Environment and Health in the Long Term

Evan D. Peet
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Abstract[1]

From global to indoor, from regional to local, the changes that human activities cause in the environment can act as stressors with multiple effects on ecology and human health both in the short and long term. The goal of this paper is to improve the understanding of the costs to human health of environmental contamination introducing a novel assessment of the quality of two environmental inputs - air and water - and relating these to long-term health outcomes by exploiting the implications of the fetal origins hypothesis. The quality of environmental inputs is assessed using various exogenous yet noisy measures to determine the underlying latent quality of water and air. The context of the study is the metropolitan area of Cebu where unique geographic data is collected and used in order to characterize the environment of a survey cohort during their early formative years of life. Initial findings suggests exposure to poor environmental quality during gestation results in insignificant or mildly improved short term health outcomes, such as birth weight and height. Additional analysis of long term outcomes is therefore needed (and forthcoming).

1 Introduction

Contamination of the environment is inherently an economic issue, resulting from the non-internalization of the costs of production. These costs of production are borne by society in a variety of ways, one of which is the cost to human health. An accurate description of costs - despite their multiple dimensions ranging from natural resource degradation, to reduced tourism and to human health - and the comparison with benefits, is fundamental to the understanding of optimal policy. Furthermore, environmental contamination and economic development are inextricably linked. Economic development generally improves welfare, however the increasing production and consumption lead to increasing amounts of environmental

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contamination. If the benefits from increased production and consumption cannot outweigh the costs to human and ecological health, then it is not truly development (Meier and Rauch 2005). The goal of this paper is to improve the understanding of the costs to human health of environmental contamination by profiling the quality of two environmental inputs - air and water - and relating these to long-term health outcomes in the developing country context of Metropolitan Cebu, Philippines.

In this study I exploit a long running survey in Metropolitan Cebu, Philippines that followed individuals collecting health data since 1983 when the individuals were in utero. I pair this health data with unique geographic data that I collected from various sources. Data such as the location of industrial polluters and mines as well as existing piped water networks and other geographic data allow for the characterization of the environment at the beginning of the survey. Combining this geographic data with locational peculiarities such as seasonal wind patterns, precipitation and topography generates variation in air and water quality that is arguably exogenous to behavioral compensations. The longitudinal characteristic of the survey enables the analysis of both short and long term effects of air and water quality variation during pregnancy. Initial findings suggests exposure to poor air quality during gestation results in insignificant or mildly improved short term health outcomes, such as birth weight and height. Additional analysis of water quality as well as long term outcomes is therefore needed (and forthcoming).

Additionally, I introduce a novel approach to assessing air and water quality, an approach based on the intuition of a literature in toxicology. A relatively new approach of toxicology aims to account for aggregate or cumulative exposures to co-occurring contaminants (Georgopoulos et al 2008, Buck et al 2003, Price et al 2005, EPA 2008). The need to examine contamination jointly arises from the fact that along the path from the source to the individual and eventually to the tissue where an effect is exerted, the contaminant can be altered from its primary form - the chemical released from the source - to some secondary form - a newly derived chemical formed through interactions with other chemicals in the media. For example, this process of chemical alteration is the main source of ozone in the troposphere. Gasoline combustion, industrial processes and chemical solvents emit oxides of nitrogen and volatile organic compounds into the atmosphere and sunlight acts as a catalyst to break down the compounds and reform them into O_3, or ozone.[2] Given the immense variety of chemicals, both known and unknown, being emitted daily into the air and water it is impossible to say that the same contaminants observed at monitor a large distance from an individual are the same that enter the individual's body. Tens of thousands of chemicals are currently used in industrial processes, with hundreds being introduced every year. The testing of both ecological and human impacts of individual chemicals are expensive and time consuming, and in general have not been performed. Only a very small fraction of the chemicals emitted daily into the environment have been adequately assessed for the risks they present to human health. In 2007, the US Environmental Protection Agency developed and began implementing ToxCast as a way to collect and analyze the effects of potentially toxic chemicals. The year 2009 saw the completion of the first phase, the profiling of approximately 300 previously researched pesticides. Current work is under way to profile the risks of hundreds more.[3] In addition to the sheer volume of chemicals with both known and unknown effects and their potential alteration in the environment, the human body's metabolic processes can further alter the compounds once inside the body. Furthermore, not only does the presence of primary or secondary compounds produce adverse effects, but the interac-

[2]See the website http://www.epa.gov/glo for additional information

[3]See the website www.epa.gov/ncct/toxcast for additional information.

tions of compounds produce effects. For instance, recent research has identified toxic interactions among metals such as arsenic, lead, and cadmium that are only observed when more than one of the substances are present (Wang et al2008, Sasso et al 2010). Therefore a variety of both primary and secondary compounds exist within the human body where they work alone or in tandem to produce an effect, facts which necessitate the need for new methods to describe the effects of environmental contamination on human health (Paustenbach 2001).

Adapting these facts from toxicology, my approach draws on the methodology proposed in Cunha, Heckman and Schennach (2010) where multiple noisy measures are used to estimate the true latent variables. Exogenous spatial and temporal variation for each of these measures (to be explained in detail in later sections) by utilizing locational peculiarities as well as the timing of human development in utero. The timing of exposure is particularly relevant because different tissues are susceptible at different times depending on the child's stage of development (Birnbaum, 2011). If exposure occurs before or after the development of an organ, it is less vulnerable and less likely to exhibit impairments than if the exposure occurs during "organogenesis", or the early stages of development (Rice and Barone 2000). Fetal insults have been shown to have persistent effects of human health through the work on the fetal origins hypothesis championed by Barker (1995). The hypothesis is that the quality of environmental inputs during those critical stages of development will have impacts on health that manifest throughout life. The longitudinal survey known as Cebu Longitudinal Health and Nutrition Survey enables this analysis because of its longitudinal nature which first collects information on a cohort of children while still in utero, next at birth and bimonthly until the children are age two, and then periodically until the most recent survey in 2007 when the children were in their mid-twenties.

The paper will proceed as follows. The next section provides a literature review, briefly touching on studies in toxicology, epidemiology and economics. Following that, I will discuss both the model and methodology (additional explanation of both can be found in appendix 1). Section four contains an explanation of the empirical implementation, in particular the location of the study and the peculiarities of the location which create exogenous levels of exposure. In section five I will discuss my current progress and in section six I will conclude.

2 Literature Review

The rapidly expanding variety and presence of potentially toxic chemicals in the environment has lead to numerous studies in the fields of toxicology and epidemiology seeking to determine the impact on human health. These studies have been performed on both human and animal subjects, each with inherent advantages and disadvantages. Animal studies allow the researcher to precisely control the magnitude and duration of both the exposure and dose administered to the subjects, however as illustrated by the failed predictions of tests of the pharmaceutical thalidomide on animals the translation from animal to human models is suspect. (Altshuler 2003). Human studies avoid the problem of interspecies outcome prediction but do not allow the researcher to precisely control the magnitude and duration of the exposure and dose. Furthermore, the samples are highly selective because participants voluntarily enroll and latent health effects are often unobserved because even the longest studies do not follow participants through adolescence. The CHAMACOS study began in 1999 by enrolling over 600 pregnant women living in the Salinas Valley in

California.[4] The Salinas Valley is one of the richest agricultural areas on earth and the inhabitants are highly exposed to a variety of pesticides such as organochlorines, organophosphates, pyrethroids as well as other common chemicals such as flame retardants (polybrominated diphenyl ether, or PBDE) and bisphenol A. These women and their children have been followed until the present time with their exposures to toxins measures through blood and urine tests. Results from studies employing CHAMACOS have shown that exposure to organophosphates and organochlorines in utero can lead to poor birth outcomes and diminished childhood neurodevelopment, while others have shown that exposure to PBDE flame retardants can lead to endocrine disruption and neurotoxicity in children (Eskenazi et al 2004). However, the 600 enrolled women have been shown to differ significantly from the over 1,100 women originally selected as eligible for the study. Women who declined to participate were more likely to be English speaking, born in the United States and have more education, and less likely to be living with an agricultural worker (Eskenazi et al 2004).. Both the observed and unobserved differences in the sample likely bias the studies and prevent the generalization of the results. In the present study I will be unable to catalog with precision the type and amount of environmental contaminants as in the above mentioned studies, however by employing measures of contamination exogenous to behavioral compensation the bias of the epidemiological and toxicological studies is avoided.

The economic literature generally focuses on describing short term responses to imprecise measures of exposures to environmental contaminants. However, three studies of note relate environmental quality to long term outcomes. First Gould et al 2009 exploits the random relocation of Yemenite refugees, some to homes with piped water and others to homes without, to show a marginal long term effect of the presence of piped water in the home at an early age on later schooling outcomes for girls. The results of this study stand firmly on the randomness of relocation, with threats to randomness biasing the results. The second study, Ebenstein 2012, similar to the present study in its use of geographic variation, combines water quality and digestive cancer measures in China to show that a deterioration of water quality by a single grade on a six-grade scale increases the digestive cancer death rate by 9.7 percent. However, even when using rainfall instruments the results are derived from comparing health outcomes in very distinct locations with unobserved differences (such as genetic differences between inhabitants of North and South China). The third study of note examining the effects of environmental quality on long term outcomes is Almond et al 2009 where the authors argue that radioactive fallout from the 1986 Chernobyl disaster. Meteorological conditions caused Sweden to receive roughly 5 percent of the Caesium fallout and precipitation differences across Sweden resulted in significant geographic variation in the level of radiation exposure. Interestingly, the analysis shows no damage to health, but cognitive ability as manifested in academic achievement is negatively affected. While natural experiments such as this one offer unique opportunities to examine generally unobservable effects, it can be argued that standard daily exposures, such as those examined in the present study, are of more general importance given the ubiquity the of ever growing number and volume of contaminants.

Economic studies of short term health responses to water quality studies have shown effects on infant mortality and diarrheal incidence due to biological contamination. These responses are highly related because fecal coliform contamination resulting in repeated bouts of diarrhea have been shown to reduce the child's ability to absorb nutrients, thus leading to death or growth stunting (Gadgil 1998). The quality of

[4]See the website www.cerch.org/research-programs/chamacos for additional information.

water in economic studies is proxied by the presence of piped water. Galiani, Gertler and Schargrodsky (2005) investigate the impact of privatizing piped water services in Argentina and conclude that privatization results in improved water quality, more households connected to piped water services and decreased infant mortality. Gamper-Rabindran, Khan and Timmins (2007) show similarly significant decreases in child mortality due to public expansion of piped water services in Brazil. However, the presence of piped water is an imprecise measure of the quality of water and thus the estimated parameters are intent to treat effects because the actual cleanliness of the water is unknown. These intent to treat effects are important parameters because piped water is the most common long term solution for the over 1 billion people on earth without access to adequate drinking water. However, as demonstrated by Bennett (2010) piped water can have unintended consequences. The study shows that clean water and sanitation are substitutes, so if people perceive their water to be cleaner then they will take fewer actions to ensure that their surroundings are sanitary. Therefore, the benefits of piped water are mitigated by behaviors that recontaminated the water. Piped water can also have non-biological contamination, though past studies of this type of contamination solely focus on the arsenic contamination in Bangladesh, a non-biological agent which has numerous reported long term health consequences . In this setting, arsenic contamination is not due to economic activities but to naturally occurring arsenic rich geology. Two studies of arsenic in Bangladesh, Soumya et al (2011) and Field, Glennerster and Hussam (2011), in combination show that the public health effort to inform people about the arsenic levels in their water sources was effective, people switched to arsenic-safe sources and did not switch back, but that many of them switched to biologically contaminated sources and diarrheal incidence has increased. Other sources of non-biological contamination are more economic, such as the use of pesticides in agriculture or the effluent of industrial processes. My study will examine the health effects of non-biological contamination of water due to economic activities using various measures which exploit geographic and infrastructure characteristics of the study's context. For example, I obtained from the Metro Cebu Water District (MCWD) a detailed map of the layout of pipes and the dates when each section became operational. The MCWD pipes are subject to high levels of seepage due to transient water pressure, thus combined with other information collected regarding the location of industrial polluters in Metro Cebu, the quality of water that arrives at the household's tap varies due to the layout of pipes, the age of the pipes and both the topography and precipitation of the surrounding area influencing the sources and amount of contamination. To date, no economic study (of which I am aware) has examined the long term health effects of non-biological contamination of water.

Air quality studies in economics have employed a few more health outcomes than water quality studies, however each study has only involved short term responses. Moreover, each study of air quality face two critical concerns: the imprecision of exposure measures and the endogeneity of exposure to locational sorting and compensating behaviors. It is clear that an individual's exposure to airborne contaminants is not closely related to the nearest air quality monitor, but rather the microenvironments - the home, the neighborhood, and the workplace - where the individual spends time determine the true exposure (Georgopoulos et al 2008). However, data is generally only available for environments defined at a much more macro level. It is also clear that the environment plays a role in housing decisions. Chay and Greenstone (2004) show that air quality is capitalized in housing prices, making some measures of air quality endogenous to unobservable characteristics of the family and individual. The study by Currie and Schmeider (2008) illustrates the empirical difficulty in addressing these issues. The US EPA's Toxic Release Inventory contains very

detailed information regarding the emissions of polluting industrial plants. However, only annual data for counties is available. Therefore the health outcomes are necessarily aggregated to the county-year level and identifying variation occurs across counties, ignoring residential sorting and differences across counties. My research will focus solely on one metropolitan area, Cebu, Philippines, with geographic distinction made at the neighborhood level (known as barangay in the Philippines) and the unit of time will be the month. Other studies have been able to refine the geography and the time to zip code and month, with the implicit assumption that sorting decisions within the particular geographical-time unit are exogenous to health outcomes (Currie et al 2009 and others). Other studies have exploited unexpected changes in emissions due to economic shocks, showing that when production decreases due to economic downturns result in lower levels of emissions fewer infant deaths and acute respiratory infections are observed (Pope 1989 and Chay 1999. However, since these lowered emissions are due to economic downturns, behavior may be altered in a way that affects health.[5] Using geographic peculiarities of Metropolitan Cebu the present study will exploit variation in air quality unrelated to sorting decisions and compensating behaviors. For example, the island of Cebu lies in the center of the Philippine islands, running on a Southwest to Northeast axis. Coincidentally, the region experiences seasonal wind patterns known as the Amihan (a northeastern wind) and the Habagat (a southwestern wind). These wind patterns mean that the sources of air pollution need not be closely proximate to the individual in order to exert an effect. I combine these wind patterns with the timing developmental utero to form exogenous measures of air quality.

3 Model and Methodology

3.1 Model and Empirical Specification

Begin with the general description of the health of the organs n of the body as $\theta_{n,t,s}$, where t denotes the time period and s the stage of development. Let $f_{n,t,s}(\cdot)$ denote the production function of the health of organ n. Imposing an intertemporal framework, inputs are accepted and production commenced during time period t and stage s with the resulting health of the organ observed in period $t' > t$ (also note that s' is not strictly greater than s). Let $I_{t,s}$ denote inputs beneficial to the production of health and let $\sigma^*_{t,s}$ denote the true, underlying latent level of detrimental inputs or contaminants. ψ denote the vector of parameters of the production function.

$$\theta_{n,t',s'} = f_{n,t,s}(I_{t,s}, \sigma^*_{t,s}, \psi) \text{ for } n \in \{1, 2, ..., N\} \text{ and } t' > t \text{ and } s' \geq s \tag{1}$$

Furthermore, let $\sigma^{q*}_{t,s}$ for $q \in \{a, w\}$ denote contaminants resulting from air and water inputs. Therefore, the health of the organ n, or $\theta_{n,t',s'}$ is a function of beneficial inputs and air and water contamination.

$$\theta_{n,t',s'} = f_{n,t,s}(I_{t,s}, \sigma^{a*}_{t,s}, \sigma^{w*}_{t,s}, \psi) \text{ for } n \in \{1, 2, ..., N\} \text{ and } t' > t \text{ and } s' \geq s \tag{2}$$

[5] Another study examined the 1950 to 1980 policy of the Chinese government that heating was a basic right and should be freely provided by the government. Due to budgetary restrictions this right was only extended to areas north of the Huai River and as a result levels of total suspended particulates were 5-8 times higher north of the Huai River **?**. This study did not analyze the health effects of this policy due to a lack of data but it is the only study examining air quality in a developing country context, where there is a general paucity of data.

Cunha, Heckman and Schennach (2010) adopts a constant elasticity of substitution empirical specification for the dynamic skill formation function similar to the production function described above. This functional form is appealing because its ability to capture a variety of interesting special cases, such as substitutes and complements. Applying the constant elasticity of substitution functional form allows the effects of investments to be non-monotonic. Suppressing the time t subscripts for simplicity and additional focus on the stages of human development (such as embryonic, fetal, etc), and adding a vector of controls, X_s, the resulting empirical specification is as follows:[6]

$$\theta_{n,s'} = \left[\alpha_s(\sigma_s^{a*})^\phi + \omega_s(\sigma_s^{w*})^\phi + \delta_s I_s^\phi + \beta X_s^\phi\right]^{1/\phi} \tag{3}$$

3.2 Methodology

With the intent of finding a measure of $\sigma_{t,s}^{q*}$, the endogeneity of this measures is a consistent concern in studies regarding the health impacts of the environment. Because of the capitalization of environmental quality in housing prices, exposure determined at some geographic area is likely endogenous to residential sorting and accompanying unobserved heterogeneity. Additionally, because dose actually effects the change within the body and not exposure, omitted and unobservable compensating behaviors are likely sources of endogeneity. However, beyond these concerns there is the issue that the available measures of air or water quality are imperfect, noisy descriptions of the individual's exposure to environmental contamination. I address this issue by employing various measures of air and water quality, each arguably exogenous but each undoubtedly imperfect and noisy. These multiple measures are used to identify the distribution of the latent air and water quality using the Cunha, Heckman and Schennach (2010) framework.

Let m denote the measure and M^q the total number of measures: $m \in \{1, 2, ..., M^q\}$ for $q \in \{a, w\}$. Recall $\sigma_{t,s}^{q*}$ is the latent quality of the input, air or water, consumed in period t during the developmental stage s. And let $\sigma_{t,s,m}^{q}$ denote the mth measure of input quality. Adopting a linear form for the relationship between the noisy measure and the latent variable, let $\mu_{t,s,m}^{q}$ be the mean function and $\epsilon_{t,s,m}^{q}$ the error term. The coefficients $\gamma_{t,s,m}^{q}$ are the factor loadings.

$$\sigma_{t,s,m}^{q} = \mu_{t,s,m}^{q} + \gamma_{t,s,m}^{q}\sigma_{t,s}^{q*} + \epsilon_{t,s,m}^{q} \tag{4}$$

Assume that $E(\epsilon_{t,s,m}^{q}) = 0$. Normalizing $E(\sigma_{t,s}^{q*}) = 0$ for all t, given measurements $\sigma_{t,s,m}^{q}$ the mean functions are identified as the means of the measures $\sigma_{t,s,m}^{q}$. The factor loadings $\gamma_{t,s,m}^{q}$ remain to be identified. One more normalization, $\gamma_{t,s,1}^{q} = 1$, is required. Therefore, computing the covariances of the measures the factor loadings are identified:

$$Cov(\sigma_{t,s,1}^{q}, \sigma_{t+1,s,1}^{q}) = Cov(\sigma_{t,s}^{q*}, \sigma_{t+1,s}^{q*}) \tag{5}$$

$$Cov(\sigma_{t,s,2}^{q}, \sigma_{t+1,s,1}^{q}) = \gamma_{t,s,2}^{q} Cov(\sigma_{t,s}^{q*}, \sigma_{t+1,s}^{q*}) \tag{6}$$

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[6]Much more expansive development of the model is given in the appendix.

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$$Cov(\sigma^q_{t,s,M^q}, \sigma^q_{t+1,s,1}) = \gamma^q_{t,s,M^q} Cov(\sigma^{q*}_{t,s}, \sigma^{q*}_{t+1,s}) \tag{7}$$

The factor loadings $\{1, \gamma^q_{t,s,2}, \gamma^q_{t,s,3}, ..., \gamma^q_{t,s,M^q}\}$ are therefore identified by the ratio of covariances, such as dividing equations (11) and (12) by equation (10).

$$\frac{Cov(\sigma^q_{t,s,m}, \sigma^q_{t+1,s,1})}{Cov(\sigma^q_{t,s,1}, \sigma^q_{t+1,s,1})} = \gamma^q_{t,s,m} \tag{8}$$

Having identified the factor loadings, $\gamma^q_{t,m}$, the measures of latent input quality can be identified for each t.

$$\frac{\sigma^q_{t,s,m}}{\gamma^q_{t,s,m}} = \frac{\mu^q_{t,s,m}}{\gamma^q_{t,s,m}} + \sigma^{q*}_{t,s} + \frac{\epsilon^q_{t,s,m}}{\gamma^q_{t,s,m}} \tag{9}$$

Collecting the measures of latent input quality for all t, the distribution of input quality can be identified.

$$\sigma^{q*} = \left(\{\sigma^{q*}_{t,s}\}^T_{t=1} \right) \text{ for } q \in \{a, w\} \tag{10}$$

Of more particular interest in this study is the input quality during the critical periods of development. How exactly to relate exposure to health is an open question. Is it the integral under the exposure-duration curve or the integral under the curve but above some threshold level of exposure, a level which the body can handle and eliminate but above which the toxins accumulate and exert and effect? And what is the appropriate threshold? This study will not address the issue of thresholds and manageable levels of exposure, rather the total integral under the exposure-duration curve will be examined for its relation to health. Furthermore, because the measures of exposure are weighted by duration, the relevant variable is the sum of exposures during the stage of development.

$$\sigma^{q*}_s = \sum_{t \in s} \sigma^{q*}_{t,s} \text{ for } q \in \{a, w\} \tag{11}$$

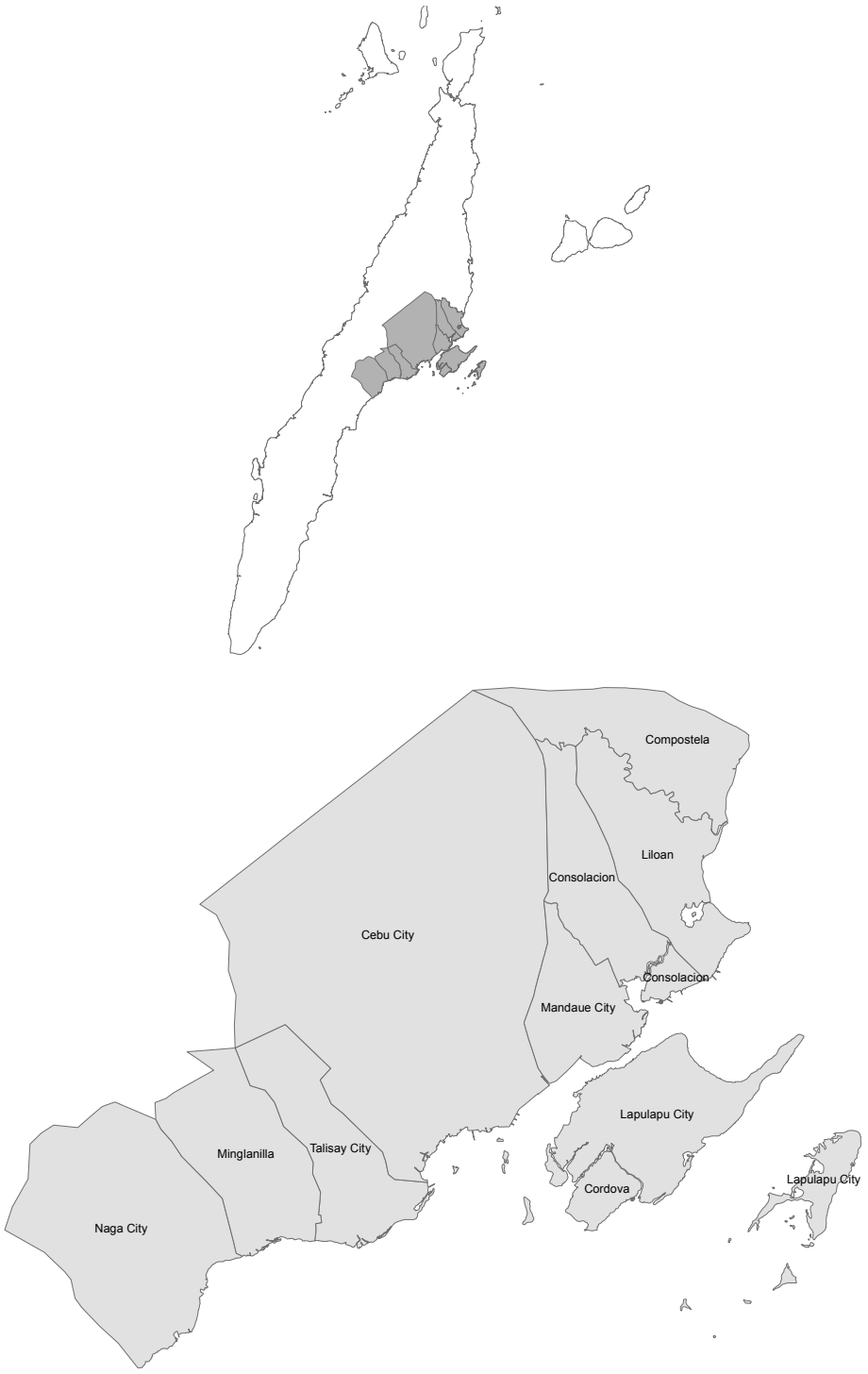
Note that the is the same as in equation 3.

4 Empirical Implementation

4.1 Location: Cebu, Philippines

Metropolitan Cebu is located in the Central Visayas region, otherwise known as Region VII, of the Philippines, on the island of Cebu. Currently, the metropolitan area encompasses seven cities and six municipalities, however in 1983 there were five cities and five municipalities - Cebu City, Mandaue City, Talisay City, Lapu-lapu City, Naga City, Consolacion, Liloan, Cordova, Minglanilla and Compostela. When the Cebu Longitudinal Health and Nutrition Survey began 33 barangay - or wards, which are the smallest administrative division in the Philippines - in 8 of the aforementioned cities and municipalities, all but Minglanilla

and Compostela.



Metropolitan Cebu is a particularly interesting context for various reasons. First, over the past few decades the area has experienced increasing amounts of industrial activities, culminating in the recent description of the region's director of the Environmental Management Bureau that Cebu today is like Manila

of the 1980s, a city and time of famously high levels of pollution.[7] Because of the development of Cebu over the recent years the environmental costs can be examined. Additionally, there are geographic and infrastructure peculiarities which give rise to arguably exogenous measures of air and water quality. Finally, and most importantly, a longitudinal survey collecting health information on individuals beginning before birth, frequently during the first two years of life, and every few years until the present time has taken place in Cebu since 1983. This survey is called the Cebu Longitudinal Health and Nutrition Survey.

Metropolitan Cebu is a particularly important economic center for the Philippines due to the large number of manufacturing plants and its location which makes it a large distribution hub for the rest of Asia. By recent count, approximately 1000 industrial establishments are located in the province of Cebu, mainly concentrated in Cebu City, Mandaue and Lapu-lapu. Cebu City is the commercial, business and international trade center of the metropolitan area. Mandaue is the industrial center of the metropolitan and Lapu-lapu is another international trade center and hub of tourism. Mineral resources are abundant in the region and mining for ore, copper, manganese, iron, gold and silver comprise one of the largest revenue sources for the region. Apart from mining, there are many other industrial firms in the area producing fertilizer, sugar, cement, and processing food. Although farming has diminished throughout the Philippines, there remain a few farms in Metropolitan Cebu and farming in the province accounting for a little under 10% of all the farming area in the Philiippines at last calculation. Furthermore, the sources of illness gives an indication that the health costs of pollution in the Philippines are quite large. The main transport media of pollutants to humans are air and water, estimated by the National Epidemiology Center of the Philippines showing 62% of illnesses are related to the respiratory system, while 31% are related to water, and just 7% other. The Department of Health reported in 2007 in the area of Metropolitan Cebu the five leading causes of mortality are infant and communicable diseases, and the five leading causes of morbidity are diarrhea, pneumonia, bronchitis, influenza and hypertension.[8]

4.1.1 Water in Cebu

The provision of clean water is a critical problem throughout the Philippines, though much more so in the urban areas such as Metro Cebu. A large portion of the country is not connected to piped water, and even fewer connected to sewage systems. Due to the shortage of investment in sanitation a major form of water pollution is organic resulting in high levels of disease such as gastroenteritis, diarrhea, dysentery and others. However, due to economic development and increased industrial activity, organic pollution of the water supply is not the only problem. Mining is increasingly prevalent in the Philippines due to the abundance of natural resources, which combined with poor monitoring, surveillance and enforcement of environmental regulations leads to high levels of mining effluent in the groundwater. Weak enforcement of environmental regulations also result in the prevalence of other industrial effluent in the groundwater. In 2004 the government passed the Clean Water Act, after passing a series of ineffectual legislation aimed at protecting this natural resource. It is currently unclear how effective this legislation has been. Prior to that, the estimated annual losses due to water pollution in the Philippines are over 1.3 billion US dollars (World Bank 2003).

[7] Allan Aranguez, Environmental Management Bureau, Region VII, quoted in Parco, B. Metro Cebu's air quality as bad as Manila's, Cebu Daily News, June 2008.

[8] The source for all of the information presented in this paragraph is the Regional State of the Brown Environment Report of 2010, produced by the Environmental Management Bureau of the Department of Environment and Natural Resources in Region VII.

The Philippines is generally known to have an abundant supply of water, but this is not true for Cebu. The water situation in Cebu is challenged by the ever growing demand and diminishing supply, as well as the continual contamination via economic activities. Nearly all of the water supply of Cebu, 98%, is from groundwater **?**. The precipitation recharge of the available groundwater in Cebu is approximately 86.4 million cubic meters per year. The amount of pumped water per year by the Metro Cebu Water District and all other private users totals approximately 84 million cubic meters per year, almost the amount of recharge. As the population grows, tourism increases, and the economy develops the demand for water will also grow and very soon begin to exceed the annual recharge. Furthermore, the permanent supply of water in the underground aquifers is threatened by seawater intrusion. Because of Cebu's location on the coast, and the location of its water supply in underground aquifers, the freshwater is vulnerable to the intrusion of saltwater into the aquifers. Both the Water Resources Center at the University of San Carlos and the Metro Cebu Water District have been monitoring seawater intrusion since that time.

Besides the location of Metro Cebu along the coast, a number of other locational peculiarities make the water situation of Metro Cebu very dire. The lack of forest cover in Cebu limits the accumulation of rainfall recharge and magnifies the problem of pesticide contamination in agricultural runoff. Furthermore, the quality of ground and surface water is threatened by economic activities such as mining and industrial production. Take for example the Butuanon River. The Butuanon River originates in the mountains of the island of Cebu and travels through the city of Mandaue until emptying into the Mactan channel that divides the islands of Cebu and Lapu-lapu. This river is closely monitored by the Environmental Management Bureau of Region VII because of its location within a heavily industrialized area. It is subject to essentially indiscriminate wastewater dumping by both domestic and industrial sources. Within the watershed, an area of land where all of the water drains into the same place, are located approximately 20 industrial sources of effluent, including a steel foundry and a furniture manufacturer (EMB 2010). Geographic information regarding watersheds in Metro Cebu and the location of mines and industrial polluters will be used to produce variation in the quality of water consumed by individuals without access to piped water.

Piped water in Metro Cebu is supplied by one agency, the Metro Cebu Water District (MCWD). The Local Water Utilities Act of 1973 created the Metro Cebu Water District, built upon the 25.4 million Philippine pesos of infrastructure assets of the Osmena Waterworks System created in 1910, including the Buhisan Dam that still provides 5% of the water in the area. Apart from the dam, the MCWD obtains the remainder of its water supply from over 100 deep wells. 58 million pesos and 112 million pesos were invested in infrastructure in 1976 and 1983 respectively, following my more large scale investments every few years.[9] There have been numerous expansion and maintenance efforts performed over the years by the MCWD, however both remain a critical need for the utility. In 2005, 55% of the population within the area of the MCWD were connected by pipes, and the leakages accounted for just under 30% of production. Furthermore, in 2005 consumers averaged 20 hours per day of service in their pipes. The service of water is interrupted regularly for various reasons such as the regulation of supply, maintenance, upgrades, and the lack of power at the pumps (ADB 2005). These service interruptions as well as the layout of pipes and the location of mines and industrial polluters will be used to produce variation in the quality of water consumed by individuals with access to piped water.

[9]For additional information on the MCWD see their website at www.mcwd.gov.ph

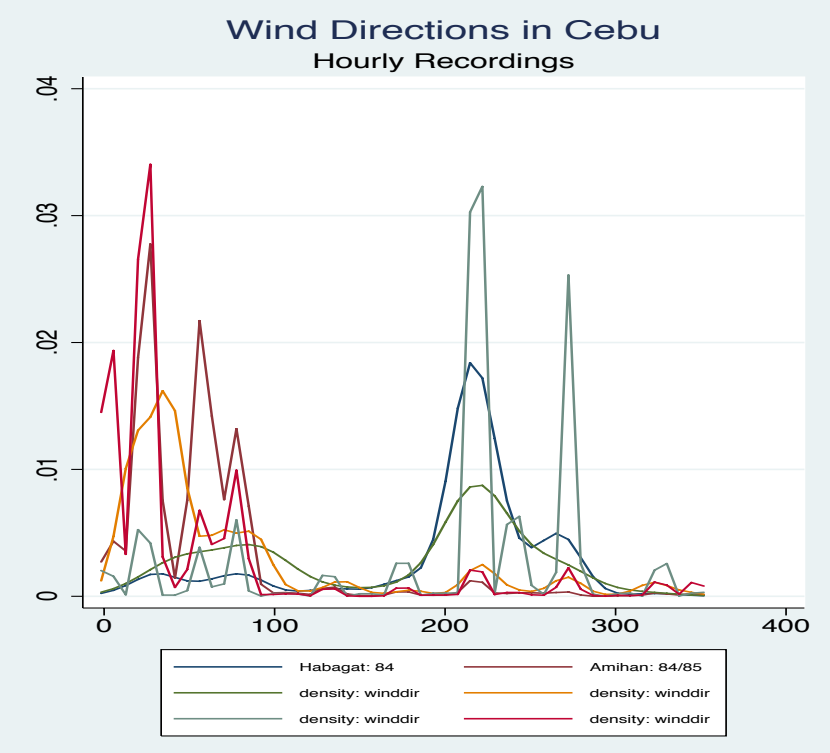
4.1.2 Air in Cebu

Air pollution occurs when the amount of chemical and particulates in the emissions from economic processes exceeds the earth's natural capacity for assimilation. Despite scarce data, one study by the World Bank has estimated the the health costs of exposure to particulates less than 10 micrometers in size, or PM_{10}, are over 400 million US dollars annually in the 4 largest urban centers of the Philippines, of which Metro Cebu is one. The government of the Philippines began during the 1990s to make progress in addressing the rising amounts of air pollution, including passing the 1999 Clean Air Act which increases monitoring and the role of the private sector, as well as employing market based instruments to reduce pollution. However, inadequate investment and weak institutional capacity have mitigated the potential effectiveness of all attempted legislation passed by the government (World Bank 2002).

Metro Cebu is rapidly urbanizing and industrializing and as a result is experiencing high levels of air pollution, however official monitoring of air quality in Metro Cebu by the Department of the Environment and Natural Resources did not begin until the mid 1990s. In general, the sources of air pollution in the area of Metro Cebu can be divided into stationary and mobile sources. Stationary sources are immobile structures, facilities or installations such as power and manufacturing plants. In Metro Cebu a variety of stationary sources of air pollution have been identified: coal-fired power plants, cement plants, sugar mills, food processing plants and beverage plants. Coal-fired power plants are a particular problem in Metro Cebu, where two plants are located and have been operating for multiple decades. These are major sources of particulate matter and sulfur dioxide. Mobile sources of air pollution are machines propelled or powered by oxidation or reduction reactions such as carbon based fuel combustion (EMB 2010). These vehicles are the major sources of carbon monoxide, ozone, particulate matter, nitrogen oxides, and lead. The types of vehicles in Cebu range from motorcycles to cars and taxis to jeepneys, a poplar mean of public transportation made from left over US military jeeps after World War II. To this day, almost all jeepneys as well as trucks, buses and utility vehicles run on diesel fuel, a main source of fine particle and nitrogen oxide emissions. Additionally, the heavy particle emitting two stroke engines dominate the motorcycle market in Cebu over the more efficient four stroke engines **?**. Furthermore, lead was not eliminated from gasoline until 2001 in the Philippines. The detrimental effects of lead - ranging from neurological disorders to cardiovascular disease - were not recognized in the Philippines until the 1990s, and in 1993 the lead content in gasoline began to be phased out until being completely eliminated in 2001 **?**. The Environmental Management Bureau in region VII has been monitoring the air quality of Cebu at least at a basic level since 1995 and between 1995 and 2000 the level of total suspended particulates exceeded the standard each year (Villafane 2001). Recent measures of air quality during the years of 2008-2010 have shown a consistent downward trend to the levels of total suspended particulates indicating government and consumer reactions to the high levels of pollution (EMB 2010). However, the exposure period of interest for this study are the years 1983-1985, a time period during which pollution abatement efforts were not undertaken, and geographic information regarding transportation will be combined with prevailing wind patterns to produce geographic variation in air quality.

A peculiar and important characteristic of Cebu which heavily influences local concentrations of air pollutants are the prevailing wind patterns, the Amihan and the Habagat. Amihan describes a season dominated by cool northeast winds, and the Habagat describes a seasons dominated by warm southwest wind. Furthermore, Metro Cebu is oriented on an axis running from the southwest to the northeast, meaning that

these wind patterns sweep cleanly across Metro Cebu from the northeast during the Amihan and from the southwest during the Habagat. The exposure of individuals to pollution sources not directly nearby is dominated by these wind patterns. The location of stationary sources of air pollution combined with the Amihan and Habagat wind patterns will be used to produce geographic variation in air quality. Generally, the Amihan begins in September or October and lasts until April or May when the Habagat begins. Using data obtained from the National Climatic Data Center containing hourly weather dating back to before the critical 1983-1985 years of the CLHNS survey, the kernel density plots below describe the prevailing wind patterns. Wind direction is recorded as degrees between 0 and 360, with 90 degrees indicating East, 180 indicating South and 270 indicating West. As depicted in the figure below, the two seasons differ markedly, with vast majority of observations during the Amihan period centered around 45 degrees (40 degrees is the mode observation), and the majority of observations during the Habagat centered around 210 (220 degrees is the mode observation).



4.2 Data

When thinking how to quantify the health costs to environmental contamination it is instructive to imagine the ideal data. The location of the study would be one where there was enough pollution to identify an effect but not so much that it would be drastically different and inapplicable to other locations. Individuals would be followed throughout their life course to observe latent and intergenerational effects. Data would be collected frequently to capture the duration of exposure. Both exposure and dose measures would need to be collected for each respondent in order to correctly asses the damage inflicted by certain levels of pollution. Optimally, an air quality monitor would be taken with individual during all hours of the

day and all food and water consumed would be sampled and tested for a huge variety of contaminants, not solely those known to be emitted by various sources but potential secondary chemicals. Additionally, blood, urine and stool samples should be frequently collected and tested for an even larger variety of substances due to the metabolic ability of the body to transform ingested substances. Furthermore, DNA samples would be collected and tested in order to understand the epigenetic effects of the toxin's dose. In addition, demographic and economic information such as income, education, household composition and time-use diaries to again attempt to control for compensating behaviors. Obtaining this data would be extraordinarily costly, practically infeasible, however by combining data from various sources I believe the data I employ is the best suited to addressing the costs of environmental contamination on health.

The Cebu Longitudinal Health and Nutrition Survey (CLHNS) randomly sampled 33 barangay (17 urban and 16 rural) in Metro Cebu in order to form a cohort of pregnant women. The barangays contained roughly 28,000 households, all of which were canvassed in search of pregnant women in late 1982 and early 1983. Women of the selected barangays who gave birth between May 1, 1983, and April 30, 1984, are included in the baseline sample taking place during the 6th or 7th month of pregnancy. The following table describes the distribution of the sample over the various cities and barangay included in the sample.

Sample Distribution by City and Barangay

	Percent	N
Cebu City	46.81	1461
Quiot Pardo	4.2	131
San Nicolas	2.95	92
Sambag II	6.98	218
Basak Pardo	4.97	155
T. Padilla	5.58	174
Labangon	10.77	336
Lorega San Miguel	7.75	242
Budla-an	1.89	59
Pamutan	1.73	54
Consolacion	11.76	367
Cansaga	0.54	17
Poblacion	3.43	107
Danglag	0.8	25
Panoypoy	0.54	17
Pulpogan	5.35	167
Tolo-tolo	1.09	34
Cordova	1.44	45
Cogon	1.44	45
Lapu-lapu	9.42	294
Basak	5.51	172
Poblacion	2.85	89
Cao-oy	0.67	21
Caohagan	0.38	12
Lilo-an	0.9	28
Santa Cruz	0.9	28
Mandaue	13.81	431
Opao	3.97	124
Mantuyong	3.4	106
Basak	2.92	91
Casuntigan	3.52	110
Naga	8.81	275
Jaguimit	1.22	38
Balirong	2.24	70
Bairan	0.83	26
Cantao-an	2.18	68
Inoburan	1.86	58
Poblacion	0.48	15
Talisay	7.05	220
San Roque	4.42	138
Mojon	2.63	82
Urban Population	76.39	2384

In total 3,327 women were surveyed at baseline and at birth 3,080 were resurveyed (attrition due to non-single live births and migration). Following the child's birth, the mother-child pair was resurveyed every two months for the first two years of the child's life. The following table provides summary statistics regarding the infant health.

Summary Statistics: Infant Health

	Mean	N
Male	0.53	3,121
Gestation Weeks (from upper bound conception)	35.96	3,097
	(3.26)	
Gestation Weeks (from lower bound conception)	38.15	3,097
	(3.27)	
Age at Death of Infants (< 2 yrs) in weeks	40.69	148
	(30.62)	
Average of 3 Skinfold Measures	12.78	3,119
	(4.02)	
Birth Length centimeters	49.25	3,051
	(2.14)	
Birth Weigh grams	3,010.97	3,085
	(477.22)	
Reported Low Birth Weight %	13.3	3,121
Birth Weight <2500g %	13.94	3,121
Delivery Complications %:		
None	85.58	3,121
Heavy Bleeding	3.33	3,121
Prolonged Labor	1.99	3,121
Breech	1.15	3,121
Forceps	0.51	3,121
Dry Labor	0.38	3,121
Coiled Cord and Other	4.01	3,121
Birth Month %:		
January	6.89	3,121
February	6.5	3,121
March	6.57	3,121
April	5.38	3,121
May	8.3	3,121
June	8.72	3,121
July	10.12	3,121
August	9.68	3,121
September	10.64	3,121
October	9.84	3,121
November	9.39	3,121
December	7.98	3,121

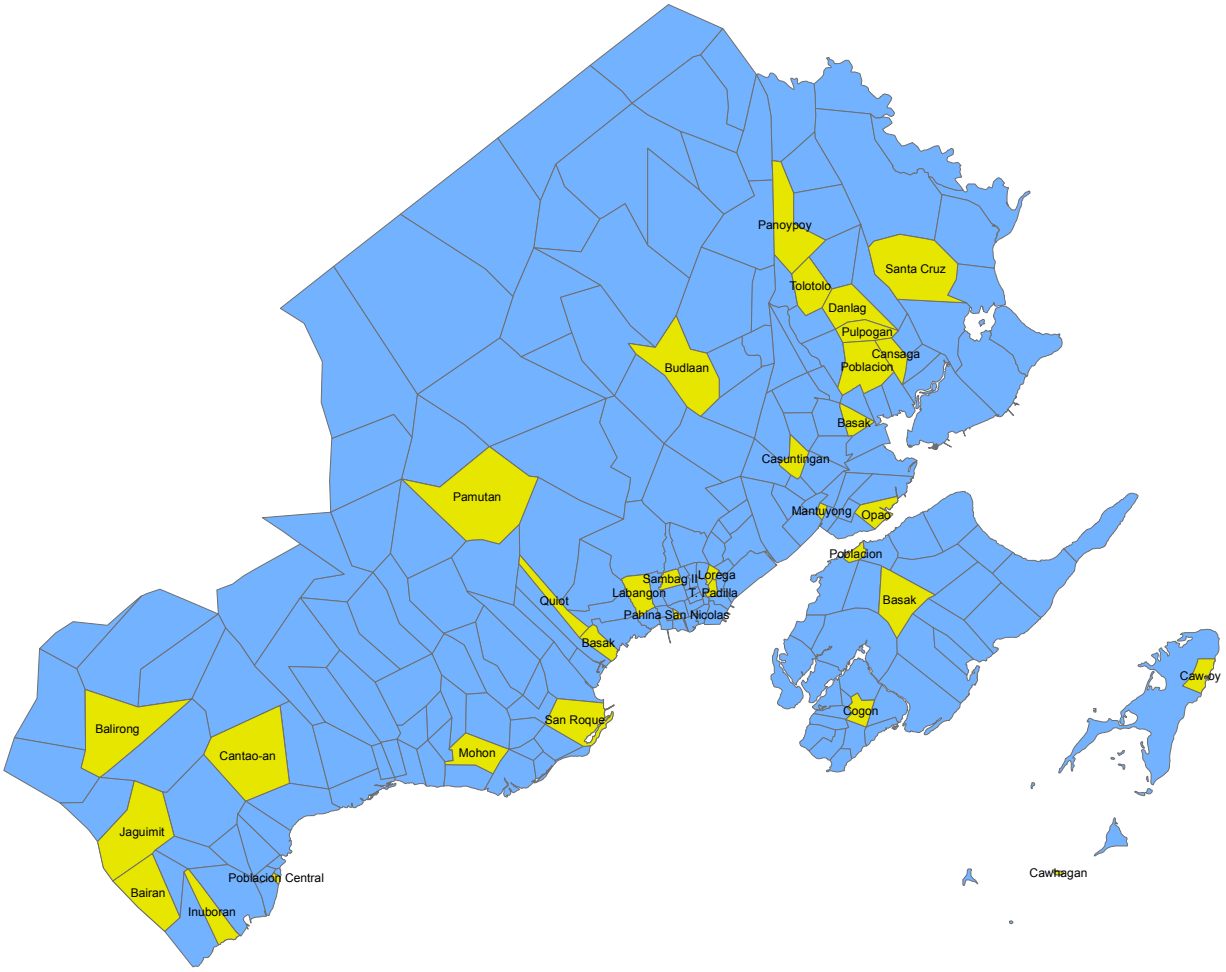
After the first two years, respondents were followed every 3-4 years with the latest survey taking place in 2007. Household composition, income and other demographics are collected in each survey wave. Community surveys were also collected with information about local businesses. Additionally, extensive anthropometric measures were collected in each wave as well as food and time diaries. Furthermore, the survey is sufficiently long to include information on the reproductive histories of the cohort of children and many adult health outcomes. In 2005 biomarkers from blood samples were collected for the entire sample following an overnight fast. These blood samples were analyze for cardiovascular risk factors with the following assays performed: c-reactive protein, homocysteine, insulin, glucose and lipids. Elevated levels of c-reactive protein and homocysteine have been shown to be related to environmental contaminants such as particulate matter (for both), pesticides and heavy metals (in the case of c-reactive protein). The following table provides basic summary statistics of later life outcomes, such as health.

Later Life Outcomes:	
Mortality:	
Died between birth and age 2 %	1.83
Died between age 2 and 5 %	0.19
Died after age 5 %	1.28
Morbidity in 2005 (n=1912):	
Chronic Illness or Disability %	12.50
Ever Smoked %	59.68
Poor Reported Health %	7.22
Biomarkers in 2005 (n=1756):	
C-Reactive Protein	1.63
	(5.56)
Homocysteine	12.53
	(4.94)
C-Reactive Protein >3 mg/L%	50.00
Homocysteine >12 mg/L%	73.00
Anthropometric in 2002 (n=2023):	
Weight in kilograms	49.78
	(8.66)
Height in centimeters	156.89
	(10.27)
Systolic Blood Pressure	102.91
	(10.76)
Diastolic Blood Pressure	66.95
	(9.11)
Pre hypertension %	6.32
Anthropometric in 2005 (n=1888):	
Weight in kilograms	51.53
	(10.20)
Height in centimeters	157.22
	(10.46)
Systolic Blood Pressure	105.89
	(13.21)
Diastolic Blood Pressure	72.31
	(10.56)
Pre hypertension %	14.00
Anthropometric in 2007 (n=1817):	
Weight in kilograms	50.69
	10.61
Systolic Blood Pressure	109.39
	(12.44)
Diastolic Blood Pressure	73.57
	(10.51)
Pre hypertension %	26.00
Other in 2005 (n=1912):	
Graduate High School	39.85
Some College	21.13

4.2.1 Identification Strategy and Measures of Exposures

An intuitive explanation of the identification strategy is to exploit the combination of the timing of critical developmental periods with geological, infrastructure and climatic peculiarities of Metro Cebu. The general problem facing all environmental health studies is that exposure that varies by geographic location is possibly endogenous to unobservable determinants of health. For this study it will be critical to identify valid counterfactuals and because of the nature of the study this will be done by location. The most specific residential information collected on the survey respondents is the barangay. Barangays are sub-city level organizations comprising approximately 3 to 4 square miles on average in Metro Cebu. For instance,

Cebu City covers a land area of 292 square kilometers and is divided into 80 barangay. The 33 barangay of the CLHNS survey were selected from 6 cities/municipalities of Metro Cebu: Cebu City, Consolacion, Lapu-lapu, Mandaue, Naga and Talisay. The following figure shows the location of each of the sample barangays.



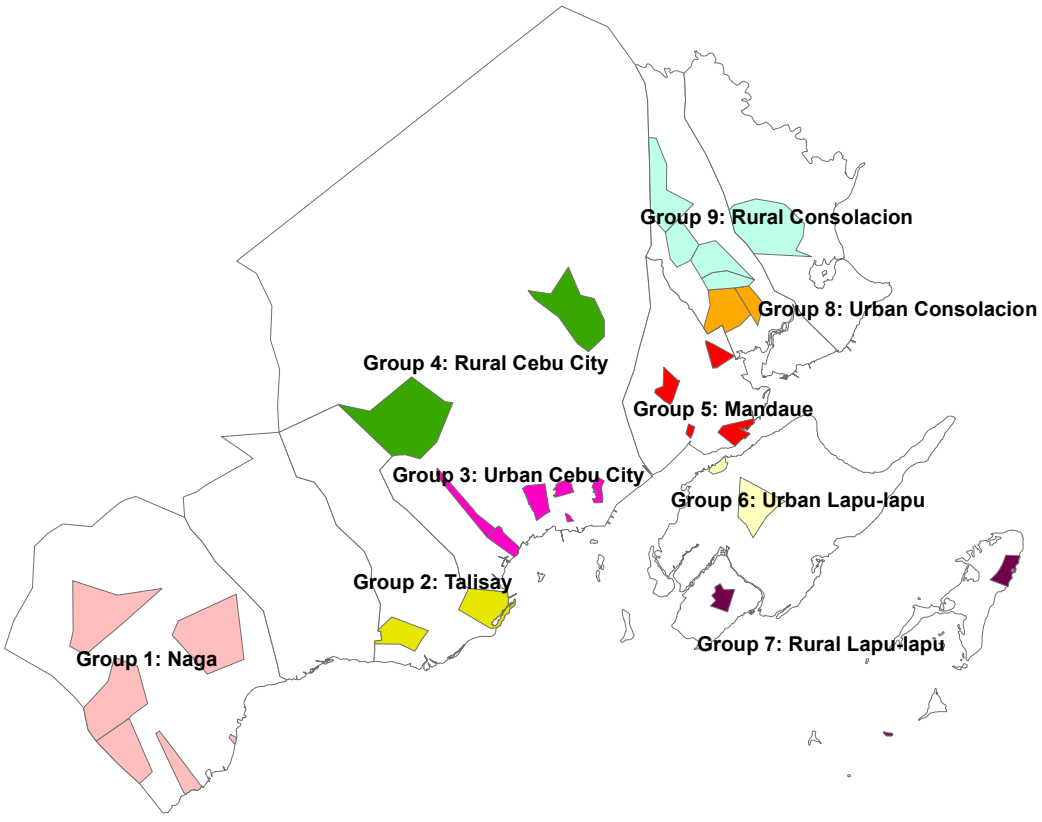
Because the survey area is concentrated in one metropolitan area, one might expect the differences between individuals living in one barangay to individuals living in another barangay to be small. Regardless the differences do exist both by reputation - such as Mandaue's reputation as the industrial hub or Lapu-lapu's reputation as the tourist hub - and by analysis. The following table shows regressions of health outcomes on city binaries, purely correlation however it is evident that differences exist between the cities.

Health Outcomes and City Correlations

	1 bth_weight	2 bth_height	3 skinfold	4 crprotein	5 homocys	6 insulin
Consolacion	-7.17	-0.22*	-0.32	0.52	-0.28	-0.72
	-28.03	-0.13	-0.23	-0.43	-0.38	-0.63
Cordova	-140.13*	-0.48	-2.33***	-0.76	0.09	2.55*
	-73.83	-0.33	-0.6	-0.94	-0.83	-1.38
Lapu-Lapu	34.76	0.40***	-0.58**	0.88**	0.70*	-1.78***
	-30.56	-0.14	-0.25	-0.44	-0.39	-0.64
Liloan	-9.05	0.26	-1.16	-1	-2.16*	1.98
	-91.03	-0.41	-0.76	-1.26	-1.12	-1.86
Mandaue	21.4	0.19	-0.55**	0.22	0.44	-0.26
	-26.36	-0.12	-0.22	-0.42	-0.37	-0.62
Naga	-31.42	-0.47***	-1.67***	0.37	-0.94**	-1.14*
	-31.77	-0.14	-0.26	-0.47	-0.42	-0.69
Talisay	39.96	-0.31**	-0.57**	0.1	-0.21	-1.91**
	-34.59	-0.16	-0.29	-0.53	-0.47	-0.79
Constant	3,007.49***	49.28***	13.18***	1.42***	12.56***	9.18***
	-12.53	-0.06	-0.1	-0.2	-0.18	-0.3
Observations	3,085	3,051	3,119	1,756	1,756	1,756
R-squared	0	0.01	0.02	0	0.01	0.01

Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

However, there are also differences within the cities across urban and rural divides. Therefore the reference groups that I will employ in my analysis within the same city and urban/rural classification except the two barangay, Santa Cruz, Liloan and Cogon, Cordova, which stand alone in their respective municipalities. These are combined with barangay in neighboring cities with the same urban/rural classification in order to create 9 reference groups. Identification is obtained by variation among barangay within the same reference group, as depicted below in the following figure.



Let us first consider the identifying variation for air quality. Variation in air quality within the barangay is due to timing. Amihan and Habagat wind patterns in the Philippines change which air pollution sources lie upwind of the household, thus in combination with the critical periods of development in early childhood the child's exposure to emitted by sources upwind is very coincidental. Variation in observed birth dates for children within the barangay as well as the timing of wind patterns and the location of air polluters gives the identifying variation. This strategy will be employed for various sources of airborne pollution such as stationary sources like industrial and coal-fired power plants and mines, as well approximations of mobile sources, and this variation is generated using sources located outside of the barangay, holding variation within the barangay constant and avoiding the problem of sorting. Information regarding the number vehicles by barangay in Metro Cebu is unavailable so I will approximate the pollution from mobile sources using road density. Using GIS maps provided by the Cebu Provincial Planning and Development Office (PPDO), I can determine the density of roads in each direction of the barangay. Combined with wind data, this give an approximation to airborne pollution exposure by mobile sources.

Stationary sources of pollution will be used both for air and water quality. I employ information regarding the stationary sources of pollution from various sources. First, the Environmental Management Bureau has provided me with lists of each industrial polluter by type (air, water, hazardous material) and industry dating back to 1999. Clearly, this does not provide a description of stationary sources during the years 1983-85. However, I was able to find and obtain phonebooks from the years 1983, 1984 and 1985 from the Directories Philippines Corporation (DPC) which provide information regarding the existence, location and industry of companies during those years. By using the EMB data to determine which types of companies would have needed pollution permits and applying that to the companies that existed during 1983-85, I can describe the industrial stationary sources of pollution during that time. Additionally, there are other stationary sources of air and water pollution for which I have locational information. Apart from industrial stationary sources of air pollution, there are also power plants and mines. Mines also contribute to water contamination as do farms using pesticides. From the Philippine Department of Energy I have obtained the locations of coal-burning power plants in Cebu.[10] I have also obtained GIS data regarding land use during the 1980s from the Provincial Planning and Development Office. Furthermore, I have obtained information regarding the locations of large and small mines in Cebu from the Mines and Geosciences Bureau (MGS) and the Provincial Mines Office (PMO). By combining the locations of stationary air polluters with wind data the exposure of individuals within particular barangay can be described.

Similarly, using the locations of stationary water polluters obtained via the sources described above I can describe the exposure of individuals to waterborne contaminants. However, instead of using wind patterns to combine with the locations of polluters, I will use the piped water network and the watershed divisions in Metro Cebu.[11] The need to use both the network of pipes and the water arises because not all households in the survey obtain their water via pipes. First, I will describe the water quality occurring in piped water. LeChevallier et al (2003) shows that when water pressure within the pipes varies, as is the case with the consistently interrupted service of Metro Cebu (ADB 2005), seepages from the surrounding soil enter the pipes and contaminate the water. Therefore, water polluters - as identified using the EMB and phonebook data, as well as the MGS and PMO mine data - located between the water treatment plant and

[10] Coal-burning was the only type of plant operating in Cebu during the 1980s

[11] The US EPA defines a watershed as "the area of land where all of the water that is under it or drains off of it goes into the same place." See http://water.epa.gov/type/watersheds/whatis.cfm for further information.

the household cause contamination in piped water. The amount of seepages will also be a function of the soil types - some more permeable than others - where the polluter is located, thus I will also employ GIS data on soil types provided by the Provincial Planning and Development Office (PPDO). As with the variation in air quality, because the sources of contamination are not immediately proximate to the household, being located anywhere along the path from the treatment plant to the tap, they are unlikely to be correlated with the residential decision. Because the exact residential locations of survey respondents is unavailable, the measure of piped water quality will be an average of the variation in each of the pipes serving the barangay. I have obtained from the Metro Cebu Water District maps of the pipelines as well as information regarding when each segment of the pipes were laid. The figure below depicts the pipes throughout Metro Cebu.[12]

For non-piped water users, variation in their water quality is due to the location of water polluters as well as soil type and elevation. Therefore, in addition to the locations of water polluters from the EMB/phonebook and MGS/PMO mine data, I also employ GIS data on watersheds, soil type and elevation from the PPDO. A watershed is defined as an area of land where all the water drains into the same place. The following figure displays both the watershed divisions and barangay divisions in Metro Cebu, the barangays of the sample are highlighted.

Households are exposed to the pollution from sources located at higher elevations within the same watershed, and the amount of pollution can be scaled by the permeability of the soil. Therefore, within a watershed all contamination originating from higher elevations should affect non-piped sources of water, but sources of contamination located within different watersheds will have no effect.

The following table summarizes the various measures of air and water quality that will be employed in this study. Cunha, Heckman and Schennach (2010) show that in order to identify the latent variable the number of measures must be greater than or equal to 2. As the number of measures grow, so too does the efficiency of estimating the latent variable.

Measures of Exposure of Air and Water Contamination

Air		Water	
Measure	Source	Measure	Source
Road Density and Wind	PPDO, NCDC	Pipelines, soil and Industrial Plants	MCWD, PPDO, EMB, DPC
Power Plants and Wind	DOE, NCDC	Pipelines, soil and mines	MCWD, PPDO, MGS, PMO
Industrial Plants and Wind	EMB, DPC, NCDC	Pipelines, soil and farms	MCWD, PPDO
Mines and Wind	MGS, PMO, NCDC	Watershed, soil and Industrial Plants	PPDO, EMB, DPC
		Watershed, soil and mines	PPDO, MGS, PMO
		Watershed, soil and farms	PPDO

[12]First, note that this figure depicts the current state of the piped water network as I have not yet been able to employ the information on when piped began to operate to describe the network in the early 1980s. Secondly, note that all of the sample barangay are served by the Metro Cebu Water District except those within Naga City. Piped water was not available during the years 1983-85 in Naga City (today a municipal utility distributes piped water) and each respondent in Naga City reports using non-piped sources of water.

5 Preliminary Results

The results presented here are preliminary and additional results are shortly forthcoming. Furthermore, the results presented here do not employ the methodology discussed above, rather they are basic reduced form relations between exposure and health outcomes. The equation of interest here is:

$$\theta_{ij} = \alpha + \beta_1 EXP_{ij} + \gamma X_{ij} + \epsilon_{ij}$$

EXP denotes the exposure. In these preliminary results the exposure occurs during the entire period of pregnancy. Various exposures measures are shown below. For instance, one exposure measure is the exposure to industrial polluters, the number of polluters in each 10 degree bearing from the individual's sample barangay weighted by their distance and the proportion of the individuals gestation subject to each wind bearing. The same basic idea holds for the remaining exposure measures: the presence of industrial and transportational city zoning weighted by distance and wind bearing, the presence of mines weighted by distance and wind bearing. Slightly different is the measure of mobile source air pollution exposure. Employing a gravity model based on the population density of each barangay throughout Metro Cebu, traffic flows are calculated. Then, after calculating the distance and bearing between each sample barangay and all barangays of the metro area, traffic flow is weighted by distance and wind bearing. All of these measures as well as the health outcomes are standardized. Included in X are reference group controls, income, gestational length and residence outside of the barangay during pregnancy.

Total Exposure Weighted by distance and Wind Direction to Industrial Polluters

	1	2	3	4
Dependent Variable: Birth Weight				
Minor Industrial Polluters	-0.08*	-0.09*	-0.09*	-0.09*
	(0.04)	(0.04)	(0.04)	(0.04)
Major Industrial Polluters	0.05***	0.05***	0.05***	0.05***
	(0.00)	(0.01)	(0.00)	(0.00)
Observations	3072	3,072	3,072	3,072
R-squared	0.01	0.09	0.09	0.09
Dependent Variable: Birth Length				
Minor Industrial Polluters	-0.01	-0.02	-0.02	-0.02
	(0.03)	(0.03)	(0.03)	(0.03)
Major Industrial Polluters	0.02**	0.02***	0.02***	0.02***
	(0.01)	(0.00)	(0.01)	(0.01)
Observations	3109	3109	3109	3109
R-squared	0.01	0.07	0.07	0.07
Dependent Variable: Low Birth Wght Binary				
Minor Industrial Polluters	0.01	0.01	0.01	0.01
	(0.01)	(0.01)	(0.01)	(0.01)
Major Industrial Polluters	-0.00	-0.00	-0.00	-0.00
	(0.00)	(0.00)	(0.00)	(0.00)
Observations	3109	3109	3109	3109
R-squared	0.01	0.07	0.07	0.07
Additional Controls:				
Gender	Yes	Yes	Yes	Yes
Location	Yes	Yes	Yes	Yes
Gestation Length	No	Yes	Yes	Yes
Part of Pregnancy Outside Barangay	No	Yes	Yes	Yes
Per Capita Income	No	No	Yes	No
Wealth and Housing	No	No	No	Yes

Standard errors clustered at the city urban/rural level in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Total Exposure Weighted by distance and Wind Direction to Mines				
	1	2	3	4
Dependent Variable: Birth Weight				
Clay Mines	-0.01	-0.03	-0.02	-0.02
	(0.04)	(0.05)	(0.05)	(0.05)
Coal Mines	0.03	0.04*	0.05**	0.05**
	(0.02)	(0.02)	(0.02)	(0.02)
Copper Mines	-0.06	-0.05	-0.04	-0.04
	(0.05)	(0.05)	(0.05)	(0.05)
Gold Mines	0.00	0.00	-0.00	-0.00
	(0.01)	(0.01)	(0.01)	(0.01)
Silver Mines	0.04**	0.04***	0.04***	0.04***
	(0.01)	(0.01)	(0.01)	(0.01)
Observations	3072	3,072	3,072	3,072
R-squared	0.01	0.08	0.09	0.10
Dependent Variable: Birth Length				
Clay Mines	-0.02	-0.03	-0.03	-0.03
	(0.04)	(0.05)	(0.05)	(0.04)
Coal Mines	0.05	0.06*	0.06*	0.07*
	(0.03)	(0.03)	(0.03)	(0.03)
Copper Mines	-0.05	-0.04	-0.04	-0.04
	(0.03)	(0.03)	(0.03)	(0.03)
Gold Mines	0.02	0.02	0.02	0.02
	(0.02)	(0.02)	(0.02)	(0.02)
Silver Mines	0.04	0.04	0.04	0.04
	(0.03)	(0.02)	(0.02)	(0.02)
Observations	3109	3109	3109	3109
R-squared	0.01	0.07	0.07	0.07
Dependent Variable: Low Birth Wght Binary				
Clay Mines	0.00	0.01	0.01	0.01
	(0.01)	(0.01)	(0.01)	(0.01)
Coal Mines	-0.01***	-0.01***	-0.01***	-0.01***
	(0.00)	(0.00)	(0.00)	(0.00)
Copper Mines	0.01	0.00	0.00	0.00
	(0.02)	(0.02)	(0.02)	(0.02)
Gold Mines	-0.00	-0.00	-0.00	-0.00
	(0.00)	(0.00)	(0.00)	(0.00)
Silver Mines	-0.01	-0.01*	-0.01*	-0.01*
	(0.01)	(0.00)	(0.00)	(0.00)
Observations	3109	3109	3109	3109
R-squared	0.01	0.07	0.07	0.07
Additional Controls:				
Gender	Yes	Yes	Yes	Yes
Location	Yes	Yes	Yes	Yes
Gestation Length	No	Yes	Yes	Yes
Part of Pregnancy Outside Barangay	No	Yes	Yes	Yes
Per Capita Income	No	No	Yes	No
Wealth and Housing	No	No	No	Yes

Standard errors clustered at the city urban/rural level in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Total Exposure Weighted by distance and Wind Direction to Zoning				
	1	2	3	4
Dependent Variable: Birth Weight				
Agricultural/Agro-Industrial Zones	-0.02	-0.01	-0.01	-0.01
	(0.04)	(0.04)	(0.04)	(0.04)
Commercial Zones	0.03	0.03	0.03	0.03
	(0.02)	(0.03)	(0.03)	(0.03)
Transportation Zones	-0.06	-0.06	-0.06	-0.06
	(0.05)	(0.05)	(0.04)	(0.04)
Industrial Zones	-0.02	-0.03*	-0.03*	-0.03*
	(0.01)	(0.01)	(0.02)	(0.01)
Observations	3072	3,072	3,072	3,072
R-squared	0.01	0.08	0.09	0.10
Dependent Variable: Birth Length				
Agricultural/Agro-Industrial Zones	0.10	0.11*	0.11**	0.11**
	(0.05)	(0.05)	(0.05)	(0.05)
Commercial Zones	0.03	0.03	0.02	0.02
	(0.02)	(0.02)	(0.02)	(0.02)
Transportation Zones	0.00	0.01	0.01	0.01
	(0.06)	(0.06)	(0.05)	(0.05)
Industrial Zones	-0.00	-0.01	-0.01	-0.01
	(0.04)	(0.04)	(0.03)	(0.03)
Observations	3051	3051	3051	3051
R-squared	0.03	0.07	0.08	0.09
Dependent Variable: Low Birth Wght Binary				
Agricultural/Agro-Industrial Zones	-0.00	-0.01	-0.01	-0.01
	(0.01)	(0.01)	(0.01)	(0.01)
Commercial Zones	-0.02**	-0.02***	-0.02***	-0.02***
	(0.01)	(0.00)	(0.00)	(0.00)
Transportation Zones	0.02	0.02	0.02	0.02
	(0.01)	(0.01)	(0.01)	(0.01)
Industrial Zones	0.00	0.00	0.00	0.00
	(0.01)	(0.01)	(0.01)	(0.01)
Observations	3109	3109	3109	3109
R-squared	0.01	0.07	0.07	0.07
Additional Controls:				
Gender	Yes	Yes	Yes	Yes
Location	Yes	Yes	Yes	Yes
Gestation Length	No	Yes	Yes	Yes
Part of Pregnancy Outside Barangay	No	Yes	Yes	Yes
Per Capita Income	No	No	Yes	No
Wealth and Housing	No	No	No	Yes

Standard errors clustered at the city urban/rural level in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Total Exposure Weighted by Distance, Wind Direction and Gestation to Population Density Based Traffic Flow

	1	2	3	4
Dependent Variable: Birth Weight				
Population Density based Traffic Flow	0.06***	0.06***	0.06***	0.06***
	(0.01)	(0.01)	(0.01)	(0.01)
Observations	3,072	3,072	3,072	3,072
R-squared	0.01	0.09	0.09	0.09
Dependent Variable: Birth Length				
Population Density based Traffic Flow	0.08**	0.08**	0.08***	0.07**
	(0.03)	(0.03)	(0.02)	(0.02)
Observations	3051	3051	3051	3051
R-squared	0.02	0.07	0.08	0.08
Dependent Variable: Low Birth Wght Binary				
Population Density based Traffic Flow	-0.00	-0.00	-0.00	-0.00
	(0.01)	(0.01)	(0.01)	(0.01)
Observations	3109	3109	3109	3109
R-squared	0.01	0.07	0.07	0.07
Additional Controls:				
Gender	Yes	Yes	Yes	Yes
Location	Yes	Yes	Yes	Yes
Gestation Length	No	Yes	Yes	Yes
Part of Pregnancy Outside Barangay	No	Yes	Yes	Yes
Per Capita Income	No	No	Yes	No
Wealth and Housing	No	No	No	Yes

Standard errors clustered at the city urban/rural level in parentheses
*** p<0.01, ** p<0.05, * p<0.1

The interpretation of these results is at the moment unclear. For the most part, the results are insignificant indicating a lack of effect of air quality on birth health. The few significant results appear to exert counter intuitive effects on birth health. It is for this reason, as well as the previously explained intuition of toxicology and the need to examine long term effects, that additional results incorporating additional measures to generate the latent air quality, as well as the latent water quality, be used.

6 Issues and Discussion

An important flaw in the CLHNS data is the high level of attrition. A portion of the attrition is due to mortality, some of which is known, but some of it is simply due to respondent relocation. This affects the representativeness of the sample and is a source of selection bias due to the potential for households to relocate because of the health of the members of the household or their preferences toward exposure to environmental contaminants. There is some reasons to believe than in the a developing country context with

low income levels the potential for potential for relocation to avoid poor air and water quality is limited.[13] There is little that can be done to remedy this problem, however there are a few different ways in which the magnitude of the problem can be described. First, some of the attrition is temporary, the respondents disappear from the survey for a wave or more and then reappear. When they reappear there is information about where they while absent from the survey, and if this location is within Metro Cebu information conceding the pollution levels within this area are available. To the extent that these attritors are similar to others this information can be informative regarding the relocation of individuals, particularly whether they relocate to areas of different contamination levels. However, temporary attritors may be different from permanent attritors. Two comparisons can be made in order to describe permanent attritors, comparing exposure levels and health. Comparing the differences in observed exposure between permanent attritors before they left the sample and the remainder of the sample will perhaps provide some insight into the role of pollution in the household's relocation decision. Next, comparing available health information of permanent attritors before they left the sample with health information of the remaining sample can perhaps address whether health concerns were a motivation in the attrition. The following table is a preliminary attempt at describing how attritors differ from non-attritors using various measures of infant health.

Health Outcomes and Attrition Correlations

	1	2	3	4	5
	bth_weight	bth_height	gestation	skinfold	conception
Died during survey	-170.10***	-0.69***	-4.75***	-0.58**	-7.53
	(32.19)	(0.15)	(1.47)	(0.26)	(6.44)
Temporary Attrition	-21.53	0.15	-2.68**	0.03	-24.03***
	(27.60)	(0.12)	(1.32)	(0.23)	(5.80)
Permanent Attrition	-26.39	0.07	1.12	0.18	-12.79***
	(22.62)	(0.10)	(1.09)	(0.19)	(4.77)
Temporary then Permanent Attrition	-39.68	-0.08	1.25	0.13	-11.23
	(35.04)	(0.16)	(1.68)	(0.30)	(7.35)
Constant	3,034.88***	49.27***	267.67***	12.79***	8,428.46***
	(11.65)	(0.05)	(0.56)	(0.10)	(2.45)
Observations	3,086	3,051	3,098	3,120	3,098
R-squared	0.01	0.01	0.01	0.00	0.01

Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

The omitted group are those that alive and in the survey. From these results, it does not appear that, apart from those that died, attritors differ greatly from non-attritors in these measures. The one area where they appear to differ is in conception date, those that temporarily or permanently attrit appear to have earlier conception dates.[14]

Another potential issue is where these pregnant women spent their pregnancies, some of which may not have spent their pregnancies in Metro Cebu where they were surveyed. The following table summarizes the prior locations of women who spent a portion of their pregnancy before the baseline interview outside of their baseline barangay.

[13] Anecdotal evidence collected from acquaintances in Metro Cebu indicates that relocation is rarely driven by environmental concerns, particularly during the time period of the 1980s. First of all, these environmental concerns have only recently come to the public consciousness. Second, because of the generally low levels of income both in the survey and in Metro Cebu in general, most relocations are economically driven as individuals transition through the labor force.

[14] The displayed measure is the lower bound for conception. The results are the same when using the upper bound for conception.

Prior Residences of Survey Respondents	
Previous Residence (n=3121):	
Urban Metro Cebu	3.94
	(19.46)
Rural Metro Cebu	0.8
	(8.92)
Other City in Cebu Province	0.13
	(3.58)
Poblacion in Cebu Province	0.54
	(7.36)
Rural Cebu	0.8
	(8.92)
Metro Manila	0.16
	(4.00)
Other Province	0.64
	(7.98)
Poblacion in Other Province	0.48
	(6.92)
Rural in Other Province	0.9
	(9.43)
Lived Outside Barangay during Pregancy:	
Upper Bound %	5.99
Lower Bound%	5.61
Gestation Days (Upper Bound, n=187)	109.49
	(66.06)
Gestation Days (Lower Bound, n=175)	100.82
	(62.31)

There is no information within the CLHNS regarding where the woman spent the later portions of the pregnancy, after being interviewed at baseline and before giving birth when location is again documented. However, there is no indication from anecdotal sources that women spend the latter portions of their pregnancies with their parents or in some other location, in fact anecdotal evidence suggests that, if anything, parents move in with their pregnant daughters.[15]

6.1 Current Progress

At this point all the data has been collected, however much of it still requires cleaning. Therefore, the main area of progress is in preparing the data for use in estimation. However, I am also still attempting to refine the model and any comments in that regard are extremely appreciated.

7 Appendix: Model and Methodology

7.1 The Utility Framework

Human health involves interactions among multiple scales of biological organization that are affected by various environmental factors **?**. Standard models of health have abstracted from biological organization and environmental factors. The seminal model of health from Grossman (1972) describes health as both a produced and a consumed good, a purely economic view of health. The model defines health to depreciate over time with investments made to remediate the depreciation. More recently, Almond and Currie (2011) proposed (yet do not employ) an application of the Heckman (2007) skill formation model to the fetal

[15]Conversation with Dr. Alan Feranil and other members of the Office of Population Studies.

origins literature. The suggested model allows early childhood inputs to exert large effects over the life course in contrast to the Grossman model which builds in a fading effect to inputs as time passes. The model I present builds on these models by allowing health to be both produced and consumed, and letting early childhood inputs exert the large effect they have been observed to have. Take as a starting point the statement in Grossman (1972) that, "most students of medical economics have long realized that what consumers demand when they purchase medical services are not these services per se but, rather, 'good health.'" 'Good health' is perceived - and is likely a very idiosyncratic perception - and it is that perception that enters into the consumer's utility function. Denote the perception of health as $H_{t,s}$ for time period t and stage s, and begin with the same basic functional form for utility:

$$U = U(H_{t,s}, X_{t,s}) \tag{12}$$

Regardless of how an individual perceives his or her health, true health is multidimensional. The multidimensionality of health is difficult to address though Heckman (2007b) gives an insight into how it might be accomplished. The author does not address the multidimensionality of health in particular, rather the multidimensionality of human capital. The framework presented separates the dimensions of health, cognitive and non-cognitive skills from the aggregate of human capital and allows for an investigation of the interactions between the various dimensions of human capital. The model I present separates health into multiple dimensions by organs. The intuition is that how an individual perceives health is a function of how the organs of the body work together. Perception is therefore modeled as some unspecified function of the health of the various components of the body, or the organs. N denotes the number of organs in the body, $h_{t,s}$ denotes the set of all organs and $\theta_{n,t,s}$ denotes the health of the particular organ $n \in \{1, 2, ..., N\}$:

$$H_{t,s} = H_{t,s}(h_{t,s}) \text{ s.t. } h_{t,s} = \{\theta_{1,t,s}, \theta_{2,t,s}, ..., \theta_{N,t,s}\} \tag{13}$$

The next question to address is how is the health of the organs determined. To address that, consider that the human body is a system that constantly accepts inputs which are used in productive processes serving numerous functions. For example, consider the productive input air. Oxygen, or O_2, is the necessary component of the air breathed by aerobic organisms. Entering through the mouth or the nose, oxygen reaches the lungs and is transferred to the blood. There it binds with hemoglobin and is pumped through the heart to eventually arrive in the tissue fluid of an organ. There cells collect the oxygen by diffusion, at the same time losing carbon dioxide. Within the cell the oxygen is transferred to the mitochondrion and is then chemically reacted with glucose, $C_6H_{12}O_6$, to produce carbon dioxide, water and adenosine triphosphate, or ATP. The key result of this reaction is ATP. The carbon dioxide is waste product released from the cell into the blood, returned to the heart and the lungs, and finally exhaled into the air. ATP is energy which, after being produced through the above process known as cellular respiration, is then used in various processes within the body. I will model the body as a system of productive processes, such as cellular respiration. I will denote the basic primary inputs for the body's productive processes as $a_{t,s}$ for air, $w_{t,s}$ for water and $i_{t,s}$ for all other inputs, including food and medicine or potentially harmful substances, where s denotes the stage of development. However, not all of the air inhaled and the water drunk are used in the body's productive processes. There are many elements in the air we breathe, the water we drink and the other inputs we consume, but only a small fraction is needed by the body, as illustrated with

the example of oxygen. I will denote the components necessary to the body's productive processes as $\tau^q_{t,s}$, where q is defined as either a for air, w for water, and i for all other inputs, or $q \in \{a, w, i\}$. The functional form will be left unspecified but can be thought of as a fraction.

$$\tau^q_{t,s} = \tau^q_{t,s}(q_{t,s})$$

Furthermore, let b_j denote the body's productive process j, a certain number of these processes accepting only primary inputs and others accepting both primary inputs and secondary inputs resulting from other productive processes.

$$b_j = b_j(\tau^a_{t,s}, \tau^w_{t,s}, \tau^i_{t,s}, \psi) \text{ for } j \in \{1, 2, ..., J\} \tag{14}$$

$$b_k = b_k(\tau^a_{t,s}, \tau^w_{t,s}, \tau^i_{t,s}, b_j, \psi) \text{ for } k \in \{J+1, J+2, ..., \mathcal{J}\} \text{ and } k \neq j \tag{15}$$

The health of a particular organ does not necessarily depend on all of the productive processes of the body, rather a subset. Let subset of productive processes particular to organ n be denoted as B_n, such that each B_n contains any number of b_j produced by the body.

$$B = \{B_1, B_2, ..., B_N\} \tag{16}$$

The parameter ψ denotes the genetic component of the productive process. It is left unspecified how this component enters into the productive processes b_j, a lack of specificity justified by recent research rendering the distinction between nature and nurture obsolete. It is clear that individual genetic variation plays a significant role in all human capital outcomes, including health. However, recent literature emphasizes epigenetic expression or the interaction between genetics and the environment, meaning that traditional additive nature and nurture models mischaracterize gene-environment interactions **?**. The genetic code for an individual can be thought of as a set of initial conditions for the dynamic biological system of the body that evolves under the influence of environmental conditions **?**.

The inputs each have demands dictated by the body, such as the need to breathe, thirst and hunger. However, these are survival demands or needs and differ drastically from common demand functions for production inputs in economics.[16] As mentioned, not all of the air, water and other inputs are necessary for the body's productive processes. The necessary components have been denoted with $\tau^q_{t,s}$. Some of the non-essential inputs are immediately expelled, like many of the elements of air that are immediately exhaled. However, some of the non-essential inputs stay in the body, are absorbed and distributed until reaching a specific tissue site where they are deposited and accumulate. The amount of the contaminant that reaches the cells, membranes, tissues or organs were adverse effects occur is called in the toxicology literature the biologically effective dose (Paustenbach 2001). The biologically effective dose will be denoted with $\sigma^q_{n,t,s}$ where $q \in \{a, w, i\}$.

The health of the organs of the body can now be defined as functions of the body's processes B_n, genetics

[16] First of all, while optimal quantities of inputs to traditional production functions are found by the firm's optimization process (i.e. the maximization of profits), it is unclear that there exists a general optimization process which applies to all of the body's productive processes. Also, in traditional production functions with multiple inputs, as long as each of the inputs is greater than zero a deficit in one of the inputs can be compensated with higher levels of the other; this is obviously not the case with the body's productive processes. Furthermore, traditional production functions are monotonically increasing in their inputs while the body's productive processes cannot accept continually increases levels of inputs.

ψ, and the biologically effective dose of environmental contaminants $\sigma^q_{n,t,s}$. Let the health of the organ n be denoted by $\theta_{n,t,s}$, a result of the health production function $f_{n,t,s}(\cdot)$. Additionally, recalling that t denotes the time period and s denotes the stage of development, I will impose an intertemporal framework on the production of the health of organ n. Inputs are accepted and production commenced during time period t and stage s, and the resulting health of the organ is observed in period $t' > t$. However, because the stages of development contain multiple time periods s' is not strictly greater than s.

$$\theta_{n,t',s'} = f_{n,t,s}(B_n, \sigma^q_{n,t,s}, \psi) \text{ for } n \in \{1, 2, ..., N\} \text{ and } t' > t \text{ and } s' \geq s \tag{17}$$

7.2 Air and Water Quality

The quantity of air, water and food, if insufficient, will lead to death in a short amount of time, however the impacts of the quality of the inputs are not always observed so quickly. An underlying axiom of toxicology is that when normal bodily function and processes are sufficiently perturbed, a response will be elicited. Sufficient toxic perturbation leads to adverse response (Sheldon and Cohen Hubal 2009, Paustenbach 2001). This perturbation is the biologically effective dose, or the amount of the contaminant that reaches the cell, membrane, tissue or organ where it can elicit an effect. The biologically effective dose has been denoted $\sigma^q_{n,t,s}$ where $q \in \{a, w, i\}$ and $n \in \{1, 2, ..., N\}$. $\sigma^q_{n,t,s}$ will be described as an unspecified function of the primary input, i.e. air a, water w, and all others i. For instance, if $q = a$ then,

$$\sigma^a_{n,t,s} = \sigma^a_{n,t,s}(a_{t,s}) \text{ for } n \in \{1, 2, ..., N\}$$

The biologically effective dose is the empirical gold standard of toxicological dose response studies. It is a standard that is rarely, if ever, met. Currently, the best approach to estimating the biologically effective dose is by using toxicokinetic models. These models are estimated using blood samples and calibrated using previously estimated parameters of absorption, distribution and elimination particular to an organ and a toxin.[17] Without measured levels of toxins in the blood, I will attempt to measure the presence of environmental contaminants within the body's system in general or,

$$\sigma^q_{t,s} = \sum_{n=1}^{N} \sigma^q_{n,t,s} \text{ for } q \in \{a, w, i\} \tag{18}$$

This will alter equation (6) by making the health of the organ a function of the general presence of contaminants within the body, $\sigma^q_{t,s}$.

$$\theta_{n,t',s'} = f_{n,t,s}(B_n, \sigma^q_{t,s}, \psi) \text{ for } n \in \{1, 2, ..., N\} \text{ and } t' > t \text{ and } s' \geq s \tag{19}$$

Recall that the necessary elements for the body's productive processes are denoted by $\tau^q_{t,s}$. They have also been described as unspecified functions of the primary inputs air, water and all others. Note that it is not the case that $\tau^q_{t,s} + \sigma^q_{t,s} = q_{t,s}$. This is for two reasons. First, the body has processes and organs which eliminate contaminants from its system, such as the process of exhaling and the kidney and liver organs. Because of this not all of the contaminants which enter the body will eventually reach the cells,

[17] Further precision can be achieved with urine and stool samples that measure the elimination of toxins from the system. Even with samples of blood, urine and stool there would be concerns of endogeneity that would be difficult to resolve.

membranes and tissues where they would exert an adverse effect. Much of the air breathed in will be immediately exhaled and have no effect on the body. And a portion of the contaminants which enter the body's system via water are expelled before they become part of the biologically effective dose of a particular organ. And the second reason is that some of the elements which enter the body are neither beneficial nor detrimental. The presence of innocuous elements and the expulsion of contaminants before they reach target cells, membranes and tissues result in the following: $\tau^q_{t,s} + \sigma^q_{t,s} \neq q_{t,s}$.

With the intent of finding a measure of $\sigma^q_{t,s}$, the endogeneity of this measures is a consistent concern in studies regarding the health impacts of the environment. Because of the capitalization of environmental quality in housing prices, exposure determined at some geographic area is likely endogenous to residential sorting and accompanying unobserved heterogeneity. Additionally, because dose actually effects the change within the body and not exposure, omitted and unobservable compensating behaviors are likely sources of endogeneity. However, beyond these concerns there is the issue that the available measures of air or water quality are imperfect, noisy descriptions of the individual's exposure to environmental contamination. I address this issue by employing various measures of air and water quality, each arguably exogenous (see section 3.2.1 for a detailed explanation of the measures employed), but each undoubtedly imperfect and noisy. These multiple measures are used to identify the distribution of the latent air and water quality using the Cunha, Heckman and Schennach (2010) framework.

Let m denote the measure and M^q the total number of measures: $m \in \{1, 2, ..., M^q\}$ for $q \in \{a, w\}$. Let $\sigma^{q*}_{t,s}$ denote the latent quality of the input, air or water, consumed in period t during the developmental stage s. And let $\sigma^q_{t,s,m}$ denote the mth measure of input quality. Adopting a linear form for the relationship between the noisy measure and the latent variable, let $\mu^q_{t,s,m}$ be the mean function and $\epsilon^q_{t,s,m}$ the error term. The coefficients $\gamma^q_{t,s,m}$ are the factor loadings.

$$\sigma^q_{t,s,m} = \mu^q_{t,s,m} + \gamma^q_{t,s,m}\sigma^{q*}_{t,s} + \epsilon^q_{t,s,m} \tag{20}$$

Assume that $E(\epsilon^q_{t,s,m}) = 0$. Normalizing $E(\sigma^{q*}_{t,s}) = 0$ for all t, given measurements $\sigma^q_{t,s,m}$ the mean functions are identified as the means of the measures $\sigma^q_{t,s,m}$. The factor loadings $\gamma^q_{t,s,m}$ remain to be identified. One more normalization, $\gamma^q_{t,s,1} = 1$, is required. Therefore, computing the covariances of the measures the factor loadings are identified:

$$Cov(\sigma^q_{t,s,1}, \sigma^q_{t+1,s,1}) = Cov(\sigma^{q*}_{t,s}, \sigma^{q*}_{t+1,s}) \tag{21}$$

$$Cov(\sigma^q_{t,s,2}, \sigma^q_{t+1,s,1}) = \gamma^q_{t,s,2} Cov(\sigma^{q*}_{t,s}, \sigma^{q*}_{t+1,s}) \tag{22}$$

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$$Cov(\sigma^q_{t,s,M^q}, \sigma^q_{t+1,s,1}) = \gamma^q_{t,s,M^q} Cov(\sigma^{q*}_{t,s}, \sigma^{q*}_{t+1,s}) \tag{23}$$

The factor loadings $\{1, \gamma^q_{t,s,2}, \gamma^q_{t,s,3}, ..., \gamma^q_{t,s,M^q}\}$ are therefore identified by the ratio of covariances, such as dividing equations (11) and (12) by equation (10).

$$\frac{Cov(\sigma^q_{t,s,m}, \sigma^q_{t+1,s,1})}{Cov(\sigma^q_{t,s,1}, \sigma^q_{t+1,s,1})} = \gamma^q_{t,s,m} \tag{24}$$

Having identified the factor loadings, $\gamma^q_{t,m}$, the measures of latent input quality can be identified for each t.

$$\frac{\sigma^q_{t,s,m}}{\gamma^q_{t,s,m}} = \frac{\mu^q_{t,s,m}}{\gamma^q_{t,s,m}} + \sigma^{q*}_{t,s} + \frac{\epsilon^q_{t,s,m}}{\gamma^q_{t,s,m}} \tag{25}$$

Collecting the measures of latent input quality for all t, the distribution of input quality can be identified.

$$\sigma^{q*} = \left(\{\sigma^{q*}_{t,s}\}^T_{t=1} \right) \text{ for } q \in \{a, w\} \tag{26}$$

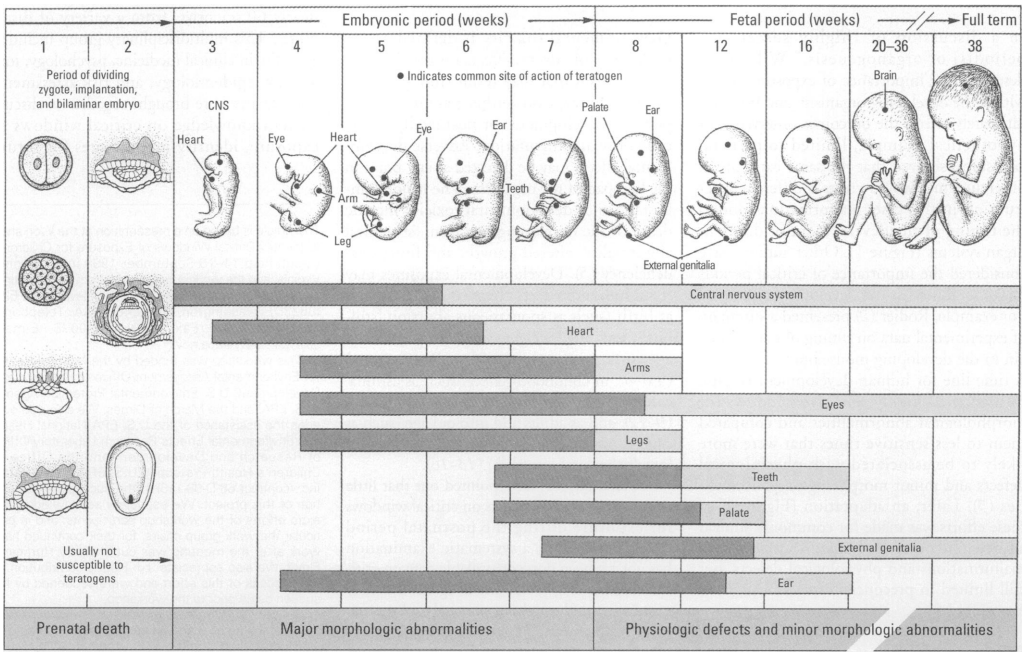
Of more particular interest in this study is the input quality during the critical periods of development. How exactly to relate exposure to health is an open question. Is it the integral under the exposure-duration curve or the integral under the curve but above some threshold level of exposure, a level which the body can handle and eliminate but above which the toxins accumulate and exert and effect? And what is the appropriate threshold? This study will not address the issue of thresholds and manageable levels of exposure, rather the total integral under the exposure-duration curve will be examined for its relation to health. Furthermore, because the measures of exposure are weighted by duration, the relevant variable is the sum of exposures during the stage of development.

$$\sigma^{q*}_s = \sum_{t \in s} \sigma^{q*}_{t,s} \text{ for } q \in \{a, w\} \tag{27}$$

7.3 Critical Periods of Development

The stage of development, s, is critical for this analysis. Note that if $t \in \{1, 2, ..., T\}$ is the time period then each $s \in \{1, 2, ..., S\}$ is a subset of the time periods. Some stages in life are more important for health than others. Medical science has identified various critical stages of development that occur during pregnancy and early in the life of a newborn child **??**. During the early period of organ development, the basic structures are formed. The rapid and diverse nature of processes that form the basic structures during the early stages of development there is an increased vulnerability to environmental contaminants. Perturbations of normal functions during the earliest stages of development can result in major disruptions such as fetal death and congenital abnormalities **?**. As development progresses, disruptions are more likely to result in impaired growth, physiological defects, functional deficiencies and pregnancy complications (Bodgen 1995, Hewitt 1998, Wergeland 1997[18]). Prenatal development is generally divided into three periods: periconceptual, embryonic and fetal. The periconceptual period is the first two weeks following conception, and exposure to significant levels of contaminants during this period typically lead to unobservable prenatal death. Weeks 3-7 are known as the embryonic period, during which most organs begin to develop. The fetal period is the last and longest, from week 8 until birth. The following figure is from Moore (1998) and shows these periods and the timing of the development of various organs.

[18]For a review of all of these studies see Altshuler 2003



As seen above, for each organ the critical stage of development is different. The heart develops during weeks 3 to 8, while the earliest stage of reproductive development begins in week 7 until week 12, however the reproductive system continues developing through the fetal period and the postnatal period. The periods of early development are the most vulnerable to exposure to environmental contaminants however exposures during later periods are still non-negligible. Therefore, a distinction will be made in the model between stages of development. As previously discussed, s denotes the stage of development and is a subset of the time periods of life $t \in \{1, 2, ..., T\}$.

In general, the development of the organs during pregnancy can be divided into an 'embryo' and a 'fetal' stage. After birth we will consider exposures during the 'postnatal' period. The goal of this study is to analyze the long term impacts of exposures to environmental contaminants during early stages of development. These long term impacts can be observed during the stages of 'child', 'adolescent' and 'adult'.

$$s \in \{embryo, fetal, postnatal, child, adolescent, adult\} \tag{28}$$

At this point t will be dropped from the notation and the focus will be on the stages of development. The nature of the study is to address the long term impacts of exposure to environmental contaminants in early life, therefore the outcomes of interest are: $\theta^n_{child}, \theta^n_{adolescent}, \theta^n_{adult}$.

7.4 Genetics

At this point let us further discuss ψ in $\theta_{n,t',s'} = f_{n,t,s}(B_n, \sigma^q_{n,t,s}, \psi)$. At best ψ will describe actual genetic information of the individual, genetic codes which have been identified to lead to certain health outcomes. However, empirical studies rarely contain this information. The second best approach involves utilizing parental information, though generally the actual genetics of the parents are generally not available. In this case, medical histories of the parents can be used. For instance, if n denotes the heart, then histories of

heart problems in the parents are relevant to the child's heart health. Exactly how relevant can be unclear because of the environmental impact on the expression of the genetic information. Because health outcomes result from the interaction of environmental inputs and genetics, medical histories contain information on both environment and genetics. Regardless, this information is often the best available approximation to genetics and that is why it is used in medical practice.

Therefore, ψ will be specified as parental health histories relative to organ n. Denote paternal health with regards to organ n as $\theta_{n,F}$, and maternal health with regards to organ n as $\theta_{n,M}$.

$$\psi = \{\theta_{n,F}, \theta_{n,M}\} \tag{29}$$

Further, consider that in the very first period of life the individual's health outcome is only a function of initial conditions or the genetic inheritance from the parents, $\theta_1^n = f_0^n(\theta_F^n, \theta_M^n)$. For all other periods in time, the health of an organ is a function of both genetics and subsets of the body's productive processes. Therefore, equation (8) can be rewritten as:

$$\theta_{n,t',s'} = f_{n,t,s}(B_n, \sigma_{t,s}^q, \theta_{n,F}, \theta_{n,M}) \text{ for } n \in \{1, 2, ..., N\} \text{ and } t' > t \text{ and } s' \geq s \tag{30}$$

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