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Maternal smoking during pregnancy and birthweight – A propensity score matching approach

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Abstract

There is accumulated evidence of the existence of a deleterious effect of smoking on birth outcomes. Whether there is a causal link or a mere statistical association is not clear. Understanding the effect of smoking on pregnancy is a critical issue because of the public policy implications for dissuading maternal smoking. This study was designed to distinguish causal links from statistical association in the relationship between fetal exposure to maternal smoking and birth outcomes. Although the task involves several aspects of estimation we restrict our focus to the issue of self-selection. We explore this issue by using the propensity score method and compare that with parametric estimators. First we estimate the treatment effect of smoking during pregnancy on different birth outcomes. Then, we extend the method to the case of the multi-treatment "intensity of smoking". The deleterious effect of smoking is found robust to the different estimation methods used.

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Introduction

Despite the remarkable decline in smoking in United States of America, smoking is still a common form of maternal substance abuse during pregnancy and is thought to be the largest modifiable risk factor for pregnancy [Kramer (1987)]. There is accumulated evidence suggesting that maternal smoking during pregnancy has a negative effect on birthweight, by increasing the risk of low birth weight (less than 2500 grams), as well as the risk for other infant health hazards [See Kramer (1987) and Walsh (1994) for reviews]. There is also strong evidence of dose responsiveness on birthweight [Walsh (1994)]. Nonetheless, the causality and the magnitude of such effects are still unclear. Skepticism regarding causal interpretation of the associations between maternal smoking and undesirable birth outcomes arises because it is believed that women who persist in smoking through pregnancy are not likely to be randomly drawn from the pregnant population. A potential bias arises because there might be persistent omitted factors that affect both the birth outcome and smoking decision. Two characteristics of smokers explain why smokers are likely to be a self-selected group among pregnant women. First, smoking is an addictive habit. The literature on addiction characterizes addicts as individuals with higher discount rates, likely to be myopic or lack self-control. Therefore cigarette smoking is perhaps most appropriately interpreted as part of a broader pattern of behavior and life-style. Second, smoking is also a health decision. It is plausible to assume that mothers have at least some information not observable to the researcher about the risks of smoking and their own health status and the health of their fetus. The sign of the bias is not obvious because the relationship between health and smoking decisions is still theoretically ambiguous.

An ideal framework for assessing the causal effects of maternal smoking would be to conduct an experimental trial in which expectant mothers would be randomly assigned into smoking and non-smoking groups. Ethical considerations, as well as costs, preclude such experiments. As an alternative to the experimental approach, several non-experimental methods have been proposed. In the econometric literature, the dominant approach has been to model causality and self-selection using a system of structural equations. A classical method often used in applied economics to obtain consistent estimators is the two step least squares, mainly the Instrumental Variable (IV) estimator. Recent contributions from economists address the problem of potential endogenity in smoking using IV methods. Overall, they find a higher impact of smoking on birthweight than previous single-parameter regression epidemiological studies. This finding contradicts the main dominant belief in the epidemiology and medical literature that mothers who

smoke have other undesirable unobservable characteristics, and therefore the higher prevalence of adverse outcomes is due to the smoker and not to the smoking per se [Yerushalmy (1971), Butler, Goldstein & Ross (1972), Silverman (1977), Hickey, Clelland & Bowers (1978)]. These studies are not convincing because the statistical methods applied rely on strong assumptions. Indeed the empirical consequences of the IV scheme depend greatly on the "quality of the instruments," as well as on the amount of heterogeneity in the population to be observed.

Our main contribution in this paper is to deal with potential bias using the propensity score matching method. Matching estimation has received increasing attention in the econometric literature as a serious alternative to structural analysis of non-experimental data [For a comprehensive survey see Angrist & Krueger (1999) and Heckman, Lalonde & Smith (1999)]. Originally developed by Rubin (1977) and Rosenbaum & Rubin (1983), matching methods were extended by Heckman, Ichimura & Tood (1998), Imbens (2000) and Lechner (1999).

As far as we know this is the first application of the propensity score in this context. The major advantages of matching procedures are that they do not require parametric functional form and exclusion restrictions. Moreover, leaving the individual causal effects completely unrestricted reduces the problem of heterogeneity in the population. The Stata command to perform Propensity Score Matching [psmatch] is implemented by Barbara Sianesi [see Sianesi (2001)].

The remainder of this paper is organized as follows. We first briefly summarize previous works. The next section presents the propensity score matching method. The following section describes the data used, including the birth outcome variables and smoking variables. Next we discuss the statistical results for Ordinary Least Squares, Probit regressions and propensity score methods, followed by a discussion of the results of Propensity score methods in comparison with the benchmark regressions and tests of the unconfoundness hypothesis. The final section presents some concluding remarks

Previous works

In this section we review some recent studies that attempted to deal with the potential bias of single equation parametric estimation. Rosenzweig & Schultz (1983) made the first attempt to investigate the causal relationship between smoking and birthweight using the IV method. They used as an additional instrument to the number of daily smoked cigarettes the price of cigarettes during the year that mother become pregnant. Their estimates show that those who smoke fourteen cigarettes a day have infants weighing, on average, 195 grams less than the infants of non-smokers. Their OLS estimates suggest a decrease of 179 grams. While a seminal paper in health

economics, the conclusions from Rosenzweig & Schultz are not robust. The set of instruments employed had little relevance in the first stage reduced form, leading to potential bias. Permutt & Hebel (1989) used data from Sexton & Hebel's (1984) smoking cessation program to estimate the magnitude of the impact of smoking participation in a simultaneous equation model. A dummy for "received intervention" was used as instrument for maternal smoking behavior, based on the assumption that participation is randomly assigned and (only) affects birthweight by reducing smoking. Alarmingly, the study suggests a much greater negative effect of smoking on birthweight than previous studies. According to the results, total cessation raises birthweight by 430 grams. The results are clouded by the important drawbacks of using IV estimates with experimental data [Angrist, Imbens & Rubin (1996), Heckman (1995, 1998)].

Evans & Ringel (1999) explored the within-state variation in taxes, in the period of 1989-1992, as an instrument for smoking participation during pregnancy. Their IV estimates suggest that fetal exposure to smoking results in a 367 gram birthweight deficit. Evans & Ringel (1999) correctly noted that their estimates may overestimate the true impact of smoking. They argued that changes in the prices of cigarette across years within states are not likely to affect all smokers. If those who quit in response to an increase in the cigarette price have marginal gains from quitting, the IV estimates are likely to be larger than the OLS but results cannot be generalized.

A different approach is found in Hamilton (2001). The author applies a Bayesian treatment models to estimate the causal impact of maternal smoking during the 8th month of gestation. Using the data from Sexton & Hebel (1984) smoking cessation program, the author estimated the treatment effects on the restricted sample of compliers, following the note of Angrist *et al.* (1996). The main strength of this approach is that Bayesian models are flexible and explicitly allow for heterogeneous treatment participation and response. The results show that late term maternal smoking reduces the birthweight by an average of 348 grams. Furthermore, quitting has a stronger effect among women who were moderate smokers prior to pregnancy, implying a birthweight difference of 430 grams.

Methods

Matching methods

Our goal was to use propensity score matching to estimate the effect of smoking on birth weight and risk for low birth weight (thereafter LBW) Using the terminology in the evaluation literature we were interested in evaluating the causal effect of the treatment of interest "smoking

during pregnancy" (S=1), relative to another treatment "no smoking during pregnancy", (S=0) on the birth outcomes (BO). Let BO_1 be the birth outcome of a smoker and BO_0 the birth outcome for non-smoker. We wanted to estimate: $E [BO_1 - BO_0 | S=1] = E [BO_1| S=1] – E [BO_0| S=1]$. In the program evaluation literature this difference, is called the "average treatment effects on the treated population" [Heckman & Robb (1995)]. It is thus necessary that each mother is potentially exposable to any treatment. From the data we can observe the first term on the right side, but we cannot observe the second term, that is, the birth outcome a smoker would have if she had chosen not to smoke. If mothers who smoke are not random the one equation parametric estimator bias is given by: $E[BO_1| S=1] - E[BO_0| S=0] = E[BO_1 - BO_0 | S=1] + \{E[BO_1| S=1]- E[BO_1| S=0]\}$

Randomization of the assignment to treatment S would solve this problem, but it is ethically unviable. The matching method provides a way to estimate treatment effects when controlled randomization is not possible. It is based on a simple idea: for each mother who smokes, find a group of comparable mothers who have similar observable characteristics among the non-smokers. Within each set of matched individuals one can then estimate the impact of maternal smoking on the individual by the difference in the sample means. Unmatched observations are discharged from analysis; therefore, the matching estimator approximates the virtues of randomization mainly by balancing the distribution of the observed attributes across smokers and non-smokers. Deheija & Wahba (1998) showed that matching provides a significantly closer estimate for the treatment effects than the standard parametric techniques.

The key assumption underlying the matching methodology is that of *unconfoundedness*. This assumption asserts that the relevant differences between smokers and non-smokers are captured by the observable characteristics of mothers and, that conditional on these characteristics, smoking status can be taken to be random. Formally;

$BO \perp\!\!\!\perp S \mid \mathbf{X}$, where $\perp\!\!\!\perp$ denotes independence. (1)

We have further to assume that there are smokers and non-smokers for each possible set of characteristics x in X, i.e. $0 < \Pr(S \mid \mathbf{X}) < 1$ (2)

Ideally, we would control for all mothers' observable characteristics thought to influence both smoking participation and birth outcomes. However, matching using all relevant variables was impractical because of computational burden. As an alternative, the empirical literature often invokes the finding of Rosenbaum & Rubin (1983) that showed that if (1) and (2) hold, and then individuals can be matched based on the propensity of smoking participation P(x), rather than conditional on X itself. In this case, the *unconfoundedness* can be re-written as $BO \perp\!\!\!\perp S \mid P(\mathbf{X})$. This

method, called the *propensity score* method, has been applied by several researchers [See e.g. Dehejia & Wabha (1999, 2002), Heckman *et al.* (1998), and Angrist & Hahn (1999)].

The *unconfoundedness* assumption validates the comparison of smokers and non-smokers with the same (or close) values of $P(\mathbf{X})$ (or **X**). Therefore, it is possible to estimate the "potential" average effect of smoking during pregnancy on the birth outcomes among smokers, by calculating the difference between the birth outcomes of smokers and what the birth would have been if they did not smoke.

Estimation of a propensity score binary matching method is therefore done in two steps. The first step is to estimate a propensity score $P(\mathbf{X})$ for smoking. The second step, given the estimated propensity score, is to apply the matching methods to the univariate non-parametric regression E [B0|S=j, P (X)], j =0, 1]. We apply the radius method of matching. This method consists of matching each smoker to non-smokers whose propensity scores are within some tolerance level å. If there are no non-smoker observations within the tolerance this smoker record is discarded. Thus, the method matches a person i if and only if $|P(X_i) - P(X_j)| \leq$ å.

Estimation of multiple treatments

Our previous analysis of smoking/non-smoking groups can be extended to allow for different levels of smoking. Using the terminology introduced by Imbens (2000) and Lechner (1999), we assume that there are K+1 exclusive treatments denoted by 0, 1,..., K+1 where the value zero correspond to the absence of treatment. Therefore in our case the different treatments correspond to four levels of smoking (non-smoking, light, moderate, and heavy) and are denote by $S \in \{0,1,23\}$. The potential outcomes denoted by $B0^0$, BO^1, BO^2, $BO^3$are associated with the different (mutually exclusive) levels.

The identification assumption means that there exists a set of observable variables **X**, such that $BO \perp\!\!\!\perp S \mid P^S(\mathbf{X})$, where P^S denotes the probability of intensity s conditional on **X**. If the assumption holds, then the distribution of smoking effects may be identified for any pair of different levels, say {0,1} as $(B0^0, B0^1) \perp\!\!\!\perp S \mid [P^0, P^1](\mathbf{X}) \cdot S \in \{0,1\}$

Our main focus is to estimate E [$B0^1 - B0^0$ | S =l], i.e. the average conditional effect given the level of smoking l relative to non-smoking 0.

Data

The main data for this study come from the birth/infant death period linked file, compiled by the United States National Center for Health Statistics for the 1995 birth cohort. The dataset links the National Natality Detail files and National Mortality Detail files, which are derived from the universe of birth and death certificates in the 57 registration areas in the Unites States. The birth certificate includes much information about the mother and infant. Information from the death certificates includes infant's race, residence, age at death and causes of death.

To obtain the data used in study, we selected 25% of the roughly 3.9 million live births that occurred in the United States in 1995. This sample was selected to include all reported births that resulted in an infant or fetal death, of which there are roughly 26,000 in each category. The remaining birth records for our sample were drawn at random, albeit with STATA procedures that can be replicated, from the remaining births that did not result in a perinatal death. Because the sub-sample over-represents the number of perinatal deaths, we used appropriate weight corrections. Due to computational limitations the selected sample of Caucasian mothers was still too large, so we randomly selected a 35% sub-sample of these records.

The birth certificates of California, Indiana, New York State (excluding New York City) and South Dakota do not have information on maternal smoking during pregnancy. For this reason these states were not included in our analysis. Therefore 20% of the original data was deleted from analysis. The exclusion of the data from California disproportionately affects the representation of Hispanics. Consequently Hispanics were also excluded from the analysis. Births by mothers who reside outside the U.S. are also not included in the analysis. Multiple births are excluded because they are significantly different from singleton births with respect to birth outcomes and mortality risk. In addition, records of live births with missing birthweight information and those coded with implausible weights (less than 400 grams) are discharged from the analysis. We excluded from the analysis the records of fetal death with fetuses less than 20 weeks old. Again the selection process can be replicated. Our final dataset includes 485,905 records in which the mother is Afro-American and 681,600 in which the mother is Caucasian. Two items on the US birth certificates record whether the mother reports smoking during pregnancy and, if she smokes, the number of cigarettes smoked per day.

Dependent variable

It is assumed that each individual is born with a certain initial endowment of health that is not directly observed. A common measure of the stock of health at birth is birthweight. To allow

for the non-linearity between birthweight and well-being, we used a dichotomous variable to identify LBW infants. Although a birthweight of 2500 grams does not represent specific biological categories, empirical studies show that this reference does well in identifying infants with high risks of mortality and morbidity [See Institute of Medicine (1985)]. The clinical and epidemiological literature on birth outcomes has shown that the health production functions of Afro-American and Caucasians should be separately estimated. [See Corman, Joyce & Grossman (1987), Liu (1988), and Frank, Jackson, Salkever & Strobino (1992)].

Smoking variables

The smoking participation decision is naturally coded as a binary variable equal to "1" if mother reports that she has smoked. The distribution of cigarette consumption has focal answers (10, 20, 40) recognized in the medical literature as different levels of addiction and health risk. Therefore we create a polycothomous variable which aggregates smokers by the quantities consumed: the variable assumes the value "0" when the mother reports no consumption, "1" if she reports *light* consumption (less than 10 cigarettes a day), "2" if she reports *moderate* consumption (10 or more and less than 20 cigarettes a day) and "3" if she reports *heavy* consumption (20 or more cigarettes a day).

Empirical results

Data on smoking behavior by race is reported in table 1. As can be seen, nearly 17.6% of Caucasian women and 10.8% of Afro-American women self reported smoking during pregnancy. The majority of mothers who continue smoking during pregnancy are moderate consumers.

Table 2 presents descriptive statistics for the sample Caucasian mothers and Table 3 for the sample of Afro-American mothers. The data suggest that smokers and non-smokers tend to differ with respect to their observable characteristics. As other have shown, mothers who smoke during pregnancy tend to be less educated, more likely to be unmarried, start prenatal care later, as well as gain significantly less weight during pregnancy. Moreover, Caucasian mothers who smoke during pregnancy are younger than their peers, while Afro-American smokers tend to be older.

Benchmark results

We estimated the hybrid and reduced form models with standard regression methods, by race. The independent variables include demographic variables, health conditions and state dummy variables. To the extent that the correlation between smoking and birth outcomes is causal, the estimated coefficient should not change much when controlling for additional pre-existing characteristic. We also report the *odds-ratio* of smoking and *population risks attributable to smoking* (*PRAS*) estimated by logistic regressions.

Parametric estimates for dichotomous treatment (smoking, no smoking) on birthweight are presented in Table 4 and Table 5. The results support previous findings that smoking has a deleterious association with health stock at birth. The estimated birthweight deficit associated with maternal smoking ranges from 200 to 280 grams, which falls close to the mean of the interval of the previous epidemiological estimates [see Walsh (1994)]. As expected, after controlling for the mother's demographic characteristics and for the level of prenatal care received, the impact of smoking decreases. The estimates are stable among the other regressions. The consistency of the results suggests that the smoking impact is causal and increases the risks independently of other key determinants of birth outcomes. A similar convergence to previous studies arises in our LBW infant estimates: the likelihood of a LBW delivery doubles among mothers who reported smoking during pregnancy (Table 6 and Table 7). Maternal smoking during pregnancy appears to be responsible for around 8% of LBW among Afro-Americans and 14% among Caucasians. Again the impact of smoking is stable across specifications.

Table 8 presents the estimates for birthweight related to smoking intensity. Tables 9 and 10 report the estimates for low birthweight in dichotomous form related to smoking intensity. . In this case we only report the results of model 3 (as defined in Table 4), for simplicity purposes. The results suggest that an increasing and strong monotonic dose relationship emerges for birthweight. Nonetheless, the dose relationship is not linear. Instead, the deleterious effects of smoking on at-birth outcomes start occurring at very low baseline consumption, which raises suspicions of behavioral influences.

Propensity score results

We selected the co-variates in the propensity score method to satisfy the balance property, which asserts that smoking participation and the observed co-variates are conditionally independent, given the propensity score. The propensity score is a function of variables in the single parametric regressions (Model 3), except infant sex. We additionally control for prices of

cigarettes when the mother was teenager (average price of cigarettes and income per-capita during the period the mother was 15 years old to 19 years old), and interaction effects between marital status and number of children, education and age. We include these additional variables to balance the scores and following the recommendation of Heckman *et al* (1998) that consider the gains of efficiency when there are variables that affect the propensity score but can be excluded from the second stage. The propensity score is naturally bounded between zero and one and was estimated using a standard Probit model. (Results upon to request).

Matching estimator for binary treatment

To identify the appropriate matches, we alternatively set the cut-off for similar probability at 10% and 5% in predicting the likelihood of being a smoker. The alternative cut-off values did not appreciably change the results. Because matching performance relies on closeness of the propensity scores, we report results for those with propensity scores that differ by less than 5%. Observations for which the estimated marginal probabilities were larger than the maximum of the corresponding probability in the counterpart group were excluded. The reverse holds for minima.

From our large set of Caucasian mothers, only 10284 of mothers who smoke were matched with 7258 non-smokers. The average number of times that a non-smoker in the control group was matched is 1.4, but some observations are heavily used. [The maximum number of replacements is 14]. For Afro-Americans, 4469 smokers were matched with 2770 non-smokers. The average number of times that a non-smoker was matched is 1.3. Again some observations are heavily used [maximum number of replacement is 21 times], which may result in an inflation of the variance.

Table 11 reports the mean impact and the variance of smoking participation on birth outcomes, based on the difference between matched observations, providing evidence that smoking has a negative impact on birthweight and increases the risks for LBW. Furthermore, these results are similar to results from the parametric methods. Nonetheless the results for the Afro-American sample suggest that our one equation parametric models slightly overestimate the effect on birthweight, as well as the risk of low birthweight.

Matching estimator for multi-treatment

We use an ordered Probit to obtain $[\hat{P}^0, \hat{P}^1, ..\hat{P}^3]$, with the same covariates used in the bivariate propensity score earlier. Pair-wise matches are based on the *Mahalonobis* distance.

Again, matching is done allowing for replacement. To ensure common support we delete all observations with probabilities larger than the smallest maximum and smaller than the largest minimum of all intensity levels.

Tables 12 and 13 report our estimates for mean differences in the birth outcome, given intensity of consumption, with reference to the non-smoking level. The results for dose-response suggest that there is a negative effect on birth outcome by going from light to heavy consumption. The effects are already present at low levels of consumption, confirming that the deleterious effect of smoking is likely to start at low levels of consumption. As with the binary treatment, the results suggest that parametric models overestimate the impact of smoking participation for the Afro-American sample. The difference is very small for low levels of consumption but it increases for heavy smokers. Nonetheless, the number of matched observations for heavy smokers is small for Afro-Americans and therefore we should be cautious in deriving any conclusion.

For the Caucasian sample, the matched results are again very similar to the parametric estimation, although among heavy smokers the results suggest that the parametric model may slightly underestimate the negative effects of smoking for heavy smokers. We can conjecture that Caucasian mothers who smoke heavily may try to compensate for the effect of smoking with other non-observable behaviors.

Unconfoundedness assumption

In this section we focus on the validity of the *unconfoundedness* assumption. The validity of the *unconfoundedness* assumption implies that the group of matched smokers does not differ from the group of matched nonsmokers in the variables that are associated to smoking participation. We tested the hypothesis at different levels of propensity score. Our results suggest that matched smokers and non-smokers have indeed similar distributions of observable variables. We grouped the observations into strata defined on the estimated propensity score and checked whether the covariates were balanced across the smoking and non-smoking sub-populations within each stratum. The usual tests for the statistical significance of the differences in the first and second moments of the distribution were performed. The means of the main variables, conditional on the propensity score are not significantly different in terms of the attributes. These results are impractical to report here but are available upon request.

The *unconfoundedness* assumption also requires that conditional on observed variables smoking participation is random, and thus independent of the potential outcomes. This means that relevant differences between any two groups are captured by observed variables. We use the

Sagan-Wu–Hausman type test to investigate whether there is bias due to lack of controls for unobservable differences between smokers and non-smokers. The test is only valid when each smoker is matched with only one non-smoker, because of the complexity of weighting. Therefore, we restricted the sample to observations matched only once. One vector of control variables (**Z**) is introduced to test the equations for birth outcomes. The identification of a linear model requires that there is at least one variable in **Z** that is not in **X.** Because of the non-linearity of the propensity score in **X** that condition is not essential. Nonetheless, we identify the regression by including the infant sex variable in Z. We estimate

$BO = \alpha + \beta S + \gamma \mathbf{R} + \rho \mathbf{Z} + \nu$; where **R** denotes the residual from the smoking participation model.

With the exception of the birthweight equation for Afro-Americans, we cannot reject the null that $\gamma = 0$ which suggests that the *unconfundness* assumption may be reasonable at least for the Caucasian sample.

Conclusions

Our main goal was to investigate the causal impact of smoking on birth outcomes. In this paper, we have utilized a method for estimating the treatment effect of smoking on birth outcomes in the presence of non-random assignment with propensity score matching. Our results strengthen the evidence that cigarette smoking during pregnancy has a significant causal impact on the health of infants at birth. We conclude that OLS estimates and Probit estimates perform empirically well in estimating the birth outcome production function, in terms of measuring the effects of tobacco. Several pieces of evidence support our conclusions. First, parametric regressions are strongly robust. This indicates that the smoking effect is not mediated by observable variables. Second, the results of OLS and Matching estimators are similar. Although our results suggest that Afro-American mothers who continue smoking during pregnancy may accumulate other undesirable health conditions or health behaviors, the results of matching estimation are not dramatically different from those of the parametric regressions.

The deleterious causal effect of smoking starts at low levels of consumption. This result suggests that the benefits of reducing smoking during pregnancy are significantly higher to mothers who achieve total cessation. Public policy messages should preferentially address the goal of zero consumption.

Our conclusions must be tempered by several factors. First there are several other

methodological problems influencing the validity of the results such as measurement errors in self-reported smoking habits and sample-selection. Second, a better specification of birth outcomes, with more refined data in particular on smoking behaviors, per-capita income, health insurance, and other substance abuse may also permit a better interpretation of coefficients and help to clarify the causality relationship.

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Table 1 -Tobacco consumption of pregnant women by race

	Afro-American sample	Caucasian Sample
% Smokers	10.80	17.60
	(31.01)	(38.00)
Intensity of smoking		
% Lighter smokers	5.1	4.8
	(22.04)	(21.24)
% Moderate smokers	4.9	11.4
	(21.50)	(31.75)
% Heavy smokers	0.3	0.8
	(5.22)	(9.07)

(Standard deviation in parentheses)

Table 2 - Descriptive statistics of independent variables by smoking status for Caucasian Sample

	Non smokers	Smoke	Light smokers	Moderat smokers	Heav smoke
Demographic variables					
Mother age (average)	27.81	25.57	24.82	25.75	26.93
	(5.82)	(5.94)	(5.97)	(5.85)	(6.23
% Adolescent	8.89	16.82	21.86	15.12	10.25
	(28.46)	(37.40	(41.33)	(35.83)	(30.36
% Adult	89.25	82.00	77.28	83.67	87.32
	(30.97)	(38.42	(41.90)	(36.96)	(33.28
% Older	1.85	1.18	0.85	1.20	2.43
	(13.47)	(10.78	(9.33)	(10.91)	(15.39
% Married	83.81	55.55	55.89	55.36	63.88
	(36.83)	(49.70	(49.65)	(49.71)	(48.03
Mother years of education	13.63	11.78	12.01	11.71	12.18
	(2.23)	(1.82)	(1.89)	(1.77)	(2.27
% W. High School education	57.96	21.81	26.17	20.35	15.95
	(49.36)	(41.30	(43.96)	(40.26)	(36.62
Obstetric History					
Number of live births	1.89	2.06	1.81	2.13	2.16
	(1.09)	(1.15)	(1.00)	(1.16)	(1.29
% First baby	45.20	40.33	45.34	37.07	24.82
	(49.79)	(49.05	(49.78)	(48.30)	(43.20
% Low parity	46.99	48.34	46.14	50.61	53.39
	(49.99)	(49.97	(49.85)	(49.99)	(49.89
% High parity	7.81	11.31	8.49	12.38	21.78
	(26.83)	(31.68	(27.87)	(32.87)	(41.28
% Previous preterm babie	0.95	1.95	1.17	2.18	2.62
	(9.74)	(13.79	(10.77)	(14.62)	(15.99
% Previous death	25.61	33.43	30.93	34.44	32.99
	(43.64)	(47.17	(46.22)	(47.52)	(47.02
Medical Conditions					
Weight Gain	0.79	0.78	0.83	0.77	0.743
	(0.31)	(0.35)	(0.35)	(0.35)	(0.364
%At least one health risk	23.77	28.05	27.02	27.97	32.99
	(42.57)	(44.93	(44.41)	(44.88)	(47.02
Prenatal care(1)					
%Inadequate	6.39	14.76	13.26	15.03	19.38
	(24.45)	(35.40	(33.86)	(35.73)	(39.53
%Intermediate	13.26	14.02	14.61	13.78	13.35
	(33.91)	(34.72	(35.32)	(34.46)	(34.0
%Adequate	49.32	40.63	41.94	40.45	37.97
	(49.99)	(49.13	(49.34)	(49.08)	(48.53
%Adequate Plus	29.67	28.99	29.15	29.11	26.85
	(45.69)	(45.39	(45.44)	(45.42)	(44.32

(Standard deviation in parentheses)

Table 3 - Descriptive statistics of independent variables by smoking status for Afro-American Sample

	Non smoke	Smoker	Light smokers	Moderate smokers	Heavy smol
Mother age (average)	24.18	27.13	26.22	27.99	27.99
	(6.14)	(6.13)	(6.15)	(5.97)	(5.96)
% Adolescent	26.16	11.54	19.93	7.98	6.59
	(943.95)	(31.94)	(35.64)	(27.10)	(24.80)
% Adult	72.70	86.85	83.73	90.06	91.48
	(44.55)	(33.79)	(36.90)	(29.91)	(27.92)
% Older	1.14	1.61	1.33	1.95	1.93
	(10.60)	(12.60)	(11.46)	(13.85)	(13.76)
% Married	30.67	40.98	16.34	17.92	18.28
	(46.11)	(49.18)	(36.98)	(38.35)	(38.70)
Mother years of education	12.23	11.55	11.58	11.54	11.18
	(2.12)	(1.68)	(1.70)	(1.66)	(1.57)
% High School education	32.47	17.21	18.08	16.60	9.74
	(46.82)	(37.74)	(38.49)	(37.21)	(29.69)
Obstetric History					
Number of live births	2.05	3.09	2.86	3.30	3.78
	(1.30)	(1.80)	(1.68)	(1.87)	(2.07)
% First baby	46.78	20.78	25.00	16.96	10.97
	(49.89)	(40.58)	(43.30)	(37.52)	(31.26)
% Low parity	39.61	41.06	42.55	41.15	35.38
	(48.90)	(49.20)	(49.44)	(49.21)	(47.81)
% High parity	13.60	38.16	32.44	41.88	53.64
	(34.28)	(48.58)	(46.81)	(49.43)	(49.87)
% Previous preterm babi	1.25	3.23	2.88	3.34	6.06
	(11.11)	(17.68)	(16.72)	(17.97)	(23.85)
% Previous death	26.94	41.97	40.62	43.06	44.79
	(45.25)	(49.35)	(49.11)	(49.51)	(49.73)
Medical Conditions					
Weight Gain	0.75	0.70	0.72	0.69	0.66
	(0.35)	(0.37)	(0.37)	(37.46)	(0.40)
% At least one health ris	28.73	41.19	38.94	42.06	50.69
	(45.25)	(49.22)	(48.76)	(49.36)	(49.99)
Prenatal care (1)					
% Inadequate	19.34	34.69	32.03	36.44	45.43
	(39.49)	(47.56)	(46.67)	(48.12)	(49.86)
% Intermediate	12.86	12.61	12.78	12.75	12.46
	(33.48)	(33.25)	(33.38)	(33.35)	(33.08)
%Adequate	33.61	23.91	26.63	22.50	15.80
	(47.23)	(42.65)	(44.20)	(41.76)	(36.51)
%Adequate Plus	31.05	24.68	25.44	24.10	19.39
	(46.27)	(43.12)	(43.56)	(42.77)	(39.60)

(Standard deviation in parentheses)

(a) Using Kotelchuck Adequacy of Prenatal Care Utilization (APNCU) Index [Kotelchuck (1994)].

Table 4- Summary of birthweight regressions for Caucasian sample. Smoking participation coefficient

	Model 1 (a)	Model 2(b)	Model 3(c)	Model 4(d)
Smoking participation	-274.26 (1.89)	-233.20 (2.26)	-231.52 (1.52)	-234.01 (2.03)
Adjusted R^2	0.031	0.090	0.123	0.128
# observations	674828	651199	572708	572708

(Robust standard deviation in parentheses)

(a)Model 1 - Without controls
(b) Model 2 – Controls for marital status, parity level, age, age squared, dummy variables for level of prenatal care received
(c)Model 3 – Controls for Model 2 variables + dummies for chronic health conditions (diabetes, renal diseases, cardiac problems, lung problems as well as herpes) logarithm of maternal weight gain during the pregnancy
(d) Model 4 – Controls for Model 3 + state dummy variables.

Table 5- Summary of birthweight regressions for Afro-American sample. Smoking participation coefficient

	Model 1	Model 2	Model 3	Model 4
Smoking participation	-250.46 (3.23)	-231.63 (3.36)	-215.66 (3.49)	-225.20 (3.51)
Adjusted R^2	0.013	0.059	0.066	0.070
# observations	481048	455678	379989	379759

Table 6- Summary of LBW regressions for Caucasian sample. Smoking participation coefficient

	Model 1	Model 2	Model 3	Model 4
Probit coefficient	0.340	0.334	0.341	0.346
	(0.006)	(0.007)	(0.007)	(0.008)
Marginal effect	0.052	0.036	0.033	0.034
	(0.001)	(0.009)	(0.001)	(0.001)
Odds ratio	2.281	1.946	1.972	1.993
	(0.026)	(0.026)	(0.030)	(0.030)
PRAS	0.169	0.142	0.146	0.148
	(0.028)	(0.003)	(0.003)	(0.003)
Pseudo R^2	0.016	0.084	0.106	0.110
# observations	674828	651199	572708	573047

(Robust standard deviation in parentheses)

Table 7- Summary of LBW regressions for Afro-American sample. Smoking participation coefficient

	Model 1	Model 2	Model 3	Model 4
Probit coefficien	0.433	0.397	0.386	0.406
	(0.007)	(0.007)	(0.008)	(0.008)
Marginal effect	0.108	0.093	0.084	0.088
	(0.002)	(0.002)	(0.002)	(0.002)
Odds ratio	2.183	2.039	2.013	2.093
	(0.025)	(0.026)	(0.030)	(0.031)
PRAS	0.090	0.082	0.079	0.083
	(0.002)	(0.002)	(0.002)	(0.002)
Pseudo R^2	0.011	0.050	0.059	0.077
# observations	481048	455678	379989	379661

(Robust standard deviation in parentheses)

Table 8- Summary of birthweight regressions. Intensity of consumption

	Afro-American sample	Caucasian sample
Light	-188.41	-180.32
	(4.62)	(3.44)
Moderate	-267.73	-254.70
	(5.08)	(2.39)
Heavy	-344.78	-306.74
	(23.62)	(8.26)
Adjusted R^2	0.099	0.128
# observations	378163	572708
Reset (p-value)	0.000	0.001

*Includes all other variables in Model 3

(Robust standard deviation in parentheses)

Table 9 - Summary of LBW for Caucasian sample. Intensity of consumption

	Light	LBW Moderat	Heavy
Probit Coefficien	0.281	0.370	0.500
	(0.023)	(0.045)	(0.021)
Marginal effects	0.029	0.034	0.054
	(0.002)	(0.001)	(0.004)
Odds ratio	1.787	2.054	2.500
	(0.045)	(0.036)	(0.123)
PRAS	0.032	0.102	0.011
	(0.002)	(0.003)	(0.001)
Pseudo R^2	0.111		
# observations	573047		
Reset	0.497		

*Includes all other variables in Model 3

(Robust standard deviation in parentheses)

Table 10 - Summary of LBW for Afro-American sample. Intensity of consumption

	LBW		
	Light	Moderat	Heavy
Probit Coefficie	0.302	0.453	0.663
	(0.015	(0.020)	(0.051
Marginal effec	0.067	0.107	0.173
	(0.003	(0.003)	(0.015
Odds ratio	1.771	2.305	3.256
	(0.035	(0.046)	(0.254
PRAS	0.031	0.044	0.004
	(0.001	(0.001)	(0.000
Pseudo R^2	0.074		
# observations	37816		
Reset (p-value	0.078		

*Includes all other variables in Model 3

(Robust standard deviation in parentheses)

Table 11 – Propensity score matching estimates

	Caucasian sample	Afro-American sample
Birthweight	-227.40	-186.90
	(27.28)	(19.47)
LBW	0.036	0.065
	(0.004)	(0.010)

(Robust standard deviation in parentheses)

Table 12 - Mean differences in the birth outcome for Caucasian sample. Reference to non-smoking level

	Birthweight	LBW
Light	-190.20	0.031
# smokers 2710	(18.13)	(0.008)
# non-smokers, 2271		
Moderate	-248.04	0.041
# smokers, 6657	(12.09)	(0.005)
#nonsmokers, 4073		
Heavy	-365.53	0.084
# smokers,462	(42.24)	(0.019)
# nonsmokers, 446		

(Robust standard deviation in parentheses)

Table 13– Mean differences in the birth outcomes for Afro-American sample. Reference to non-smoking level

	Birthweight	LBW
Light # smokers 2182 #non-smokers,1914	-174.18 (23.32)	0.062 (0.012)
Moderate # smokers, 1984 #nonsmokers, 1634	-234.98 (26.60)	0.085 (0.014)
Heavy # smokers, 99 # nonsmokers, 97	-222.03 (117.58)	0.111 (0.059)

(Robust standard deviation in parentheses)