Protect Young Mothers from Cigarettes, Help Their Babies?
A Regression Discontinuity Study on Minimum Cigarette Purchase Age*

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Abstract

A key target in the U.S. health policies is to reduce costly adverse birth outcomes to which prenatal smoking is one of the most significant contributors. This paper is the first to address whether implementing minimum cigarette purchase age of 21 at Pennsylvania can improve infant health through curbing smoking among young mothers. My research question is crucial because young mothers are heavily engaged in smoking and have more low birth weight babies, and smoking prevalence among mothers in Pennsylvania also exceeds the national average. The potential scope of this regulation is therefore large. I use a unique large dataset to find there is a 16 percent decrease in the average cigarettes smoked per day and a 20 percent decrease in low birth weight for mothers subject to the regulation at the cutoff. The 2SLS regression discontinuity estimates indicate that smoking 1 more cigarette per day during pregnancy worsens a variety of birth outcomes among all mothers. For smokers, it reduces birth weight by 61.17 grams, increases the probability of low birth weight by 2.8 percentage points, and decreases the APGAR 1 minute score by 0.13 points. The large intergenerational benefits induced by the law shed new light on the current political debate in many other states on whether enforcing MCPA 21.

Keywords: Infant Health, Maternal Smoking, Minimum Cigarette Purchase Age, Regression Discontinuity Design

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1 Introduction

Improving infant health has been one of the key targets in U.S. health policies. In the short run, augmenting birth weights lowers health care cost. This impact is particularly salient for low birth weight babies. The estimates by Almond et al (2005) [6] imply the excess hospital cost associated with a typical infant declines sharply from $6806 to $604 if increasing the baby's birth weight from the 1500-2000 gram(g) range to the 2000-2500 g range. The literature also suggests that compared to normal birth weight children, low birth weight children are more likely to have cognitive deficits (Hack et al, 1995 [30]), experience health problems (Corman and Chaikind, 1998 [20]), and have lower educational attainments (Currie and Hyson, 1999 [22]). In the long term, a poor birth outcome especially low birth weight is found to negatively affect adulthood health, employment and socioeconomic status (Case et al, 2005 [16]). Behrman and Rosenzweig(2004) [8] use a dataset of Minnesota female monozygotic twins to find a 1 pound increase in an infant's birth weight solely due to fetal growth rate increase would increase her schooling, adult height and earnings, respectively by one third of a year, 0.6 inches and 7 percent[1].

One of the most significant contributors for adverse birth outcomes is maternal cigarette smoking during pregnancy. Kramer(1987) [37] points to it as the single largest modifiable risk for low birth weight. A 2001 Surgeon General report further links maternal smoking with small for gestational age, preterm delivery, birth defects, infant mortality and a host of other poor birth outcomes (CDC, 2001 [17]). Although there is a consensus that prenatal smoking does harm to newborns, estimating the size of this effect is difficult due to unobserved heterogeneity across childbearing women. Previous studies which use different econometric approaches have yielded a broad range of estimates, with even the most conservative causal estimate implying a startling cost on American society. Yet thus far the direct government policy interventions are limited to facilitate pregnant women' accesses to smoking cessation services[2]. In contrast, there are many state level anti-smoking policies [3] that may effectively reduce cigarette consumption among childbearing women and therefore potentially increase their birth outcomes. The existing studies however pay much attention only on state cigarette taxes, leaving other important policies little addressed. Ringel and Evans (2001) [51] find pregnant women are very responsive to tax changes. A tax hike of $0.55 per pack would decrease maternal smoking by about 22%. There are

[1]The birth weight effects on both short and long run outcomes are also quantified in the context of countries other than U.S., see Black et al(2007) [10] for a Norwegian study.

[2]36 state Medicaid programs have covered at least one treatment for smoking cessation by 2001 (Halpin et al, 2003 [32]). From April 2000 to September 2005, American College of Obstetricians and Gynecologists (ACOG) provided training for clinicians to implement the newly established smoking cessation guideline.

[3]In addition to cigarette excise taxes, these policies include various youth access laws especially minimum cigarette purchase ages, restrictions on tobacco products, educational programs and smoke free indoor air laws, etc.

also evidences that a higher state cigarette tax or the introduction of a cigarette hike increases birth weight and decreases the prevalence of low birth weight babies(Evans and Ringel, 1999 [28]; Lien and Evans, 2005 [40]).

In this paper, I provide the first evaluation on whether and how much minimum cigarette purchase age (MCPA) of 21 effective between 1992 and 2002 at Pennsylvania (PA) has curbed prenatal smoking and improved infant health. This regulation has been an important state level anti-smoking policy directly targeted at young mothers yet rarely examined in previous studies. Figures 2-5 to 2-8 show that compared to older mothers, young mothers aged between 20 and 22 at PA were highly engaged in smoking during pregnancy with a higher level of average cigarette consumption, while they appeared to face higher risks of delivering lower birth weight infants[4]. The smoking prevalence at PA also exceeded the national average, as shown in figures 2.1-2.2. Therefore the scope for policy intervention of MCPA 21 is potentially large. Indeed, I find that the law can effectively curb prenatal smoking and improve a variety of infant health outcome measures. To address the concern of generalizability, it is worth noting that the distribution of prenatal smoking and infant health among young mothers at PA is close to the overall pattern in the United States (Figure 2.1-2.4). This law therefore would have a similar impact in promoting infant health, once established in other states. In this sense, my finding on the intergenerational benefits induced by MCPA 21 also provides novel insights for the current political debate on whether implementing minimum cigarette purchase age to 21 at other states[5]. My paper also advances the literature by exploiting the exogenous variation in maternal cigarette consumption induced by the legal purchase age of 21 to shed new light on the impact of prenatal smoking on infant health, under a regression discontinuity (RD) framework. The advantage of this identification strategy is that it is not subject to policy endogeneity, a concern often raised for state cigarette taxes, because MCPA 21 at PA has been kept for one decade[6]. It is also immune to confounding effects from changes in other tobacco control policies, which is another critique on using state cigarette taxes as instruments. Over the period when MCPA 21 was effective, other tobacco control policies at PA were nearly invariant except an increase in the level of smoke free air protection at public schools. This change and other county level policies were unlikely to differentially affect the smoking behavior or birth outcomes of the mothers around

[4]The occurrence of low birth weight babies in this subpopulation exceeded the average of the entire mother population, only less than teenager mothers.

[5]In June 2009, President Obama signed the Family Smoking Prevention and Tobacco Control Act, the nation's strictest anti-smoking law to date. One of the final stipulations of this bill orders a study to be conducted on the public health implications of implementing the minimum tobacco product purchase age above the current 18 years.

[6]As I discuss below, the historic increase in the legal purchase age to 21 at PA is unlikely due to a high level of state smoking prevalence, but it may not be the case on the later shift back to MCPA 18. Therefore policy endogeneity could be a concern if both changes in MCPA at PA are used to examine the impact of maternal smoking

the law induced cutoff.

I use a restricted version of PA Natality data to find that there is a significant jump in average prenatal cigarette consumption among mothers who have legal access to cigarette just before or after the end of first trimester, with a flexible piecewise polynomial specification. The identification assumption on the conception age cutoff is consistent with epidemiological findings on the maternal smoking cessation pattern and the substantial infant health improvement due to smoking cessation or reduction by the end of the first trimester. I find there is a 16 percent decrease in the average cigarettes smoked per day for mothers subject to the age restriction. The reduced form regressions show that enforcing MCPA 21 can result in a large improvement in a variety of newborn health measures, in particular, a 20 percent decrease in the number of low birth weight babies. Combining estimates from previous two steps, I find more cigarette consumption per day during pregnancy increases the incidence of low birth weight infant, reduce gestation and decrease APGAR scores. For smoking mothers, consuming 1 more cigarette per day during pregnancy would reduce infant birth weight by 61.17 g, increase the probability of having a low birth weight infant by 2.8 percentage points and decrease the APGAR 1 minute score by 0.13 points. Interestingly, my findings are salient if the sample is restricted to women with conception ages at least one week older or younger than 21 by the end of first trimester because in this case there is a sharper difference in mothers' incentive to curb smoking, or if I focus on the period when the law was more seriously implemented at PA. Finally, I also find that these adverse impacts concentrate on women who are non Hispanic white, or have low educational attainments.

The remainder of the paper proceeds as follows. The next section reviews the epidemiological literature on maternal smoking cessation, previous studies on the impact of prenatal smoking on infant health, and the institutional background of the minimum cigarette purchase age at PA and the rest of United States. Section 3 presents the empirical strategy. Section 4 describes the restricted Pennsylvania Natality live birth data used for this study. Section 5 reports findings and conducts a variety of robustness checks. Section 6 concludes.

2 Background

2.1 Maternal Smoking during Pregnancy

Numerous studies have addressed maternal smoking and its consequences, while the epidemiological literature has provided two strands of evidence on prenatal smoking cessation which are often neglected in economics studies but closely related to this paper. First, the epidemiological studies listed in table 3 consistently show that the reduction in prenatal smoking mostly occurs

during the first trimester of pregnancy[7]. This pattern is robust across different decades although there is a lasting decline in the number of smoking mothers. Second, these studies link timing of fetal exposure to smoking with various birth outcomes[8].

The studies in the first three rows of table 3 report optimistic findings that the negative impact of prenatal smoking on birth weights would be nullified if mothers stop smoking by as late as 32^{nd} week in pregnancy. However, the mothers in Rush and Cassano (1983) [56] exposed to an important information shock on the adverse effect of prenatal smoking. With the overall low cessation rate among them, it is possible that quitters more aware of this shock had some unobserved characteristics beneficial for infant health. If this selection was more prevalent among earlier quitters, the estimated impact of smoking up to the second trimester can be biased toward zero. Lieberman et al(1994) [39] only focuses on the term (37 or more weeks of gestation, the mid of the third trimester) singleton births. If prenatal smoking shortens gestation and thus birth weights, they dropped from the sample mothers who gave preterm births because of smoking in the first and second trimester[9]. This would result in attenuation of the estimate on early smoking cessation. A salient problem in Lindley et al (2000) [41] is that they use a convenience sample of women whose health care providers chose to ask about smoking at the 32^{nd} week visit. If a mother's unobserved ability for earning is positively correlated good health counseling which regularly encourages smoking cessation, their estimates on smoking termination by the second trimester will be again biased toward zero.

Butler et al (1972) [13], Macarthur and Knox (1988) [42], and McCowan et al (1992) [43] provide more conservative evidences that smoking cessation should take place by 15^{th} or 16^{th} week to significantly reduce the negative impact of maternal smoking. The finding by Butler et al (1972) [13] is compelling since smoking behavior of the mother cohort in this study is much less subject to selection (Fertig, 2009) [29]. Besides, Macarthur and Knox [42] indicate that smoking cessation after the 16^{th} week only mediates the negative impact of maternal smoking.

In contrast, recent studies show that the end of first trimester is actually the critical deadline for smoking cessation or reduction if pregnant women want to avoid a substantial damage on infant health due to smoking. McDonald et al (1992) [45] use a dataset of 40445 pregnant women, the largest in published works to address this topic.They find there is no significant

[7]Also see Pickett et al(2003) [49].

[8]The standard datasets used by economists do not code maternal smoking across three trimesters until 2002 when the National Survey on Drug Use and Health (NSDUH) started to do so. The Natality data began collecting this information in 2003. None of them covers the period when MCPA 21 was effective at PA. Other datasets such as National Longitudinal Survey of Youth (NLSY), Pregnancy Risk Assessment Monitoring System (PRAMS), National Health Interview Survey (NHIS) and Behavioral Risk Factor Surveillance System (BRFSS) either only records a mother's smoking status in pregnancy, or only asks her smoking behavior at the first and late half of the pregnancy.

[9]Indeed, their sample contains an unusual high portion of mothers who started to smoke in the second and third trimester.

difference in excess risk of low birth weight between mothers who quit smoking by the end of the first trimester and non-smokers. But continuation of smoking in the second trimester leads to a sharp increase in excess risk. However, this risk increase can be substantially mediated among continuing smokers through reduction in cigarette consumption, especially for mothers who smoke 1 pack (20 cigarettes) per day in the first trimester. Wisborg et al (2001) [63] and Wang et al (2002) [61] reach the same conclusions as to other birth outcomes such as birth weight, gestation, stillbirths and infant mortality. Yan (2009) [64] uses a much larger panel data set of mothers with multiple births between 2003 and 2006, based on birth records from Pennsylvania and Washington. This paper applies both ordinary least square (OLS) and mother fixed effect (FE) approaches to find cessation within the first trimester nullifies the adverse impact on birth outcomes, but mothers who continue smoking up to the second trimester will be much more likely to have poor health infants even they quit during the second trimester. Besides, there is suggestive evidence that both smoking cessation and reduction by the end of the first trimester are important to lessen the overall negative impact of prenatal smoking.

Finally, it should be noted that the fluctuation of maternal smoking can be correlated with other substance uses. Alcohol use is the most prominent one. Yet except for heavy drinking, the impact of low to medium level drinking on birth outcomes is not well established (IOM,1990 [35]). McDonald et al (1992) [45] find that consumption of alcohol is below 6 drinks per week during pregnant even leads to a small but significant reduction in the risks of low birth weight, low birth weight for gestation and preterm birth.

2.2 Maternal Smoking and Birth Outcomes

There are in general three approaches to identify the causal relation between maternal smoking and birth outcomes. The first is using observed covariates to correct for selection into prenatal smoking. This strategy is arguably plausible when researchers can use datasets with rich sets of controls which simultaneously determine maternal smoking and infant health. The existing evidences show that in addition to standard controls, including measures on psychosocial stress (Brooke et al,1989 [12]), drug use (Noman et al,2007 [46]), or other typically unobserved variables as well as non-standard covariates (Reichman et al,2008 [50]) do not appreciably affect the typical estimated impact of prenatal smoking on infant birth weight (-200 to -250 g). Almond et al (2005) [6] further shows there is little association between maternal smoking and the fraction of infants with birth weights less than 1500 g. As to heterogeneous effects across different subpopulations, Walker et al (2009) [60] applies both ordinary least square (OLS) and matching estimation to find that the detrimental impact of prenatal smoking is smaller if a mother is black or teenager. However, the Natality data used in these two recent studies do not contain some important covariates such as income, employment status, or any substance use other than

smoking and drinking. This poses a threat on the identification assumption of selection on observables.

Using mother fixed effects model is the second approach to handle unobserved heterogeneity in maternal smoking. The seminal work by Rosenzweig and Wolpin (1991) [53] justifies this estimation strategy in a model of intra family resource allocation where the mother specific health endowment both directly affects infant health and determines prenatal inputs. Their within mother estimates, based on a panel of 3384 births from NLSY1979, indicate that smoking above (below) 1 pack/day is associated with a reduction in birth weight by 160 g (83 g), but smoking has an insignificant impact on weeks of gestation. Besides, the estimate on the correlation between maternal health endowment and prenatal smoking is negative, suggesting an exacerbation of endowed health inequality and an upward biased OLS estimate(in the absolute value). Rosenzweig and Wolpin (1995) [54] evaluate five alternative statistical formations in estimating an infant health production technology, with focus on maternal age effects. They find the estimated impact of parental smoking is very close to Rosenzweig and Wolpin (1991) [53] when the preferred mother fixed effect with instrumental variable approach (FE-IV) is applied to capture the biological effects of maternal age at birth.

Recent studies tend to utilize much larger datasets. The matched panel in Abrevaya (2006) [1], based on the publicly released Natality data, includes more than 80000 mothers of multiple births. His estimate on the impact of prenatal smoking varies from -100 to -150 g. Abrevaya and Dahl (2008) [2] use a more precisely matched sample of 101268 mothers to find the quantile estimates on mother smoking are significant across all but the lowest 10% quantile. However, two pronounced problems on the FE model raised by Abrevaya (2006) [1] so far have not been well addressed in the literature. First, a mother's smoking behavior can be responsive to earlier birth outcomes. To handle this feedback effect, researchers can apply FE-IV directly on prenatal smoking[10] among mothers who have three births. No study on prenatal smoking has formally used it. Second, the change in prenatal smoking across births can be correlated with change in other behaviors during pregnancy. When some of these behaviors are unobservable, the direction of bias on the FE estimates is ambiguous.

The third approach is to use instrumental variables. Rosenzweig and Schultz (1983) [52] use input prices for household production, income and parental education as instruments to identify a health technology, under a strong assumption that family specific health endowment only affects maternal behavior in pregnancy yet doesn't correlate with children's endowment[11].

[10]This is the same idea as Rosenzweig and Wolpin (1995) [54] which uses FE-IV approach to control for the feedback effect on childbearing ages. A recent paper by Royer (2004) [55] extends this framework such that maternal age can have varying effects across different births.

[11]Therefore parental characteristics or family background variables which affect prenatal behaviors are not correlated with unobserved children's health endowment, and so can be used as instruments for prenatal inputs.

Their estimates imply the birth weight for infants of smokers would be 230 g less than nonsmokers. Permutt and Hebel (1989) [48] use a randomized late-term smoking cessation intervention conducted by Sexton and Hebel (1984) [58] to be the instrument for prenatal smoking. The estimated adverse impact of -430 g is large. Hamilton (2001) [33] applies a Bayesian finite mixture model on the same dataset and finds the treatment effect is -348 g for all compliers, -430 g for compliers who smoked fewer than 20 cigarettes per day prior to pregnancy[12]. However this clinical trial sample of Sexton and Hebel (1984) [58] is small, and highly selective because the experiment enrollees were restricted to mothers who smoked at least 10 cigarettes per day before pregnancy.

Researchers also seek for plausible state level experiments to instrument for prenatal smoking. The pioneering work of Evans and Ringel (1999) [28] explores whether a higher state cigarette tax would reduce maternal smoking and improve birth outcomes. Their dataset comes from 1989 to 1992 Natality detail files with 10.5 million singleton births. The two stage least square (2SLS) estimates indicate that smoking during pregnancy lowers birth weight by 353 to 594 g, increases the incidence of low birth weight by 10 to 16 percentage points, but has little effect on the incidence of very low birth weight. Lien and Evans (2005) [40] use large tax hikes that occurred in four states to identify the consequences of maternal smoking on infant health, with a sample of 1991 to 1997 Natality detail files. Their 2SLS estimates range from -94.62 to -2765.17 g on birth weight and 3 to 40 percentage increase on low birth weight, for different treatment states. Pooling all the treatment states gives a decline of 181.86 g on birth weight and a 6.8 percentage increase on low birth weight. Using cigarette taxes as the instrument are subject to three problems. First, changes in state taxes can be correlated with other time variant changes in tobacco control policies. Failure to control for all of them would bias upward the response of prenatal smoking on the tax change and bias downward the 2SLS estimates. Second, there can be a reverse causality that the state government increases cigarette taxes as a reaction to smoking prevalence, or the decrease in smoking prevalence and increase in state cigarette tax is actually driven by unobserved antismoking sentiments (DeCicca et al, 2007 [23]). Third, some states use the tax revenue to improve unobserved health behaviors or knowledge of the pregnant women[13].

[12]He argues that mothers who smoke heavily may compensate for smoking cessation with other unobserved unhealthy behaviors. Yet in this study, pregnant mothers were young and more likely to treat cigarette smoking as a complement to other substance uses. Dee (1999) [24] provides the evidence on the complementarity of smoking and drinking among young people.

[13]Lien and Evans (2005) [40] shows that the 1992 cigarette tax increase at Massachusetts provides funding to educational programs for pregnant women.

2.3 Minimum Cigarette Purchase Age

As an important youth tobacco control intervention, the minimum cigarette purchase age law prohibits selling cigarette to young people under a specific age which varies by states. Table 1 indicates that 43 states had set up the legal purchase ages by year 1988, mostly at 18. The law has been established in every state over the next two decades when some states adjust their legal cigarette purchase ages multiple times. In 2007, every state set MCPA as 18 except Alabama, Alaska, New Jersey and Utah which had legal ages of 19. Pennsylvania is the only state which had placed cigarette purchase restriction to young people up to age 21 between year 1992 and 2002[14].

The existing literature on MCPA focuses on gauging its implementation across different states. DiFranza et al (1987) [26] shows the law was not strictly enforced at Massachusetts in 1985. However a recent paper by DiFranza and Dussault (2005) [25] reports that the states were forced to more seriously enforce the law starting from the mid 1990s for two reasons. First, the Congress required a reduction in block grant funding to non-compliance states since 1994. Second, the direct federal regulations had been enacted by the Food and Drug Administration, although this FDA's enforcement was terminated on Mar 2000. According to this study, Pennsylvania has kept good effort in complying with the law, with a 50 percent decline in the average law violation rate from year 1996 to 2000. Surprisingly, only two previous studies have directly examined the impacts of MCPA on the short run youth smoking or long term adult health. Chaloupka and Grossman (1996) [19] find the cigarette purchase age regulation did not have a significant impact on youth cigarette smoking, using the Monitoring the Future Project data of year 1992 to 1994. Nevertheless during that period states faced little pressure from the federal government to curb cigarette sales to the youth. In contrast, Ahmad and Billimek (2007) [4] find if shifting the legal purchase age from 18 to 21 can delay the age specific smoking initiation rate by 3 years, it would lead to not only a decline in adult (more than 18 years) smoking prevalence comparable to a large 40% tax-induced price increase[15]but also an increased accumulation of quality adjusted life years comparable to what would be observed following a 20% tax-induced price across a 75-year period. Therefore the existing meager evidences on the health benefits implied by MCPA law are quite mixed. More important, so far no study ever tries to uncover the MCPA induced intergenerational health benefit, that is, whether enforcing this law can improve birth outcomes through curbing smoking among young pregnant mothers.

The SLATI indicates PA enforced MCPA 21 between 1992 and 2002, without reporting the exact starting or ending date on the law enforcement. Correspondence with PA public health

[14]The data on MCPA are based on "State Legislated Actions on Tobacco Issues"(SLATI), 1988 to 2007. First published in 1988, it is the only up-to-date and comprehensive summary of state tobacco control laws.

[15]The literature suggests that the pass through from cigarette exercise tax to the retail price is almost cent for cent (Chaloupka and Warner, 2000 [18]).

officials provides me with suggestive clues on the initiation of legal purchase age 21. Back in June 1990, a discrepancy was found in enforcing legal purchase age of 18. While the amended Section 6305 of Pennsylvania Crime Code, Title 18, prohibited tobacco sales to anyone under age 18 years, Section 6306(a) prohibited any cigarette or cigarette paper to any "minor" which was however not defined within that section. Pursuant to the Statutory Construction Act, a "minor" is defined as individual under the age of 21 not 18. Since the early 1992, PA public health administration had launched a series of campaigns to promote awareness of MCPA 21. The public were unprepared for these events. The subsequent newspaper reports deepened the public's impression on this law. They can be dated back to as early as June 1, 1992. On that day the Patriot News stated, "if you are under 21 (yes, it's 21, not 18 as most people believe) it's against the law to smoke[16]." As proponents of this new law, health officials and anti-smoking groups kept on boosting the law enforcement[17]and even proposing more stringent legislative bills to set age 21 as the legal age for sales of other tobacco products, over the subsequent decade. Yet these attempts were often thwarted by tobacco companies' efforts to ensure youth access[18].The law of MCPA 21 was repealed by PA legislation with the minimum legal purchase age reduced to 18 on July 10, 2002[19]. This shift was accompanied by a series of more stringent regulations to restrict the youth's access to cigarettes[20]. Therefore the PA state government appeared to seek alternative tobacco control policies among young people such that mothers below age 20 who were not exposed to higher cigarette purchase age faced other restrictions in getting cigarettes.

Besides, there was little change in other PA tobacco control policies over the period when MCPA 21 was effective. No change occurred in terms of the PA total Alciati score, a measure on the extensiveness of state tobacco youth access laws. The levels of smoke free air protection at 12 different types[21]of sites were also invariant, except that the protection level at public school increased from 1 to 3 in year 2001.

3 Empirical Strategy

This paper examines whether and how much MCPA 21 has affected maternal smoking and infant health, and then uses the law-induced variation in maternal smoking to address the impact of

[16]Phil Galewitz,"Youngsters get burned by lax cigarette laws", Patriot News, June 1, 1992, Page C8.

[17]Godshall,"Tougher law would reduce teen smoking", Valley News Dispatch, Mar 20, 1994, Page A9.

[18]Phil Galewitz,"Cigarettes illegally sold to teenagers", Patriot News, June, 1993, Page B1.

[19]Pennsylvania Legislative Reference Bureau

[20]The annual data on youth tobacco access laws and smoke-free air laws are available at the ImpacTeen State Level Tobacco Control Policy and Prevalence Database. It also codes the existence of any regulation on minors' possession, use and purchase of tobacco products, which are however ambiguous because this dataset provides no clear definition on "minors" for different years.

[21]They include government worksites, private worksites, child care centers, health care facilities, restaurants, recreational facilities, cultural facilities, public transit, shopping malls, public schools, private schools and free standing bars.

prenatal smoking on infant health. My first step is to estimate the following baseline first stage equation:

$$S_{ij} = \alpha_0 + \alpha_1 A_{ij} + \alpha_2 A_{ij}^2 + \alpha_3 Over21_{ij} + \alpha_4 Over21_{ij} \times A_{ij} + \alpha_5 Over21_{ij} \times A_{ij}^2 + t_j + csize_i + \epsilon_{ij} \quad (1)$$

where S_{ij} is one measure of cigarette consumption during pregnancy for mother i[22]who conceived in year j. The variable A is the normalized mother age on the day of conception, defined as the time distance between the last day of mother's first trimester (90 days after mother's conception day, see figure 1-1) and her 21 years birthday. It is measured by *days* in the regression analysis. For ease of graphical presentation, I convert it into a measure in *years*. For instance, while A is equal to 0 unit of *day* at the cutoff, it always graphically appears to be *20.75* units of *years*. Below I call A as *age at conception* or *conception age* interchangeably when it doesn't cause any confusion. $Over21$ is an indictor function which equals 1 if A is nonnegative. In addition, t is the year of mother's conception fixed effect, and the variable *csize* is the size of mother's residential county[23] fixed effect, and ϵ is a random error. Later, I also augment this model by adding different combination of numerous controls including infant sex, live birth order, parental characteristics, fertility history, as well as maternal health status and health behavior. To summarize, my specification in equation (1) allows a flexible piecewise quadratic polynomial in age at conception fully interacted with an indictor on whether a pregnant woman is older than 21 by the last day of first trimester.

One challenge of this research is that it is not quite as straightforward to establish the conception age cutoff for maternal smoking as other RD studies based on age laws[24] of school entry (Berlinski et al , 2008 [9]; McCrary and Royer, 2006 [44]), school leaving (Oreopoulos, 2006 [47]) and alcohol use (Carpenter and Christopher, 2008 [14] and 2009 [15]). In equation (1) I assume the cutoff should be placed such that it splits the sample of pregnant women into two groups which differed in exposure to MCPA at the first trimester. As shown in figure 1-1, the younger group to the left of cutoff was prohibited from buying cigarette during the entire first trimester. In contrast the older one to the right had legal access to cigarette during at least part of the first trimester. The parameter of α_3 in equation (1) then captures how pregnant women near to the cutoff changed their smoking behavior over the pregnancy due to a sudden legal exposure to cigarette at the first trimester[25]. An immediate concern is that there could

[22]As explained in the next section, my sample only consists of mothers giving singleton births.

[23]I divide residential counties at PA into three categories according to county population: above 1 million; below 1 million and above 0.1 million; and below 0.1 million (base).

[24]Lee and Lemieux (2009) [38] provides a comprehensive discussion on the application of discontinuities in age.

[25]Intuitively, both groups of mothers were randomly assigned around the cutoff due to MCPA 21. They were identical in all the characteristics except prenatal smoking behaviors and therefore birth outcomes. The treatment group of younger mothers was exogenously more likely to curb smoking and improve birth outcomes than the control group of older mothers. The magnitude of the difference between the two groups, which was the impact

be more than one cutoff. For instance, women who conceived within 180 days prior to their 21 year old birthdays can legally purchase cigarettes in the first or second trimester, but younger mothers to the left of this potential cutoff can not until the third trimester. One might wonder if these two groups of mothers were also remarkably different in prenatal cigarette consumption. However, next I justify that only the case of A being 0 (or, the age at conception is 20.75 as in figure 3-1) is the only plausible MCPA induced cutoff for prenatal smoking.

The underlying story behind the present empirical approach can be captured in a simple model[26] where mothers make smoking decisions based on a tradeoff between dependence on cigarettes during pregnancy and their infant health. Only mothers whose costs from curbing smoking exceed their perceived infant health gains are affected by the instrument MCPA 21. As the previous review of epidemiological literature suggests, increasing cigarette consumption or smoking initiation just before or after the end of the first trimester can make a fundamental difference on birth outcomes. Henceforth it is reasonable to assume only at that period [27] mothers can perceive the largest fraction of smoking cost which suddenly accrues to their infants. Then they compare it with the potential gains from continuous smoking and finally choose the optimal amount of average cigarette consumption in pregnancy. Under this decision making mechanism, the positively selected mothers who are affected by the instrument would stop cessation effort or start to smoke within the first trimester[28] in the absence of MCPA 21, without severely harming their babies. Later this offers an explanation on why my IV estimation uncovers a large negative effect. Overall the predicted result for equation (1) is that mothers with conception age just above 20.75 would on average have higher level of current and successive cigarette consumption throughout pregnancy than those just below the cutoff. Besides, despite strong evidences to support this cutoff only, I will experiment with other potential cutoffs in section 5 and find their impacts are small as well as statistically insignificant.

There are two noticeable advantages of using the MCPA 21 induced cutoff to instrument for prenatal smoking. First, this age regulation of cigarette purchase was stable over a decade. Therefore my new instrument is no longer subject to policy endogeneity characterized by a third factor driving the change in both the purchase age regulation and maternal smoking. Second, this approach is not confounded by changes in other tobacco control policies, one important concern on using state cigarette taxes as instruments. As discussed above, there was little change in other state level tobacco control policies between 1992 and 2002 at PA. The only time

of this regulation, turned out to be large as shown in the next section.

[26]In Rosenzweig and Wolpin(1991 [53],1995 [54]), mothers choose the optimal amount of cigarettes smoked in pregnancy solely as an input for infant health production. Here a mother's addiction to cigarettes has been added into the model, since smoking at different trimesters directly enters her utility function.

[27]Most pregnant women receive smoking cessation counseling during routine prenatal care in the first trimester. For a recent paper on this topic, see Evans and Lien(2005) [27].

[28]Because of addiction, they are also less likely to reduce cigarette smoked or quit smoking in the second or third trimesters.

variant increase on the smoking free air protection at public schools can not affect or generate the discrete change in maternal smoking at the MCPA 21 induced cutoff. Similarly for other county level tobacco control policies if they also changed somewhat over the 10 years.

My second step is to estimate the reduced form model:

$$BO_{ij} = \beta_0 + \beta_1 A_{ij} + \beta_2 A_{ij}^2 + \beta_3 Over21_{ij} + \beta_4 Over21_{ij} \times A_{ij} + \beta_5 Over21_{ij} \times A_{ij}^2 + t_j + csize_i + \nu_{ij} \quad (2)$$

where BO_{ij} is one of the eight birth outcomes for mother i who conceived in year j. By estimating equation 1 and 2, I can recover γ_1 the causal parameter which describes the impact of smoking on birth outcomes in the structural equation (3) below by forming the ratio of β_3 (the estimated discontinuity in infant outcomes) to α_3 (the estimated increase in prenatal smoking at the cutoff).

$$BO_{ij} = \gamma_0 + \gamma_1 S_{ij} + t_j + csize_i + \upsilon_{ij} \quad (3)$$

as before I will check the sensitivity of the estimate γ_1 by adding into equation (1) and (2) a number of controls. In short, the estimation strategy amounts to a two stage least square with semi parametric specification[29]where I use an indictor of $Over21$ to instrument for prenatal smoking. Finally, the same empirical framework can be applied to evaluate the MCPA 18 at PA before 1992 or after 2002, or at other 35 states since 1989. However, because Walker et al (2009) [60] finds that the adverse impact of maternal smoking on birth weight is smaller for teen mothers than adult mothers, there is a narrower scope of strictly enforcing the purchase age 18 than 21[30].

4 Data

I use a restricted version of 1992 to 2002 Pennsylvania Natality birth data, a 100 percent sample of annual certificates on all live births. It was provided by the Pennsylvania's Bureau of Health Statistics. A unique feature of this dataset is that it contains the exact birth dates for all mothers and their infants which are not available in the publicly released dataset. I use this information and weeks of gestation to compute the normalized mother age on the day of conception which

[29]Although the nonparametric estimation proposed by Hahn, Todd and Van Der Klaauw [31] could be an alternative method, this paper focuses on the semi parametric approach.

[30]Another potential problem in studying birth outcomes of teenage mothers with the global polynomial specification is that a mother's age profile on infant weight may not be quite a smooth function around the cutoff of age 18. The medical literature suggests that the theoretical ages at which girls cease to grow in stature lies in somewhere between age 17 to 19. Alex and Davila (1972) [5] finds that the median age for girls to cease growth in stature is 17.3 years. Adolescent mothers below the growth termination age tend to have lower birth weight infants because they and their fetus compete for nutrients (Scholl et al, 1994 [57]) while those above the threshold age are no longer subject to this competition. Therefore it is possible for late teenager mothers, the relation between maternal age and infant birth weight is not continuous at some interval to either side of the MCPA 18 induced cutoff.

is nonnegative if a mother's 21 year birthday lies within the first trimester[31]. This calculation is done in two steps. First, because gestation is the time distance between the date characterized by 14 days before the conception day and the date when the baby is born, I use gestation and the exact infant birth dates to work out the date when each mother conceives. Second, as discussed before, I use the date of mother's conception day and the her exact date of birth to calculate the normalized mother age on the day of conception(figure 1-1).

I place several restrictions in constructing the final sample. First, mothers who were born outside U.S.[32]or resided in states other than PA are excluded. Second, the sample only retains mothers whose ages at conception were within a 10 month interval to both sides of the cutoff, such that every mother would be old enough to have legal access to cigarette by the end of her pregnancy[33]. Third, for the primary analysis I restrict the sample in a conservative way to mothers who became pregnant between Oct 1, 1992 and July 10, 2001. This is because diffusion of the new information on MPCA 21 among the public is assumed to take four months, and backing one year from the law ending day can guarantee all the pregnant mothers[34] in the sample were under the regime of MCPA 21, before it was repealed[35]. Below the robustness check shows the results are not sensitive to the specific law starting day of Oct 1, 1992. Fourth, following the literature I also exclude mothers who had non-singleton births and gave more than one birth when their conception ages are restricted to the interval of about 20 to 21.5 years.

Table 2 presents that summary statistics for all variables. The restricted Natality data have two measures on each mother's smoking behavior, whether she smoked during pregnancy and if yes how many cigarettes smoked per day. The valid response rate exceeds 98 percent for both questions. About 27 percent of the young women were smokers who on average consumed 10.4 cigarettes per day. Smoking mothers were more likely to be single, white and had lower educational attainments.

There is a rich set of birth outcome variables in the dataset. The infant health measures used in this paper consist of infant birth weight, indictors of birth weight less than 2500 g (low birth weight), 2000 g or 1500 g (very low birth weight), gestation, indictor of premature birth(or preterm birth, weeks of gestation less than 37 weeks), APGAR 1 minute and 5 minute scores. Birth weight has been the primary measure of newborns in almost all the studies on infant health. Low birth weight is a costly outcome both in the short and long term. It is caused by

[31]The duration of pregnancy is usually divided into three trimesters. According to a standard definition (Wikipedia), the first trimester is from the last menstrual period to the 13^{th} week, the second from the 14^{th} to 26^{th} week, and the third trimester from the 27^{th} week to the 44^{th} week.

[32]Mother birth places are restricted to 50 states,DC,Puerto Rico, Virgin Islands and Guam.

[33]The average gestational age in the sample is 39 weeks.

[34]They gave births no later than July 10, 2002.

[35]This restriction essentially avoids the cases that some young mothers who had no legal access to cigarette under MCPA 21 were suddenly able to sometime during pregnancy due to the law shifting back to MCPA 18 in 2002.

two factors: intrauterine growth retardation and premature birth. Maternal smoking is found to be an important modifiable contributor for the former factor, while the evidence of its impact on the latter is mixed because the factors for premature births are not well understood (Kramer, 1987 [37]). However, premature birth is an important health measure because premature infants are at greater risk for impediments in growth and mental development. And it is responsible for the majority of neonatal deaths and nearly one half of all cases of congenital neurological disability. The APGAR score is an critical indictor by which doctors will determine whether a newborn requires immediate medical care. It is calculated by evaluating a newborn by five tests on a scale ranging from 0 to 2, then summing up the scores of five tests to reach the final value. These tests are usually made at 1 and 5 minutes after birth. In my sample, infants of smoking women on average have lower birth weights and have greater risks of being low birth weight than those born to the general population. The differences are not pronounced in the fraction of very low birth weight, weeks of gestation or APGAR scores.

The covariates in table 2 include birth characteristics, parental socioeconomic background, mother's fertility history and maternal health status. The dataset has separately coded mother's Hispanic origin and race. Because infant health is known to differ substantially between Hispanic and non-Hispanic parents, I construct three non-overlapped indicators to differentiate the race/ethnicity of mothers (fathers): non-Hispanic white, non-Hispanic black, and Hispanic. I also construct an indictor on father's age missing and later verify whether the sudden decline of birth weight around the cutoff is actually driven by unplanned pregnancies[36] rather than prenatal smoking. The final sample contains 60710 young adult pregnant women, 16262 of whom reported smoking during pregnancy.

5 Results and Robustness Checks

5.1 Prenatal Smoking

I start by reporting the results from the first stage regression. Figure 3-1 visually shows the relation between age at conception and average cigarette consumption during pregnancy. To reduce the data noise, I take the average of smoking measures over weekly cells of conception age. Over these means there is a superimposed fitted line from a piecewise quadratic regression on the underlying micro data[37]. In this figure, there is a noticeable discrete increase in average cigarette consumption at the cutoff.

[36] Whether father's age is missing has been used as a good proxy for unintended pregnancies (Watson and Fertig, 2009 [62]).

[37] It should be noted whether the regression is based on average smoking measures over weekly cells of conception age or daily cells (weighted by the number of observations within each cell), the estimates and their statistical significance at the cutoff are virtually the same.

Column (1) in table 4 indicates that the estimate of this jump in cigarette consumption is equal to 0.453 and statistically significant at 1%. It amounts to about a 16 percent increase for mothers just one week below the cutoff, or a 17 percent increase at the sample mean. Alternatively, mothers subject to the law would reduce overall cigarette consumption by about 16 to 17 percent. Adding controls of birth and basic parental characteristics, parental socioeconomic variables, mother fertility history and health status only shrinks the estimate by a small magnitude but does not change its significance, as indicated in column (2), (3) and (4). It is worth mentioning that maternal health variables include an indicator of alcohol drinking and a continuous measure of drinks per week. The little difference of the estimates between column (3) and (4) is reassuring, because this implies even if there were any sudden increase in alcohol assumption at the cutoff due to the minimum legal drinking age (MLDA) of 21, it could not be the driving force for the discrete increase in cigarette smoking, given that the two substance uses are complements for young mothers. In table 6 I further show that there is actually no evidence of any discrete jump in alcohol consumption at the cutoff[38].

The change of 0.453 cigarettes per day can be attributed to changes at the extensive or intensive margin, or both around the cutoff. I plot the conception age profiles for smoking participation during pregnancy in figure 3-2. There is some visual evidence of an increase in the smoking participation, although the size is small. Few pregnant nonsmoking mothers at the cutoff start to smoke due to a sudden legal exposure to cigarette. Column (5) in table 4 shows that the corresponding estimate is about 0.02 (on a base of 27 percentage points) and only marginally significant. The estimate becomes smaller and insignificant when all the covariates are included as in column (8). In contrast, figure 3-3 uncovers a large jump in average cigarette consumption for smokers at the cutoff. Column (9) of table 4 indicates that the estimated impact is 0.98 cigarettes per day, about a 9.1 percent increase for mothers just one week below the cutoff, or a 9.4 percent increase at the sample mean. Adding a number of controls which correlate with smoking intensity only makes the estimate smaller by at best 9 percent in the magnitude, as in column(10)-(12). These estimates are always highly significant.

Table 5 presents a number of robustness checks for the results in table 4. In column (1) across all three panels, I apply the specification of cubic piecewise polynomials in conception age. The estimated impact is similar in magnitude and significant at 5% for average cigarettes smoked per day. The one for smoking participation is slightly larger and still marginally significant. The only exception is that the estimated increase for smokers' average cigarette consumption

[38]This finding differs from Carpenter and Christopher (2009) [14] which uncovers discrete increases of alcohol drinking induced by MLDA 21 among the young female at California. One possible reason for this difference is that alcohol consumption in the Natality data is severely underreported, therefore shrinking the discrete change. The underreported drinking measures can also be systematically correlated with some unobservable. However if the gap in drinking does not shrink close to zero and the correlated unobservable is orthogonal to the cutoff, then a statistically significant discrete jump in alcohol consumption should be detected when it does exist.

becomes insignificant. However, I also conduct three Wald tests for the joint significance on the cubic terms, none of which yields a p-value sufficiently small. Since the quadratic specification is actually the preferred model, I use it for all the remaining robustness checks. In column (2) and (3), I assume that the starting date for MCPA 21 to be June 1, 1992 and Jan 1, 1992, respectively. The estimates and their significances across three panels are very close to table 4, except that the increase on smoking participation is imprecisely estimated. Therefore my estimates are not sensitive to any specific initial date of the law enforcement.

In the fourth robustness check, I drop one week of observations to each side of the cutoff. The retained mothers above the cutoff had legal access to cigarettes for at least one week in the first trimester. They benefited more from smoking initiation or resistance to cessation earlier in pregnancy than those mothers who were dropped but closer to the original cutoff. By the same argument, mothers below the cutoff in the remaining sample had stronger incentives to curb smoking compared to those who were also dropped yet nearer to the original cutoff[39]. I therefore expect the estimated impacts should be at least as large as in table 4. This check also helps addressing concerns on the potential errors in calculating conception ages around the cutoff[40]. Mothers who could have been mistakenly placed above or under the original cutoff within a week's window were completely removed from the new sample. If the law did work, the discrete increase in the cutoff needs to be at least as significant as before. As expected, the estimates and their significances across three panels are again significant and somewhat lager than table 4. The impact on smoking participation is insignificant but similar in magnitude as before. In column (5) I restrict the sample period to 1994-1999 when there was a continuous federal initiative for the purchase age law implementation at the state level. The corresponding estimates are consistent with this story. Although both samples shrink by about one third, all the estimated impacts for three smoking measures are not only highly significant but also larger in magnitude than those in table 4.

I next consider one more potential cutoff which locally differentiates women by whether legally exposed to cigarette before the end of second trimester, as in column (6). The modified specification with two cutoffs is in the same spirit as Van de Klaauw (2002) [59]. The estimates for the original cutoff in panel 1 and 2 are statistically significant and somewhat larger than those in table 4, while the impact on smoking at the intensive margin is insignificant. The additional cutoff(cutoff2 in table 5) is never precisely estimated in any panel. I then experiment another potential cutoff (cutoff3 in table 5)which differentiates pregnant women by whether

[39]Note that in the original sample, the difference in the gains from cigarette consumption was small, but the cost difference on infant health was quite large for mothers around the original cutoff. As to mothers in the new sample, the cost difference remained as large as before but the difference in the benefits of prenatal cigarette consumption was further enlarged for mothers around the new cutoff.

[40]A small portion of gestational ages are imputed. Besides, each mother's gestation reported in the Natality dataset is the integer part of the actual gestation.

legally exposed to cigarette before the middle of third trimester in column (7). All the three estimates based on the original cutoff from panel 1 to 3 are significant and similar in size as table 4, with the additional cutoff never precisely estimated in any case. Column (8) combines all three cutoffs in one specification. It shows that the estimates for all the smoking measures at the original cutoff are still robust, while none of the new cutoffs is precisely estimated. Finally, I carry on two placebo experiments in column (9) and (10). First the full sample is divided into two subsamples by the original cutoff. Then for each subsample, I pick up its median to be the new cutoff and apply the same piecewise quadratic polynomial specification to test for any jump at the new cutoff. None of the six estimates in the final two columns turns out to be statistically significant.

I conduct another robustness check on the continuity of the running variable's density, as suggested by Imbens and Lemieux(2008) [34]. It is possible that some mothers rely heavily on smoking to relieve stress during pregnancy, such that they delay conception to guarantee easier and legal access to cigarette as early as in the first trimester. This manipulation on the running variable, if exists, poses a threat to the exclusion assumption on the cutoff. Figure 1-2 presents the case for all mothers where I group them by age at conception in weekly cells. It doesn't exhibit strong evidence of non-random clustering of maternal age at conception to either side of the cutoff. Similarly for smoking mothers, as shown in figure 1-3.

Next I check the smoothness of all the covariates around the cutoff. Table 6 gives estimates from regressions similar to equation (1) with the dependent variables being birth characteristics, maternal and paternal socioeconomic status, maternal fertility history, or maternal health status and health behavior. The tests are conducted for both the sample of all mothers and the one of smoking mothers. The 40 estimates[41] in table 6 shows for each covariate there is no evidence of significant change just at the cutoff. I also graph the conception age profiles of all the covariates in figure 12.1 to 32.2. They exhibit either no or small jumps at the cutoffs.

5.2 Infant Health

Table 7 reports the reduced form estimates from equation (2). The first row of column (1) indicates that having legal access to cigarette just before the end of the first trimester would lower an infant's birth weight by 16.67 g. Adding more controls, this estimate shrinks to 11.37 g although still insignificant. The corresponding impact for smoking mothers is highly significantly at about -60 g no matter which sets of covariates are included, as in columns (5) to (8). Since the previous estimate on smoking participation at the cutoff is insignificant and small, this adverse

[41]The missing values on father's age are replaced with the sample mean when I uses it as a covariate in the regression analyses of smoking or birth outcomes. Here whether this imputation on father's age is done or not, the estimated discrete increase on this variable at the cutoff is always statistically insignificant.

impact due to more cigarettes smoked at the intensive margin is the primary driving force for the law induced effect among all mothers. Figure 4.1 and 4.2 plot the age at conception profiles of birth weights grouped in weekly cells for all mothers and smoking mothers. The discontinuity in birth weight for smokers is noticeable.

The second row presents the estimated discrete change in the low birth weight indictor. It suggests that for all mothers, there is a significant decrease of 1.5 percentage points or a 20 percent reduction (a 23 percent decrease at the sample mean) in the incidence of low birth weight babies among those subject to the regulation. The estimate is very robust to inclusion of more covariates. Columns (5) to (8) reveal that the discontinuity in low birth weight for smoking mothers due to earlier exposure to cigarette is about 2.7 percentage points across all the specifications. This is equal to about a 31 percent increase for mothers just one week below the cutoff, or a 30 percent increase at the sample mean. Figure 5-1 and 5-2 present these two salient discontinuities[42]. In contrast, the third and fourth rows in table 7 indicate that the estimated discontinuity is insignificant for infant birth weight less than 2000 g or 1500 g. This is also visually shown in the figures 6.1 to 7.2.

The next four rows in table 7 report the discontinuities for four other infant health measures. Gestation would be significantly reduced by about 0.09 to 0.1 weeks for all mothers just above the cutoff, and by 0.19 to 0.2 weeks for smoking mothers. The probability of preterm birth would increase by about 1.3 percentage points for all mothers yet the same impact is insignificant for smoking mothers. Including more controls make these estimates less precisely estimated. The corresponding small jumps at the cutoffs are presented in figure 8.1 to 9.2. Finally, the estimated discrete changes for APGAR 1 minute and 5 minute scores for the all mothers above the cutoff are about -0.09 and -0.04 points respectively while the impacts are -0.13 and -0.07 points for smokers. All these four estimates are significant and robust to adding various covariates. Figure 10.1 to 11.2 illustrate the four remarkable discontinuities in the corresponding conception age profiles.

5.3 Effects of Prenatal Smoking on Infant Health

Table 8 presents the 2SLS estimates based on three different smoking measures. Column (1) shows that the impact of smoking 1 more cigarette per day among all mothers on birth weight is insignificantly at -37 g. However, its effect on low birth weight indicator is significant at 3.7 percentage points, robust to various covariates. The impacts of smoking in pregnancy are imprecisely estimated whichever measure on infant health is used, as in column (5) to (8). This is not surprising since the first stage estimated impact on smoking participation is only

[42]The jumps in the graphs can occur one week before or late at the cutoff which might be due to the measurement problem in gestation as discussed above.

marginally significant. In contrast, the first row in Column (9) reports that for smoking mothers, 1 more cigarette per day significantly reduce infant birth weight by 61.17 g. It also increases the probability of having a low birth weight infant by 2.8 percentage point, a 33 percent increase for mothers just one week below the cutoff, or a 31 percent increase at the sample mean. Because the influence of MCPA 21 on smoking concentrates on the intensive margin and smokers are more likely to have lower birth weight infants, the adverse impacts of prenatal smoking induced by the law mainly work at the lower end of the birth weight distribution. As to other infant health measures, prenatal smoking has an insignificant impact on the fraction of infants less than 2000 or 1500 g among all mothers or smokers. Column (1) also indicates that the gestation would decrease by 0.22 weeks due to 1 more cigarette per day among all mothers, yet it becomes insignificant when more controls are added[43].The impact of the law induced smoking on premature birth is also imprecisely estimated. In the final two rows, I use the estimates for all mothers with no additional controls and find a one standard deviation increase in average cigarettes per day will reduce APGAR 1 minute score by 1.11 points, or a 0.85 standard deviation around the sample average. The same amount of increase in cigarette consumption results in a decrease in the APGAR 5 minute score by 0.47 points, or 0.64 of one standard deviation. As to smokers, 1 more cigarette per day due to the law is associated with an increase of 0.13 points in APGAR 1 minute score. Adding all the covariates makes these estimates slightly larger yet still statistically significant (Column 10 to 12).

Overall my estimated impacts of prenatal smoking on infant health are quite large especially for smokers. One concern is that there may be discrete changes in other substance uses driven by MLDA 21 or other regulations around the cutoff, which can overstate the impact of smoking on birth outcomes and bias the 2SLS estimate upward. I can not test the discrete change for each substance use because my dataset only codes prenatal smoking and drinking. However, as I find no evidence of a significant sudden jump in drinking (most likely due to MLDA 21), the resulting bias from alcohol use may be small[44]. Second, my estimate might reflect a total effect since it can affect birth outcomes indirectly through other unobservable channels. I find among smoking mothers there is a 20 percent increase (at the cutoff) in the number of frequent smokers[45] who smoke more than 10 cigarettes per day. If they account for most of the compliers, and are more heavily engaged in other unobservable risky or unhealthy behaviors determined by smoking, the estimated total effect of smoking should be large. Third, Brachet(2005) [11] shows

[43]As mentioned above, whether prenatal smoking shortens gestation is not well understood in the medical literature. My finding here suggests that the law induced smoking impact on gestation may be small. However, it is possible that the determinants for gestation highly correlate with smoking, leading to a discrete change in the previous reduced form estimation on gestation.

[44]Even the discrete change in drinking does exists, it is unlikely to largely bias the estimate on the impact of smoking given that some smokers are light to moderate alcohol users and more alcohol drinking imply no further detrimental impact on birth outcomes

[45]They include both moderate (10-20 cigarretes/day) and heavy smokers (more than 20 cigarretes/day).

that misclassification of endogenous smoking status can lead to attenuation in the first stage estimate on the response of smoking to cigarette taxes, and inflate the second stage estimate on the adverse impact of smoking on infant health. By the same argument, smoking status misreporting or underreporting in the amount of cigarette smoked around the cutoff can bias upward my 2SLS estimates. Fourth, my 2SLS estimates capture the adverse smoking impacts for a group of positively selected mothers. They would stop smoking cessation or reduction without doing much harm to their babies without the MCPA intervention, implying a large local average treatment effect. Fifth, however, the potential bias of my IV estimate can also be downward. Adda and Cornaglia (2006) [3] argue that the impact of excise taxes on the number of cigarette smoked is overstated if smokers compensate for a price increase by extracting more nicotine per cigarette. If young pregnant women just above the cutoff tend to suddenly reduce smoking intensity[46] for each cigarette while smoking more cigarettes, then the effect of MCPA 21 in reducing average cigarette consumption will be overestimated under the assumption that the extracted amount of cotinine per cigarette is constant. This in turn downward biases the two stage estimate.

Table 9 assesses the robustness of the findings in table 8. Since the impact of smoking participation during pregnancy is not precisely estimated for any birth outcome, I focus on the other two smoking measures. Using a cubic specification reduces the precision of all estimates as in column (1). But the specification tests are unable to reject that the coefficients of all the cubic terms are equal to 0. In column (2) and (3), I try different starting dates on enforcing MCPA 21. The results are virtually the same as table 8. In column (4), I drop one week of observations at each side of the cutoff. For the sample of all mothers, the estimated impacts on the probabilities of low birth weight, APGAR 1 minute and 5 minute scores are very close to table 8. For the sample of smokers, I also find similar impact estimates on birth weight and low birth weight as before. Next I restrict the sample period to year 1994-1999 when the law was presumably more seriously implemented. Column (5) shows that the new results are mostly consistent with table 8. I conduct two placebo experiments in the final two columns, by splitting the original sample into two and using the median of each sample to be the new local instrument. As expected, the estimated impacts are always insignificant and sometimes wrong signed.

5.4 Effects of Prenatal Smoking on Infant Health: Subgroups

In this subsection I stratify the sample by race and years of school to explore the heterogeneous effects across different subpopulation. Table 10 focuses on non-Hispanic white and non-Hispanic

[46] Adda and Cornaglia (2006) [3] find that the relation between cigarettes per day and cotinine is linear up to 10 cigarettes a day, close to the average just below the cutoff (10.77) or the mean (10.42) of the smoker sample.

black mothers. The specifications in the odd number columns include no additional covariates, while the even number columns include all the covariates (same for table 11). Column (1) and (2) indicate that 1 more cigarette per day will significantly increase the incidence of low birth weight by 2.5 to 2.8 percentage points, reduce APGAR 1 minute score by 0.19 to 0.22 points and APGAR 5 minute score by 0.07 to 0.08 points. For smoking mothers, it will on average reduce infant birth weight by 61.37 to 68.63 g, as well as decrease APGAR 1 minute score by 0.14 to 0.16 points, as in column (3) and (4). In contrast, the estimates in column (5) to (8) are ambiguous and imprecise for non-Hispanic black mothers.

Table 11 presents the results separately for mothers with no more than twelve years of schooling and those who have been to the college. For low educated mothers, the first two columns indicate that 1 more cigarette per day will significantly increase the probability of low birth weight by 4.2 to 4.3 percentage points, decrease APGAR 1 minute score by 0.18 to 0.2 points and APGAR 5 minute score by 0.07 to 0.08 points. For smokers among the low educated mothers, it will on average reduce infant birth weight by 60.65 to 62.45 g, and increase the probability of low birth weight by 2.8 to 2.9 percentage points as shown in column (3) and (4). The impact of the law induced prenatal smoking is not precisely estimated for higher educated mothers, as in column (5) to (8). To summarize, the adverse impacts of prenatal smoking around the cutoff in the general mother population or smoking mothers are largely due to the negative effects that occur among low educated, non Hispanic white mothers.

6 Conclusion

This paper provides the first evaluation on whether and how much MCPA 21 at PA have reduced prenatal smoking and improved infant health. I find young pregnant mothers just below the MCPA 21 induced cutoff would consume 0.453 less cigarettes per day than those just above the cutoff. The estimated decrease is 9.4 percent at the intensive margin, while for the smoking participation the increase is small and marginally significant. These estimates are robust to inclusion of various covariates, alternative specification, different initial law enforcement dates and multiple cutoffs, etc. The reduced form regressions indicate a 20 percent decrease in low birth weight for mothers subject to the age restriction. Besides, there is suggestive evidence that this law can increase gestation and reduce the probability of preterm birth. For smoking mothers, the improvements in birth weight and low birth weight are an increase of 60 g and a reduction of 2.7 percentage points, respectively. The corresponding benefits of MCPA 21 on APGAR 1 minute and 5 minute are increases of 0.127 and 0.073 points. I also use the exogenous variation of maternal cigarette consumption driven by the law to provide new evidences on the impact of prenatal smoking on infant health. The regression discontinuity estimates indicate that

consuming 1 more cigarette per day during pregnancy worsens a variety of birth outcomes among all mothers. For smokers, it reduces infant birth weight by 61.17 g, increase the probability of having a low birth weight infant by 2.8 percentage points and decrease the APGAR 1 minute score by 0.13 points.

Taken together, these new findings show that enforcing MCPA 21 can substantially curb prenatal smoking among young women and enhance their birth outcomes. They also highlight the intergenerational benefit of keeping a healthy fetal environment (Currie, 2009 [21]). Third, this study suggests that even the law induced smoking reduction can lead to a noticeable saving in the short run health care cost. To show this, I use both the cross sectional and fixed effect estimates on the impact of birth weight on hospital costs in Almond et al (2005) [6], together with my estimate which indicates for smokers 1 more cigarette per day during pregnancy reduces birth weight by 67 g (the estimate with all controls added). I also assume that adverse effect on the incidences of having babies lighter than 2500 g, 2000 g and 1500 g[47]are the same. Then I calculate that a smoking mother would avoid an extra hospital cost varying from \$30 to \$114 (in \$2000) if due to the MCPA 21 she had consumed 1 less cigarette per day during pregnancy[48]. Fourth, I also use the birth weight estimates by Behrman and Rosenzweig (2004) [8] and my new estimate on smoking reduction to calculate the longer term MCPA 21 induced benefit[49]. Given that a smoking mother consumes 1 less cigarette per day due to MCPA 21 and her baby is a female, then the resulting lighter baby will later on have more schooling of 0.6 months, be about 0.1 inches taller and have higher earnings by 1 percent. Finally, my paper opens new paths for future research. For instance, it is interesting to apply the RD approach to earlier years when MCPA 21 was not established to construct an additional counterfactual. Or, instead of the current RD approach, researchers can use a difference in difference strategy among mothers of different age groups, based on the historical shifts in the cigarette purchase age at PA[50].

[47]There is a rapid nonlinear increase in the hospital cost for newborns across different (decreasing) birth weight segments at the lower end of the overall distribution, as shown in Almond et al(2005) [6]. Joyce(1999) [36] reports a similar pattern in the cost saving for newborn delivery at the higher end of hospital discharges.

[48]This cost saving is not small, compared to the total hospital cost saving due to smoking cessation that varies from \$53 to \$907 in Almond et al (2005) [6].

[49]Because both Almond et al (2005) [6], and Behrman and Rosenzweig (2004) [8] use samples of twins with the same gestations, their birth weight effect estimates are entirely based on augmenting the fetal growth rate. This study did not find any significant impact of prenatal smoking on gestation especially conditioning on smoking mothers, it is reasonable to assume the -67 g per cigarette effect stems from the retarded fetal growth rate rather than the shortened gestation. Therefore my new estimates is directly applicable with theirs.

[50]This is similar to Watson and Fertig (2005) [62] which addresses whether a lower drinking age leads to more prenatal drinking and worse birth outcomes. But notice there were many more state level experiments in changing drinking ages than smoking ages, and the second shift in MCPA at PA may be subject to policy endogeneity.

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Table 1: State Minimum Cigarette Purchase Age (1989-2007)

Year	Number of States by State Minimum Cigarette Purchase Age					
	16	17	18	19	21	None
1988	5	4	31	3	0	8
1989	2	4	35	3	0	6
1990	2	4	36	3	0	5
1991	1	3	40	3	0	3
1992	0	2	42	3	1	2
1993	0	0	46	3	1	0
1994	0	0	46	3	1	0
1995	0	0	46	3	1	0
1996	0	0	46	3	1	0
1997	0	0	46	3	1	0
1998	0	0	46	3	1	0
1999	0	0	46	3	1	0
2000	0	0	46	3	1	0
2001	0	0	46	3	1	0
2002	0	0	46	3	1	0
2003	0	0	47	3	0	0
2004	0	0	47	3	0	0
2005	0	0	47	3	0	0
2006	0	0	46	4	0	0
2007	0	0	46	4	0	0

Source: State Legislated Actions on Tobacco Issues.

Table 2: Descriptive Statistics

Variables	All Mothers		Mothers(Smokers)	
	Mean	Std.dev	Mean	Std.dev
Maternal Smoking				
Smoker	0.268	0.443	1	0
Cigarettes per day	2.792	5.832	10.423	6.888
Birth Outcomes				
Birth Weight (grams)	3302.224	559.141	3173.197	549.912
Birth Weight< 2500grams	0.064	0.245	0.089	0.285
Birth Weight< 2000grams	0.021	0.142	0.026	0.16
Birth Weight< 1500grams	0.01	0.097	0.011	0.105
Gestation	39.071	2.046	38.961	2.137
Premature Birth	0.074	0.251	0.083	0.276
1 minute APGAR score	8.056	1.306	8.079	1.303
5 minute APGAR score	8.987	0.728	9	0.732
Birth Characteristics				
Infant Male	0.512	0.5	0.514	0.5
Live Birth Order	1.617	0.811	1.773	0.878
Maternal Characteristics				
Mothers Age	20.982	0.62	20.978	0.618
Mother Non-Hispanic White	0.753	0.431	0.847	0.36
Mother Non-Hispanic Black	0.179	0.384	0.108	0.31
Mother Hispanic Origin	0.066	0.249	0.041	0.199
Mother Years of Education= 12	0.567	0.495	0.537	0.499
Mother Years of Education> 12	0.246	0.43	0.126	0.332
Mother Married	0.39	0.488	0.289	0.453
Maternal Fertility Characteristics				
Number of Terminations	0.317	0.66	0.382	0.727
Father Age Missing(unwanted birth)	0.008	0.086	0.011	0.104
Previous Infant Preterm of Small	0.018	0.132	0.026	0.158
Maternal Health Status and Health Behaviors				
Drinker	0.008	0.086	0.019	0.136
Drinks per week	0.018	0.417	0.04	0.399
Weight Gain (pounds)	31.255	13.776	30.449	14.288
Paternal Characteristics				
Fathers Age	24.156	4.089	24.55	4.438
Father Non-Hispanic White	0.702	0.457	0.779	0.415
Father Non-Hispanic Black	0.218	0.413	0.157	0.364
Father Hispanic Origin	0.078	0.268	0.059	0.235
Father Years of Education= 12	0.567	0.495	0.592	0.492
Father Years of Education> 12	0.207	0.405	0.126	0.332
Number of observations	60710		16262	

Note: The data come from PA annual birth files (singleton births), with mothers who conceived between Oct 1, 1992 and July 10, 2001.

Table 3: Mother Smoking Cessation and Birth Outcomes (Epidemiological Literature)

Research	Timing of Smoking Cessation	Findings on Birth Outcomes
Rush and Cassano (1983)	Mother smoking rate (1): 46.90% Cessation by the 24^{th} week (2): 8.23% Cessation between 24^{th} and 32^{nd} week: 1.9% Smoking throughout three trimesters (3): 89.23%	No significant difference in the average birth weights between mothers who quit smoking by the 32^{th} week and non-smokers.
Lieberman et al (1994)	Mother smoking rate: 30.12% Cessation by the end of the 1^{st} trimester: 16.39% Cessation by the end of the 2^{nd} trimester: 3.80% Smoking throughout three trimesters: 72.74%	No significant difference in the risk of giving birth to undersized infants between mothers who quit smoking by the end of the second trimester in pregnancy and non-smoking mothers.
Lindley et al (2000)	Mother smoking rate: 35.45% Cessation by the 32^{nd} week: 17.57%	No significant difference in the average birth weights between mothers who quit smoking by the 32^{nd} week and non-smoking mother.
Macarthur and Knox(1988)	Mother smoking rate: 28.45% Cessation by the 16^{th} week:16.28% Smoking throughout three trimesters: 74.82%	No significant difference in average birth weights between mothers who quit smoking by the end of the 16^{th} week in pregnancy and non-smoking mothers. Smoking cessation after the 16^{th} week only mediates the negative impact of maternal smoking on infant birth weight.
Butler et al (1972)	Mother smoking rate: 36.49% Cessation by the 16^{th} week:20.49%	No significant difference in infant mortality rate and average birth weights between mothers who quit smoking by the end of the 16^{th} week in pregnancy and non-smoking mothers.
McCowan et al(2009)	Mother smoking rate: 20.45% Cessation by the 1^{st} trimester:48.05% Cessation between 12^{th} and 15^{th} week: 2.9%	No significant difference in the rate of spontaneous preterm birth and undersized infants between mothers who quit smoking by the 15^{th} week and non-smoking mothers.
McDonald et al(1992)	Mother smoking rate: 35.50%	No significant difference in excess risk of low birth weight between mothers who quit smoking by the end of the first trimester and nonsmoking mothers. Continuation of smoking in the second trimester leads to significant excess risk of low birth weight.
Kristen et al(2001)	Mother smoking rate: 40.45% Cessation by the 1^{st} trimester: 26.11%	No significant difference in the risks of stillbirths and infant mortality between mothers who quit smoking by the end of first trimester and non-smoking mothers.
Wang et al (2002)	Mother smoking rate: 24.37% Cessation by the 1^{st} trimester: 28.74% Smoking throughout three trimesters: 71.26%	No significant difference in average birth weight, the risk of low birth weight and gestation between mothers who quit smoking by end of the first trimester and nonsmoking mothers.

Note: (1) Ratio of the number of smoking mothers to the number of all mothers. (2) Ratio of the number of mothers who quit smoking to the number of smoking mothers. (3) Ratio of the number of mothers who smoke over three trimesters to the number of smoking mothers.

Table 4: Mother's Age at Conception and Smoking during Pregnancy

	Dependent Variable											
	Cigarettes Per day				Smoker				Cigarettes Per Day (Smokers)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Over21	0.453	0.463	0.402	0.387	0.018	0.019	0.014	0.013	0.983	0.933	0.876	0.901
	(0.141)***	(0.137)***	(0.134)***	(0.132)***	(0.011)*	(0.011)*	(0.010)	(0.010)	(0.334)***	(0.316)***	(0.315)***	(0.310)***
Year of Conception?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Size of Residence County?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Birth Characteristics?	No	Yes	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Basic Parental Controls?	No	Yes	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Parent Socioeconomic Status?	No	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes
Fertility History?	No	No	No	Yes	No	No	No	Yes	No	No	No	Yes
Mother Health Status?	No	No	No	Yes	No	No	No	Yes	No	No	No	Yes
Num of Observations	60710	60710	60710	60710	60710	60710	60710	60710	16262	16262	16262	16262

Note: Each specification includes a quadratic in age at conception fully interacted with a dummy of being over age 21. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%. Birth characteristics include infant male and live birth order. The basic parental controls include mother's race/ethnicity, father's race/ethnicity, father's age and father's age missing. The parental socioeconomic controls include mother's education equal to 12 years, mother's education more than 12 years, mother married, father's education equal to 12 years, father's education more than 12 years. Variables on mother fertility history consist of number of terminations, previous infant preterm of small. Mother health status variables include mother alcohol use, drinks per week and weight gain during pregnancy.

Table 5: Mother's Age at Conception and Smoking during Pregnancy (Robustness Checks)

	Dependent Variable of Panel 1: Cigarettes per Day									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Over21	0.398	0.439	0.437	0.460	0.538	0.513	0.483	0.513	-0.183	-0.254
	(0.193)**	(0.137)***	(0.138)***	(0.132)***	(0.163)***	(0.252)**	(0.173)***	(0.252)**	(0.185)	(0.182)
Cutoff2	No	No	No	No	No	0.209	No	0.095	No	No
						(0.203)		(0.223)		
Cutoff3	No	No	No	No	No	No	0.040	0.184	No	No
							(0.206)	(0.229)		
Piecewise Polynomial	Cubic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic
Num of Observations	60710	63486	64627	59411	40905	60710	60710	60710	31283	29315
	Dependent Variable of Panel 2: Smoker									
Over21	0.024	0.016	0.016	0.017	0.026	0.034	0.025	0.034	-0.016	-0.011
	(0.015)*	(0.011)	(0.011)	(0.011)	(0.013)**	(0.018)*	(0.013)*	(0.018)*	(0.015)	(0.015)
Cutoff2	No	No	No	No	No	0.018	No	0.021	No	No
						(0.015)		(0.017)		
Cutoff3	No	No	No	No	No	No	-0.001	0.008	No	No
							(0.014)	(0.016)		
Piecewise Polynomial	Cubic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic
Num of Observations	60710	63486	64627	59411	40905	60710	60710	60710	31283	29315
	Dependent Variable of Panel 3: Cigarettes per Day(Smokers)									
Over21	0.569	1.029	1.023	1.101	1.018	0.643	0.845	0.643	-0.123	-0.520
	(0.441)	(0.324)***	(0.320)***	(0.323)***	(0.387)***	(0.531)	(0.400)**	(0.531)	(0.413)	(0.448)
Cutoff2	No	No	No	No	No	0.058	No	-0.452	No	No
						(0.527)		(0.612)		
Cutoff3	No	No	No	No	No	No	0.150	0.347	No	No
							(0.531)	(0.632)		
Piecewise Polynomial	Cubic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic
Num of Observations	16262	16985	17276	15896	10860	16262	16262	16262	8366	7868

Note: Each specification includes year of conception and size of residence county fixed effects. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%.

Table 6: Age Profiles of Potential Confounders

Dependent Variables	"Over 21" Coefficient Estimate	
	All Mothers	Mothers(Smokers)
Infant Male	-0.011	-0.004
	(0.011)	(0.023)
Live Birth Order	0.021	0.055
	(0.020)	(0.041)
Mother Non-Hispanic White	-0.014	-0.003
	(0.010)	(0.016)
Mother Non-Hispanic Black	0.009	0.010
	(0.009)	(0.014)
Mother Hispanic Origin	0.005	-0.006
	(0.007)	(0.010)
Mother Years of Education=12	-0.019	-0.023
	(0.012)	(0.021)
Mother Years of Education> 12	-0.004	-0.007
	(0.011)	(0.016)
Mother Married	-0.016	-0.015
	(0.012)	(0.021)
Father's Age	0.003	0.021
	(0.105)	(0.199)
Father Non-Hispanic White	-0.006	0.017
	(0.011)	(0.019)
Father Non-Hispanic Black	0.008	-0.002
	(0.011)	(0.017)
Father Hispanic Origin	-0.002	-0.015
	(0.007)	(0.012)
Father Years of Education= 12	-0.017	-0.032
	(0.012)	(0.024)
Father Years of Education> 12	-0.001	-0.011
	(0.011)	(0.016)
Number of Terminations	0.015	-0.036
	(0.015)	(0.035)
Father Age Missing(unwanted birth)	-0.002	-0.001
	(0.002)	(0.006)
Previous Infant Preterm of Small	0.004	0.01
	(0.003)	(0.007)
Drinker	0.003	0.002
	(0.002)	(0.006)
Drinks per week	-0.007	-0.008
	(0.014)	(0.031)
Weight Gain(pounds)	0.038	0.195
	(0.359)	(0.677)
Num of Observations	60710	16262

Note: Each specification includes a quadratic in age at conception fully interacted with a dummy of being over age 21,together with year of conception and size of residence county fixed effects. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%.

Table 7: Reduced Form Estimates: Mother's Age at Conception and Birth Outcomes

Dependent Variables	"Over 21" Coefficient Estimate							
	All Mothers				Mothers(Smokers)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Birth Weight	-16.669	-13.785	-11.723	-11.387	-60.097	-60.157	-58.349	-60.422
	(12.787)	(12.714)	(12.893)	(12.161)	(27.556)**	(27.507)**	(27.592)**	(25.809)**
Birth Weight< 2500g	0.015	0.015	0.015	0.014	0.027	0.028	0.027	0.027
	(0.006)***	(0.006)**	(0.006)**	(0.006)**	(0.012)**	(0.012)**	(0.013)**	(0.012)**
Birth Weight< 2000g	0.006	0.006	0.006	0.006	0.008	0.008	0.008	0.008
	(0.004)*	(0.004)	(0.004)	(0.004)	(0.008)	(0.008)	(0.008)	(0.008)
Birth Weight< 1500g	0.002	0.002	0.002	0.002	0.004	0.004	0.004	0.004
	(0.002)	(0.002)	(0.003)	(0.002)	(0.005)	(0.005)	(0.005)	(0.005)
Gestation	-0.101	-0.096	-0.095	-0.092	-0.195	-0.189	-0.188	-0.191
	(0.049)**	(0.049)**	(0.049)*	(0.049)*	(0.114)*	(0.115)	(0.115)	(0.112)*
Premature Birth	0.013	0.013	0.013	0.012	0.005	0.005	0.005	0.004
	(0.007)**	(0.007)*	(0.007)*	(0.007)*	(0.013)	(0.013)	(0.013)	(0.013)
1-Min APGAR Score	-0.087	-0.088	-0.087	-0.086	-0.127	-0.133	-0.131	-0.130
	(0.029)***	(0.029)***	(0.029)***	(0.029)***	(0.066)*	(0.066)**	(0.066)**	(0.066)**
5-Min APGAR Score	-0.036	-0.036	-0.036	-0.036	-0.073	-0.076	-0.074	-0.075
	(0.017)**	(0.017)**	(0.017)**	(0.017)**	(0.038)*	(0.038)**	(0.038)**	(0.038)**
Num of Observations	60710	60710	60710	60710	16262	16262	16262	16262

Note: Specifications (1) and (5) only include a quadratic in age at conception fully interacted with a dummy of being over age 21, together with year of conception and size of residence county fixed effects. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%. Specifications (2) and (6) further add infant male, live birth order, and the basic parental controls which include mother's race/ethnicity, father's race/ethnicity, father's age and father's age missing. Specification (3) and (7) expand model (2) and (6) by adding parent socioeconomic controls such as mother's education equal to 12 years, mother's education more than 12 years, mother married, father's education equal to 12 years, father's education more than 12 years. Specifications (4) and (8) have additional controls on maternal fertility history (number of terminations, previous infant preterm of small) and those on maternal health status (mother alcohol use in pregnancy, drinks per week and weight gain during pregnancy).

Table 8: Mother Smoking and Birth Outcomes(2SLS Estimates)

Dependent Variable	Smoking Measures in Pregnancy											
	Cigarettes per Day				Smoker				Cigarettes per Day (Smokers)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Birth Weight	-36.758	-29.776	-29.128	-29.408	-910.107	-724.812	-821.920	-906.878	-61.165	-64.465	-66.585	-67.042
	(30.687)	(28.819)	(33.278)	(33.291)	(886.296)	(780.611)	(1065.98)	(1212.536)	(34.111)*	(35.643)*	(38.248)*	(37.170)*
Birth Weight< 2500g	0.034	0.033	0.036	0.037	0.845	0.792	1.022	1.133	0.028	0.030	0.031	0.030
	(0.017)**	(0.016)**	(0.019)*	(0.019)*	(0.602)	(0.544)	(0.829)	(1.007)	(0.015)*	(0.016)*	(0.017)*	(0.017)*
Birth Weight< 2000g	0.013	0.013	0.015	0.015	0.333	0.320	0.417	0.462	0.008	0.008	0.009	0.009
	(0.009)	(0.009)	(0.010)	(0.011)	(0.288)	(0.270)	(0.395)	(0.472)	(0.008)	(0.009)	(0.009)	(0.009)
Birth Weight< 1500g	0.004	0.004	0.004	0.004	0.093	0.089	0.115	0.128	0.004	0.004	0.004	0.004
	(0.006)	(0.006)	(0.006)	(0.007)	(0.154)	(0.148)	(0.203)	(0.232)	(0.005)	(0.006)	(0.006)	(0.006)
Gestation	-0.223	-0.208	-0.237	-0.237	-5.519	-5.053	-6.674	-7.312	-0.198	-0.203	-0.215	-0.212
	(0.131)*	(0.123)	(0.145)	(0.150)	(4.312)	(3.877)	(5.866)	(6.962)	(0.133)	(0.139)	(0.151)	(0.146)
Premature Birth	0.029	0.028	0.031	0.031	0.712	0.674	0.884	0.964	0.005	0.005	0.005	0.005
	(0.018)	(0.017)*	(0.020)	(0.020)	(0.561)	(0.516)	(0.777)	(0.924)	(0.013)	(0.014)	(0.015)	(0.014)
1-Min APGAR Score	-0.191	-0.190	-0.217	-0.222	-4.739	-4.626	-6.112	-6.843	-0.129	-0.142	-0.150	-0.145
	(0.087)**	(0.083)**	(0.100)**	(0.105)**	(3.210)	(2.986)	(4.740)	(5.860)	(0.078)*	(0.083)*	(0.090)*	(0.086)*
5-Min APGAR Score	-0.080	-0.078	-0.090	-0.092	-1.976	-1.900	-2.531	-2.838	-0.075	-0.081	-0.085	-0.083
	(0.043)*	(0.042)*	(0.050)*	(0.052)*	(1.409)	(1.325)	(2.068)	(2.540)	(0.046)	(0.049)*	(0.053)	(0.051)
Num of Observations	60710	60710	60710	60710	60710	60710	60710	60710	16262	16262	16262	16262

Note: Specifications (1), (5) and (9) only include a quadratic in age at conception fully interacted with a dummy of being over age 21, together with year of conception and size of residence county fixed effects. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%. Specifications (2), (6) and (10) further add infant male, live birth order, and the basic parental characteristics which include mother's race/ethnicity, father's race/ethnicity, father's age and father's age missing. Specification (3), (7) and (11) expand model (2),(6) and (10) by adding parental socioeconomic controls which consists of mother's education equal to 12 years, mother's education more than 12 years, mother married, father's education equal to 12 years, father's education more than 12 years. Specifications (4), (8) and (12) have additional controls on maternal fertility history (number of terminations, previous infant preterm of small) and those on maternal health status (mother alcohol use in pregnancy, drinks per week and weight gain during pregnancy).

Table 9: Mother Smoking and Birth Outcomes(2SLS Estimates, Robustness Checks)

Dependent Variable	Smoking Measure in Pregnancy of Panel 1: Cigarettes per Day						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Birth Weight	-67.164	-39.183	-42.276	-15.389	-57.568	-28.747	-8.038
	(56.411)	(31.483)	(31.197)	(27.463)	(35.375)	(99.707)	(68.932)
Birth Weight< 2500g	0.037	0.035	0.036	0.031	0.039	0.021	0.036
	(0.028)	(0.017)**	(0.018)**	(0.016)*	(0.018)**	(0.046)	(0.045)
Birth Weight< 2000g	0.018	0.013	0.013	0.015	0.019	-0.029	0.009
	(0.016)	(0.009)	(0.009)	(0.010)	(0.010)*	(0.039)	(0.022)
Birth Weight< 1500g	0.000	0.003	0.003	0.005	0.010	-0.048	0.001
	(0.009)	(0.006)	(0.006)	(0.006)	(0.006)	(0.053)	(0.011)
Gestation	-0.185	-0.234	-0.237	-0.198	-0.270	0.414	-0.152
	(0.194)	(0.131)*	(0.131)*	(0.125)	(0.141)*	(0.626)	(0.291)
Premature Birth	0.043	0.034	0.035	0.024	0.029	0.021	0.036
	(0.032)	(0.018)*	(0.019)*	(0.016)	(0.019)	(0.055)	(0.048)
1-Min APGAR Score	-0.248	-0.189	-0.195	-0.170	-0.183	-0.040	0.044
	(0.144)*	(0.088)**	(0.090)**	(0.089)*	(0.085)**	(0.283)	(0.166)
5-Min APGAR Score	-0.111	-0.077	-0.075	-0.076	-0.091	-0.101	-0.042
	(0.071)	(0.044)*	(0.043)*	(0.043)*	(0.044)**	(0.196)	(0.104)
Piecewise Polynomial	Cubic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic
Num of Observations	60710	63486	64627	59411	40905	31283	29315
Dependent Variable	Smoking Measure in Pregnancy of Panel 2: Cigarettes per Day(Smokers)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Birth Weight	-91.967	-60.138	-58.284	-51.008	-68.605	244.435	-14.751
	(95.535)	(31.775)*	(31.172)*	(28.396)*	(43.230)	(929.982)	(68.791)
Birth Weight< 2500g	0.046	0.025	0.026	0.024	0.036	0.019	0.034
	(0.043)	(0.014)*	(0.014)*	(0.013)*	(0.021)*	(0.143)	(0.045)
Birth Weight< 2000g	0.026	0.008	0.008	0.010	0.010	-0.090	0.017
	(0.026)	(0.007)	(0.007)	(0.008)	(0.010)	(0.322)	(0.027)
Birth Weight< 1500g	0.001	0.004	0.004	0.004	0.009	-0.096	0.005
	(0.012)	(0.005)	(0.005)	(0.005)	(0.007)	(0.329)	(0.014)
Gestation	-0.393	-0.193	-0.187	-0.187	-0.249	1.518	-0.135
	(0.406)	(0.122)	(0.120)	(0.122)	(0.167)	(5.333)	(0.265)
Premature Birth	0.015	0.008	0.009	0.007	0.010	0.006	0.035
	(0.033)	(0.012)	(0.012)	(0.012)	(0.016)	(0.135)	(0.047)
1-Min APGAR Score	-0.254	-0.120	-0.121	-0.108	-0.084	-0.401	-0.103
	(0.233)	(0.071)*	(0.072)	(0.074)	(0.075)	(1.563)	(0.199)
5-Min APGAR Score	-0.235	-0.064	-0.064	-0.058	-0.052	-0.134	-0.040
	(0.200)	(0.042)	(0.041)	(0.042)	(0.046)	(0.686)	(0.098)
Piecewise Polynomial	Cubic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic	Quadratic
Num of Observations	16262	16985	17276	15896	10860	8336	7868

Note: Each specification includes a quadratic in age at conception fully interacted with a dummy of being over age 21,together with year of conception and size of residence county fixed effects. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%.

Table 10: Mother Smoking and Birth Outcomes(2SLS Estimates, By Race)

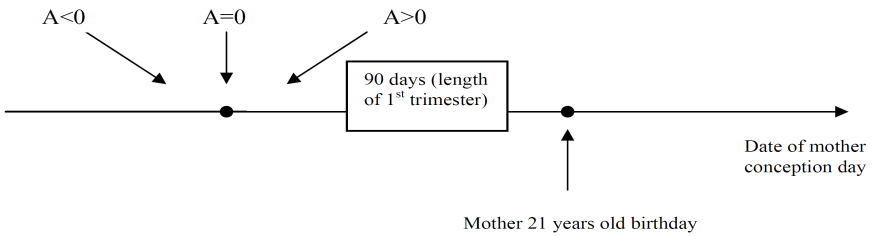
Dependent Variable	Smoking Measure (Non Hisp White Mothers)				Smoking Measure (Non Hisp Black Mothers)			
	Cigarettes per Day		Cigarettes per Day(Smokers)		Cigarettes per Day		Cigarettes per Day(Smokers)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Birth Weight	-40.200	-39.640	-61.374	-68.257	121.375	116.211	14.287	-3.695
	(26.270)	(30.142)	(35.678)*	(40.955)*	(174.811)	(203.452)	(76.812)	(69.900)
Birth Weight< 2500g	0.025	0.028	0.025	0.028	0.023	0.032	0.012	0.012
	(0.013)*	(0.015)*	(0.016)	(0.018)	(0.078)	(0.097)	(0.043)	(0.042)
Birth Weight< 2000g	0.011	0.013	0.008	0.009	0.010	0.016	0.011	0.013
	(0.008)	(0.009)	(0.009)	(0.010)	(0.043)	(0.054)	(0.027)	(0.027)
Birth Weight< 1500g	0.004	0.004	0.005	0.005	-0.008	-0.004	-0.009	-0.003
	(0.004)	(0.005)	(0.006)	(0.007)	(0.031)	(0.037)	(0.016)	(0.013)
Gestation	-0.166	-0.188	-0.239	-0.266	-0.448	-0.580	0.227	0.209
	(0.100)*	(0.120)	(0.144)*	(0.167)	(0.679)	(0.881)	(0.394)	(0.377)
Premature Birth	0.013	0.015	0.010	0.010	0.122	0.149	-0.029	-0.029
	(0.013)	(0.015)	(0.014)	(0.016)	(0.118)	(0.163)	(0.046)	(0.046)
1-Min APGAR Score	-0.187	-0.223	-0.142	-0.156	0.177	0.180	0.018	-0.047
	(0.078)**	(0.099)**	(0.083)*	(0.094)*	(0.366)	(0.431)	(0.192)	(0.190)
5-Min APGAR Score	-0.070	-0.084	-0.075	-0.084	0.014	-0.010	-0.044	-0.072
	(0.039)*	(0.049)*	(0.050)	(0.057)	(0.193)	(0.232)	(0.106)	(0.113)
Num of Observations	45717	45717	13769	13769	10740	10740	1749	1749

Note: Each specification includes a quadratic in age at conception fully interacted with a dummy of being over age 21,together with year of conception and size of residence county fixed effects. Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%.

Table 11: Mother Smoking and Birth Outcomes(2SLS Estimates, By Education)

Dependent Variable	Smoking Measure (Mother Schooling≤ 12)				Smoking Measure (Mother Schooling> 12)			
	Cigarettes per Day		Cigarettes per Day(Smokers)		Cigarettes per Day		Cigarettes per Day(Smokers)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Birth Weight	-42.651	-34.511	-60.647	-62.447	55.478	78.197	-87.000	-143.816
	(29.996)	(29.551)	(34.405)*	(35.752)*	(266.818)	(375.702)	(191.514)	(296.111)
Birth Weight< 2500g	0.042	0.043	0.028	0.029	-0.076	-0.111	0.029	0.047
	(0.019)**	(0.023)**	(0.015)*	(0.016)*	(0.162)	(0.280)	(0.084)	(0.119)
Birth Weight< 2000g	0.011	0.012	0.004	0.004	0.046	0.058	0.062	0.076
	(0.008)	(0.009)	(0.008)	(0.008)	(0.104)	(0.157)	(0.104)	(0.141)
Birth Weight< 1500g	0.006	0.007	0.003	0.003	-0.039	-0.057	0.014	0.019
	(0.006)	(0.006)	(0.005)	(0.006)	(0.075)	(0.133)	(0.029)	(0.040)
Gestation	-0.185	-0.189	-0.157	-0.162	-0.721	-0.772	-0.810	-1.011
	(0.122)	(0.129)	(0.128)	(0.136)	(1.514)	(2.074)	(1.375)	(1.884)
Premature Birth	0.027	0.028	0.003	0.002	0.053	0.056	0.033	0.051
	(0.017)	(0.018)	(0.013)	(0.014)	(0.134)	(0.182)	(0.093)	(0.131)
1-Min APGAR Score	-0.184	-0.203	-0.082	-0.091	-0.339	-0.381	-0.783	-0.905
	(0.084)**	(0.095)**	(0.073)	(0.079)	(0.772)	(1.071)	(1.241)	(1.600)
5-Min APGAR Score	-0.069	-0.077	-0.058	-0.064	-0.256	-0.284	-0.304	-0.329
	(0.041)*	(0.051)*	(0.045)	(0.049)	(0.517)	(0.662)	(0.502)	(0.572)
Num of Observations	45797	45797	14090	14090	14913	14913	2172	2172

Note: Each specification includes a quadratic in age at conception fully interacted with a dummy of being over age 21,together with year of conception and size of residence county fixed effects.Robust standard errors clustering in age-at-conception cells are reported in parentheses. *** means statistically significant at 1%, ** at 5%, * at 10%.



Note:A=90+date of conception day-date of 21 years old birthday

Figure 1-1: Definition of Variable A

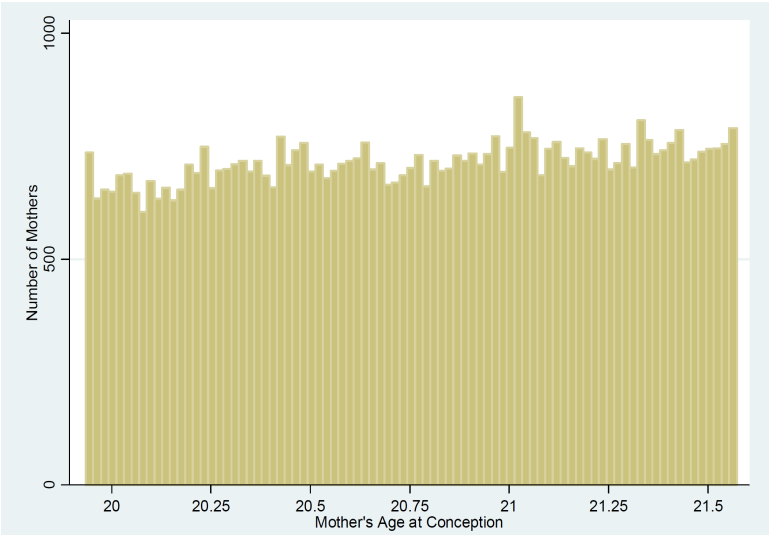


Figure 1-2: Frequency of Maternal Age at Conception

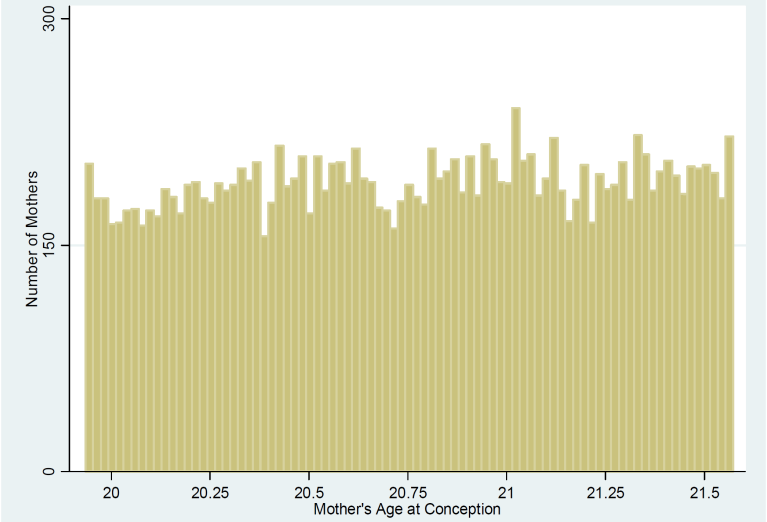


Figure 1-3: Frequency of Maternal Age at Conception(Smokers)

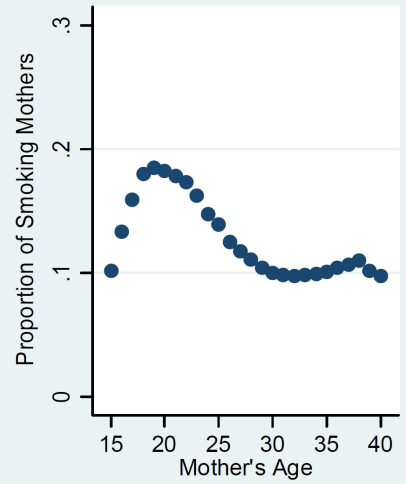


Fig2-1: Prenatal Smoking for Mothers at Various Ages
Source: 5% National Sample(1992-2002)

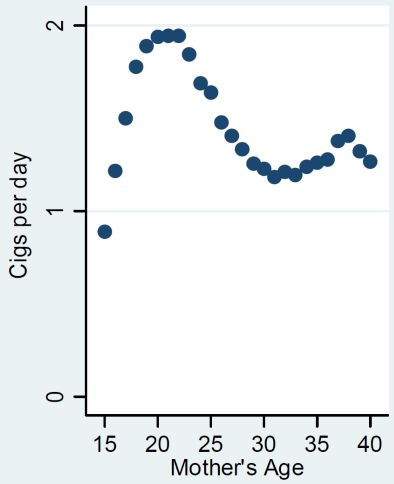


Fig2-2: Cigs per day for Mothers at Various Ages
Source: 5% National Sample(1992-2002)

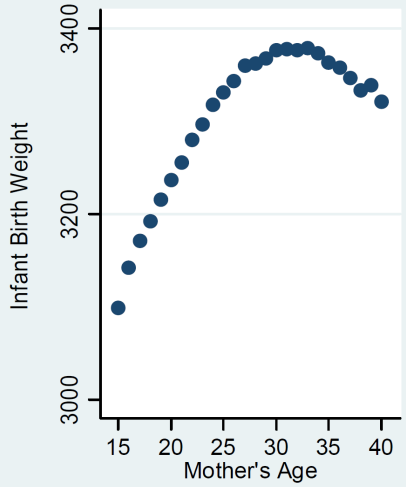


Fig2-3: Infant BW for Mothers at Various Ages
Source: 5% National Sample(1992-2002)

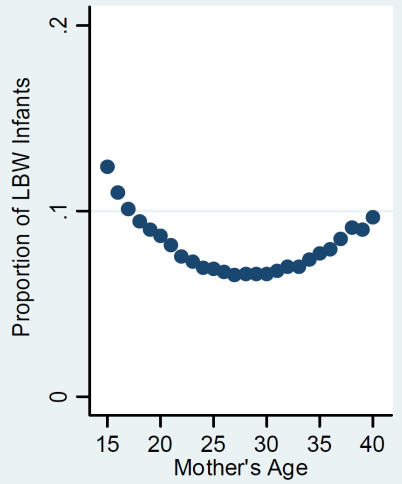


Fig2-4: LBW Infants for Mothers at Various Ages
Source: 5% National Sample(1992-2002)

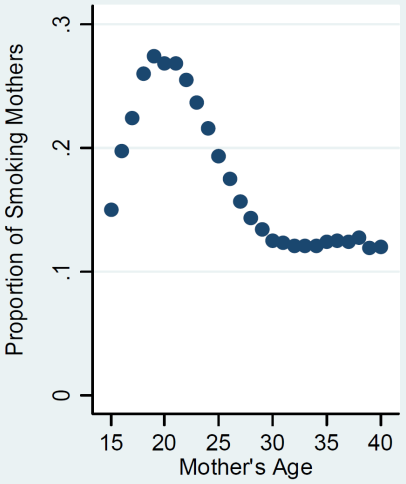


Fig2-5: Prenatal Smoking for Mothers at Various Ages
Source: 100% PA Sample(1992-2002)

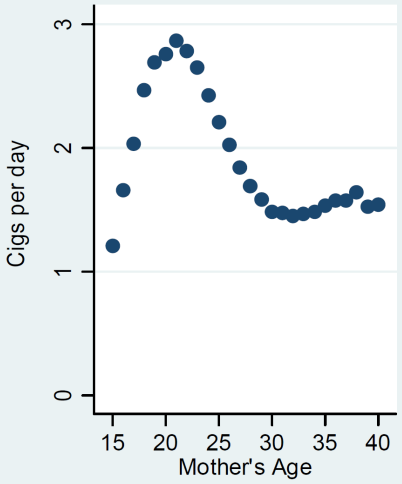


Fig2-6: Cigs per day for Mothers at Various Ages
Source: 100% PA Sample(1992-2002)

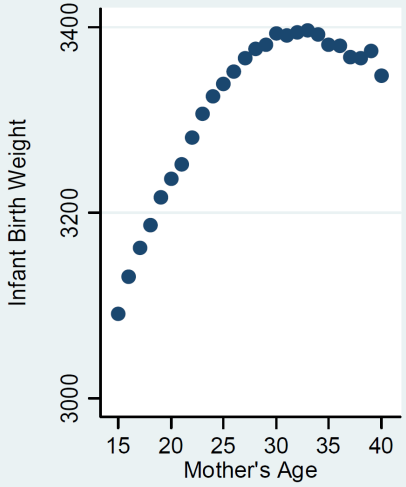


Fig2-7: Infant BW for Mothers at Various Ages
Source: 100% PA Sample(1992-2002)

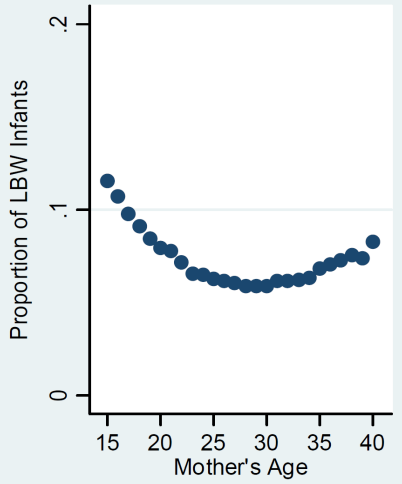


Fig2-8: LBW Infants for Mothers at Various Ages
Source: 100% PA Sample(1992-2002)

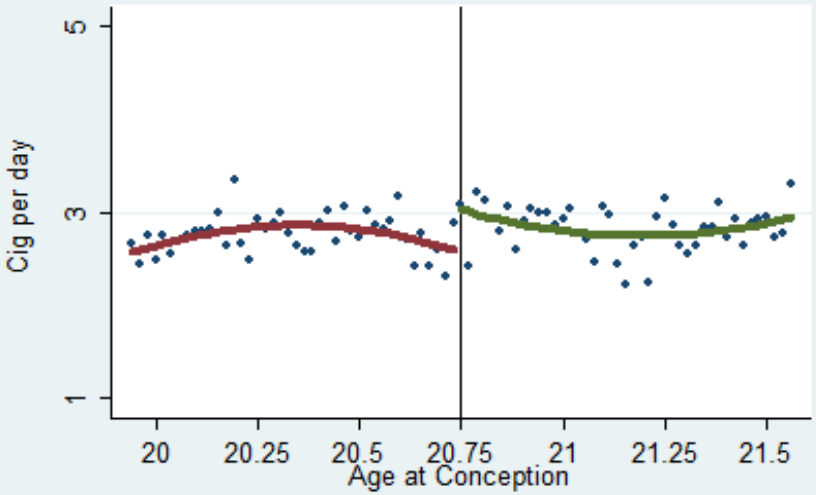


Figure 3-1: Age at Conception and Cigarettes per day in Pregnancy

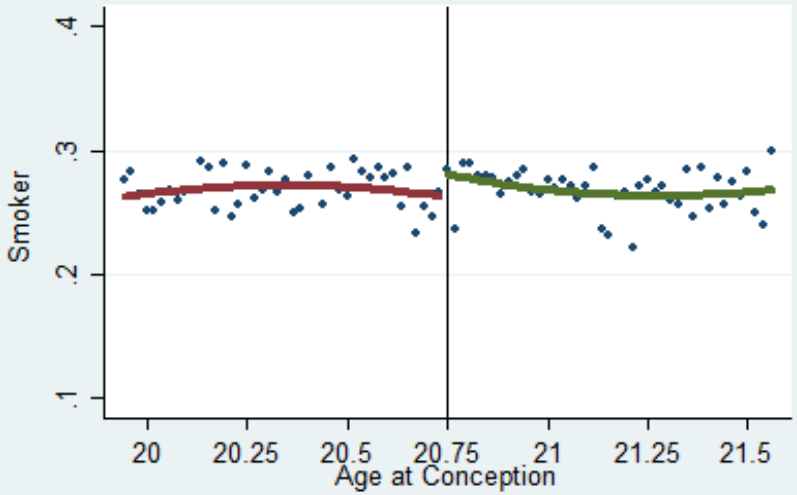


Figure 3-2: Age at Conception and Smoking in Pregnancy

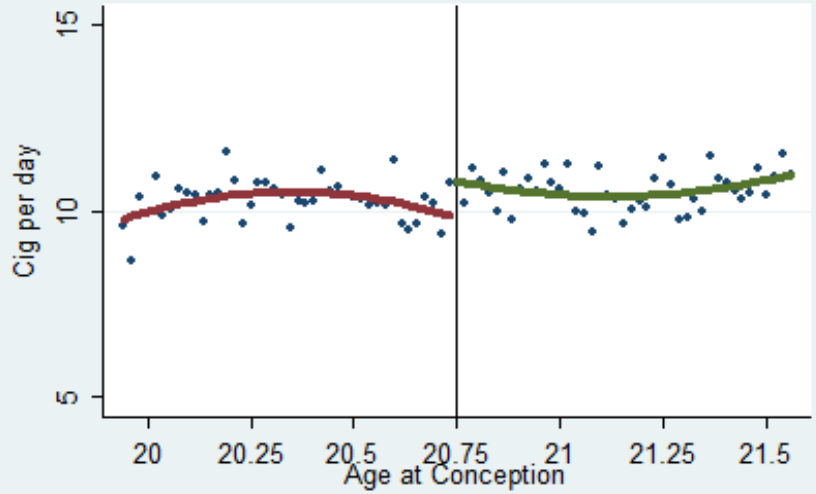


Figure 3-3: Age at Conception and Cigarettes per day in Pregnancy(Smokers)

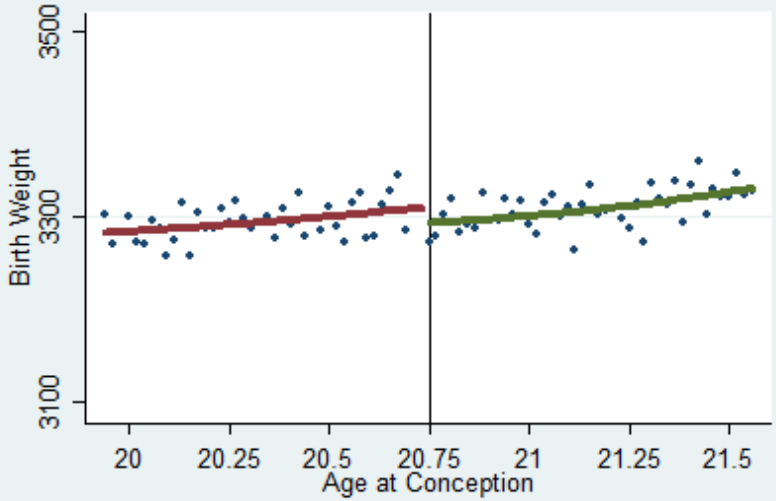


Figure 4-1: Age at Conception and Infant Birth Weight

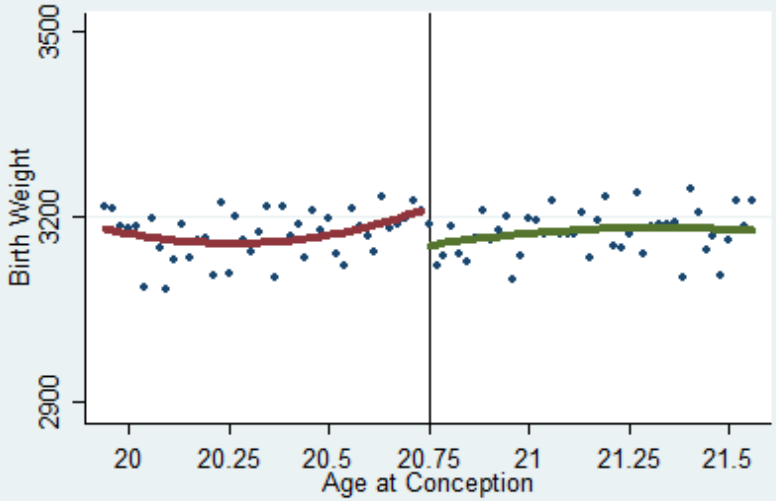


Figure 4-2: Age at Conception and Infant BW(Smokers)

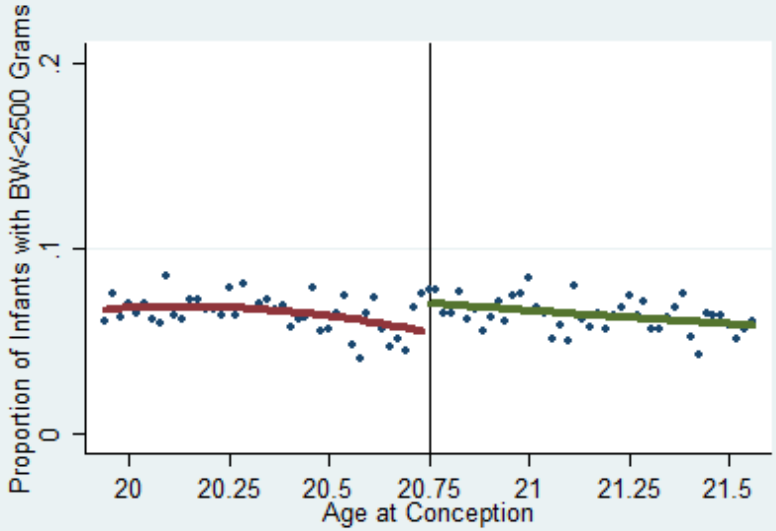


Figure 5-1: Age at Conception and Infant BW<2500

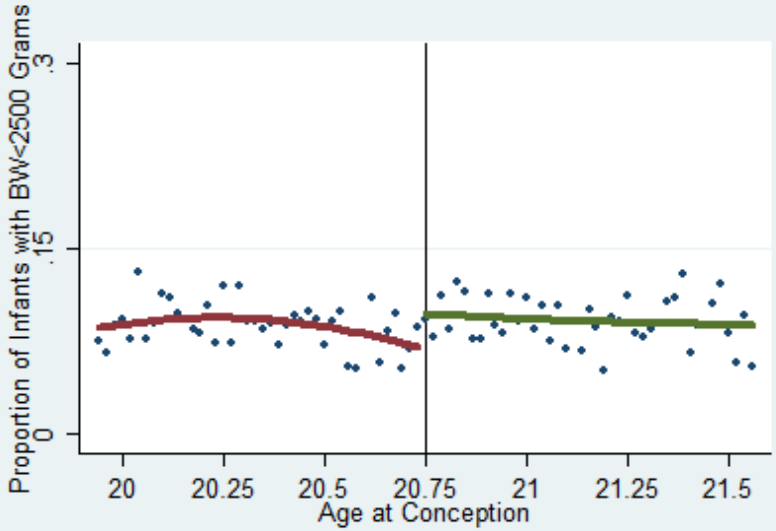


Figure 5-2: Age at Conception and Infant BW<2500(Smokers)

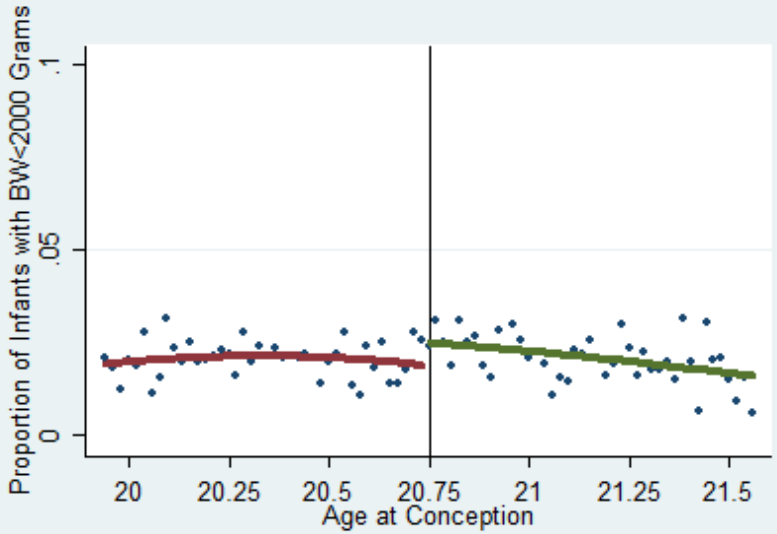


Figure 6-1: Age at Conception and Infant BW<2000

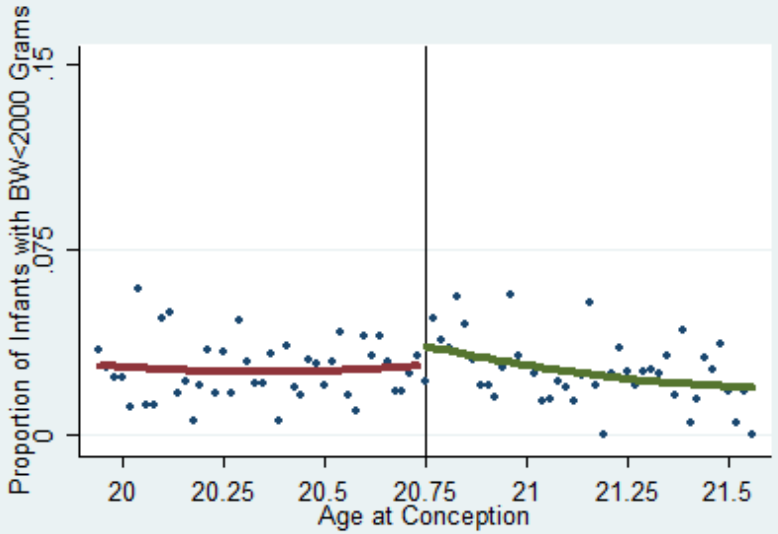


Figure 6-2: Age at Conception and Infant BW<2000(Smokers)

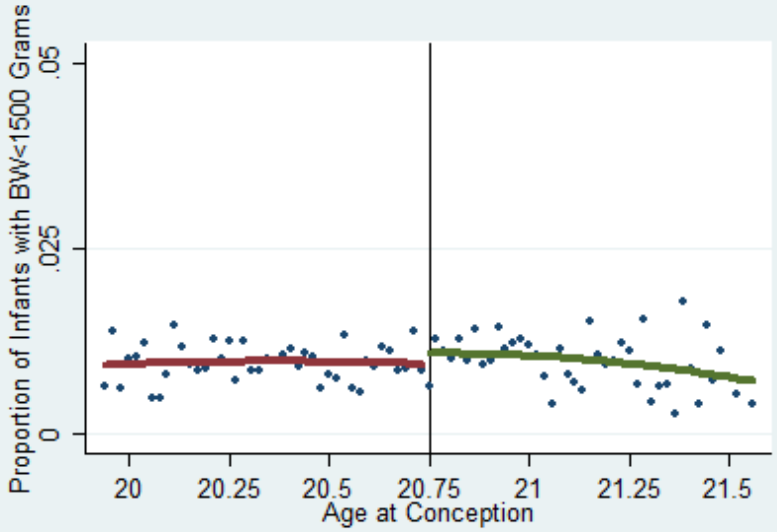


Figure 7-1: Age at Conception and Infant BW<1500

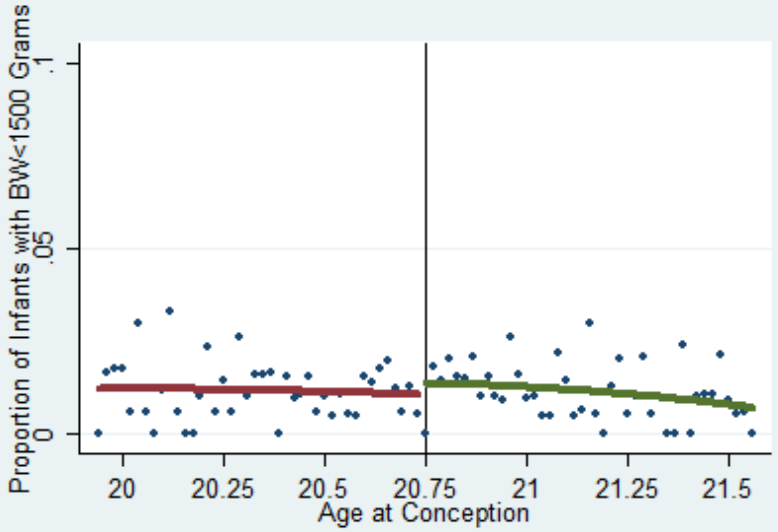


Figure 7-2: Age at Conception and Infant BW<1500(Smokers)

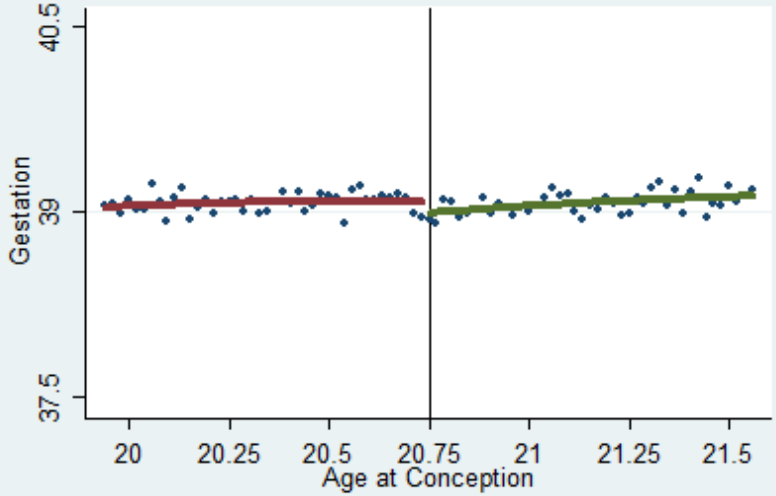


Figure 8-1: Age at Conception and Gestation

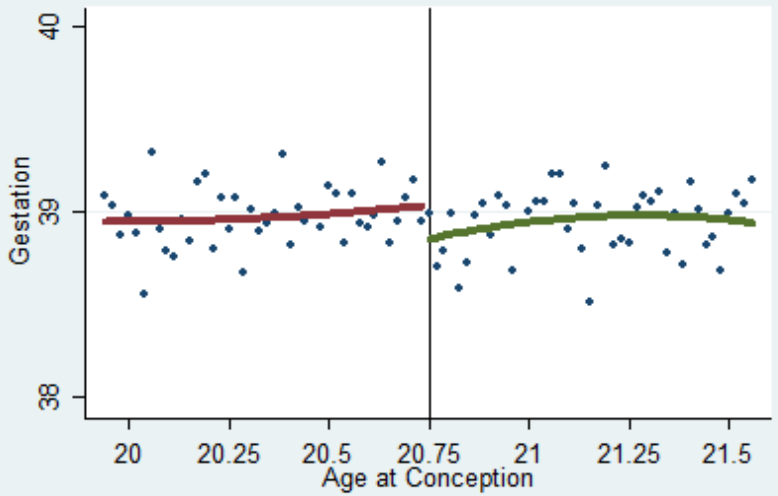


Figure 8-2: Age at Conception and Gestation(Smokers)

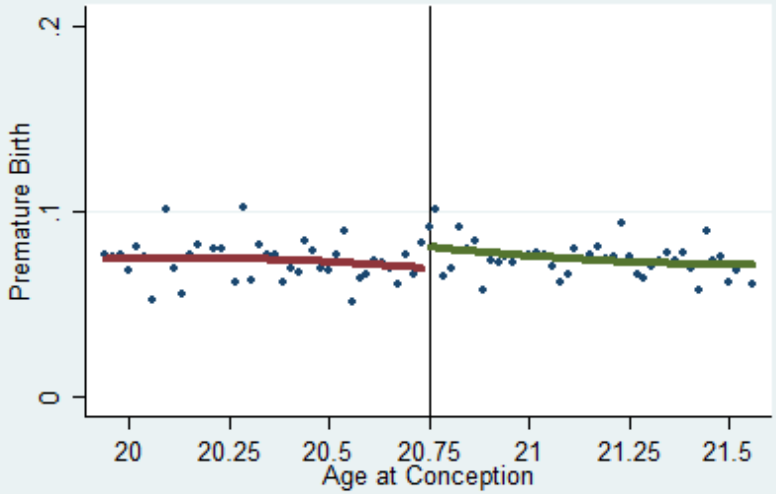


Figure 9-1: Age at Conception and Prematurity

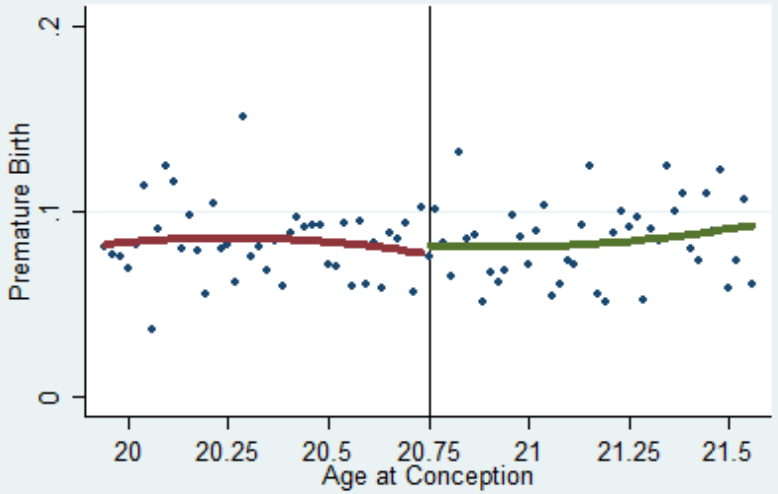


Figure 9-2: Age at Conception and Prematurity(Smokers)

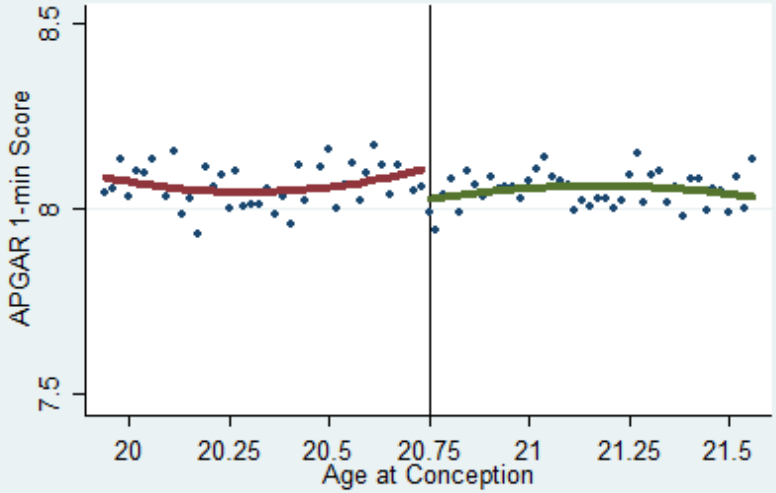


Figure 10-1: Age at Conception and APGAR 1-min

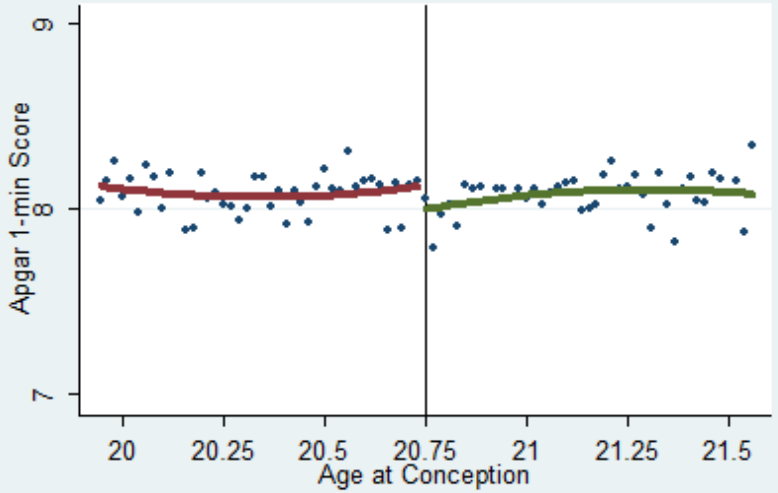


Figure 10-2: Age at Conception and APGAR 1-min (Smokers)

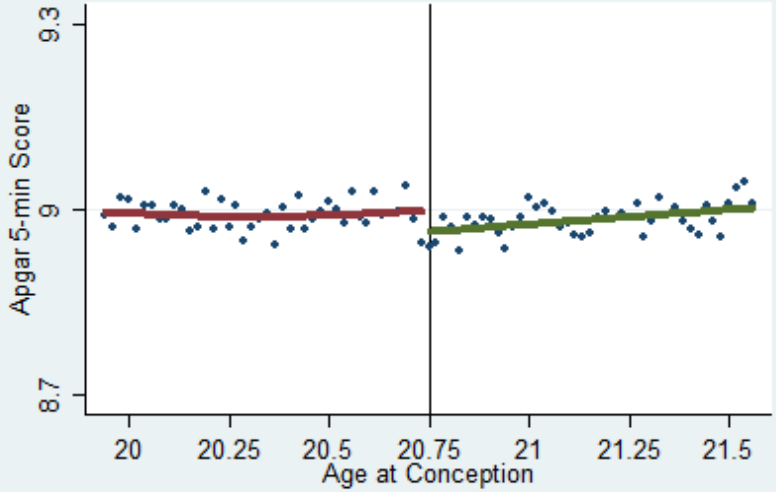


Figure 11-1: Age at Conception and APGAR 5-min

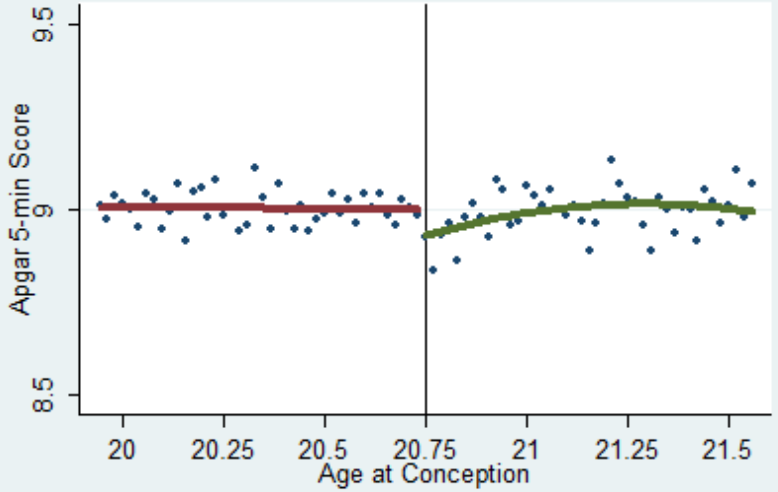


Figure 11-2: Age at Conception and APGAR 5-min (Smokers)

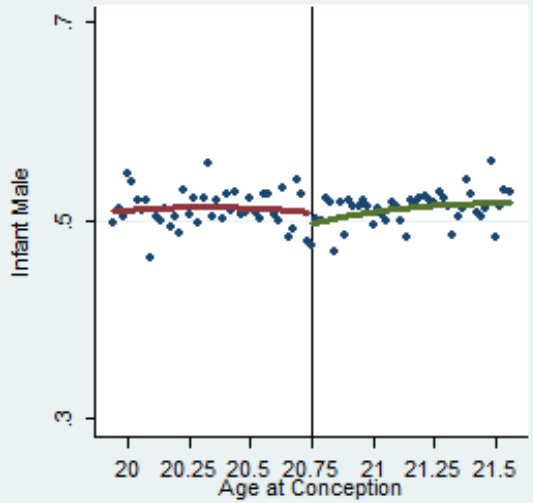


Fig 12-1: Age at Conception and Infant Male

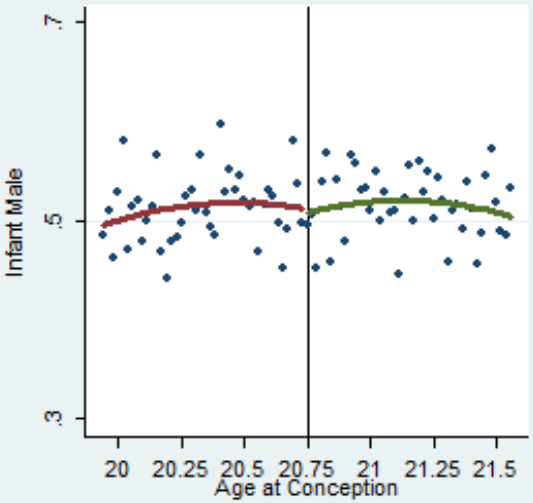


Fig 12-2: Age at Conception and Infant Male(Smokers)

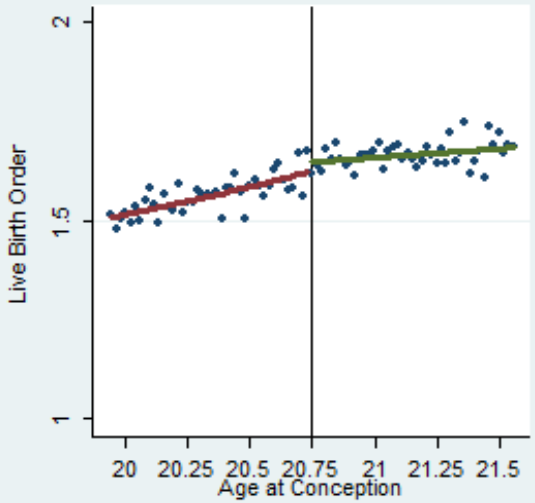


Fig 13-1: Age at Conception and Live Birth Order

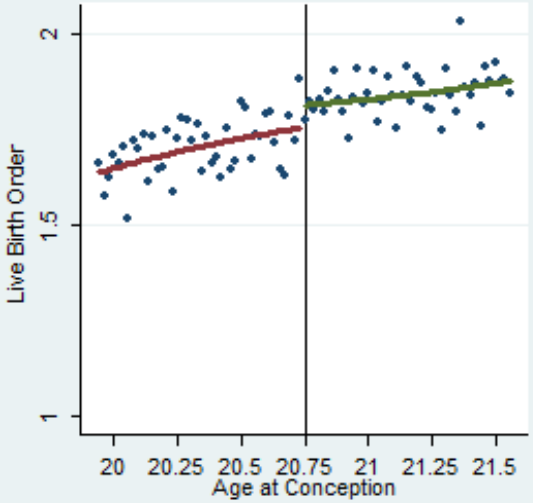


Fig13-2: Age at Conception and Live Birth Order(Smokers)

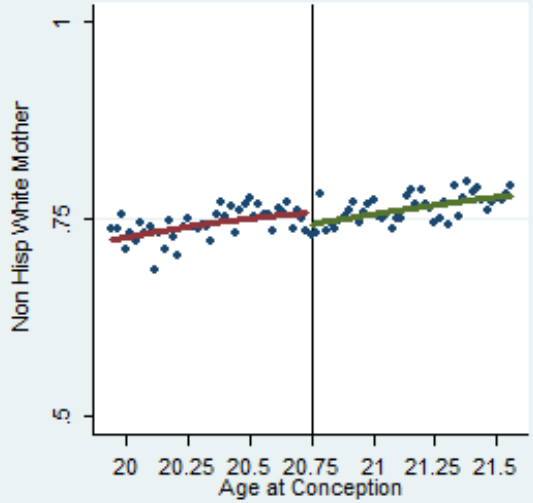


Fig 14-1: Age at Conception and White Mother

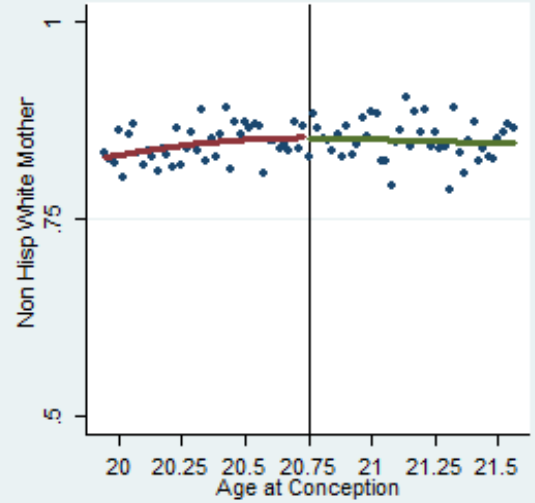


Fig 14-2: Age at Conception and White Mother(Smokers)

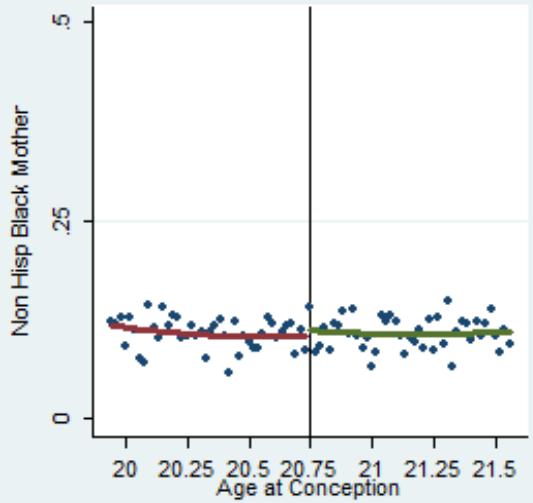


Fig 15-1: Age at Conception and Black Mother

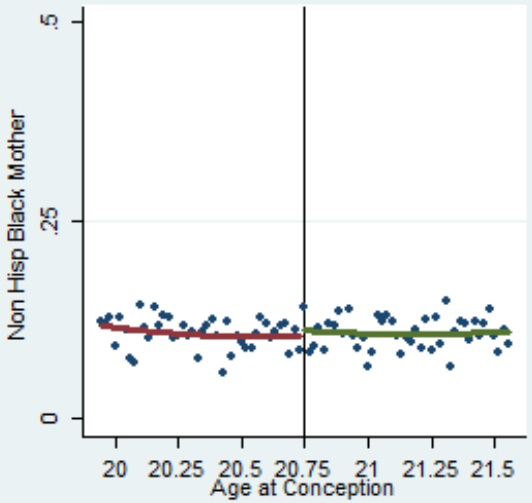


Fig15-2: Age at Conception and Black Mother(Smokers)

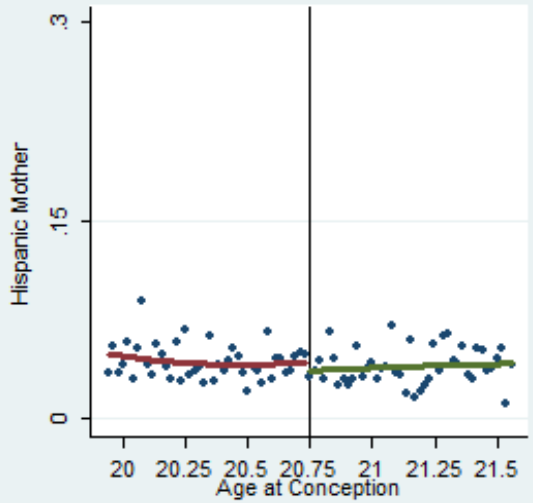


Fig 16-1: Age at Conception and Hispanic Mother

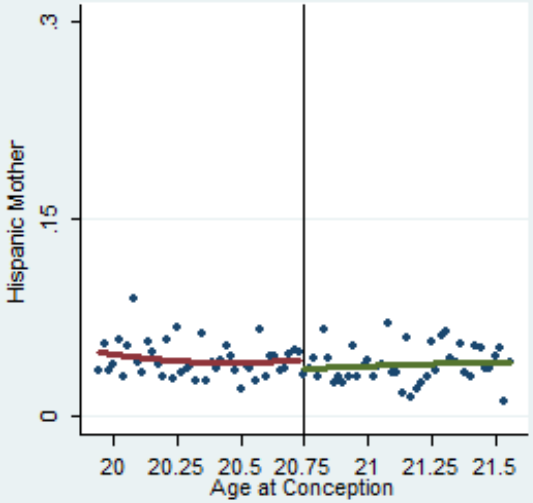


Fig16-2: Age at Conception and Hispanic Mother(Smokers)

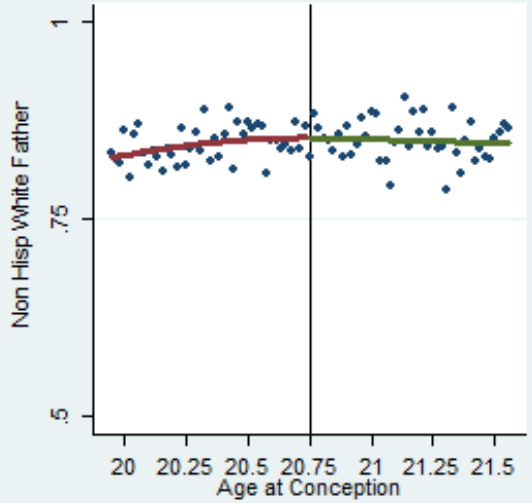


Fig 17-1: Age at Conception and White Father

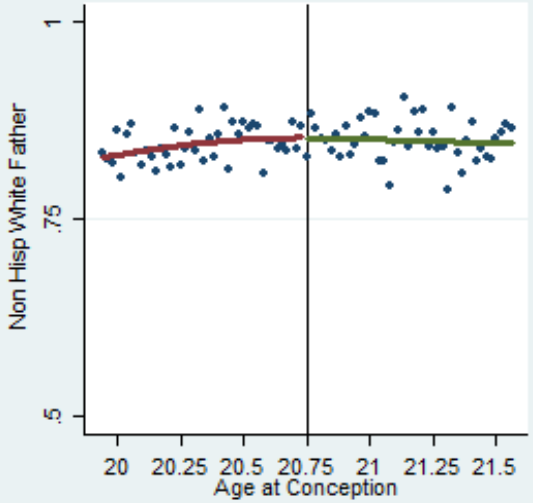


Fig 17-2: Age at Conception and White Father(Smokers)

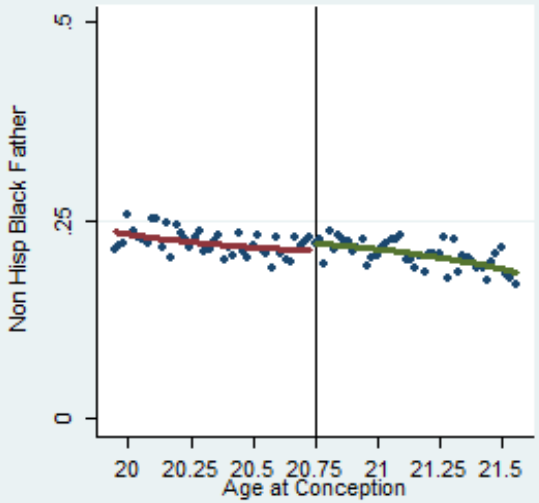


Figure 18-1: Age at Conception and Black Father

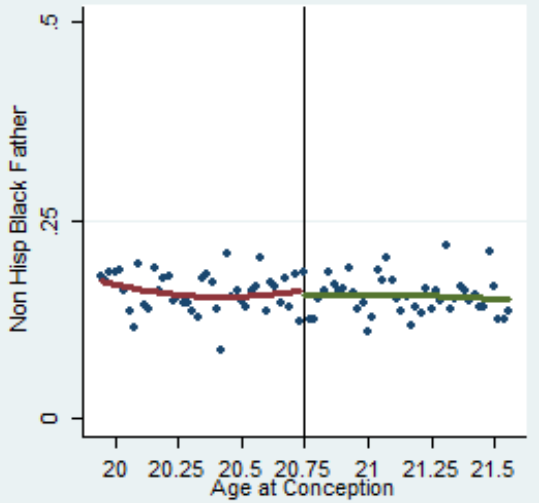


Fig 18-2: Age at Conception and Black Father(Smokers)

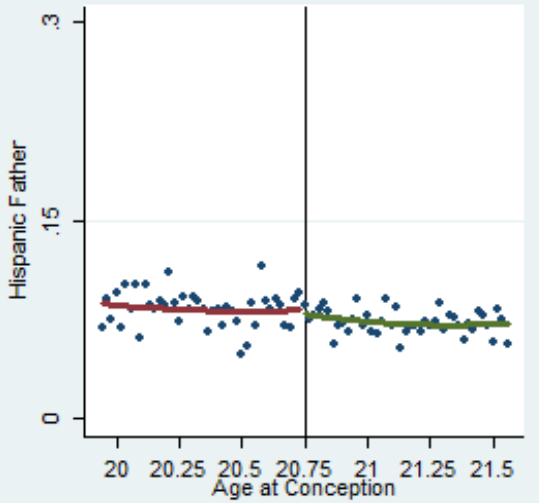


Figure 19-1: Age at Conception and Hispanic Father

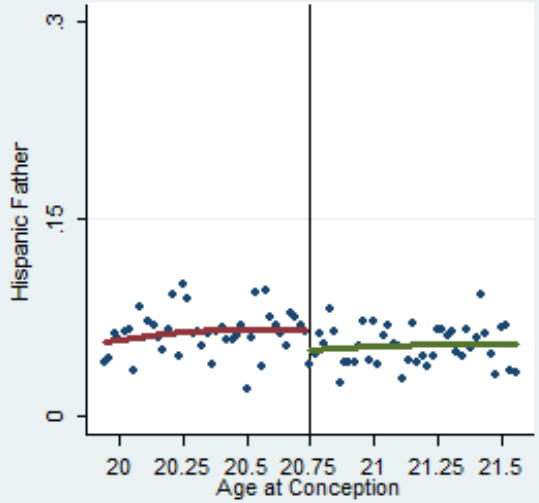


Fig 19-2: Age at Conception and Hispanic Father(Smokers)

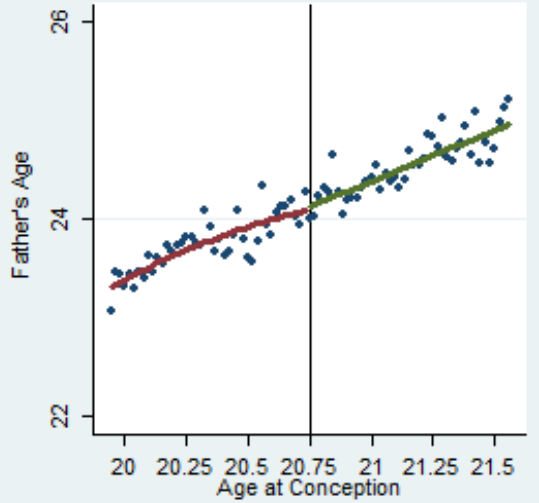


Fig 20-1: Age at Conception and Father's Age

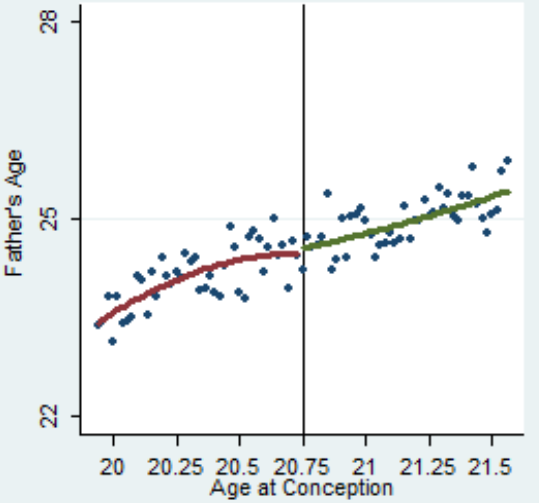


Fig 20-2: Age at Conception and Father's Age(Smokers)

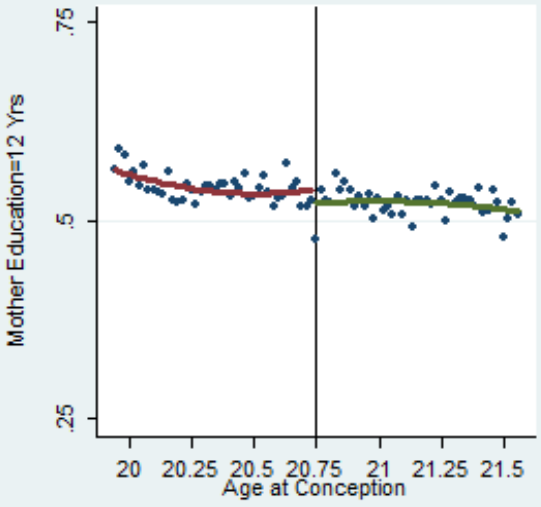


Fig21-1: Age at Conception and Mom Edu=12 Yrs

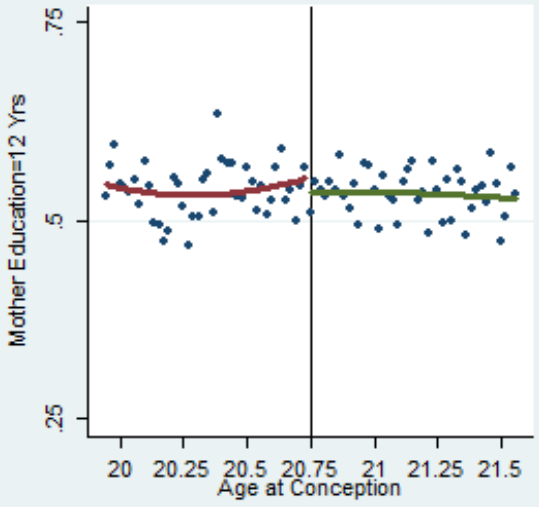


Fig21-2:Age at Conception and Mom Edu=12Yrs(Smokers)

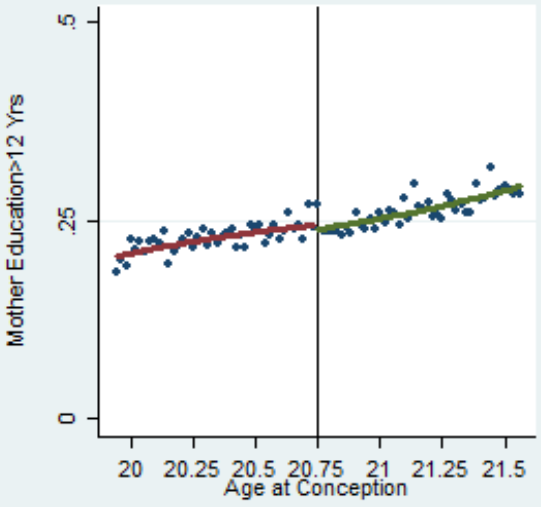


Fig 22-1: Age at Conception and Mom Edu>12Yrs

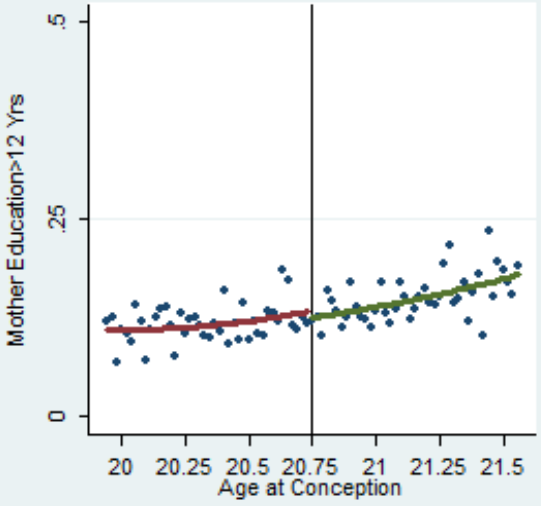


Fig22-2:Age at Conception and Mom Edu>12Yrs(Smokers)

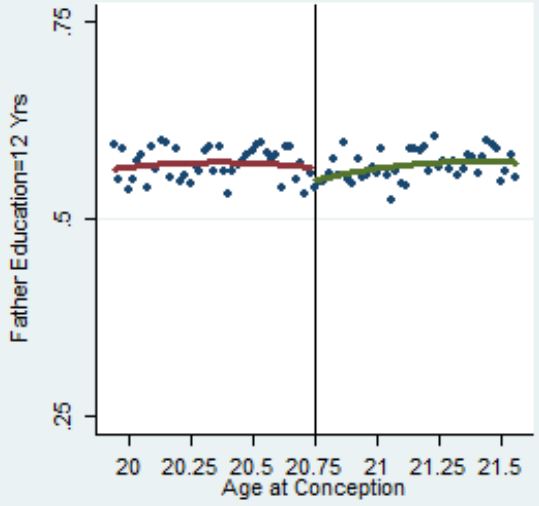


Fig 23-1: Age at Conception and Dad Edu=12 Yrs

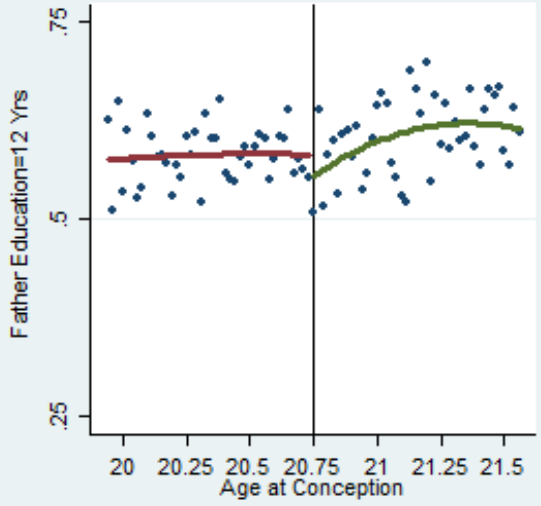


Fig23-2: Age at Conception and Dad Edu=12 Yrs(Smokers)

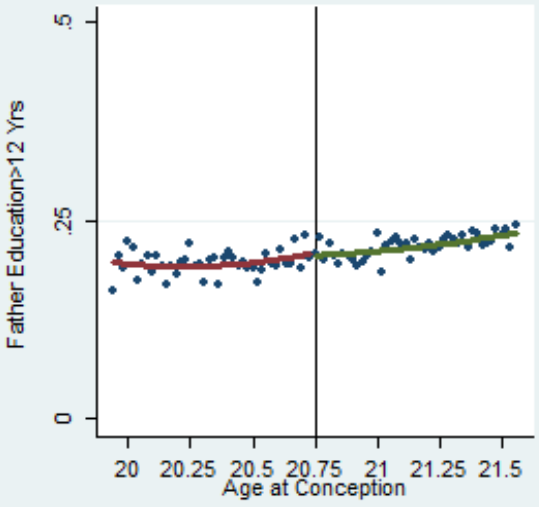


Fig24-1: Age at Conception and Dad Edu>12 Yrs

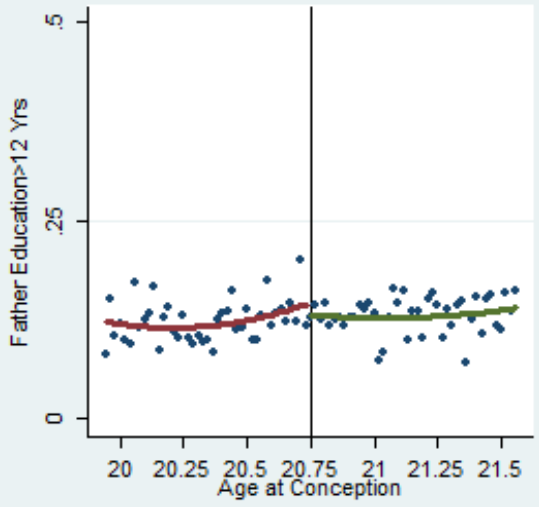


Fig24-2:Age at Conception and Dad Edu>12 Yrs(Smokers)

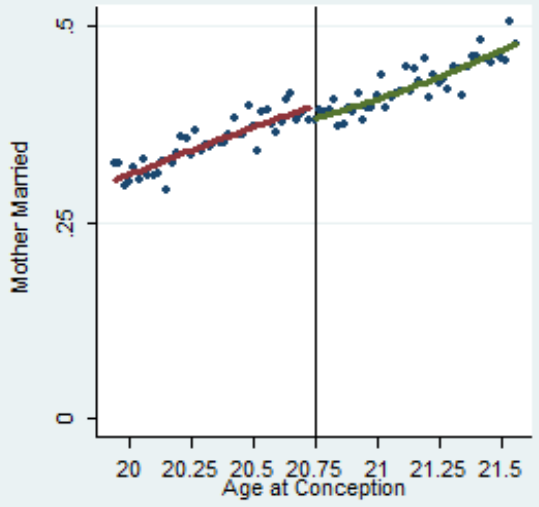


Fig25-1: Age at Conception and Mother Married

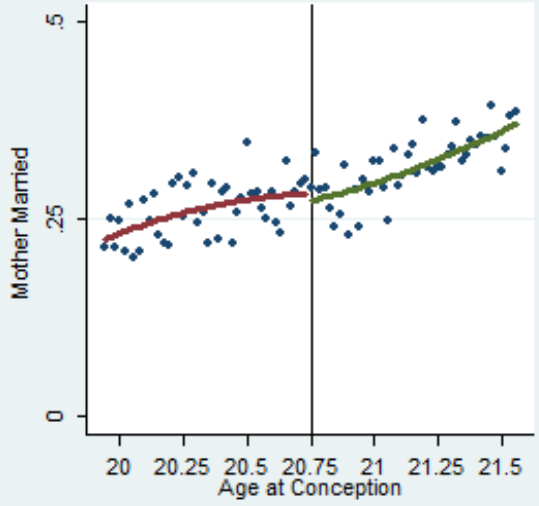


Fig 25-2: Age at Conception and Mother Married(Smokers)

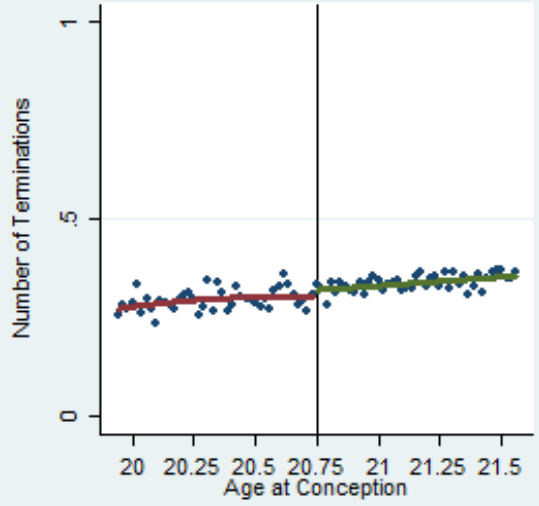


Fig26-1: Age at Conception and Terminations

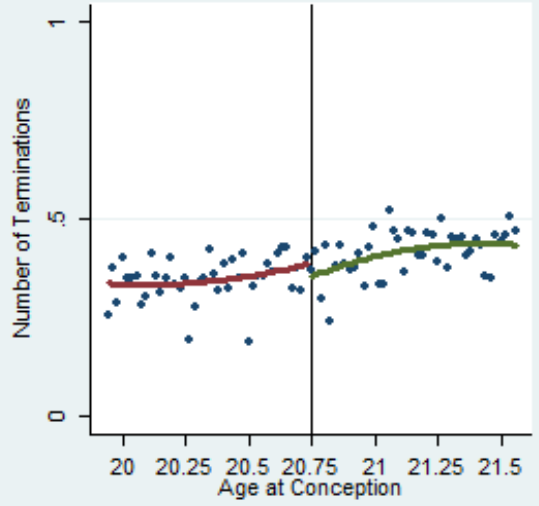


Fig26-2:Age at Conception and Terminations(Smokers)

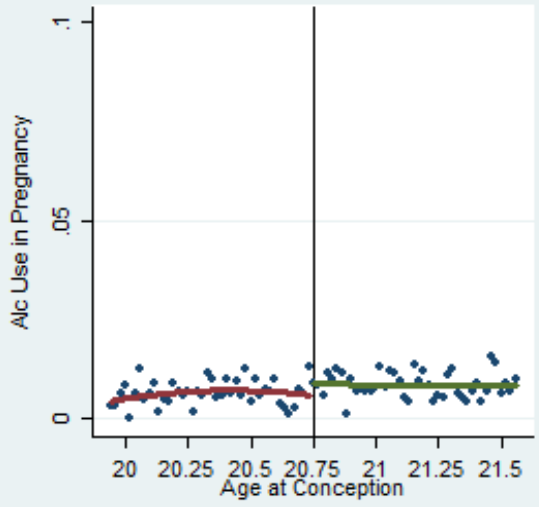


Fig 27-1: Age at Conception and Drink in Pregnancy

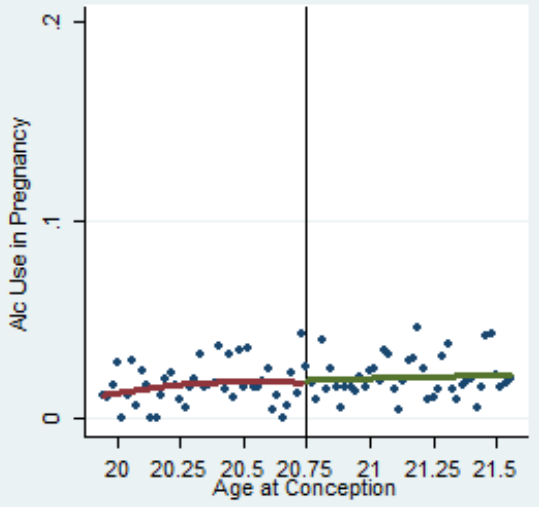


Fig27-2: Age at Conception and Drink in Preg(Smokers)

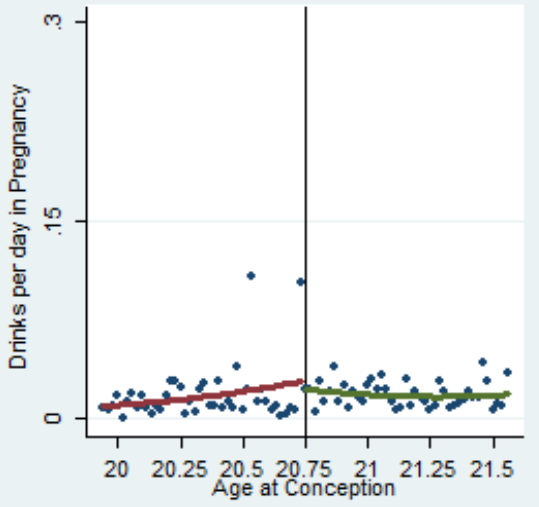


Figure 28-1: Age at Conception and Drinks per day

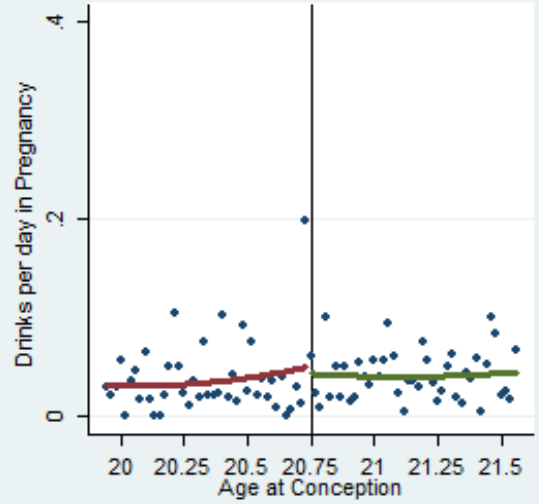


Fig28-2: Age at Conception and Drinks per day(Smokers)

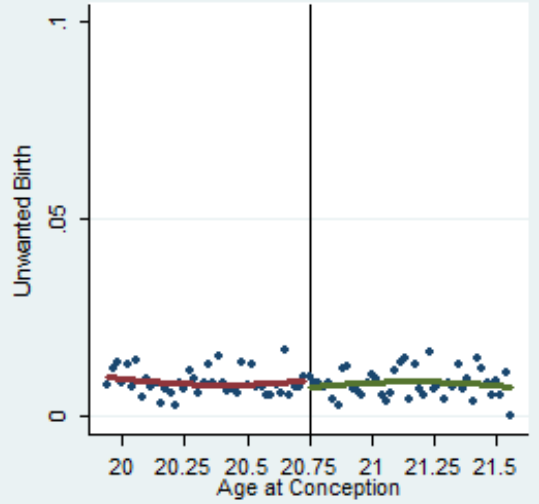


Fig 29-1: Age at Conception and Dad Age Missing

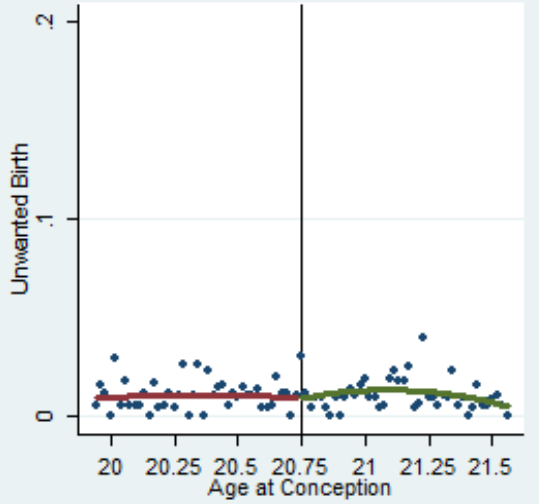


Fig29-2: Age at Conception and Dad Age Missing(Smokers)

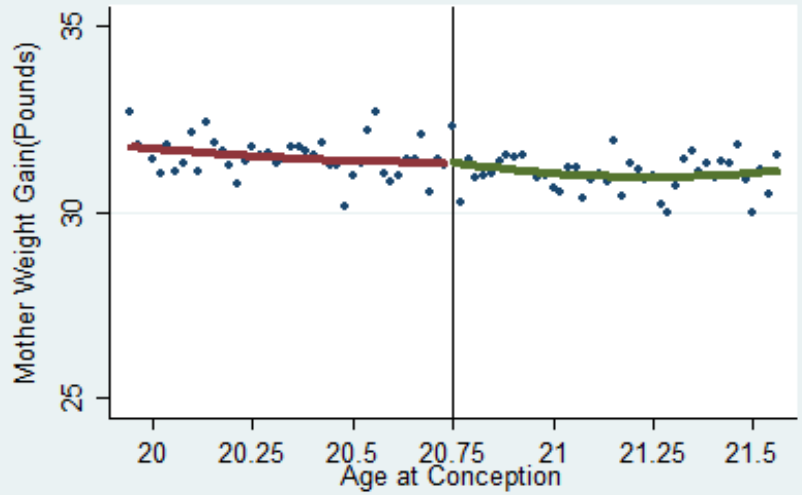


Fig 30-1: Age at Conception and Mother Weight Gain

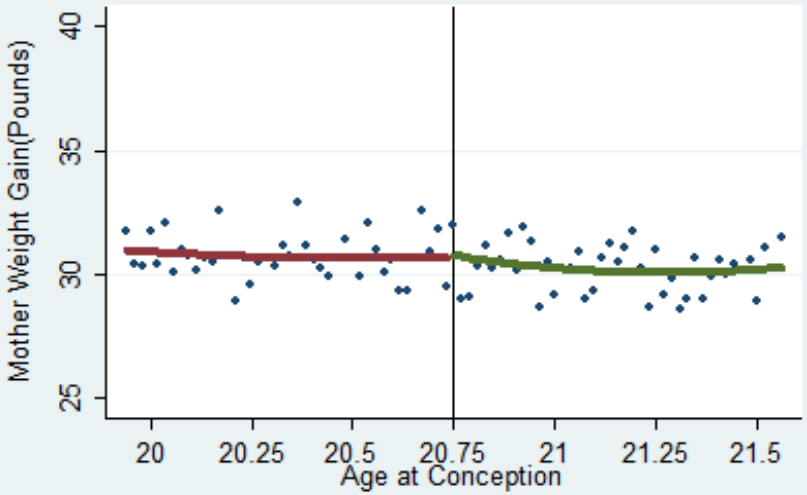


Fig 30-2: Age at Conception and Mother Weight Gain(Smokers)

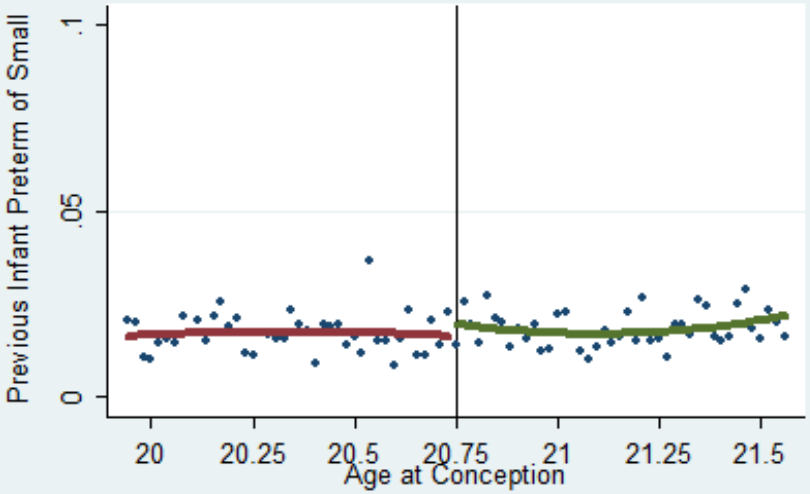


Fig 31-1: Age at Conception and Previous Infant Preterm of Small

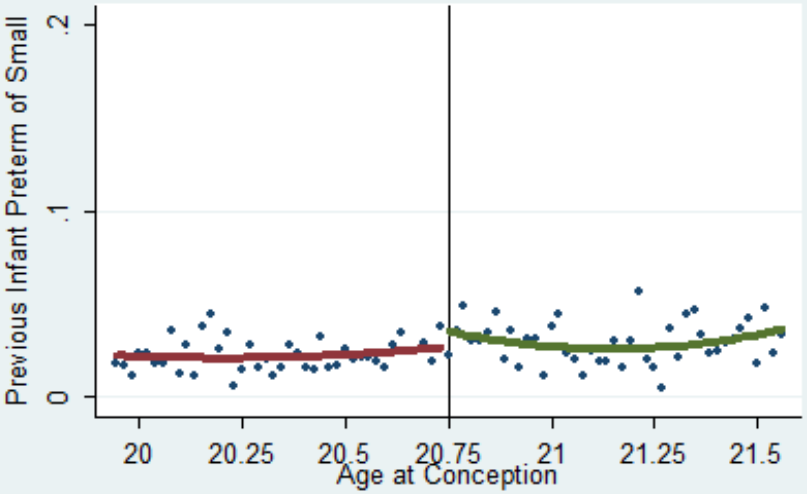


Fig 32-2: Age at Conception and Previous Infant Preterm of Small(Smokers)