Examining the Relationship Between Low Birth Weight Occurrence and Passive Measures of Environmental Arsenic by Census Tract in Escambia and Santa Rosa Counties, Florida

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ABSTRACT: Arsenic is a public health concern because of its widespread distribution and high toxicity, even when doses are small. Low birth weight (LBW) occurrence, birth weights less than 2500g, may be associated with prenatal exposure of arsenic from environmental factors and consuming contaminated drinking water and food. The objective of this study was to examine whether mothers living in areas of Escambia and Santa Rosa counties with varying levels of background arsenic in surface soil and water were associated with the occurrence of LBW. Inverse distance weight in ArcGIS was used to interpolate arsenic concentrations from environmental samples and estimate arsenic concentrations by census tracts in the two counties. After excluding multiple births and displaced geocoding addresses, birth data were obtained for the years of 2005 (n = 5845), 2010 (n = 5569), and 2015 (n = 5770) from the Bureau of Vital Statistics at the Florida Department of Health to assess temporal differences. Generalized linear models were used to analyze and compare the association between child and maternal demographic information, socioeconomic characteristics, and the environmental estimates of arsenic with LBW. No significant association was found between environmental arsenic concentration and LBW, suggesting that environmental contamination of the pregnant mother’s census tract may not be a useful proxy in assessing risk for LBW.

KEYWORDS: Pregnancy, arsenic, smoking, low birth weight, environmental health

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INTRODUCTION

Chronic exposure to arsenic during pregnancy can affect fetal growth and development, leading to low birth weight (LBW) and other adverse birth outcomes.1-3 LBW infants, infants whose birth weights are less than 2500 g, are at risk for many developmental complications, and arsenic exposure during pregnancy increases this risk.4,5 Arsenic exposure in infants may result from placental transfer of arsenic concentrations from mother to child.6 Concerns have been raised that pregnant women living in areas with high arsenic levels in soil and drinking water may have increased risk for LBW. Chronic exposure to arsenic during pregnancy has been found to increase the likelihood of stillbirth and neonatal deaths.7 Arsenic exposure could eventually lead to stunted growth in young children.8 Humans are exposed to arsenic through ingestion, inhalation, and dermal contact from contaminated sources.9 A common cause of arsenic exposure among pregnant women is the ingestion of arsenic-contaminated foods and lifestyle choices, including firsthand or secondhand tobacco smoke.1,10,11

In this study, the relationship between residence in areas suspected to have higher environmental arsenic levels in private drinking wells and soil and LBW was investigated. In a previous study, it was found that individual counties in the state of Florida, including Escambia and Santa Rosa, had high distributions of arsenic in the environment from both natural and anthropogenic sources.12 As part of a United States Environmental Protection Agency (USEPA) program to assess pollution and community health in Escambia and Santa Rosa counties, surface soil samples were collected from 126 sites with test results identifying arsenic levels that ranged from 0.13 to 14.0 mg/kg, with a mean of 1.38.13 Although generally low, 33 of those sites exceeded Florida’s residential soil cleanup target level of 2.1 mg/kg. Arsenic concentrations from private drinking wells in the two counties have been recorded by the Florida Department of Health and Department of Environmental Protection. For this study, the relationship between environmental arsenic proximity and LBW was investigated. The objective of this study was to examine whether mothers in Escambia and Santa Rosa counties living in areas with higher levels of background arsenic in surface soil and water were at a higher risk for delivering a newborn with LBW.

MATERIAL AND METHODS

Ethics review and consent

The study was approved by the Florida Department of Health Institutional Review Board Committee on July 12, 2018 (Florida Department of Health [FDOH] IRB Study # 2018012). This study was approved by the University of Illinois Urbana-Champaign Institution Review Board Committee on November 29, 2017 (IRB Study #18359). Due to the nature of...
the data, informed consent from the mothers was not deemed necessary and was waived by both Institutional Review Board committees.

Data sources

Birth data. The study areas of interest were Escambia and Santa Rosa County in the Florida Panhandle. Both areas are predominantly rural, with the largest principal city being Pensacola. To measure LBW, we obtained data on birth weight, child and maternal demographic information, and residential location data from the Bureau of Vital Statistics at the Florida Department of Health. Only singleton births were used for our study, meaning records with multiple births were excluded from the study. The data sets included infant information, such as a birth month, birth year, gestational age, sex, birth weight, plurality, and birth facility. Parental information, such as mother and father’s age, residential address, race, and ethnicity, were also included in the data set. Data that account for social and lifestyle characteristics, such as Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) program enrollment, tobacco use during pregnancy, and alcohol use, were also included in the data set.

Surface soil and private well data. One hundred and sixty-five surface soil samples were used for this study (Escambia County, n = 104; Santa Rosa County, n = 61). One hundred and seven surface soil arsenic concentrations were compiled from multiple sites around Escambia and Santa Rosa Counties using secondary data from previous studies. The soil and water arsenic concentrations were highly skewed, and 2018 to monitor arsenic concentrations changes over time. We obtained data on birth weight, child and maternal demographic information, and residential location data from the Bureau of Vital Statistics at the Florida Department of Health. Only singleton births were used for our study, meaning records with multiple births were excluded from the study. The data sets included infant information, such as a birth month, birth year, gestational age, sex, birth weight, plurality, and birth facility. Parental information, such as mother and father’s age, residential address, race, and ethnicity, were also included in the data set. Data that account for social and lifestyle characteristics, such as Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) program enrollment, tobacco use during pregnancy, and alcohol use, were also included in the data set.

Generalized linear models (GLMs) were used to estimate the relationship between LBW probability and arsenic concentrations from surface soils and private water wells, respectively. Inverse distance weight (IDW) was used in ArcGIS to interpolate arsenic concentrations from soil samples and predict arsenic concentrations with the assumption that each measured sampling point was influenced by the distance between points, meaning points that are closer together have a stronger weight than points further away. A separate IDW was performed to predict the estimate of arsenic concentrations from private well data. Private well IDW were only assessed for census tracts that contained private wells. Census tracts with no private wells present were not included in the interpolation. The results of the IDW interpolation were depicted as a GIS raster grid, which was used to calculate the zonal statistics (mean of the arsenic concentrations) by census tract.

These zonal statistics were then spatially joined to the census tract shapefiles by adding mean soil and private well arsenic concentrations to the census tract shapefile data, respectively. The same mean soil and private well arsenic concentration estimations were used for all 3 data sets: 2005, 2010, and 2015. The mean environmental arsenic concentrations from both the soil and private wells were spatially joined to the geocoded birth data for Escambia and Santa Rosa counties at the census tract level. The final data sets consisted of point data for each birth, along with covariates, and mean private well and surface soil arsenic estimation for each of the three years.

Statistical analysis

Generalized linear models (GLMs) were used to estimate the relationship between LBW probability and arsenic concentrations from surface soils and private water wells, demographic data, birth data, and social and lifestyle covariates listed in Tables 1 and 2. Parental ages were recategorized into two groups representing mothers: under the age of 25 years and age 25 years and older. Parental races were categorized into three groups: white, black, and other races. Other races include Asian, Native American, Pacific Islander, and people who identified as more than one race. Parental education was recategorized into two categories to represent parents who had a less than high school education and those who had a high school education or higher. The birth facility was recategorized into two groups representing hospital births and nonhospital births (i.e., ambulance births and at-home births). Medical pay source or insurance information was recategorized into private insurance, nonprivate insurance, and other (self-pay or affiliated program pay). Maternal

Spatial analysis

Residential address geocoding. Residential addresses for the mothers were provided as part of the vital statistics data set and geocoded using the geocoding toolbox in ArcGIS. To geocode, full addresses were submitted to the ArcOnline World Geocoding Service through ArcGIS. Births that occurred in Escambia and Santa Rosa Counties for which the mother resided in other counties were excluded from the study as we had no way to account for how long they have been in either of the two counties.

Spatial interpolation of environmental data. For this study, spatial interpolation techniques were used to estimate arsenic concentrations in surface soil and private well data, respectively. The same mean soil and private well arsenic concentration estimations were used for all 3 data sets: 2005, 2010, and 2015. The mean environmental arsenic concentrations from both the soil and private wells were spatially joined to the geocoded birth data for Escambia and Santa Rosa counties at the census tract level. The final data sets consisted of point data for each birth, along with covariates, and mean private well and surface soil arsenic estimation for each of the three years.
tobacco and alcohol usage refers to whether mothers used tobacco and alcohol during pregnancy. Maternal tobacco usage notes if mothers smoked during pregnancy, quit while pregnant, or did not smoke during pregnancy. For all groups, missing or not available data were classified as not available.

A logistic regression model was fitted for each of the three years. The outcome (dependent) variable for each model was LBW, and explanatory (independent) variables included arsenic concentration from surface soils and private water wells, demographic (parents race, ethnicity, age, and education) and birth data (infant sex and birth facility), and social and lifestyle covariates (medical source pay, maternal alcohol, and tobacco usage, and WIC enrollment). Medical source pay and WIC program enrollment were used as proxies for socioeconomic status. Variance inflation factors (VIF) were used to assess the explanatory variables for multicollinearity. Stepwise selection was used to optimize our model for the best fit. Pseudo $R^2$ was reported for each model to explain how well the best-fitted variables of the model explain LBW. Receiver operator characteristic (ROC) curves were constructed to determine model prediction accuracy for the probability of LBW occurrence at the census tract level and to quantify the area under the curve (AUC) (Supplemental Figure S1). R code for this study was archived on GitHub. All data were analyzed using R version 3.4.1.

Results

Descriptive statistics

After excluding multiple births and displaced geocoding addresses, birth data were obtained for the years of 2005 (n = 5845), 2010 (n = 5569), and 2015 (n = 5770). Arsenic levels in soil samples across both counties ranged from 0.58 to 121.0 mg/kg, with a mean of 5.75 mg/kg. Arsenic concentrations in private wells across both counties ranged from 0 to 3.3 µg/L with a mean of 0.45 (SD = 0.61), which are below the maximum contaminant level (MCL) in drinking water and do not pose an immediate threat to human health according to the EPA. Arsenic levels in soil samples and private wells were not significant associated with LBW occurrence in our GLM. Our findings showed that infants born to white fathers were 3.15 times as likely to experience LBW occurrence compared with infants born to black and white mothers (OR = 0.56; CI = 0.33-0.90). Infants born to mothers aged 25 years and above were 38% more likely to experience LBW occurrence compared with infants born to mothers under the age of 25 years (OR = 1.38; CI = 1.08-1.76).

Results of the GLMs

The results of the logistic regression are noted in Figure 1A to C and expressed as odds ratios. In all three models, private well and surface soil arsenic concentrations in the environment were shown to have no significant association with LBW occurrence. For the year of 2005, our study found that the probability of infant born from mother with private health insurance experiencing LBW occurrence was 31% less likely to occur when mothers had private insurance compared with mothers with nonprivate and other forms of medical payment (odds ratio [OR] = 0.69; confidence interval [CI] = 0.49-0.98). Infants born of mothers of other races (excluding black and white) were 54% (OR = 0.46; CI = 0.25-0.80) less likely to experience LBW compared with infants born to black or white mothers, whereas the probability of infant born of white mother were 58% less likely to experience LBW occurrence (OR = 0.42; CI = 0.32-0.57).

For the year of 2010, no significant association was found between LBW occurrence and arsenic concentrations associated with surface soil and private wells. Infants born from mothers who had other or private insurance were 75% (OR = 0.25; CI = 0.15-0.40) and 23% (OR = 0.77; CI = 0.60-0.99) less likely to have LBW occurrence compared with mother with nonprivate insurance, respectively. Infants born to mothers from other races (excluding black and white) were predicted to have a 44% decrease in LBW occurrence compared with infants born to black and white mothers (OR = 0.56; CI = 0.33-0.90). Infants born to white mothers were predicted to have a 52% decrease in LBW occurrence compared with black mothers and mothers of other races (OR = 0.48; CI = 0.37-0.63). Infants born to mothers aged 25 years and above were 38% more likely to experience LBW occurrence compared with infants born to mothers under the age of 25 years (OR = 1.38; CI = 1.08-1.76).

For the years 2015, environmental arsenic concentrations associated with private wells and surface soils were found to have no significant association with LBW in our GLM. Our findings showed that infants born to white fathers were 3.15 times as likely to experience LBW occurrence compared with black fathers and fathers of other races (OR = 3.15; CI = 1.67-6.11). Infants born of fathers above the age of 25 years were predicted to have a 32% decrease in the likelihood of LBW occurrence (OR = 0.68; CI = 0.46-0.99). Infants of mothers who had other forms of medical payment were 50% less likely to experience LBW occurrence compared with private and nonprivate insurance (OR = 0.50; CI = 0.31-0.78). Infants born to mothers of other races, excluding black and white, are 68% less likely to experience LBW occurrence compared with white and black mothers (OR = 0.32; CI = 0.14-0.65).

Infants born to white mothers had an 84% decreasing probability of experiencing LBW occurrence compared with black mothers and mothers of other races (OR = 0.16; CI = 0.08-0.30). Infants born from mothers who used tobacco were 95% more likely to experience LBW occurrence (OR = 1.95; CI = 1.3-2.86).

Prediction model of LBW occurrence

Predicted probability models were created to estimate the risk of LBW occurrence by census tract for the three years. By calculating the measures of effect from our best-fitted GLMs for each
### Table 1. Demographic and birth characteristics of parental and infants for the years of 2005, 2010, and 2015.

| DEMOGRAPHIC AND BIRTH CHARACTERISTICS (N AND %) | 2005 (N = 5845) | 2010 (N = 5569) | 2015 (N = 5770) |
|-------------------------------------------------|-----------------|-----------------|-----------------|
| **Maternal age**                                |                 |                 |                 |
| < 25                                            | 2493 (42.7)     | 2236 (40.2)     | 1885 (32.7)     |
| ≥ 25                                            | 3351 (57.3)     | 3333 (59.8)     | 3885 (67.3)     |
| Not available                                   | 1 (0.0)         | 0 (0.0)         | 0 (0.0)         |
| **Paternal age**                                |                 |                 |                 |
| < 25                                            | 1539 (26.3)     | 1338 (24.0)     | 1022 (17.7)     |
| ≥ 25                                            | 3896 (66.7)     | 4231 (76.0)     | 4001 (69.3)     |
| Not available                                   | 410 (7.0)       | 0 (0.0)         | 747 (12.9)      |
| **Maternal ethnicity**                         |                 |                 |                 |
| Hispanic                                        | 217 (3.7)       | 218 (3.9)       | 241 (4.2)       |
| Non-Hispanic                                    | 5553 (95.0)     | 5243 (94.1)     | 5436 (94.2)     |
| Other or Unknown Hispanic                       | 75 (1.3)        | 108 (1.9)       | 91 (1.6)        |
| Not available                                   | 0 (0.0)         | 0 (0.0)         | 2 (0.0)         |
| **Paternal ethnicity**                         |                 |                 |                 |
| Hispanic                                        | 179 (3.1)       | 176 (3.2)       | 189 (3.3)       |
| Non-Hispanic                                    | 4461 (76.3)     | 4158 (74.7)     | 4427 (76.7)     |
| Other or Unknown Hispanic                       | 53 (0.9)        | 1235 (22.2)     | 82 (1.4)        |
| Not available                                   | 1152 (19.7)     | 0 (0.0)         | 1072 (18.6)     |
| **Maternal race**                               |                 |                 |                 |
| Black                                           | 1286 (22.0)     | 1278 (22.9)     | 1276 (22.1)     |
| Other races                                     | 331 (5.7)       | 315 (5.7)       | 371 (6.4)       |
| White                                           | 4228 (72.3)     | 3976 (71.4)     | 4121 (71.4)     |
| Not available                                   | 0 (0.0)         | 0 (0.0)         | 2 (0.0)         |
| **Paternal race**                               |                 |                 |                 |
| Black                                           | 813 (13.9)      | 810 (14.5)      | 896 (15.5)      |
| Other races                                     | 229 (3.9)       | 1412 (25.4)     | 270 (4.7)       |
| White                                           | 3655 (62.5)     | 3347 (60.1)     | 3533 (61.2)     |
| Not available                                   | 1148 (19.6)     | 0 (0.0)         | 1071 (18.6)     |
| **Maternal education**                         |                 |                 |                 |
| < High school                                   | 1058 (18.1)     | 901 (16.2)      | 730 (12.7)      |
| ≥ High school                                   | 4783 (81.8)     | 4649 (83.5)     | 5031 (87.2)     |
| Not available                                   | 4 (0.1)         | 19 (0.3)        | 9 (0.2)         |
| **Paternal education**                         |                 |                 |                 |
| < High school                                   | 584 (10.0)      | 467 (8.4)       | 443 (7.7)       |
| ≥ High school                                   | 4109 (70.3)     | 3910 (70.2)     | 4252 (73.7)     |
### Table 1. Social and lifestyle characteristics of mothers for the years of 2005, 2010, and 2015.

| SOCIAL AND LIFESTYLE CHARACTERISTICS  | 2005 (N = 5845) | 2010 (N = 5569) | 2015 (N = 5770) |
|---------------------------------------|-----------------|-----------------|-----------------|
| Not available                         | 1152 (19.7)     | 1192 (21.4)     | 1075 (18.6)     |
| Sex of infant                         |                 |                 |                 |
| Female                                | 2842 (48.6)     | 2755 (49.5)     | 2839 (49.2)     |
| Male                                  | 3003 (51.4)     | 2813 (50.5)     | 2931 (50.8)     |
| Not available                         | 0 (0.0)         | 1 (0.0)         | 0 (0.0)         |
| Birth weight                          |                 |                 |                 |
| Birth weight ≥ 2500 g                 | 5303 (90.7)     | 5028 (90.3)     | 5199 (90.1)     |
| LBW (< 2500 g)                        | 542 (9.3)       | 541 (9.7)       | 569 (9.9)       |
| Birth facility                        |                 |                 |                 |
| Hospital                              | 5793 (99.1)     | 5538 (99.4)     | 5724 (99.2)     |
| Out of hospital                       | 51 (0.9)        | 26 (0.5)        | 44 (0.8)        |
| Not available                         | 1 (0.0)         | 5 (0.1)         | 2 (0.0)         |
| Source pay                            |                 |                 |                 |
| Private insurance                     | 2173 (37.2)     | 1869 (33.6)     | 1907 (33.1)     |
| Nonprivate insurance                  | 2957 (50.6)     | 2982 (53.5)     | 3009 (52.1)     |
| Other                                 | 713 (12.2)      | 714 (12.8)      | 849 (14.7)      |
| Not available                         | 2 (0.0)         | 4 (0.1)         | 5 (0.1)         |

Abbreviation: WIC, Women, Infants, and Children.

### Table 2. Social and lifestyle characteristics of mothers for the years of 2005, 2010, and 2015.

| SOCIAL AND LIFESTYLE CHARACTERISTICS  | 2005       | 2010       | 2015       |
|---------------------------------------|-----------|-----------|-----------|
| Mother in WIC program                 |           |           |           |
| Yes                                   | 2796 (47.8)| 3079 (55.3)| 2841 (49.2)|
| No                                    | 3038 (52.0)| 2453 (44.0)| 2924 (50.7)|
| Not available                         | 11 (0.2)  | 37 (0.7)  | 5 (0.1)   |
| Maternal tobacco use                  |           |           |           |
| Yes                                   | 605 (10.4) | 606 (10.9) | 547 (9.5) |
| Yes, but quit                         | 127 (2.2)  | 53 (1.0)   | 0 (0.0)   |
| No                                    | 5112 (87.5)| 4870 (87.4)| 5196 (90.1)|
| Not available                         | 1 (0.0)   | 40 (0.7)   | 27 (0.5)  |
| Maternal alcohol use                  |           |           |           |
| Yes                                   | 26 (0.4)  | 32 (0.6)  | 43 (0.7)  |
| No                                    | 5816 (99.5)| 5530 (99.3)| 5722 (99.2)|
| Not available                         | 3 (0.1)   | 7 (0.1)   | 5 (0.1)   |

Abbreviation: WIC, Women, Infants, and Children.
year in R, we were able to predict the likelihood of LBW occurrence at the census tract level for both counties. The models predicted higher incidences of LBW in smaller census tract clusters in Escambia County and Santa Rosa County for all years (Figure 2A to C). These areas with high predicted probabilities were primarily around the urban area of Pensacola and neighboring cities. In all years, low and moderate cases of LBW were predicted for most of the census tracts in both counties, and this could be due to the distribution of mothers above the age of 25 years who gave birth, which was significant in our logistic regression model. The distribution of LBW incidences may account for drastic changes we see in 2010 compared with the other year as more census tracts were noted to have predicted LBW occurrence.

From the ROC plot, estimates of the AUC were used to test the validity and utility of all the predictors used in our LBW predictive models (Supplemental Figure S1). From the AUC, we found that all the models were similar in predicting the outcome of LBW occurrence for each year: 2005 model (AUC = 0.60), 2010 (AUC = 0.65), and 2015 (AUC = 0.60).

Discussion
Concentrations of arsenic in soil and water were not associated with the incidence of LBW by census tract, meaning no significant association was found between environmental arsenic concentrations in the census tract of maternal residence and the occurrence of LBW. Many studies have noted a relationship between arsenic exposure and LBW. However, some studies have found inconsistencies in the relationship when looking at arsenic concentrations and LBW. Limitations in this study may account for the lack of association between environmental arsenic and LBW. One limiting factor is the lack of direct exposure measurements for mothers, which would explain how much background arsenic is absorbed by the mother and the infant.

Mothers can be exposed to arsenic through water, soil, food, or dust, with its main routes of exposure being ingestion of food and contaminated water and inhalation of polluted air and chemicals. Arsenic can persist in pregnant mothers’ tissues for up to 7 days but can vary; however, repeated measures

![Figure 1. Odds ratios of GLM for the years of (A) 2005, (B) 2010, and (C) 2015. GLM indicates generalized linear model; LBW, low birth weight. P-value (reference): *** P < 0.001; ** P < 0.01; * P < 0.05.](image-url)
of exposure to arsenic due to diet, smoking, or drinking water can prolong exposure during pregnancy. Food source could be local, domestic, or imported, depending on the mother’s preference, which can be hard to assess in studies that focus on passive measures. Passive measures of arsenic exposure are sometimes not the most reliable indicators of associations due to lack of knowledge of direct exposure and different routes of arsenic exposures the mothers may encounter. This limitation could be strengthened by assessing biomarkers for arsenic exposure in pregnant mothers and comparing those concentrations with the confounding factors associated with LBW.

Using biomonitoring, Laine et al found that more than half the mothers in their study had arsenic concentrations in their drinking water that exceeded 10 µg-As/L (the World Health Organization guideline limit) and was linked to urinary concentration in mothers and infant gestational age and length. Also, humans are exposed to other xenobiotics and inorganic pollutants, which might cause LBW and should be accounted for in future studies. This study could also be strengthened by increasing the sample size of private wells in both counties. The usage of spatial autocorrelation techniques is reliant on a large and highly disturbed sample population to estimate arsenic concentration accurately. With our small sample size, it may be difficult to compare soil samples, which were a mix of secondary and collected data and private well data. The private wells were secondary data that rely on self-reporting from well owners and government assistance in monitoring concentration. Access to these unreported private wells could strengthen our spatial autocorrelation techniques (IDW) and give a more precise estimate of arsenic from private drinking wells.

It was also found that the mothers’ education level and social lifestyle choices, such as maternal tobacco usage, while pregnant increased the likelihood of LBW in infants. Tobacco has been known to contain heavy metals such as lead and arsenic and that arsenic in biological samples of smokers was found to be higher than nonsmokers. Similar to previous studies, a strong association between maternal smoking and the occurrence of LBW and preterm births were reported was noted in 2015. It is known that smoking while pregnant and secondhand exposure to tobacco during late pregnancy could lead to LBW and preterm births. Smoking has been linked to LBW, and the more tobacco smoked during pregnancy has been linked to intrauterine growth retardation, thus affecting birth weight.

In addition to social lifestyle choices, socioeconomic status can affect pregnancy and infants’ birth weight; the mother’s education level, insurance type, and WIC enrollment were used as a proxy for socioeconomic status in this study. We found that mothers with high school education or higher had a lower risk of birthing an infant with LBW. Maternal stress is often defined as a mix of low education and income, marital status (single), age, and ethnicity and associated stressors, and has been found to increase the risk of LBW and preterm births. It has also been noted that high poverty and low education at the neighborhood level were found to increase the risk of LBW. Women from poor neighborhoods and education levels below 14 years of schooling have been found to have increased risk of adverse birth outcomes such as LBW, preterm birth, stillbirth, neonatal, and postnatal death.

In this study, higher predicted LBW was noted in areas with higher populations and, by relation, more births. These higher populated urban areas may see less environmental arsenic due to better water treatment practices and government assistance compared with rural regions. The prediction models had high accuracy in reporting LBW occurrence, as noted in the ROC curves and, thus, the GLM results may provide useful insight into factors related to LBW.

Figure 2. Maps created from the response predictions of the GLMs depicting the predicted probability of LBW in Escambia and Santa Rosa for each of the three years (2005, 2010, and 2015). GLM indicates generalized linear model; LBW, low birth weight.
Conclusions
In conclusion, no significant association was found between environmental arsenic concentrations and the occurrence of LBW, suggesting that the environmental contamination of the pregnant mother’s census tract level may not be a useful proxy in assessing risk for LBW occurrence. Results showed that smoking and the mother’s race was associated with LBW in infants. In addition, women who had private insurance had a lower risk of giving birth to LBW infants in both Santa Rosa and Escambia County, Florida. These findings are consistent with previous studies and relevant to assisting in future public health studies focused on vulnerable populations. Future research should measure arsenic biomarkers in expectant mothers to allow us to assess how direct exposure to arsenic from the environment affects both mothers and infant birth outcomes as part of a cohort study.

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Author Contributions
MSR: Conceptualization, Methodology, Investigation, Formal Analysis, Visualization, Writing – original draft preparation, Writing – Reviewing and Editing.
MOHR: Supervision, Conceptualization, Methodology, Validation, Writing – Reviewing and Editing.

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Supplementary Material
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REFERENCES
1. Vahter M. Effects of arsenic on maternal and fetal health. Annu Rev Nutr. 2009;29:381-399. doi:10.1146/annurev-nutr-080508-141102.
2. Fei DL, Koesterl DC, Li Z, et al. Association between in utero arsenic exposure, placental gene expression, and infant birth weight: a US birth cohort study. Environ Health. 2013;12:37-58. doi:10.1186/1476-069X-12-58.
3. Claus Henn B, Ettersger AS, Hopkins MR, et al. Prenatal arsenic exposure and birth outcomes among a population residing near a mining-related superfund site. Environ Health Perspect. 2016;124:1308-1315. doi:10.1289/ehp.1500700.
4. Ahmad SA, Sayed MH, Barua S, et al. Arsenic in drinking water and pregnancy outcomes. Environ Health Perspect. 2001;109:629-631. doi:10.1289/ehp.0110926.
5. Huyck KL, Kile ML, Mahulidian G, et al. Maternal arsenic exposure associated with low birth weight in Bangladesh. J Occup Environ Med. 2007;49:1097-1104. doi:10.1097/JOM.0b013e318156b6a0.
6. Hall M, Gamble M, Slavovich V, et al. Determinants of arsenic metabolism: blood arsenic metabolites, plasma folate, cobalamin, and homocysteine concentrations in maternal-newborn pairs. Environ Health Perspect. 2007;115:1503-1509. doi:10.1289/ehp.9906.
7. von Ehrenstein OS, Guha Mazumder DN, Hira-Smith M, et al. Pregnancy outcomes, infant mortality, and arsenic in drinking water in West Bengal, India. Am J Epidemiol. 2006;163:662-669. doi:10.1093/aje/kwj089.
8. Gardener RM, Kippler M, Toifl P, et al. Environmental exposure to metals and children’s growth to age 5 years: a prospective cohort study. Am J Epidemiol. 2013;177:1356-1367. doi:10.1093/aje/ksw437.
9. Hughes MF, Thomas DJ, Kenyon EM. Toxicology and epidemiology of arsenic and its compounds. In: Henke K, ed. Arsenic. Chichester, UK: John Wiley & Sons; 2009:237-275. doi:10.1002/9780470741122.ch4.
10. Hoppenhain C, Ferreccio C, Browning SR, et al. Arsenic exposure from drinking water and birth weight. Epidemiology. 2003;14:593-602. doi:10.1097/00001620015314541457.
11. Farzan SF, Korick S, Li Z, et al. In utero arsenic exposure and infant infection in a United States cohort: a prospective study. Environ Res. 2013;126:24-30. doi:10.1016/j.envres.2013.05.001.
12. Scott-Richardson M, O’Hara Ruiz M, Smith R. Florida Arsenic Distribution Index: quantifying the distribution of past and present arsenic usage. Int J Environ Res Public Health. 2019;16:744. doi:10.3390/ijerph16050744.
13. Liebens J, Molhuver CJ, Rao KR. Pollution of surface soils in Escambia and Santa Rosa Counties, FL. http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.4545&rep=rep1&type=pdf.
14. Scott-Richardson M, Smith RL. mspscot2/R-low-birth-weight-and-arsenic-Rcode: release of LBW and R codes. July 2019. doi:10.5281/ZENODO.334890.
15. Team RC. R: A Language and Environment for Statistical Computing (Version 3.4. 2) [Computer software]. Vienna, Austria: R Foundation for Statistical Computing; 2017.
16. Singh N, Kumar D, Sahu AP. Arsenic in the environment: effects on human health and possible prevention. J Environ Biol. 2007;28:359-365.
17. Rahman MM, Sengupta MK, Ahamed S, et al. The magnitude of arsenic contamination in groundwater and its health effects to the inhabitants of the Jalgazi-one of the 85 arsenic affected blocks in West Bengal, India. Sci Total Environ. 2005;338:189-200. doi:10.1016/j.scitotenv.2004.06.022.
18. McDermott S, Bao W, Aelon CM, BAI, LWB. Does the metal content in soil around a pregnant woman’s home increase the risk of low birth weight for her infant? Environ Geochem Health. 2014;36:1191-1197. doi:10.1007/s10653-014-9617-4.
19. Rahman A, Vahter M, Smith AH, et al. Arsenic exposure during pregnancy and size at birth: a prospective cohort study in Bangladesh. Am J Epidemiol. 2009;169:304-312. doi:10.1093/aje/kw332.
20. Shirai S, Suzuki Y, Ohata J, Mizumoto Y. Maternal exposure to low-level heavy metals during pregnancy and birth size. J Environ Sci Health A Toxic Hazard Subst Environ Eng. 2010;45:1468-1474. doi:10.1080/109074010.2009042.
21. Chou WC, Chung YT, Chen HY, et al. Maternal arsenic exposure and DNA damage biomarkers, and the associations with birth outcomes in a general population from Taiwan. PLoS One. 2014;9:e86398. doi:10.1371/journal.pone.0086398.
22. Thomas S, Aruckle TE, Fisher M, Fraser WD, Ettinger A, King W. Metals exposure and risk of small-for-gestational age birth in a Canadian birth cohort: the MIREC study. Environ Res. 2015;140:430-439. doi:10.1016/j.envres.2015.04.018.
23. Hu X, Zheng T, Cheng Y, et al. Disturbances of heavy metals in maternal and cord blood and the association with infant birth weight in China. J Reprod Med. 2015;60:21-29. http://www.ncbi.nlm.nih.gov/pubmed/25745747. Accessed July 18, 2019.
24. Bloom MS, Buck Louis GM, Sundaram R, Maisog JM, Steurerwald AJ, Parsons PJ. Birth outcomes and background exposures to select elements, the Longitudinal Investigation of Fertility and the Environment (LIFE). Environ Res. 2015;138:118-129. doi:10.1016/j.envres.2015.01.008.
25. Röllin HB, Channa K, Olotua BG, Oddy JO. Evaluation of in utero exposure to arsenic in South Africa. Sci Total Environ. 2017;575:338-346. doi:10.1016/j.scitotenv.2016.10.044.
26. Neeti K, Prakash T. Effects of heavy metal poisoning during pregnancy. Int Rev J Environ Sci. 2013;2:88-92.
27. Jia L, Zhang L, Li Z, Liu Y, Ren A. Placental concentrations of mercury, lead, cadmium, and arsenic and the risk of neural tube defects in a Chinese population. Reprod Toxicol. 2013;35:25-31.
28. Laine JE, Bailey KA, Rubio-Andrade M, et al. Maternal arsenic exposure, arsenic methylation efficiency, and birth outcomes in the biomarkers of exposure to arsenic (BEAR) pregnancy cohort in Mexico. Environ Health Perspect. 2015;123:186-192. doi:10.1289/ehp.1300747.
29. Lazarević K, Nikolić D, Stosić L, Mihljević S, Videnović J, Bogdanović D. Determination of lead and arsenic in tobacco and cigarettes: an important issue of public health. Cent Eur J Public Health. 2012;20:62-66. doi:10.21101/cejph.a3728.
30. Ventura SJ, Hamilton BE, Mathews TJ, Chandra A. Trends and variations in smoking during pregnancy and low birth weight: evidence from the birth certificate, 1990-2000. Pediatrics. 2003;111:1176-1180. https://www.ncbi.nlm.nih.gov/pubmed/12728134. Accessed July 18, 2019.
31. Pereira PF, Da Mata FA, Figueredo AC, de Andrade KR, Pereira MG. Maternal active smoking during pregnancy and low birth weight in the Americas: a
systematic review and meta-analysis. *Nicotine Tob Res.* 2017;19:497-505. doi:10.1093/ntr/ntw228.

32. Soneji S, Beltrán-Sánchez H. Association of maternal cigarette smoking and smoking cessation with preterm birth. *JAMA Netw Open.* 2019;2:e192514. doi:10.1001/jamanetworkopen.2019.2514.

33. Jaddoe VWV, Troe Hofman A, Mackenbach JP, Moll HA, Steegers EA, Witterman JC. Active and passive maternal smoking during pregnancy and the risks of low birthweight and preterm birth: the generation R study. *Paediatr Perinat Epidemiol.* 2008;22:162-171. doi:10.1111/j.1365-3016.2007.00916.x.

34. Horta BL, Victora CG, Menezes AM, Halpern R, Barros FC. Low birthweight, preterm births and intrauterine growth retardation in relation to maternal smoking. *Paediatr Perinat Epidemiol.* 1997;11:140-151. doi:10.1046/j.1365-3016.1997.d01-17.x.

35. Zheng W, Suzuki K, Tanaka T, Kohama M, Yamagata Z. Association between maternal smoking during pregnancy and low birthweight: effects by maternal age. *PLoS ONE.* 2016;11:e0146241. doi:10.1371/journal.pone.0146241.

36. Mannocci A, Vaschetto C, Semyonov L, et al. Maternal smoking and sociodemographic characteristics in correlation with low birth weight: a Turin (piedmont) study. *Zdr Varst.* 2014;53:221-225. doi:10.2478/zjph-2014-0022.

37. Nkansah-Amankra S, Dhawain A, Hussey JR, Luchok KJ. Maternal social support and neighborhood income inequality as predictors of low birth weight and preterm birth outcome disparities: analysis of South Carolina pregnancy risk assessment and monitoring system survey, 2000-2003. *Matern Child Health J.* 2010;14:774-785. doi:10.1007/s10995-009-0508-8.

38. Luo Z-C, Wilkins R, Kramer MS; for the Fetal and Infant Health Study Group of the Canadian Perinatal Surveillance System. Effect of neighbourhood income and maternal education on birth outcomes: a population-based study. *CMAJ.* 2006;174:1415-1420. doi:10.1503/cmaj.051096.

39. Campbell EE, Seabrook JA. The influence of socioeconomic status on adverse birth outcomes. *Can J Midwifery Pract Res.* 2016;15:10-20.

40. Allen A, Dávila JD, Hofmann P. The peri-urban water poor: citizens or consumers? *Environ Urban.* 2006;18:333-351. doi:10.1177/0956247806069608.