## The Impact of Diabetes Mandates on Infant Health

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#### Abstract

Among the factors suspected for the lagging improvements in infant health in recent period are increasing obesity and diabetes prevalence among women of childbearing age. This paper investigates the impact of mandated insurance coverage for diabetes on adverse pregnancy outcomes. Among infants born to educated women, who have high rates of coverage through private insurance affected by mandates, diabetes mandates are associated with a reduction in premature births and a decrease in low birth weight prevalence. These gains are concentrated among older women and are larger for African-Americans. There is a weaker effect on the prevalence of high birth weight (>4000 grams), potentially due to the deleterious effects of an increased probability of weight gain in excess of 35 pounds among diabetic women in mandates states.

Keywords: diabetes insurance mandates, prematurity, low birth weight, moral hazard

JEL Classifications: I18, I13, K32

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### INTRODUCTION

Despite significant gains made during the latter part of the 20<sup>th</sup> Century<sup>1</sup>, United States lags behind other developed countries in improvements in infant health outcomes.<sup>2</sup> Certain trends over time are particularly concerning: between 1990 and 2005 the preterm birth rate as well as the prevalence of low birth weight in the United States has increased (CDC, NCHS data). Moreover, during the period of 2000-2005 the United States experienced the first sustained period of lack of decline in infant mortality rates since the 1950s (MacDorman et al., 2008). Increased use of assisted reproductive therapies explains part of these trends, as do maternal characteristics such as increased age, incidence of obesity, and incidence of diabetes.

In particular, some demographic trends are especially worrisome because they represent preventable sources of poor infant outcomes. The incidence of diabetes among women of childbearing age more than doubled between 1980 and 2009 to reach approximately 4%.<sup>3</sup> In addition, there is also evidence of an increase in the incidence of gestational diabetes (GDM). A Northern California study found that GDM prevalence increased by ~50% between 1991-1997 (from 5.1% to 7.4%) (Ferrara et al, 2002) while a Colorado study reported an increased in prevalence of ~95% between 1994-2002 (Dabelea et al, 2005). Such changes in diabetes prevalence can explain why there was twice the number of births to women with diabetes in 2005 than there was in 1999 (Lawrence et al, 2008).

The increase in the incidence of diabetes among future mothers mirrors a wider trend of increased diabetes prevalence in total population. This trend along with the high health care costs of complications from diabetes caught policy makers' attention. Poor information about complications from diabetes, or lack of access to medical means of diabetes management could explain the relatively high incidence of complications. In response to a perceived need for improved management of diabetes, many states enacted legislation requiring health care

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<sup>&</sup>lt;sup>1</sup> From 1981 to 1989 the infant mortality rate declined at an average of 2 percent per year (Arias et al 2003). Over the more recent period, 1990 to 2001, the infant mortality rate experienced an average decrease of 3 percent per year (Kochanek, et al, 2002)

<sup>&</sup>lt;sup>2</sup> For instance, in 2005 the United States ranked 30<sup>th</sup> in infant mortality behind such countries as the Czech Republic or Cuba (NCHS, 2009)

<sup>&</sup>lt;sup>3</sup>Data Source: Centers for Disease Control and Prevention (CDC), National Center for Health Statistics, Division of Health Interview Statistics, data from the National Health Interview Survey. Data computed by personnel in CDC's Division of Diabetes Translation, National Center for Chronic Disease Prevention and Health Promotion. Data retrieved from http://www.cdc.gov/diabetes/statistics/incidence/fig5.htm on July 28, 2011.

insurance plans to provide coverage for the equipment, supplies, services and medications used for treating diabetes without charging higher premiums for coverage.

While improved coverage of diabetes allows for better management of diabetes, mandates also generate incentives for individuals to substitute away from preventive measures such as healthy eating and exercise. The presence of moral hazard makes for less than clear-cut theoretical predictions of the impact of mandates on public health. Empirically, Klick and Stratmann (2007) found evidence that diabetes mandates lead to an increase in obesity rates among people with diabetes. However, for a comprehensive understanding of the costs and benefits of these policies, any cost-benefit analysis should account for the impact on all affected categories and not only people with diabetes. Another group to consider are infants born to mothers with diabetes. As shown in the literature review below, uncontrolled diabetes is associated with higher incidence of macrosomia, prematurity, and low birth weight. Because of their high medical and future cost, these represent a significant component of the impact of diabetes and, thus, diabetes mandates.

When diabetes mandates are associated with an increase in obesity rates and, as shown below, both mothers' diabetes and obesity status have negative effects on infant outcomes, the net effect of diabetes mandates on pregnancy outcomes is theoretically ambiguous. Assessing the impact of better medical care when accompanied by poorer preventive-care (moral hazard) is of first-order importance in understanding how to effectively address the issue of poor infant outcomes derived from the obesity-diabetes nexus through policies that affect women's health during their fertile years. Our results are also a contribution to the literature documenting the impact of mandated insurance benefits and can provide some insight into the possible effects of federal health reform. Surprisingly, while there is substantial literature documenting the effect of insurance mandates on utilization (diabetes treatment Li et al., 2010; infertility treatment Schmidt, 2007, Bitler and Schmidt, 2012; mental health care Harris et al., 2006, Busch and Barry, 2008, Pacula and Sturm 2000; mammography Bitler and Carpenter, 2011; Pap tests Bitler and Carpenter, 2012) the literature on the effect on health outcomes is sparse (Kick and Stratmann, 2007).

In this paper, we use 1992-2003 panel data from NCHS's Vital Statistics Natality Data to evaluate the population impact of diabetes mandates on infant health. Our econometric approach builds on triple difference (DDD) procedures commonly used in policy evaluation literature

(Meyer, 1995; Gruber, 1994; Bertrand, Duflo, and Mullainathan, 2004). In this framework the treatment group consists of infants born to diabetic mothers, while all other infants serve as the control group. We compare changes in the outcomes of infants born to diabetic mothers versus infants born to non-diabetic mothers in states that enacted diabetes mandates pre- to post-adoption with changes in the same groups and time periods in states that did not enact such mandates.

Because health insurance mandates only apply to private insurance plans, diabetes mandates will only apply to a portion of the population, namely people with diabetes covered by private health insurance. Thus, the impact may be hard to identify in the total population. We focus our analysis on a specific subgroup: infants born to diabetic mothers with more than 12 years of education, known to be more likely to have private insurance. <sup>4,5</sup> Our results suggest that diabetes mandates significantly decreases the prevalence of low birth weight and prematurity but does not significantly change the incidence of high birth weight among women with more than 12 years of education. These effects are plausibly larger where more women are covered through private fully insured health plans and are smaller and insignificant in the case of births to women with 12 or fewer years of education or to non-diabetic women.

#### 1 THEORETICAL PERSPECTIVES AND LITERATURE REVIEW

We briefly summarize the relevant literature in this section. We begin by discussing the likely effect of diabetes insurance on private markets and the expected effect of diabetes mandates. We then present existing evidence of the effect of diabetes on pregnancy outcomes. To put our study into perspective we also briefly discuss previous studies on the economic impact of adverse pregnancy outcomes.

### 1.1. Diabetes Mandates – Conceptual Framework

The high incidence of diabetes in US population along with the high cost of complications due to poor disease management made for a total cost of diabetes in terms of direct medical care and indirect productivity losses of approximately \$174 billion in 2007 (2011 National Diabetes Fact

<sup>&</sup>lt;sup>4</sup> Note that insurance status is endogenous because added diabetes coverage with no increase in cost likely makes coverage more attractive. Thus a separation by insurance status would not be appropriate because the treatment group would change from pre- to post-period.

Some estimates of the impact of diabetes mandates on infants born to women with 12 years of education or less are reported in Table 6. Further estimates obtained using this sample are reported in the Supplemental Results Appendix available on request.

Sheet). It is this high cost that led the majority of states' legislatures to pass laws mandating that health insurance providers cover supplies, services, medications, and equipment for treating diabetes as part of their basic coverage without charging higher premiums for the coverage (see Table 1).

Like any insurance problem, there are two possible opposing effects of providing diabetes coverage. First, because people can get medical treatment much more cheaply than before, they will be more likely to use this treatment and thus become healthier. Second, the provision of health insurance has the potential to create moral hazard problems.<sup>6</sup> In short, insurance lowers the price of medicine, which causes some people to move from non-medical solutions such as healthy life styles to medical solutions (Kahn, 1999).<sup>7</sup> Should this substitution occur, some of the gains from medication would be offset and the costs savings from reduced medical care after including diabetes medication in their coverage would not be as high as originally predicted.

As a result, private health insurance that included diabetes care as a benefit would have a higher price after factoring in this moral hazard problem than if it hadn't been considered. Moreover, sometimes the moral hazard can be difficult to quantify. Thus, it may be problematic, even if moral hazard is recognized, to determine the optimal pricing structure. Possible methods that could be undertaken are for insurance companies to just not insure diabetes medicine at all, to price insurance which includes diabetes medicine substantially higher even for those who are not substituting this medicine for living a "healthy life style" or, to refuse to insure any other side effects of poor health habits.

It is here where arguments for government intervention seem to spring. When insurance companies attempt to avoid these moral hazard effects by either not including certain conditions in the insurance policy or by charging what is perceived as being an extraordinary high price for insurance that covers it, there is a tendency to think that this will be very costly to society in many ways. For example, there is likely to be large medical costs that society must bear if people cannot afford this insurance; there is the cost to society by the drop in productivity by those who have this diabetes; and there can be large political costs to legislators who do not pass mandates when there is interest group pressure. Those who worry about such costs often suggest

<sup>6</sup> See Pauly (1974), Neiman (1999), and Zweifel and Manning (2000).

<sup>&</sup>lt;sup>7</sup> This is opposed to using medication as a complement to healthy living.

that governments should mandate that insurance companies cover diabetes treatment at a reasonable price. It is the long term costs and benefits of these mandates that is the focus of this paper.

In practice, diabetes mandates require private insurance companies to provide coverage of medication, equipment, supplies and sometimes education for at-home treatment. Noting that some states do not in fact always define what each of the above terms means, medication usually implies coverage of insulin, glucagon, but also other prescriptive medication, while equipment and supplies could include coverage of insulin syringes, blood glucose monitors, insulin infusion devices, podiatric appliances to prevent complications associated with diabetes, visual reading and urine test strips, lancets and lancet devices, and injection aids. The coverage sometimes includes outpatient self-management training, and education and medical nutrition therapy. People with diabetes likely to be affected by mandates generally belong to employer-sponsored group health plans or individual health plans.<sup>8</sup>

Diabetes mandates are likely to be associated with the same problems detailed above. Previous literature (Klick and Stratman, 2007) found evidence that diabetes mandates are associated with an increase in Body Mass Index (BMI) of people with diabetes. However, estimating the direct impact on people with diabetes may understate/overstate the actual effect of the mandates if third parties are affected. Another group to consider is pregnant women with diabetes and the health of their infants. In this paper, our focus will be on low/high birth weight babies as one of the potential effects of diabetes mandates.<sup>9</sup>

### 1.2. The Effect of Diabetes on Pregnancy Outcomes

During pregnancy women tend to develop hypoglycemia<sup>10</sup> between meals and during sleep. However, if the maternal insulin response is inadequate, hyperglycemia results and the mother experiences recurrent postprandial hyperglycemic episodes. These episodes are a significant source of accelerated growth of the fetus. High fetal glucose levels are accompanied by fetal hyperinsulinemia, which promotes excess nutrient storage, resulting in macrosomia. Numerous studies, of which for the sake of brevity we will mention only few, find that poor diabetes control

<sup>&</sup>lt;sup>8</sup> The Employment Retirement Income Security Act (ERISA) limits the role of state mandates. State health insurance laws only apply to the fully insured plans in which mostly individuals and small and medium sized businesses and organizations pay premiums.

<sup>&</sup>lt;sup>9</sup> Very low birth weight (VLBW) occurs at less than 1500 grams; low birth weight (LBW) occur at less than 2500 grams, high birth weight (HBW) occurs at greater than 4000 grams and very high birth weight (VHBW) at greater than 4500 grams.

<sup>&</sup>lt;sup>10</sup> plasma glucose mean = 65-75 mg/dL

during pregnancy is associated with macrosomia (Jovanovic-Peterson et al., 1991; Combs et al., 1992; Ostlund et al., 2003). In addition, maternal obesity, which is common among people with type 2 diabetes, also has a strong effect on fetal macrosomia (Ehrenberg et al, 2004; Yogev and Langer, 2008, Owens et al, 2010).

This is a potentially significant cost for the society because high birth weight is a risk factor for increased emergency visits (Sin et al, 2004), for child obesity (Danielzik et al, 2004), diabetes (Harder et al, 2007), 11 cancer (Hjalgrim et al., 2003; Harder et al., 2008), rheumatoid arthritis (Mandl et al., 2009), asthma (Yuan et al., 2002; Remes et al. 2008), and atopic dermatitis (Kerkhof et al., 2003). There is also some evidence that high birthweight is associated with poorer cognitive function (Richards et al., 2001), reading difficulties (Kirkegaard et al, 2006), and lower test scores (Cesur and Rashad, 2010). Note however that while there have been many articles associating high birth-weight with present and/or future medical problems, there has been little research examining the economic costs of these medical problems.

Although most fetuses of diabetic mothers exhibit growth acceleration, growth restriction occurs with significant frequency in pregnancies in women with preexisting diabetes. The effect is explained by underlying maternal vascular disease associated with diabetes. Previous literature found that maternal diabetes is correlated with low birth-weight (Rosenberg et al, 2005; McDonald et al, 2010). Diabetes during pregnancy was also found to be associated with higher incidence of pre-term labor (Hedderson et al, 2003). These effects are again compounded by obesity as researchers found a positive correlation between obesity and both pre-term and low birth weight babies (Naeye, 1990; Chen et al, 2009).

An extensive literature has examined the costs associated with pre-term and low birth-weight babies. These costs include both higher medical costs (McCormick et al, 1991; Gilbert et al, 2003; Tomiska et al, 2003; and Schmitt et al, 2006) as well as less education (Corman and Chaikind, 1998; Conley and Bennett, 2001; Tsou et al., 2008), poorer longer term health (Paneth, 1995) and economic outcomes (Currie and Hyson, 1998; Behrman and Rosenzweig, 2004; Black et al, 2007; Behrman and Butler, 2007; Johnson, forthcoming). Note that the above is by no means a comprehensive description of the literature; those who are interested in a more extensive

<sup>&</sup>lt;sup>11</sup> Harder et al (2007) find evidence of a U-shaped relationship between birth weight and risk of type 2 diabetes with equal increase in risk in both tails of the distribution relative to babies with birth weights between 2500 and 4000 grams.

grams.

12 In particular, pregnant women with diabetes-associated retinal or renal vasculopathies and/or chronic hypertension are most at risk for growth restriction.

review of the costs associated with low birth-weight should find Zupanic (2007), Petrou et al (2001) and Hack et al (1995) worthwhile reading.

While all studies agree that the cost of low birthweight babies is likely substantial, aggregating these cost estimates of several different factors into a total cost estimate has produced quite varying results. The National Academy of Science estimated the total cost of pre-term babies in 2005 was "at least \$26.2 billion or \$51,600 per infant". Alderman and Behrman (2006) find a "benefit of \$510 per infant moved from the low-birth weight status, amounting to a total benefit of \$180,709,830 in 2007. Lewit et al (1995) estimates the cost of low birth weight in 1988 of being between \$5.4 and \$6 billion, about 2 times as high as AIDS and around 2/3 of the cost of alcohol abuse. On the other hand Almond et al (2005) suggest that it is quite possible that "existing estimates overstate the true costs and consequences of low birth weight by at least a factor of four and by as much as a factor of 20."

Thus the cost of diabetes to third parties could be quite substantial and the benefit from better control of diabetes significant. Nevertheless when diabetes mandates are associated with weight gain, and diabetes and obesity are independent factors in infant outcomes (Ehrenberg et al., 2004; Rosenberg et al., 2005; Knight et al., 2011) the actual effect of diabetes mandates on high birth-weight and pre-term and low birth weight is theoretically ambiguous and remains to be answered empirically. Note that the hypothesis that diabetes mandates can lead to a significant change in high and low birth weight prevalence, and thereby have a substantial long term benefit has to jump through two hoops to be confirmed. First, it must be the case that poor diabetes management is due to poor access to medical care and not completely determined by behavioral factors. There is evidence that state diabetes mandates are associated with higher utilization rates of diabetes management devices (Li et al, 2010), an indication that not all

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<sup>&</sup>lt;sup>13</sup> This included \$16.9 billion for medical care, \$1.9 b for maternal delivery costs, \$611 m for early intervention costs, \$1.1 b for disabling conditions such as hearing and mental retardation and \$5.7 b for lost labor market productivity associated with the disabilities caused by low birth-weight.

productivity associated with the disabilities caused by low birth-weight.

14 They admit, however, that their estimates are focused on, for the most part, estimated gains in labor productivity and avoiding infant medical costs as well as the cost of infant deaths.

<sup>&</sup>lt;sup>15</sup> In a later paper Almond et al (2010) find that "the cost of saving a statistical life for newborns near 1500 g is on the order of \$550,000 with an upper bound of approximately \$1.2 million in 2006 dollars. Although the cost measures may not fully capture the additional care provided to VLBW newborns, the magnitude of the cost-effectiveness estimates suggests that returns to medical care are large for this group."

<sup>&</sup>lt;sup>16</sup> Wong et al. (2002) finds that maternal BMI has a more direct and greater effect on incidence of large for gestational age (LGA) births than glucose control.

<sup>17</sup> To our knowledge there are no direct estimates of the effect of diabetes mandates on the coverage of diabetic

<sup>&</sup>lt;sup>17</sup> To our knowledge there are no direct estimates of the effect of diabetes mandates on the coverage of diabetic population. However, Pollitz et al (2005) mention several state reports indicating that diabetes mandates will increase coverage.

existing private health insurance plans were covering or offering diabetes coverage at the time. Second, the moral hazard inducing women to substitute medical treatments for life style as identified by Klick and Stratman (2007) must not completely offset the benefit of medical care. To the degree that the empirical analysis finds a significant impact of diabetes mandates on low and/or high birth weight babies, this result is even more powerful.

### 2. EMPIRICAL STRATEGY

Given that the literature supports the proposition that low birth weight and high birth weight babies impose a substantial cost on society, no matter which estimate one believes, it then leads to the question of whether diabetes mandates have a significant impact on the number of babies in the tails of the birth weight distribution. Fortunately, we have a natural experiment to consider this question since these mandates have been imposed on some states while not others. Likewise, these mandates were instituted at different times in different states (Table 1).

The goal of the empirical work is to identify the causal effect of diabetes mandates on population prevalence of adverse birth outcomes. To identify this effect we need to control for all shocks to the affected group in treated states that are correlated with the adoption of the legislation. For this purpose we use all birth data for the period 1992-2003, which covers most instances of diabetes mandates adoptions, <sup>19</sup> in a difference-in-difference-in-difference analysis. We compare the treatment individuals in the experimental states to a set of control individuals in those same states and measure the change in the treatments' relative outcomes, relative to states that did not pass diabetes mandates. The identifying assumptions of this "differences-in-differences-in-differences" (DDD) estimator are fairly weak. It only requires that there be no contemporaneous shock that affects the relative outcomes of the treatment group in the same state and year as the law.

In our case, the treatment group comprises births to women with diabetes. The control group represents all other births. Due to the very large sample size and the fact that the relevant legislative variable varies only at state/year level, the data is collapsed into state/year/mother diabetic status cells. The data is also divided by mother's age and race because the trends in

<sup>19</sup> The only exception is the Wisconsin. In this state diabetes mandate became effective in 1988 when Natality Data does not report diabetic status of the mother.

<sup>&</sup>lt;sup>18</sup> In addition, findings that diabetes mandates affected the health of people with diabetes (Klick and Stratmann, 2007) imply that mandates lead to changes in utilization rates.

diabetes incidence vary by age group (Lawrence et al., 2008) and because African-American women with diabetes tend to have different rates of diabetic complications and to be more likely to have low birth-weight babies than do Caucasian women (Nicholson et al., 2006). The age groups are: age below 25, 25 to 29, 30 to 34, and 35 and over. Thus, all observations are averages for the year/state/mother's age/mother's race/mother's diabetes, and the regressions run at that level, weighted by cell size.

We estimate the following equation:

(1) 
$$Y_{gdst} = \alpha + \beta_1 X_{gdst} + \beta_2 \gamma_s + \beta_3 \lambda_{tg} + \beta_4 \mu_d + \beta_5 \partial_{td} + \beta_6 \delta_{sd} + \beta_7 \varphi_{st} + \beta_8 \theta_{std} + \varepsilon_{gdst}$$
  
where g indexes age/race group, d diabetic status of the mother, s states, and t time.

X is a vector of time-varying determinants of birth outcomes such as female infant, plural birth, first child, mother's education, marital status, and prenatal care.  $\gamma_s$  is a fixed state effect,  $\lambda_{tg}$  is a fixed year effect that varies by age group, and  $\mu_d$  is a dummy for treatment group (1 if mother was diabetic, 0 otherwise).

We include state fixed effects,  $\gamma_s$ , to control for differences in birth outcomes that are common to people in the same state (for instance, secular differences in the overall level of health due unmeasured cultural factors such as cuisine specificity or weather). Second, we include year fixed effects,  $\lambda_{tg}$ , to capture any time-varying differences in the health common to the infants, such as changes in federal level health care policies. Because, as we mentioned above, there are different trends in infant outcomes by age and race of the mother, we allow the time effects to vary by age-race demographic groups. The interactions between the time effects and the diabetic status of the mother,  $\partial_{td}$ , account for differential changes over time in the health status of infants born to diabetic mothers (such as those due to changes in diabetes management technologies). The interactions between state effects and diabetic status,  $\delta_{sd}$ , of the mother are included to control for systematic differences in outcomes of infants born to diabetic mothers across states. The equation includes state-by-year fixed effects,  $\varphi_{sb}$  that control for differential changes over time in states that adopted mandates.

In this framework  $\beta_8$ , the coefficient of the triple interaction between diabetic status, mandate state, and post-treatment status,  $\theta_{std}$ , captures the variation in health specific to infants of diabetic mothers (relative to non-diabetic mothers) in states with diabetes mandates (relative to states without such mandates) in the years after the law (relative to before the law). Note that

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<sup>&</sup>lt;sup>20</sup> In other words we have a dummy for each age-race demographic group in each year.

because the effects of diabetes mandates on birth outcomes likely lag at least several months behind its adoption, we are agnostic ex-ante as to whether a change in diabetes mandates status should be recorded with a lag, two lags, or no lags. Hence, we will experiment with different specifications and let the data indicate the relevant time period. The dependent variable could be percent low birth weight babies (defined as either <2500 grams or <1500 grams), percent high birth weight babies (defined as either >4000 grams or >4500 grams), and percent premature (defined as either  $\le36$  weeks or  $\le32$  weeks).

Some issues regarding the estimation strategy should be mentioned. First, the unit of observation is more detailed than the level of variation of the independent variable of interest, the state level. Second, there are no instances of repeals in the data. It is thus likely that the error terms are correlated within each state over time. In the presence of autocorrelation, estimated standard errors tend to be biased downward, making coefficient estimates spuriously statistically significant. Moreover, misspecification of the autocorrelation process, which is likely to occur with short time series like the ones used in this paper, can also lead to downward bias in the standard error estimates. To correct for all these potential problems, this paper reports robust standard errors clustered at the state level, a method that allows for an arbitrary autocorrelation process (Bertrand et al., 2004).

A different issue is that only a subset of women who give birth are likely to be affected by mandates because state mandates apply only to individuals who have private insurance. In addition, self-insured plans are exempted under the Employment Retirement Income Security Act (ERISA). While the coefficients estimated from the specification above are valid estimates of the population impact of diabetes mandate, we may also want to know the impact among those subject to mandates (effect of treatment on the treated). In the analysis below we follow previous literature on state insurance mandates (Bitler and Carpenter, 2011) and other types of policies affecting subsets of population (Almond et al., 2011) and inflate the coefficients by the estimated treatment rates (i.e. the estimated proportion of women in our investigated demographic group having the type of insurance subject to diabetes mandates) to obtain the

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Source: http://www.nlm.nih.gov/medlineplus/ency/article/003402.htm

<sup>&</sup>lt;sup>21</sup> In some specifications we will also consider the 5 minutes Apgar score. Note however that the data is less reliable because of a larger fraction of missing values. The Apgar score is a summary measure of the newborn's condition based on heart rate, respiratory effort, muscle tone, reflex irritability, and skin color. It takes values from 1 to 10, where higher is better. Values of 8 and above are considered normal. Any score lower than eight indicates the child needs assistance.

estimated treatment effect on the treated. Additionally, in the empirical analysis we will test the robustness of results by exploiting this information about the likelihood of treatment.

In the subsequent analysis, we use detailed individual level data to estimate the impact of diabetes mandates for each of the eight separate age/race groups (mother's age <25;  $\geq 25$  and <30;  $\geq 30$  and <35; and  $\geq 35$  and mother's race White or Black). For this purpose we use a similar equation:

(2)  $Y_{ist} = \alpha + \beta_1 X_{ist} + \beta_2 \gamma_s + \beta_3 \lambda_t + \beta_4 \mu_d + \beta_5 \partial_{td} + \beta_6 \delta_{sd} + \beta_7 \varphi_{st} + \beta_8 \theta_{std} + \varepsilon_{ist}$  where *i* indexes individuals. In addition, in this specification we also include county level controls for log wages, the number of physicians in the county per 1,000 residents, and the number of hospitals per 100,000 individuals.<sup>22</sup>

#### 3. DATA

Birth outcomes data comes from National Center for Health Statistics's (NCHS) Vital Statistics Detail Natality Data and covers all births<sup>23</sup> in the 50 US states. The analysis is performed on 1992-2003 data. As shown in Table 1, this data covers most instances of diabetes mandates. The only exception is Wisconsin, where diabetes mandates became effective in 1988 when the data on diabetic status of mothers was not reported in NCHS Natality Data. As such the sample in this study is extremely representative of the country as a whole.

Infant characteristics such as gender, plurality (single versus higher order birth) and parity (whether it was a first or subsequent birth) also come from Natality Data. In addition, Natality Data provides information about the demographic characteristics of mothers such as age, race, education, marital status, and state of residence but does not provide income information. However, education can act as a proxy for income and in the state level regressions the state-by-year-by-demographic group fixed effects likely absorb most of the variation in

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<sup>&</sup>lt;sup>22</sup> State by year fixed effects control for such factors in the analysis using data collapsed in state cells.

<sup>&</sup>lt;sup>23</sup> We retain all births including multiple births, thus our results are valid for the entire population. Note that we find no evidence that diabetes mandate led to any change in the proportion of plural births among women with diabetes relative to non-diabetics (the estimated effect is 0.088 with standard errors of 0.203 and thus highly insignificant). The results hold on the sample of singletons although the estimates are slightly lower and sometimes with larger standard errors function of specification, understandable given that we loose power. This is because we loose significant variation in our dependent variable: more than 25% of low birth weight babies are plural births. In state cells regressions this leads to very thin cell sizes for women with diabetes in some age groups. We need this variation given that we control for a very large number of fixed effects relative to the sample size in our state level regressions. Results using individual level data on the sub-sample of singleton births are reported in Table A4 Supplemental Results Appendix (not for publication).

income. Because practically all means-tested programs are administered at state level, the stateby-year fixed effects absorb the impact of such programs on infant outcomes and thus any source of bias from the introduction or expansion of such programs.

When using individual level data we also use county level income as an additional proxy. This data comes from The Bureau of Labor Statistics Quarterly Census of Employment and Wages. In the analysis using individual level data we also include variables acting as controls for access to medical care: county number of physicians per 1000 individuals, and county hospitals per 100,000 individuals, come from U.S. Department of Health and Human Services, Area Resource Files.

Natality Data also provides information about the diabetic status of the mother.<sup>24</sup> While it is true that pregestational diabetes has more severe consequences on infant outcomes than gestational diabetes we cannot distinguish between type 1, type 2 diabetes, and gestational diabetes in this data. To investigate the differential effect of diabetes mandate on women with pregestational diabetes versus gestational diabetes, we use the Behavioral Risk Factor Surveillance System (BRFSS) to estimate the prevalence of pregestational versus gestational diabetes among women of childbearing age.

For the purpose of obtaining an estimate of the proportion of women with the type of health insurance falling under diabetes mandates, we use the private insurance prevalence and employment by firm size data from the March Supplement of the Current Population Survey (March CPS), and the percentage of workers in self-insured plans by firm size from Kaiser/HRET Survey of Employer-Sponsored Health Benefits and Medical Expenditure Panel Survey.

Due to the specific target of diabetes mandates, we concentrate our study on the sample of infants born to mothers with more than high-school education. Specifically, diabetes mandates require health care insurers to provide coverage for medically necessary equipment, supplies, services, and medication for individuals with diabetes, and thus affect women that have private health insurance. According to Wyn and Peckham (2010) study 42% of women who had not graduated from high-school are uninsured and 25% of women with high-school education also lack insurance compared to just 11% of women with a college degree. Moreover, only 23%

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<sup>&</sup>lt;sup>24</sup> The diabetes information is quite comprehensive: there are between 0.5 and 3% missing values in each year investigated. Observations with missing diabetes status were dropped.

of the women with less than high school education that do have health insurance are covered through their employer. The rate of employment-based coverage among women with high-school education is somewhat larger: 49% but still significantly lower than the 75% rate of employer coverage among women with a college degree. As such diabetes mandates are more likely to affect women with college education because a significantly larger proportion will experience a change in insurance coverage. We focus on this sub-sample where mandates are binding because regressions that impose the constraint of equal effect for the entire population may conclude that the policy had little or no effect (Bitler and Schmidt, 2012). Results obtained using the sample of infants born to mothers with high-school education or less are discussed in the text when relevant or reported in the Supplemental Results Appendix available on request.

The caveat is that these results may not be generalizable to women with less education even if they were privately insured because education may be a good predictor of individuals' discount rates (Fuchs, 1982) and, thus, of their propensity to invest in health. Another reason why the results may not be generalizable beyond educated women is that education may predict consumer knowledge of coverage, which is correlated with their propensity to use medical care. And finally education may simply affect the allocation of health inputs, medical care versus self-care, in a way that shifts the health production function (Grossman, 2006). Despite these limitations, our study represents an important step in understanding the costs and benefits of insurance mandates.

Another potential caveat regards the fact that there are many legitimate ways to code the legislative data. If the transaction costs associated with insurance contractual arrangements are high enough, the enactment of the law could prompt insurance companies to include diabetes coverage on new insurance contracts even before the law becomes effective knowing that within months they will have to change the contract anyway. In this case there may be an impact on coverage even before the law becomes effective. However, if additional coverage is costly enough insurance companies may prefer to wait until the law becomes effective to include additional coverage for diabetes. Consequently, coverage changes would occur only after the law becomes effective. However, because of the difference between the timing of changes in coverage and the date the effect is recorded, only after birth, it is unlikely there is any impact on infants born between the enactment and the effective dates. For this reason in the main analysis

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<sup>&</sup>lt;sup>25</sup> Note that the year of enactment and year the law became effective are identical for some states.

we measure the impact using the diabetes mandates effective date.<sup>26</sup> If the effective date of the reform was on or after July 1<sup>st</sup>, the law was coded as belonging to the year after because for most of the year the law did not apply and thus could not affect births in that year. In the sensitivity analysis we also show that our results are robust to a different coding of the diabetes insurance variable.

Summary statistics for the main sample used for our analysis are reported in Table 2. The means and standard errors of variables used are shown for all births and separately for births to diabetic mothers only.<sup>27</sup> These means were calculated for the state-years with no mandates, separated by treatment status in the following year: no diabetes mandate in columns 1 and 4; and diabetes mandates adoption in columns 2 and 5. Columns 3 and 6 report the results of t-tests for the equality of means. Because infant outcomes exhibit time trends and the means calculated in columns 2 and 5 tend to use later data than the means from columns 1 and 4, we report t-test of equality of means conditional on time fixed effects. Adopting and non-adopting states appear to be virtually identical with respect to the variables characterizing the environment and infant outcomes of infants born prior to mandates adoption. Similarly, there is no statistically significant difference between the characteristics of infants born to diabetic mothers in experimental versus nonexperimental states.

### 4. RESULTS

### 4.1. Main Results

### 4.1.1.Graphical Evidence

Figures 1 to 6 showing pre- and post-diabetes mandates trends in the incidence of high birth weight and low birth weight separately for diabetic mothers and non-diabetic mothers anticipate our main result.<sup>28</sup> They are all consistent with a decrease in the tails of the distribution of babies born to diabetic mothers in mandates states. Among non-diabetic mothers the trend in prevalence of poor infant outcomes before mandates is indistinguishable from the trend after mandates.

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<sup>&</sup>lt;sup>26</sup> We also report some results obtained using enactment date in the sensitivity analysis.

<sup>&</sup>lt;sup>27</sup> For the sake of parsimony the summary statistics for the non-diabetic mothers were reported only in the Supplemental Results Appendix.

28 In all graphs year t is the year mandates became effective.

In addition, this graphical evidence shows no indication of a transitory pre-treatment increase in the tails of the distribution, the equivalent of an "Ashenfelter dip" for this case (Ashenfelter, 1978), which would suggest the estimates indicate just mean reversion and thus are falsely attributed to diabetes mandates. A formal test for pre-trends performed by entering leads of the diabetes mandates in the main specification, reported in Table 9 also finds no evidence of a pre-trend. At the same time, however, the graphs are also consistent with an upward trend in low birth weight and prematurity and a downward trend in high birth weight that may vary by state, highlighting the need to control for state specific time effects.

### 4.1.2. Econometric Results

It is difficult to say with certainty whether diabetes mandates should have an instantaneous effect or a lag effect. However, previous research shows that fetal growth acceleration in large for gestational age fetuses of diabetic mothers begins in the second trimester (Wong et al, 2002) and in fact high-glycemia appears to have the most impact on fetal growth in the third trimester (Schaefer-Graf et al, 2003). On the other hand, if diabetes leads to prematurity it likely does so through its effect on fetal environment before the third trimester. As a result there could be heterogeneity in the timing of the effect on each tail of the birth weight distribution. In Table 3 we present estimates of the instantaneous impact, one year lag impact and two year lag impact obtained using equation (1). Each cell represents the coefficient from a different regression.

As shown in the second column of Table 3 we find that the effects of diabetes mandate lag one year behind the year the legislation became effective. This is highly plausible because small fetal size was correlated with maternal glycemic control during the first trimester (Pedersen et al. 1984; Visser et al. 1985).<sup>29,30</sup> Also ostensibly a significant proportion of low

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<sup>&</sup>lt;sup>29</sup> Other studies found no difference in early fetal growth among diabetic and non-diabetic pregnancies (Brown et al, 1992)

The prevalent opinion appears to be that the frequency of congenital malformations is about 3 times higher in the children of insulin dependent pregestationaly diabetic women (Coustan 1998; Dunne et al 2009). However, we found no evidence of a significant impact of diabetes mandates on the two most common types of congenital malformations: neural tube malformations and heart malformations. The estimate coefficient of the lag effect of diabetes mandate on the prevalence of neural tube malformations (anencephalus, spina bilfida/meningocele, hydrocephalus, microcefalus, and other central nervous system anomalies) is -0.007 with standard error of 0.019 and the coefficient in the case of heart malformations (all heart and circulatory malformations) is -0.035 with standard error of 0.052. Note that studies indicate that congenital malformations occur almost exclusively in offspring of women whose diabetic disease had an early onset and in whom it was severe enough; a severity evidenced by the fact that many of them had vascular complications during their childbearing years (Comess et al 1969). Given that in our data we cannot separate between pregestational and gestational diabetes and that the prevalence of congenital

birth weight babies are premature babies. Because the effect on prematurity seems to be concentrated among births with less than 32 weeks of gestation it seems plausible that whatever affected the fetal environment that led to premature birth took place relatively early during pregnancy. It is thus more likely to be the case that the effect of diabetes mandates would show in the year following the effective date of legislation. In either case, it is reassuring to find a lag between the adoption and any significant correlation with infant outcomes, suggesting that the adoption of diabetes mandates is exogenous and providing further confidence in our identifying strategy. For the remaining part of the paper we report only the lag effect of diabetes mandate.

We find that diabetes mandates are associated with a statistically significant 0.34 percentage points, the equivalent of a 3.6 percent decrease in low birth weight (<2500 grams) prevalence among births to women with diabetes, and a 0.19 percentage points, the equivalent of 11% reduction from the mean of the prevalence of very low birth weight (<1500 grams) among births to women with diabetes. The adoption of diabetes mandates also leads to a 0.22 percentage points, the equivalent of 10 percent decrease in prematurity (≤32 weeks) prevalence among births to women with diabetes. <sup>31,32</sup> These are estimates of the investigated population impact of diabetes mandate. We follow Almond et al (2011) and calculate the implied impact on those who were affected by mandates by dividing these coefficients by an estimate of the share of population investigated subject to mandates.<sup>33</sup> After inflating the estimated effect by this share

malformation is very low (less than 1% for either type of malformations considered) it is possible that we cannot detect the effect of mandates in our data.

<sup>&</sup>lt;sup>31</sup> The only women who should be affected are those with private insurance. Thus zero impact on other women is being averaged with a larger effect on women affected by mandates, thus leading to a small overall effect.

<sup>&</sup>lt;sup>32</sup> The effect of mandates on the prevalence of low 5 minutes Apgar score (< 8) among infants born to diabetic mother is negative but not statistically significant at conventional significance levels. Note that this variable has a high non-reporting rate ( $\sim$ 18%). Most non-response ( $\sim$ 65%) is driven by one state only, California. All other results are robust to the exclusion of California.

<sup>&</sup>lt;sup>33</sup> We do not have information about private insurance in the Natality data, nor about the share of women with private insurance from self-insured plans that are not subjected to diabetes mandate. Instead we use data from Kaiser/HRET Employer Health Benefits Annual Survey to obtain the percentage of insured workers in self-insured plans by firm size (in particular we averaged the 1996 and 1998 data to obtain self-insurance rates in 1997, the middle of our sample period). This information along with the data regarding the share of educated workers (more than 12 years education) in each type of firm as defined by firm size allows us to obtain the share of population with more than high-school education in self-insured plans. (We used the March CPS to obtain the share of population 18-45 years old with more than high-school education by firm size.) Our calculations indicate that approximately 44.8% of insured people with more than high-school education were in self-insured plans. For comparison this estimate is very close, though slightly lower that the 48.3% estimate obtained by Bitler and Carpenter (2011) using the 2000 Medical Expenditure Panel Survey (MEPS) Household Component for 2000. This is due to the fact that according to Kaiser/HRET data in year 2000 there were more workers in self-funded plans in large firms than in previous years, 1996, 1998 and than in following year 2001. Replicating our calculations using 1997 MEPS data indicates that ~43.07% of private sector enrollees are in self-insured plans, a lower estimate. March CPS data

we find that diabetes mandate reduces low birth weight prevalence by 0.73-0.76 percentage points (or approximately 7.9-8.1 percent), very low birth weight prevalence by 0.41-0.43 percentage points (or approximately 24-25 percent), and prematurity by 0.48-0.50 percentage points (or approximately 21.6-22.3%) among diabetic women subject to diabetes mandate.<sup>34</sup> These estimates however represent a partial equilibrium and could be an overestimate of the effect if, for instance, self-insured plans respond to the introduction of diabetes mandates, and thus the share of women treated in population is in fact larger than estimated. Second, these might also represent underestimates if mandated benefits laws would cause employers to reduce offers of health insurance.<sup>35</sup>

We find no statistically significant effect on high birth weight prevalence. It is unlikely this result is driven by endogeneity. It would require a contemporaneous shock that affects the relative outcomes of the treatment group in the same state-years as the law. In addition the tests we performed did not find evidence of endogeneity. <sup>36</sup> The alternative hypothesis is that perhaps the incidence of moral hazard identified by Klick and Stratmann (2007), increased obesity prevalence among people with diabetes, offsets the expected impact of diabetes mandates in reducing the prevalence of high birth weight. Previous research found that maternal BMI has a greater effect on incidence of large for gestational age (LGA) births than glucose control (Wong et al, 2002), and, as shown below, we find evidence of higher pregnancy weight gain among women with diabetes living in mandate states.<sup>37</sup>

indicates that 81.28% of 18-45 year old women with more than 12 years of education had private insurance in our sample period. This means that between 44.87% (HRET based estimate: (100-44.8)\*81.28/100) and 46.27% (MEPS based estimate: (100-43.07)\*81.28/100) of educated women of childbearing age would have had private insurance subject to this regulation. Note that we assume that the rates of self-insurance are the same for group insurance and for individually purchased insurance, while in fact individually purchased insurance is more likely to be fully funded (Klick and Stratman, 2007) and, thus, subject to mandates.

Conover, 1998).

<sup>&</sup>lt;sup>34</sup> The lowest estimate is based on the 1997 MEPS data on the prevalence of self-funded insurance plans while the highest estimate is based on the 1996-1998 HRET data on the prevalence of self-funded insurance plans. The empirical evidence to support this claim is mixed (Gabel and Jensen, 1992; Gruber 1994b; Sloan and

<sup>&</sup>lt;sup>36</sup> The graphs do not indicate pre-trends, nor is there any correlation between our measures of infant health and leads of diabetes mandates. We also found no evidence of sensitivity to sample size as shown in sensitivity analysis. <sup>37</sup> It should be mentioned, here, that while this weight gain seems to be associated with decreases in low and very low birth rate babies and no change in high birth rate babies, this doesn't mean that such mandates do not have harmful effects. After all, weight gains, if caused by these mandates, could lead to other health problems such as hypertension, future heart attacks etc. Thus, although diabetes mandates seem to lead to obesity in mothers, without having adverse affects to their babies, these mandates may still be harmful and would suggest that research beyond birth rates would be a fruitful area to pursue.

Note that even if insulin restores fertility of diabetic women some studies remarked that those with onset of diabetes at young ages are still more likely to suffer from infertility than non-diabetics (Livshits and Seidman, 2009). Thus, the diabetic pregnancies investigated here may be among the healthiest diabetic women. To the extent to which mandates providing better ability to control diabetes may lead to more diabetic pregnancies our study relying on year-to-year variation in pregnancy outcomes may not be able to fully account for general equilibrium effects of diabetes mandates.<sup>38</sup>

While we cannot point out with certainty the mechanism of the effect, we can attempt to identify the demographic categories that benefit most from diabetes mandates and the categories still vulnerable. The state level data has the advantage of reducing noise and improving tractability of regressions as we deal with significantly fewer observations. At the same time it reduces the power of our tests and the ability to identify the effect in the presence of the large number of fixed effects required by the DDD identification strategy. Because cutting the data to investigate the impact of diabetes mandates by demographic group reduces available variation, and thus, the power to identify the effect of the law, we use highly detailed individual level data to identify the effect of diabetes mandates by age-race demographic group.<sup>39</sup> The age groups are: age below 25, 25 to 29, 30 to 34, and 35 and over. Because the sample investigated is babies born to educated women, pregnancies before the age of 25 would be more likely to be unplanned than post age 25 pregnancies. While women younger than 25 are more likely to be healthy, it could also be that the investment in healthy behaviors during pregnancy may be lower. Similarly older mothers, over 35 years old, are more likely to suffer from diabetes but they appear to have experienced a slower increase in diabetes incidence than younger age groups (Lawrence et al., 2008) which could be driven by different lifestyle choices. These women are also less likely to have an unplanned pregnancy. As a result there may be significant heterogeneity in the effect of diabetes mandates.

As reported in Table 4, we find that the benefit of diabetes mandates is concentrated among infants born to women over 30 years old. This is consistent with a higher prevalence of

<sup>&</sup>lt;sup>38</sup> Using our panel of annual data we find no evidence of a change in the proportion of births to diabetic women in total births (coefficient 0.024 with standard error of 0.118).

<sup>&</sup>lt;sup>39</sup> Note that the estimates are similar when using state-year cells but the standard errors are understandably larger – results reported in the Supplemental Results Appendix and available on request.

diabetes among these women.<sup>40</sup> We also find a larger effect on the prevalence of low birth weight among babies born to Black women with diabetes, consistent with the previous finding that low birth weight incidence is higher among this demographic group than it is among Caucasian diabetic women (Nicholson et al., 2006).

To investigate the hypothesis that the effect of better access to medical care is attenuated by behavioral changes we estimate the effect of diabetes mandates on pregnancy weight gain among diabetic women. Because our data do not report weight before pregnancy it is difficult to assess whether changes in pregnancy weight are in fact an improvement or else putting women in a weight category that could generate problems for their babies. Consequently, we concentrate on weight gain of at least 35 pounds that would be problematic for most women in our sample. The Institute of Medicine pregnancy weight gain guidelines are 28-40 pounds for underweight women, 25-35 for women of normal weight, 15-25 for overweight women and 11-20 for obese women. 41 Given that approximately 59.5% of all women age 20-39 are overweight, 54.9% of Non-Hispanic White women are overweight and 78% of Non-Hispanic Black women are overweight (Flegal et al., 2010) while less than 3% of women over 20 years old are underweight (Fryar and Ogden, 2010) a pregnancy weight gain of more than 35 pounds is too much for the large majority of women in our sample. Note that in our data there is heaping at 35 pounds. It could be that women control their weight as to gain exactly 35 ponds or else misreporting causes the heaping. We believe it is more likely to be caused by under-reporting than over-reporting and treat a weight gain of 35 pounds as problematic.

We find that diabetes mandates are in fact associated with an increase in pregnancy weight gain among diabetic women, but the change is statistically significant only among Black women 30-35 year old. This change in behavior could attenuate the positive effect of diabetes mandates and at least partially explain why we find no effect on the prevalence of high birth weight babies. About 18% non-response rate of weight gain.

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<sup>&</sup>lt;sup>40</sup> The CPS data also indicates somewhat higher prevalence of private health insurance coverage among older women.

<sup>&</sup>lt;sup>41</sup> "Weight Gain During Pregnancy: Reexamining the Guidelines," Institute of Medicine of the National Academies, May 28, 2009 available at <a href="www.iom.edu/pregnancyweightgain">www.iom.edu/pregnancyweightgain</a> (downloaded August 2011)

<sup>&</sup>lt;sup>42</sup> Another way to test this hypothesis is to look at women over 35 years old having their first child. We believe these women would be more likely to avoid any type of behavior that has the potential of harming the pregnancy. We find that among Black women over 35 years old there is evidence of significant increase in the probability of significant weight gain (35 pounds or more) only among those already having a child (coefficient 2.146 with standard error of 1.176 and significant at 5%) but not among those having their first child (coefficient 0.616 with standard errors of 4.227). In addition, we find a significant decrease in very high birth weight prevalence among first births to Black

65% of the missing values come from California for which there are no data on weight gain. Nevertheless the remaining missing values represent a significant enough proportion of the population to suggest the data on weight gain represents a selected sample. There is no evidence however that the non-response rate is different for diabetics versus non-diabetics. Using the data from all other states we find that the non-response rate among people with diabetes is 6.516% and among non-diabetics is 6.215%.

A different question is raised by the differential response of male versus female fetus to changes in fetal environment because that the human male is more fragile than the female (Kraemer 2000). To investigate the hypothesis of heterogeneity in the effect of mandates by infant gender we separate the state/year/demographic group cells by infant gender and run separate regressions for male versus female infant. The results reported in Table 5 suggest that diabetes mandates are associated with a statistically significant reduction in prematurity among female infants but not among male infants. One explanation is that there are more female premature births because females have a higher probability of survival if premature. It is possible that the fetal health threshold at which a pregnancy results in a live birth is lower when maternal health improves. As a result diabetes mandates may lead to a decrease in prevalence of fetal deaths. A larger decrease in male fetal deaths would be consistent with our estimates. Because our data does not have reliable information about stillbirths and miscarriages we defer this question for future research.

An alternative hypothesis is that of different mechanisms behind the decrease in low birth weight among male and female infants. As suggested by Kramer (1987a, 1987b), birth weight is determined by gestation length, and by the growth conditional on gestation length (intrauterine growth or IUG). To test whether the decrease in low birth weight prevalence among female infants is driven by a decrease in prematurity while the decrease in low birth weight prevalence among male infants is driven by fetal growth restrictions we differentiate premature low birth

women over 35 years old (coefficient -1.782 with standard error of 0.863 and significant at 5%) but a lower such decrease among second or subsequent births (coefficient -0.559 with standard error of 0.394). Although the coefficients follow a similar pattern they are not significant in the case of White women.

<sup>&</sup>lt;sup>43</sup> By fetal deaths we denote any attrition between conception and live births.

<sup>&</sup>lt;sup>44</sup> Matthews et al (2008) found that poor maternal nutrition skews the sex ration in favor of girls.

<sup>&</sup>lt;sup>45</sup> The NCHS Fetal Death datasets have missing values for diabetic status of mother for approximately 40% of observations, which makes it unsuitable for our analysis. We found no evidence of a significant change in sex ratio of infants born to diabetic women in states with mandates. The estimated coefficient of the lag diabetes mandate on the proportion of female births is -0.054 with a standard error of 0.251 (both multiplied by 100 to improve readability).

weight births from full term low birth weight births. We find that most of the effect on low birth weight is concentrated among premature births for both male and female babies, <sup>46</sup> suggesting that even if there is no significant change in the proportion of premature male births, the resulting births are either slightly longer gestation or just less fragile. This is important to document because prematurity is more important than IUG in determining low birth weight but so far it has proved to be more difficult to manipulate.

### 4.2. Falsification Tests

One way to test whether we are capturing the effect of diabetes mandates is to investigate whether this legislation is associated with any change in infant outcomes where there should be none.

### 4.2.1 Effect by Education

We first show there is no effect when mothers are less likely to experience a change in their insurance status. In the data section we explained that the focus of this paper is on infants born to mothers with more than high-school education because data show that these women are likely to have private health insurance and, thus, are more likely to be affected by the adoption of diabetes mandates. It follows that if indeed our empirical strategy captures the effect of diabetes mandate we should observe a lower effect where mandates are less likely to be binding. Women with high-school education or less are less likely to have obtained health insurance through their employer. In Table 6, Panel A we report results obtained by replicating our main empirical specification on the sample of infants born to women with high-school education or less. The coefficients are indeed not significant and even change sign in some cases. Moreover, when we restrict the sample to women with 16 or more years of education, among which according to March CPS data almost 90% have private insurance, the effect is larger and more precisely estimated. These findings provide further reassurance that our identifying strategy can successfully isolate the impact of diabetes mandates.

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<sup>&</sup>lt;sup>46</sup> Mandates are associated with a ~6% (coefficient -0.415) decrease in prevalence of premature low birth weight male infants but with a ~2.2% (coefficient -0.046) decrease in prevalence of full term low birth weight male infants. <sup>47</sup> We report the coefficients obtained under the assumption that timing of the effect of diabetes mandates is the same across sub-samples. A complete replication of our investigation of the dynamics of the impact using the sample of births to women with 12 years of education or less is reported in the Supplemental Results Appendix. There is no evidence that diabetes mandates have any significant effect on this sample at any point in time.

### 4.2.2. Effect by Diabetic Status of Mother

In addition, we look at infants born to non-diabetic mothers. Any change in outcomes among these women should be small because they do not benefit directly from mandates. A positive impact, if any, would be driven by access to education about preventive behaviors and it would be smaller. Mandates may also have an effect if they resulted in premium changes that affected the decision to obtain coverage or if they reduce incentives to engage in preventive behaviors. We find that there is no statistically significant effect on infants born to non-diabetic mothers (Table 6, Panel B) and the estimated coefficients imply changes several times smaller. For instance, our DD estimates imply an  $\sim$ 4.4% decrease in low birth weight prevalence among mothers with diabetes but an  $\sim$ 0.7% decrease among women with no diabetes; similarly the implied decrease in prematurity ( $\leq$ 32 weeks) is of  $\sim$ 12% among women with diabetes but of only  $\sim$ 1.7% among all other women. The lack of worsening outcomes is consistent with previous literature that found no evidence of moral hazard associated with diabetes mandates among non-diabetics (Klick and Stratmann, 2007).

# 4.2.3. Effect by Type of Diabetes: Pregestational versus Gestational

In Panel C of Table 6 we investigate whether the effect of mandates differs by types of diabetes (pre-gestational or gestational). Because Natality data does not include the information about the type of diabetes we use BRFSS data to obtain the relative prevalence of gestational diabetes among 18-45 years old women with diabetes for each state/year cell<sup>48</sup> and interact it with the triple interaction (diabetic status, mandate state, post-treatment status). The analysis is more tentative in nature not only due to measurement error in this variable but also because it has been recognized that gestational diabetes does in fact record cases of unrecognized pregestational diabetes (Kim and Ferrara, 2010). Thus, a higher share of gestational diabetes among pregnant women diagnosed with diabetes likely indicates more cases of gestational diabetes but it could

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<sup>&</sup>lt;sup>48</sup> BRFSS includes pregnant women, however the sample-size is very small (an average of ~60 women in a state) thus making the sample of diabetic pregnant women too small (an average of ~3 in each state) for any meaningful estimates of relative prevalence of gestational versus pre-gestational diabetes. Consequently, we obtain estimates for the entire sample of women age 18-45. There is significant measurement error in this variable. We do not have a very good estimate of the actual instances of gestational diabetes in a year because the question asked in BRFSS is "Have you EVER been told by a doctor that you have diabetes?" Thus, for instance among women 30-44 some may have had gestational diabetes in their 20s. Nevertheless, the mean estimates appear to be quite similar to the estimates of prevalence of diabetes from other sources. Approximately 2.3% of women age 18-45 have diabetes slightly higher than the 1.82% pre-gestational diabetes among pregnancies in 2005 (Lawrence et al, 2008). Also approximately 2.2% of women 18-45 have ever had gestational diabetes compared to estimated ranges of 2 to 10% of pregnancies (CDC, 2011).

also be indicative of more undiagnosed pregestational diabetes.<sup>49</sup> It may not be entirely appropriate to interpret the results only through the lenses of expected effects associated with diabetes early in pregnancy (the case of pregestational diabetes) versus the effects associated with diabetes late in pregnancy (most likely the case of gestational diabetes). In addition, the interpretation has to account for potentially different effects of mandates on insurance rates by type of gestation. It is likely that women with type I diabetes, diagnosed early in their life<sup>50</sup>, would have obtained some sort of coverage even without mandates. The same is not necessarily true for women with type II diabetes, believed to be driven by life style, or for women with gestational diabetes. The price of insurance may have changed due to mandates,<sup>51</sup> or else the demand for insurance may change with increased coverage possibly leading to differential changes in insurance rates by type of diabetes.

Our estimates point to larger decreases in the share of macrosomic infants in areas with larger shares of women diagnosed with gestational diabetes. We also find smaller improvements in low birth-weight/prematurity prevalence in areas with larger shares of gestational diabetes, i.e. smaller shares of pregestational diabetes. This is consistent with the medical literature that indicates that diabetes early in pregnancy leads to low birth-weight and prematurity, while diabetes later in pregnancy, such as gestational diabetes is more likely to lead to macrosomia.

# 4.3.4. Effect by Likelihood of Treatment as Determined by Type of Insurance

Even among women with more than high-school education not all have private insurance. We do not have information whether women in our data had private insurance. Instead we follow Schmidt (2007) and use March CPS data to calculate the share of women age 18-45 with more than 12 years of education in each state and year who are covered by private insurance and test whether the effect of diabetes mandates varies with private insurance coverage. For this purpose we add to our main specification, equation (1), an interaction term between our triple

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<sup>&</sup>lt;sup>49</sup> The variable is the share of women with gestational diabetes among women with diabetes, thus, a larger share could be driven by fewer women with pre-gestational diabetes, or by more women with gestational diabetes when the number of women with pre-gestational diabetes is the same.

<sup>&</sup>lt;sup>50</sup> Peak age at diagnosis in US is 14.

<sup>&</sup>lt;sup>51</sup> We are not aware of any such estimates, however the "2003 Diabetes Mandate Report" issued by Utah Insurance Department (October 28, 2003) found that diabetes mandates increased costs by less than 0.1%, the equivalent of 2 USD per year per policyholder. Similarly Louisiana Department of Insurance "A Study of the Costs Associated with Healthcare Benefits Mandated in Louisiana" issued on 28 February 2003 also found that the cost of diabetes mandate was less than 0.006 percent of total cost paid by insurers.

<sup>&</sup>lt;sup>52</sup> Because we cannot calculate coverage through private insurance separately for women with diabetes versus non-diabetics we impose the constraint that the coverage is the same in both groups.

interaction (diabetic status, mandate state, post-treatment status) and the share of privately insured women. Consistent with our hypothesis, the results reported in Table 7 column 1 indicate a systematic gradient in the size of the effect by prevalence of private coverage. Specifically, diabetes mandates lead to larger decreases in both tails of the birth weight distribution where more women are covered by private insurance.

In addition, not all women with private insurance will experience a change in their coverage even when living in a state with diabetes mandates. Under the Employment Retirement Income Security Act of 1974 (ERISA) firms that self-insure are exempt from mandates. If the effect we identify is driven by mandates, we should observe a larger impact on births to women that do not obtain health insurance coverage through firms that self-insure. Detailed information on the share of employees in such firms is not available. However, previous empirical analysis found that large firms are more likely to self-insure (Park, 2000, Gabel et al., 2003). We follow Schmidt (2007) and use the employment by firm size data as a proxy for the share of employment in firms that self-insure or not. Specifically, we use March CPS data to obtain the share of 18-45 years old employees with more than 12 years of education who work in firms with less than 500 employees<sup>53</sup> in each state and year and interact this variable with our triple interaction (diabetic status, mandate state, post-treatment status). Although not all estimates are statistically significant, we find the decrease in the tails of birth weight distribution is systematically larger where the share of employment in small and medium size firms is more significant.

Furthermore, we obtain an estimate of the share of population in fully-insured plans and investigate whether we observe a larger effect where more people are enrolled in fully-insured plans. For this purpose we use the Kaiser/HRET Employer Health Benefits Annual Survey data to obtain an estimate of the share of covered workers in self-insured plans by firm size.<sup>55</sup> We use this data along with the share of 18-45 years old workers with more than 12 years of education

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<sup>&</sup>lt;sup>53</sup> March CPS reports categories of less than 25 employees, 25-99, 100-499, 500-999, and 1000 or more employees. We chose the 500 employees cut-off because Gabel et al (2003) found that only 13% of employees in firms with less than 200 employees were enrolled in self-insured plans, 46% in firms with 200-999 employees, but over 60% of employees in firms with 1000 or more employees.

<sup>&</sup>lt;sup>54</sup> The share of employees working in firms that employ less than 500 workers is absorbed by the state-by-year fixed effects.

<sup>&</sup>lt;sup>55</sup> Because the data is not available for all years in our sample we average 1996 and 1998 data to obtain the 1997 (middle of sample) estimate of share of workers in self-insured plans by firm size.

by firm size<sup>56</sup> to obtain an estimate of the share of 18-45 years old workers with more than 12 years of education enrolled in self-funded insurance plans and, thus, also the share enrolled in fully-insured plans. For an estimate of the share of educated (>12 years education) women of childbearing age covered by fully insured plans we apply the private insurance rates among this population (March CPS data) to the share of fully insured workers.<sup>57</sup> The coefficients of the variable obtained from interacting our estimate of the share of fully insured with our triple interaction (diabetic status, mandate state, post-treatment status) are reported in column 3 of Table 7. Again we find the same pattern of health improvements where the treatment population is likely to be larger.

Lastly, we test how the diabetes mandates effect varies with employment in industries where workers tend to obtain health insurance through fully insured plans (Table 7, column 4). For this purpose we retain the following industries: retail, wholesale, service and finance. Both the Kaiser/HRET Employer Health Benefits Annual Survey data and the Form 5500 filings as described by Brien and Panis (2011) indicate that retail, finance and service have lower rates of self-insurance than other industries. In addition, Kaiser/HRET data indicate that mining/construction/wholesale have low rate of self-insurance but Brien and Panis (2011) only find low rates of self-insurance in wholesale and not mining and not construction.<sup>58</sup> These observations made us retain wholesale employment in our analysis but not mining and constructions. We find a similar gradient as in our previous analyses suggesting mandates have larger effects in areas known to have larger populations covered by fully funded private health insurance.

All these tests point in the same direction: the effect of diabetes mandate is larger where the likelihood of treatment is larger, providing further support for our estimates.

### 4.3. Sensitivity Analysis

All but three states implemented diabetes mandates that require private insurance policies to provide coverage for diabetes treatment. Mississippi, Missouri, and Washington laws require only that insurers offer coverage, i.e. make available for purchase a policy which covers diabetes

56 Source: March CPS.57 As it is also explained in footnote 33.

<sup>&</sup>lt;sup>58</sup> MEPS data confirms high-rates of self-insured plans in mining, but not in constructions. However, March CPS data indicates significantly lower rates of private insurance in construction, thus even if many insured workers may be in fully funded plans, the proportion of total workers in fully funded plans is still lower than for other industries.

treatment, but do not require all insurance policies to cover diabetes. So far we have treated both types of laws similarly, however, they could have different implications with respect to their potential of generating moral hazard. If coverage is only offered, perhaps only those for which the benefits outweigh the cost of additional coverage would chose to be covered. In other words, only those who find it most difficult to manage diabetes through life style would. The implication for the insurance companies is that they add to their pool of insured only the frailest, but for the population at large the implication is that only those who need diabetes coverage the most will get it. This might limit the potential of moral hazard in population, because perhaps fewer people with easy to manage diabetes would substitute life style for medication. It could thus be the case that requiring to offer coverage is associated with larger improvements in health than requiring to provide coverage.

On the other hand, if people do not assess correctly their ability to manage diabetes and/or the costs associated with the failure to manage their diabetes on a permanent basis, mandating the inclusion of coverage for everybody could imply faster intervention to correct poor diabetes management. Coverage may be associated with more contact with physicians that likely are better at assessing the individuals' ability to successfully manage diabetes and are able to recommend corrective measures.

We find no statistically significant difference between the impact of the mandate to offer coverage versus the mandate to provide coverage. The coefficients for the mandate to offer are larger but also less precisely estimated due to less variation in this variable. We do find a marginally significant effect on high birth weight prevalence in the case of the mandates to provide coverage. Given that this coefficient is positive, while the coefficient on the mandate to offer coverage is negative, it is possible that it is due to higher moral hazard of increased weigh gain associated with the mandate to provide coverage.

Second, we test to see if the obtained effect is sensitive to changes in functional form and to our choice of coding the law. We find the results are robust to using log dependent variable<sup>59</sup> and to coding of the timing of impact. For instance, because in many states the effective date of diabetes mandates was either exactly on July 1<sup>st</sup> (the cut-off used to distinguish between a year with mandates and one without) (9 states) or immediately afterwards, in row 3 of Table 9 the

added before taking the log.

26

<sup>&</sup>lt;sup>59</sup> Because the dependent variable can take zero values, we add 1 (one) to the dependent variable before taking the log. We prefer the specification in levels because it is known the estimates are somewhat sensitive to the value

diabetes mandates variable is coded 1 if diabetes mandates became effective before July 1<sup>st</sup> of that year, and 0.5 if diabetes mandates became effective in the first week of July. Reassuringly, the results obtained using this specification are substantially the same. Similarly, we find that our results are robust to using the enactment date. Note that statistically significant effects occur two years after enactment, to be expected given the lag between enactment and effective date.

Third, we test if secular differences in trends between adopting and non-adopting states (not already captured by controls) confound our results. In row 5 of Table 8, we find that our results are robust in regressions restricted to states that passed diabetes mandates. In Table 6 we also reported that the difference-in-difference estimates obtained on the sample of infants born to diabetic mothers are substantially the same with our main estimates. These results remove any concerns that potential secular differences between infant health of diabetic versus non-diabetic mothers (not already captured by controls) confound our estimates.

We further investigate whether our estimates are biased by potential endogeneity due to pre-existing trends by using leads of diabetes mandates in our main specification. The lead diabetes mandate variable is not significant, providing support for our identification. A similar test involves using leads of the enactment year. To the extent to which there is endogeneity, it would be more likely to show prior to enactment than it is prior to the year the law became effective. We find no evidence of a significant correlation between the enactment of diabetes mandates and prior infant outcomes.

The results are also robust to the sample choice. In the introduction we mentioned that the incidence of diabetes in population has increased. This could raise the concern that there may be changes in the characteristics of the treatment group before and after the introduction of mandates that may confound our estimates.<sup>60</sup> To eliminate this concern in row 9 of Table 8 we concentrate on a smaller sample of years before and after the period when most diabetes mandates laws became effective, 1998-1999. Estimates obtained using the 1995-2001 data are substantially the same as those obtained using the entire sample, providing support for our identifying strategy.<sup>61</sup>

<sup>60</sup> We also find no evidence that the mandates led to a change in share of births to diabetic mothers (coefficient 0.024 with standard errors 0.118).

<sup>61</sup> In addition, we found no evidence that the time passed since implementation matters for the magnitude of the effect (results reported in The Supplemental Results Appendix available on request).

In addition, we test the robustness of the results to adding years. The initial choice of sample reflected the need to cover as many instances of reforms while maintaining a manageable sample to test our results on individual level data. Here we show that adding one year at the beginning of the sample and one year at the end does not alter the results.

### 5. CONCLUSION

The question of how to best improve infant outcomes is of great importance in a country that lags behind other developed countries. This is becoming even more pressing when current trends predict women will experience even higher rates of obesity and diabetes, known predictors of poor infant health. This paper investigates the impact of diabetes mandates, which improve access to medical care but may be accompanied by the moral hazard of decreased preventive self-care among women during their fertile years. This paper contributes to the empirical literature on the effect of mandated benefits on health by estimating the impact of diabetes mandates on one measure of infant health – abnormal birth weight infants.

There are four prime conclusions from our analysis:

- 1. The mandates are associated with an reduction in the prevalence of premature birth (≤32 weeks) among diabetic mothers with more than 12 years of education. Among the same demographic group it is also associated with a decrease in the prevalence of very low birth weight (<1500 grams), and the prevalence of low birth weight (<2500 grams). Our estimates point out the demographic categories that experience the most gains: African Americans and older women.
- 2. There is no evidence of a significant change in the mean prevalence of high birth weight, however there is some evidence of an effect among the demographic groups most likely to be treated: those with the highest prevalence of private insurance and especially fully-funded private insurance.
- 3. A positive correlation between the adoption of diabetes mandates and the likelihood that diabetic mothers over 30 years old gain more than 35 pounds during pregnancy. The effect is significant at conventional significance levels only among African-American mothers of 30 to 35 years old. This result is quite plausible since infants of older women and African-American women were found to have experienced the largest improvement in the left tail of the birth weight distribution. Thus, it seems likely that these are the categories for which mandates are

binding. Nevertheless, the result raises concerns about the potential deleterious effect of behavioral changes triggered by diabetes mandates.

4. No effect was found on the outcomes of infants born to women with high-school education or less. This seems reasonable since such women are less likely to hold private insurance and, thus, to be affected by diabetes mandates.

Of the above results, the weak effect of diabetes mandates on the incidence of high birth weight babies is perhaps the most thought provoking. One explanation is that the biological mechanisms behind the correlation between maternal diabetes and high birth weight are different from the mechanisms linking maternal diabetes, and prematurity and low birth weight. Another is that moral hazard offsets the positive impact of mandates, while possibly affecting each tail of the birth weight distribution differently. The finding that diabetic women over thirty are more likely to gain more than 35 pounds during pregnancy provides supportive evidence of the latter proposed explanation, but does not reject the hypothesis that other factors also play a role.

If it is indeed true that moral hazard explains the poor improvement in high birthweight prevalence, it might also be the case that without this moral hazard effect, the decrease in premature and low birth rate prevalence associated with the diabetes mandates would be greater. Nevertheless, overall our results suggest that a comprehensive investigation of all parties affected by mandates in warranted. While it is possible that the health of people with diabetes suffered due to an increase in obesity rates, we found an improvement in outcomes of infants born to diabetic women. Likely further investigation of the effects of diabetes mandates on other conditions such as chronic hypertension, vasculopathies, and heart problems might be useful in determining the efficacy of these mandates.

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### References:

Almond, Douglas, Hilary W. Hoynes, and Diane Whitmore Schanzenbach "Inside the War on Poverty: The Impact of Food Stamps on Birth Outcomes" *The Review of Economics and Statistics*, May 2011, 93(2): 387-403

Ashenfelter, Orley. "Estimating the effect of training programs on earnings." *The Review of Economics and Statistics*, 1978, 60(1): 47-57.

Alderman, Harold, and Jere E. Behman, "Reducing the Incidence of Low Birth Weight in Low-Income Countries Has Substantial Economic Benefits," *The World Bank Research Observer*, 21 No. 1 (2006), 25-48.

Almond, Douglas, Kenneth Y. Chay, and David S. Lee, "The Costs of Low Birth Weight," *The Quarterly Journal of Economics*, August 2005, 1031-1083.

Almond, Douglas; Joseph J. Doyle Jr.; Amanda G. Kowaski; and Heidi Williams, "Estimating Marginal Returns to Medical Care: Evidence from At-Risk Newborns," *The Quarterly Journal of Economics*, May 2010, 593-634.

Arias E.; Anderson RN; Kung HC; Murphy SL; Kochanek KD. Deaths: Final data for 2001. National vital statistics reports; vol 52 no 3. Hyattsville, Maryland: National Center for Health Statistics. 2003

Behrman, Jere R., and Mark R. Rosenzweig, "Returns to Birthweight," *The Review of Economics and Statistics*, May 2004, 586-601.

Behrman, Richard E.; and Adrienne Stith Butler, Editors, "Preterm Birth: Causes, Consequences, and Prevention," Committee on Understanding Premature Birth and Assuring Healthy Outcomes, National Academies Press, 2007

Bertrand, Marianne; Esther Duflo; and Sendhil Mullainathan. "How much should we trust differences- in-differences estimates?" *Quarterly Journal of Economics*, February 2004, 119(1): 249-275.

Bitler, Marianne P. "Effects of Increased Access to Infertility Treatment on Infant and Child Health: Evidence from Health Insurance Mandates." 2008 available at http://www.socsci.uci.edu/~mbitler/research.html

Bitler, Marianne P. and Christopher S. Carpenter. "Insurance Mandates and Mammography," NBER Working Paper No. 16669, 2011.

Bitler, Marianne P. and Christopher S. Carpenter. "Effects of State Cervical Cancer Insurance Mandates on Pap Test Rates," 2012.

Bitler, Marianne P., and Lucie Schmidt. 2012 "Utilization of Infertility Treatments: The Effects of Insurance Mandates." *Demography*, 49(1): 124-149

Black, Sandra E.; Paul J. Devereux; and Kjell G. Salvanes, "From the Cradle to the Labor Market? The Effect on Adult Outcomes," *The Quarterly Journal of Economics*, February 2007, 122(1): 404-439.

Brown ZA, Mills JL, Metzger BE et al. "Early sonographic evaluation for fetal growth delay and congenital malformations in pregnancies complicated by insulin-requiring diabetes." *Diabetes Care*, 1992, 15: 613–619.

Susan H. Busch and Colleen L. Barry, "New Evidence on the Effects of State Mental Health Mandates," *Inquiry*, Fall 2008, 45(3): 308-322.

Centers for Disease Control and Prevention (CDC). National Diabetes Fact Sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 2011.

Cesur Resul and Inas Rashad Kelly. "From Cradle to Classroom: High Birth Weight and Cognitive Outcomes" *Forum for Health Economics and Policy*, 2010; 13(2) (Health Economics), Article 2, 2010

Chen Aimin, Shingairai A. Feresu, Cristina Fernandez, and Walter J. Rogan, "Maternal Obesity and the Risk of Infant Death in the United States" *Epidemiology*, January 2009, 20(1): 74-81

Combs CA; Gunderson E; Kitzmiller JL; Gavin LA; Main EK. "Relationship of fetal macrosomia to maternal postprandial glucose control during pregnancy." *Diabetes Care*, October 1992;15(10):1251-1257.

Comess LJ; Bennett PH; Burch TA et al. "Congenital anomalies and diabetes in the Pima Indians of Arizona." *Diabetes*, 1969, 18: 471–477

Conley, Dalton and Neil G. Bennett, "Birth Weight and Income: Interactions Across Generations," *Journal of Health and Social Behavior*, December 2001; 42: 450-465.

Corman, Hope and Stephen Chaikind, "The Effect of Low Birthweight on the School Performance and Behavior of School-Aged Children," *Economics of Education Review*, 1998, 17(3): 307-316.

Coustan Donald R. "Pre-conception planning: the relationship's the thing." *Diabetes Care*, 1998, 21(6): 887–888

Currie, Janet and Rosemary Hyson. "Is the Impact of Health Shocks Cushioned by Socioeconomic Status? The case of Low Birthweight." *American Economic Review*, May 1999, 89(2): 245-250.

Dabelea D, Snell-Bergeon JK, Hartsfield CL, Bischoff KJ, Hamman RF, McDuffie RS. "Increasing prevalence of gestational diabetes mellitus (GDM) over time and by birth cohort: Kaiser Permanente of Colorado GDM Screening Program." *Diabetes Care*, March 2005, 28(3): 579–584.

Danielzik S; Czerwinski-Mast M; Langnäse K; Dilba B; Müller MJ. "Parental overweight, socioeconomic status and high birth weight are the major determinants of overweight and obesity in 5–7 y-old children: baseline data of the Kiel Obesity Prevention Study (KOPS)." *International Journal of Obesity and Related Metabolic Disorders*, November 2004, 28(11): 1494-1502.

Dunne FP; Avalos G; Durkan M; Mitchell Y; Gallacher T; Keenan M; Hogan M; Carmody LA; Gaffney G; ATLANTIC DIP collaborators. "ATLANTIC DIP: Pregnancy outcome for women with pre-gestational diabetes along the Irish Atlantic seaboard." *Diabetes Care*, July 2009. 32(7): 1205-6.

Ehrenberg Hugh M.; Brian M. Mercer; and Patrick M. Catalano. "The influence of obesity and diabetes on the prevalence of macrosomia." *American Journal of Obstetrics and Gynecology*, September 2004, 191(3): 964-968.

Ferrara, Assiamira; Hedderson Monique M., Quesenberry Charles P., Selby Joseph V. "Prevalence of Gestational Diabetes Mellitus Detected by the National Diabetes Data Group or the Carpenter and Coustan Plasma Glucose Thresholds." *Diabetes Care*, September 2002, 25(9): 1625–1630.

Flegal Katherine M., Margaret D. Caroll, Cynthia L. Ogden, Lester R. Curtin. "Prevalence and Trends in Obesity Among US Adults, 1999-2008," *JAMA*, January 13, 2010, 303(3): 235-241.

Fryar, Cheryl D., Cynthia L. Ogden. "Prevalence of Underweight Among Adults Aged 20 Years and Over: United States, 2007-2008," CDC, National Center for Health Statistics, October 2010.

Fuchs, Victor. "Time Preference and Health: An Exploratory Study," in Victor Fuchs Editor, *Economic Aspects of Health*, Chicago: University of Chicago Press, 1982: 93-120.

Gabel, Jon R., Gail A. Jenson, and Samantha Hawkins. "Self-Insurance in Times of Growing and Retreating Managed Care," *Health Affairs*, March/April 2003, 22(2): 201-210.

Gilbert, William M., Thomas S. Nesbitt, and Beate Danielson, "The Cost of Prematurity: Quantification by Gestational Age and Birth Weight," *Obstetrics & Gynecology*, September 2003, 102(3): 488-492.

Grossman, Michael. "Education and Nonmarket Outcomes," in Eric Hanushek and Finis Welch Editors, *Handbook of the Economics of Education* Volume1, North Holland, 2006.

Gruber, Jonathan. 1994a. "The Incidence of Mandated Maternity Benefits." *The American Economic Review*, June 1994, 84 (3): 622-41.

Gruber, Jonathan. 1994b. "State Mandates Benefits and Employer Provided Insurance," *Journal of Public Economics*, 55(3): 433-464.

Hack, Maureen, Nancy K. Klein, and Taylor H. Gerry, "Long-Term Developmental Outcomes of Low Birth Weight Infants," *The Future of Children*, Spring 1995, 5(1): 176-196.

Harder, Thomas; Andreas Plagemann; and Anja Harder. "Birth Weight and Subsequent Risk of Childhood Primary Brain Tumors: A Meta-Analysis," *American Journal of Epidemiology*, 2008, 168 (4): 366-373.

Harder, Thomas; ElkeRodekamp, Karen Schellong, Joachim W. Dudenhausen, and Andreas Plagemann. "Birth Weight and Subsequent Risk of Type 2 Diabetes: A Meta-Analysis," *American Journal of Epidemiology*, 2007, 165(8): 849-857.

Harris Katherine M., Cristopher Carpenter, and Yuhua Bao, "The Effects of State Parity Laws on the Use of Mental Health Care," *Medical Care*, June 2006, 44(6): 499-505.

Hedderson, Monique M.; Ferrara, Assiamira; Sacks, David A. "Gestational Diabetes Mellitus and Lesser Degrees of Pregnancy Hyperglycemia: Association With Increased Risk of Spontaneous Preterm Birth", *Obstetrics & Gynecology*, October 2003, 102(4): 850-856.

Hjalgrim LL; Westergaard T; Rostgaard K; Schmiegelow K; Melbye M; Hjalgrim H; Engels EA. "Birth weight as a risk factor for childhood leukemia: a meta-analysis of 18 epidemiologic studies." *American Journal of Epidemiology*, October 2003,158(8):724–735.

Jensen Gail and Gabel Jon R. 1992. "State Mandated Benefits and the Small Firm's Decision to Offer Insurance," *Journal of Regulatory Economics* 4(4): 379-404.

Johnson, Rucker C. and Robert F. Schoeni, "The Influence of Early-Life Events on Human Capital, Health Status, and Labor Market Outcomes over the Life Course," forthcoming *B.E. Journal of Economic Analysis & Policy: Advances*.

Jovanovic-Peterson L; Peterson CM; Reed GF; Metzger BE; Mills JL; Knopp RH; Aarons JH. "Maternal postprandial glucose levels and infant birth weight: the Diabetes in Early Pregnancy Study. The National Institute of Child Health ad Human Development - Diabetes in Early Pregnancy Study." *American Journal of Obstetrics and Gynecology*, January 1991, 164(1 Pt 1):103-111.

Kahn, Matthew, "Diabetic Risk Taking: The Role of Information, Education, and Medication." *Journal of Risk and Uncertainty*, 1999, 18: 147-164.

Kerkhof M.; Koopman L.P.; Van Strien R.T.; Wijga A.; Smit H.A.; Aalberse R.C.; Neijens H.J., Brunekreef B.; Postma D.S.; Gerritsen J. "Risk factors for atopic dermatitis in infants at high risk of allergy: the PIAMA study." *Clinical and Experimental Allergy Journal of the British Society for Allergy and Clinical Immunology*, 2003, 33(10): 1336-1341.

Kirkegaard I; Obel C; Hedegaard M; Henriksen T. "Gestational Age and Birth Weight in Relation to School Performance of 10-Year-Old Children: A Follow-up Study of Children Born After 32 Completed Weeks." *Pediatrics*, October 2006, 118(4): 1600-1606.

Kim, Catherine and Assiamira Ferrara. "Gestational Diabetes During and After Pregnancy" Springer; 1<sup>st</sup> Edition, 2010.

Klick, Jonathan and Thomas Stratmann. "Diabetes Treatments and Moral Hazard," *Journal of Law and Economics*, 2007, 50(3): 519-638.

Knight, Kristin M.; Eva K. Pressman; David N. Hackney; Loralei L. Thornburg. "Perinatal outcomes in type 2 diabetic patients compared with non-diabetic patients matched by body mass index." *The Journal of Maternal-Fetal and Neonatal Medicine*, July 2011

Kochanek, Kenneth D.; and Joyce A. Martin, "NCHS Health E-Stat: Supplemental Analyses of Recent Trends in Infant Mortality," 2002.

Kraemer, Sebastian. "The Fragile Male." BMJ, 2000, 321: 1609–1612.

Kramer, Michael S. (1987a) "Intrauterine Growth and Gestational Determinants," *Pediatrics*, 80: 502-511.

Kramer, Michael S. (1987b) "Determinants of Low Birth Weight: Methodological Assessment and Meta-Analysis," *Bulletin of the World Health Organization* 65(5): 633-737.

Lawrence, Jean M.; Richard Contreras; Wansu Chen; and David A. Sacks. "Trends in the Prevalence of Preexisting Diabetes and Gestational Diabetes Mellitus Among a Racially/Ethnically Diverse Population of Pregnant Women, 1999–2005," *Diabetes Care*, May May 2008, 31(5): 899-904

Lewit, Eugene M.; Linda Schuurmann Baker; Hope Corman; and Patricia H. Shiono, "The Direct Cost of Low Birth Rate," *The Future of Children*, Spring 1995, 5(1): 35-56.

Li, Rui; Ping Zhang, Lawrence Barker, and DeKeely Hartsfield. 2010 "Impact of state mandatory insurance coverage on the use of diabetes preventive care." *BMC Health Services Research*, 10: 133

Livshits A.; Seidman DS. "Fertility issues in women with diabetes." *Womens Health (Lond Engl)*, 2009, 5:701–707.

Mandl, Lisa A.; Karen H. Costenbader; Julia Simard; and Elizabeth W. Karlson. "Is Birthweight Associated with Risk of Rheumatoid Arthritis? From a Large Cohort Study" *Annals of the Rheumatic Diseases*, April 2009; 68(4): 514-518

MacDorman; Marian F; T.J. Mathews. "Recent Trends in Infant Mortality in the United States," *NCHS Data Brief*, No. 9, October 2008

Mathews, Fiona; Paul J. Johnson; and Andrew Neil. "You are What Your Mother Eats: Evidence for Maternal Preconception Diet Influencing Foetal Sex in Humans." *Proceedings of the Royal Society B: Biological Sciences*, 2008: 1-8

McCormick, Marie C.; Judy C. Bembaum; John M. Eisenberg; Sharon Lee Kustra; and Emily Finnegan. "Costs Incurred by Parents of Very Low Birth Weight Infants After the Initial Neonatal Hospitalization." *Pediatrics*, September 1991, 88(3): 533-541.

McDonald, Sarah D; Zhen Han; Sohail Mulla; and Joseph Beyene. "Overweight and obesity in mothers and risk of preterm birth and low birth weight infants: systematic review and meta-analyses." *British Medical Journal*, July 2010; 341:c3428.

Meyer, Bruce D. "Natural and Quasi-Experiments in Economics." *Journal of Business & Economic Statistics*, April 1995, 13(2): 151-161.

Naeye, Richard L. "Maternal body weight and pregnancy outcome," *American Journal of Clinical Nutrition*, August 1990, 52(2): 273-279

National Center for Health Statistics, "Health, United States 2008, with Chart book" Hyattsville, MD: 2009.

Nicholson, Wanda K.; Harold E. Fox; Lisa A. Cooper; Donna Strobino; Frank Witter; Neil R. Powe. "Maternal Race, Procedures, and Infant Birth Weight in Type 2 and Gestational Diabetes," *Obstetrics & Gynecology*, September 2006, 108(3) Part 1: 626-634.

Ostlund, Ingrid; Ulf Hanson; Anders Björklund; Ragnhild Hjertberg; Nord Eva; Elisabeth Nordlander; Marja-Liisa Swahn ML; and Jan Wager. "Maternal and Fetal Outcomes if Gestational Impaired Glucose Tolerance is Not Treated." *Diabetes Care*, July 2003; 26(7): 2107-2111.

Owens, Lisa A.; Eoin P. O'Sullivan; Breeda Kirwan; Gloria Avalos; Geraldine Gaffney; Fidelma Dunne, and for the ATLANTIC DIP Collaborators. "ATLANTIC DIP: The Impact of Obesity on Pregnancy Outcome in Glucose-Tolerant Women." *Diabetes care*, March 2010, 33(3): 577-579.

Pacula, Rosalie Liccardo and Roland Sturm. "Mental Health Parity Legislation: Much Ado about Nothing?" *Health Services Research*, April 2000, 35(1 Pt 2):263-275.

Paneth, Nigel S., "The Problem of Low Birth Rate." *The Future of Children*, Spring 1995, 5(1): 19-34.

Park, Christina H. "Prevalence of Employer Self-Insured Health Benefits: National and State Variation." *Medical Care Research and Review*, 2000, 57(3): 340-60.

Pedersen JF, Mølsted-Pedersen L, Mortensen HB. "Fetal growth delay and maternal hemoglobin A<sub>1c</sub> in early diabetic pregnancy." *Obstetrics Gynecology*, 1984, 64: 351–352

Petrou, S.; T. Sach; and L. Davidson, "The Long-Term Costs of Preterm Birth and Low Birth Weight: Results of a Systematic Review," *Child: Care, Health and Development*, March 2001, 27(2): 97-115.

Pollitz, Karen; Eliza Bangit; Kevin Lucia; Mila Kofman; Kelly Montgomery, and Holly Whelan. "Falling through the Cracks: Stories of How Health Insurance Can Fail People with Diabetes." Washington, D.C.: Georgetown University Health Policy Institute and the American Diabetes Association, 2005.

Remes ST; Patel SP; Hartikainen AL; Jarvelin MR; Pekkanen J. "High birth weight, asthma and atopy at the age of 16 years." *Pediatric Allergy and Immunology*, 2008; 19(6):541-543

Richards Marcus; Hardy Rebecca; Kuh Diana; Wadsworth Michael EJ. "Birth weight and cognitive function in the British 1946 birth cohort: longitudinal population based study." *British Medical Journal*, 2001,322: 199-203.

Rosenberg, Terry J.; Samantha Garbers; Heather Lipkind; and Mary Ann Chiasson. "Maternal Obesity and Diabetes as Risk Factors for Adverse Pregnancy Outcomes: Differences Among 4 Racial/Ethnic Groups." *American Journal of Public Health*, September 2005, 95(9): 1545-1551.

Schaefer-Graf, Ute M.; Siri L. Kjos; ÖmerKilavuz; Andreas Plagemann; Martin Brauer; Joachim W. Dudenhausen; and Klaus Vetter. "Determinants of Fetal Growth at Different Periods of Pregnancies Complicated by Gestational Diabetes Mellitus or Impaired Glucose Tolerance," *Diabetes Care*, January 2003, 26(1): 193-198.

Schmitt, Susan K.; LaShika Sneed; and Ciaran S. Phibbs, "Costs of Newborn Care in California: A Population Based Study," *Pediatrics*, January 2006; 117(1): 154-160.

Sin, Don D.; Sheldon Spier; Lary W. Svenson; Don P. Schopflocher; Ambikaipakan Senthilselvan; Robert L Cowie; S.F. Paul Man. "The Relationship Between Birth Weight and Childhood Asthma. A Population-Based Cohort Study." *Archives of Pediatrics & Adolescent Medicine*. January 2004; 158(1): 60-64.

Sloan, Frank A., and Christopher J. Conover. Fall 1998. "Effects of State Reforms on Health Coverage of Adults," *Inquiry* 35: 280-93.

Tommiska, Viena; Risto Tuominen and Vineta Fellman, "Economic Costs of Care in Extremely Low Birthweight Infants during the First Two Years of Life," *Pediatrics Critical Care Medicine*, April 2003, 4(2): 157-163.

Tsou, Meng-Ting; Meng-Wen Tsou; Ming-Ping Wu; Jin-Tan Liu. "Academic Achievement of Twins and Singletons in Early Adulthood: Taiwanese Cohort Study," *British Medical Journal*, July 2008; 337:a438.

Visser GH; Bedekam DJ; Mulder EJ et al. "Delayed emergence of fetal behavior in type-1

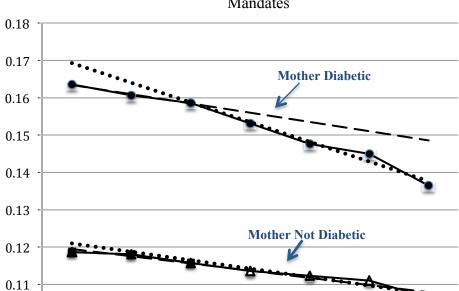
diabetic women." Early Human Development, 1985, 12:167–172

Wong, Shell Fean; Fung Yee Chan; Jeremy J.N. Oats; David H. McIntyre. "Fetal Growth Spurt and Pregestational Diabetic Pregnancy," *Diabetes Care*, October 2002, 25(10): 1681-1684.

Yogev Y, Langer O. Pregnancy outcome in obese and morbidly obese gestational diabetic women. *Eur J Obstet Gynecol Reprod Biol*. Mar 2008; 137(1): 21-6

Yuan, Wei; Basso Olga; Sorensen Henrik T. and Jorn Olsen. "Fetal growth and hospitalization with asthma during early childhood: a follow-up study in Denmark." *International Journal of Epidemiology* 2002;31(6):1240-1245

Zupanic, John A. F. "A Systematic Review of Costs Associated with Preterm Birth," Appendix D in Behrman, Richard E. and Adrienne Stith Butler, Editors, "Preterm Birth: Causes, Consequences, and Prevention," National Academies Press, 2007: 688-724.



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Figure 1. High Birthweight Incidence Before and After Diabetes Mandates

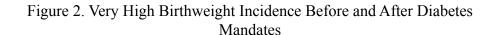
The solid line represents the incidence of high birth weight (>4000grams) among infants born in the years preceding and following diabetes mandates adoption at time t. The long dash line represents the trend in high birth weight determined by data 3 years before diabetes mandates became effective and up to and including the year prior to adoption. The short dash line is the trend in high birth weight after the adoption determined by data starting the year of adoption and up to the third year following adoption.

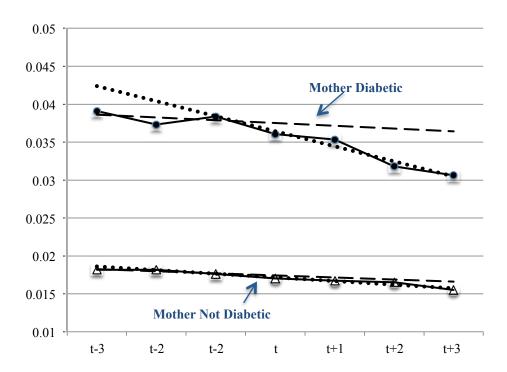
t+1

t

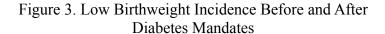
t+2

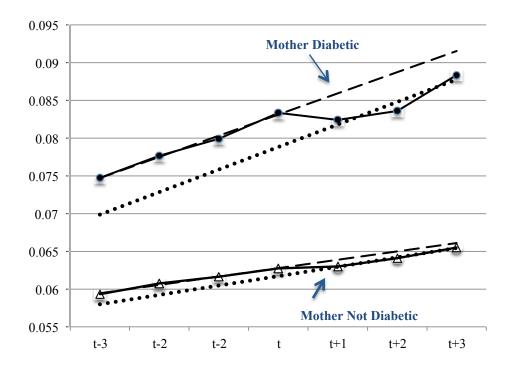
t+3



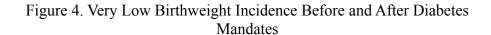


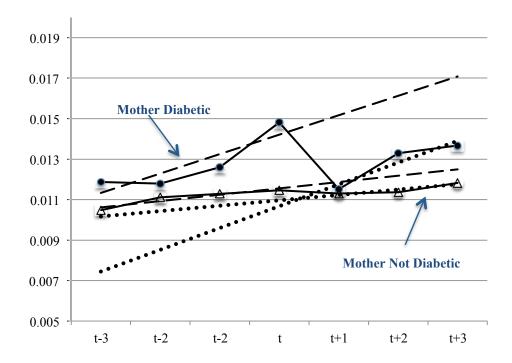
The solid lines represent the incidence of very high birth weight (>4000grams) among infants born in the years preceding and following diabetes mandates adoption at time t. The long dash lines represent the trend in very high birth weight determined by data 3 years before diabetes mandates became effective and up to and including the year prior to adoption. The short dash lines are the trend in very high birth weight after the adoption determined by data starting the year of adoption and up to the third year following adoption.



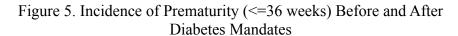


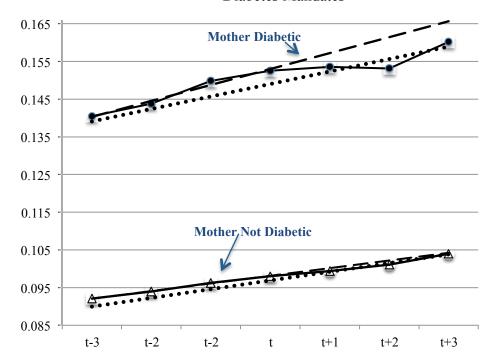
The solid lines represent the incidence of low birth weight (<2500grams) among infants born the years preceding and following diabetes mandates adoption at time t. The long dash lines represent the trend in low birth weight determined by data 3 years before diabetes mandates became effective and up to and including the year of adoption. The short dash lines are the trend in low birth weight after the adoption determined by data starting the year following adoption and up to the third year following adoption.



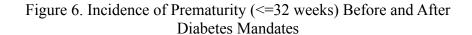


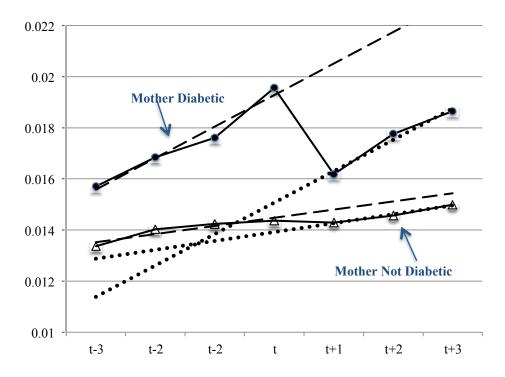
The solid lines represent the incidence of very low birth weight (<1500grams) among infants born in the years preceding and following diabetes mandates adoption at time t. The long dash lines represent the trend in very low birth weight determined by data 3 years before diabetes mandates became effective and up to and including the year of adoption. The short dash lines are the trend in very low birth weight after the adoption determined by data starting the year following adoption and up to the third year following adoption.





The solid lines represent the incidence of premature birth ( $\leq$  36 weeks) among infants born in the years preceding and following diabetes mandates adoption at time t. The long dash lines represent the trend in premature births determined by data 3 years before diabetes mandates became effective and up to and including the year of adoption. The short dash lines are the trend in premature births after the adoption determined by data starting the year following adoption and up to the third year following adoption.





The solid lines represent the incidence of very premature birth ( $\leq$  32 weeks) among infants born in the years preceding and following diabetes mandates adoption at time t. The long dash lines represent the trend in very premature births determined by data 3 years before diabetes mandates became effective and up to and including the year of adoption. The short dash lines are the trend in very premature births after the adoption determined by data starting the year following adoption and up to the third year following adoption.

Table 1: Diabetes Mandates Legislation

Table 1: Diabetes Mand		
State	Effective Date	Legislation
Alaska	July 27, 2000	ST §21.42.390
Arizona	January 1, 1999	A.R.S. §20-826(P), §20-934
Arkansas	August 1, 1997	ST §23-79-603
California	January 1, 2000	HLTH & S §1367.51
Colorado	July 1, 1998	ST §10-16-104 (subsection 13)
Connecticut	October 1, 1997	§38a-492(d)
Delaware	September 29, 2000	18§3560
District of Columbia	October 21, 2000	DC CODE §31-3001
Florida	July 1, 1995	FL ST §627.65745
Georgia	July 1, 2002	§33-24-59.2
Hawaii	July 1, 2001	HI ST §432:1-612
Illinois	January 1, 1999	215 ILCS 5/356w - (H. 3427)
Indiana	January 1, 1998	IN ST 27-8-14.5-4
Iowa	July 1, 1999	IA ST §514C.18
Kansas	January 1, 1999	KS ST § 40-2,163
Kentucky	July 15, 1998	KY ST §304.17A-148
Louisiana	January 1, 1998	LA R.S. 22:1034
Maine	July 4, 1996	ME ST T. 24 §2332-F:
Maryland	October 1, 1997	MD INSURANCE §15-822
Massachusetts	August 2, 2000	MA ST 118E §10C
Michigan	March 28, 2001	MI ST 500.3406p
Minnesota	August 1, 1994	MN ST §62A.45
Mississippi	January 1, 1999	MS ST § 83-9-46
Missouri	January 1, 1998	MS ST § 83-9-46
Montana	January 1, 2002	MT ST 33-22-129
Nebraska	October 1, 1999	NE ST § 44-790
Nevada	January 1, 1998	NV ST 689A.0427
New Hampshire	January 1, 1998	NH ST §415:6-e
New Jersey	January 5, 1996	NJ ST 17:48-6n
New Mexico	January 1, 1998	
New York	<u> </u>	NM ST §59A-22-41
North Carolina	January 1, 1994	NY INS §3216:
	October 1, 1997	NC ST § 58-51-61:
Oklahoma	November 1, 1996	OK ST T. 36 §6060.2.
Oregon	January 1, 2002	OR ST §743.694.
Pennsylvania	February 13, 1999	40 P.S. §764e.
Rhode Island	January 1, 1997	RI ST §27-18-38
South Carolina	January 1, 2000	SC ST § 38-71-46.
South Dakota	July 1, 1999	SD ST §58-18-83
Tennessee	January 1, 1998	TN ST § 56-7-2605
Texas	January 1, 1998	TX INS §1358.001-TX INS §1358.005
Utah	July 1, 2000	UT ST §31A-22-626.
Vermont	October 1, 1997	VT ST T. 8 §4089c.
Virginia	July 1, 1999	VA ST §38.2-3418.8
Washington	January 1, 1998	WA ST 48.20.391
West Virginia	June 8, 1996	WV ST § 33-15C-1:
Wisconsin	April 7, 1988	WI Stat Ann §632.895[6]
Wyoming	July 1, 2001	WY ST §26-20-201

Table 2: Summary Statistics

		All		Moth	er Diabetic	
	Non-Adopting	Adopting		Non-Adopting	Adopting	
	States	States	t	States	States	t
	[1]	[2]	[3]	[4]	[5]	[6]
HBW >4000g	12.003	11.551	-0.65	16.491	15.726	-0.29
	(2.989)	(2.828)		(4.166)	(3.573)	
HBW >4500g	1.875	1.797	0.45	3.980	3.822	0.51
	(0.748)	(0.703)		(1.966)	(1.766)	
LBW <2500g	6.125	6.485	0.75	7.563	8.196	-0.43
	(2.379)	(2.253)		(3.535)	(3.637)	
LBW <1500g	1.150	1.256	0.90	1.179	1.322	-1.13
	(0.780)	(0.773)		(1.405)	(1.395)	
Premature ≤36	9.548	10.071	0.19	14.407	15.224	-1.21
weeks	(2.867)	(2.677)		(4.813)	(4.528)	
Premature ≤32	1.487	1.585	1.17	1.667	1.868	-0.46
weeks	(0.975)	(0.923)		(1.668)	(1.730)	
Female	48.736	48.749	-0.18	47.924	48.295	0.92
	(1.155)	(1.043)		(4.758)	(4.291)	
Plural	3.097	3.404	-0.33	4.249	4.665	-0.56
	(1.081)	(1.182)		(2.759)	(2.630)	
First child	42.604	42.595	-0.21	39.930	39.472	-1.42
	(14.843)	(14.199)		(14.529)	(13.844)	
Mother's Age	29.352	29.603	0.97	31.040	31.295	0.80
	(4.880)	(4.969)		(4.848)	(4.902)	
Mother-Black	11.805	11.783	-0.06	12.751	13.509	0.39
	(32.270)	(32.3263)		(33.362)	(34.232)	
Mother's	15.035	15.113	1.26	14.992	15.045	1.10
Education	(0.537)	(0.538)		(0.424)	(0.425)	
Mother	84.979	83.957	-1.16	86.857	85.464	-1.32
Married	(16.651)	(17.088)		(13.301)	(13.833)	
No Prenatal	0.548	0.473	-0.60	0.238	0.237	-0.64
Care	(0.701)	(0.526)		(0.608)	(0.526)	
Log (wage)	2.576	2.638	0.74	2.570	2.638	0.91
	(0.141)	(0.153)		(0.142)	(0.153)	
Physicians/	2.283	2.494	1.25	2.280	2.554	1.50
1000 pop	(0.544)	(0.571)		(0.545)	(0.591)	
Hospitals/	2.475	2.240	-0.68	2.486	2.216	-0.93
100,000 pop	(1.109)	(0.972)		(1.087)	(0.941)	

All means are calculated for the sub-sample of infants born to mothers with more than 12 years of education. Columns 1, 2, 4, and 5 report averages of state-year observations with no non-economic damages caps. Columns 1 and 4 isolate the observations corresponding to states that did not adopt the caps in the following year. Columns 2 and 5 isolate the observations corresponding to states that adopted the caps in the following year. Standard errors clustered at state level are reported in parentheses. Columns 3 and 6 report t-test of equality of means conditional on time fixed effects.

<sup>\*</sup>significant at 10% significance level, \*\* significant at 5% significance level, \*\*\* significant at 1% significance level.

Table 3. The Impact of Diabetes Mandates on Infant Outcomes

Timing of Impact	t	t+1	t+2
High Birth Weight			
>4000g	0.435*	0.323	0.223
	(0.245)	(0.238)	(0.254)
>4500g	0.091	0.059	-0.063
	(0.114)	(0.113)	(0.109)
Low Birth Weight			
<2500g	-0.128	-0.339**	-0.260**
	(0.119)	(0.151)	(0.129)
<1500g	-0.008	-0.192**	-0.073
	(0.089)	(0.080)	(0.072)
Premature Birth			
≤36 weeks	-0.215	-0.335	-0.187
	(0.275)	(0.281)	(0.245)
≤32 weeks	-0.042	-0.223**	-0.134*
	(0.100)	(0.099)	(0.080)
5 minutes Apgar Score			
<8	-0.025	-0.088	-0.019
	(0.140)	(0.135)	(0.117)

The dependent variable is the incidence of high birth weight, low birth weight, prematurity, or a 5 minutes Apgar Score less than 8 among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. Due to missing values for the 5 minutes Apgar Score all regressions reported in the last row use 8968 observations. All other regressions run on 9352 observations from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality, birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table 4: The Impact of Diabetes Mandates on Infant Outcomes by Mother's Age-Race

Demographic Group

Demographic		th Weight	Low Bir	th Weight	Prematu	re Birth	Weight
	>4000 g	>4500 g	<2500 g	<1500 g	≤36 weeks	≤32 weeks	Gain ≥35
				Panel A: WI	HITE		_
Age <25	-0.501	0.261	0.194	-0.069	0.689	0.321	-0.004
	(0.646)	(0.417)	(0.435)	(0.184)	(0.524)	(0.273)	(0.990)
$25 \ge Age > 30$	0.424	-0.235	-0.132	-0.185	-0.299	-0.315**	-0.137
	(0.384)	(0.162)	(0.208)	(0.136)	(0.375)	(0.141)	(0.463)
205 4 525	0.264	0.20044	0.206	0.164*	0.062**	0 404444	0.041
$30 \ge Age > 35$	0.264	0.308**	-0.306	-0.164*	-0.863**	-0.404***	0.041
	(0.431)	(0.141)	(0.194)	(0.090)	(0.383)	(0.110)	(0.345)
$Age \ge 35$	0.239	0.041	-0.303	-0.223***	0.459	-0.165	0.397
Agc ≥ 33	(0.347)			(0.082)	(0.362)	(0.130)	(0.425)
	(0.347)	(0.219)	(0.253)	Panel B: BL		(0.130)	(0.423)
Age <25	1.645	0.352	0.406	0.534	0.426	0.410	1.073
Age <23							
	(1.118)	(0.621)	(0.972)	(0.419)	(1.368)	(0.478)	(2.094)
25\ge Age >30	1.157	-1.287	-0.006	0.350	-2.194*	0.226	1.659
20_1180 00	(0.886)	(5.137)	(0.675)	(0.380)	(1.166)	(0.512)	(1.311)
	(0.000)	(0.157)	(0.070)	(0.200)	(1.100)	(0.012)	(1.011)
$30 \ge Age > 35$	0.005	-0.422	-1.516*	-0.828*	-0.321	-0.374	2.471**
-	(0.669)	(0.538)	(0.821)	(0.466)	(0.769)	(0.436)	(1.215)
$Age \ge 35$	-0.137	-0.692*	-2.150**	-1.053**	-1.090	-1.227**	2.001*
	(0.906)	(0.346)	(0.915)	(0.517)	(1.283)	(0.594)	(1.019)

Each cell reports estimates from a separate regression using individual level data. All regressions retain the subsample of infants born to mothers with more than 12 years of education. All regressions use data from the period 1992-2003. The number of observations varies across regressions from a low 298,593 births to Black women over 35 to a high 5,703,032 births to White women age 30-35. To improve readability all coefficients and standard errors were multiplied by 100. All regressions control for mother age, race, education, marital status, and prenatal care, infant gender, plurality, and birth order (dummy equal to 1 if first child and zero otherwise). All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table 5. The Impact of Diabetes Mandates on Infant Outcomes – by Gender

	High Bir	th Weight	Low Birt	h Weight	Prematu	Premature Birth	
	>4000 g	>4500 g	< 2500 g	<1500 g	≤36 weeks	≤32 weeks	
Female	0.061	-0.056	-0.150	-0.216**	-0.226	-0.256**	
	(0.252)	(0.116)	(0.220)	(0.098)	(0.274)	(0.115)	
	,	, ,	` ,	, ,	, ,	,	
Male	0.463	0.150	-0.464**	-0.154	-0.402	-0.167	
	(0.305)	(0.168)	(0.195)	(0.095)	(0.401)	(0.110)	

The dependent variable is the incidence of high birth weight, low birth weight or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. All regressions use data from the period 1992-2003. Regressions reported in first row use 9142 observations, while regressions reported in second row use 9154 observations. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

\* significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table 6: The Impact of Diabetes Mandates on Infant Outcomes - Falsification Tests

	High Birtl	n Weight	Low Bir	Low Birth Weight		re Birth		
	>4000 g	>4500 g	<2500 g	<1500 g	≤36 weeks	≤32 weeks		
		Panel A. Mandates effect by mothers' education: DDD						
Baseline: >12 years of	0.323	0.059	-0.339**	-0.192**	-0.335	-0.223**		
education	(0.238)	(0.113)	(0.151)	(0.080)	(0.281)	(0.099)		
Sample: $\leq 12$ years of	-0.047	0.039	0.079	-0.018	0.100	0.026		
education	(0.296)	(0.126)	(0.187)	(0.093)	(0.237)	(0.108)		
			***	ate ate ate		ate ate ate		
Sample: ≥16 years of	0.355	0.114	-0.580***	-0.323***	-0.450	-0.367***		
education	(0.301)	(0.153)	(0.208)	(0.118)	(0.386)	(0.136)		
	Panel B. Mandates effect by diabetic status: DD							
Diabetic	0.308	0.057	-0.368***	-0.225***	-0.389	-0.270***		
	(0.266)	(0.116)	(0.136)	(0.079)	(0.289)	(0.091)		
Non-diabetic	0.059	0.022	-0.043	-0.026*	-0.040	-0.024		
	(0.075)	(0.022)	(0.037)	(0.014)	(0.062)	(0.017)		
		Panel C. M	landates effe	ect by type of di	abetes: DDD	ate ate		
Diabetes•Mandate	0.362	0.098	-0.304**	-0.206**	-0.328	-0.227**		
	(0.237)	(0.116)	(0.141)	(0.078)	(0.271)	(0.100)		
	***	***	**	**		**		
Diabetes•Mandate• %	-0.046	-0.008	$0.009^{**}$	0.004**	-0.004	0.005**		
Gestational Diabetes	(0.008)	(0.003)	(0.004)	(0.002)	(0.006)	(0.002)		

The dependent variable is the prevalence of high birth weight, low birth weight or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions use data from the period 1992-2003. Regressions in rows 1, and 4-7 retain the sub-sample of infants born to mothers with more than 12 years of education. The regressions in row 2 retain the sub-sample of infants born to mothers with 12 or fewer years of education. The regressions in row 3 retain the sub-sample of infants born to mothers with 16 or more years of education. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects. In addition regressions in rows 1-3 and 6-7 include all two-way interactions between diabetic status, year FE, and state FE. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table 7. The Impact of Diabetes Mandates by Likelihood of Treatment

	Diabetes•	Diabetes• Mandate•	Diabetes•	Diabetes•Mandate•
	Mandate•Private	Small/Medium Firm	Mandate•Fully	Mostly Fully
	Insurance	Employment	Insured	Insured Industry
	[1]	[2]	[3]	[4]
High Birth Weight				
>4000g	-0.077***	-0.069*	-0.151***	-0.071*
	(0.025)	(0.035)	(0.041)	(0.035)
>4500g	-0.026*	-0.035*	-0.057**	-0.037**
	(0.015)	(0.020)	(0.025)	(0.017)
Low Birth Weight				
<2500g	-0.013	-0.014	-0.017	-0.009
	(0.017)	(0.025)	(0.032)	(0.025)
<1500g	-0.008	-0.012	-0.011	-0.004
	(0.010)	(0.009)	(0.017)	(0.011)
Premature Birth				
≤36 weeks	-0.074***	-0.092**	-0.164***	-0.068
	(0.020)	(0.045)	(0.044)	(0.052)
≤32 weeks	-0.002	-0.019	-0.002	-0.027**
	(0.013)	(0.012)	(0.021)	(0.013)

Each estimate comes from a different regression. The dependent variable is either the prevalence of high birth weight, low birth weight, or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. All regressions run on data from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table 8. The Impact of Diabetes Mandates: Mandate to Provide Coverage versus Mandate to Offer Coverage

			1101 00 101480			
	High Birt	th Weight	Low Birt	h Weight	Premat	ure Birth
	>4000 g	>4500 g	<2500 g	<1500 g	≤36 weeks	≤32 weeks
Mandate to:	_					
Cover diabetes	0.399*	0.093	-0.328**	-0.184**	-0.309	-0.218**
treatment	(0.230)	(0.112)	(0.150)	(0.080)	(0.286)	(0.100)
Offer coverage for	-0.770	-0.435	-0.501	-0.307*	-0.708**	-0.289
diabetes treatment	(0.740)	(0.293)	(0.343)	(0.155)	(0.318)	(0.205)
F-test of joint significance	2.34	1.70	2.63*	3.24**	2.48*	2.60*
F-test of equality of coefficients	2.47	3.19*	0.29	0.75	1.85	0.13

The dependent variable is the prevalence of high birth weight, low birth weight, or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. All regressions run on 9352 observations from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality, birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

\* significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table 9. Robustness check

	High Birt	th Weight	Low Bir	th Weight	Prematu	ıre Birth
	>4000 g	>4500 g	<2500 g	<1500 g	≤36 weeks	≤32 weeks
[1] Main	0.323	0.059	-0.339**	-0.192**	-0.335	-0.223**
	(0.238)	(0.113)	(0.151)	(0.080)	(0.281)	(0.099)
[2] I D 1 4	0.010	0.011	0.022*	0.072**	0.020	0.072**
[2] Log Dependent	0.018	0.011	-0.033*	-0.073**	-0.020	-0.073**
Variable	(0.014)	(0.026)	(0.017)	(0.030)	(0.016)	(0.033)
[3] Different Law	0.356	0.078	-0.322**	-0.183**	-0.304	-0.203*
Coding	(0.247)	(0.116)	(0.144)	(0.087)	(0.295)	(0.107)
543.79	0.060	0.06	0.0004	0.45044	0.240	0.00
[4] Enactment, t+2	0.363	0.062	-0.288*	-0.178**	-0.348	-0.207**
	(0.237)	(0.115)	(0.146)	(0.080)	(0.283)	(0.100)
[5] Adopting states	0.176	0.025	-0.286*	-0.135**	-0.176	-0.185*
only	(0.218)	(0.117)	(0.164)	(0.065)	(0.299)	(0.100)
Ollry	(0.210)	(0.117)	(0.104)	(0.003)	(0.299)	(0.100)
[6] Lead 1	0.488*	0.193*	-0.076	-0.038	-0.164	0.000
	(0.249)	(0.112)	(0.121)	(0.092)	(0.255)	(0.095)
[7] [ 10	0.170	0.001	0.012	0.001	0.207	0.020
[7] Lead 2	0.159	0.001	0.013	-0.091	-0.287	-0.020
	(0.264)	(0.122)	(0.136)	(0.091)	(0.228)	(0.083)
[8] Lead 1	0.163	0.023	0.049	-0.077	-0.296	-0.009
enactment	(0.269)	(0.124)	(0.137)	(0.093)	(0.232)	(0.085)
[9] 1995-2001	0.202	-0.034	-0.365*	-0.271***	-0.091	-0.253**
	(0.250)	(0.147)	(0.187)	(0.080)	(0.292)	(0.121)
[10] 1991-2004	0.307	0.024	-0.301**	-0.193**	-0.315	-0.206**
[10] 1991-2004						
	(0.251)	(0.115)	(0.141)	(0.075)	(0.289)	(0.083)

The dependent variable is the prevalence of high birth weight, low birth weight or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. Unless otherwise specified these regressions use data from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table A1. Summary Statistics

-		Mother Not Diabetic					
	Non-Adopting	Adopting					
	States	States	t				
HBW >4000g	11.879	11.427	-0.71				
	(2.851)	(2.707)					
HBW >4500g	1.817	1.737	0.28				
_	(0.585)	(0.539)					
LBW <2500g	6.085	6.434	0.76				
	(2.327)	(2.179)					
LBW <1500g	1.149	1.254	1.00				
	(0.756)	(0.747)					
Premature ≤36	9.414	9.917	0.20				
weeks	(2.671)	(2.442)					
Premature ≤32	1.482	1.577	1.23				
weeks	(0.949)	(0.887)					
Female	48.758	48.762	-0.28				
	(0.852)	(0.753)					
Plural	3.065	3.367	-0.34				
	(0.977)	(1.089)					
First child	42.678	42.688	-0.15				
	(14.846)	(14.208)					
Mother's Age	29.305	29.553	0.94				
	(4.874)	(4.965)					
Mother-Black	11.779	11.731	-0.08				
	(32.242)	(0.322)					
Mother's	15.036	15.115	1.27				
Education	(0.539)	(0.541)					
Mother Married	84.927	83.912	-1.16				
	(16.732)	(17.185)					
No Prenatal Care	0.556	0.480	-0.59				
	(0.702)	(0.524)					
Log (wage)	2.576	2.638	0.73				
	(0.141)	(0.153)					
Physicians/ 1000	2.283	2.492	1.24				
pop	(0.544)	(0.571)					
Hospitals/	2.475	2.241	-0.67				
100,000 pop	(1.110)	(0.974)					

All means are calculated for the sub-sample of infants born to mothers with more than 12 years of education. Columns 1 and 2 report averages of state-year observations with no non-economic damages caps. Column 1 isolates the observations corresponding to states that did not adopt the caps in the following year. Column 2 isolates the observations corresponding to states that adopted the caps in the following year. Standard errors clustered at state level are reported in parentheses. Column 3 reports t-test of equality of means conditional on time fixed effects. \* significant at 10% significance level, \*\* significance level, \*\* significance level, \*\* significance level.

Table A2. The Impact of Diabetes Mandates on Infant Outcomes: Sub-Sample Mothers with 12 or Fewer Years of Education

	of rewel rears c	71 Education	
Timing of Impact	t	t+1	t+2
High Birth Weight			
>4000g	-0.145	-0.048	0.032
	(0.252)	(0.296)	(0.269)
>4500g	0.115	0.039	0.110
	(0.086)	(0.126)	(0.137)
Low Birth Weight			
<2500g	0.137	0.079	0.185
	(0.167)	(0.188)	(0.158)
<1500g	-0.052	-0.018	0.007
C	(0.087)	(0.093)	(0.078)
Premature Birth			
≤36 weeks	0.213	0.100	0.184
	(0.280)	(0.237)	(0.207)
≤32 weeks	-0.001	0.026	0.039
	(0.099)	(0.108)	(0.092)

The dependent variable is the prevalence of high birth weight, low birth weight or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with 12 or fewer years of education. All regressions run on 9314 observations from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

\* significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table A3: The Impact of Diabetes Mandates on Infant Outcomes by Mother's Age-Race Demographic Group: Data collapsed by state/year/demographic group cells

_>	>4000 g	>4500 g	.2500				Weight	
			<2500 g	< 1500 g	≤36 weeks	≤32 weeks	Gain≥35	
	Panel A: WHITE							
All ages 0.	.271	0.092	-0.213	-0.162**	-0.240	-0.206**	-0.056	
(0	0.263)	(0.123)	(0.134)	(0.076)	(0.278)	(0.090)	(0.321)	
Age < 25 -0	0.951	0.002	0.109	0.070	0.429	0.328	-0.732	
•		(0.605)	(0.523)	(0.298)	(0.882)	(0.414)	(1.421)	
(1	1.023)	(0.003)	(0.323)	(0.298)	(0.882)	(0.414)	(1.421)	
$25 \ge Age > 30$ 0.	.321	-0.243	-0.353	-0.172	-0.418	-0.276	-0.382	
0)	0.550)	(0.258)	(0.289)	(0.212)	(0.547)	(0.219)	(0.726)	
20> 4 = 25 0	.243	0.355	-0.143	-0.155	-0.789	-0.313*	-0.095	
ě .								
(0	0.616)	(0.216)	(0.289)	(0.133)	(0.509)	(0.157)	(0.470)	
Age $\geq 35$ 0.	.407	0.069	-0.215	-0.219	0.388	-0.157	0.528	
_(0	0.534)	(0.297)	(0.348)	(0.132)	(0.522)	(0.196)	(0.671)	
			]	Panel B: BL	ACK			
All ages 0.	.374	-0.213	-1.024**	-0.358	-0.951	-0.320	1.450**	
(0)	0.458)	(0.326)	(0.472)	(0.250)	(0.582)	(0.293)	(0.577)	
A = = < 25 1	.224	0.111	0.462	0.252	0.649	-0.008	0.294	
· ·								
(1	1.667)	(0.907)	(1.688)	(0.696)	(2.220)	(0.734)	(3.196)	
25≥ Age >30 0.	.706	0.016	-0.045	0.226	-1.477	0.118	0.666	
(1	1.357)	0.871	(1.027)	(0.552)	(1.742)	(0.794)	(1.790)	
Č		-0.286	-1.650	-0.682	-0.530	-0.383	2.157	
(0	0.947)	(0.859)	(1.248)	(0.646)	(1.123)	(0.710)	(1.648)	
$Age \ge 35 \qquad -0$	0.013	-0.470	-2.326*	-0.962	-1.902	-1.086	2.127	
ě	1.350)	(0.547)	(1.280)	(0.743)	(1.810)	(0.865)	(1.659)	

Each cell reports estimates from a separate regression. The dependent variable is the prevalence of high birth weight, low birth weight, prematurity or mother pregnancy weight gain above 35 pounds among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. All regressions use data from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level.

Table A4: The Impact of Diabetes Mandates on Singleton Birth Outcomes by Mother's Age-Race

Demographic Group

	High Birth Weight		Low Birth Weight		Premature Birth					
	>4000 g	>4500 g	<2500 g	<1500 g	≤36 weeks	≤32 weeks	Weight Gain ≥35			
		Panel A: WHITE								
Age <25	-0.503	0.263	0.449	0.158	0.470	0.281	-0.230			
	(0.657)	(0.424)	(0.408)	(0.150)	(0.536)	(0.194)	(1.081)			
25≥ Age >30	0.446	-0.236	0.022	-0.083	-0.092	-0.122	0.008			
	(0.401)	(0.168)	(0.196)	(0.103)	(0.401)	(0.100)	(0.473)			
30≥ Age >35	0.278	0.326	-0.227	0.060	-0.690**	-0.153**	0.091			
	(0.460)	(0.150)	(0.129)	(0.060)	(0.327)	(0.072)	(0.377)			
$Age \ge 35$	0.231	0.042	-0.060	0.146**	0.568	0.102	0.615			
<i>v</i> –	(0.374)	(0.237)	(0.230)	(0.068)	(0.341)	(0.091)	(0.396)			
	Panel B: BLACK									
Age <25	1.629	0.328	0.187	0.273	0.894	-0.095	1.604			
	(1.179)	(0.637)	(0.903)	(0.342)	(1.265)	(0.447)	(2.043)			
25≥ Age >30	1.241	-0.140	-0.253	0.065	-2.404**	-0.112	1.575			
	(0.936)	(0.536)	(0.740)	(0.337)	(1.152)	(0.464)	(1.300)			
30≥ Age >35	0.089	-0.444	-0.996	-0.396	-0.108	-0.185	2.343*			
C	(0.688)	(0.570)	(0.800)	(0.378)	(0.765)	(0.401)	(1.231)			
$Age \ge 35$	-0.223	-0.723**	-1.760**	-0.714	-0.813	-1.008**	1.452			
<b>U</b> –	(0.920)	(0.361)	(0.878)	(0.473)	(1.276)	(0.438)	(1.039)			

Each cell reports estimates from a separate regression using individual level data. All regressions retain the subsample of singleton births to mothers with more than 12 years of education. All regressions use data from the period 1992-2003. To improve readability all coefficients and standard errors were multiplied by 100. All regressions control for mother age, race, education, marital status, and prenatal care, infant gender, plurality, and birth order (dummy equal to 1 if first child and zero otherwise). All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table A5: Effect of Diabetes Mandates by Likelihood of Treatment: Proportion of Population with Individually Purchased Private Health Insurance

	Diabetes•Mandate•Individually purchased private insurance
High Birth Weight	
>4000g	0.031
C	(0.046)
>4500g	0.001
2	(0.019)
Low Birth Weight	,
<2500g	-0.031
	(0.023)
<1500g	0.010
	(0.013)
Premature Birth	(*******)
≤36 weeks	-0.015
	(0.052)
≤32 weeks	-0.009
	(0.019)

Each estimate comes from a different regression. The dependent variable is either the prevalence of high birth weight, low birth weight, or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. All regressions run on data from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

<sup>\*</sup> significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level

Table A6. The Effect of Diabetes Mandate: Time since Implementation

	High Birth Weight		Low Birth Weight			Premature Birth	
	>4000 g	>4500 g	<2500 g	<1500 g	≤36 weeks	≤32 weeks	
Diabetes•	0.104	0.081	-0.286*	-0.168**	-0.520*	-0.205*	
Mandate	(0.250)	(0.118)	(0.160)	(0.078)	(0.278)	(0.106)	
Diabetes• Time since Mandate	0.061	-0.001	-0.050	-0.034	0.111	-0.026	
	(0.041)	(0.023)	(0.053)	(0.022)	(0.087)	(0.028)	

Each estimate comes from a different regression. The dependent variable is either the prevalence of high birth weight, low birth weight, or prematurity among infants born to mothers from a demographic group defined by the race, age and diabetic status in a particular state and year. The variable "Time since Mandate" is equal to 1 in the year following the effective date of the mandate, 2 in the second year following the mandate adoption, 3 in the third, etc, and 0 in all state-years with no mandate (the effective date is also coded 0 because of the lag effect of the mandate). All regressions retain the sub-sample of infants born to mothers with more than 12 years of education. All regressions run on data from the period 1992-2003. All regressions control for mother's education, marital status, and prenatal care (percent women starting prenatal care in 2<sup>nd</sup> trimester, 3<sup>rd</sup> trimester, or that did not receive prenatal care), infant gender, plurality and birth order (percent first child), age-race demographic group fixed effects that are allowed to vary over time. All regressions control for diabetic status of the mother, state fixed effects, year fixed effects, and all their interactions. Robust standard errors clustered at state level are reported in parentheses.

\* significant at 10% significance level; \*\* significant at 5% significance level, \*\*\* significant at 1% significance level