# Working Paper Series

## No. 32

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

Paula Veiga

Ronald P. Wilder

January 2006

**Núcleo de Investigação em Microeconomia Aplicada**
**Universidade do Minho**

Fundação para a Ciência e a Tecnologia
MINISTÉRIO DA CIÊNCIA E DA TECNOLOGIA

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

**Paula Veloso da Veiga**

Department of Economics and NIMA, University of Minho

Email: paulav@eeg.uminho.pt

Corresponding author

**Ronald P. Wilder**

Department of Economics, University of South Carolina

Email: wilder@moore.sc.edu

## Abstract

There is accumulated evidence of the existence of a deleterious effect of smoking on birth outcomes. Understanding the effect of smoking on pregnancy is a critical issue because of the public policy implications for dissuading maternal smoking. 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.

Abstract word count: 149

Key words: Smoking, Birth outcomes, Causal effects, Propensity score and matching,

JEL Classification: I12, C12, C21

Full word count: 6748

## 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.

With few exceptions the literature relies on "selection on observables [Heckman and Rob(1985) ] and the standard practice consist of entering characteristics in levels in a linear model such as birthweight regression.. 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.

An ideal framework for assessing the 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 [Rosenzweig & Schultz (1983), Permutt & Hebel (1989), Evans & Ringel (1999) ] and Bayesian treatment models [ Hamilton (2001) ]. Overall, they find a higher impact of smoking on birthweight than previous 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 use the propensity score matching method to estimate the impact of smoking on birth outcomes. 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).

Matching allow us to address some limitations of OLS estimates. First, the linearity assumption of OLS can hide the failure of "common support" conditions Matching methods rely on "common support" assumption. Although it does not solve the support problem, matching methods allow us to get a clear sense of the extent of the problem. Second, 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. 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. The final section presents some concluding remarks

## Methods

### Matching methods

Proposed initially in the evaluation literature, propensity matching allow for correcting the estimation of the treatment effects controlling for the existence of confounding factors that maybe in the origin of biased estimates. Confounding factors may result from the fact that assignment in treatment and control groups is not random. Using the terminology in the evaluation literature we were interested in evaluating the 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: $E\,[BO_1 - BO_0 \mid S{=}1] = E\,[BO_1 \mid S{=}1] - E\,[BO_0 \mid S{=}1]$. 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: $E[BO_1 \mid S{=}1] - E[BO_0 \mid S{=}0] = E[BO_1 - BO_0 \mid S{=}1] + \{E[BO_1 \mid S{=}1] - E[BO_1 \mid S{=}0]\}$

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. Any standard probability model can be used to estimate it. 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 $E[B0^1 - B0^0 \mid S = l]$, 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.

### 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.

## Conclusions

Our main goal was to investigate the 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 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, as similar.

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. Nonetheless, the result leaves scope for behavioral explanations suggesting that smokers may indeed be self-selected group among pregnant woman.

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.

## References

Adams, E., & Melvin, C. (1998). Costs of Maternal Conditions Attributable to Smoking During Pregnancy, *American Journal of Preventive Medicine*, October, 15(3), 212-219.

Angrist, J., & Hahn, J. (1999). When to Control for Covariate Panel – Asymptotic Results for Estimates of Treatment Effects, *Technical Working Paper*, 241, National Bureau of Economic Research, Cambridge, MA.

Angrist, J., & Krueger, B. (1999). Empirical Strategies in Labor Economics, in Handbook of Labor Economics, O. Ashenfelter & D. Card (eds.). Vol. III A., Chapter 23, 1277-1366.

Angrist, J., Imbens, G., & Rubin, D. (1996). Identification of Causal Effects Using Instrumental Variables, *Journal of American Statistical Association,* 91(434), 444-455.

Butler, N., Goldstein, H. & Ross M. (1972). Cigarette Smoking in Pregnancy: Its Influence on Birthweight and Perinatal Mortality, *British Medical Journal*, April*,* 2(806), 127-130.

Corman, H., Joyce, T., & Grossman, M. *(*1987). Birth Outcome Production Function in the United States, *The Journal of Human Resources*, 22, 322-360.

Dehejia, R., & Wabha, S. (2002). Propensity Score Matching methods for Non-experimental Causal Studies, *The Review of Economics & Statistics*, February 48(1), 151-161

Dehejia, R., & Wahba, S. (1999). Causal Effects in a Non-Experimental Studies: Re-Evaluation The Evaluation of Training Programs, *Journal of the American Statistical Association*, 94, 1053-1062.

Evans, W., & Ringel, J. (1999). Can Higher Cigarette Taxes Improve Birth Outcomes? *Journal of Public Economics*, April*,* 72(1), 135-154.

Frank, R., Jackson, C., Salkever, D., & Strobino, D. (1992). Update Estimates of the Impact of Prenatal Care on Birth Weight Outcomes by Race, *Journal of Human Resources*, 27(4), 629-642.

Hamilton, B. (2001). Estimating Treatment Effects in Randomized Clinical Trials With Non-Compliance: the Impact of Maternal Smoking on Birthweight, *Health Economics*, July*,* 10 (5), 399-410.

Heckman, J. (1995). Instrumental Variables: a Cautionary Tale, *Technical Working Paper,* September, 85, National Bureau of Economic Research, Cambridge, MA.

Heckman, J. (1998). Instrumental Variables: Study of Implicit Behavioral Assumptions Used in Making Program Evaluations, *Journal of Human Resources*, 32(3), 441-462.

Heckman, J., & Robb, R. (1995). Alternative Methods For Evaluating The Impact of Interventions, in Longitudinal Analysis of Labor Market Data, J. Heckman, & B. Singer (eds), 156-246. Cambridge: Cambridge University Press.

Heckman, J., Ichimura, H., & Todd, P. (1998). Matching as an Econometric Evaluation Estimator, *Review of Economic Studies*, 65, 265-294.

Heckman, J., Lalonde, R., & Smith, J. (1999). The Economics & Econometrics of Active Labour Market Programs, in Handbook of Labor Economics, O. Ashenfelter & D. Card (eds). Vol. III A., Chapter 31, 1865-2097. Available at http, //lily.src.uchicago.edu/papers/papers.html

Hickey, R., Clelland, R. & Bowers, E. (1978). Maternal Smoking, Birthweight, Infant Death, & the Self-selection Problem, *American Journal of Obstetrics & Gynecology*, 131, 805-811.

Imbens, G. (2000). The Role of the Propensity Score in Estimating Dose Response Functions, *Biometrika*, 87(3), 706-710.

Institute of Medicine (1985). Committee to Study the Prevention of Low Birthweight. Preventing Low Birthweight, Washington D.C, National Academy Press.

Kotelchuck, M. (1994). The Adequacy of Prenatal Care Utilization Index, its US Distribution & Association with Low Birthweight, *Journal of Public Health,* 84(9), 1489-1468.

Kramer, M. (1987). Determinants of Low Birthweight, Methodological Assessment & Meta-Analysis, *Bulletin of the World Wealth Organization*, 65(5), 663-733.

Lechner, M. (1999). Identification & Estimation of Causal Effects of Multiple Treatments Under the Conditional Independence Assumption, Discussion Paper 9908, University of St. Gallen. Unpublished manuscript.

Liu, G. (1998). Birth Outcomes and Effectiveness of Prenatal Care, Health Services Research, 1998, 32(6) 805-823.

Permutt, T., & Hebel J. (1989). Simultaneous Equation Estimation in a Clinical Trial of the Effect of Smoking on Birthweight, *Biometrics*, June, 45(2), 619-622.

Rosenbaum, P., & Rubin, D. (1983). The Central Role of the Propensity Score in Observational Studies for Causal Effects, *Biometrika*, 70, 41-55.

Rosenzweig, M., & Schultz, T. (1983). Estimating a Household Production Function, Heterogeneity, the Demand for Health Inputs & their Effects on Birth Weight, *Journal of Political Economy*, 91, 723-746.

Rubin, D. (1977). Assignment to a Treatment Group on the Basis of a Covariate, *Journal of Educational Statistics*, 2, 1-26.

Sexton, M., & Hebel, J. (1984). A Clinical Trial of Change in Maternal Smoking & its Effects on Birth Weight, *Journal American Medical Association*, February, 251 (7), 911- 915.

Sianesi, B. (2001). Implementing Propensity Score Matching Estimators With STATA, Prepared for UK Stata Users Group, VII Meeting. London, May.

Silverman, D. (1977). Maternal Smoking and Birthweight, *Journal of Epidemiology,* June, 105(6), 513-521.

Sowards, K. (1999). What is the Leading Cause of Infant Mortality? A Note on the Interpretation of Official Statistics, I, November, 89(11), 1752-1754.

Tobacco Institute, *The Tax Burden on Tobacco* (Washington, DC, The Tobacco Institute, Historical Compilation #30, 1995). Available at http, //www.tobaccoinstitute.com.

Walsh, R. (1994). Effects of Maternal Smoking on the Adverse Pregnancy Outcomes, Examination of the Criteria Causation, *Human Biology*, December, 66(6), 1059-1092.

Yerushalmy, J. (1971). The Relationship of Parents' Cigarette Smoking to Outcome of Pregnancy. Implication as to the Problem of Inferring Causation from Observed Associations, *Journal of Epidemiology,* 93(6), 443- 446.

**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 ri | 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<br>(1.89) | -233.20<br>(2.26) | -231.52<br>(1.52) | -234.01<br>(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 | -231.63 | -215.66 | -225.20 |
| | (3.23) | (3.36) | (3.49) | (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**

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

(Robust standard deviation in parentheses)

Working Papers - NIMA series

No.

1. Lígia Pinto , Glenn Harrison, *Multilateral negotiations over climate change policy*, May 2000

2. Paulo Guimarães, Douglas Woodward, Octávio Figueiredo, *A tractable approach to the firm location decision problem*, May 2000

3. Miguel Portela , *Measuring skill: a multi-dimensional index*, September 2000

4. Rosa Branca Esteves , Paulo Guimarães, *Price discrimination and targeted advertising: a welfare analysis*, November 2000

5. Anabela Botelho , Lígia Pinto , *Has Portugal gone wireless? Looking back, looking ahead*, December 2000

6. Pedro Barros, Clara Dismuke , *Hospital production in a national health service: the physician's dilemma*, December 2000

7. Anabela Botelho , Mark A. Hirsch, Elisabet E. Rutström, *Culture, nationality and demographics in ultimatum games*, December 2000

8. Miguel Portela , *The impact of segregation on wage inequality: a look at recruitment and pay policies at the firm level*, January 2001

9. Pedro Portugal, Ana Rute Cardoso, *Disentangling the minimum wage puzzle: an analysis of job accessions and separations from a longitudinal matched employer-employee data set*, April 2001

10. Ana Rute Cardoso, Priscila Ferreira , *The dynamics of job creation and destruction for University graduates: why a rising unemployment rate can be misleading*, May 2001

11. Octávio Figueiredo, Paulo Guimarães, Douglas Woodward, *Asymmetric information and location*, July 2001

12. Anabela Botelho , Lígia Pinto , *Hypothetical, real, and predicted real willingness to pay in open-ended surveys: experimental results*, September 2001

13. Anabela Botelho , Lígia Pinto , Miguel Portela , António Silva, *The determinants of success in university entrance*, September 2001

14. Anabela Botelho , *Strategic behavior at trial. The production, reporting, and evaluation of complex evidence*, September 2001

15. Paulo Guimarães, *The state of Portuguese research in economics: an analysis based on publications in international journals*, September 2001

16. Anabela Botelho , Glenn Harrison, Marc Hirsch, Elisabet E. Rutström, *Bargaining behavior, demographics and nationality: a reconsideration of the experimental evidence*, December 2001

17. João Cerejeira da Silva , *Identification of the Portuguese industrial districts*, February 2002

18. Octávio Figueiredo, Paulo Guimarães, Douglas Woodward, *Modeling industrial location decisions in U.S. Counties*, April 2002

19. Aslan Zorlu , Joop Hartog, *The effect of immigration on wages in three European countries*, October 2002

20. Elvira Lima , David K. Whynes, *Finance and performance of Portuguese hospitals*, February 2003

21. Aslan Zorlu , *Do ethnicity and sex matter in pay? Analyses of 8 ethnic groups in the Dutch labour market*, June 2003

22. Cécile Wetzels , Aslan Zorlu , *Wage effects of motherhood: a double selection approach*, June 2003

23. Natália Barbosa , *What drives new firms into an industry? An integrative model of entry*, October 2003

24. Elvira Lima , Teresa Josefina Lopes Esquerdo , *The economic costs of alcohol misuse in Portugal*, October 2003

25. Anabela Botelho , Lígia Pinto , Isabel Rodrigues , *How to comply with environmental regulations?The role of information*, October 2003

26. Natália Barbosa , Helen Louri, *Corporate performance: does ownership matter? A comparison of foreign - and domestic - owned firms in Greece and Portugal*, October 2003

27. Anabela Botelho , Lígia Pinto , *Students' expectations of the economic returns to college education. Results of controlled experiment*, December 2003

28. Paula Veiga , *Income-related health inequality in Portugal*, July 2005

29. Anabela Botelho , Glenn Harrison, Lígia Pinto , Elisabet E. Rutström, *Testing static game theory with dynamic experiments: a case study of public goods*, November 2005

30. Anabela Botelho , Glenn Harrison, Lígia Pinto , Elisabet E. Rutström, *Social norms and social choice*, November 2005

31. Anabela Botelho , Glenn Harrison, Lígia Pinto , Elisabet E. Rutström, Paula Veiga , *Discounting in developing countries: a pilot experiment in Timor-Leste*, November 2005

32. Paula Veiga , Ronald P. Wilder , *Maternal smoking during pregnancy and birthweight - A propensity score matching approach*, January 2006

---

The Working Papers of the Applied Microeconomics Research Unit (NIMA) can be downloaded in PDF format from http://nima.eeg.uminho.pt

---