

# **Are Environmental Toxins a Source of Health Shocks to Children?**

## **Evidence from Hispanic Children in the U.S.**

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### **Abstract**

Chronic diseases are among the most prevalent, costly and preventable of all health problems: representing three-quarter of the nation's \$1.4 trillion medical care costs. Childhood cognitive disorders have emerged as a leading contributor to education spending: special education spending alone accounts for 14 percent of the \$360 billion on elementary and secondary education. It is important to identify factors that influence the incidence of chronic childhood conditions. We examine whether exposure to environmental toxins, pesticides particularly, impacts children's cognitive and physical health. Using the Hispanic Health and Nutrition Examination Survey, we find that exposure to pesticides through farming or pesticide production significantly increases a child's chance of developing heart, respiratory and other chronic diseases. Furthermore, parental occupational exposure to pesticides significantly increases their children's chance of exhibiting cognitive disorders, respiratory diseases including asthma, heart diseases and other chronic diseases. The medical costs and the reduction in children's future earnings associated with poor cognitive and physical health amount to 30 percent of their future lifelong earnings.

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## **I. Introduction**

Chronic diseases are among the most prevalent, costly, and preventable of all health problems in the United States, accounting for three-quarters of the nation's \$1.4 trillion medical care costs. Childhood cognitive disorders have emerged as a leading contributor to education spending: special education spending alone accounts for 14% of the \$360 billion spent on elementary and secondary education. It is therefore important to identify factors that influence the incidence of chronic childhood conditions. We examine whether exposure to environmental toxins, pesticides in particular, impacts children's cognitive and physical health.

Although chronic low-level exposure to pesticides is prevalent in the entire U.S. population, the age group with the highest organophosphate pesticides exposures is children aged 11 and younger (reported by the National Health and Nutritional Examination Surveys in Hill et al. 1995, Stephenson 2003)<sup>1</sup>. Children of agricultural workers and pesticide applicators are subject to great levels of pesticide exposure from helping their parents on the farm, living on or near a farm, and living with their exposed parents who inadvertently bring pesticides home on their clothing, skin, and hair. Occupational pesticide exposure of one family member increases pesticide exposure of the whole family (Shealy et al. 1997 and Simcox et al.1999). Children of farming parents are more likely than children of non-farming parents to be exposed to doses that exceed the U.S. Environmental Protection Agency reference dose (Fenske et al. 2000 and Curl et al.2002).

Many published studies report on the increased child health hazard of parental pesticide exposures. Feychting et al. (2001) find there is an increased risk of nervous system tumors in children related to paternal occupational pesticides exposure after following a cohort study with 235,635 children from birth to age 14. Ma et al. (2002) find that exposure to household

pesticides is associated with an elevated risk of childhood leukemia. Guillette et al. (1998) find that exposed children living in a farming community with heavy use of pesticides exhibited remarkably impaired hand-eye coordination and short-term memory, difficulty in drawing, and more aggressive and anti-social behavior than the control children, who lived in a similar community with little use of pesticides. In a study of 210,723 live births in rural Minnesota between 1989 and 1992, Garry (1996 and 2002) finds pesticide applicators' children had significantly higher birth defect rates. Garry et al. (2002) find increased risks of birth defect or development disorders in farming families.

What have received little attention in the literature is that there is a link between pesticides exposures and the dysfunction of hormone and immune system, and that an impaired immunity would increase the susceptibility to the toxicity of pesticides on a host of health problems besides birth defects and cancers. Children may be particularly prone to the impact of pesticides on their immune system because they are still developing and their immune system is immature. Chronic childhood diseases are important health outcomes in examining the health consequence of pesticides on children because many chronic childhood diseases persist across lifespan and chronic diseases have become the leading contributor to U.S. health care costs.

In this paper, to advance our understanding of health effects of pesticides, we test the following hypothesis: do pesticides impact children's cognitive and physical health including chronic conditions? In particular, we examine two questions and make three contributions to the literature. In the first question, we focus on older children who may be directly exposed to pesticides through farming, pesticide manufacturing or other pesticide-related occupational settings; we examine whether directly exposed children more likely to develop chronic conditions or poor overall health than children who are not directly exposed. We make our first

methodological contribution in examining this question; we fully control for confounders originating from observed and unobserved family-related factors through the use of within-sibling estimation, that is, the difference in siblings' health outcome that is attributable to the difference in their exposure.

In the second question, we focus on younger children who may not be directly exposed to pesticides occupationally themselves but whose parents may be; we examine whether children whose parents are occupationally exposed to pesticides are more likely to develop cognitive disorders or childhood chronic diseases or poor overall health than children whose parents are not. We make our second methodological contribution in examining this question; we fully allow for complex and nonlinear interaction effects among the covariates through the use of matching analysis, that is, the difference in the health outcome between an indirectly exposed child and his/her unexposed pseudo-twin where a pseudo-twin is a child whose characteristics are identical or closest to those of the exposed child.

Our third methodological contribution is that we examine whether occupational pesticide exposure is a valid proxy for actual exposure to pesticide. We perform validity check by regressing a child's serum pesticide metabolites on whether he/she is occupationally exposed to pesticides, as well as on whether he/she is indirectly exposed through his/her parents' occupational exposure. We find evidence that occupational pesticide exposure is a valid proxy for actual direct and indirect exposure to pesticide.

We present our main results in Figure 1 and we use the Hispanic Health and Nutrition Examination Survey (HHANES) in testing our hypothesis. We use fixed-effect (or within-sibling difference) estimation to quantify the impact of occupational exposure to pesticide among older children (aged 12–17), as the survey contains data on whether they were occupationally exposed

themselves<sup>2</sup>. We find that occupational exposure to pesticides at late childhood increases the incidence of having poor overall health by 23.8% and increases their risk of developing a chronic heart-related disease by 26.1%, a chronic respiratory disease by 4.8%, and a chronic condition related to heart, lung, or kidney by 48.8%.

We use random-effect and matching analysis to quantify the impact of parental occupational pesticide exposure on younger children (aged 11 and younger), as the survey contains no data on their occupational exposure to pesticides. We find that parental occupational pesticide exposure increases their risk of exhibiting cognitive disorders by 5.7%—equivalent to the impact of having an additional 11 µg/dl (microgram per deciliter) of lead, and serum lead is known to increase childhood cognitive disorders. Living with occupationally exposed parents increases a child's chance of developing asthma and other chronic respiratory disease by 9.6%, a heart-related disease by 4.2%, a kidney disease by 4.0%, and a chronic condition related to heart, lung, or kidney by 25%. The estimates from matching analysis are very similar to those from random effect, indicating that our estimates are robust to complex interaction effect among all other covariates.

For children in both age groups, the economic costs of parental occupational exposure on these children, including medical costs and the children's future earnings, are approximately 30% of the children's lifelong earnings.

The next section defines the parameter of interest and describes our estimation strategies. Section III describes the data and provides descriptive analysis, Section IV presents our main findings, and Section V implements our robustness checks. We discuss the policy implications of our results in Section VI and conclusions in Section VII.

## II. Econometric Framework

This section discusses our framework for quantifying the health risks of pesticide exposure. In particular, we define the exposure variable and the parameter of interest, describe our identification strategies, discuss functional form issues for estimating the impact of pesticide exposure on these outcomes, discuss the etiology of health risks of pesticide exposure, justify the exposure and outcome variables used in the study, and discuss other econometric issues.

### A. Specifications

Let

$$(1) \quad h_{ij} = \alpha + p_{ij}^* \beta + X_i' \gamma + a_i + Z_{ij}' \pi + u_{ij}$$

where  $h_{ij}$  is the health outcome of child  $j$  in family  $i$ ,  $p_{ij}^*$  is pesticide exposure,  $X_i$  is a vector of family-specific observable determinants of health (e.g., race and parental education),  $a_i$  reflects the family-specific unobservable determinants of health (e.g., genetic factors and parenting),  $Z_{ij}$  is a vector of child-specific observable determinants of health (e.g., age and gender), and  $u_{ij}$  represents other child-specific idiosyncratic factors.

The variable of interest is  $p_{ij}^*$ , and parameter of interest is  $\beta$ . We address our questions to both direct exposure and indirect exposure. When  $p_{ij}$  measures a child's direct (indirect) exposure to pesticide,  $\beta$  quantifies the impact of direct (indirect) exposure. Furthermore, we do not observe a child's true exposure to pesticide,  $p_{ij}^*$ , and we observe a measure of pesticide exposure,  $p_{ij}$ , which contains a measurement error,  $e_{ij}$ :

$$(2) \quad p_{ij} = p_{ij}^* + e_{ij}$$

Substituting (2) into (1), we have an estimable equation:

$$(3) \quad h_{ij} = \alpha + p_{ij} \beta + X_i' \gamma + a_i + Z_{ij}' \pi + u_{ij} - \beta e_{ij}$$

## B. Identification Strategy

We use a family fixed effect in the regression in estimating  $\beta$  when we observe the child's own exposure to pesticides. Siblings in the family share the same parents<sup>3</sup>, so the inclusion of family fixed effect controls for all family-specific observable determinants of health, such as parents' education, age, race, family income, housing location and living environment, and parents' health behavior, as well as all other family-specific unobservable determinants of health, such as parents' preferences for their children's health. The genetic factors are controlled for to the extent that siblings share the same set of genetics, though the within-sibling genetic differences are not. The fixed effect estimate,  $\beta_{FE}$ , is unbiased only when the within-sibling differences in observable factors ( $W_{ij}$ ), unobservable factors ( $u_{ij}$ ), and measurement errors ( $e_{ij}$ ) are all uncorrelated with the differences in the child's pesticide exposure ( $p_{ij}$ ).

We use a family random effect in the regression in estimating  $\beta$  when we have no data on the child's own exposure to pesticides. The cross-sectional estimate of  $\beta$ ,  $\beta_{OLS}$ , can be confounded by observable factors such as parental education and child's age (elements in  $X_i$  and  $Z_{ij}$ ) that simultaneously influence child's pesticide exposure and health. We perform several robustness checks to assess the sensitivity of our results. To allow for complex and nonlinear interactions between all observable factors, we employ a matching estimator, described below.

## C. Matching Analysis and the Applicability of Matching Estimator

To allow for flexible functional form, we employ a linear matching estimator for a binary treatment or exposure variable in estimating (3)<sup>4</sup>. Let  $H$  denote a health outcome of interest. Pool

all children, and let  $k$  denote the  $k$ -th child in the sample of  $N$ . Let  $E_k$  denote whether child  $k$  was exposed. Let  $H_{k1}$  denotes the health outcome if child  $k$  was exposed, and  $H_{k0}$  denotes the health outcome if child  $k$  was not exposed. Let  $X$  denote a vector of characteristics or health determinants other than  $E_k$ . We are interested in the sample average treatment (or exposure) effect for the treated  $\tau_T$ , conditional on  $X$  where  $N_T$  is the number of exposed children:

$$(4) \quad \tau_T = \frac{1}{N_T} \sum_{k:E_k=1} (H_{k1} - H_{k0})$$

However, we cannot observe both  $H_{k1}$  and  $H_{k0}$  simultaneously for child  $k$  because this child was either exposed or not. A matching estimator imputes  $H_{k0}$  for exposed child  $k$  by finding some unexposed children whose characteristics are identical or closest to those of the exposed child  $k$ . The closeness is determined by a metric. Formally<sup>5</sup>, let  $\|x\|_V = (x'Vx)^{1/2}$  be the vector norm with positive definite matrix  $V$ . We define  $\|z-x\|_V$  as the distance between the vectors  $x$  and  $z$ . Let  $d_M(k)$  be the distance from the covariate value for child  $k$ ,  $X_k$ , to the  $M$ th nearest match<sup>6</sup>. Every match of  $X_k$  must have the opposite treatment than unit  $k$ ; if  $k$  is exposed, the matches are unexposed and vice versa. Let  $J_M(k)$  denote the set of indices for the matches for unit  $k$  that are as close as the  $M$ th match and let  $\#J_M(k)$  denote the number of elements in  $J_M(k)$ . A simple matching estimator calculates the sample average treatment effect for the treated children as follows:

$$(5) \quad \tau_M^t = \frac{1}{N_1} \sum_{k:E_k=1} \left( H_k - \frac{1}{\#J_M(k)} \sum_{l \in J_M(k)} H_l \right)$$

The identification in the matching analysis (Imbens 2003) is based upon two assumptions: conditional on covariates  $X$ , whether the child is treated is independent of the outcomes and the probability of being treated is bounded away from zero and one. The first assumption states that whether or not parents are occupationally exposed to pesticides is

independent of their children's health, and it is not testable empirically. We cannot rule out the possible correlation that parents who had not been occupationally exposed to pesticides consciously chose to not engage in farming or other pesticide-related activities because of the prior poor health of their children. Given that there is virtually no study on the adverse health effect of parental pesticide exposure to children, we think such a correlation is unlikely or small for this survey.

The second assumption requires an overlap in covariates between the exposed and the unexposed; this is testable by comparing the distributions of covariates. Figure 1 plots the distributions of four key characteristics variables: mother's education, father's education, log of per capita family income, and child's serum lead level. The distributions of these key variables exhibit similar shape and support, and have large overlaps within the support, though they may differ at the sample means.

#### D. Measures of Health Outcomes

We use two sets of health outcome variables ( $h_{ij}$ ) to characterize the impact of pesticide exposure on children's health: incidence of chronic childhood conditions and overall health status. First, we consider particular chronic conditions, such as the presence of respiratory symptoms, heart-related diseases, or thyroid-related conditions. The biomedical literature provides guidelines and justifications of these health outcomes. Low-level exposure to pesticides causes abnormal levels of thyroid, impaired production of antibodies to foreign protein, and other immune system dysfunctions (Porter et al. 1999). Some pesticides are found to apparently target the developing brain during the critical period of cell division (Chanda and Pope 1996, Eriksson 1996); others are found to be toxic to the immune systems of animals (Thomas 1995).

Some animal studies find that nicotine and chlopyrifos (an organochlorine pesticide) alter early brain development in a similar way (Moore 2003, Slotkin 1998 and 1999).

The advantage of using particular chronic condition as the outcome variable is that it is a stable and somewhat objective measure of severe health problems. Chronic conditions are often identified by health-care professionals and persistent for an extended period of time rather than an instantaneous event. The disadvantage, however, is that the diagnosis of chronic conditions is not uniform among all affected children due to various reasons, such as the lack of medical services access; thus this variable may under-measure the true health outcomes.

In addition to particular chronic conditions, we consider the presence and the number of chronic conditions of all types combined. We treat this separately because the development of related chronic conditions is often correlated: pesticide exposure is known to weaken the immune system, and a compromised immune system can have a profound and complex impact on a host of health issues.

Second, we consider overall health status as a five-scale categorical variable with value 1 = Excellent and 5 = Poor<sup>7</sup>. The advantage of this measure is that it takes into account severe health problems, such as chronic diseases, as well as moderate health problems not yet identified or treated by medical professionals. Many reported health risks of chronic pesticide exposure relate to immune system dysfunctions, which implicate a wide range of health issues. Based on the health status variable, we construct indicators of good health, defined by a health status of Excellent or Very Good, or whether poor health, defined by a health status of Fair or Poor.

#### E. Other Econometric Issues

Of special concern are three cases that would bias the estimate of  $\beta$  and the survey we use are lack of appropriate data to address these issues. The first case arises if the child's exposure, direct or indirect, were endogenous. For example, children with poor health may be less likely to help their parents on farms, which would lead to a downward bias. Conversely, sick children may be more likely to spend more time in the contaminated house or have greater contact with exposed parents, which subject them to a greater level of indirect exposure; this would cause an upward bias. Unfortunately, the HHNES survey data does not discriminate among such variations.

The second case arises when the family fixed effect varies among the siblings. It may be plausible that parents value their children's health differently depending upon the child's health, age, or other characteristics. For example, parents may have greater preferences for the younger child than for the older child or vice versa, or they may value the health of the sick child more than that of the healthy one. Unfortunately, the survey provides no additional information on such potential heterogeneity.

The third case arises when there are omitted variables. There may be unobserved child-specific individual heterogeneity, such as personality traits, that impact both health and pesticide exposure. For example, sick children may be more likely to have poor eating habits or hygiene than healthy children. Unfortunately, the survey provides no data on such information, so we assess the direction and magnitude of the omitted variable bias in the robustness checks.

### **III. Data and Descriptive Analysis**

This section describes the survey used and presents the summary statistics. We discuss several measures of pesticide exposure including the occupational and biomedical, and assess the

validity of parents' occupational pesticide exposure history as a proxy for children's chronic indirect exposure to pesticides. We end the section by summarizing the differences in characteristics between the exposed and unexposed children.

#### A. The Survey

We use the Hispanic Health and Nutrition Examination Survey (HHNES), conducted by the National Center for Health Statistics, a division of the Center for Disease Control and Prevention (CDC). This survey, conducted between 1982 and 1984, is a nationwide cross-section probability sample of approximately 16,000 individuals ranging in age from 6 months to 74 years. The goal of the survey was to collect data on the health and nutritional status of Hispanic groups in selected areas of the U.S. It is the only national survey that contains detailed pesticide exposure history coupled with serum pesticide metabolites, thus allowing for the construction of occupational exposure measures and assessment of whether occupational exposure measures are valid proxies for chronic exposures to pesticides. The serum pesticide metabolites data, collected in 1982–84, were released from CDC to the public in August 2003.

The Hispanics in this survey represent ethnicity groups from three regions: 1) Mexicans and Mexican-Americans residing in selected counties of Texas, Colorado, New Mexico, Arizona, and California; 2) Cubans and Cuban-Americans residing in Miami, Florida (Dade County); and 3) Puerto Ricans and Puerto Rican-Americans residing in the greater metropolitan area of New York, including parts of New Jersey and Connecticut. After discarding missing data, we have 2,692 usable observations of children under age 17<sup>8</sup>.

## B. Summary Statistics

The first column of Table 2 shows the summary statistics of health outcomes plus socioeconomic and health behavioral data for all the children. Half the children are in good health and 15% have one or more chronic conditions. The children are on average 8.8 years old, and their mothers and fathers completed an average of 5.6 and 4.0 years of schooling, respectively. Average per capita family income is \$3,704 in 1984 dollars. The vast majority of children have adequate nutrition based on physician assessment. Approximately 91% of the children have a place to go for routine checkups or emergency care, with the date of their most recent routine checkups approximately six months prior. The children have on average 10 microgram per deciliter ( $\mu\text{g}/\text{dl}$ ) lead in blood serum. Damages have been documented beginning at a blood lead concentration of 10  $\mu\text{g}/\text{dl}$ , and even under 5  $\mu\text{g}/\text{dl}$  (AAP 2003)<sup>9</sup>.

## C. Measures of Pesticide Exposure

There are two types of exposure estimates to pesticides: external and internal. The former measures the amount and types of pesticides in the environment that an individual is exposed to, and the latter, also referred as biomonitoring, measures the amount and types of pesticides that exist in an individual's body.

Broadly speaking, there are two categories of pesticides: organochlorine and organophosphate (and carbamate) pesticides. Organochlorine pesticides are effective against many insects. Fat-soluble, they concentrate in animal fats and are thus more concentrated higher on the food chain. They decay very slowly and are extremely persistent in the environment. Organophosphate pesticides decay more rapidly than organochlorine pesticides. As they are

mostly water soluble, they do not bio-accumulate up the food chain and are not persistent in the environment.

We first consider an external measure. We construct the pesticide exposure variables using the pesticide exposure history available for individuals aged 12 and older. The first indicator of pesticide exposure considers the child's exposure to pesticides through occupational settings, specifically those related to farming or pesticide production activities, including pesticide manufacturing, processing, and handling. It is set to 1 if the individual was engaged in farming or pesticide production and 0 otherwise. Among the 1,967 observations of the children in this age group, about 18% were occupationally exposed to pesticides. Let  $e_{ij}$  denote the occupational exposure by individual  $j$  in family  $i$ . This is the direct exposure indicator and used to estimate the impact on older children.

The second indicator, denoted as  $E_i$ , applies to the household and is set to 1 if anyone in household  $i$  is directly exposed in occupational settings and 0 otherwise. This is the indirect exposure indicator and is used to estimate the impact on younger children (aged 11 and younger), who were not asked in the survey about their direct occupational exposure. Among the 4,046 observations of children under age 12, about 21% had parents exposed to pesticides through farming (but not pesticide production), 12% had parents exposed to pesticides through pesticide production activities (but not farming), and 7.3% had parents exposed to pesticides through both farming and pesticide production activities.

There are two kinds of internal measures of pesticide exposures: blood serum and urine. The pesticide metabolites detected in blood serum measure the persistent organochlorine pesticide residues or metabolites. The level of organochlorine pesticide metabolites in blood serum represents chronic and/or recent exposure and is likely to be less than the true

accumulation of pesticides because organochlorine pesticides are fat-soluble and can deposit in lipids and organs. The organochlorine pesticides can be classified into four categories: dichlorodiphenylethanes (e.g., DDT), cyclodienes, chlorinated benzenes (e.g., hexachlorobenzene [HCB]), and cyclohexanes (e.g., hexachlorocyclohexane [HCH]) (CDC 2003).

The pesticide metabolites detected in urine represent the organophosphate (and some organochlorine) pesticides that the body excretes; this excretion is positively correlated with the recent exposure to pesticides, although the exact correlation is largely unknown.

The survey contains data on several organochlorine and organophosphate pesticide metabolites in individuals aged 12 and older, as shown in Table 1A in the Appendix. Most serum organochlorine pesticide metabolites have little or no variations, except for DDT metabolites, (pp-DDE in particular) among the usable children observations.

#### D. Is the Occupational Exposure Indicator a Valid Indicator for Pesticide Exposure?

If we find that children in exposed families have a higher concentration than those in unexposed families of organochlorine pesticide metabolites in the blood serum, we could justify the use of the household-level occupational exposure indicator,  $E_i$ , as a proxy of children's chronic exposure to pesticides. Let

$$(6) \quad m_{ij} = \alpha + E_j \delta + X_i' \gamma + M_i' \eta + a_i + Z_{ij}' \pi + u_{ij}$$

where  $m_{ij}$  is the pesticide metabolites concentration level of child  $j$  in family  $i$ ,  $M_i$  are the pesticide metabolites concentration levels of mother and father of the same type as  $m_{ij}$ , and the rest of the variables are the same as those in (1). A positive and significant  $\delta$  would justify the use of  $E_i$  as a representation of  $p_{ij}$  in (3) to estimate the impact of pesticide exposure on children's health.

We use the Principal Component Analysis on the three types of persistent pesticide metabolites with the largest variations, that is, DDT (pp-DDT), HCB, and HCH (beta-BHC). We use one principal component and apply (6) under the assumption that the unobservable family effect,  $a_i$ , is uncorrelated with other covariates. We use two household-level exposure variables: the indicator of whether anyone in the household was exposed and that of exposed persons in the household. Results are shown in Table 3.

When we use the family occupation exposure indicator as the exposure variable, we find that the principal component of the pesticide metabolites is positively correlated with the indicator and correlations are significant at the 10% level. Children born in the United States have a significantly lower level of metabolites than immigrant children. Children's pesticide metabolites are positively associated with their smoking habits and the population density of their living location; children from the metropolitan statistical areas in a major city have lower levels of metabolites than rural children outside any standard metropolitan statistical areas.

When we use the number of exposed persons in the family as the exposure variable, we obtain qualitatively similar results. Children's pesticide metabolites are positively correlated with the number of exposed person in the family, and correlations are significant at the 1% level<sup>10</sup>. Taking together the estimates of (6), we conclude that children's pesticide metabolites are strongly and significantly correlated with (a) the occupational exposure of any family member, and (b) the number of exposed family members. This justifies using these two exposure variables as proxies for children's chronic exposure to pesticides.

### E. Differences due to Indirect Exposure

Using the family occupational pesticide exposure indicator as a valid proxy to children's chronic exposure to pesticides, we then stratify the sample into exposed and unexposed children based on  $E_i$ , the family occupational exposure indicator, and present the summary statistics in the columns 2 and 3 in Table 1. Compared to unexposed children, exposed children are on average 6% less likely to attain good health and 1% more likely to have chronic conditions. There is some difference in the basic demographics: exposed children are on average slightly older, more likely to be male, and more likely to be Mexican or Cuban Hispanics. In addition, they tend to live in more densely populated areas. There is little difference in serum lead levels or per capita family income. There is considerable difference in parents' education and health-related behaviors of the two groups. Parents of exposed children were better educated, made fewer recent routine checkups, and were more likely to have adequate nutrition and better accessibility to medical services. The  $t$ -stat column indicates that exposed and unexposed children differ in the sample average of many characteristics.

## **V. Empirical Results**

This section presents the main results of our analysis. We start with estimating the impact of direct occupational pesticide exposure on older children's chronic conditions and overall health status, and then estimate the impact of parental occupational pesticide exposure on younger children's cognitive disorders and physical health based on chronic conditions and overall health status.

### A. The Impact of Direct Exposure

Table 4 is an expanded version of the first column of Table 1 and provides more details on the health impact of direct occupational exposure. We use the indicator of the child's occupational exposure to pesticides as the exposure variable and estimate (3) with a fixed effect estimator to fully account for family effects. We control for child-specific characteristics such as the child's country of birth, smoking history, adequacy of nutrition, serum white blood count, serum total cholesterol level, serum lead level, age, and gender. The inclusion of serum variables allows us to account for the differences in immune system (white blood count), dietary impact (cholesterol), and exposure to other toxins (e.g., lead); these differences may result from the interaction between genetics, environment, and behavior.

We find that, compared to their siblings who were not directly exposed to pesticides, directly exposed children are nearly 55% more likely to have heart diseases and the increased risk is significant at the 5% level; they are 5% more likely to have chronic respiratory conditions such as tuberculosis and the increased risk is significant at the 1% level; and they are also about 16% more likely to have chronic kidney diseases though the increased risk is insignificant at the conventional levels.

Combining all the chronic conditions related to heart, lung, and kidney, we find that directly exposed children are nearly 33% more likely to develop one or more chronic conditions than their directly unexposed siblings, and the increased risk is significant at the 1% level. The chance of directly exposed children developing one chronic condition is increased by nearly 49%, and the increased risk is significant at the 1% level.

## B. The Impact of Indirect Exposure

Table 5 is an expanded version of the second and third columns of Table 1, and provides more details on the health impact of indirect exposure. The exposure variable indicates whether anyone in the child's family was occupationally exposed to pesticides. We estimate (3) with a random effect estimator, controlling for a large set of child- and family-specific characteristics. We find that children whose parents were occupationally exposed to pesticides have a greater likelihood than children from unexposed families to exhibit cognitive disorders, develop specific chronic conditions, and experience poor overall health. After controlling for an extensive set of characteristics, we find the increased risks in all health outcomes are large and highly significant.

The child-specific characteristics comprise the child's birthplace (United States or elsewhere), presence of any birth defects or complications, nutritional adequacy, serum white blood count, serum total cholesterol level<sup>11</sup>, serum lead level, ethnicity, age, and gender. The family-specific characteristics are parents' education, household income<sup>12</sup>, current farming activity, smoking history, heavy alcohol use, and recentness of their last routine checkup. In addition, since environmental health shocks may vary in types and concentrations across geographical regions, we control for the population density of the area the child lives in<sup>13</sup>, whether the family lives in a standard metropolitan statistical area (MSA), whether they live in a central city, and ethnicity—which also captures the variations among the major regions, since health shocks in California or Texas may differ from those in New York City

### *Cognitive Disorders*

Table 5 presents the estimates of the impact of indirect pesticide exposure to children's cognitive disorders. We find that children whose parents were occupationally exposed to

pesticides are 5.7% more likely to exhibit one cognitive disorder than children whose parents were not occupationally exposed to pesticides, and the increased risk is significant at the 5% level. Diagnosed by the child's physician, these cognitive disorders include problems relating to speech and language, coordination, psychology or behavior, and convulsions, but exclude mental retardation<sup>14</sup>.

Consistent with the literature that blood lead causes mental dysfunction in children, we find that for every 1  $\mu\text{g}/\text{dl}$  increase in serum lead, a child's chance of exhibiting one cognitive disorder increases 0.5%, and the increased risk is significant at the 1% level. The cognitive impact of parents' occupational pesticide exposure on their children is as large as the impact of having an additional 11  $\mu\text{g}/\text{dl}$  of lead.

We also find that children with birth problems are 10% more likely to have cognitive disorders. To isolate the effect of birth problems including birth defects and complications, we estimate on children with and without birth problems separately. We find that the impact of pesticide exposure in children with birth problems is statistically insignificant and twice as large as that in children without birth problem, while the impact is statistically significant in those without birth problems. Similarly, the impact of lead is statistically significant in children without birth problems and statistically insignificant in those with birth problems. This is not to say that children with birth problems are not cognitively affected by exposures to pesticides or lead.

We then estimate the impact of lead using a family fixed-effect estimator to explore the potential confounding of the unobservable family factors. We find that the marginal impact of lead is 0.9% on all children and significant at the 1% level; 0.6% on children without birth problems and significant at 1% level; and 8.3% on children with birth problems at the 5% level.

The considerable difference of the impact of lead between the random and fixed effect estimates indicates that, without accounting for the unobservable family factors, the estimate of the lead on children with birth problem would be biased downward as much as one magnitude. Though this analysis does not provide definitive evidence on how the unobservable family factors influence our estimates of the impact of pesticide exposure, the fact that the cognitive impact of lead on children without birth problems is robust to the unobservable family factors provides may suggest that the cognitive impact of pesticide exposure on the same group of children is robust to the unobservable family factors.

In addition, we find that cognitive disorders are less pronounced in Mexican-American children than other Hispanic children, and this may be the ethnic and genetic difference in the susceptibility to pesticides. Childhood cognitive disorders are positively associated with the child's age, although a younger child may be more likely to be diagnosed than an older one, and this may also be that the impact is cumulative.

The random effect estimate of the cognitive impact of parental occupational exposure is robust to the nonlinear interactions between all the covariates. The matching analysis, adjusted for bias and heteroskedasticity, shows that the cognitive impact of indirect pesticide exposure is 5.1% and significant at the 5% level.

#### *Physical Health: Chronic Conditions and Health Status*

Table 6 shows cross-sectional estimates of the impact of indirect pesticide exposure on children's likelihood to develop specific chronic conditions and on overall health status; we focus on children aged 4–11 who were born in the United States. Compared to unexposed children, we find that children whose parents were occupationally exposed are 9.7% more likely

to develop respiratory diseases including asthma and the increased risk is significant at the 1% level.

The physician-assessed nutritional adequacy is found to be positively associated with the incidence of respiratory diseases. The positive association may be because parents who work more hours (and are thus more exposed) earn more and can therefore better feed their children. Children with birth problems are 7.6% more likely to have respiratory diseases and the estimate is significant at the 10% level. Mexican-American children living in Texas, Colorado, New Mexico, Arizona, and California plus the Cuban-American children in Florida are 13% less likely to develop respiratory diseases than Puerto Rican-American children in the New York City area and the difference is significant at the 1% level.

We find that occupational pesticide exposure of the parents increases the children's chance of developing kidney-related diseases by 4.0% and heart-related diseases by 4.2%; increased risks are significant at the 5% levels. Occupational pesticide exposure of the parents increases the children's chance of developing a chronic respiratory, kidney or heart condition by 25% and the estimate is significant at the 1% level. The indirect exposure from their exposed parents gives these children a 33.8% higher chance to be in a next worse health status, and the estimate is significant at the 1% level.

These estimates of impact of parental occupational exposure on children's physical health are robust to the nonlinear interactions between the covariates. Estimates by the matching analysis, adjusted for bias and heteroskedasticity, are 7.5% for respiratory diseases and significant at the 5% level, 5.8% for heart-related diseases and significant at the 1% level, and 4.7% for kidney-related diseases and significant at the 5% level. The matching estimates are

22.7% for all chronic diseases and 36% for health status and both estimates are significant at 1% levels.

## **VI. Robustness of the Findings**

This section explores the sensitivity of the results to (1) an alternative construction of the indirect exposure variable; (2) inter-generational transmission of health; (3) different age and maternal education groups; and (4) functional form issues. We present the results of our focus on the impact of indirect exposure on cognitive disorders, number of chronic diseases, and overall health status in Table 7. We explore the sensitivity of the results to (5) birthplace and (6) gender-specific reporting error differences and focus on the impact of direct exposure on heart disease incidence and number of chronic diseases, displaying the results in Table 8<sup>15</sup>.

### A. Alternative Constructions of Exposure Variable

We examine the sensitivity of the results to three alternative constructions of the indirect exposure variable. We have used an indicator that measures whether anyone in the family was exposed to pesticides through farming and/or pesticide manufacturing and processing activities. First, we estimate the impact of indirect exposure through farming activities by excluding the children whose parents were exposed through pesticide manufacturing and processing activities only. Second, we estimate the impact of indirect exposure through pesticide manufacturing and processing activities by excluding the children whose parents were exposed through farming only. Third, we use the number of persons in the household who were occupationally exposed to pesticides as the exposure variable. For the usable data, 61% are unexposed, 22% have one

exposed person in the family, 12.8% have two, 3.7% have three, 1.3% have four, and 0.25% have five.

The cognitive impact is larger in children whose parents were exposed to pesticides through farming than in the children of those exposed through pesticide manufacturing, though the difference is statistically insignificant. The per-exposed-person impact on children's cognitive disorders is 3.5% and significant at the 1% level. The impacts on children's chronic diseases and health status are stronger in children whose parents were exposed to pesticides through pesticides manufacturing than farming, though the differences are statistically insignificant. The per-exposed-person impact on children's cognitive disorders is 3.5% and significant at the 1% level.

#### B. Inter-Generational Transmission of Health

We examine whether children's poor health is primarily driven by their parents' poor health by including maternal health as an additional regressor. The inclusion of maternal health may over-control for the inter-generational transmission of health since the mother is generally exposed for a longer period than the child, and the estimate of the impact on the child may be downward-biased. We include the mother's serum pesticide metabolites, more specifically, the first principal component based upon the Principal Component Analysis, using three major persistent pesticides (DDT, HCB, and HCH) to estimate the cognitive impact. We use the presence of the mother's chronic diseases to estimate the impact on a child's physical health. We find the estimates accounting for maternal health are unchanged in either the magnitude or the significance level.

### C. Results by Different Age and Maternal Education

We examine whether the impact of indirect exposure varies across different child age groups or maternal education. We find that the cognitive impact of pesticide exposure is more pronounced in older children (aged 8–11) than in those under age seven; however, the impact of lead is more pronounced in the younger children. A child whose mother has more than a high-school education is more affected cognitively by pesticide exposure than one whose mother has no more than high school education, though the difference is statistically insignificant. The cognitive impact of lead is statistically significant only in children whose mothers did not finish high school.

The impact of pesticide exposure on chronic childhood diseases is stronger in older children (above age seven) than in younger children (under age seven), though the impact is equally statistically significant and the difference in the impact is statistically insignificant. The effect of pesticide exposure on children with better-educated mothers is stronger for developing chronic diseases, 39.1% versus 24.8%, and the difference is statistically insignificant. We obtain qualitatively similar findings for the health status variable<sup>16</sup>.

### D. Functional Form Issues

We examine the appropriateness of the linear probability model that we have used for specific categories of chronic diseases: respiratory, kidney, and heart diseases. We use a logistic probability model for the incidence of respiratory diseases, and the marginal effect of indirect pesticide exposure is 9.2% and significant at the 5% level with the robust standard error of 0.040, while the linear probability model estimate is 9.6% and significant at the 1% level with a robust standard error of 0.037.

We examine the appropriateness of the linear model that we have used for the 5-point scale health status variable by using an ordered probit model. The marginal effect of indirect pesticide exposure is 0.6% for poor health status and significant at the 10% level, 4% for fair health status and significant at the 5% level, 3.9% for good health status and significant at the 5% level, 0.9% for very good health status and significant at the 10% level, and -7.6% for excellent health status and significant at the 5% level.

#### E. Birthplace

We examine whether the fixed-effect estimate of the impact of occupational exposure varies with the children's birthplace, specifically, whether they were born in the United States, and present the results in the first two rows of Table 8. Children born outside the United States are slightly more affected than those born in the United State by heart disease incidence: 27.2% versus 23.7%, respectively, and the difference is statistically insignificant. The impact on chronic diseases is greater and statistically more significant in foreign-born children than in domestic-born children: 53.7% versus 22.8%, though the difference is statistically insignificant.

#### F. Gender-Specific Reporting Errors

We examine whether the fixed-effect estimate of the impact of occupational exposure is sensitive to reporting errors that are gender-specific, such as the fact that female teenagers may systematically over-report while male teenagers may under-report their outcomes. The third and fourth rows of Table 8 show the estimates by gender. We find that the estimate of the impact on heart diseases is larger in males than in females, 39.1% versus 22.3% and that the estimate of the

impact on all chronic diseases is slightly larger in females than in males, 39.6% versus 35.5%. None of the differences is statistically significant.

## **VI. Policy Implications**

In this section, we start with estimates from existing literature of the cost of chronic diseases and poor overall health in children. We perform the economic cost-benefit analysis on eliminating the externality of (parental) occupational pesticide exposure and discuss the relevance of our findings to current policy.

### A. Economic Costs of Cognitive Disorders, Chronic Diseases, and Poor Health

The economic costs of childhood chronic conditions and poor overall health include medical costs, special education cost (for the cognitive disorders), loss in parental income, missed school days, and generally compromised quality of life. The costs also include the loss in children's future earnings as adults.

The CDC estimates that more than 90 million Americans live with chronic illnesses. Chronic diseases account for 70% of all deaths in the United States. The medical care costs of people with chronic diseases account for more than 75% of the nation's \$1.4 trillion medical care costs. In 2001, approximately \$300 billion was spent on chronic cardiovascular diseases, and \$129 billion in lost productivity due to chronic cardiovascular disease.

We calculate the lower bound of the costs of chronic heart diseases as \$3,330 direct medical costs plus \$1,430 indirect costs (lost productivity) per patient, if all the 90 million Americans with chronic conditions have cardiovascular diseases. This amounts to an average of \$4,760 per year per person children and adults included.

We use asthma to calculate the costs of chronic childhood respiratory diseases. The direct medical costs are \$393 and indirect costs, including loss in parental income, are \$244 per child under age 17 with asthma per year; these costs are higher for persons with asthma above age 18: \$535 and \$373 for direct and indirect costs, respectively. This amounts to \$637 per year per child and \$908 per year per adult for asthma (Weiss and Sullivan 2001). We mention adults here because asthma is generally contracted in childhood.

We calculate the lower bound of the medical costs of chronic childhood kidney diseases. There are about 20 million U.S. residents with chronic kidney disease. The medical costs of kidney diseases may vary with the stage of the disease. Medicare claims for about 300,000 patients with end-stage renal disease, the final stage of chronic kidney disease, which requires dialysis, total more than \$16 billion annually, or \$53,000 per year per patient at the final stage. Assuming no medical costs at prior stages (an unrealistic assumption), the medical costs of chronic kidney disease would be \$795 per year per patient.

We estimate the costs of cognitive disorder using the estimates of the cost relating only to lead. The CDC estimates that each 1- $\mu$ g/dl reduction in blood lead would render some special education and medical costs unnecessary and save approximately \$2,000 per child (Needleman 1998).

The economic costs of poor childhood health include the reduction in children's future income as adults. Case, Fertig, and Paxson (2003) find that poor childhood health persists over the individual's lifespan, and that poor childhood health, including the presence of chronic conditions, is associated with lower educational attainment and lower earnings as adults. They find that for men, being in fair or poor health status at age 23 reduces their earnings by 64% at

age 33 and 24% at age 42. The loss of earnings (in log) is approximately 5% for every chronic condition present at age seven.

## B. The Economic Benefit-Cost Analysis

We apply the costs of cognitive disorders, chronic conditions, and poor health to our findings on the impact of childhood pesticide exposure (Table 1), and we perform the benefit analysis on the direct occupational exposure on older children, and indirect exposure from parental occupational exposure on younger children separately.

### *Costs of Direct Occupational Pesticide Exposures*

For older children, our fixed-effect estimates show that occupationally exposed children are 26% more likely to develop chronic heart diseases than unexposed children; this exposure incurs costs of \$1,237 annually per affected child. The present value of the costs associated with the increased chance of having heart diseases is \$24,740 with a 5% discounting rate and \$41,233 with a 3% discounting rate. The present value of the costs associated with the increased incidence of chronic respiratory diseases is \$600 with a 5% discounting rate and \$1,000 with a 3% discounting rate.

The occupationally exposed children are 49% more likely to develop a chronic condition. Given the average Hispanic annual individual income of approximately \$30,000 and the 5% reduction in log earnings as adults per chronic condition, the loss in annual income due to childhood occupational pesticide is \$7,556, a 25% reduction in adult earnings. The present value of the loss in exposed affected children's lifetime earning stream amounts to \$151,000 with a 5% discounting rate and \$250,000 with a 3% discounting rate.

Taking the medical costs and children's future earnings together, the economic costs of childhood occupational exposure amounts to \$175,000 with a 5% discounting rate and \$300,000 with a 3% discounting rate. These costs represent 30% of these children's lifetime earnings.

#### *Costs of Indirect Exposure from Parental Occupational Pesticide Exposure*

For the younger children, our cross-sectional estimates show that the occupational pesticide exposure of their parents increased their chance of having cognitive disorders by 5.7%. The impact of parental pesticide exposure to cognitive disorders is quantitatively equivalent to an increase of 11- $\mu$ g/dl in lead. The medical costs of the impact of parental pesticide exposure to cognitive disorders amounts to \$1,254 per year per affected child; the present value of these costs is \$25,000 with a 5% discounting rate and \$41,800 with a 3% discounting rate.

The medical costs associated with the increased risk resulting from parental occupational pesticide exposure of contracting asthma or other respiratory diseases or heart- and kidney-related diseases are \$300 per year per affected child; the present value of these costs is \$6,000 and \$10,000 with a 5% or 3% discounting rate, respectively. The reduction of their future incomes as adults is approximately 12.5%, based on the average individual Hispanic annual income of \$30,000. The present value of the loss in exposed children's lifetime earning stream is \$75,000 and \$125,000 with a 5% and 3% discounting rate, respectively.

Taking together the medical costs and the impact on children's future earnings, the economic costs of parental occupational exposure amount to \$100,000 and \$165,000 with a 5% and 3% discounting rate, respectively. These costs represent approximately 30% of these children's lifetime earnings.

### *The Cost Analysis*

There are several policy instruments: income transfer, terminating secondary exposure pathways, and reducing parental direct exposures. Our findings imply that income transfer is unlikely to be an effective instrument. Terminating the pathway of parental occupational exposure can be an effective instrument. For example, farms and other pesticide-related jobsites can provide sanitary facilities to allow farmers with children at home to clean themselves immediately after leaving the field contaminated with pesticides and related toxins. Frisvold, Mines, and Perloff (1988) report that lack of field sanitation on agricultural jobsites increases by 60% the probability of agricultural workers reporting gastrointestinal disorders. The marginal cost of providing such a facility is several magnitudes smaller than the benefit on a per-worker basis.

### C. Relevance to the Current Population

Our estimates of the impact of indirect pesticide exposure during childhood on children's cognitive and physical health is based upon the Hispanic Health and Nutrition Examination Survey that was conducted over twenty years ago. Many organochlorine pesticides introduced in the 1940s are used rarely in the United States today because of their environmental persistence. The Environmental Protection Agency banned many of the uses of organochlorine pesticides during the 1970s and 1980s.

However, trace amounts of organochlorine pesticides continue to be produced and used in the United States. Many developing countries continue to manufacture and process organochlorine pesticides, and some of them may be imported into the United States either as pesticides or as residue in foods. Our findings may be relevant to the Hispanic children in the

United States, as many of their parents are engaged in farming and pesticide production activities. The recent National Nutrition Examination Surveys show that Mexican-Americans have on average higher levels of persistent pesticide metabolites than the non-Hispanic, white, or black population. Future work should assess the relevance of our findings to the current generation of Hispanic children in the United States. Our findings are particularly relevant to regulating organochlorine pesticides in developing countries.

## **VII. Conclusions**

We estimate a detrimental impact of childhood pesticide exposure on children's cognitive and physical health. Among older children whose occupational exposure to pesticides and serum pesticide metabolites data are observed, we find that children who were occupationally exposed to pesticides through farming or pesticide production have significantly higher levels of persistent pesticide metabolites in their blood serum than those who were not, after adjusting for a large set of family-specific characteristics. The strong correlation between occupational exposure history and serum metabolites provides evidence that the occupational exposure history is a valid proxy for children's chronic exposure to pesticides.

Among older children aged 12–17, we find that occupational exposure to pesticides during late childhood increases their risk of developing a chronic heart-related disease by 26.1%, a chronic respiratory disease by 4.8%, and a chronic condition related to heart, lung, or kidneys by 48.8%; it also increases the incidence of poor overall health status by 23.8%. Our estimates account for all observed and unobserved family factors, a child's birthplace, smoking history, nutritional factors, age, and gender; the estimates are insensitive to maternal education, child's birthplace, and gender-specific reporting errors. The economic costs of childhood occupational

exposure, including medical costs and future earnings reduction, are 30% of children's lifetime earnings, which amounts to \$175,000 with a 5% discounting rate and \$300,000 with a 3% discounting rate.

Among younger children (aged 11 and younger) whose occupational pesticide exposure history and serum pesticide metabolites are not unobserved, we find that parental occupational pesticide exposure increases their risk of exhibiting cognitive disorders by 5.7%; this is equivalent to the impact of having an additional 11  $\mu\text{g}/\text{dl}$  of lead in the bloodstream, which is also found to increase childhood cognitive disorders. Living with occupationally exposed parents increases a child's chance of developing asthma and/or other chronic respiratory disease by 9.6%, a heart-related disease by 4.2%, a kidney-related disease by 4.0%, and a chronic condition related to heart, lung, or kidney by 25%.

Our estimates account for birth defects or complications, nutritional factors, serum lead, ethnicity, age, and gender; and parents' education level, income, smoking and drinking history, preventive care practices, and current farming activity; and finally, where the family lives and the population density there are also considered. Evidenced by the matching analysis, our results are robust to complex nonlinear interactions among the covariates. Our results are insensitive to the occupational settings where parents were exposed to pesticides: farming versus pesticide production; using the number of exposed persons as the exposure variable; maternal health; child's age; and maternal education.

The economic costs of parental occupational exposure, including medical costs and children's future earnings are approximately 30% of these children's lifetime earnings, which amounts to \$100,000 and \$165,000 with a 5% and 3% discounting rate, respectively.

Our findings are particularly relevant to regulating organochlorine pesticides in the developing countries where persistent pesticides are still produced and used. Our findings may also be relevant to the current generation of Hispanic children in the United States and the assessment of the relevance should be an emphasis of future work.

## References

- Case A, Fertig, A and Paxson, C., "From Cradle to Grave? The Lasting Impact of Childhood Health and Circumstance", NBER Working Paper No. 9788, 2003.
- Chanda S. M. and Pope C. N., "Neurochemical and Neurobehavioral Effects of Repeated Gestational Exposure to Chlorpyrifos in Maternal and Developing Rats". *Pharmacol Biochem Behavior*, 1996, 53:771-6.
- Curl, C. L., et al. "Evaluation of Take-Home Organophosphorus Pesticide Exposure Among Agricultural Workers and Their Children", *Environmental Health Perspectives*, 2002, 110(12).
- Eriksson P., "Developmental Neurotoxicology in the Neonate-Effects of Pesticides and Polychlorinated Organic Substances". *Archives of Toxicology Supplement*, 1996; 18:81-8.
- Fenske, R., A., Kissel, J. C., Lu, C., et al, "Biologically Based Pesticide Dose Estimates for Children in an Agricultural Community", *Environmental Health Perspectives*, 2000, 108(6).
- Feychting, M., Plato, N., Nise, G. and Ahlbom A., "Paternal Occupational Exposures and Childhood Cancer", *Environmental Health Perspectives*, 2001, 109(2).
- Frisvold G., Mines, R. and Perloff J., "The Effects of Job Site Sanitation and Living Conditions on the Health and Welfare of Agricultural Workers", *American Journal of Agricultural Economics*, 1988, 70(4): 875-95.
- Garry, V. et al., "Pesticide Applicators, Biocides, and Birth Defects in Rural Minnesota", *Environmental Health Perspectives*, 1996, 104(4):394-9.
- Garry, V. F., Harkins, M. E., et al, "Birth Defects, Season of Conception, and Sex of Children Born to Pesticide Applicators Living in the Red River Valley of Minnesota, USA", *Environmental Health Perspectives*, 2002, 110(3).
- Guillette, E and et al, "An Anthropological Approach to the Evaluation of Preschool Children Exposed to Pesticides in Mexico", *Environmental Health Perspectives*, 1998, 106(6).
- Hill R., Head S., Baker S., Gregg M., Shealy D., "Pesticide Residues in Urine of Adults Living in the United States: Reference Range Concentrations". *Environmental Research* 1995; 71:99108.
- Hill R., To T., Holler J., Fast D., "Residues of Chlorinated Phenols and Phenoxy Acid Herbicides in the Urine of Arkansas Children". *Archives of Environmental Contamination and Toxicology* 1989; 18:469-474.
- Imbens, G. "Semiparametric Estimation of Average Treatment Effects under Exogeneity: A Review", University of California at Berkeley, Working Paper, 2003.
- Ma, X., Buffler, P. A., et al, "Critical Windows of Exposure to Household Pesticides and Risk of Childhood Leukemia", *Environmental Health Perspectives*, 2002, Volume 110, Number 9.

Moore, Colleen F., *Silent Scourge – Children, Pollution, and Why Scientists Disagree*, Oxford University Press, 2003.

Needleman, H.L., “Childhood Lead Poisoning: The Promise and Abandonment of Primary Prevention”, *American Journal of Public Health*, 1998, 88, 1871-7.

O’Rourke, M. K., Lizardi, P. S., Rogan S. P., Freeman, N. C., Aguirre, A., and Saint, C. G., “Pesticide Exposure and Creatinine Variation Among Young Children”, *Journal of Exposure Analysis and Environmental Epidemiology*, 2000, 10:672-681.

Porter, W., Jaeger J. and Carlson, I., “Endocrine, Immune and Behavioral Effects of Aldicarb (carbamate), Atrazine (triazine) and Nitrate (fertilizer) Mixtures at Groundwater Concentrations”, *Toxicology and Industrial Health*, 1999, 15(1-2):133-50.

Shealy D., Barr J., Ashley D., Patterson D., et al. “Correlation of Environmental Carbaryl Measurements with Serum and Urinary 1-naphthol Measurements in a Farmer Applicator and His Family”. *Environmental Health Perspectives*, 1997; 105:510-513.

Simcox, N. J., Fenske, R. A., Wolz, S. A., Lee, I. Kalman, D. A., “Pesticides in Household Dust and Soil: Exposure Pathways for Children of Agricultural Families”, *Environmental Health Perspectives*, 1999, 107(supplement 3):409-419.

Stephenson, Joan, “CDC Report on Environmental Toxins: Some Progress, Some Concerns”, *Journal of American Medical Association*, 2003, 289: 1230-1231.

Thomas P., “Pesticide-Induced Immunotoxicity: are Great Lakes Residents at Risk?” *Environmental Health Perspectives* 1995; 103:55-61.

Weiss, K.B. and Sullivan, S.D., “The Health Economics of Asthma and Rhinitis: Assessing the Economic Impact”, *The Journal of Allergy and Clinical Immunology*, 2001, 107(1): 3 –8.

Table 1: Summary of Basic Results

Outcome Variables	Direct Exposure	Indirect Exposure	
	Fixed Effects	Random Effects	Matching
Cognitive disorders	--	0.057 ** (0.025)	0.051 ** (0.025)
Respiratory diseases	0.048 *** (0.015)	0.096 *** (0.037)	0.075 ** (0.035)
Heart diseases	0.261 *** (0.067)	0.042 ** (0.021)	0.058 *** (0.018)
Kidney diseases	0.157 (0.105)	0.040 ** (0.020)	0.047 ** (0.027)
Number of diseases	0.488 *** (0.143)	0.250 *** (0.069)	0.227 *** (0.063)
Health status	0.549 ** (0.259)	0.338 *** (0.122)	0.360 *** (0.132)
Poor health incidence	0.238 ** (0.097)	0.065 (0.041)	0.034 (0.042)
Good health incidence	-0.170 (0.144)	-0.156 *** (0.051)	-0.181 *** (0.056)

Notes: Data are from the Hispanic Health and Nutrition Examination Surveys 1982–4. Presented are marginal effect estimates of specification (3) using a fixed-effect for the directly exposed older children in the first column and using a random effect for the indirectly exposed younger children in the second column, and matching analysis (5) in the third column. Standard errors are in parentheses. \*\*\*: 1% significance level. \*\*: 5% significance level. \*: 10% significance level.

Table 2. Summary Statistics and Difference due to Pesticide Exposure

	All	$E_i = 0$	$E_i = 1$	<i>t</i> -stat for diff in sample mean
Good health	0.488	0.518	0.459	3.04
Chronic conditions	0.148	0.139	0.159	1.44
Serum lead	10.427	10.433	10.420	-0.08
Adequate nutrition	0.99	0.99	0.99	0.00
Age	8.800	8.301	9.446	6.10
Female	0.492	0.502	0.482	-22.22
Mother education	5.607	4.430	7.127	13.99
Father education	3.994	2.589	5.808	18.74
Per capita income, log	8.217	8.266	8.154	-2.41
Parents' medical access	0.558	0.419	0.739	17.75
Parents' routine checkup	0.685	0.514	0.906	25.47
Parents' nutrition	1.018	0.719	1.404	24.22
Mexican Hispanic	0.652	0.617	0.697	4.36
Cuban Hispanic	0.127	0.094	0.169	5.65
Population density	4.247	4.068	4.479	4.42
Sample Size	2,692	1,517	1,175	

Notes: Un-weighted sample means are shown and standard deviations in parentheses. Good health is an indicator set to 1 if the child's health status is very good or excellent. Chronic conditions indicator is set to 1 if the child has one or more chronic conditions. Education is measured in years of completed schooling. Serum level of lead is in microgram per deciliter. Adequate nutrition is an indicator. Last routine checkup is a categorical variable with a smaller value indicating a most recent checkup.

Table 3: Sources of the Variations in Serum Metabolites using Principal Component Analysis

	Weather anyone was exposed		# Exposed persons	
Direct & indirect exposure	0.485* (0.282)	-	0.140*** (0.055)	-
Indirect exposure	-	0.655* (0.380)	-	0.291*** (0.110)
Born in U.S.	-0.593* (0.354)	-0.440 (0.492)	-0.581 (0.362)	-0.465 (0.503)
Ever smoked	0.581** (0.250)	0.800*** (0.294)	0.637** (0.265)	0.802*** (0.307)
Adequate nutrition	0.277 (0.669)	0.403 (0.817)	0.225 (0.615)	0.233 (0.693)
Age	0.059 (0.056)	0.094 (0.066)	0.054 (0.053)	0.095 (0.063)
Female	0.172 (0.223)	0.113 (0.225)	0.178 (0.209)	0.162 (0.225)
Mexican Hispanic	-0.058 (0.252)	-0.065 (0.285)	-0.003 (0.258)	-0.029 (0.285)
Cuban Hispanic	-0.493 (0.342)	-0.436 (0.422)	-0.400 (0.342)	-0.328 (0.431)
Mother PCA	-0.073 (0.078)	0.108 (0.325)	-0.097 (0.081)	0.153 (0.322)
Father PCA	-0.012 (0.109)	-0.007 (0.119)	-0.039 (0.104)	-0.026 (0.111)
Mother education	-0.022 (0.044)	-0.012 (0.049)	-0.026 (0.047)	-0.018 (0.053)
Father education	-0.037 (0.023)	-0.044 (0.032)	-0.033 (0.024)	-0.030 (0.033)
Per capita income	0.092 (0.092)	0.078 (0.116)	0.110 (0.096)	0.094 (0.120)
Parents' nutrition	-0.367 (0.361)	-0.390 (0.382)	-0.399 (0.341)	-0.511 (0.386)
Population density	-0.163* (0.090)	-0.222** (0.111)	-0.151* (0.083)	-0.201** (0.102)
MSA central city	-1.220** (0.559)	-1.420** (0.661)	-1.191** (0.544)	-1.292** (0.623)
$R^2$ [sample size]	0.23 [170]	0.26 [142]	0.22 [170]	0.25 [142]

Notes: Presented are the coefficient estimates of (6) using a random effect estimator where the dependent variable is the child's pesticide metabolites serum level, specifically, the first principal component in the Principal Component Analysis on the DDT, HCB and HCH pesticide metabolites in serum. Robust standard deviations are in parentheses. Estimates of "indirect exposure" are restricted to observations that were not directly exposed. Additional covariates are child's serum white blood cell count, cholesterol and vitamin A, as well as whether mother (father) ever smoked, and was ever a heavy drinker; coefficients on these covariates are statistically insignificant.

Table 4: Fixed Effects Estimates of the Impact of Direct Occupational Exposure

	Respiratory diseases	Heart diseases	Number of diseases	Poor health	Health status
Direct exposure	0.048*** (0.015)	0.261*** (0.067)	0.488*** (0.143)	0.233** (0.097)	0.549** (0.259)
Born in U.S.	0.011 (0.023)	0.068 (0.133)	0.481 * (0.282)	0.066 (0.191)	0.049 (0.512)
Ever smoked	-0.002 (0.010)	-0.007 (0.037)	0.008 (0.077)	0.016 (0.053)	0.009 (0.141)
Serum white blood count	-0.000 (0.002)	0.005 (0.008)	0.025 (0.017)	-0.023** (0.011)	-0.076** (0.031)
Adequate nutrition	-0.021 (0.039)	-0.120 (0.129)	-0.173 (0.273)	-0.024 (0.185)	0.733 (0.496)
Age	-0.001 (0.002)	0.005 (0.010)	0.047 ** (0.022)	0.011 (0.015)	-0.033 (0.039)
Female	-0.010 (0.002)	0.059 (0.034)	0.095 (0.077)	0.023 (0.054)	0.051 (0.43)
$R^2$	0.07	0.16	0.18	0.15	0.14

Notes: Presented are coefficient estimates of (3) using a fixed-effect estimator on children aged 12–17. Additional covariates are serum cholesterol and lead levels, and coefficients of these variables are insignificant in all columns. Standard errors are in parentheses. \*\*\*: 1% significance level. \*\*: 5% significance level. \*: 10% significance level. Sample size is 432.

Table 5: The Impact of Indirect Exposure on Children's Cognitive Disorders

	Random Effects			Fixed Effects		
	All	w/ bp	w/o bp	All	w/ bp	w/o bp
Pesticide exposure $E_i$	0.057** (0.025)	0.129 (0.102)	0.058** (0.026)	-	-	-
Serum lead	0.005*** (0.002)	0.006 (0.009)	0.005** (0.002)	0.009*** (0.003)	0.083** (0.037)	0.006*** (0.002)
Birth problems	0.103** (0.042)	-	-	-0.035 (0.066)	-	-
Age	0.007** (0.003)	0.015 (0.014)	0.004 (0.003)	0.009 (0.008)	-0.009 (0.053)	-0.001 (0.006)
Female	-0.027 (0.024)	0.027 (0.087)	-0.026 (0.020)	-0.013 (0.046)	-0.185 (0.053)	-0.061 (0.006)
Mexican Hispanic	-0.081* (0.039)	-0.235** (0.108)	-0.030 (0.036)	-	-	-
Cuban Hispanic	-0.059 (0.050)	-0.186 (0.130)	0.002 (0.052)	-	-	-
Parents farming now	-0.034 (0.048)	-0.270* (0.147)	-0.048 (0.036)	-	-	-
Mother education	-0.000 (0.003)	0.011 (0.007)	-0.002 (0.003)	-	-	-
Father education	-0.004 (0.003)	-0.009 (0.011)	-0.003 (0.003)	-	-	-
Per capita income, log	-0.003 (0.010)	-0.070* (0.037)	0.007 (0.011)	-	-	-
$R^2$	0.06 [866]	0.13[183]	0.05 [683]	0.04	0.34	0.07

Notes: Presented are coefficient estimates of (3) on children aged 11 and younger and born in the United States. The dependent variable is an indicator of whether the child was diagnosed with problems in speech and language, coordination, psychology or behavior, and convulsions, but excluding mental retardation. The exposure variable is whether anyone in the family was occupationally exposed to pesticides. Additional covariates are serum cholesterol and lead levels, nutritional adequacy, recentness of parents' last routine checkup, parental smoking and drinking history, population density of the living place, and SMA inside or outside the central city; coefficients of these variables are insignificant in all columns. Standard errors are in parentheses. "w/ bp" means children with birth problems; "w/o bp" means children without birth problem. Sample size is 866 for all, 183 for with birth problems, and 683 for without birth problems. \*\*\* 1% significance level. \*\* 5% significance level. \*: 10% significance level.

Table 6: The Impact of Indirect Exposure on Children's Physical Health

Dependent Variables	Respiratory diseases	Kidney diseases	Heart diseases	Number of diseases	Health status
Pesticide exposure $E_i$	0.097*** (0.037)	0.040** (0.020)	0.042** (0.021)	0.250*** (0.069)	0.338*** (0.122)
Birth problems	0.076* (0.041)	-0.010 (0.021)	0.017 (0.025)	0.307*** (0.094)	0.262** (0.123)
Age	-0.002 (0.006)	-0.003 (0.004)	-0.002 (0.004)	0.000 (0.012)	-0.001 (0.020)
Female	-0.036 (0.026)	0.082*** (0.016)	0.010 (0.017)	0.001 (0.054)	0.003 (0.091)
Mexican Hispanic	-0.130*** (0.045)	0.038** (0.018)	-0.032 (0.025)	-0.171* (0.086)	-0.177 (0.141)
Cuban Hispanic	-0.060 (0.068)	0.069* (0.040)	0.019 (0.046)	0.007 (0.130)	-0.622*** (0.205)
Parents farming now	-0.068 (0.077)	-0.012 (0.045)	-0.056* (0.032)	-0.333** (0.141)	0.842** (0.362)
Mother education	0.002 (0.005)	0.003 (0.003)	-0.001 (0.003)	0.007 (0.009)	-0.031** (0.015)
Father education	0.005 (0.005)	0.005* (0.003)	0.000 (0.003)	0.004 (0.010)	-0.066*** (0.018)
Per capita income, log	0.014 (0.013)	0.005 (0.008)	-0.001 (0.008)	0.010 (0.023)	-0.016 (0.050)
$R^2$	0.09	0.09	0.03	0.10	0.16

Notes: Presented are coefficient estimates of (3) using a random-effect estimator on children aged 4–11 and born in the United States. Dependent variables are (a) respiratory diseases, an indicator whether the child was diagnosed to have asthma or other lung diseases; (b) kidney diseases, an indicator for urinary diseases or infections; and (c) heart diseases, an indicator for rheumatic heart or other heart diseases. The exposure variable is whether anyone in the family was occupationally exposed to pesticides. Additional covariates are serum cholesterol and lead levels, recency of parents' last routine checkup, parental smoking and drinking history, population density of the living place, and SMA inside or outside the central city; coefficients of these variables are insignificant in all columns. Standard errors are in parentheses. Sample size is 588. \*\*\* 1% significance level. \*\* 5% significance level. \*: 10% significance level.

Table 7: Robustness Checks of the Estimates of the Impact of Indirect Exposure

	Cognitive disorders		# Chronic diseases	Health status
	Pesticides	Lead	Pesticides	Pesticides
Exposure through farming <sup>i</sup>	0.081*** (0.032) [0.07, 733]	0.005*** (0.002)	0.265*** (0.090) [0.09, 496]	0.311** (0.146) [0.15, 498]
Exposure through pesticides manufacturing <sup>i</sup>	0.064** (0.029) [0.07, 729]	0.005*** (0.002)	0.299*** (0.082) [0.10, 502]	0.388*** (0.133) [0.19, 503]
Number of exposed persons	0.035*** (0.014) [0.06, 866]	0.005*** (0.002)	0.110*** (0.034) [0.09, 586]	0.136*** (0.051) [0.16, 587]
Mother's health <sup>ii</sup>	0.057** (0.025) [0.06, 866]	0.005*** (0.002)	0.251*** (0.089) [0.10, 586]	0.341*** (0.121) [0.17, 587]
Age under 7	0.025 (0.025) [0.06, 606]	0.005** (0.002)	0.195*** (0.077) [0.09, 327]	0.384*** (0.150) [0.21, 327]
Age 8-11	0.133** (0.065) [0.12, 260]	0.003 (0.004)	0.315*** (0.125) [0.16, 259]	0.271 (0.178) [0.15, 260]
Mother has high school diploma or less	0.063** (0.029) [0.06, 771]	0.005*** (0.002)	0.248*** (0.072) [0.10, 520]	0.299** (0.131) [0.13, 530]
Mother has more than high school	0.074* (0.039) [0.43, 95]	-0.002 (0.003)	0.391** (0.149) [0.59, 57]	0.490* (0.278) [0.56, 57]

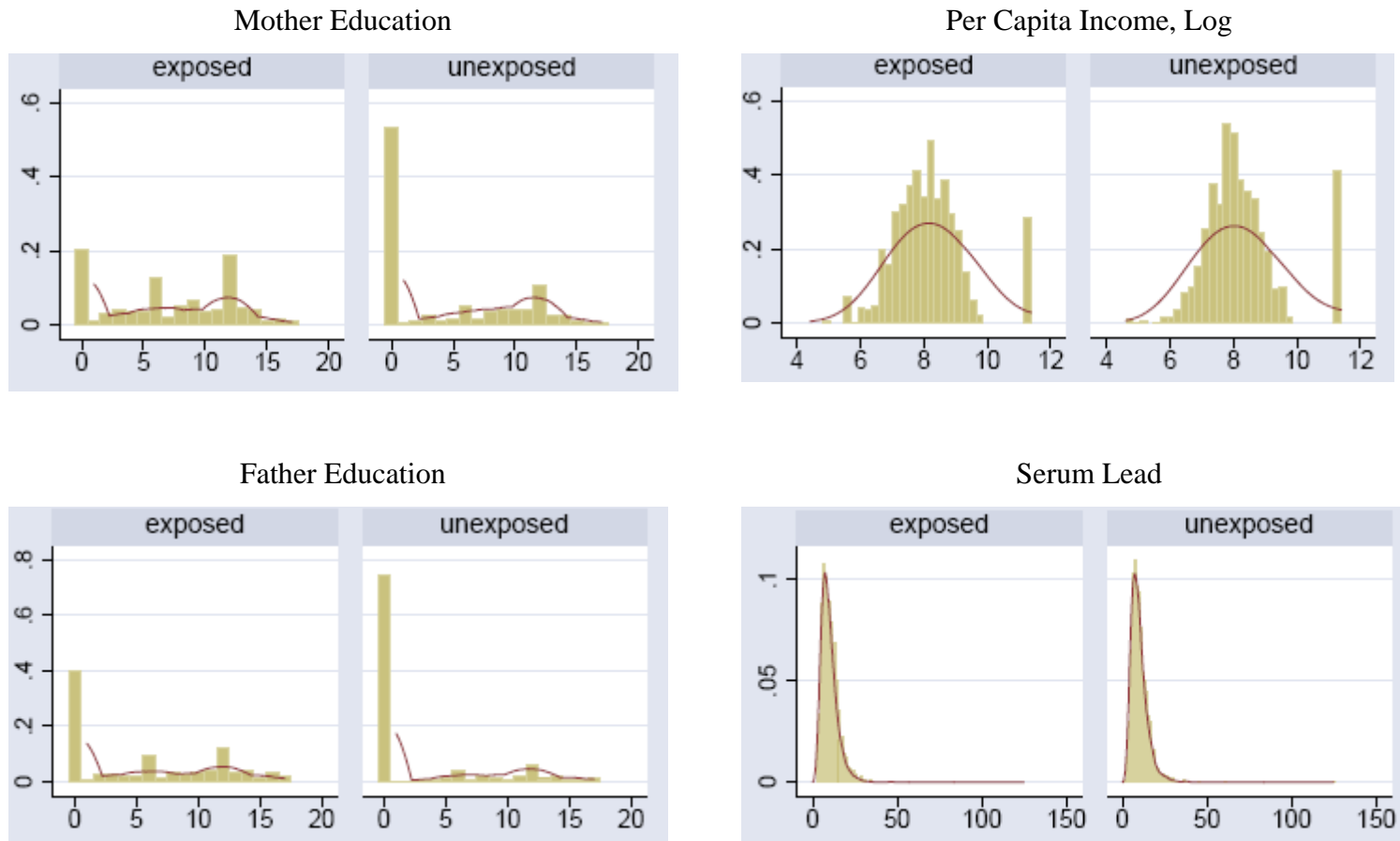
Notes: See Notes in Table 5. i: The estimates are obtained on subsamples that exclude observations on children whose parents were exposed to pesticides only through pesticide manufacturing (farming). ii: Mother's health is represented by the serum pesticide metabolites (the first principal component, see Table 3) for the cognitive disorders, and mother's chronic conditions in physical health. In brackets are R-squared and sample size.

Table 8: Robustness Checks of the Fixed-Effect Estimates of the Impact of Direct Exposure

	Heart diseases	# Chronic diseases
Born in the U.S.	0.237 *** (0.090) [0.17, 333]	0.228 * (0.127) [0.17, 333]
Not born in the U.S.	0.272 ** (0.109) [0.37, 99]	0.537 ** (0.202) [0.51, 99]
Female	0.223 ** (0.104) [0.31, 214]	0.396 * (0.199) [0.21, 214]
Male	0.391 *** (0.100) [0.50, 218]	0.355 ** (0.138) [0.37, 218]
Mother's education less than 10 years	0.260 *** (0.077) [0.16, 299]	0.338 *** (0.111) [0.15, 299]
Mother's education 10 or more years	0.296 * (0.168) [0.34, 133]	0.331 (0.287) [0.30, 133]

Notes: See Notes in Table 4. Presented are fixed-effect estimates of the coefficient of the direct occupational pesticide exposure indicator in (3). In brackets are R-squared and sample size.

Figure 1: Distributions of Key Variables by Family Exposure Indicator



Appendix: Table 1A: Percent Detected Positive in Biomonitoring Data

Pesticides	Urine metabolite	Serum metabolite	M	C	PR
<i>Organochlorine pesticides</i>					
DDT		pp'-DDT (2)	15.2%	7.3%	1.9%
		pp'-DDE	99.7%	97.0%	90.5%
Hexachlorobenzene [HCB]		Hexachlorobenzene	4.8%	6.3%	
		Pentachlorophenol (2)	29.7%		
Hexachlorocyclohexane [HCH]		alpha-BHC		1.0%	
	[HCB] & [HCH]	2,4,5 Trichloropheol (5)	1.1%		
		2,4,6- Trichloropheol (2)	5.5%		
Chlordane, Heptachlor		trans-Nonachlor	8.3%	1.1%	1.0%
		Oxychlordane	4.4%	5.5%	1.0%
		Heptachlor epoxide	1.0%	1.0%	
Dieldrin		Dieldrin	3.5%	1.5%	1.0%
PCB		PCB (15)	1.0%		
<i>Organophosphate pesticides</i>					
Malathion		Monocarboxylic acid (10)	6.2%		
		Dicarboxylic Acid (10)	2.2%		
(methyl) Parathion		para-Nitrophenol (10)	1.1%		
Chlorpyrifos (methyl)		3,5,6-Trichloro-2- pyridinol (3)	18.4%		
<i>Herbicides</i>					
		Dicamba(3.0)	1.0%		
		2,2,4-D (10)	1.0%		

Notes: MDL: Minimal detectable level. MDL is 1.0 ppb unless specified otherwise in parentheses. M: Mexican-Americans. C: Cuban-Americans. PR: Puerto Rican-Americans. Sample size: M only for all urine metabolites where n = 2,022 for urine malathion, n = 2,008 for other urine metabolites. For serum metabolites, n = 2,034 for M, n = 399 for C and n = 671 for PR.

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- <sup>1</sup> Various regional studies report similar findings (an Arkansas study by Hill et al. 1989, a Minnesota study by Adgate et al. 2001, and an Arizona study by O'Rourke et al. 2000).
- <sup>2</sup> The pesticide exposure history data was available for individuals aged twelve or older.
- <sup>3</sup> However, in the cases of divorces and subsequent merging of families, children may not live with their biological parents, or siblings in the family do not share the same biological parents. We are unable to screen for this possibility from the survey.
- <sup>4</sup> See Imbens 2003 for a review on semiparametric estimation of average treatment effects, and Abadie and Imbens 2002 for a detailed discussion of the properties on the matching estimators we use in this paper.
- <sup>5</sup> The following description draws from Abadie, Drukker, Herr, and Imbens 2002.
- <sup>6</sup> Allowing for the possibility of ties, this is the distance such that fewer than  $M$  units are closer to unit  $k$  than  $d_M(k)$ . If there are no ties, there would be exactly  $M$  matches as close to  $X_k$  as  $d_M(k)$ .
- <sup>7</sup> Other values are 2 = Very Good, 3 = Good, 4 = Fair and 5 = Poor.
- <sup>8</sup> Children aged 12 or older responded to the survey themselves and children under aged 11 and under had their parents respond to the survey for them.
- <sup>9</sup> The CDC reports that in the early 1990s, 4.4% of children had elevated blood lead levels, defined as levels  $\geq 10$   $\mu\text{g}/\text{dl}$ ; in 2000, the percentage of children aged 1-5 years with elevated blood lead levels decreased to 2.2% (CDC 2004). However, CDC and AAP currently use 15  $\mu\text{g}/\text{dl}$  as the level of concern in an individual child (CDC 1991, AAP 1998).
- <sup>10</sup> Then we estimate (6) by mother's education (high school dropouts versus high school diploma and higher) and child's age (12–14 versus 15–17). The correlation between metabolites and the exposure variable is large and significant in children whose mothers have a high school education or higher. The correlation is larger in the 15–17 group than the 12–14 group. None of these differences by groups is statistically significant.
- <sup>11</sup> The inclusion of these serum variables is motivated by the same reason as in the estimates of impact of direct exposure.
- <sup>12</sup> Ideally, we would use measures of permanent incomes such as asset ownership. However, the survey does not contain measures of permanent incomes other than parental education.
- <sup>13</sup> Ideally, we would use more detailed data on children's living locations (such as the zip code) as well as whether their homes have running water. However, these details are not available in the survey.
- <sup>14</sup> Among the 3,692 usable observations, more than 7% had one cognitive disorder, and an additional 1% had two or more.
- <sup>15</sup> Ideally we would like to empirically test the assumption in the fixed-effect estimator that siblings are "perfectly exchangeable" in estimating the impact of occupational pesticide exposure on their health outcomes. Such a test is made infeasible by the small number of usable observations.
- <sup>16</sup> We find the fixed-effect estimate of the impact of direct occupational pesticide exposure is insensitive to maternal education in magnitude of the impact, shown in the last two rows of Table 8. The impact on heart diseases incidence and number of chronic conditions is statistically more significant in children with less educated mothers than those with more educated mothers, and the difference in the point estimates is statistically insignificant.