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Country Department Southern Cone

Sebastian Miller
J. Cristobal Ruiz-Tagle

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Sebastian Miller
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J. Cristobal Ruiz-Tagle*
Environmental Defense Fund

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Abstract

This paper examines the effect of exposure to air pollution on the probability of a pregnancy ending in a stillbirth delivery. We use official registry data of pregnancy outcomes and municipality-week level data on PM10 and CO air pollution concentrations for 84 municipalities in Chile over the period 2008-2015. Using a hazard function approach to account for *time at risk* throughout the duration of pregnancies, we find that acute exposure to PM10 and CO pollution has a significant adverse effect on the probability of stillbirth delivery, and find no significant effect of chronic exposure to these pollutants. For a stillbirth rate of 5.7 per every thousand pregnancies in our sample, we find that a one-standard-deviation increase in acute exposure to PM10 pollution (S.D.=23.34) results in a 10.5 percent increase in the probability of stillbirth. Similarly, a one-standard-deviation increase in acute exposure to CO pollution (S.D.=.54) results in a 5.3 percent increase in probability of stillbirth. Furthermore, we further examine the most likely pathway in which air pollution adversely affects a fetal death that results in stillbirth delivery (via lack of oxygen flowing to the fetus that may eventually result in a stillbirth due to hypoxia). We find indeed larger effects of acute exposure to air pollution (both PM10 as well as CO) on the probability of stillbirth due to hypoxia.

* Email: cristobal.ruiztagle@gmail.com

I. Introduction

There is a large literature documenting the effects of exposure to airborne air pollutants on human health across several cities and regions in both developing as well as the industrialized world (Nadadur and Hollingsworth 2015; Šrám et al. 2005). Much of the literature focuses on the adverse effects on particularly vulnerable subpopulations, namely infants and the elderly. In this paper, we examine the adverse effects of airborne air pollution on a fundamental pregnancy outcome; that is, whether a pregnancy results in a livebirth delivery, or whether a fetal death occurs so that the pregnancy ends in a stillbirth delivery. Although there is a growing literature documenting the adverse effects of air pollution on several pregnancy outcomes —such as low birth weight and preterm birth—there is little evidence on the adverse effects of air pollution on the probability of pregnancy ending in a stillbirth delivery (Nadadur and Hollingsworth 2015). In fact, the empirical literature is, so far, inconclusive on whether air pollution has any significant effect on fetal deaths and stillbirth deliveries (Veras et al. 2015).

In this paper, we look at the effects of two airborne pollutants, particulate matter of diameter less than 10 microns (PM10) and Carbon Monoxide (CO) on the probability of pregnancy resulting in a stillbirth delivery. We examine the effects of these two pollutants throughout two distinctive periods of exposure: *chronic* exposure, that is, exposure throughout the entire length of pregnancy, and *acute* exposure, exposure on the week of delivery (either a livebirth or stillbirth).

On the other hand, as air pollution diminishes the capacity of the pregnant woman to transmit nutrients and oxygen to the fetus, a severe inhibition of this capacity may result in a fetal death due to the lack of oxygen in the fetus. Therefore, we also examine the effects of these two pollutants and these two periods of exposure on the most likely consequences of air pollution, a stillbirth due to hypoxia.

We find evidence of adverse effects of *acute* exposure to both PM10 and CO pollution on the probability of stillbirth. Furthermore, we find larger effects for the probability of stillbirth due to hypoxia. Conversely, we find only weak evidence of *chronic* exposure to these air pollutants.

The next section briefly reviews the existing literature. We present the data we employed in section III and in section IV we explain the statistical methods we use for econometric analysis. Section V presents econometric results and in section VI we contrast these results with those of the existing related literature. We discuss on the policy implications of our results in section VII and present concluding remarks in section VIII.

II. Literature Review

There is a growing body of literature that looks at the effects of air pollution on births' health outcomes. For instance, Edwards et al. (2015) reviews 139 articles and extracts extensive information about the effects of exposure to criteria pollutants (carbon monoxide, particulate matter, nitrogen dioxide, sulfur dioxide, ozone) polycyclic aromatic hydrocarbons and traffic-related air pollutants. Edwards et al. (2015)'s review examines six pregnancy outcomes: gestational-age, preterm birth, small for gestational-age, full term low birth-weight and both continuous and threshold low birth-weight. However, few studies look at the association between maternal exposure to air pollutants and fetal death or stillbirth delivery. Moreover, most of these studies examine the effects of exposure to air pollution during specific periods of the pregnancy (the days immediately before delivery, specific months and trimesters of pregnancy) as well as exposure during the entire length of pregnancy. The table in Appendix 1 summarizes the existing related literature and its main findings.

The existing literature that looks at the effects of exposure to air pollution on the probability of stillbirth finds (i) significant effects of *acute* exposures (just a few days immediately before delivery) to *peak* levels of carbon monoxide pollution (Faiz et al. 2013)[1]; or,

[1] *Peak* levels of pollution refers to pollution events in which pollution is higher than the sum of the mean pollution and inter-quantile range (> mean + IQR). If pollution has a normal distribution, this would be equivalent to episodes of pollution in the top 2.5% percent of its distribution.

(ii) only weak evidence of exposure to air pollution when measured in levels, significant only at 90% confidence (Pereira et al. 1998)[2]. Regarding exposure to particular matter, both PM10 and PM2.5 (also known as fine particulate matter), the literature finds significant effects of different windows of exposure (third trimester, first two months, and entire length of pregnancy) and at different intensities. Whereas Kim et al. (2007) find statistical evidence on the probability of stillbirth from exposure to PM10 during the third trimester of pregnancy in Seoul, Korea; Hwang et al. (2011) find evidence of exposure during the first two months of pregnancy in Taiwan. Furthermore, De Franco et al. (2015) find evidence of exposure to *peak* levels of PM2.5 during the third trimester of pregnancy in the State of Ohio; and Green et al. (2015) find only weak evidence of exposure to levels of PM2.5 for the entire pregnancy in the State of California.

However, because of to the focus on trimester-specific exposure, the existing literature misses those stillbirth deliveries that could have been due to exposure to air pollution but did not lasted through the third trimester of pregnancy. This misses those pregnancies that may have already resulted in a stillbirth but that did not last passed the second trimester of pregnancy.[3] If those early stillbirth deliveries are due to exposure to air pollution, then this selection of observations introduces a (selection) bias. This bias would lead us to accept the null hypothesis of no statistically significant effect of air pollution on stillbirth deliveries. Same is true for those studies that focus on month-specific exposure through the eighth and ninth month of pregnancy.

Moreover, the existing literature does not find conclusive evidence of an effect of exposure to CO pollution on stillbirth deliveries (other than at *peak* levels), nor does it find evidence of acute exposure to PM10 pollution on stillbirth deliveries. In this paper we examine the effects on the probability of stillbirth of both *acute* and *chronic* exposure to PM10 and CO pollution. Moreover, the existing literature fails to control for potential confounders such as

[2] Notice that Pereira et al. (1998) is an *ecological study*. Most ecological studies have only one city-wide measure of pollution exposure and do not include mother-specific controls, such as mother's medical history, demographics and socioeconomic characteristics.

[3] In fact, as we will show below, about half of the stillbirth deliveries in our sample to not last passed the second trimester of pregnancy (passed the twenty sixth week of pregnancy).

those given by seasonal patterns of conception and delivery, and, most importantly, for possible location-specific unobservable effects (for example, municipality-specific confounders that may introduce bias to the estimation of the effect of exposure to air pollution). In this paper we address all these issues and, in doing so, we do not introduce a selection bias due to missing observations on pregnancies that resulted in stillbirth before reaching the third trimester of pregnancy.[4] Furthermore, we control for potential seasonal and location-specific confounders.

Moreover, in this paper we also look at cause-specific stillbirths by focusing on those stillbirths that are most likely to be driven by exposure to air pollutants; stillbirths due to hypoxia.[5] Thus, in this paper we also we provide supporting evidence of the more likely pathway in which air pollution may affect the fetus, via severely inhibiting the capacity of the pregnant woman to provide the necessary nutrients and oxygen to the fetus. Therefore, we also examine whether exposure to air pollution is more likely trigger stillbirths due to hypoxia.

III. Data

We obtained individual-level data on stillbirth deliveries from official records of the Department of Health Statistics of Chile's Ministry of Public Health. This data contains nationwide hospitals' records on those women who have delivered livebirths and stillbirths. The data records the municipality where the mother lived at the time of delivery as well as the date of delivery. This data also provides information on mother's pregnancy histories (number of

[4] We refer to acute exposure as exposure to air pollution on the week of delivery, and to chronic exposure as to average exposure throughout the entire duration of the pregnancy.

[5] Hwang et al. (2011) and Green et al. (2015) suggest that one possible pathway linking air pollution exposure and stillbirth would be via the oxygen uptake by the fetus. They suggest that pollutants may increase maternal methemoglobin levels, which can oxidize fetal hemoglobin levels and inhibit the oxygen transport needed by the fetus (Green et al., 2015). Thereby, a severe lack of oxygen by the fetus could potentially result in fetal death due to hypoxia. Furthermore, Veras et al. (2015) suggest that air pollution can affect the utero-placental and umbilical cord flow and consequently the transport of glucose and oxygen through the placenta. Airborne particulate matter can affect pregnancy outcomes due to inflammation of the placenta, which could impair transplacental nutrient exchange thus affecting nutrition of the fetus and reducing oxygenation of maternal blood Kannan et al. (2007). Similarly, Maisonet et al. (2004) suggest that alveolar inflammation can lead to increased difficulties with blood flow, impacting placental functions.

previous livebirths deliveries, whether she had delivered a stillbirth in the past, etc), and mother's personal characteristics (mother's age, years of schooling, etc). The data also reports the number of weeks of pregnancy at the time of delivery. We restrict our data to pregnancies lasting more than thirteen weeks as miscarriages of pregnancies lasting less than that often go unreported.[6] In this way, we have data on 4,915 stillbirth deliveries and 857,820 livebirth deliveries over the period 2008-2015, yielding a rate of 5.7 stillbirths for every thousand pregnancies lasting more than thirteen weeks.

Additionally, the health data allows us to categorize those stillbirths according to the leading cause of death as diagnosed by the physician at the time of delivery. When a pregnancy ends in a delivery of a stillbirth, the physician must write down the leading diagnostic according to the tenth version of the international classification of disease (ICD-10). In our dataset, of those 4,915 stillbirth deliveries in our data, 1,365 correspond to stillbirth due to hypoxia whereas 3,550 correspond to stillbirths due to other causes (different from hypoxia). This yields a rate of 1.6 stillbirths due to hypoxia out of every thousand pregnancies (lasting more than thirteen weeks).

Air pollution data comes from Chile's Air Quality National Information System; a network of air quality monitoring stations of Chile's Environmental Ministry. The monitoring stations provide daily records of both particulate matter with diameter less than 10 microns per cubic meter (PM10) and carbon monoxide concentrations (CO) in parts per million. We build a municipality-week level dataset by assigning pollution exposure to each municipality for each week as follows. First, we take the week-average pollution from each monitoring station. Second, we construct a spatial mapping of pollutants by using the geographical coordinates of the monitoring stations to impute pollution levels to spatial reference points. As we assume that most people live within certain vicinity of schools we use the geographical location of schools within a given municipality as spatial reference points to spatially impute air pollution

[6] We also conducted analysis restricting the sample to those pregnancies that lasted more than twenty weeks. The results remain largely unchanged.

concentrations.[7] That is, we impute air pollution concentrations to the geographical location of each school within a municipality and then we average the imputed air pollution data across all (selected) schools for each municipality.[8] In this way we generate a municipality-week dataset for air pollution data that we then merge with the individual-level pregnancy data outlined above.

A. Correlation between Air Pollution and Pregnancy Outcomes

Our data allows us to assign municipality-level PM10 and CO pollution on the week of delivery of each pregnancy (either a livebirth or stillbirth). We refer to this as *acute* exposure. Furthermore, since we also have data on the number of weeks of pregnancy, we also assign average PM10 and CO pollution exposure throughout the entire length of pregnancy, to which we refer to as *chronic* exposure. Figure 1 below plots average chronic and acute exposure to PM10 pollution (left plot) and average chronic and acute exposure to CO pollution (right plot) for each pregnancy outcome (either a livebirth or a stillbirth delivery). The figure also adds its corresponding 95% confidence intervals.

-- Figure 1 about here --

The left plot of Figure 1 shows that the average *chronic* exposure to PM10 pollution of those livebirths is not statistically different from the average *chronic* exposure to PM10 pollution of those pregnancies that ended in a stillbirth. The same is true for the average *acute*

[7] Furthermore, we restrict our sample of schools to those that are located (i) at a distance of no more than 5 kilometers from the nearest air quality monitoring station, and, (ii) between 5 to 10 kilometers to the nearest monitoring station, but no more than 20 kilometers to the second-nearest monitoring station. If there is more than one monitoring station within a 20 kilometer radius from the school, we take the average by weighting the pollution data of each station by the inverse of the distance to the school. This methodology for spatial imputation of pollution data is known as the Inverse of the distance as weights (IDW) and has been previously been employed in (Currie and Neidell 2005) and (Arceo, Hanna, and Oliva 2016), to name a few.

[8] We only consider those municipalities for which we have pollution data for at least 70% of the weeks of any given year.

exposure to PM10 pollution. However, this figure is somehow different for CO pollution. The right plot of Figure 1 shows that the average chronic exposure to CO pollution of those livebirths is higher than that of stillbirths, whereas this difference becomes statistically insignificant for *acute* exposure to CO pollution.

We also look at cause-specific stillbirth, by distinguishing between those stillbirths diagnosed as due to hypoxia from those diagnosed as due to causes other than hypoxia ('SB Other' in Figure 2 below). The left plot of Figure 2 below shows that the average chronic exposure to PM10 pollution for those stillbirths due to hypoxia is statistically higher than that of both stillbirth due to causes other than hypoxia (SB Other) and for livebirths. This difference is even larger for acute exposure to PM10. On the other hand, although the right plot of Figure 2 shows that the average chronic exposure to CO pollution for those stillbirths due to hypoxia is not statistically different from that of livebirths. However, the average acute exposure to CO pollution is statistically higher than both that of stillbirths due to other causes and that of livebirths.

-- Figure 2 about here --

In addition, Table 1 below shows both mean and standard deviation of PM10 and CO pollution (both for chronic and acute exposure), for pregnancies that resulted in a livebirth or a stillbirth, and within those stillbirths, whether the stillbirth was due to hypoxia or due to other causes. These are the same numbers that we use to generate both Figure 1 and Figure 2. Also, Table 1 presents the number of observations for each category.[9]

-- Table 1 about here --

[9] So that 95% confidence intervals shown in figures 1 and 2 can easily be calculated by using the simple formula $Mean\ \pm SD/\sqrt{N}$.

In addition to presenting statistics for PM10 and CO pollutants by birth outcome, Table 1 above also presents descriptive statistics (means and standard deviations) for mother's personal characteristics (mother's age, history of past stillbirths, marital status and years of education) and the season of the year at both the beginning and at the end of the pregnancy (season of conception and season of delivery). Table 1 above shows that the average age at time of delivery is statistically higher for those pregnant women that delivered a stillbirth than for those that delivered a livebirth. However, when looking at the average age of those that delivered a stillbirth due to hypoxia, the average age of those women is not statistically different from the average age of those women who delivered a livebirth. Those who delivered a stillbirth due to causes other than hypoxia (SB Other) are statistically older than those that delivered livebirths. Table 1 also shows that those women that delivered a stillbirth (either due to hypoxia or due to other causes) present a higher incidence of history of delivering stillbirths in the past (41 per every thousand pregnant women) than those that delivered a livebirth (19 per every thousand pregnant women). Those women that delivered a stillbirth are more likely to be single (39 percent) and have fewer years of education (12.9) than those that delivered a livebirth (of which 33 percent are single and have an average of 12.6 years of education). Finally, the bottom panel of Table 1 shows descriptive statistics for season of conception and season of delivery. Among those that delivered a stillbirth, 29 percent of them became pregnant during the winter, which is statistically higher than that of those women that delivered a stillbirth and became pregnant during any other season of the year. This difference is further accentuated when focusing on those stillbirths due to hypoxia (31 percent). In sum, Table 1 shows that there are important differences in the personal characteristics and the patterns of pregnancy between those women that delivered a stillbirth and those that delivered a livebirth. In assessing the effect of air pollution exposure on pregnancy outcomes we explicitly control for the personal characteristics of the woman as well as for the seasonal pattern of the pregnancy.

B. Stillbirth Rates and Air Pollution across municipalities and time

To further explore the relationship between air pollution and pregnancy outcomes we look at the average stillbirths rates and pollution exposure both across different municipalities and across time, for the period 2008-2015. Figure 3 below presents average acute exposure to PM10 and CO pollution and stillbirth rate (upper plots) and rate of stillbirth due to hypoxia (lower plots) for the 84 municipalities in our dataset, where each blue dot represents average for each municipality. We have added a trend (red line) to highlight the possible correlations between these variables. The figure suggests that, when aggregated at the municipality level, there is little correlation between average acute exposure to PM10 pollution and average stillbirth rate across municipalities (upper left plot). However, the figure suggests a weak positive correlation between average acute exposure to CO pollution and stillbirth rate across municipalities (upper right plot). The lower plots of Figure 3, however, show a relatively stronger positive correlation between average acute exposure to PM10 and CO pollution and stillbirth rate due to hypoxia across municipalities (lower plots). On the other hand, the plots for chronic exposure to both PM10 and CO pollution (omitted here) present very similar patterns as those of acute exposure, in Figure 3.

-- Figure 3 about here –

On the other hand, Figure 4 presents month-average acute exposure to PM10 and CO pollution and stillbirth rates and stillbirth due to hypoxia, for the period 2008-2015. The figure suggests little serial correlation between acute exposure to PM10 and CO pollution and stillbirth (upper plots), as well as little serial correlation between acute exposure air pollution and stillbirth due to hypoxia (lower plots).

-- Figure 4 about here –

C. Variation of air pollutants and stillbirth

Our analysis thus far suggests that there is a positive correlation between acute exposure to air pollution and stillbirth delivery, and a stronger positive correlation between acute exposure to air pollution and stillbirth due to hypoxia. In this section we further examine how the *variation* in exposure to air pollution relates to the variation in stillbirth rate and stillbirth due to hypoxia.[10] Table 2 below presents means and standard deviations of stillbirth rate for all causes, stillbirth rate due to hypoxia and both chronic as well as acute exposure to PM10 and CO pollution for the sample of 84 municipalities over the period 2008-2015. The table shows that the stillbirth rate (all causes) in our sample is 0.057, and the stillbirth rate due to hypoxia is 0.016.

Also, as it can be expected, Table 2 shows that both chronic and acute exposure yield pretty similar means but they differ in their variation in terms of their respective standard deviations (where the more disaggregated week-level acute pollution exposure presents much larger standard deviations).

Table 2 also presents a decomposition of the standard deviation across municipalities and weeks. The table shows that the overall variation of stillbirth rate and stillbirth due to hypoxia is evenly distributed across the space and time dimension of our data (that is, across both municipalities and weeks), ranging from 9 percent of the overall variation (for municipality-level variation of stillbirth rate) to 14 percent (for both the municipality-level variation of stillbirth due to hypoxia as well as for the week-level variation of stillbirth). For chronic exposure to PM10 pollution, the variation is also evenly distributed across municipalities and time (weeks) at 67 percent and 65 percent of the total variation, respectively. However, the more disaggregated measure for PM10 pollution, acute exposure, varies largely across time (weeks), representing 79 percent of the overall variation of acute exposure to PM10 pollution. On the other hand, most of the variation of exposure to CO

[10] The regression analysis discussed in detail in the subsequent section exploits this variation to examine how exposure to air pollutants affects stillbirth rates.

pollution occurs across weeks for both chronic exposure to CO (68 percent) as well as for acute exposure to CO (77 percent).

-- Table 2 about here --

IV. Duration of Pregnancies and Hazard Function Approach

A. Duration of Pregnancies by Pregnancy Outcome

One of the fundamental differences between stillbirths and livebirths is that the pattern of duration of pregnancy of those stillbirths is remarkable different to that of livebirths. Most fetal deaths and stillbirths deliveries occur before full-term gestation.[11] Figure 5 below presents histograms for both livebirths and stillbirths according to the number of weeks of pregnancy at the time of delivery. The figure below show that whereas most livebirth deliveries occur between weeks 38 and 40 (indeed, more than 75 percent of total livebirth deliveries), very few stillbirths occur during that period of gestation (less than 10 percent of total stillbirth deliveries). Stillbirth deliveries seem to distribute relatively evenly over the entire pregnancy, with a relatively larger amount of stillbirth deliveries concentrated between the 16th and 24th week of pregnancy and another (although smaller) concentration of stillbirth deliveries in the weeks before the completion of a full term of pregnancy.[12] On the other hand, the distribution of pregnancies varies little between those stillbirths due to hypoxia and stillbirths due causes other than hypoxia (not shown here).

[11] Full term pregnancy is defined in the medical literature as those pregnancies that last at least 36 weeks since conception.

[12] The remarkable differences in the distribution of weeks of pregnancies for livebirths and stillbirths seem to have been overlooked by the existing literature that examines the relationship between air pollution and pregnancy outcomes. As explained earlier, this omission is likely to have introduced selection bias in previous studies. We should also note that the duration of the pregnancy is intrinsically connected with the pregnancy outcome (either a livebirth or a stillbirth) and those observed or unobserved factors that determine the duration of pregnancies are very likely to also determine the pregnancy outcome. Thus, if not properly controlling for these factors, this would introduce further biases in the estimation of the effects of exposure to air pollution on pregnancy outcomes.

-- Figure 5 about here --

In addition, for each week of actual delivery, Figure 6 below shows the ratio of those pregnancies ending in a stillbirth in week *t* over all pregnancies ending in week *t*, and contrasts the time pattern of those pregnancies that end in a Stillbirth delivery to those that end in a livebirth delivery. The figure shows that whereas most pregnancies that end early (say, before the 26th week of pregnancy) present a very large probability of ending on a stillbirth (indeed, the probability is always greater than .8), those pregnancies that end close to full term (after the 36th week of pregnancy) have a very low probability of ending in a stillbirth (probabilities lower than .05).

-- Figure 6 about here --

B. Hazard Function Approach

To estimate the effect of air pollution exposure on the probability of stillbirth in this paper we employ the hazard function approach. The hazard function approach allows to explicitly account for the *time at risk* throughout the duration of the pregnancy, and to gauge how exposure to air pollution during the pregnancy may affect whether a pregnancy results in a stillbirth or livebirth delivery. [13]

In terms of the biostatistics terminology, a stillbirth or livebirth delivery can be thought of as a transition out of a current state of pregnancy, resulting in an interruption of the pregnancy. In this paper, we focus on how exposure to air pollution affects these transitions out of pregnancy and how such exposure to air pollution may make it more likely for any specific pregnancy outcome to occur (either a livebirth or a stillbirth delivery).

[13] Currie and Neidell (2005) employ a linear hazard functions approach to estimate the effect of PM10, O3 and CO on infant mortality in California. While controlling for location specific unobserved factors at the zip-code level, the authors find significant effects of acute (week-level) exposure to CO.

The hazard function approach consists on estimating the probability that the status of a pregnant woman changes, at a given week (say week T), from pregnant to no longer pregnant, where this no-longer-pregnant status can be either a livebirth or a stillbirth delivery. More formally, the hazard function approach consists on estimating the probability that a given pregnancy that has lasted through period $T \geq t-1$, transitions out of its current state at period $T = t$. In other words, this approach consists of estimating the (discrete-time) hazard function $h(X,t)$ defined by

$$h(X,t) = \frac{f(X,t)}{Prob(X,T > t-1)} = \frac{f(X,t)}{1 - F(X,t-1)} \tag{1}$$

where $f(.)$ denotes the (discrete-time) probability distribution function and $F(.)$ denotes the (discrete-time) cumulative distribution function. Both need to be specified.

Whereas the numerator in (1) denotes the unconditional probability of a transition out of a state of pregnancy, the denominator in (1) denotes the *survival probability*, that is, the probability that the pregnancy lasts at least up to period $T \geq t-1$. Hence, the hazard $h(t)$ denotes the probability of a transition out of a state of pregnancy at a given period $T = t$, occurs, given that such a transition has not occurred yet.

Under this framework, exposure to air pollution may have two effects that are captured simultaneously by the hazard of stillbirth in (1). First, exposure to air pollution may shorten the elapsed time in the current state (shorten the duration of pregnancies), which is captured by a decrease in the survival probability (the denominator in equation (1)). Second, for those pregnancies that end at a given period $T \geq t-1$, exposure to air pollution may increase the probability that those transitions out of pregnancies result in stillbirth deliveries (as opposed to resulting in livebirth deliveries), yielding an increase in the numerator of equation (1) above.

In order to assess the effects of covariates on the hazard probability, it is common practice in the empirical literature to assume that the underlying continuous-time model follows a proportional hazard rate $\theta(X,t) = \lambda(X)\theta_0(t)$, so that the hazard rate of transitioning out of the current state depends on both the elapsed time in that state (pregnancy time, $\theta_0(t)$) as well as on covariates X (a function $\lambda(X)$). Where $\theta_0(t)$ is known as the baseline hazard, and $\lambda(X) = e^{\beta' X}$ denotes a vector of covariates where each covariate X_i has a proportional effect on the hazard probability via β_i. Assuming a logit specification for $f(.)$ in (1) above, the proportional hazard rate assumption in a leads to the *complementary log-log* regression model discrete-time setting (Jenkins 2005):

$$h(X,t) = 1 - \exp[-\exp(\beta' X + \gamma_i(t))]$$

or (2)

$$Log\big[-Log[1 - h(t,X)]\big] = \beta' X + \gamma_i(t)$$

where $\gamma(t)$ is the cumulative hazard ($\gamma(t) = \int_{t-1}^{t} \theta_0(u)du$) that depends on elapsed time only (not on covariates Xs).

Using this hazard function approach, in the next section we estimate both the probability of a pregnancy transitioning out to a stillbirth (that is, the probability of Stillbirth) and the probability of a pregnancy transitioning to a stillbirth due to hypoxia (Stillbirth Hypoxia). When estimating such probabilities we focus in particular on how both chronic and acute exposure to PM10 and CO pollution shapes this probability. That is, for any given week of pregnancy in which the pregnant mother is *at risk* of suffering from of the effects of exposure to air pollution, we focus on how such exposure may affect the probability of her pregnancy ending in a stillbirth delivery.

From equation (2) above, the regression framework for assessing the effect of air pollution on probability of stillbirth can be expressed as follows.

$$\begin{aligned} Log\big[-Log[1 - h_i(t,X)]\big] &= \beta' X_i + \gamma(t)_i \\ &= \beta_1 P_{it} + \beta_2 z_i + \beta_3 w_t + \gamma_i(t) \end{aligned} \qquad (2')$$

where P_{it} denotes exposure to air pollution over period t (chronic or acute exposure to either PM10 or CO pollution); z_i denotes mother's characteristics (such as age, history of past stillbirth deliveries, marital status and years of education) as well as controls for seasonality of the pregnancy (season of conception and season of delivery); and w denotes a set of month and year dummies. Furthermore, $\gamma_i(t)$ is a function that captures the effect of the duration of the pregnancy (in weeks) on the hazard of stillbirth delivery. In this case, we use a semi-parametric piece-wise constant hazard with breaks at pregnancy weeks 21, 28, and 36.

To implement this discrete-time estimation strategy, we treat a pregnancy that lasted for n weeks as if it contributes n observations to the sample.[14] Thus, this model can be implemented by estimating equation (2') using a *complementary log-log* regression model where the dependent variable takes on value 1 when a pregnancy ends in a stillbirth and on value 0 in all other cases (both when a pregnancy continues from one week to the next one, and when a pregnancy ends in a livebirth delivery). Alternatively, in the presence of competing end states (say, a stillbirth delivery or a livebirth delivery) the *complementary log-log* regression model reduces to a multinomial logit (Jenkins 2005). In our case, the multinomial logit has three potential outcomes. Whereas the base outcome is that of remaining pregnant (from one week to the following one), the two other outcomes are a stillbirth delivery and a livebirth delivery. Therefore, to estimate the effect of exposure to pollution on the probability of stillbirth as a transition out of a state of pregnancy we could use either the complementary log-log model or the multinomial model (they indeed yield both the same results).[15]

In the next section, using this hazard function approach, we estimate the effect of air pollution on the probability of a pregnancy transitioning out to a stillbirth by estimating a

[14] In this framework, we calculated chronic exposure to air pollution as the moving average of the week-level air pollution variable, averaging out up to the *n*-th week of pregnancy.

[15] By changing the base outcome to that of a livebirth, the multinomial logit also allows to estimate the effect of exposure to air pollution on—once a delivery occurs— whether that delivery is a stillbirth or a livebirth. If both stillbirth and livebirth deliveries where to occur around the same week of pregnancy, then contrasting stillbirth deliveries directly with livebirth deliveries would be the appropriate model. However, as Figure 5 shows, most of stillbirth deliveries do not occur around the same week of pregnancy as livebirths deliveries do (livebirths occur around the 38th week of pregnancy). Therefore, in this paper we focus on estimating the effect of exposure to air pollution on a transition out of pregnancy towards a stillbirth delivery, and contrast that with the probability of remaining pregnant on that particular week.

multinomial logit. Furthermore, we also estimate the effect on the probability of a pregnancy transitioning to a stillbirth due to hypoxia by estimating a multinomial logit with three end states (namely, stillbirth delivery due to hypoxia, stillbirth delivery due to other than hypoxia and livebirth delivery). When estimating these multinomial logits we focus in particular on how both chronic and acute exposure to PM10 and CO pollution shapes these probabilities for any given week of pregnancy.

V. Econometric Analysis

In this section we present the parameter estimates of the model outlined above. We first examine the effect of air pollution on the probability of a pregnancy ending in a stillbirth delivery and in the next sub-section we look at the effect of air pollution on the probability of a pregnancy ending in stillbirth due to hypoxia.

A. Effect of Exposure to Air Pollution on Probability of Stillbirth

Table 3 below presents hazard function parameter estimates for the effect of exposure to air pollution on the probability of stillbirth (the β_1s). Each cell presents parameter estimates associated to exposure to PM10 and CO pollution (and its corresponding standard error) from an independent regression. Each regression introduces only one pollutant (first two rows for PM10 and third and fourth rows for CO), and only one specification for each period of exposure to each pollutant (either chronic exposure or acute exposure). That is, two pollutants and two periods of exposure to each pollutant. For the estimates presented in column 1 we do not introduce any additional controls. The estimates presented in column 2 introduce controls for pregnancy weeks (piecewise hazard with breaks at 21, 28, and 36 pregnancy weeks), mother's age, whether the mother has experienced stillbirth deliveries in the past, marital status and years of education.[16] In column 3 we introduce dummies for months, year and seasonality of pregnancy (both for the season of conception as well as for the season of delivery). Column 4 introduces all the controls from columns 2 and 3. Finally, Column 5 adds dummies by municipality to control for unobserved municipality-specific effects. Due to computer processing capacity the regressions used only a 10 percent random sample of those pregnancies that resulted in a livebirth delivery. Accordingly, regressions coefficients and standard errors were adjusted by frequency weights in order to account for this sampling of livebirth deliveries.

The results presented in Table 3 show that the parameter estimates associated to acute exposure to PM10 pollution (second row of Table 3) are statistically significant across all specifications, as shown in columns 1 through 5. Furthermore, the parameter estimates associated to acute exposure to CO pollution (fourth row of Table 3) are statistically significant only across those specifications that introduce time and municipality-specific controls. On the other hand, the results shown in Table 3 show that both chronic exposure to PM10 and CO pollution have mostly a non-significant effect on the probability of stillbirth. Most parameter estimates on chronic exposure to both PM10 and CO pollution are statistically non-significant at conventional levels.

-- Table 3 about here --

The hazard estimates of the parameter associated to acute exposure to PM10 vary from .00112 (column 1) to 0.00449 (column 5), depending on the set of controls we introduce. When introducing only acute exposure to PM10 pollution (column 1), we obtain that the parameter estimate is small and only weakly significant. Adding further controls for mother's characteristics (column 2), dummies for month, year, season of conception and delivery (columns 3 and 4), and dummies for municipality (column 5) yields larger and more significant

[16] Although we do not directly observe the smoking habits of the pregnant woman, mother's years of education, marital status and age, together, can act as a good proxy for smoking habits of the pregnant woman.

estimates. Moreover, once we introduce time and municipality controls, the parameter estimates turn relatively stable across all these different specifications (columns 3, 4 and 5).

According to the parameter estimate in column 5 (which introduces all available controls), our estimate suggests that the probability that a pregnancy ends in a stillbirth (as opposed to remaining pregnant) increases by .449 percent due to a 1-unit change in acute exposure to PM10. Table 2 shows that the standard deviation of acute exposure to PM10 is 23.34 and that the stillbirth rate (baseline probability of stillbirth) in our sample is 5.69 per every thousand pregnancies. Thereby, our estimate suggests that a one-standard-deviation increase in acute exposure to PM10 pollution would increase the stillbirth rate by 10.5 percent, bringing it up to 6.29 per every thousand pregnancies [6.29=.00569 *(1+23.34*.00449)]. Thereby, for our sample (spanning 84 municipalities over the years 2008-20015), a one-standard-deviation increase in acute exposure to PM10 pollution would result in 508.4 additional stillbirth deliveries over the period [508.4 = 0.00629*(4,915+857,820) - 4,915], an average of 63.6 additional stillbirths a year.

On the other hand, the hazard estimates associated to the effect of acute exposure to CO pollution on probability of stillbirth varies from -.000205 (column 1) to .0999 (column 4), depending on the set of controls we introduce. Once we introduce controls for month, year, season of conception and delivery (columns 3 and 4), and dummies for municipality (column 5) the parameter estimates turn statistically significant and relatively stable across all these specifications.

The parameter estimate that introduces all available controls (column 5) yields an estimate for the effect of acute exposure on the probability of stillbirth of .0976. Thereby, this estimate suggests that a one-standard-deviation increase in acute exposure to CO pollution (see Table 2) would increase the stillbirth rate by 5.3 percent, bringing it up to 5.99 per every thousand pregnancies [=0.00529*(1 + .54*.0976)].[17] Therefore, such an increase in acute exposure to CO pollution would result in 252.7 additional stillbirth deliveries over the period considered in this paper [=0.00599*(4,915+857,820) - 4,915], an average of 31.6 additional stillbirths a year.

[17] Table 2 shows that the standard deviation of acute exposure to CO pollution is .54.

B. Effect of Exposure to Air Pollution on Probability of Stillbirth Due to Hypoxia

One of the channels that air pollution is thought to adversely affect the fetus during pregnancy is via the nutrients and oxygen that the pregnant woman transmits to the fetus. Thereby, exposure to air pollution is likely to restrict her capacity to properly transmit nutrients and oxygen to the fetus. In situations where this inhibition is severe enough to end the pregnancy in a stillbirth, it is likely that the cause of fetal death is diagnosed by the physician as hypoxia. Therefore, the adverse effects of exposure to air pollution on pregnancies are likely to be more directly reflected in those fetuses that die due to hypoxia.

In Table 4 below we present estimates of the hazard function regression analysis for stillbirths due to hypoxia. Similar to Table 3, the first column presents estimates without introducing any controls, and then columns 2 to 5 introduce further controls. Each cell in Table 4 refers to an independent regression with only one pollutant (and only type exposure) where the first statistic refers to the parameter estimate associated to that pollutant and the second statistic refers its corresponding standard error.

-- Table 4 about here --

The estimates presented in Table 4 show that acute exposure to PM10 and CO pollution has a statistically significant effect on the probability of stillbirth due to hypoxia. Furthermore, these parameter estimates turn relatively stable throughout all different specifications of Table 4 (columns 1 through 5). On the other hand, the estimates for the effect of chronic exposure to PM10 and CO pollution are not consistently significant throughout different model specifications in Table 4.

The estimates presented in column 5 of Table 4 suggest that the probability of a pregnancy ending in a stillbirth due to hypoxia increases by .633 percent due to a 1-unit increase in acute exposure to PM10. Given that the stillbirth rate due to hypoxia is 1.58, per every thousand (see Table 2), a one-standard-deviation increase in acute PM10 pollution would bring this rate up to 1.82 per every thousand [=.00158*(1+23.34*.00633)], which corresponds to a 14.8 percent increase. This means that a one-standard-deviation increase in acute exposure to PM10 pollution would result in 201.7 additional stillbirth deliveries due to hypoxia, [=0.00182*(4,915+857,820)-1,365], an average of 25 additional stillbirths due to hypoxia a year.

Similarly, the estimates for acute exposure to CO pollution in column 5 show that a one-standard-deviation increase in acute CO pollution would bring the probability of stillbirth due to hypoxia up to 1.76 per every thousand [=.00158*(1+.54*.212)], which corresponds to a 11.4 percent increase. That is, such an increase in acute exposure to CO pollution would result in additional 154.2 stillbirths due to hypoxia [=0.00182*(4,915+857,820) - 1,365], an average of 19.3 additional stillbirth due to hypoxia a year.

Our findings support the notion that the effect of exposure to air pollution is stronger among those stillbirths due to hypoxia. Table 2 shows that stillbirths due to hypoxia amount to only 27.8 percent of total stillbirths (1,365 out of 4,915). However, the additional stillbirths due to hypoxia that would result from an increase in pollution exposure amounts to a much larger proportion. Whereas the additional stillbirths due to hypoxia that would result from a one-standard-deviation increase PM10 pollution would represent 37.9 percent of the total additional stillbirths, the figure from an increase in one-standard-deviation in CO pollution would represent 61 percent of the total additional stillbirths. This finding support our prior that the pathway in which air pollution adversely affects pregnancies is primary manifested through those fetal deaths that are caused by hypoxia.

An additional finding that is worth noticing is that the hazard estimates for the probability of stillbirth due to hypoxia are quite similar for both chronic as well as acute exposures to both PM10 as well as CO pollution. This is particularly true for columns 3 and 4 of Table 4 (first and second row for PM10 pollution, and third and fourth row for CO pollution). This suggests a consistent estimate for the effects of both PM10 and CO pollution regardless of whether it is chronic exposure or acute exposure. This may be because the aggregation of the pollution exposure variable throughout many weeks of pregnancy (in the case of chronic exposure) reduces the variability of the pollution variable, which, in turn, generates larger standard errors in the parameter estimate. This suggests that our statistical methods may be limited when it comes to reliable reporting significant effects of chronic exposures to air pollutants. Although chronic exposure may actually have significant adverse effects on pregnancies, our statistical methods cannot actually capture that effect.

VI. Discussion

In this paper we estimate that acute exposure to a 10-unit increase in acute exposure to PM10 pollution increases the probability of stillbirth by 4.49 percent. On the other hand, about half of the stillbirth deliveries in our sample occur during the third trimester of pregnancy—after the 26th week of pregnancy (see Figure 5). Thereby, our measure of acute exposure to PM10 can be somewhat compared to measures of exposure to PM10 pollution in the last trimester of pregnancy of those stillbirth deliveries (either second or third trimester of pregnancy). For instance, Kim et al. (2007) find that a 10-unit increase exposure to PM10 pollution in the third trimester of pregnancy increases the probability of stillbirth by 8 percent. However, whereas the stillbirth rate that reported in Kim et al. (2007) for the city of Seoul is 4.45 percent, the stillbirth rate in our data is only .57 percent. For stillbirth rates similar to the one reported in our paper, Hwang et al. (2011) find that only exposure to *peak* levels of PM10, during the first and second month of pregnancy, increases the probability of stillbirth by about 2 percent.[18] Moreover, in a study that looks at the effects of exposure to fine particulate matter (PM2.5), De Franco et al. (2015) find that exposure to *peak* levels of PM2.5 during the third trimester of pregnancy increase the probability of stillbirth by 42 percent. Similarly, Green et al. (2015) find some evidence that exposures to *peak* levels of PM2.5 during the entire pregnancy increase the probability of stillbirth by 6 percent.

[18] *Peak* levels of pollution refers to pollution events in which pollution is higher than the sum of the mean pollution and inter-quantile range (> mean + IQR). If pollution has a normal distribution, this would be equivalent to episodes of pollution in the top 2.5% percent of its distribution.

VII. Policy Implications

Air pollution is a growing global concern, particularly in middle income and emerging economies. Consequently, estimating the effects of air pollution on human health can provide policy makers with the necessary tools to back efforts to reduce concentrations of air pollutants.

For example, Chile's environmental authority has developed Air Pollution Control and Prevention Plans (PPDAs) for Santiago since the late 1990s, and more recently also for other smaller cities throughout the rest of the country. PPDAs consist of a battery of provisions for reducing air pollution. As of today, Chile's environmental authority has developed PPDAs for 18 cities, covering largely the same geographical area as the set of municipalities considered in this paper.

The goal of these PPDAs is to bring air pollution concentrations below Chile's national standard, which is 50 micrograms per cubic meter for PM10. These PPDAs are evaluated ex-ante estimating social costs and benefits of expected improvements in air quality using estimates from different international studies that look at the effect of changes in pollution concentrations on specific health outcomes (these estimates are known in the literature as *dose-response functions*). However, the effect of air pollutants on stillbirth deliveries has not yet been included in the evaluation of Chile's PPDAs. As a consequence, it is likely that the benefits from the efforts to reduce air pollution concentration have been largely underestimated in the cost-benefit analysis for Chile's PPDAs.

Indeed, the average PM10 concentration in our sample is 63.93, exceeding Chile's standard for this pollutant (see Table 2). To bring PM10 concentrations to meet Chile's standard for PM10 would require an average reduction in concentration of 13.93 units. According to our estimates from Table 3, such a reduction would translate into 300 fewer stillbirth deliveries over the period 2008-2015, and an average of 37.6 fewer stillbirth deliveries a year. Chile's

Minister of Social Development has recently set the official value of statistical life at US$ 0.431 million (MINDES 2017). Therefore, the value of those fewer stillbirth deliveries resulting from reducing concentrations of PM10 pollution to meet Chile's standard would amount to US$16.2 million a year. These benefits have not been valued in the cost-benefit evaluations of the efforts to reduce air pollution concentrations under Chile's PPDAs.

VIII. Concluding Remarks

In this paper we examined the relation between exposure to airborne air pollution and the effect that this may have on the probability of a pregnant woman ending their pregnancy in a stillbirth delivery. The existing literature shows only weak evidence of the relationship between exposure to air pollution and stillbirth delivery.[19]

We looked at the effect of both chronic as well as acute exposure to PM10 and CO pollutants on the probability of stillbirth by estimating hazard functions that explicitly account for the *time at risk* throughout the duration of the pregnancy. We controlled for number of weeks of pregnancy, mother's characteristics, seasonal pregnancy characteristics, month and year-specific dummies, and municipality-specific dummies. We find that acute exposure to PM10 has an adverse effect on the probability of stillbirth. We estimate that a one-unit change in acute exposure to PM10 pollution increases the probability of stillbirth by .449 percent. This means that, for our sample of 84 municipalities over the period 2008-2015 in Chile, a one-standard-deviation change (increase/decrease) in PM10 concentrations would translate into 63.6 (additional/fewer) stillbirths a year.

Furthermore, to the best of our knowledge, the existing literature does not examine the effect of exposure to air pollution on the probability of cause-specific stillbirths, and the likely pathway of the effects of pollution exposure on fetal deaths that result in a stillbirth delivery. In this paper we looked at the effects of PM10 and CO on the probability of stillbirth due to hypoxia (those fetuses that die due to the lack of oxygen). We find that acute exposure to both

[19] As noted earlier, only exposure to very high levels of air pollutants is associated to a statistically significant higher probability of stillbirth (De Franco et al., 2015).

PM10 and CO increase the probability of stillbirth due to hypoxia and has a larger effect than that of the one on stillbirth due to all causes.

On the other hand, it could be argued that the lack of controls for smoking habits of the pregnant woman may introduce bias in our parameter estimates. However, as long as smoking habits does not systematically correlate with exposure to air pollution after controlling for mother's years of education, age, marital status and location of residence (municipality of the mother), the lack of control for smoking habits should not introduce bias in our estimates.

Moreover, as with most of epidemiological studies in the literature, this study assigns exposure to air pollutants only imperfectly. In our case, we assign exposure based on a sound average of the pollution concentration at the municipality of residence. A more accurate measure of pollution exposure would be such that it would allow to record air quality at an exact location for each pregnant woman in each period of time during her pregnancy. Unfortunately, our data does not allow for such accurate recordings. Furthermore, it is likely that pregnant women are more aware of the health risks of the surrounding environment. Pregnant women attend regular checkups with their gynecologist and health practitioners who may alert them of the health hazards of exposure to high levels of air pollution. They may also be more alert at the news and warnings of the local environmental and health authorities. As a consequence, pregnant women may change their behavior to limit their exposure to air pollutants. For example, they may choose not to exercise and to reduce time spent outside in days of high pollution concentrations. They may even be inclined to wear a breathing or dust mask during days of high pollution concentrations, although this would be rare in a country like Chile. Nevertheless, both assigning only an imperfect measure of actual pollution exposure as well as a possible change in behavior to minimize pollution exposure would bias our results towards the null of no effect. The fact that we find a statistically significant effect under these limitations likely represents a lower bound of the true effect of air pollution on the probability of stillbirth.

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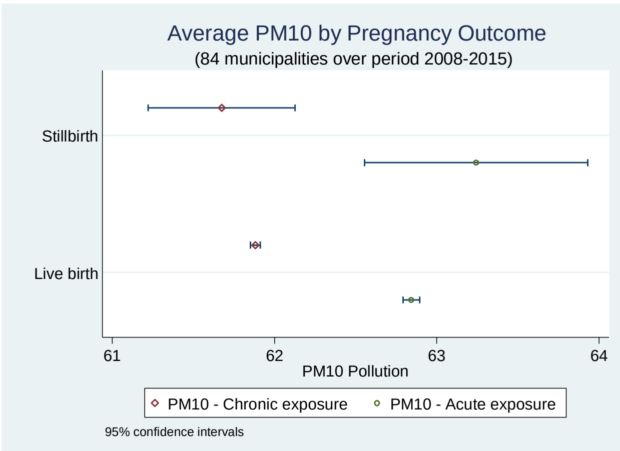
Figure 1: Mean PM10 and CO Pollution, by Pregnancy Outcomes

PM10 Pollution



CO Pollution

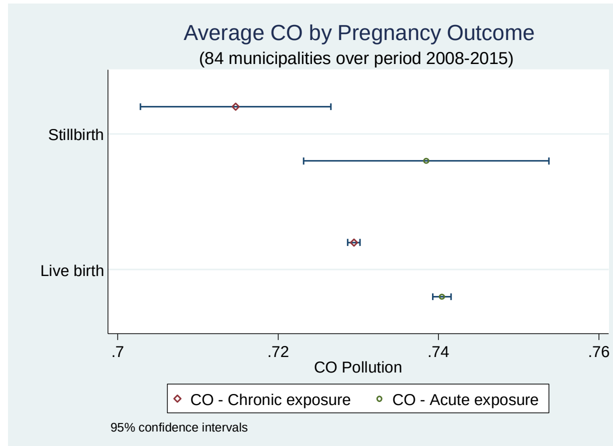
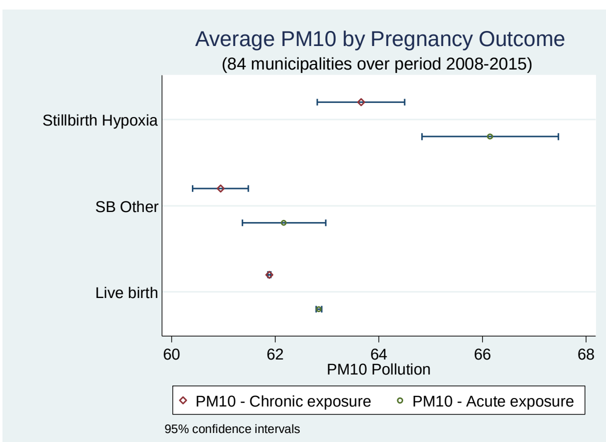


Figure 2: Mean PM10 and CO Pollution, by Causes of Stillbirth Delivery

PM10 Pollution



CO Pollution

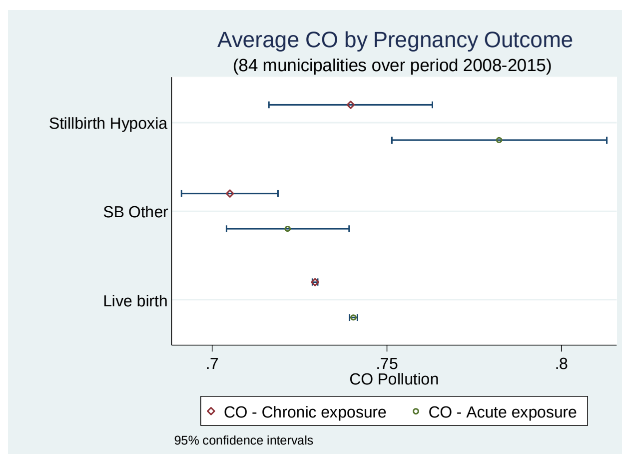


Figure 3: Municipality-level Stillbirth Rate, Stillbirth Due to Hypoxia and Acute Exposure to PM10 & CO Pollution,.

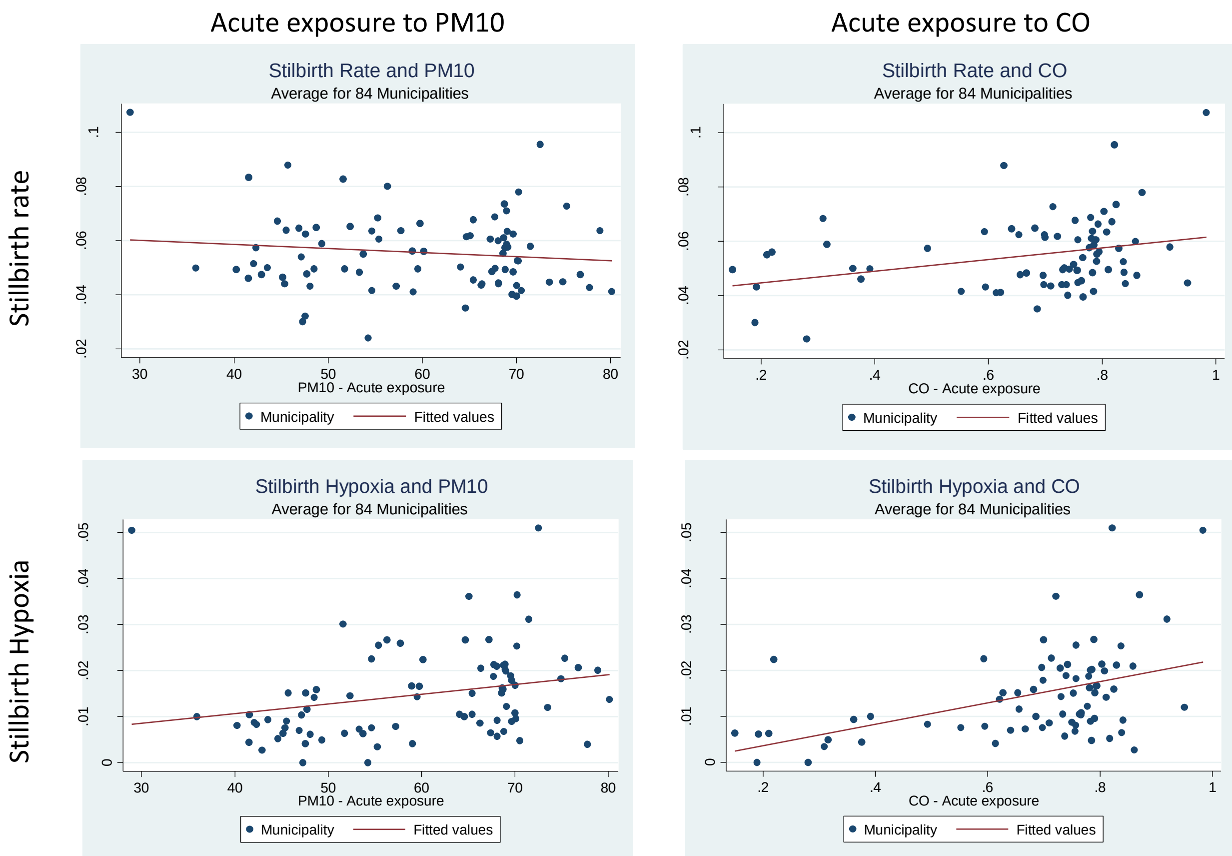


Figure 4: Monthly average Stillbirth Rate, Stillbirth Due to Hypoxia and Acute Exposure to PM10 & CO Pollution.

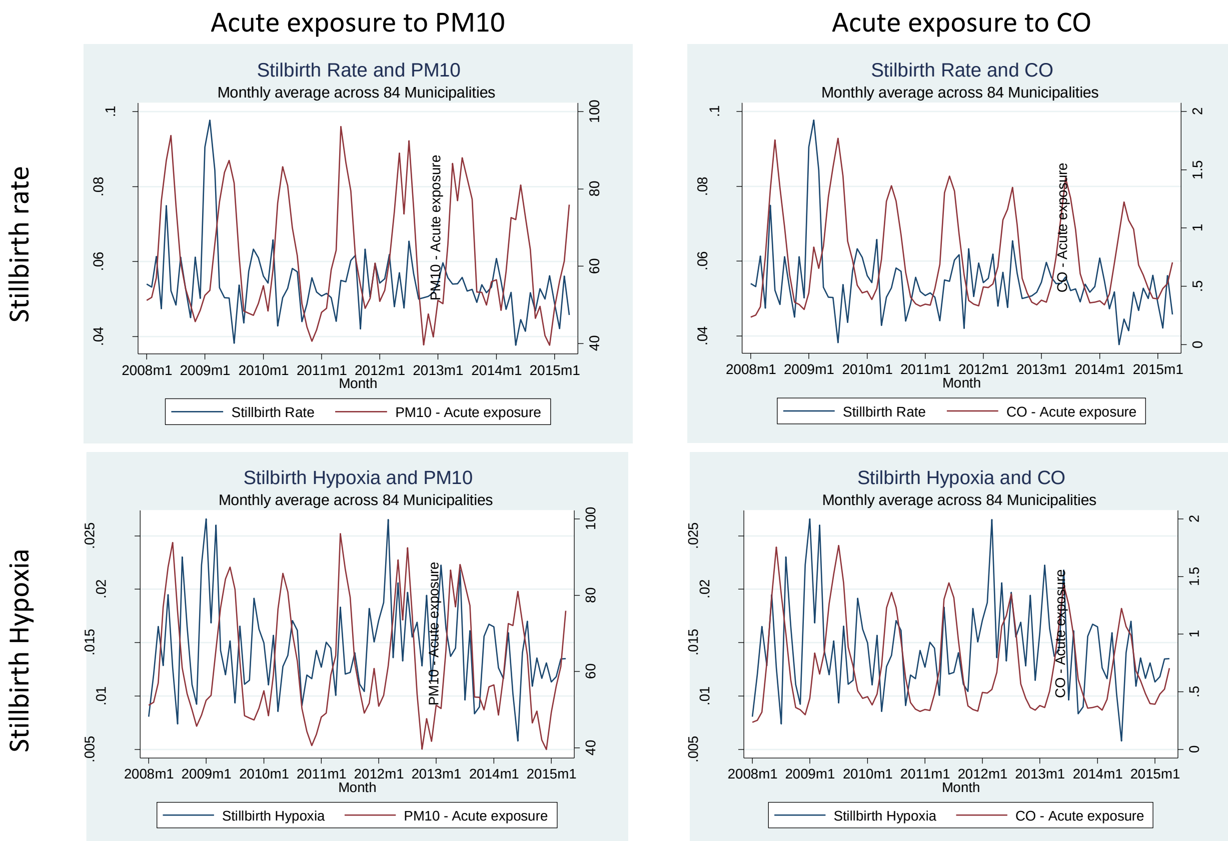


Figure 5: Histogram of Livebirths and Stillbirths

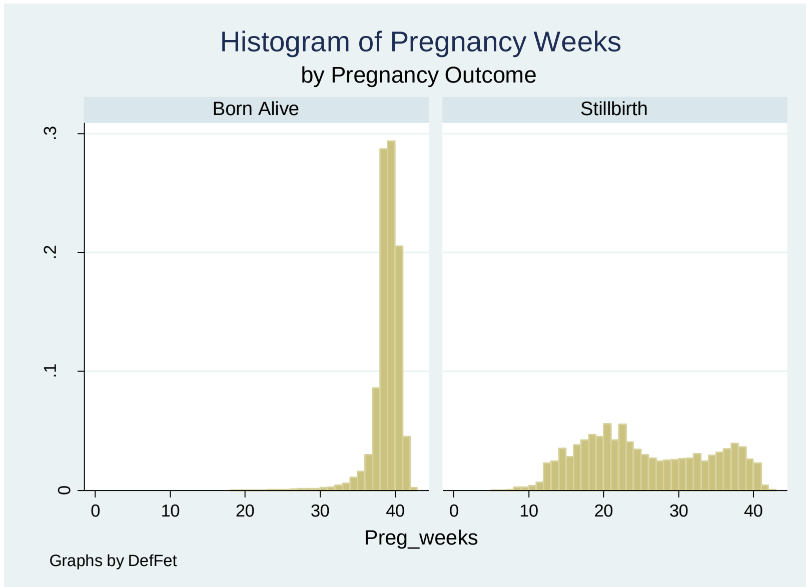


Figure 6: Hazard of Stillbirth delivery for those pregnancies ending at a given week t.

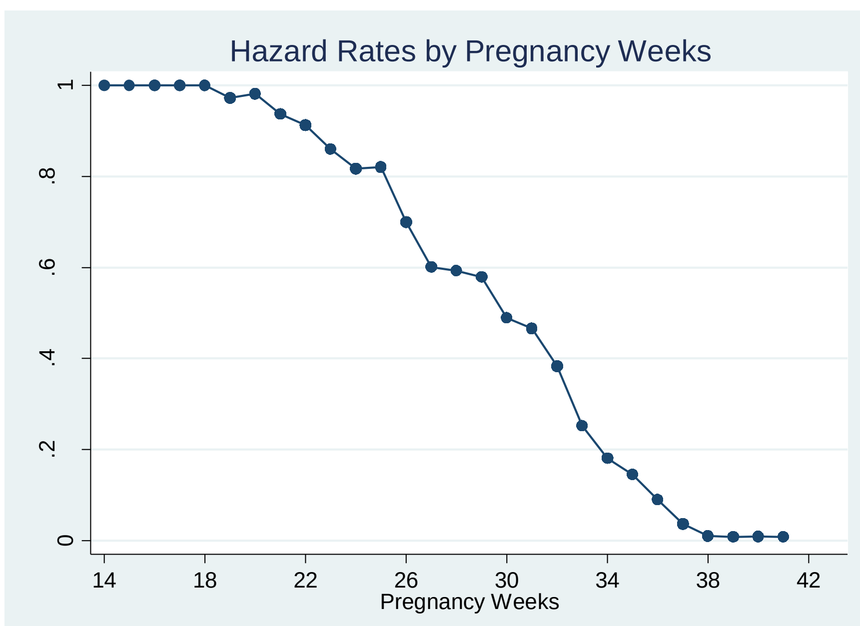


Table 1: PM10, CO Pollution and Covariates, by Pregnancy Outcome.

	Stillbirths						Live Births	
	All Stillbirths (N = 4,915)		Stillbirths Hypoxia (N = 1,365)		Stillbirths Other (N = 3,550)		(N = 857,820)	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
PM10 - Chronic exposure	62.51	16.03	64.17	15.52	61.87	16.18	62.79	0.36
PM10 - Acute exposure	64.41	24.32	67.13	24.09	63.35	24.33	63.90	23.29
CO - Chronic exposure	0.718	0.425	0.740	0.441	0.709	0.418	0.730	0.363
CO - Acute exposure	0.741	0.548	0.781	0.577	0.726	0.535	0.741	0.541
Mother's age	28.7	7.5	27.8	7.5	29.0	7.4	27.7	6.7
Mother's past stillbirths	0.041	0.199	0.044	0.205	0.040	0.196	0.019	0.136
Marital status (0=married, 1=single)	0.39	0.49	0.36	0.48	0.40	0.49	0.33	0.47
Mother's years of education	12.1	3.0	11.6	2.9	12.3	3.1	12.6	2.9
Season of Conception								
Summer	0.22	0.42	0.23	0.42	0.22	0.42	0.23	0.42
Autumn	0.23	0.42	0.21	0.41	0.24	0.43	0.26	0.44
Winter	0.29	0.45	0.31	0.46	0.28	0.45	0.27	0.45
Spring	0.25	0.43	0.25	0.43	0.25	0.43	0.24	0.42
Season of Delivery								
Summer	0.27	0.44	0.27	0.45	0.27	0.44	0.26	0.44
Autumn	0.28	0.45	0.30	0.46	0.28	0.45	0.28	0.45
Winter	0.23	0.42	0.23	0.42	0.23	0.42	0.24	0.43
Spring	0.22	0.41	0.20	0.40	0.22	0.42	0.23	0.42

Table 2: Descriptive Statistics for Stillbirth, Stillbirth due to Hypoxia, PM10 & CO Pollution Over Length of Pregnancy and on Week of Birth or Fetal Death

Variable	**Mean**	**Standard Deviation**			**Obs.**
		Overall	**Municipality**	**Week**	
Stillbirth rate (all causes)	0.0057	0.106	0.009	0.015	90,697
Stilbirth Hypoxia	0.0016	0.058	0.008	0.008	90,697
PM10 - Chronic exposure	62.77	13.87	9.26	9.05	90,697
PM10 - Acute exposure	63.93	23.34	9.22	18.44	90,697
CO - Chronic exposure	0.73	0.37	0.11	0.25	90,697
CO - Acute exposure	0.74	0.54	0.11	0.42	90,697

Table 3: Effect of PM10 & CO pollution on the probability of stillbirth. Hazard estimates.

	(1)	(2)	(3)	(4)	(5)
PM10 - Chronic exposure	-0.00212**	-0.00170	0.000116	0.000589	0.00196
	(0.00100)	(0.00116)	(0.00149)	(0.00176)	(0.00270)
PM10 - Acute exposure	0.00112*	0.00118**	0.00343***	0.00354***	0.00449***
	(0.000581)	(0.000600)	(0.000759)	(0.000758)	(0.000893)
CO - Chronic exposure	-0.0882*	-0.0937*	-0.0617	-0.0641	-0.0908
	(0.0455)	(0.0488)	(0.0676)	(0.0688)	(0.0736)
CO - Acute exposure	-0.000205	-0.00334	0.0859***	0.0999***	0.0976***
	(0.0311)	(0.0302)	(0.0302)	(0.0310)	(0.0321)
• Controls: Pregnancy weeks, Mother's age, Past stillbirth delivery, Marital Status and Years of education.	N	Y	N	Y	Y
• Dummies for Month, Year and Seasonality of pregnancy (season of the year for both conception & delivery)	N	N	Y	Y	Y
• Municipality Dummies	N	N	N	N	Y
Obs. expanded by pregnancy weeks	2,261,543	2,261,543	2,261,543	2,261,543	2,261,543
Observations	**90,697**	**90,697**	**90,697**	**90,697**	**90,697**

Standard errors clustered by municipality. *** p<0.01, ** p<0.05, * p<0.1.

Table 4: Effect of PM10 & CO pollution on the probability of stillbirth due to hypoxia. Hazard estimates.

	(1)	(2)	(3)	(4)	(5)
PM10 - Chronic exposure	0.00326	0.00436*	0.00976***	0.0119***	0.00181
	(0.00205)	(0.00227)	(0.00305)	(0.00361)	(0.00530)
PM10 - Acute exposure	0.00565***	0.00560***	0.00874***	0.00878***	0.00633***
	(0.00147)	(0.00144)	(0.00181)	(0.00179)	(0.00198)
CO - Chronic exposure	0.0344	0.0220	0.194*	0.198*	0.0808
	(0.0806)	(0.0840)	(0.102)	(0.111)	(0.109)
CO - Acute exposure	0.131**	0.111**	0.252***	0.255***	0.212***
	(0.0591)	(0.0552)	(0.0651)	(0.0659)	(0.0660)
• Controls: Pregnancy weeks, Mother's age, Past stillbirth delivery, Marital Status and Years of education.	N	Y	N	Y	Y
• Dummies for Year and Seasonality of pregnancy (season of the year for both conception & delivery)	N	N	Y	Y	Y
• Municipality Dummies	N	N	N	N	N
Obs. expanded by pregnancy weeks	2,261,543	2,261,543	2,261,543	2,261,543	2,261,543
Observations	**90,697**	**90,697**	**90,697**	**90,697**	**90,697**

Standard errors clustered by municipality. *** p<0.01, ** p<0.05, * p<0.1.

APENDIX 1: Summary of current literature

Authors	Location and Years	Pollutants & Imputation of Exposure	Length of Exposure in the Analysis	PM and CO Mean (SD)	Stillbirth rate (%)	Methodology and Econometrics	Controls	Effects on Stillbirth (Odd Ratios)
Kim et. al. (2007)	Seoul, Korea. May 1st 2001 – May 31st 2004.	PM10 Nearest monitoring station to mother's address using 27 monitoring stations in Seoul.	Trimesters, Last six weeks. Months,	89.3 (45.7)	4.4	Hospital-based cohort study. Logistic regression	Demographics, mother's SES, medical history, pregnancy complications, exposure to smoking, alcohol use	1.1 for Third trimester
Pereira et. al. (2008)	Sao Paulo, Brazil. Jan 1991 – Dec 1992.	PM10, CO (also SO2, NO2 & O3). City-wide daily average using 13 and 5 monitoring stations for PM10 and CO, respectively.	3 and 14 days before delivery for CO and PM10, respectively.	CO: 5.73 (1.89) PM10: 65.0 (27.3)	8.36	Citywide ecological study. Poisson regression.	Season (month and day dummies) and weather (temperature, relative humidity).	CO: Positive & significant at 90%
Hwang et. al. (2011)	Taiwan. Jan 1st 2001- Dec 31st 2007.	PM10, CO (also SO2, NO2 & O3) Interpolated using IDW to postal-code area (one block face) using 72 monitoring stations.	Months, Trimesters and entire pregnancy.	PM10: 72.9 (23.3) CO: 0.66 (0.18)	0.62	Nationwide population-based. Logistic regression.	Sex, maternal age, gestational age, municipality-level SES, season of conception and year of birth.	PM10: 1.02 for First and Second Month
Faiz et. al. (2012)	State of New Jersey, USA. Jan 1st 1998 – Dec 31st 2004.	PM2,5, CO (also SO2 & NO2) Nearest monitoring station to mother's address (10Km and 5Km) using 25 and 16 monitoring stations for PM2.5 and CO, respectively	Trimesters and entire pregnancy	PM2.5: 13.8 (1.6) CO: 0.92 (0.3)	0.42	Statewide population-based. Logistic regression	Maternal age, race and educational attainment, participation in prenatal care and self-reported smoking. Also, neighborhood SES, mean temperature, calendar year and month of conception.	CO: 1.14 of *peaks* (> mean+IQR) during Second and Third trimesters
Faiz et. al. (2013)	State of New Jersey, USA. Jan 1st 1999 – Dec 31st 2004	PM2,5, CO (also SO2 & NO2) Nearest monitoring station to mother's address (10Km and 5Km) using 5 and 13 monitoring stations for PM2.5 and CO, respectively	2 to 6 days before delivery	PM2.5: 14.7 (8.8) CO: 0.85 (0.4)	0.42	Statewide population-based. Logistic regression	Maternal age, race and educational attainment, participation in prenatal care and self-reported smoking. Also, mean apparent temperature.	CO: 1.20 and 1.17 of *peaks* (> mean+IQR) for lag-2 days and lag-3 days, respectively.
De Franco et. al. (2015)	State of Ohio, USA. 2006 - 2010	PM2.5 Nearest monitoring station to mother's address (10Km) using 57 monitoring stations.	Trimesters and entire pregnancy	PM2.5: 13.3 (1.8)	0.53	Statewide population-based. Logistic regression.	Maternal age, race, educational attainment, prenatal care and season of conception.	PM2.5: 1.42 of *peaks* (> mean+IQR) during Third trimester.
Green et. al. (2015)	State of California, USA. Jan 1st 1999 – Dec 31st 2009	PM2,5, CO (also SO2, NO2 & O3) Nearest monitoring station to the centroid of mother's postal-code. 20Km and 5Km for PM2.5 and CO, respectively.	Trimesters and entire pregnancy	PM2.5: 15.2 (5.1) CO: 1.29 (0.67)	0.46	Statewide population-based. Logistic regression.	Maternal education, race/ ethnicity and age. Season and year of conception, sex of the infant/fetus, relative humidity, and air basin.	PM2.5: 1.06 of *peaks* (> mean+IQR) for entire pregnancy (significant at 90%)