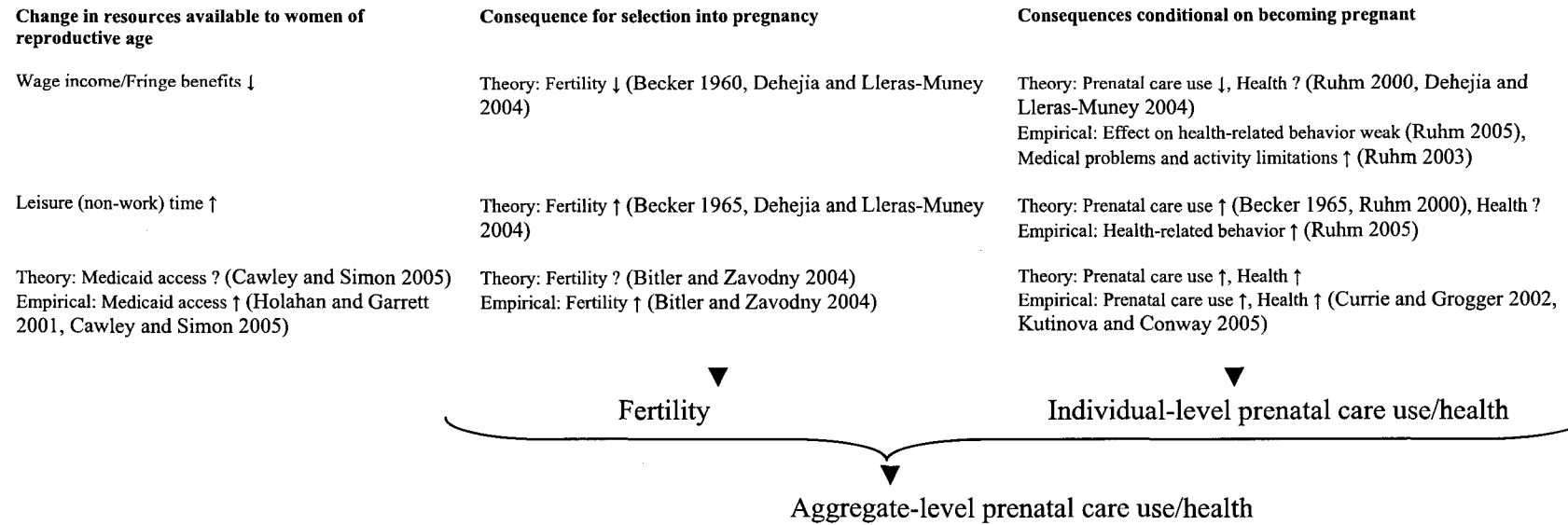
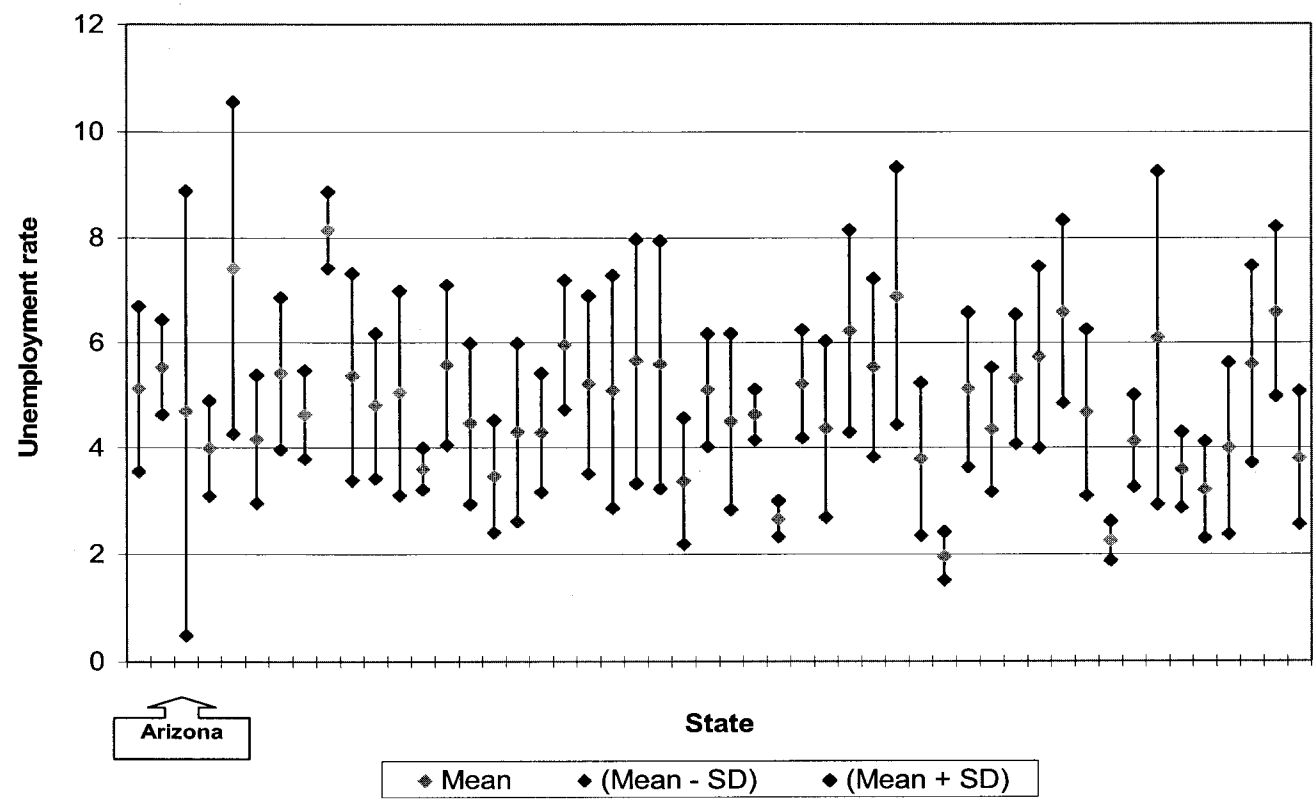
Figure 1. The Effects of an Increase in Unemployment

Figure 2. Variation in County-Level Unemployment within and across States, 1991-1999



'Mean' is the state unemployment rate constructed as a weighted average of county unemployment rates. 'SD' is a weighted standard deviation of county unemployment rates within state.

Table 1. Characteristics of Race Cohorts

Percentage or mean	Black (3,426 obs.)	White (5,125 obs.)
PNC in 1 st trimester	70.68	88.30
Adequate/intermediate PNC	87.84	96.79
Low birth weight	9.06	3.73
Very-low birth weight	2.64	0.72
Placental abruption	0.70	0.56
Anemia	3.19	1.43
Hypertension	3.01	3.05 ^C
Any maternal complication	6.64	4.90
30 ≤ age < 40	26.54	41.55
Age ≥ 40	1.49	2.11
Less than high school education	18.96	8.59
At least college education	11.54	32.23
Married	36.28	83.72
Unemployment rate – county	5.92	5.34
Unemployment rate – state	5.90	5.80 ^A
Per-capita income (USD)	23,544	23,654 ^C
Medicaid eligibility threshold (% federal poverty line/100)	1.64	1.65 ^C

Unless otherwise noted, all differences between blacks and whites are statistically significant at the 99% confidence level. ^A, ^B, and ^C indicate significance at the 95%, 90%, and less than 90% confidence level, respectively.

Table 2. The Effects of Unemployment on Prenatal Care Use, Infant and Maternal Health; By Race

	Black	White
Y = f(Unemployment), 1989-1999, county-level		
PNC in 1 st trimester	U: 0.537** (0.242)	U: -0.068 (0.078)
Adequate/intermediate PNC	U: 0.390** (0.188)	U: 0.014 (0.033)
Low birth weight	U: -0.041 (0.056)	U: -0.021** (0.008)
Very-low birth weight	U: -0.002 (0.017)	U: -0.008** (0.003)
Placental abruption	U: 0.015 (0.099)	U: 0.010 (0.045)
Anemia	U: -0.466 (1.121)	U: 0.093 (0.282)
Hypertension	U: -0.330 (0.547)	U: 0.001 (0.214)
Any maternal complication	U: -0.765 (1.513)	U: 0.010 (0.373)
Y = f(Unemployment, Medicaid, Unemployment*Medicaid), 1989-1999, county-level		
PNC in 1 st trimester	U: 0.438 (0.450) M: 0.158 (1.448) U*M: 0.050 (0.245)	U: -0.576*** (0.139) M: -0.713* (0.385) U*M: 0.282*** (0.072) (M [^] = 204%FPL, U [^] = 2.53)
Adequate/intermediate PNC	U: 0.142 (0.308) M: 0.257 (0.911) U*M: 0.128 (0.184)	U: -0.259*** (0.074) M: -0.285 (0.219) U*M: 0.151*** (0.041) (M [^] = 171%FPL)
Low birth weight	U: 0.074 (0.086) M: 0.157 (0.271) U*M: -0.064 (0.054)	U: 0.007 (0.019) M: 0.050 (0.056) U*M: -0.015* (0.009) (M [^] = 44%FPL)
Very-low birth weight	U: 0.009 (0.035) M: -0.021 (0.089) U*M: -0.006 (0.019)	U: -0.013* (0.007) M: -0.018 (0.025) U*M: 0.003 (0.004)
Placental abruption	U: -0.041 (0.218) M: -0.406 (0.569) U*M: 0.036 (0.126)	U: 0.201** (0.099) M: 0.151 (0.290) U*M: -0.106** (0.050) (M [^] = 191%FPL)
Anemia	U: 0.793 (2.087) M: 3.176 (4.634) U*M: -0.722 (0.978)	U: 0.413 (0.528) M: -0.748 (1.246) U*M: -0.170 (0.233)
Hypertension	U: 0.217 (1.471) M: 2.570 (3.001) U*M: -0.333 (0.695)	U: 0.299 (0.460) M: 0.763 (1.344) U*M: -0.168 (0.221)
Any maternal complication	U: 0.747 (2.907) M: 4.802 (6.208) U*M: -0.883 (1.327)	U: 0.830 (0.757) M: 0.035 (1.989) U*M: -0.449 (0.360)

Estimated with OLS. All models include county and year fixed effects. NDF have been aggregated by race. Observations are weighted by the number of births. SEs (in parentheses) have been corrected for heteroskedasticity and clustering at the county level. *, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. U is the unemployment rate and M the Medicaid eligibility threshold. M[^] indicates a "break-point" level of Medicaid eligibility (as a percent of the federal poverty line). U[^] indicates a "break-point" level of the unemployment rate.

Table 3. Characteristics of Race Cohorts; State-Level Cells

Proportion or mean	Black (504 obs.)	White (597 obs.)
PNC in 1 st trimester	70.05	87.04
Adequate/intermediate PNC	88.02	96.58
Low birth weight	9.00	3.86
Very-low birth weight	2.58	0.73
Placental abruption	0.71	0.59
Anemia	3.32	1.51
Hypertension	3.15	3.37
Any maternal complication	6.91	5.32
30 ≤ age < 40	25.33	37.45
Age ≥ 40	1.42	1.86
Less than high school education	19.29	10.22
At least college education	10.66	27.96
Married	36.31	83.28
Unemployment rate – state	5.86	5.69 ^c
Medicaid eligibility threshold (% federal poverty line/100)	1.63	1.62 ^c

Unless otherwise noted, all differences between blacks and whites are statistically significant at the 99% confidence level. ^a, ^b, and ^c indicate significance at the 95%, 90%, and less than 90% confidence level, respectively.

Table 4. The Effects of Unemployment on Prenatal Care Use, Infant and Maternal Health; By Race; State-Level Unemployment

	Black	White
Y = f(Unemployment), 1989-1999, county-level		
PNC in 1 st trimester	U: 0.756** (0.317)	U: -0.059 (0.107)
Adequate/intermediate PNC	U: 0.481* (0.251)	U: 0.015 (0.042)
Low birth weight	U: -0.060 (0.073)	U: -0.036*** (0.011)
Very-low birth weight	U: -0.009 (0.023)	U: -0.009** (0.004)
Placental abruption	U: -0.150 (0.114)	U: 0.063 (0.067)
Anemia	U: -1.418 (1.552)	U: 0.114 (0.325)
Hypertension	U: -0.415 (0.728)	U: 0.033 (0.259)
Any maternal complication	U: -1.926 (2.128)	U: 0.098 (0.439)
Y = f(Unemployment, Medicaid, Unemployment*Medicaid), 1989-1999, county-level		
PNC in 1 st trimester	U: 0.555 (0.805) M: -0.193 (2.278) U*M: 0.110 (0.420)	U: -0.913*** (0.220) M: -1.939*** (0.647) U*M: 0.490*** (0.121) (M [^] = 186%FPL)
Adequate/intermediate PNC	U: 0.012 (0.620) M: -0.466 (1.611) U*M: 0.258 (0.308)	U: -0.434*** (0.135) M: -0.915** (0.393) U*M: 0.257*** (0.072) (M [^] = 169%FPL)
Low birth weight	U: 0.047 (0.114) M: 0.167 (0.393) U*M: -0.060 (0.080)	U: 0.003 (0.034) M: 0.098 (0.100) U*M: -0.022 (0.018)
Very-low birth weight	U: -0.020 (0.056) M: -0.084 (0.140) U*M: 0.008 (0.027)	U: -0.024* (0.013) M: -0.052 (0.043) U*M: 0.009 (0.008)
Placental abruption	U: -0.036 (0.385) M: 0.147 (1.123) U*M: -0.063 (0.228)	U: 0.400** (0.179) M: 0.648 (0.543) U*M: -0.192* (0.098) (M [^] = 208%FPL)
Anemia	U: -2.199 (3.309) M: -2.150 (9.516) U*M: 0.462 (1.821)	U: -0.820 (0.919) M: -4.436* (2.465) U*M: 0.562 (0.508)
Hypertension	U: 0.456 (1.955) M: 3.902 (4.874) U*M: -0.551 (0.981)	U: 0.241 (0.897) M: 0.604 (2.649) U*M: -0.121 (0.472)
Any maternal complication	U: -2.115 (4.357) M: 0.766 (12.093) U*M: 0.081 (2.306)	U: -0.147 (1.412) M: -2.907 (3.984) U*M: 0.166 (0.763)

Estimated with OLS. All models include county and year fixed effects. NDF have been aggregated by race. Observations are weighted by the number of births. SEs (in parentheses) have been corrected for heteroskedasticity and clustering at the county level. *, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. U is the unemployment rate and M the Medicaid eligibility threshold. M[^] indicates a "break-point" level of Medicaid eligibility (as a percent of the federal poverty line).

Table 5. The Effects of Unemployment on Prenatal Care Use, Infant and Maternal Health; By Race; State-Level Unemployment and Cells

	Black	White
Y = f(Unemployment), 1989-1999, state-level		
PNC in 1 st trimester	U: 0.730* (0.395)	U: -0.083 (0.147)
Adequate/intermediate PNC	U: 0.472* (0.254)	U: 0.018 (0.062)
Low birth weight	U: -0.055 (0.090)	U: -0.035*** (0.013)
Very-low birth weight	U: -0.010 (0.032)	U: -0.011*** (0.004)
Placental abruption	U: -0.106 (0.107)	U: 0.070 (0.064)
Anemia	U: -1.451 (1.561)	U: 0.236 (0.275)
Hypertension	U: -0.480 (0.786)	U: -0.035 (0.380)
Any maternal complication	U: -1.968 (2.142)	U: 0.192 (0.372)
Y = f(Unemployment, Medicaid, Unemployment*Medicaid), 1989-1999, state-level		
PNC in 1 st trimester	U: 0.506 (0.964) M: 0.111 (2.808) U*M: 0.114 (0.497)	U: -0.878** (0.355) M: -1.723* (1.018) U*M: 0.463** (0.188) (M [^] = 189%FPL)
Adequate/intermediate PNC	U: -0.134 (0.671) M: -0.825 (1.878) U*M: 0.339 (0.357)	U: -0.443** (0.191) M: -1.008* (0.532) U*M: 0.269** (0.100) (M [^] = 165%FPL)
Low birth weight	U: 0.059 (0.162) M: 0.145 (0.550) U*M: -0.063 (0.113)	U: 0.016 (0.047) M: 0.149 (0.154) U*M: -0.030 (0.027)
Very-low birth weight	U: -0.020 (0.074) M: -0.126 (0.198) U*M: 0.009 (0.037)	U: -0.018 (0.015) M: -0.038 (0.045) U*M: 0.005 (0.009)
Placental abruption	U: 0.132 (0.412) M: 0.534 (1.195) U*M: -0.139 (0.243)	U: 0.307 (0.194) M: 0.529 (0.640) U*M: -0.138 (0.117)
Anemia	U: -2.541 (3.512) M: -3.846 (9.679) U*M: 0.676 (1.929)	U: -0.578 (1.046) M: -4.076 (2.648) U*M: 0.508 (0.575)
Hypertension	U: 0.591 (2.013) M: 3.617 (5.361) U*M: -0.659 (1.033)	U: 0.361 (1.232) M: 0.756 (3.895) U*M: -0.229 (0.679)
Any maternal complication	U: -2.114 (4.565) M: -0.738 (12.928) U*M: 0.097 (2.491)	U: 0.115 (1.516) M: -2.495 (4.597) U*M: 0.079 (0.867)

Estimated with OLS. All models include state and year fixed effects. NDF have been aggregated by race. Observations are weighted by the number of births. SEs (in parentheses) have been corrected for heteroskedasticity and clustering at the state level. *, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. U is the unemployment rate and M the Medicaid eligibility threshold. M[^] indicates a “break-point” level of Medicaid eligibility (as a percent of the federal poverty line).

Table 6. Characteristics of Race/Marital Status Cohorts

Percentage or mean	Single Black (3,002 obs.)	Married Black (2,554 obs.)	Single White (4,864 obs.)	Married White (5,123 obs.)
PNC in 1 st trimester	64.36	81.72	72.72	91.31
Adequate/intermediate PNC	84.22	94.06	91.16	97.87
Low birth weight	10.28	6.94	6.00	3.29
Very-low birth weight	2.86	2.28	1.23	0.62
Placental abruption	0.75	0.60	0.73	0.53
Anemia	3.36	2.81	1.92	1.33
Hypertension	2.86	3.23	3.07	3.05 ^c
Any maternal complication	6.71	6.40	5.55	4.77
30 ≤ age < 40	18.84	40.26	19.94	45.75
Age ≥ 40	1.01	2.36	1.47	2.23
Less than high school education	25.06	8.33	24.22	5.57
At least college education	4.83	23.39	7.56	37.00
Unemployment rate – county	6.03	5.76	5.55	5.30
Unemployment rate – state	5.91	5.91 ^c	5.80	5.80 ^c
Per-capita income (USD)	23,516	23,694 ^c	23,091	23,767
Medicaid eligibility threshold (% federal poverty line/100)	1.64	1.65 ^c	1.67	1.64 ^a

Unless otherwise noted, all differences between single and married women within a racial cohort are statistically significant at the 99% confidence level. ^a, ^b, and ^c indicate significance at the 95%, 90%, and less than 90% confidence level, respectively.

Table 7. The Effects of Unemployment on Prenatal Care Use, Infant and Maternal Health; By Race and Marital Status

	Single Black	Married Black	Single White	Married White
Y = f(Unemployment), 1989-1999, county-level				
PNC in 1 st trimester	U: 0.690* (0.350)	U: 0.130 (0.180)	U: -0.522*** (0.141)	U: -0.070 (0.077)
Adequate/intermediate PNC	U: 0.507* (0.271)	U: 0.115 (0.114)	U: -0.147* (0.077)	U: 0.012 (0.030)
Low birth weight	U: -0.054 (0.071)	U: -0.006 (0.042)	U: -0.018 (0.031)	U: -0.009 (0.008)
Very-low birth weight	U: -0.008 (0.027)	U: 0.017 (0.022)	U: -0.027* (0.015)	U: -0.002 (0.004)
Placental abruption	U: -0.011 (0.107)	U: 0.051 (0.132)	U: -0.014 (0.088)	U: 0.018 (0.045)
Anemia	U: -0.224 (1.308)	U: -0.963 (0.954)	U: 0.011 (0.399)	U: 0.132 (0.283)
Hypertension	U: -0.454 (0.623)	U: -0.055 (0.541)	U: -0.221 (0.277)	U: 0.028 (0.222)
Any maternal complication	U: -0.665 (1.755)	U: -0.960 (1.329)	U: -0.275 (0.546)	U: 0.074 (0.368)
Y = f(Unemployment, Medicaid, Unemployment*Medicaid), 1989-1999, county-level				
PNC in 1 st trimester	U: 0.893 (0.608) M: 0.886 (2.088) U*M: -0.122 (0.339)	U: -0.064 (0.325) M: -0.258 (0.844) U*M: 0.110 (0.171)	U: -1.060*** (0.257) M: -0.606 (0.685) U*M: 0.291** (0.127) (M [^] =364%FPL)	U: -0.537*** (0.127) M: -0.735** (0.354) U*M: 0.261*** (0.065) (M [^] =206%FPL, U [^] =2.81)
Adequate/intermediate PNC	U: 0.492 (0.420) M: 1.021 (1.334) U*M: -0.010 (0.251)	U: -0.184 (0.194) M: -0.264 (0.479) U*M: 0.167* (0.098) (M [^] =110%FPL)	U: -0.611*** (0.171) M: -0.055 (0.500) U*M: 0.249*** (0.091) (M [^] =246%FPL)	U: -0.201*** (0.062) M: -0.282 (0.183) U*M: 0.119*** (0.033) (M [^] =170%FPL)
Low birth weight	U: 0.052 (0.113) M: 0.189 (0.314) U*M: -0.059 (0.060)	U: 0.104 (0.097) M: 0.076 (0.291) U*M: -0.061 (0.058)	U: 0.072 (0.056) M: 0.194 (0.146) U*M: -0.049** (0.023) (M [^] =147%FPL)	U: 0.010 (0.017) M: 0.049 (0.049) U*M: -0.011 (0.009)
Very-low birth weight	U: 0.016 (0.059) M: -0.042 (0.148) U*M: -0.011 (0.030)	U: -0.046 (0.050) M: -0.108 (0.136) U*M: 0.036 (0.024)	U: 0.013 (0.028) M: -0.002 (0.085) U*M: -0.021 (0.015)	U: -0.017** (0.007) M: -0.019 (0.022) U*M: 0.008** (0.004) (M [^] =206%FPL)
Placental abruption	U: -0.196 (0.276) M: -0.837 (0.672) U*M: 0.112 (0.157)	U: 0.181 (0.268) M: 0.206 (0.741) U*M: -0.074 (0.150)	U: 0.126 (0.211) M: -0.295 (0.579) U*M: -0.074 (0.103)	U: 0.218** (0.101) M: 0.235 (0.293) U*M: -0.111** (0.051) (M [^] =196%FPL)
Anemia	U: 2.153 (2.452) M: 5.527 (5.348) U*M: -1.348 (1.137)	U: -1.617 (1.945) M: -1.171 (4.513) U*M: 0.373 (0.964)	U: 1.377* (0.822) M: 0.896 (1.834) U*M: -0.735** (0.329) (M [^] =187%FPL)	U: 0.157 (0.518) M: -1.258 (1.255) U*M: -0.005 (0.236)
Hypertension	U: 0.287 (1.813) M: 2.700 (3.712) U*M: -0.438 (0.872)	U: 0.274 (1.235) M: 2.316 (2.672) U*M: -0.211 (0.561)	U: 0.421 (0.668) M: 2.689 (1.810) U*M: -0.356 (0.318)	U: 0.215 (0.457) M: 0.279 (1.337) U*M: -0.105 (0.220)
Any maternal complication	U: 1.874 (3.525) M: 6.644 (7.498) U*M: -1.452 (1.619)	U: -1.114 (2.473) M: 1.170 (5.356) U*M: 0.069 (1.155)	U: 1.814 (1.198) M: 3.211 (3.026) U*M: -1.133** (0.550) (M [^] =160%FPL)	U: 0.524 (0.735) M: -0.859 (1.923) U*M: -0.241 (0.352)

Estimated with OLS. All models include county and year fixed effects. NDF have been aggregated by race and marital status. Observations are weighted by the number of births. SEs (in parentheses) have been corrected for heteroskedasticity and clustering at the county level. *, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. U is the unemployment rate and M the Medicaid eligibility threshold. M[^] indicates a "break-point" level of Medicaid eligibility (as a percent of the federal poverty line). U[^] indicates a "break-point" level of the unemployment rate.

Table 8. Characteristics of Race/Education Cohorts

Percentage or mean	Black Edu <12 (1,630 obs.)	Black Edu 12-15 (3,179 obs.)	Black Edu 16+ (1,244 obs.)	White Edu <12 (3,987 obs.)	White Edu 12-15 (5,121 obs.)	White Edu 16+ (4,988 obs.)
PNC in 1 st trimester	56.58	71.82	88.40	69.18	87.31	95.33
Adequate/intermediate PNC	77.03	89.48	96.60	89.02	96.79	98.91
Low birth weight	12.03	8.66	6.45	6.41	3.85	2.76
Very-low birth weight	2.78	2.57	2.36	1.10	0.75	0.52
Placental abruption	0.82	0.67	0.57	0.77	0.57	0.47
Anemia	3.51	3.13	2.49	2.01	1.47	1.21
Hypertension	2.19	3.09	3.74	2.29	3.24	2.91
Any maternal complication	6.28	6.63	6.56 ^C	4.91	5.13	4.46
30 ≤ age < 40	17.96	24.65	52.56	15.77	34.60	61.23
Age ≥ 40	1.18	1.27	3.42	0.86	1.55	3.44
Married	15.64	35.83	72.89	54.43	81.34	96.22
Unemployment rate – county	6.27	5.92	5.59	5.72	5.46	5.03
Unemployment rate – state	6.01 ^B	5.92	5.80 ^B	5.91 ^B	5.84	5.73
Per-capita income (USD)	23,266 ^C	23,375	25,423	21,713	22,885	25,531
Medicaid eligibility threshold (% federal poverty line/100)	1.63 ^C	1.64	1.70	1.60 ^A	1.63	1.68

Unless otherwise noted, all differences compared to women with '12 to 15 years' of education within the same racial cohort are statistically significant at the 99% confidence level. ^A, ^B, and ^C indicate significance at the 95%, 90%, and less than 90% confidence level, respectively.

Table 9. The Effects of Unemployment on Prenatal Care Use, Infant and Maternal Health; By Race and Education

	Black Edu <12	Black Edu 12-15	Black Edu 16+	White Edu <12	White Edu 12-15	White Edu 16+
Y = f(Unemployment), 1989-1999, county-level						
PNC in 1 st trimester	U: 1.145** (0.476)	U: 0.526** (0.232)	U: 0.030 (0.145)	U: -0.342** (0.155)	U: -0.028 (0.083)	U: -0.012 (0.056)
Adequate/intermediate PNC	U: 0.942** (0.389)	U: 0.334* (0.175)	U: -0.070 (0.095)	U: 0.006 (0.093)	U: 0.026 (0.034)	U: -0.012 (0.025)
Low birth weight	U: -0.048 (0.109)	U: -0.063 (0.051)	U: 0.032 (0.053)	U: -0.046 (0.035)	U: -0.017* (0.010)	U: -0.021* (0.012)
Very-low birth weight	U: -0.009 (0.044)	U: -0.004 (0.017)	U: 0.029 (0.034)	U: 0.003 (0.014)	U: -0.011*** (0.004)	U: -0.000 (0.005)
Placental abruption	U: 0.129 (0.179)	U: -0.023 (0.104)	U: 0.102 (0.268)	U: -0.099 (0.114)	U: 0.027 (0.048)	U: 0.042 (0.064)
Anemia	U: 0.402 (1.388)	U: -0.510 (1.147)	U: -1.921 (1.548)	U: -0.039 (0.457)	U: 0.012 (0.255)	U: 0.325 (0.423)
Hypertension	U: -0.217 (0.671)	U: -0.359 (0.519)	U: 0.254 (1.250)	U: -0.081 (0.295)	U: 0.045 (0.222)	U: 0.098 (0.258)
Any maternal complication	U: 0.350 (1.839)	U: -0.885 (1.504)	U: -1.509 (2.316)	U: -0.285 (0.585)	U: 0.003 (0.371)	U: 0.314 (0.503)
Y = f(Unemployment, Medicaid, Unemployment*Medicaid), 1989-1999, county-level						
PNC in 1 st trimester	U: 0.948 (0.886) M: -0.515 (3.104) U*M: 0.113 (0.485)	U: 0.441 (0.398) M: 0.352 (1.348) U*M: 0.040 (0.219)	U: -0.200 (0.461) M: -0.160 (0.967) U*M: 0.127 (0.242)	U: -0.635* (0.342) M: 0.113 (1.108) U*M: 0.157 (0.171)	U: -0.466*** (0.144) M: -0.558 (0.407) U*M: 0.243*** (0.074) (M^ = 192)	U: -0.202** (0.092) M: -0.211 (0.223) U*M: 0.105** (0.043) (M^ = 191)
Adequate/intermediate PNC	U: 0.595 (0.672) M: 0.720 (2.107) U*M: 0.168 (0.377)	U: 0.122 (0.249) M: 0.319 (0.749) U*M: 0.108 (0.143)	U: -0.360 (0.254) M: -0.485 (0.559) U*M: 0.162 (0.134)	U: -0.371* (0.217) M: 0.369 (0.762) U*M: 0.200* (0.121) (M^ = 185)	U: -0.177** (0.071) M: -0.110 (0.216) U*M: 0.111*** (0.038) (M^ = 159)	U: -0.094* (0.049) M: -0.149 (0.145) U*M: 0.046* (0.024) (M^ = 204)
Low birth weight	U: -0.115 (0.222) M: -0.298 (0.548) U*M: 0.041 (0.105)	U: 0.116 (0.090) M: 0.302 (0.297) U*M: -0.100* (0.059) (M^ = 116)	U: 0.003 (0.163) M: -0.279 (0.346) U*M: 0.018 (0.081)	U: 0.091 (0.072) M: 0.299 (0.252) U*M: -0.076** (0.035) (M^ = 120)	U: -0.003 (0.025) M: 0.009 (0.075) U*M: -0.008 (0.012)	U: 0.013 (0.031) M: 0.116 (0.073) U*M: -0.019 (0.016)
Very-low birth weight	U: 0.054 (0.089) M: -0.023 (0.217) U*M: -0.032 (0.043)	U: 0.001 (0.034) M: -0.032 (0.099) U*M: -0.002 (0.018)	U: -0.013 (0.111) M: -0.173 (0.254) U*M: 0.025 (0.060)	U: 0.001 (0.027) M: -0.110 (0.086) U*M: 0.002 (0.015)	U: -0.007 (0.010) M: 0.002 (0.032) U*M: -0.002 (0.005)	U: -0.006 (0.011) M: 0.017 (0.031) U*M: 0.003 (0.006)
Placental abruption	U: 1.019* (0.537) M: 1.622 (1.225) U*M: -0.497* (0.293) (M^ = 205)	U: -0.182 (0.234) M: -0.476 (0.627) U*M: 0.092 (0.122)	U: 0.591 (0.426) M: 0.290 (1.099) U*M: -0.268 (0.258)	U: 0.210 (0.251) M: -0.198 (0.763) U*M: -0.165 (0.123)	U: 0.187* (0.108) M: 0.035 (0.309) U*M: -0.087 (0.053)	U: 0.263* (0.140) M: 0.422 (0.367) U*M: -0.124* (0.071) (M^ = 212)
Anemia	U: 5.625 (3.820) M: 13.247 (10.003) U*M: -2.989 (2.025)	U: 0.243 (2.013) M: 1.379 (4.402) U*M: -0.424 (0.930)	U: -3.934 (3.183) M: -1.961 (6.238) U*M: 1.112 (1.478)	U: 1.418 (1.232) M: 1.028 (2.900) U*M: -0.791 (0.547)	U: 0.497 (0.499) M: -0.389 (1.260) U*M: -0.262 (0.221)	U: -0.188 (0.663) M: -2.146 (1.388) U*M: 0.294 (0.308)
Hypertension	U: 1.443 (2.353) M: 4.583 (4.942) U*M: -0.957 (1.167)	U: 0.082 (1.487) M: 2.418 (3.114) U*M: -0.274 (0.709)	U: -1.067 (2.360) M: -2.160 (4.687) U*M: 0.738 (1.035)	U: 0.949 (0.719) M: 1.930 (1.932) U*M: -0.567* (0.318) (M^ = 167)	U: 0.596 (0.475) M: 0.877 (1.465) U*M: -0.306 (0.229)	U: 0.058 (0.578) M: 1.122 (1.504) U*M: 0.015 (0.296)
Any maternal complication	U: 7.411 (5.050) M: 17.656 (12.292) U*M: -4.036 (2.576)	U: -0.031 (2.835) M: 3.008 (6.048) U*M: -0.504 (1.280)	U: -4.040 (4.498) M: -3.786 (8.507) U*M: 1.411 (1.960)	U: 2.488 (1.570) M: 2.735 (4.106) U*M: -1.510** (0.717) (M^ = 165)	U: 1.162 (0.761) M: 0.293 (2.121) U*M: -0.634* (0.361) (M^ = 183)	U: 0.063 (0.906) M: -0.650 (1.993) U*M: 0.141 (0.451)

Notes to Table 9:

Estimated with OLS. All models include county and year fixed effects. NDF have been aggregated by race and education. Observations are weighted by the number of births. SEs (in parentheses) have been corrected for heteroskedasticity and clustering at the county level. *, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. U is the unemployment rate and M the Medicaid eligibility threshold. M^{\wedge} indicates a “break-point” level of Medicaid eligibility (as a percent of the federal poverty line).

Table 10. The Effects of Unemployment on Cohort Characteristics; By Race

	Black	White
Y = f(Unemployment), 1989-1999, county-level		
Less than high school education	U: 0.082 (0.091)	U: -0.006 (0.031)
At least college education	U: 0.037 (0.056)	U: -0.002 (0.072)
Married	U: 0.425** (0.216)	U: 0.310*** (0.083)
30 <= age < 40	U: 0.395*** (0.085)	U: 0.093 (0.070)
Age >= 40	U: 0.044*** (0.012)	U: 0.055*** (0.019)
Y = f(Unemployment, Medicaid, Unemployment*Medicaid), 1989-1999, county-level		
Less than high school education	U: -0.066 (0.205) M: -0.207 (0.619) U*M: 0.082 (0.121)	U: 0.220** (0.090) M: 0.371 (0.269) U*M: -0.126** (0.053) (M^ = 175%FPL)
At least college education	U: 0.671*** (0.167) M: 1.545*** (0.469) U*M: -0.362*** (0.091) (M^ = 185%FPL, U^ = 4.26)	U: 0.929*** (0.158) M: 2.953*** (0.501) U*M: -0.528*** (0.856) (M^ = 176%FPL, U^ = 5.59)
Married	U: 0.610 (0.378) M: 0.822 (1.058) U*M: -0.112 (0.227)	U: 0.813*** (0.172) M: 1.914*** (0.522) U*M: -0.287*** (0.095) (M^ = 283%FPL, U^ = 6.66)
30 <= age < 40	U: 1.174*** (0.263) M: 2.235** (0.985) U*M: -0.451*** (0.147) (M^ = 260%FPL, U^ = 4.96)	U: 1.005*** (0.158) M: 2.832*** (0.532) U*M: -0.517*** (0.088) (M^ = 194%FPL, U^ = 5.48)
Age >= 40	U: 0.085*** (0.032) M: 0.099 (0.122) U*M: -0.024 (0.019)	U: 0.147*** (0.030) M: 0.361*** (0.110) U*M: -0.053*** (0.020) (M^ = 277%FPL, U^ = 6.80)

Estimated with OLS. All models include county and year fixed effects. NDF have been aggregated by race. Observations are weighted by the number of births. SEs (in parentheses) have been corrected for heteroskedasticity and clustering at the county level. *, **, and *** denote statistical significance at the 90%, 95%, and 99% confidence levels, respectively. U is the unemployment rate and M the Medicaid eligibility threshold. M^ indicates a "break-point" level of Medicaid eligibility (as a percent of the federal poverty line). U^ indicates a "break-point" level of the unemployment rate.

3. PATERNITY DEFERMENTS AND THE TIMING OF BIRTHS: U.S. NATALITY DURING THE VIETNAM WAR

3.1. Background

During the conflict in Vietnam, men between 18½ and 25 years of age were subject to the draft. Several exemptions to this rule existed. For example, students were exempt. Importantly for the purposes of this study, married men with dependents could also obtain a deferment from the draft, and the particulars of this policy underwent substantial changes in the 1960's. In August 1965, President Johnson issued Executive Order 11241, which formally eliminated deferments for childless men who got married after August 26, 1965, and, in October 1965, the Selective Service declared that childless married men (irrespective of the date of marriage) were to be called up. Both announcements came as a surprise (The New York Times, 1965a). Since married men with children remained exempt, the declarations provided a strong incentive for young couples to conceive a (first-born) child. Before August 1965, marriage had been a sufficient condition for a deferment. Even just a few hours before the August 26 midnight deadline, desperate couples tried to make use of this provision by quickly scheduling their wedding. Between August and October, couples that had missed the deadline had to satisfy an additional condition – conceiving a child. In October 1965, the risk of induction was further extended to *all* couples that had remained childless. Finally, in April 1970, the family deferments were entirely eliminated by Executive Order 11527 (The Selective Service System 2004).

Past research has demonstrated that taxes and expenditure programs can affect fertility (e.g., Whittington et al 1990; see also Milligan 2002 for an excellent review) as well as the timing of delivery (Dickert-Conlin and Chandra 1999). The goal of this paper is to extend this reasoning to a dramatic yet unexamined government intervention - the

effects of the Vietnam War paternity deferments on the decision to conceive a first-born child. As discussed in the popular press, Vice President Dick Cheney's first daughter, Elizabeth, was born nine months and two days after the Selective Service System announced that childless married men were to be drafted (The Boston Globe 2000, Slate Chatterbox 2004). Did other draft eligible men react to the announcement the way Vice President Cheney apparently did? And, if so, how fast was the response?

To my knowledge, no one has investigated the impacts of the Vietnam draft on natality. By using the Vietnam draft rules to identify a causal effect, however, I build on several prior studies. Joshua Angrist, for example, uses the exogeneity of the Vietnam draft rules to identify the effects of military service on lifetime earnings (Angrist 1990) and to measure the racial differences in the value of military service (Angrist 1991). Gullason (1989) and Card and Lemieux (2001, 2002) estimate the effects of the Vietnam draft on schooling explicitly recognizing that college attendance often served as a vehicle to avoid the draft. Both studies find a significant effect of the probability of being drafted on school enrollment.

The fact that the changes in the Selective Service rules were both unexpected and widely publicized makes this an ideal example to study the effects of policy on fertility decisions. Milligan (2002) argues that the assumptions made about the timing of the response to policy are arbitrary since a reaction will be delayed not only by a nine-month gestational lag but also by the time necessary for the diffusion of information about the change in policy. In the case examined here, however, the criticism seems less relevant. Newspaper clippings from August 27, 1965 suggest that the issuance of the Executive Order 11241 did receive broad attention. For example, the story was listed on the front

pages of *The New York Times* (1965c) and *The Washington Post* (1965b). The benefits of becoming a father were made explicit: “From now on, a draft-age man who gets married and becomes a father before being called into service will go into the same deferred class as other fathers.” (*The Washington Post*, 1965b, p. A12) Similarly, on October 27, 1965, one day after the Selective Service declared that childless married men were to be called up, the top U.S. newspapers commented on the policy change (*The New York Times*, 1965a; *The Washington Post*, 1965a).²³ It is reasonable to assume that the general public was well aware of the news.

Also, given the urgency of the situation for the potential draftees, any behavioral response was likely to be fast. In the mid 1960’s, the risk of induction facing young American men was increasing dramatically. In the year 1965, when the new policies were announced, the number of men inducted each month increased by more than sixfold (Figure 1). And, as anecdotal evidence suggests, young couples were ready to react almost immediately. For example, after President Johnson’s Executive Order was issued on August 26, 1965 limiting the eligibility for marital deferments to men married on or before that date, many couples quickly scheduled their wedding in order to beat the midnight deadline (*The New York Times*, 1965b).

Finally, information about the fecundity of the U.S. population in the early 1960’s confirms that young women were, on average, able to conceive a child quickly. In the year 1960, 52% of Americans 20-24 years old were able to conceive within a month from trying and 77% were successful within two months (Crist 2004). Thus, a fast and

²³ Unfortunately, only a few transcripts of television news are available for years prior to 1968 (*Vanderbilt Television News Archive*, *NBC News Archive*, and *Burrell’s Transcript Service*) and none of them is relevant to the issue at hand.

relatively strong reaction to the Executive Order issuance and the Selective Service announcement is realistic.

3.2. Data and Methods

To empirically investigate the effects of the Vietnam draft on natality, I focus on the impacts of President Johnson's Executive Order 11241 and the October 1965 Selective Service announcement and make use of the fact that these policies affected different groups of young men differently. In particular, I use a difference-in-differences type of approach and compare the effects of the policy changes on the behavior of treatment and control groups of young men.

Ideally, all American men in the draft-eligible age would constitute the treatment group. Unfortunately, however, the dataset most suitable for the study – the Vital Statistics of the United States (U.S. Department of Health, Education, and Welfare 1963-77) – does not provide detailed information on paternal characteristics.²⁴ Therefore, I use maternal age as a proxy for the father's age and adjust for the possibility of bias due to misclassification. In my baseline model, I use women 20-24 years old as the treatment group since only men up to 25 years of age were eligible for the draft and since women,

²⁴ I have explored several micro datasets but none of them was suitable for this study. For example, the Natality Detail File series only started in the year 1968. The Current Population Survey reports age in years but not the month of birth (making it impossible to focus on children as respondents) and only asks females questions related to fertility (making it impossible to link children to their fathers and to focus on fathers as respondents). The National Longitudinal Survey of Young Men (NLSYM) includes information on individuals 14 to 24 years old in the year 1966 but contains no appropriate control group. Also, the sample size in the NLSYM is too small to permit reliable inferences from a stratified analysis (for example, in the summer of 1966, sampled men 19-24 years old had only 22 first-born children). The Childbirth and Adoption History File of the Panel Study of Income Dynamics collected since 1985 does not contain enough first births in the control group to support reliable difference-in-differences estimation.

on average, tend to be younger than their partners (Table 1).²⁵ In an alternative specification, I add teenagers (15 to 19 years old) to the treatment group. Women 25-29 years old - with husbands likely to be 26 years old or older and thus ineligible for the Vietnam draft – comprise the control group.

To assess the validity of my treatment and control groups, I use the U.S. Natality Detail Files for the years 1969-1971²⁶ and calculate the percentages of fathers 19 to 25 years old (the draft eligible cohort) by maternal age (Table 2). Maternal age is a reasonably good proxy for paternal age. In particular, 65% of mothers 20 to 24 years old (the baseline treatment group) had babies with fathers 19 to 25 years old in each of the years 1969, 1970, and 1971. The corresponding percentage was 68% for women 15 to 24 years old (an alternative treatment group) and 11% for mothers in the 25 to 29 year old cohort (the control group). These estimates prove useful in adjusting for possible misclassification, as I discuss shortly.

Since the *existence* of children rather than their number played a role in determining draft eligibility, I focus on the birth of a first-born child when estimating the effects of the Executive Order and the Selective Service announcement. Also, the

²⁵ The Vital Statistics report the number of births for the following age cohorts: under 15 years, 15-19 years, 20-24 years, 25-29 years, 30-34 years, 35-39 years, 40-44 years, 45-49 years, and 50 years and over. I exclude teenagers from the baseline analysis since women under 15 years of age were unlikely to be affected by the government policy and since mothers 15-19 years old seem diverse with respect to their fertility responsiveness (the attitudes towards family planning will likely differ among women in this group). Also, my calculations suggest that between 17.3% (year 1963) and 26.7% (year 1968) of mothers 15-19 years old were single in the period under study. The corresponding estimates are 5.7% and 8.3% for mothers 20-24 years old. (<http://www.cdc.gov/nchs/data/statab/t941x18.pdf>, <http://www.cdc.gov/nchs/data/statab/t941x19.pdf>, <http://www.cdc.gov/nchs/data/statab/t941x07.pdf>, and <http://www.census.gov/popest/archives/pre-1980/PE-11.html>; Accessed 03/19/2006) In an alternative model specification, however, I add women 15-19 years old to the treatment group and the results remain qualitatively the same.

²⁶ These are the first years when the age of both parents was recorded.

outcome measure needs to be corrected for the overall effects of the war on fertility. In particular, it needs to isolate the potential changes in the number of first births in reaction to the new deferment rules from the overall changes in natality in a country where many young men had been sent to war. Therefore, I use the age-specific ratio of the number of first-born infants to all infants (reported by month and year of delivery) as the dependent variable. If the 1965 declarations had a significant effect on the fertility behavior of the potential draftees, the “first-born infants/all infants” ratio should increase in the summer of 1966 (about 9 months after the policy changes were enacted) for women in the treatment group and stay unchanged (or to increase less) for women in the control group. Thus, a comparison of the monthly “first-born infants/all infants” series (purged of a linear time trend and seasonal variation) for the treatment and control groups yields an estimate of the causal relationship between the new government draft policies and fertility.²⁷ More formally, I estimate:

$$Y_{ij} = \alpha + \beta * T_j + \gamma * M_t + \delta * T_j * M_t + \varepsilon_{ij},$$

where t indexes time periods (months from January 1963 to December 1968) and j indexes cohorts (treatment or control). Y is the detrended and deseasonalized “first-born infants/all infants” ratio, T is a dummy variable denoting the treatment group membership (age 20-24 in the baseline specification), and M is a set of dummy variables, one for each month following the first policy change (August 1965). $T * M$ are interaction dummies denoting the treatment group membership in months following the

²⁷ In one of my robustness checks below, I verify that the number of subsequent births is not driving my results.

policy changes, and ε is an error term. In the above model, the estimated δ 's on months 9 and 10 after each policy change are the difference-in-differences estimates of interest.

3.3. Results

3.3.1. Descriptive Analysis

Figure 2 plots the proportions of first births for American women 20 to 24 and 25 to 29 years old by month and year of delivery. From 1963 to 1968, the two ratios grew about linearly with only small deviations from the trend. The series, however, exhibited a spike in the summer of 1966 – approximately nine months after the new draft policies were announced. As hypothesized, the spike was more remarkable for the younger cohort.

Based on the descriptive analysis, it seems reasonable to focus on the relatively stable period from January 1963 to December 1968 when estimating the effects of the new draft policy on fertility. This time period includes several years preceding the Executive Order 11241 issuance (pre-August 1965) as well as several years following the expected effects of the new policies on fertility (post-July/August 1966). Limiting the period studied to the mid-1960's also simplifies the analysis by avoiding the substantial changes to the draft process associated with the introduction of the draft lottery in late 1969.²⁸ Finally, a relatively short follow up is sufficient for studying the immediate decision of affected young couples to conceive a first-born child. Due to the construction

²⁸ Beginning in 1970, young men were at risk of induction for only one year rather than for the entire period between ages 18½ and 25, as was the case previously. As Card and Lemieux (2001, 2002) note, the shortened period of exposure together with the relatively low rate of inductions after 1969 significantly reduced the incentives to pursue draft-avoidance strategies.

of the outcome measure – the proportion of first births to the total number of births - investigating long-term fertility dynamics would be complicated as the corrective decrease in the number of first deliveries and an increase in the number of second and subsequent deliveries would be difficult to separate out. For the purposes of this study, year 1968 therefore seems like a reasonable cutoff. Unfortunately, limiting attention to years prior to 1969 excludes the effects of the family deferment elimination of April 1970 from the analysis.

Since the two series of the first-birth ratio likely followed a different (linear) time trend in 1963 to 1968 and since their seasonal pattern might have also differed, appropriate detrending and deseasonalizing had to be performed.²⁹ A simple visual examination of the detrended and deseasonalized series (Figure 3) suggests that the government draft policies very likely did have a significant impact on the fertility of the potential draftees. In particular, while the residual ratios for the treatment and control groups followed a similar time path in the years 1963 to 1965, the treatment mothers experienced a much sharper increase in the proportion of first births in the summer of 1966. That the control mothers experienced any increase at all may stem from the fact that maternal age is an imperfect proxy for paternal age and so that some of the women in the control group might have also reacted to the draft. As further obvious from Figure 3, the two cohorts of mothers behaved somewhat differently towards the end of the studied period. More specifically, the treatment mothers had a lower residual ratio of first-born babies about 12 and 22 months after the 1966 spike. This is consistent with the fertility

²⁹ A continuous time variable and a full set of month dummies have been used. This approach is similar to that in Card and Lemieux (2001, 2002) who regress the annual education outcomes on a linear inter-cohort time trend when estimating the effects of the Vietnam draft on college attendance.

behavior (birth spacing in particular) prevalent in the U.S. at that time. Based on data from the Natality Detail Files for the years 1969-1971,³⁰ the distribution of the length of time between the first and the second live birth peaked at months 13 and 23 in the late 1960's and early 1970's (Figure 4). A decreased number of first births coupled with an increased number of subsequent births in the years 1967 and 1968 by women who had responded to the Vietnam draft by advancing their first delivery to summer 1966 may thus be responsible for the observed pattern.

3.3.2. Regression Results

To formally estimate the size and significance of the effect of the Vietnam draft rules on natality, I employ a difference-in-differences type of methodology. In the baseline specification of my model, I regress the detrended and deseasonalized “first-born infants/all infants” ratio on a dummy variable set equal to one for the treatment group, seven dummy variables set equal to one for months 8 to 14 after the August 1965 Executive Order issuance (i.e., months 6 to 12 after the October 1965 Selective Service announcement), and seven interaction dummies set equal to one for observations on the treatment group in the exposed months.³¹ If the new policies did induce young women to time the conception of their first-born child in order to make the baby's father exempt from the draft, the coefficients on the interaction dummies for months 9 and 10 after each

³⁰ These are the first years when the information on birth spacing was recorded by at least some states. Obtaining this information for the years 1966-1968 would have been preferable since, if the *number* of first births was exogenously affected by the policy change, birth *spacing* might have been affected as well. Nevertheless, the stability of the birth spacing distribution in the 1969-1971 period makes extrapolation to the earlier years seem justifiable.

³¹ As a robustness check, I have also estimated the main equation with detrending and deseasonalizing in one step. This modification had no substantial impact on the results.

of the new policies was announced should be positive and statistically significant. In addition, since the announcements were made on August 26 and October 26, 1965, even a quick response by the potential draftees would likely increase the number of infants born in June 1966 (10 months after the Executive Order issuance) and August 1966 (10 months after the Selective Service announcement) by more than the number of infants born in May and July 1966. Therefore, the coefficients on the interaction dummies for months 10 and 12 after the Executive Order issuance (i.e., months 8 and 10 after the October 1965 Selective Service announcement) should be larger in magnitude.

Results from my baseline OLS estimation are reported in the first column of Table 3. Two of the interaction variables are positive and significant at the 95% confidence level: the interaction dummies for months 10 and 12 after the Executive Order issuance, i.e., months 8 and 10 after the Selective Service announcement. The increased natality in June 1966 very likely represents a direct response to the Executive Order issuance and the increased natality in August 1966 is likely caused by the Selective Service announcement. Even though statistically insignificant, the proportions of first births among treatment women are higher in July, September, and October 1966 as well and the gap diminishes over time.

The second column of Table 3 reports results from a specification where teenagers (mothers 15-19 years old) are added to the treatment group. In this case, the interaction dummies for June, July, August, and September 1966 are all positive, large, and statistically significant. Taken together, the above results thus provide strongly suggestive evidence that the Vietnam War draft policy played a role in determining the timing, and perhaps the number, of births.

3.3.3. Correction for Misclassification

After estimating the baseline model, I explicitly acknowledge that some women might have been misclassified into the treatment and/or control group. A recent paper (Lewbel 2003) demonstrates that as long as the misclassification probabilities are known to the researcher (for example from a validation sample or from aggregate population proportions), the true average treatment effect can be calculated as: $\tau^* = \tau / (p_0 + p_1 - 1)$, where τ denotes the estimated (biased) treatment effect, p_0 is the proportion of untreated individuals in the control group, p_1 the proportion of truly treated individuals in the treatment group, and $p_0 + p_1 > 1$. (The Technical Appendix includes a more extensive discussion of this result and its use in adjusting my estimates.) As obvious from the above formula, the true treatment effect is zero if and only if the estimated treatment effect is zero. Furthermore, any misclassification into the treatment and/or control group will bias the estimated treatment effect downward. Therefore, my estimates of the effect of the deferment rules on natality are conservative. If, for example, 65% of women in the baseline treatment group and 11% of women in the control group had babies with men of the draft-eligible age (as suggested by the Natality Detail File estimates), the correct magnitude of the baseline coefficients on the interaction dummies for months 10 and 12 after the Executive Order issuance (i.e., months 8 and 10 after the Selective Service announcement) would be nearly double ($0.016 / (0.89 + 0.65 - 1) = 0.030$ and $0.017 / (0.89 + 0.65 - 1) = 0.031$, respectively). Similarly, in the specification where teenagers are added to the treatment group, the corrected statistically significant coefficients (months 10 to 13 after the Executive Order issuance) would be 0.040, 0.025, 0.037, and 0.026, respectively.

To attach meaning to these estimates, I calculate the predicted increase in the number of births. First, I consider the baseline case with no correction for misclassification. Using the actual number of deliveries obtained from the Vital Statistics suggests that the number of first births increased by 6,488 as a result of the new draft policy announcements.³² Next, using the corrected treatment effects and recognizing that 65% of mothers 20-24 years old and 11% of mothers 25-29 years old were “at risk” modifies the estimate to 8,283. And, finally, using the baseline estimates but taking into account that a fraction of the teenage group could have been affected by the new draft policies further increases the predicted effect to 15,532.

When teenagers are directly added to the treatment group, the magnitude of the estimated effect increases further. In particular, my results indicate that the number of first births might have increased by as many as 19,540 in June and August 1966. In fact, when all the statistically significant coefficients from the alternative specification are employed, the predicted number of additional first births delivered between June and September 1966 rises to 32,914.

3.3.4. Robustness Checks

To check the robustness of the baseline results, several alternative specifications of the difference-in-differences model are estimated.³³ First, I add dummy variables for

³² Let Y_1 denote the number of first births, Y_2 the number of subsequent births, and Z the policy change of interest. Then, $\tau = \partial(Y_1/Y_1+Y_2)/\partial Z = [(\partial Y_1/\partial Z) * (Y_1+Y_2) - Y_1 * (\partial Y_1/\partial Z + \partial Y_2/\partial Z)] / (Y_1+Y_2)^2$, where $\partial Y_2/\partial Z$ is assumed to be 0 (this assumption is verified in my analysis of subsequent births). Thus, $\partial Y_1/\partial Z = \tau * (Y_1+Y_2)^2 / Y_2$. Using the actual numbers of first and subsequent births to women 20-24 years old reported in the Vital Statistics yields: $\partial Y_1/\partial Z = (0.016 * 107,042^2 / 61,796) = 2,967$ (June 1966) + $(0.017 * 116,886^2 / 65,966) = 3,521$ (August 1966) = 6,488.

all the remaining months after the Executive Order issuance as well as their interactions with the treatment dummy to the baseline regression. This way, the Executive Order of August 1965 and the Selective Service announcement of October 1965 are allowed to have an effect on fertility throughout the entire period from September 1965 to December 1968. Results from this specification are very similar to those reported in the first column of Table 3.

Next, I consider the possibility that the trend in the “first-born infants/all infants” ratio was not linear (for either the treatment or the control cohort) in the mid-1960’s. To allow for this possibility, I follow Card and Lemieux (2001, 2002) and add a quadratic time variable to the simple linear time term and the full set of month dummies when detrending and deseasonalizing the original series. I then use the residuals from this analysis in the difference-in-differences type of model. The magnitude of the coefficients on the interaction dummies of interest (10 months after each of the policy changes) decreases only very slightly and both variables remain highly statistically significant. None of the other interaction dummies reaches statistical significance at the 95% confidence level. As before, the main conclusions do not change when the full model (with dummies for all months after September 1965) is estimated.

To verify the causality of the relationship, I also estimate the above models for an artificial (i.e., unreal) policy change. In particular, I assume that instead of being announced in the summer of 1965, the new draft rules were announced, alternatively, in the summer of 1962, 1963, 1964, 1966, or 1967. As hypothesized, the policy coefficients of interest never approach statistical significance in these models.

³³ Results from all alternative estimations are available upon request.

Further, to verify that the number of subsequent births is not driving my results, I use the number of subsequent births instead of the “first-born infants/all infants” ratio as the dependent variable. As expected, there is no difference between the treatment and control groups of mothers following the policy changes.

Also, since the use of the “first-born infants/all infants” ratio imposes a functional restriction on the model, I replace this variable with the number of first birth and add the number of subsequent births (as well as its interaction with the treatment group membership) on the right-hand side (Table 4). Both of the new regressors are positive and highly statistically significant but the main results remain qualitatively the same. The magnitude of the estimates is very similar as well. In particular, the new results indicate that the number of first births increased by 2,576 and 3,759 in June and August 1965, respectively. The sum of these two effects, i.e., 6,355 additional first births, is very close to the 6,488 additional births predicted by the baseline model (without correction for misclassification).

Finally, in order to formally test the joint hypothesis that the proportion of first births increased significantly in months 9 and 10 after each of the policy changes, I replace the individual dummies for months 9 to 12 after the Executive Order issuance (i.e., months 7 to 10 after the October 1965 Selective Service announcement) by a single dummy variable. As expected, the coefficient on this variable interacted with the treatment group membership is large and highly statistically significant ($\hat{\delta}=0.010$, $SE=0.004$ for the baseline treatment group and $\hat{\delta}=0.017$, $SE=0.004$ for the treatment group including teenagers). Other coefficients in the model are unaffected by this change.

3.4. Conclusions

The magnitude of the effect of the Vietnam War paternity deferments on the decision to start a family estimated in this paper is quite substantial. In particular, the calculated conservative increase in the number of first births by 15,532 in June and August 1966 represents over 7% of the total number of first deliveries in those two months. It also corresponds to about 28% of the Selective Service System calls for inductees in those months (The Selective Service System 1968). This finding adds to a growing body of evidence that government interventions may indeed affect individuals' reproductive behavior. It also adds to the list of potentially long lasting effects of the Vietnam War draft policies.

An interesting question that remains is to what extent the increase in the number of births in the summer of 1966 translated into an increase in completed fertility and to what extent it represented a mere change in birth timing. Unfortunately, this issue is difficult to address with existing data.³⁴ The consequences of either change - in terms of maternal education, labor market behavior, marital decisions, maternal and child health,

³⁴ I have examined the CPS 1995 Fertility and Marital History Supplement and obtained the distribution of the lifetime number of births (completed fertility) for women 20-24 years old at their first delivery whose first child was born in the summer of 1966. I then compared this distribution to the corresponding distributions for women whose first delivery occurred in the summers of 1962-1965 and 1967-1970. Unfortunately, the number of observations (about 80 each year - 753 in total) was too low to enable reliable comparisons. Furthermore, the methodology used made it impossible to study the proportion of women with no births. This is an important limitation since, as Ananat et al. (2004) note, zero is the only point in the fertility distribution for which there is an unambiguous prediction: in the case examined here, the proportion of childless women should fall. Following Ananat et al. (2004), I have therefore considered a complementary approach. In particular, I have used the method of cohort analysis to study completed fertility of women 10-14, 15-19, 20-24 (the exposed group), 25-29, and 30-34 years old in the year 1966. For the purposes at hand, however, this analysis proved too crude as the general decline in fertility over time strongly dominated any other fertility pattern.

and other outcomes – could potentially be important.³⁵ Thus, by influencing natality, the draft deferments likely had other long lasting effects.

³⁵ See, for example, Rosenzweig and Schultz (1985), Shapiro and Mott (1994), Angrist and Evans (1998), and Jacobsen et al. (1999) for the effects of childbearing on women's labor supply and earnings and Royer (2004) for the effects of maternal age on infant health.

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Figure 1.

**Monthly Number of Inductions
United States, 1963-1966**

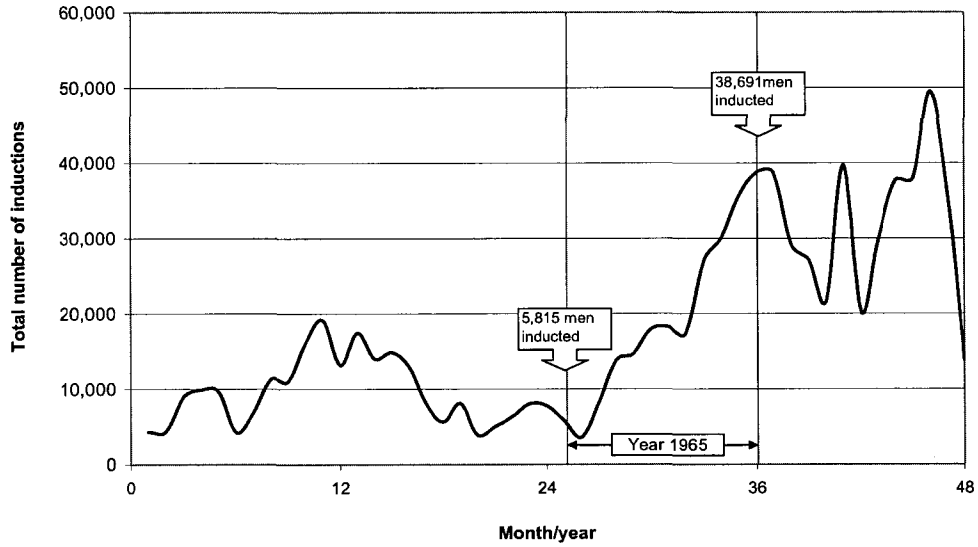


Figure 2.

**Proportion of 1st Births by Month and Year of Birth
United States, 1961-1970**

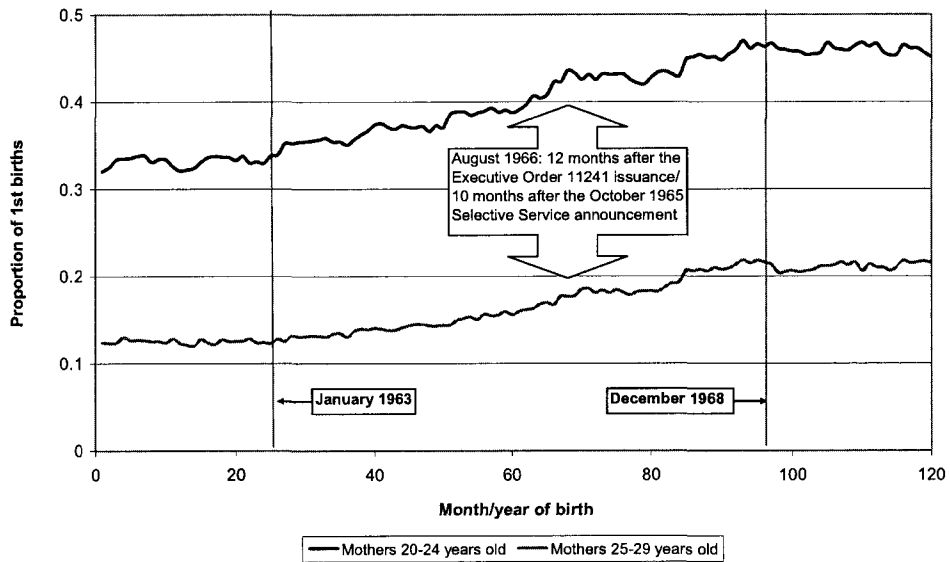


Figure 3.

**Proportion of 1st Births by Month and Year of Birth
United States, 1963-1968; Detrended and Deseasonalized**

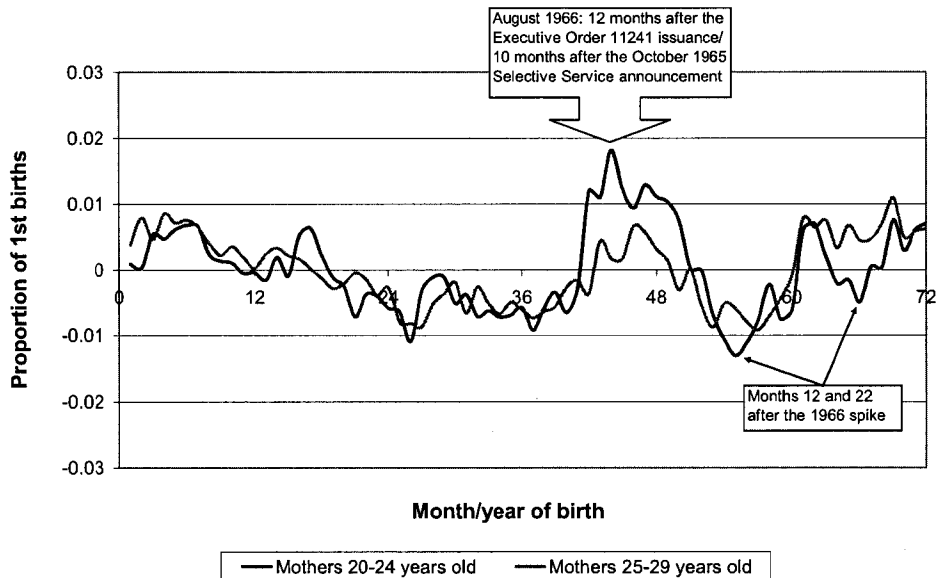
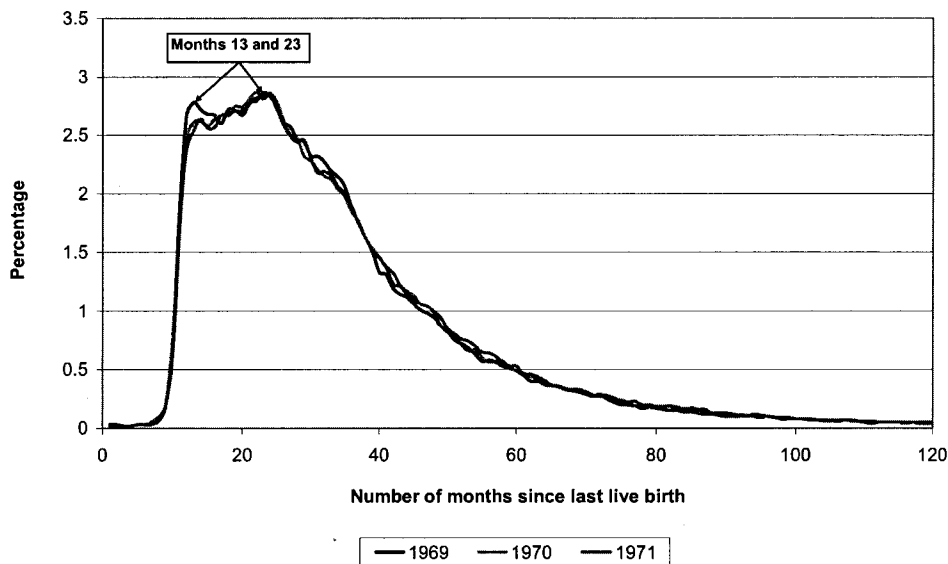


Figure 4.

**Interval Since Last Live Birth for Second Births
United States, 1969-1971**



Source: Natality Detail Files 1969-1971

**Table 1. Median Age of Brides and Grooms at the Time of First Marriage
United States, 1963-1968**

Year	Median age of brides	Median age of grooms	Difference in median age
1963	20.3	22.5	2.2
1964	20.5	23.0	2.5
1965	20.4	22.5	2.1
1966	20.3	22.6	2.3
1967	20.8	22.9	2.1
1968	20.6	22.4	1.8

Source: National Center for Health Statistics, CDC (1967-71)

**Table 2. Percentages of Fathers 19 to 25 Years Old by Mother's Age
United States, 1969-1971**

Year		1969	1970	1971
% missing info on father's age		9.18	9.70	9.89
Mother's age cohort	20 to 24 years old (baseline treatment group)	64.82	65.10	65.46
	15 to 24 years old (alternative treatment group)	68.30	68.33	68.62
	25 to 29 years old (control group)	11.18	10.84	11.20

Source: U.S. Dept. of Health and Human Services, National Center for Health Statistics (1970-72)

**Table 3. The Effects of the Vietnam War Paternity Deferments on the Proportion of
1st Births; United States, 1963-1968; OLS Estimation**

Variable	Parameter estimate	
	Baseline treatment group (20-24 years old)	Alternative treatment group (15-24 years old)
Treatment cohort	-0.001 (0.001)	-0.001 (0.001)
8 months after the Executive Order 11241 issuance/ 6 months after the October 1965 Selective Service announcement	-0.003 (0.006)	-0.003 (0.005)
9 months after the Executive Order 11241 issuance/ 7 months after the October 1965 Selective Service announcement	-0.001 (0.006)	-0.001 (0.005)
10 months after the Executive Order 11241 issuance/ 8 months after the October 1965 Selective Service announcement	-0.004 (0.006)	-0.004 (0.005)
11 months after the Executive Order 11241 issuance/ 9 months after the October 1965 Selective Service announcement	0.004 (0.006)	0.004 (0.005)
12 months after the Executive Order 11241 issuance/ 10 months after the October 1965 Selective Service announcement	0.002 (0.006)	0.002 (0.005)

13 months after the Executive Order 11241 issuance/ 11 months after the October 1965 Selective Service announcement	0.002 (0.006)	0.002 (0.005)
14 months after the Executive Order 11241 issuance/ 12 months after the October 1965 Selective Service announcement	0.007 (0.006)	0.007 (0.005)
8 months after the Executive Order 11241 issuance/ 6 months after the October 1965 Selective Service announcement * Treatment cohort	-0.003 (0.008)	0.007 (0.008)
9 months after the Executive Order 11241 issuance/ 7 months after the October 1965 Selective Service announcement * Treatment cohort	-0.001 (0.008)	0.011 (0.008)
10 months after the Executive Order 11241 issuance/ 8 months after the October 1965 Selective Service announcement * Treatment cohort	0.016** (0.008)	0.023*** (0.008)
11 months after the Executive Order 11241 issuance/ 9 months after the October 1965 Selective Service announcement * Treatment cohort	0.007 (0.008)	0.014* (0.008)
12 months after the Executive Order 11241 issuance/ 10 months after the October 1965 Selective Service announcement * Treatment cohort	0.017** (0.008)	0.021*** (0.008)
13 months after the Executive Order 11241 issuance/ 11 months after the October 1965 Selective Service announcement * Treatment cohort	0.011 (0.008)	0.015** (0.008)
14 months after the Executive Order 11241 issuance/ 12 months after the October 1965 Selective Service announcement * Treatment cohort	0.003 (0.008)	0.005 (0.008)

The dependent variable is the linearly detrended and deseasonalized proportion of first births to all U.S. births. An intercept term (not statistically significant) was included in the model. Standard errors are given in parentheses. ***, **, and * denote statistical significance at the 99%, 95%, and 90% confidence levels, respectively.

Table 4. The Effects of the Vietnam War Paternity Deferments on the Number of 1st Births; United States, 1963-1968; OLS Estimation

Variable	Parameter estimate
	Baseline treatment group (20-24 years old)
Treatment cohort	455.19 (6423.44)
Number of subsequent births	0.31*** (0.05)
Number of subsequent births* Treatment cohort	0.35*** (0.09)
8 months after the Executive Order 11241 issuance/ 6 months after the October 1965 Selective Service announcement	-20.28 (950.42)
9 months after the Executive Order 11241 issuance/ 7 months after the October 1965 Selective Service announcement	419.63 (978.87)
10 months after the Executive Order 11241 issuance/ 8 months after the October 1965 Selective Service announcement	140.84 (969.92)
11 months after the Executive Order 11241 issuance/ 9 months after the October 1965 Selective Service announcement	1358.64 (1015.25)
12 months after the Executive Order 11241 issuance/ 10 months after the October 1965 Selective Service announcement	862.29 (982.75)
13 months after the Executive Order 11241 issuance/ 11 months after the October 1965 Selective Service announcement	841.22 (976.81)

14 months after the Executive Order 11241 issuance/ 12 months after the October 1965 Selective Service announcement	1357.21 (978.84)
8 months after the Executive Order 11241 issuance/ 6 months after the October 1965 Selective Service announcement * Treatment cohort	-1256.40 (1344.63)
9 months after the Executive Order 11241 issuance/ 7 months after the October 1965 Selective Service announcement * Treatment cohort	-966.06 (1379.01)
10 months after the Executive Order 11241 issuance/ 8 months after the October 1965 Selective Service announcement * Treatment cohort	2576.34* (1391.64)
11 months after the Executive Order 11241 issuance/ 9 months after the October 1965 Selective Service announcement * Treatment cohort	1476.56 (1446.25)
12 months after the Executive Order 11241 issuance/ 10 months after the October 1965 Selective Service announcement * Treatment cohort	3758.99*** (1407.67)
13 months after the Executive Order 11241 issuance/ 11 months after the October 1965 Selective Service announcement * Treatment cohort	2430.89* (1382.85)
14 months after the Executive Order 11241 issuance/ 12 months after the October 1965 Selective Service announcement * Treatment cohort	1134.80 (1372.37)

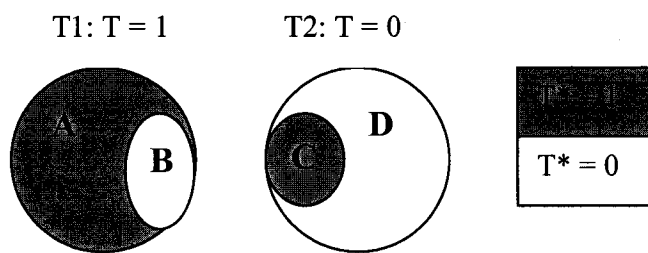
The dependent variable is the linearly detrended and deseasonalized number of first U.S. births. An intercept term was included in the model. Standard errors are given in parentheses. *** and * denote statistical significance at the 99% and 90% confidence levels, respectively.

Technical Appendix: Estimation of Average Treatment Effects with Misclassification

Based on: Lewbel, Arthur. 2003. "Estimation of Average Treatment Effects with Misclassification," Working Paper. (Especially "Identification by Known Misclassification Probabilities" – pp. 5-8 and "Proof of Theorem 1" – pp. 24-25) - <http://www2.bc.edu/~lewbel/mistrea11.pdf>

Notation:

Y	observed outcome
T*	actual treatment
T	reported treatment
t = 1	receiving treatment
t = 0	no treatment
Y(t)	outcome from treatment T* = t
X	vector of observable covariates



Definitions:

$$p_0(x) = E[I(T^*=0)|X=x, T=0] = D/(C+D) = D/T2 \quad (= 0.89)$$

$$p_1(x) = E[I(T^*=1)|X=x, T=1] = A/(A+B) = A/T1 \quad (= 0.65)$$

→ the relative sizes of groups "T=1" and "T=0" do not matter for the calculation of $p_0(x)$ and $p_1(x)$

$$b_0(x) = E[I(T=1)|X=x, T^*=0] = B/(B+D)$$

$$b_1(x) = E[I(T=0)|X=x, T^*=1] = C/(A+C)$$

→ the relative sizes of groups "T=1" and "T=0" do matter for the calculation of $b_0(x)$ and $b_1(x)$

$$r^*(x) = E[T^*|X=x]$$

$$h_t^*(x) = E[Y|X=x, T^*=t] = (\text{from assumption \#2 below}) E[Y|X=x, T^*=t, T]$$

$$\tau^*(x) = E[Y|X=x, T^*=1] - E[Y|X=x, T^*=0] = h_1^*(x) - h_0^*(x) = (\text{from assumption \#1 below}) E[Y(1)-Y(0)|X=x] \rightarrow \text{the average treatment effect}$$

$$\tau(x) = E[Y|X=x, T=1] - E[Y|X=x, T=0]$$

Assumptions (pp. 5-7 in Lewbel 2003):

1. unconfoundedness: $E[Y(t)|T^*, X] = E[Y(t)|X] \rightarrow$ treatment group membership has no effect on outcomes other than through the effects of treatment itself \rightarrow O.K.
2. $E[Y|X, T^*, T] = E[Y|X, T^*] \rightarrow$ assignment into the treatment group has no effect on outcomes when true treatment group membership is controlled for \rightarrow O.K.
3. i) $b_0(x) + b_1(x) < 1$
 $(b_0(x) + b_1(x) = 0.35*T1/(0.35*T1+0.89*T2) + 0.11*T2/(0.11T2+0.65T1)$
 $= (0.0385*T1*T2 + 0.2275*T1^2 + 0.0385*T1*T2 + 0.0979 T2^2)/$
 $(0.0385*T1*T2 + 0.2275*T1^2 + 0.5785*T1*T2 + 0.0979 T2^2) < 1)$
 \rightarrow O.K.
- ii) $E[T^*|X=x, T=1] \neq E[T^*|X=x, T=0]$
 $(E[T^*|X=x, T=1] = 0.65 \neq E[T^*|X=x, T=0] = 0.11)$
 \rightarrow O.K.
- iii) $0 < r^*(x) < 1$
 $r^*(x) = (A+C)/(A+B+C+D) \rightarrow$ O.K.
4. $\tau(x)$ is identified, i.e., consistently estimated

Derivation (pp. 24-25 in Lewbel 2003):

$$E[Y|X=x, T=1] = E[Y|X=x, T=1, T^*=0]*Pr(T^*=0|X=x, T=1) + E[Y|X=x, T=1, T^*=1]*Pr(T^*=1|X=x, T=1) = h_0^*(x)[1 - p_1(x)] + h_1^*(x) p_1(x)$$

$$E[Y|X=x, T=0] = E[Y|X=x, T=0, T^*=0]*Pr(T^*=0|X=x, T=0) + E[Y|X=x, T=0, T^*=1]*Pr(T^*=1|X=x, T=0) = h_0^*(x) p_0(x) + h_1^*(x)[1 - p_0(x)]$$

$$\begin{aligned} \rightarrow \tau(x) &= h_0^*(x)[1 - p_1(x)] + h_1^*(x) p_1(x) - h_0^*(x) p_0(x) - h_1^*(x)[1 - p_0(x)] \\ &= h_0^*(x)[1 - p_0(x) - p_1(x)] + h_1^*(x)[-1 + p_0(x) + p_1(x)] = [h_1^*(x) - h_0^*(x)][p_0(x) + p_1(x) - 1] \\ &= \tau^*(x) [p_0(x) + p_1(x) - 1] \end{aligned}$$

$$\rightarrow \underline{\tau^*(x) = \tau(x) / [p_0(x) + p_1(x) - 1]}$$

$$(\tau^*(x) = \tau(x) / [0.89 + 0.65 - 1] = \tau(x) / 0.54)$$