Published in final edited form as:
Econ Hum Biol. 2009 March ; 7(1): 84–95. doi:10.1016/j.ehb.2008.10.001.

Prenatal Care Demand and its Effects on Birth Outcomes by Birth Defect Status in Argentina

George L. Wehby, Ph.D. [Assistant Professor],
Dept. of Health Management and Policy, University of Iowa, E204, GH, Iowa City, IA 52242, Phone: 319- 384-5133, Fax : 319-384-5125, email: george-wehby@uiowa.edu

Jeffrey C. Murray, MD [Professor],
University of Iowa, College of Medicine, Department of Pediatrics, Iowa City, IA, USA

Eduardo E. Castilla, MD, PhD [Professor],
ECLAMC (Latin American Collaborative Study of Congenital Malformations) at CEMIC, Buenos Aires, Argentina, ECLAMC at Instituto Oswaldo Cruz, Rio de Janeiro, Brazil

Jorge S. Lopez-Camelo, PhD [Professor], and
ECLAMC at Imbice, La Plata, Argentina

Robert L. Ohsfeldt, PhD [Professor]
Health Policy & Management, School of Rural Public Health, Texas A&M Health Science Center, College Station, TX, USA

Abstract

Our objective was to identify determinants of prenatal care demand and evaluate the effects of this demand on low birth weight and preterm birth. Delay in initiating prenatal care was modeled as a function of pregnancy risk indicators, enabling factors, and regional characteristics. Conditional maximum likelihood (CML) estimation was used to model self-selection into prenatal care use when estimating its effectiveness. Birth registry data was collected post delivery on infants with and without common birth defects born in 1995-2002 in Argentina using a standard procedure. Several maternal health and fertility indicators had significant effects on prenatal care use. In the group without birth defects, prenatal care delay increased significantly LBW and preterm birth when accounting for self-selection using the CML model but not in the standard probit model. Prenatal care was found to be ineffective on average in the birth defect group. The self-selection of higher risk women into earlier initiation of prenatal care resulted in underestimation of prenatal care effectiveness when using a standard probit model with several covariates. Large improvements in birth outcomes are suggested with earlier initiation of prenatal care for pregnancies uncomplicated with birth defects in Argentina, implying large opportunity costs from the long waiting time observed in this sample (about 17 weeks on average). The suggested ineffectiveness for pregnancies complicated with common birth defects deserves further research.

Improvement in health care over the past several decades has contributed to a substantial increase in the likelihood of survival of infants born prematurely and/or at low birth weight (LBW) as well as those born with birth defects and disabilities. Preterm birth and LBW have been widely associated with an increased risk for delayed development, physical disabilities and reduced life expectancy. Child disability imposes a substantial burden on the affected child, family and community. Further, LBW has recently been shown to be related to lower human capital accumulation including education and wealth and lower maternal birth weight has also

Correspondence to: George L. Wehby.

been related to lower birth weight in the offspring in a sample of low and middle income countries (Victora et al, 2008).

A 7% LBW rate has been reported in Argentina in 1998 (Kramer et al. 2005). The rates of preterm birth and LBW have been increasing in the past two decades in several countries including by about 28% and 16% respectively in the US (Arias et al. 2003)[1]. The rates of LBW and preterm birth have been estimated to have increased over the past decade in Argentina.[2]

The increasing prevalence of preterm and LBW births has generally been parallel to increases in multiple births and maternal age, yet the main causes of these outcomes remain unknown. LBW can occur due to shorter gestational age at birth and/or restricted uterine fetal growth. About 16% (20 million) of babies worldwide are born at LBW, with overall occurrence in developing countries being more than double of that in developed countries (16 versus 7%) (Lawn, Cousens, and Zupan 2005). Reliable estimates of the prevalence of preterm birth are less available for developing countries. Up to 28% of the 4 million neonatal deaths worldwide are directly related to preterm birth, and up to 80% occur among LBW infants.

Prenatal care use has generally been associated with some improvements in birth outcomes yet studies for less developed countries have been limited. This study augments this body of research using data from Argentina to evaluate the effects of risk indicators on prenatal care utilization, estimate the effects of prenatal care utilization on LBW and preterm birth in subgroups of normal versus abnormal births and explore the effects of other inputs and risk factors including multivitamin use, immunization and maternal health on these outcomes. Given that children born with birth defects are generally at a higher risk for preterm birth and LBW, the study assesses the effectiveness of prenatal care utilization in subgroups of infants born with selected common birth defects and normal infants to check for any heterogeneous impacts of prenatal care.

PRENATAL CARE EFFECTIVENESS

There have been several studies of the effects of prenatal care use on birth outcomes. A meta-analysis (Carroli et al. 2001) of seven randomized clinical trials (RCTs) found no significant differences in LBW occurrence between standard and reduced prenatal visit models (generally consistent with the results reported in each trial)[3]. Such results led to conclusions that increasing prenatal care may not be an effective intervention for improving perinatal outcomes in developing countries (Bhutta et al. 2005). However, this may not hold true given the rarity of such studies in developing countries and that results on more developed countries may not be very applicable to less developed ones due to country differences in the rates of adverse birth outcomes, intensity of prenatal care use, quality of prenatal care, as well as the prevalence and type of risk factors that may modify the effects of prenatal care (such as maternal health risks and socioeconomic factors). Further, the RCTs mentioned above studied the effects of changes in number of visits but not in timing of prenatal care initiation, which may be a more relevant aspect of prenatal care use for fetal health.

[1]In the US, the rates of LBW and preterm birth in 2002 were 13.3% and 17.5% respectively among African-American Infants compared to 6.9% and 11% respectively among White infants, and 6.5% and 11.6% respectively among Hispanic infants (Arias et al. 2003). In Brazil, about 11.8% and 16.2% of a representative sample of births in 2004 were born at LBW and preterm respectively (Barros et al, 2005).

[2]Kramer et al. (2005) reported a slight increase of 1.4% in the LBW rate between 1989 and 1998. Data from the Department of Statistics and Health Information of the Ministry of Health in Argentina suggest that the LBW and preterm birth rates might have increased over the past few years but these data should be viewed with caution since 2.8% and 15% of reported births had unspecified birth weight and gestational age respectively.

[3]Four of these studies were in developed countries and three were in less developed countries.

Several econometric studies using birth registry and survey data have been also conducted. These studies highlighted the analytical complications due to maternal self-selection of prenatal care. Pregnant women may have some expectations of their pregnancy outcomes, based on their health status, pregnancy complications, pregnancy history and so on, and these expectations may affect their prenatal care demand. Women who perceive a larger risk for the ongoing pregnancy due for instance to a bad pregnancy history (e.g. prior low birth weight infant or spontaneous abortion) are expected to demand more prenatal care than those who perceive a lower risk, yet the indicators (e.g. pregnancy history) contributing to these risk perceptions may themselves be correlated with the health outcomes of the expected infant. For instance, having a previous low birth weight infant may predispose a future pregnancy to result in an underweight birth. Adverse self-selection (Rosenzweig and Schultz 1982, 1983) entails opposite effects of perceived maternal risks on prenatal care demand (higher risk increases demand) and infant health (higher risk deteriorates infant health). Unfortunately, perceived risks are inadequately observed in typically available data as they may be related to many biologic and psychosocial indicators that might signal to the mother potential fetal health risks. Given that women who demand more prenatal care in this context have a higher propensity for adverse infant outcomes, the effectiveness of prenatal care use in producing infant health when estimated by classical regression models may be biased downward. On the other hand, prenatal care use is likely to be correlated with other health behaviors given that they are in part determined by the same set of individual preferences for health and for risk tolerance. For instance, women who are more risk averse and who value health more are expected to adopt healthier lifestyles (e.g. better nutrition and stress management, less smoking and drinking) than those who are less risk averse and value health less. The first group of women is expected to both demand more prenatal care and have larger propensities for healthy infants than the latter group. Given that health preferences and behaviors are also incompletely observed in typically available data, this type of self-selection is expected to result in overestimation (biased upward) of the effectiveness of prenatal care utilization unlike adverse self-selection, and is typically referred to as favorable self-selection. The net bias depends on the analytical model applied and the extent to which it controls for both these two types of self-selection.

Previous econometric studies have primarily applied an instrumental variable (IV) or sample selection models to identify the effects of endogenous prenatal care use (e.g. Rosenzweig and Schultz 1982, 1983, 1988; Joyce, 1985, 1987, 1994; Warner 1995, 1998; Grossman and Joyce 1990; Liu 1998; Rous, Jewell, and Brown 2004, Evans and Lien 2005). These studies have generally reported modest effects of prenatal care use on birth outcomes with an overall support for adverse self-selection theory. However, the estimates based on US data may not be applicable to less developed countries due to differences in quality of care, in population characteristics and in health care systems. Using instrumental variable quantile regression (Chernozhukov and Hansen, 2004, 2005 and 2006), we found large effects of prenatal care use on low but not high birth weight quantiles in infants without birth defects from Argentina (Wehby et al, 2008). Further, previous econometric studies have generally analyzed random samples of births that have few infants with health complications. Due to potential heterogeneous impacts of health inputs by fetal health endowments, it is important to obtain specific estimates of effectiveness of health inputs by endowment level, which can be enhanced by using datasets that oversample infants with health complications. Applying a finite mixture model, Conway and Deb (2005) reported that prenatal care is ineffective for complicated pregnancies. Another limitation of previous studies is focusing primarily on continuous birth weight measures and excluding clinically relevant measures such as LBW and preterm birth.

This study evaluates those research questions in two birth samples from the third most populated country in South America, Argentina, which has a substantially lower prenatal care utilization rate compared to the US, so that specific inference can be made to guide policy makers as to the potential gains from improvement in prenatal care utilization for two distinct

infant groups with different fetal health endowments. The effects of prenatal care on birth outcomes are estimated separately for infants without birth defects and for infants with the five most common birth defects that occur early on in pregnancy and that have a large genetic etiologic component, allowing the birth defect status to be used as a good proxy indicator for lower health endowments that are exogenous to prenatal care initiation. These were cleft lip and/or palate, neural tube defects, trisomy 21, congenital heart disease and polydactyly.[4] This approach arguably provides a more natural identification of two main groups of infants that are expected to be different in the extent of their health endowment and more directly interpretable and generalizable results than a statistical identification of complicated pregnancies. This estimation approach evaluates if prenatal care impacts the birth outcomes of pregnancies complicated with major birth defects similarly as pregnancies uncomplicated with birth defects and has therefore direct implications for prenatal care delivery. Further, the study focuses on estimating the effects of prenatal care on clinically relevant outcomes including LBW and preterm birth. The study also estimates the effects of previously understudied inputs including multivitamin use, immunizations and maternal health indicators, on LBW and preterm birth. Finally, the demand for prenatal care is studied as a function of several risk indicators and other potentially relevant determinants in order to identify variables that can be modified by policy interventions to improve utilization.

METHODS

Data and Study Sample

The data for this study was obtained from the Latin American Collaborative Study of Congenital Malformations (ECLAMC), which is a WHO Collaborating Center that has been involved in birth defects surveillance and research through most of South America since 1967 (Castilla and Orioli 2004). ECLAMC involves a network of health professionals who continuously monitor births in their hospitals to identify infants with any birth defects.[5] Each newborn with a birth defect in affiliated hospitals is enrolled in the program. An infant born without birth defects is matched to each infant with a birth defect by sex, hospital and date of birth and enrolled. Similar data is collected on both groups. Data are collected through in-person interviews with the parents (mainly mothers) as well as extracted from medical records using a standard instrument. This program presents a unique secondary data source to analyze the proposed research questions in other South American countries as well.

The study sample included 2417 infants with at least one of the five most common birth defects which are cleft lip and/or palate, neural tube defects, trisomy 21, congenital heart disease and polydactyly, and a sample of 2325 infants with no birth defects, all registered between 1995 and 2002 in 34 hospitals.[6] The study sample is limited to singleton live births, with recorded birth weights between 500 and 6000 grams, and gestational age (based on last day of menstrual period) between 19.5 and 46.5 weeks[7]. These restrictions on birth weight and gestational age were used to avoid potential recording errors during data collection or entry. Similar to most previous econometric studies of prenatal care, mothers who are below 20 years of age are also

[4]Other birth defects were not analyzed due to their rarity and substantial heterogeneity in developmental complications.

[5]ECLAMC's headquarters are located at the Instituto Oswaldo Cruz, Rio de Janeiro, Brazil and at the Center of Medical Studies and Clinical Investigations (CEMIC) in Buenos Aires, Argentina (Castilla and Orioli, 2004).

[6]The difference in sample size between the infants with and without birth defects is due to the added exclusion criteria and missing data. Cleft lip and palate are craniofacial anomalies that include the lack of fusion of the lip and/or palate. About 16.9% of the birth defect group had oral clefts. Neural tube defects include defects of the brain and/spinal cord such as spina bifida (lack of closure of the spinal column. About 19.2% had neural tube defects. About 23% of the sample had Trisomy 21. Trisomy 21 indicates Down syndrome. About 23% of the sample had Trisomy 21. Congenital heart disease includes different abnormalities of the heart such as holes in the walls of the heart. About 33.7% had congenital heart disease. Polydactyly refers to having additional fingers or toes. About 16.6% had polydactyly. Infants in the birth defect group had one or more of these birth defects.

[7]An alternative sample limited to gestational age between 19.5 and 43 weeks provided virtually similar results. Results are available from the authors upon request.

excluded to avoid potential problems of endogenous selection of maternal education and other characteristics such as employment at younger ages (about 300 data observations excluded). Cases with father's age of 16 years or less or more than 65 years are also excluded to avoid potential erroneous data or influential observations (only 6 data observations were excluded). The highest reported maternal age was 48 years so no upper limit was placed on maternal age. Cases from hospitals in five provinces with a very small representation in the sample with complete data (<1% of sample each) were excluded to limit heterogeneity of area effects and influential observations. Data from these provinces were not combined into a single geographical cluster because of their general dissimilarity in terms of their location and rural/urban representation.

Prenatal Care and Infant Health Measures

Prenatal care use was measured by time elapsed prior to prenatal care initiation and by number of prenatal care visits. Prenatal care delay represents the number of weeks between the first day of the last menstrual period and the first prenatal care visit. Subjects who reported no prenatal use had the delay set to 43 weeks (no difference in results when set to 40 weeks). We focus on reporting the results of prenatal care delay given that it has been the most commonly used measure in previous econometric studies and only report the main results of prenatal visits.

LBW and preterm birth are regarded as important and clinically relevant indicators that predict significantly future child mortality and development. Binary (0,1) indicators of LBW (< 2500 grams) and preterm birth (<37 weeks of gestation) were evaluated as the health outcomes. It is important to evaluate both LBW and preterm birth as LBW is also affected by fetal growth rate in addition to gestational length.[8]

Prenatal Care Demand Function

Prenatal care use is modeled as a function of risk indicators including presence of family members/relatives who also had the birth defects included in this study, occurrence of acute illness such as flu or rubella during pregnancy and chronic illnesses such as diabetes or hypertension, reporting any difficulty in conception (with current pregnancy or previous periods), occurrence of vaginal bleeding during the first trimester of pregnancy, number of previous live births, number of miscarriages and still births and maternal age (and age squared). Also included are maternal education and employment as these represent efficiency and enabling factors[9] and regional variables at the province level that represent availability of and access to health care including clinic and hospital bed concentration, unemployment and female uninsured rates, and a wealth measure related to the percentage of the population with unmet basic needs[10]. Other individual-level characteristics that may influence demand including infant ethnic ancestry and year of pregnancy occurrence are also included.

The prenatal care delay function was estimated by ordinary least squares (OLS) with heteroscedasticity-robust standards (White 1980). The demand function was estimated separately as well as pooled for infants with and without birth defects to evaluate potential differences in the demand function parameters between the two groups. The lack of differences

[8]Other measures such as very low birth weight (<1500 grams) or very preterm (<32 weeks of gestation) were not used due to the infrequency of these outcomes in the group of infants without birth defects (about 1%). Ordinal measures (e.g. normal, low and very low birth weight) were not used for the same reason.

[9]Maternal employment may also affect the time costs of obtaining prenatal care.

[10]Data on these area characteristics were obtained online (www.indec.mecon.gov.ar) through the National Institute of Statistics and Census of Argentina (INDEC). Unemployment rates were matched to the urban region including the city of hospital of birth to the best extent possible. For instance, the 9 cities in the Buenos Aires province were matched to 4 urban areas. INDEC defined unmet basic needs to include any of the following conditions: overcrowding (more than three individuals per room), inconvenient housing (such as unstable or unsafe establishments), lack of a lavatory in the house, presence of a school age child that does not attend school, and a low education worker supporting more than 4 individuals in household.

would suggest that the studied variables have relatively comparable effects on prenatal care demand by fetal health endowments that might vary between the infant groups.

Infant Health Production Function

LBW and preterm birth were modeled as a probit function of prenatal care use, other prenatal inputs including use of multivitamins or prenatal vitamins anytime during pregnancy, indicators for tetanus and varicella immunization during the first trimester of pregnancy[11] and exposure to trauma and physical shocks (more than 80% coded as severe traumatism) during the first trimester in addition to maternal education and employment, infant's sex and the risk indicators, ancestry and pregnancy year listed in the demand function above.

In order to account for the endogenous selection of prenatal care, the probit function for the health outcome and the demand function for prenatal care were simultaneously estimated by conditional maximum likelihood (Wooldridge 2002) with heteroscedasticity-robust standard errors[12]. In order to achieve identification, the prenatal care demand function included all the exogenous model variables in addition to the identifying instruments, which were excluded from the outcome equation. This estimation is more efficient than two-stage procedures (Rivers and Vuong 1988; Bollen, Guilkey, and Mroz 1996; Norton, Lindrooth, and Ennett 1998) and marginal effects in the outcome model can be readily estimated.[13] Further, a direct test of whether prenatal care use is exogenous involves testing the hypothesis of whether the correlation of the error terms of the two equations is equal to 0 – failing to reject this hypothesis indicates that there are unlikely to be omitted variables correlated with both prenatal care use and the health outcome and that prenatal care use is exogenous.

The identifiers or instruments for prenatal care use should satisfy two primary assumptions. First, the identifiers should be significantly related to the endogenous explanatory variable (i.e. to be able to affect or predict prenatal care use). Second, they should not be related to the outcome variable except through their effect on the endogenous variable (i.e. they should not be related to the outcome through unobservable confounders or through direct effects). Failing to satisfy either of these assumptions would imply that the identifiers are not appropriate and that the model will produce inconsistent results.

The regional characteristics representing accessibility to and availability of health care as well as wealth and time costs were used as instruments. The following instruments were used for the group without birth defects: clinic and hospital bed concentration, unemployment rates, and the percentage of the population with unmet basic needs. For the group with birth defects, the instruments were clinic concentration, the rate of uninsured females, and the percentage of the population with unmet basic needs. Different instrument combinations were used in order to ensure that the identifying assumptions described above are satisfied for both groups based on the standard tests. The assumption of the instruments being correlated with prenatal care utilization was evaluated using a Wald test of the joint significance of instrument coefficients in the prenatal care use function. The second assumption cannot be directly tested because of

[11]Given that immunizations are generally not recommended during first trimester of pregnancy even for tetanus, evaluating the effects of first trimester immunization is interesting to assess any potential adverse effects on the studied infant outcomes. Local public clinics in Argentina are available to provide very basic preventive care (including immunizations), yet these do not usually provide prenatal care (Personal correspondence with ECLAMC coordinators, 2005), which might explain the lack of association between prenatal care and immunization for the Argentinean sample observed in this study. This may explain in part why reporting immunization during first trimester was not significantly associated with reporting initiation of prenatal care in first trimester. Other types of immunizations were so infrequent, and were not evaluated in this study.

[12]The model was estimated using ivprobit in Stata. We tested for random province effects using the Breusch -Pagan test (Breusch and Pagan, 1980) in order to evaluate the appropriateness of adjusting for clustering at the province level when estimating the variance-covariance matrix (Moulton, 1986) but the null hypothesis of no random effects could not be rejected.

[13]Two stage techniques include those that substitute the predicted value of prenatal care use from the first stage for the actual value of prenatal care or add the residual from the first stage to the outcome equation as well as other techniques.

the role of unobserved factors, yet can be partially evaluated through over-identification tests, which evaluate if the added restrictions (more than one instrument in this model) are appropriate. We tested the over-identification restrictions based on the minimum-distance two-stage IV probit model (Newey 1987) and Lee's (1992) over-identification test (Baum et al. 2007). Probit models of LBW and preterm birth treating prenatal care use as exogenous were also estimated for comparison to the IV probit results.

RESULTS

Comparison of the Groups with and without Birth Defects

Table 1 lists the descriptions, means and standard deviations of all model variables for the groups of infants with and without birth defects and highlights the variables with significant distributions between the two infant groups. As expected, much higher LBW and preterm birth rates were observed in the group with birth defects compared to the group without birth defects. The average prenatal care delay was 17 weeks, which is longer by more than a month than the US, and was comparable between the two groups.[14] A slightly higher rate of varicella vaccination but lower rates of exposure to physical shocks, history of birth defects, acute and chronic illnesses and vaginal bleeding, as well as lower numbers of previous live births and miscarriages/stillbirths and maternal age were observed in the group without birth defects compared to the group with birth defects. No other major differences were observed between the two groups.[15]

Demand for Prenatal Care

Table 2 lists the coefficients of the estimated demand function for the pooled sample since the coefficients of the demand function were not statistically different between the two infant groups based on a Chow test (p=0.9). Table A in the Appendix reports the regression coefficients separately for the two infant groups.

Acute and chronic illnesses decreased prenatal care delay by about 1 week as did vaginal bleeding in the first trimester (by about a week and a half). Maternal age also decreased delay by about half a week per year. Previous live births increased delay by one week per live birth.

Higher mother's education decreased prenatal care delay (by about one and two weeks with secondary and university education respectively compared to primary school education, which in turn reduced delay by one week compared to less than primary school education). Maternal employment increased delay by about one week.

Native ancestry increased delay by about two weeks, while European Latin ancestry reduced delay by about half a week.[16] Prenatal care delay decreased in 2002 by two weeks compared to 1994. Finally, delay decreased with higher geographic concentration of public clinics and hospitals and with the rate of uninsured females and increased with the proportion of population with unmet basic needs (marginally significant). The unemployment rate had a negative but statistically insignificant effect on delay. In the group without birth defects, delay decreased the rate of residents per hospital bed.

[14]About 41% of the study sample initiated prenatal care within the first trimester. About 74% of women in a representative Brazilian sample were reported to have initiated prenatal care in the first trimester in Brazil (Barros et al, 2005).

[15]The small differences in the rates of uninsured females and unmet basic needs are due to differences in the geographic distribution between the two infant samples due to the added inclusion restrictions and missing data.

[16]Due to the admixture of ancestries in Argentina (i.e. each infant can have more than one ancestry), indicators were included for reporting Native, European-Latin, European non-Latin and other ancestries (Table 1). Therefore, the effect of reporting a certain ancestry is conditional on reporting other ancestries.

Effects of Prenatal Care Delay

Table 3 reports the marginal probability effects of prenatal care delay and their percentages of the mean predicted probabilities of LBW and preterm birth under the various model specifications in addition to the results of the exogeneity and the over-identification tests. In the group without birth defects, prenatal care delay did not have a significant effect on LBW and preterm birth when treated as exogenous and the coefficient had an unexpected negative sign. However, prenatal care delay increased significantly the probabilities of LBW and preterm birth when treated as endogenous using the conditional maximum likelihood (CML) estimation. A one week delay in prenatal care initiation increased LBW and preterm birth probabilities by 0.029 and 0.027 respectively, or by about 14 and 12% of mean predicted outcome probabilities.[17] The hypothesis of exogenous selection of prenatal care use was rejected ($p<0.01$) in both outcome models, and the over-identification restrictions of using more than one instrument to identify the model could not be rejected ($p=0.2$-0.5). The instruments were significantly predictive of prenatal care use ($p<0.01$).

In the group with birth defects, prenatal care delay had a small coefficient with the unexpected negative sign for LBW under both the standard probit and the CML models. The coefficient assuming exogenous prenatal care was statistically significant (one week delay decreased LBW probability by 0.002 or by 1% of mean predicted probability). Prenatal care delay also had a small negative coefficient when treated as exogenous in the preterm birth model. The coefficient switched signs but remained small and insignificant when prenatal care was treated as endogenous. The hypothesis of exogenous selection of prenatal care initiation could not be rejected in this group. The over-identfiication restrictions were similarly not rejected ($p=0.2$-0.4). The instruments were also significantly predictive of prenatal care use in this group ($p<0.01$).

Effects of Other Inputs and Risk Factors

Table 4 reports the marginal effects of other inputs and risk factors that had, for at least one of the infant groups, significant coefficients in a reduced form probit health production function that included all the inputs and risk factors listed above (except prenatal care use) in addition to the regional instruments used for prenatal care use. In both infant groups, acute illnesses increased preterm birth risk while maternal age decreased this risk. Moreover, number of previous live births decreased LBW risk in both groups. Acute illnesses and number of previous miscarriages and stillbirths also increased LBW risk in the group without birth defects, while maternal age decreased this risk. In the birth defect group, varicella immunization in the first trimester, male gender and European non-Latin origin decreased LBW risk, while European Latin origin increased this risk. In the group without birth defects, multivitamin use during pregnancy and maternal education decreased preterm birth risk, while exposure to physical shocks in the first trimester increased this risk. In the birth defect group, chronic illnesses and number of miscarriages and stillbirths increased preterm birth risk. Pregnancies in this group occurring in recent study years (2001 and 2002) had also increased risk for preterm birth compared to pregnancies in 1994. Finally, European non-Latin ancestry decreased preterm birth risk in the group with birth defects.

DISCUSSION

A clear pattern of adverse self-selection in prenatal care utilization was observed in this sample, whereby women at a potentially higher risk for adverse infant health outcomes initiated prenatal

[17]Given that the mean predicted probability estimated from the CML model might not necessarily be close to the unconditional outcome probability, it is important to compare the marginal probabilities to the mean predicted probability from that model to get a clearer interpretation of the magnitude of the effect.

care earlier than those at lower risk. Indicators such as illness occurrence during pregnancy and previous live births had significant effects on timing of prenatal care initiation, but also had effects on infant health that support the adverse self-selection hypothesis. Prenatal care delay showed no effect on LBW or preterm birth in models that ignored self-selection into prenatal care use even with direct adjustment for several relevant risk indicators. However, large increased risks with prenatal care delay were found for pregnancies uncomplicated with birth defects when accounting for the potential endogenous selection of prenatal care. It is not surprising that even an expanded direct adjustment for several relevant inputs including maternal health and fertility history does not alleviate the adverse self-selection bias, due to the theoretical role of several unobservable risks (e.g. having a previous LBW birth or the mother being LBW). Other studies have also found that the effects of prenatal care were overall insensitive to adding several risk indicators that were typically not observed in previous studies, emphasizing the role of unobservable risks in this self-selection (Reichman et al, 2006).

The estimated effects may be seen as unreasonably large, but given the long average waiting time before prenatal care initiation in this sample (17 weeks), a one or two week change at the mean may reflect more a transition between low and intermediate level of care than between intermediate and high care, which has been reported to be more effective on average than the latter change (Joyce, 1994). One potential reason for the large effects may be that the instruments were able to account for adverse self-selection bias related to unobservable health risks, but were unable to adequately account for potential favorable self-selection bias (i.e. instruments related to positive health behaviors in the same direction as with prenatal care use), which may lead to over-estimated effects of prenatal care utilization. This remains a possible limitation in this study though the instruments performed well through both being predictive of prenatal care use and also being excludable from the birth outcome production functions by passing the over-identification tests, which provides some assurance against such a bias.

In order to test the sensitivity of the study results to the used instruments, we evaluated alternative specifications including adding father's education and employment to the regional instruments as well as using an instrument specification of only individual level characteristics including maternal and father's education and employment.[18] Maternal and father's education and father's employment are expected to improve access to prenatal care (both through increasing information and financial resources), while maternal employment might have mixed effects as it could provide financial resources but also increase the time costs of obtaining prenatal care. Arguably, these individual-level characteristics might, at least theoretically, still influence the birth outcomes through other ways besides prenatal care and that they would be expected to suffer more from favorable-selection bias than the regional variables. For instance, maternal education is expected to increase access to prenatal care but also to contribute to adopting healthier behaviors (such as better nutrition and reduced risk taking behaviors such as smoking). We found comparable CML effects of prenatal care using these alternative instrument specifications to those reported in Table 3.[19] This provides support for the results based on the primary instrument specification of regional characteristics. However, further research is needed using individual-level instruments that are more predictive of prenatal care and that are also more exogenous theoretically such as distance to prenatal care facilities. In general, most available data sources lack data on such instruments, which highlights the importance of obtaining such measures in birth outcome registries and surveillance programs

[18]In the second instrument sensitivity specification, maternal education and employment were excluded from the birth outcome function.

[19]The individual level instruments passed the over-identification tests. In the group without birth defects, the over-identification test p values were 0.5 and 0.9 in the LBW and preterm birth models respectively when adding father's education and employment to the regional instruments and p=0.65 and 0.6 in the LBW and preterm birth models respectively when using maternal and father's education and employment alone as instruments. In the group with birth defects, the over-identification test p values were 0.12 and 0.7 in the LBW and preterm birth models respectively when adding father's education and employment to the regional instruments and p=0.8 and 0.98 in the LBW and preterm birth models respectively when using maternal and father's education and employment alone as instruments.

to expand the potential of future econometric research studies aimed at studying infant and child health production.

No protective effects of earlier initiation of prenatal care against LBW and preterm birth were suggested for the birth defect group. One potential explanation may be heterogeneity in prenatal care effectiveness by birth defect severity which attenuates the overall effectiveness estimate or due to genetic and structural constraints that prevent any potential benefits on the study outcomes. In a sensitivity analysis, we estimated the LBW and preterm birth functions under scenarios of exogenous and endogenous prenatal care as exogenous and endogenous for the subgroups with only one of the studied birth defects though the sample size is limited to allow a robust analysis by birth defect type.[20] Prenatal care delay had no effect on LBW or preterm birth when treated as exogenous for all these subgroups, but LBW risk by about 9.5% (marginally significant at p=0.06) for the subgroup with trisomy 21 only (374 cases) when treated as endogenous (over-identification p =0.76). When treated as endogenous, prenatal care delay increased preterm birth risk by 8.6% (p=0.08), 11.5% (p<0.01) and 10.2% (p<0.01) for the subgroups with trisomy 21, congenital heart disease and polydactyly respectively.[21] These analyses suggest potential heterogeneity in prenatal care effectiveness by birth defect type and that pregnancies complicated with certain isolated birth defects including trisomy 21, congenital heart disease and polydactyly might still have improved birth outcomes with earlier initiation of prenatal care. Further research is needed to investigate the heterogeneity in prenatal care effectiveness by pregnancy complications and to identify contributors to the potential lack of prenatal care effects on birth outcomes among pregnancies complicated with certain common birth defects.[22]

We evaluated alternative estimation techniques including two-stage probit models (Bollen, Guilkey, and Mroz 1996; Newey 1987) and 2SLS and found comparable results which provide support for the results based on the conditional maximum likelihood model. In alternative models, we measured prenatal care use by number of visits and observed overall similar results in terms of rejection of exogenous selection into prenatal care in the LBW model for the group without birth defects and larger effects of prenatal care visits when accounting for self-selection using the conditional maximum likelihood. Prenatal care visits had a small negative effect on LBW in the group with birth defects when treated as exogenous (6.7% decrease in LBW risk per visit), yet had a positive signed coefficient when treated as endogenous, and though the effect of prenatal visits was statistically insignificant, the exogeneity of prenatal care visits was rejected at p=0.01 in this group. We used prenatal care delay as the primary utilization measure since number of visits is likely to be confounded by reverse effects of length of gestation (longer gestation will allow more visits to be made) which complicates the assessment of the effects of prenatal visits on preterm birth.[23]

One limitation of the study relates to lacking certain relevant variables, such as smoking, alcohol use and marital status, which were not available in the study data. Previous research has suggested no effect of excluding smoking and illicit drug use on estimated effects of prenatal care on birth outcomes (Joyce 1994; Warner 1998; Reichman et al. 2006).[24] The fact that 92% of the mothers in the analyzed sample reported living with the child's father at birth

[20]The CML models did not converge for some birth defects due to small birth defects. These were estimated by based on the minimum-distance two-stage IV probit model (Newey 1987).

[21]The overidentification restrictions could not be rejected at p=0.78, 0.43 and 0.87 for the subgroups with trisomy 21, congenital heart disease and polydactyly.

[22]It is unlikely that prenatal care delay affects the occurrence of the included birth defects given that the majority of the mothers initiated prenatal care after the 12th week of gestation. If prenatal care delay has small effects on preventing these birth defects, then the benefits of prenatal care on reducing LBW and preterm birth at the population level might be underestimated in this study given the high rates of LBW and preterm birth in the group with birth defects.

[23]Indeed, the over-identification restrictions were rejected in the preterm birth outcome model in the group without birth defects at p=0.02 suggesting persistent reverse effects of gestational age on number of prenatal care visits.

and that all the included observations had complete data on father's characteristics (age, education, employment) suggests that married mothers or those in a stable relationship formed the majority of the analyzed sample. Therefore, we expect no real impact of these limitations on the primary results of the study. However, it is important to reinvestigate this research question in the future using datasets that include information on these unmeasured inputs.

Another contribution of this study is its evaluation of the effects of risk indicators such as maternal health and family history of birth defects on prenatal care utilization and prenatal inputs such as multivitamin use, immunization and maternal health on birth outcomes, which have been less studied in an econometric framework. The increased risks of LBW and preterm birth risks with adverse maternal health including both chronic and acute illnesses suggests that developing policies and interventions to improve maternal health will also result in improving birth outcomes. The effects of multivitamin use on reducing preterm birth risk emphasizes the need for further research to assess the role of multivitamins in improving birth outcomes especially using designs that can account for self-selection into multivitamin use such as instrumental variable analyses or randomized clinical trials. Given that multivitamins are relatively cheap, interventions to improve multivitamin use are likely to be cost-effective if multivitamin use is truly effective in reducing preterm birth risk. Further, the increased preterm birth risk with exposure to physical shocks (trauma) highlights the complex etiology of birth outcomes as being a function of several individual and environmental factors and highlights the importance of further research to understand the determinants and contexts of maternal exposure to trauma during pregnancy (for example, to identify the role of occupational, household and transportation hazards).

This study has found evidence of self-selection in prenatal care utilization in samples from Argentina, and to our knowledge, is the first observational study that attempted to account for self-selection using an instrumental variables model when estimating the effects of prenatal care utilization on birth outcomes of infants with birth defects. The study suggests large infant health benefits from earlier initiation of prenatal care for pregnancies uncomplicated with birth defects. Given that prenatal care has low cost on average, improving prenatal care utilization in Argentina seems an appropriate health policy intervention. Since prenatal care delay decreased with increased availability of health services, one approach to improve utilization might be increasing the availability of prenatal health services particularly in communities that may be underserved[25]. The observed increase in prenatal care delay with increased health coverage rates is not surprising given that about half of ambulatory care facilities in Argentina are publicly owned, implying comparable access to prenatal care between insured and uninsured people.[26] Given that most prenatal care services may be adequately performed in outpatient settings, increasing insurance may not bring large improvements to prenatal care access in Argentina. This of course deserves further investigation and is outside the scope of the study. Another approach to improve prenatal care utilization would be to increase women's awareness of potential benefits of earlier prenatal care initiation. This is supported by the observation that women responded to potential risk indicators. An inexpensive intervention would be to launch media campaigns that highlight the importance of prenatal care. The longer prenatal care delay among employed mothers is likely due to increased time costs. This highlights the importance of having work policies (related to both scheduling and payment) that facilitate the initiation of prenatal care among employed mothers. Further, prenatal care standards may need to be modified in Argentina in order to improve utilization rates. While

[24]This is primarily due to the limited correlation between smoking (a usual behavior) and time of prenatal care initiation (a pregnancy specific behavior).

[25]The number of ambulatory care centers increased by 100% between 1980 and 1995 in Argentina (Belmartino 2000). It is unclear how willing policy makers would be to further increase the availability of these clinics.

[26]Many underinsured patients also seek care at the publicly owned facilities which are tax-funded and usually provide care at no or minimal cost to patients.

prenatal delay seemed to have decreased significantly in 2002, the average waiting time is still substantially longer than waiting time in the US and other developed countries.

Appendix

Table A
OLS Coefficients of the Demand Function for Time before Prenatal Care Initiation in Weeks by Birth Defect Status

Model Covariates	*Sample without Birth Defects (N=2325)*	*Sample without Birth Defects (N=2417)*
Intercept	49.7*** (7.8)	48.8*** (7.4)
Birth defect history	-1.2* (0.7)	-0.2 (0.5)
Difficulty in conception	-0.5 (0.7)	-0.7 (0.6)
Acute illness	-1.4*** (0.4)	-1.2*** (0.4)
Chronic illness	-1.1** (0.4)	-0.6 (0.4)
First trimester bleeding	-2.2*** (0.7)	-1.4** (0.6)
Live births	1.0*** (0.1)	1.0*** (0.1)
Miscarriages/stillbirths	0.1 (0.3)	-0.4* (0.2)
Maternal age	-0.6* (0.3)	-0.6** (0.3)
Maternal age squared	0.01 (0.01)	0.01 (0.004)
Maternal education-Less than primary	1.4** (0.6)	0.7 (0.6)
Maternal education-Incomplete secondary	-0.1 (0.5)	-0.7 (0.5)
Maternal education-Secondary	-0.5 (0.5)	-1.7*** (0.6)
Maternal education-University	-1.8** (0.7)	-2.1*** (0.8)
Maternal employment	0.7 (0.5)	1.1** (0.5)
Native ancestry	1.5*** (0.6)	2.0*** (0.6)
European Latin ancestry	-0.7 (0.4)	-0.6 (0.4)
European non-Latin ancestry	-0.7 (0.6)	-0.05 (0.6)
All other ancestry	0.8 (0.9)	-1.5* (0.8)
Pregnancy year 95	-0.1 (0.8)	-0.7 (0.9)
Pregnancy year 96	-0.2 (0.9)	0.2 (0.9)
Pregnancy year 97	0.02 (0.8)	0.5 (0.9)
Pregnancy year 98	-0.3 (0.8)	-0.6 (0.8)
Pregnancy year 99	-0.2 (0.8)	-0.3 (0.8)
Pregnancy year 00	-0.9 (0.8)	0.2 (0.9)
Pregnancy year 01	0.01 (0.8)	-0.2 (0.8)
Pregnancy year 02	-2.4** (1.0)	-1.9* (1.0)
Clinics concentration (2003)	-0.7*** (0.2)	-0.7*** (0.1)
Residents per hospital bed (1995)	-0.01** (0.004)	0.003 (0.004)
Unemployment (2002)	-0.03 (0.1)	-0.1 (0.1)
Female uninsured (2001)	-0.4*** (0.1)	-0.5*** (0.1)
Unmet basic needs (2001)	0.2** (0.1)	0.04 (0.1)
R squared	*0.12*	*0.13*

Note: Robust standard errors of regression parameters are listed in parentheses. *, **, and *** indicate significant coefficients at p<0.1, p<0.05 and p<0.01 respectively.

Acknowledgements

The authors would like to thank the health professionals involved in ECLAMC. This work benefited from review by Drs. John Brooks, Julie Urmie and John Schneider and was supported by NIH grants 1R03DE018394-01 (GLW), U01 DE-017958 (JCM and GLW), and the Agencia Nacional de Promoción Científica y Tecnológica ANPCyT PICTO-CRUP 2005 # 31101 and Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET)CNPq/MS/DECIT 40.3444/2004-7 and 40.1467/2004-0 from Argentina.

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Table 1
Means and Standard Deviations of Study Variables

Variable name	Definition	Infants without Birth Defects (N=2325)	Infants with Birth Defects (N=2417)
Infant Health Outcomes			
Low birth weight	Indicator (0,1) for low birth weight (<2500 grams)	0.061*** (0.239)	0.226 (0.418)
Preterm Birth	Indicator (0,1) for gestational age <37 weeks	0.129*** (0.336)	0.252 (0.434)
Prenatal Care Measures			
Weeks	Pregnancy weeks elapsed prior to initiating prenatal care	17.102 (9.188)	17.054 (9.556)
Other Health Inputs			
Multivitamin	Indicator (0,1) for multivitamin use during pregnancy	0.054 (0.226)	0.065 (0.246)
Tetanus	Indicator (0,1) for tetanus immunization in 1st trimester	0.053 (0.225)	0.046 (0.208)
Varicella	Indicator (0,1) for varicella immunization in 1st trimester	0.092* (0.289)	0.077 (0.266)
Physical shocks	Indicator (0,1) for maternal exposure to physical factors in 1st trimester	0.033*** (0.179)	0.055 (0.228)
Risk Indicators (also potential health determinants)			
Birth defect history	Indicator (0,1) for reporting any relatives of the child with the studied birth defects	0.061*** (0.240)	0.183 (0.387)
Difficulty in conception	Indicator (0,1) for reporting any difficulty in conception	0.086 (0.280)	0.085 (0.279)
Acute maternal illness	Indicator (0,1) for any acute illnesses during pregnancy	0.364*** (0.481)	0.406 (0.491)
Chronic maternal illness	Indicator (0,1) for any chronic illnesses during pregnancy	0.167*** (0.373)	0.213 (0.409)
First trimester bleeding	Indicator (0,1) for vaginal bleeding in 1st trimester	0.063*** (0.243)	0.108 (0.311)
Live births	Number of previous live births	1.994*** (2.067)	2.448 (2.303)
Miscarriages/stillbirths	Number of spontaneous previous abortions/stillbirths	0.287*** (0.615)	0.376 (0.764)
Maternal age	Maternal age in years at delivery	27.4*** (5.7)	29.5 (6.8)
Maternal age squared	Maternal age in years at delivery squared	781.4*** (334.8)	914.4 (423.6)
Efficiency/Enabling Factors			
Maternal education-Less than primary ⊨	Indicator (0,1) for below primary school education	0.149 (0.356)	0.165 (0.372)
Maternal education-Incomplete secondary ⊨	Indicator (0,1) for incomplete secondary school education	0.249 (0.432)	0.247 (0.432)
Maternal education-Secondary ⊨	Indicator (0,1) for completed secondary school education	0.174* (0.379)	0.153 (0.361)
Maternal education-University ⊨	Indicator (0,1) for attending university	0.075 (0.263)	0.065 (0.247)
Maternal employment	Indicator (0,1) for maternal employment status	0.205 (0.404)	0.212 (0.409)

Variable name	Definition	Infants without Birth Defects (N=2325)	Infants with Birth Defects (N=2417)
Other Characteristics (some may affect both demand and infant health)			
Native ancestry	Indicator (0,1) for native ancestry	0.864* (0.343)	0.88 (0.325)
European Latin ancestry	Indicator (0,1) for Latin European ancestry	0.454 (0.498)	0.456 (0.498)
European non-Latin ancestry	Indicator (0,1) for non-Latin European ancestry	0.078 (0.268)	0.078 (0.268)
Other ancestry	Indicator (0,1) for other ancestry	0.041 (0.199)	0.045 (0.207)
Male	Indicator (0,1) for a male sampled subject	0.505 (0.5)	0.516 (0.5)
Pregnancy year 95≠	Indicator (0,1) for pregnancy in 1995	0.127 (0.333)	0.123 (0.328)
Pregnancy year 96≠	Indicator (0,1) for pregnancy in 1996	0.096 (0.295)	0.103 (0.304)
Pregnancy year 97≠	Indicator (0,1) for pregnancy in 1997	0.116 (0.320)	0.11 (0.313)
Pregnancy year 98≠	Indicator (0,1) for pregnancy in 1998	0.129 (0.335)	0.125 (0.331)
Pregnancy year 99≠	Indicator (0,1) for pregnancy in 1999	0.144 (0.351)	0.143 (0.350)
Pregnancy year 00≠	Indicator (0,1) for pregnancy in 2000	0.121 (0.327)	0.124 (0.329)
Pregnancy year 01≠	Indicator (0,1) for pregnancy in 2001	0.135 (0.342)	0.142 (0.349)
Pregnancy year 02≠	Indicator (0,1) for pregnancy in 2002	0.035 (0.184)	0.044 (0.205)
Area Characteristics (province level)			
Clinics concentration (2003)	Number of public clinics and hospitals per 100 Km^2 in 2003	5.333 (7.218)	5.105 (7.080)
Residents per hospital bed (1995)	Number of residents per public hospital bed in 1995	461.237 (93.239)	465.593 (95.414)
Unemployment (2002)	Unemployment rate in 2002 in urban areas of the province	21.084 (4.205)	19.561 (3.310)
Female uninsured (2001)	% of females without public or private insurance in 2001	39.228* (9.825)	39.701 (9.768)
Unmet basic needs (2001)	% of population in 2001 with basic needs unmet	14.169*** (5.461)	14.708 (5.729)

Note: Standard Deviations are listed in parentheses.

⊨ Omitted category is completed primary school

≠ Omitted category is year 1994. The decrease of frequency of pregnancy occurrence in 2002 is due to the fact that the sample included cases born in 1995 through 2002.

*, ** and *** indicate significant differences in the distribution of the variables between the samples of infants with and without birth defects at p<0.1, <0.05 and <0.01 respectively.

Table 2

OLS Coefficients of the Demand Function for Time before Prenatal Care Initiation in Weeks

Model Covariates	***Pooled Birth Sample (N=4742)***
Intercept	48.7*** (5.3)
Family history of birth defects	-0.5 (0.4)
Difficulty in conception	-0.6 (0.5)
Acute maternal illness	-1.3*** (0.3)
Chronic maternal illness	-0.8*** (0.3)
First trimester bleeding	-1.7*** (0.4)
Prior live births	1.0*** (0.1)
Prior miscarriages/stillbirths	-0.2 (0.2)
Maternal age	-0.6*** (0.2)
Maternal age squared	0.01** (0.003)
Maternal education-Less than primary	1.0** (0.5)
Maternal education-Incomplete secondary	-0.4 (0.3)
Maternal education-Secondary	-1.1*** (0.4)
Maternal education-University	-1.9*** (0.5)
Maternal employment	0.9*** (0.3)
Native ancestry	1.8*** (0.4)
European Latin ancestry	-0.7** (0.3)
European non-Latin ancestry	-0.4 (0.4)
All other ancestry	-0.4 (0.6)
Pregnancy year 95	-0.4 (0.6)
Pregnancy year 96	-0.04 (0.6)
Pregnancy year 97	0.3 (0.6)
Pregnancy year 98	-0.5 (0.6)
Pregnancy year 99	-0.2 (0.6)
Pregnancy year 00	-0.4 (0.6)
Pregnancy year 01	-0.1 (0.6)
Pregnancy year 02	-2.1*** (0.7)
Clinics concentration (2003)	-0.7*** (0.1)
Residents per hospital bed (1995)	-0.002 (0.003)
Unemployment (2002)	-0.1 (0.1)
Female uninsured (2001)	-0.4*** (0.1)
Unmet basic needs (2001)	0.1* (0.1)
R squared	*0.12*

Note: The variables and reference categories of the dummy variables are as listed in Table 1. Robust standard errors of regression parameters are listed in parentheses. *, **, and *** indicate significant coefficients at p<0.1, p<0.05 and p<0.01 respectively.

Table 3

Marginal Probability Effects of Prenatal Care Delay on LBW and Preterm birth

Model	Group without Birth Defects				Group with Birth Defects			
	ME (SE)	***% of probability of positive outcome***	***Instrument Significance X^2[df]***	***Over-identification test X^2[df], (p value)***	***ME (SE)***	***% of probability of positive outcome***	***Instrument Significance X^2[df]***	***Over-identification test X^2[df], (p value)***
LBW (birth weight <2500 grams)								
Probit (prenatal care use exogenous)	-0.00006 (0.0005)	-0.13%			-0.002** (0.001)	-1.0%		
CML (prenatal care use endogenous	0.029*** ⊭ (0.008)	13.7%	24.8*** [4]	4.6 [3], (0.2)	-0.004 (0.007)	-1.7%	54.1*** [3]	3.3 [2], (0.2)
Preterm birth (gestation <37 weeks)								
Probit (prenatal care use exogenous)	-0.0002 (0.0007)	-0.23%			-0.001 (0.001)	-0.6%		
CML (prenatal care use endogenous	0.027*** ⊭ (0.008)	11.9%	23.6*** [4]	2.3 [3], (0.5)	0.006 (0.007)	2.4%	54.0*** [3]	2.1 [2], (0.4)

Note: Marginal probability effects (ME) of prenatal care delay in weeks and the probability of positive outcome are estimated holding model covariates at their means. Standard errors (SE) of marginal effects are listed in parentheses. The Chi-square test statistics (X^2) and their degrees of freedom [df] for testing the joint significance of the instruments and for testing the appropriateness of the over-identification restrictions of the instruments are reported.

** and *** indicate significant effects at p<0.05 and p<0.01 respectively.

⊭ indicates rejection of the hypothesis of exogenous selection of time of prenatal care initiation at p<0.01.

Table 4

Marginal Effects of Other Inputs and Risk Factors on LBW and Preterm birth

Input/Risk Factor	Group without Birth Defects		Group with Birth Defects	
	ME (SE)	*% of mean outcome probability*	*ME (SE)*	*% of mean outcome probability*
	LBW (birth weight <2500 grams)			
Acute illnesses	0.021** (0.009)	47.4%	0.004 (0.017)	2.1%
Miscarriages/stillbirths	0.013** (0.006)	30.9%	0.012 (0.011)	5.9%
Maternal age	-0.005* (0.003)	-12.1%	-0.004 (0.006)	-1.9%
Live births	-0.007** (0.003)	-15.6%	-0.017*** (0.005)	-7.9%
Varicella immunization	-0.004 (0.017)	-8.7%	-0.068* (0.036)	-32.3%
Male	-0.005 (0.008)	-12.4%	-0.042** (0.017)	-19.7%
European Latin	-0.009 (0.01)	-21.3%	0.036* (0.02)	16.8%
European non-Latin	0.019 (0.018)	44.4%	-0.052* (0.029)	-24.6%
	Preterm birth (gestation <37 weeks)			
Multivitamin	-0.053** (0.021)	-50.3%	-0.009 (0.035)	-3.9%
Physical shocks	0.093** (0.046)	88.9%	-0.03 (0.037)	-13.4%
Maternal education-Less than primary versus primary school	0.035* (0.021)	33.7%	0.017 (0.027)	7.3%
Maternal education-secondary versus primary school	-0.047*** (0.016)	44.9%	0.006 (0.027)	2.6%
Maternal age	-0.013** (0.005)	-12.6%	-0.012** (0.006)	-5.3%
Acute illnesses	0.026* (0.013)	24.9%	0.039** (0.018)	17.2%
Chronic illnesses	0.017 (0.017)	16.4%	0.066*** (0.022)	29.2%
European non-Latin ancestry	0.046 (0.028)	43.5%	-0.058* (0.029)	-25.5%
Miscarriages/stillbirths	0.01 (0.01)	9.2%	0.027** (0.011)	11.8%
Pregnancy year 2001	0.058 (0.042)	54.9%	0.1* (0.054)	44.0%
Pregnancy year 2002	0.092 (0.063)	87.6%	0.149** (0.07)	65.7%

Note: Marginal probability effects (ME) and mean predicted probabilities are estimated holding model covariates at their means. A reduced form probit function was estimated for LBW and preterm birth for each infant group. The inputs and risk factors included above are the ones that had significant coefficients and marginal effects for at least one infant group. The mean predicted probabilities of LBW and preterm birth were 0.044 and 0.105 respectively in group without birth defects, and 0.211 and 0.227 respectively in the group with birth defects. Standard errors (SE) of marginal effects are listed in parentheses.

*, ** and *** indicate significant marginal effects at $p<0.1$, $p<0.05$ and $p<0.01$ respectively.