Pesticides and Child Health: Evidence from Hispanic Children in the U.S.

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Abstract

This paper examines whether there is an externality of parental occupational exposure to pesticides on children's health, and whether some children are more severely affected by the externality than others. Using the Hispanic Health and Nutrition Examination Survey, we find children of exposed parents are more likely to develop chronic conditions and less likely to attain good health than children of unexposed parents, after controlling for a large set of child and family characteristics. Furthermore, children from low socioeconomic status are most vulnerable to health shocks resulting from pesticides and other related environmental toxins. Our analysis suggests that terminating the pathway of parental occupational exposure would be cost effective to correct the externality. Taken together with earlier findings that poor childhood health is associated with lower adult earnings, our results suggest more attention to be paid to the health shocks from environmental toxins for the poor as a potential mechanism through which the increasing poverty across generations at the very poor takes place: poverty makes individuals more susceptible to health shocks at childhood, which is associated with worse poverty for their children.

Introduction

There is substantial toxicological evidence that chronic exposure to pesticide affects neurodevelopment in developing animals. Children are more susceptible to environmental toxins than adults because their bodies and brains are immature. They are more exposed because they have greater contact with environmental contaminants. A recent United Nation Report, "*Children in the New Millennium: Environmental Impact on Health*", states that nearly one third of the global disease burden is the result of environmental factors, and more than 40% of this burden is borne by children under 5 years of age - some 600 million children. Such factors include exposure to toxic chemicals, such as lead and pesticides, a particular risk for the millions of children worldwide whose parents work in agriculture (Stephenson 2002).

We ask two questions. First, is there an externality of pesticide-related activities on children's health? Is the externality a causal? Second, are some children bearing more from this externality than others? We focus on the Hispanic children in the United States because their parents often engage in farming and other pesticide related activities, bring home pesticides and subject them to chronic exposure.

We think this paper is the first economic study on the externality of pesticide related activities on children's health. Our second question is also related to the recent SES-gradient literature, which states that a negative relationship between socioeconomic status (SES) and health exists in childhood, and becomes more pronounced with age (Case, Lubotsky and Paxson 2002). One explanation for the SES-gradient is that poor children experience more health shocks than non-poor children (Currie and Stabile, 2003).

Our second question is motivated by animal studies showing that undernourished animals are more vulnerable to pesticides poisoning, which implies that poor and undernourished children may be at greater risk. There is an association between higher residues of organochlorine pesticides in blood serum and black race and lower social class (Davies et. al. 1972). Poor children in large cities are likely to be exposed to high level of pesticides; for example, in the state of New York, the heaviest use of pesticide occurs in

Manhattan and Brooklyn where apartments are sprayed monthly in late 1990s (Landrigan et al. 1999).

The rest of the paper is organized as follows. Section 1 is literature review, followed by data description in Section 2. Section 3 describes a conceptual framework. Section 4 is empirical specification. Section 5 reports estimation results. Section 6 concludes.

1. Literature Review

1.1 Health Risks of Pesticides Exposures

Low-level exposures to pesticides are extremely prevalent in the U.S. population, and children under age 11 have the highest organophosphate pesticides exposures of all age groups, as reported by the National Health and Nutritional Examination Surveys. (Hill et al. 1995, Stephenson 2003). 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).

Animal studies show that a *single* low-level exposure to certain organophosphates during the early brain development can cause permanent changes in brain chemistry and consequently, changes in learning and behavior. Low-level exposures cause abnormal levels of thyroid, impaired production of antibody to foreign protein, and other immune dysfunctions (Porter et al. 1999).

Some pesticides are found to appear to target the developing brain during the critical period of cell division (Chanda and Pope 1996, Eriksson 1996), and some are found to be toxic to the immune system in animals (Thomas 1995). Some studies find that nicotine and chlopyrifos (an organochlorine pesticide) alter early brain development in a similar way (Moore 2003, Slotkin 1998 and 1999). Complex sex reversal is observed in both laboratory experiments and field studies at 0.1 ppb dose (Hayes et al. 2003).

Guillette et al. (1998) is a remarkable study on children. They find neurological and behavioral differences among the exposed and control children, where the exposed children lived in a farming community with heavy use of pesticides, and the control children lived in a similar community with little use of pesticides. They report that the

exposed children exhibited remarkably impaired hand-eye coordination, decreased physical stamina, short-term memory impairment, difficulty in drawing, and more aggressive and anti-social behavior, compared to the control children.

Adults' studies show that chronic organophosphates exposure can cause adverse effects on neurological function, cancer and reproductive function. Pesticides exposures lead to fourfold increased risk of early-onset of Parkinson's disease for adults (Butterfield et al. 1993, Gorell et al. 1998). Epidemiological studies (NAS 2000) find an increase in the prevalence of neurological and psychiatric symptoms. Antle and Pingali (1994) show that pesticide use has a negative effect on farmer health in a case study on Philippine rice farmers.

Children are more susceptible to the toxicity of pesticides because of their physical immaturity. Animal studies show that immature animals are more susceptible to the neurotoxic effects of organophosphate insecticides (Harbison 1975, Benke and Murphy 1975). The lethal dose of some organophosphate insecticides in immature animals is only one percent of the lethal dose of adult animals (Spyker and Avery 1977). The maximum tolerated dose of chloypyrifos in infant rats was one-sixth of the maximum tolerated dose in the adult rats on weight-adjusted basis (Whitney et al. 1995).

Zahm and Ward (1998) reported that many childhood malignancies were linked to pesticides, and that the reported increased risks are of greater magnitude than those observed in adults' studies, suggesting that children may be more sensitive to the carcinogenic effects of pesticides than adults.

1.2 Parental Occupational Exposures

Children of agricultural workers and pesticide applicators are subject to greater levels of pesticide exposures from helping their parents on the farm, living on or near a farm, and living with their exposed parents who bring home pesticides. Parental occupational exposures may subject their children to chronic exposure.

Numerous studies find that occupational pesticide exposure of one family member increases pesticide exposure of everyone else in the family. Shealy et al. (1997) of the Agricultural Health Study shows that all family members had increased exposure after

one family member used a pesticide applicator. Simcox et al. (1999) collected household dust and soil samples and found that farming households contain greater levels of pesticides than non-farming households. Fenske et al. (2000) found that children of farming parents were more likely to be exposed to doses that exceeded the U.S. Environmental Protection Agency reference dose than children of non-farming families. Curl et al. (2002) reports the similar findings. Children of farming families usually live near the farms where they are subject to additional exposure from agricultural drift (Richter et al. 1986).

There are several studies on the child health hazard of parental pesticide exposures. Feychting et al. (2001) found there was an increased risk of nervous system tumors in children related to paternal occupational exposure to pesticides in a cohort study based on a population of 235,635 children, who were followed from birth to 14 years. Ma et al. (2002) of the Northern California Childhood Leukemia Study found that exposure to household pesticides is associated with an elevated risk of childhood leukemia.

Children's susceptibility to pesticides begins with prenatal exposure. Garry (1996) is a notable study on birth defects. He finds pesticide applicators' children had significantly higher birth defect rates, from 210,723 live births in rural Minnesota between 1989 and 1992. Garry et al. (2002) found increased risks in birth defect or development disorders in the farming families.

2. The Model

2.1 A Theoretical Model

Our theoretical model draws heavily from Currie (2000). Parents are assumed to solve an intertemporal utility maximization as follows:

[1] $$\max_{\{C_t,H_t,V_t,G_t\}_{t=1}^T} \sum_{t=1}^T E_t \delta^t U(Q_{1t},Q_{2t},...,Q_{Lt},C_t,L_t;X_t,u_1,\varepsilon_{1t}) + B(A_{T+1})$$
subject to:
$$Q_{lt} = Q_l(Q_{l,t-1},G_t,V_t;Z_t,u_2,\varepsilon_{2t}),$$
$$C_t = Y_t - P_{gt}G_t - (A_{t+1} - A_t),$$
$$Y_t = I_t + w_t H_t + r A_t,$$
$$L_t + V_t + H_t = 1,$$

Where L is number of children in the family, δ is the discounting factor, U is the parents' utility function, B is a bequest function and A denotes assets. Q_{kt} is the health stock of child k at period t. C is consumption of other goods, L is leisure, X is a vector of exogenous taste shifters, u_1 is a vector of permanent individual specific taste shifters, and ε_1 is a shock to preferences.

In the constraint set, G and V denote material and time inputs into health production, Z is a vector of exogenous health productivity shifters, u_2 is a vector of permanent individual specific health productivity shifters, ε_2 is a shock to health productivity. Q_l is a child health production function with inputs, productivity shifters and shocks. Parental occupational pesticides exposure is an example of exogenous health productivity shifters Z.

Y is total income, P are prices, I is unearned income, w is the wage, H is hours of paid work, r is the interest rate, and endowments of health and wealth, Q_0, and A_0, are assumed to be given. Let R_k denote the set of the exogenous factors and shocks at period s, $R_s = \{X_s, Z_s, w_s, P_s, I_s, \varepsilon_{1s}, \varepsilon_{2s}\}$.

This model can be solved to yield the demand functions for C_t, H_t, G_t, and V_t of the following reduced form:

[2]
$$C_t = F^c(\lambda_t, R_t, M_t, r, \delta, u_1, u_2)$$
$$H_t = F^H(\lambda_t, R_t, M_t, r, \delta, u_1, u_2)$$
$$V_t = F^V(\lambda_t, R_t, M_t, r, \delta, u_1, u_2)$$
$$G_t = F^G(\lambda_t, R_t, M_t, r, \delta, u_1, u_2)$$

Where λ_t is the marginal utility of wealth and M_t is a vector of moments of the distribution of future R_s with $s = t+1...T$. F^C, F^H, F^V and F^G are functions. Repeatedly substituting the solutions for health inputs G and V into the health production function gives the demand for health stock Q_t:

[3]
$$Q_{lt} = Q^*(Q_0, J_t, \lambda_t, R_t, r, \delta, u_1, u_2)$$

Where J_t denotes all past realizations of R_s. with $s = 1... t-1$. Substituting the determinants of λ_t into [3] gives a demand function for Q_{lt}:

[4] $$Q_{lt} = Q^{**}(Q_0, A_0, R_t, J_t, M_t, r, \delta, u_1, u_2)$$

If we are willing to assume the distribution of future *R* is irrelevant, we can considerably simplify the model to the following:

[5] $$Q_{lt} = Q^{***}(R_t, J_t, \eta)$$

Where η denotes the child-specific or family-specific fixed effect that captures the set of initial conditions and unobserved factors $\{Q_0, A_0, r, \delta, u_1, u_2\}$.

2.2 Children's Health Determinants

This model helps identify the determinants of the child's health, that is, the variables that should enter the right hand side of the child health demand function [5]. It implies that parental taste shifters, family resources and information influence a child's health stock, which suggests family income and parental education are important determinants.

The model also implies that a child's health stock is an investment good whereby past investments or disinvestments in health determine current health stock. This suggests that parental health-related behavior; including material or time inputs to the health production may be important determinants.

A child's initial health stock influences the evolvement of a child's health stock. There may be heterogeneity of initial health stock. Children may be born with different levels of health endowment (See Case, Lubotsky and Paxson 2002 for an example).

Pesticides are not the only environmental toxins that children grow up with, and other environmental toxins may also have adverse effects on their health.

3. The Data

We use the Hispanic Health and Nutrition Examination Survey, conducted by the National Center for Health Statistics, a division of Center for Disease Control and Prevention. This survey is a cross-section nationwide probability sample of approximately 16,000 persons, 6 months-74 years of age, conducted between 1982 and 1984. It is conducted 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, allowing us to construct a comprehensive measurement of occupational exposure. After discarding missing data, we have 2,692 usable observations of children under age 17.

3.1 Summary Statistics

Table 1 contains the summary statistics of health outcomes, socioeconomic and health-related behavioral data. Based upon the self-reported or parents-reported health outcomes, half of the children are in good health, and good health is defined as being in the two best health status; 15% of the children have one or more chronic conditions.

The children are on average 8.8 years old. Mothers of the children completed on average 5.6 years of schooling, and fathers had on average 4.0 years of schooling. Average per capita family income is 3,704in 1984 dollar. Most of the children have adequate nutrition assessed by the physicians; *91%* of them have a place for routine checkups or emergence care, and their most recent routine checkups are approximately six months ago.

The children have on average 10 microgram per deciliter (µg/dL) lead in blood serum. According to American Academics of Pediatrics (AAP), as of 2003 there is yet no reliable threshold for lasting effects of lead exposure on cognitive development. Damages have been documented beginning at a blood lead concentration at 10 µg/dL, and even under 5 µg/dL in most recent studies (AAP 2003)[1].

3.2 Descriptive Analysis

We construct the pesticide exposure variables using the pesticide exposure history, which is available for individuals aged 12 and older. We construct our first indicator for each individual whether he or she was exposed to pesticides through occupational settings, and it is set to one if the individual had any of the following channels of exposure: pesticides were sprayed in the environment where they worked, they were involved in farming activities, worked in pesticide manufacturing plants, or used pesticides-containing

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[1] The Centers for Disease Control and Prevention (CDC) report that in the early 1990s, 4.4% of children had elevated blood lead levels, which are defined as levels ≥ 10 µg/dL; in 2000, the percentage of children aged 1-5 years with elevated blood lead levels has decreased to 2.2% (CDC 2004). However, CDC and AAP currently use 15 µg/dL as the level of concern in an individual child (CDC 1991, APP 1998).

products at home. We construct our second indicator for each household whether anyone in the household is directly exposed in occupational settings.

We stratify the sample into exposed and unexposed groups of children based on whether their families are exposed. Exposed children are on average 6% less likely to attain good health and 1% more likely to have chronic conditions than unexposed children. There is some difference in the basic demographics: exposed children are on average slightly older, more likely to be male, Mexican Hispanic or Cuban Hispanics than unexposed children. In addition, they live in more densely populated areas. There is little difference in serum lead levels or per capita family income.

However, there is considerable difference in parents' education and health-related behaviors where parents of exposed children were better educated, more likely to be have adequate nutrition, had better accessibility to medical services, and made less recent routine checkups than parents of unexposed children. The t-stat column indicates that exposed and unexposed children differ in the sample average of many characteristics.

4. The Specifications

There is substantial evidence from toxicological and children studies that chronic exposure to pesticides may increase risk to children's physical health and neurological development. While the exact biological mechanisms are largely unknown, we hypothesize that chronic exposure may prevent them from attaining good health, or put them at higher risk of developing chronic conditions. We focus on two health outcome indicators: the presence of one or more chronic conditions, and whether the child attains good health where good health is defined as having two best health status out of the five-scale self- or parents-reported health status.

Furthermore, without knowing whether different physiological mechanisms affect different health outcomes in different ways, we suppose that the observed health outcomes, those we are interested in estimating, are various representations of the health stock in [5]. For example, we suppose that the determination of a child's chance of attaining good health has the same structure as the determination of a child's health stock [5]. We provide panel specifications and the semi-parametric matching specification.

4.1 Logit Specifications

Let i denote family and j denote a child in the family. Let e_i is an indicator whether anyone in the household is exposed occupationally. Assuming health determinants are additively separable, a logit probability specification for binary outcomes can be described as follows:

$$\Pr\left(GH_{ij} = 1 \mid \eta_i, e_i, X_{ij}\right) = \Lambda\left(\eta_i + \alpha^I e_i + X_{ij}{}'\beta + \mu_{ij}\right) \qquad [6]$$

Where X are health determinants other than pesticide exposure. η is the unobserved family-specific effect. Λ is logit cumulative density function. α^I is the parameter of interest.

Parameter α^I is identified by the variation of parental exposure among the households. It conveys the effect of indirect exposure on a child's chance of attaining good health. For example, a negative estimate of α^I suggests that indirect exposure reduces a child's chance to attain good health. We apply the same specification to other health outcome indicators so that the identification and interpretation of α^I are consistent across multiples health indicators.

In addition, we observe the direct exposure for the older children between age 12 and 17, denoted as e_{ij}. Variations in e_{ij} allow us to identify the effect of direct exposure, α^D, in the following specification:

$$\Pr\left(GH_{ij} = 1 \mid \eta_i, e_{ij}, X_{ij}\right) = \Lambda\left(\eta_i + \alpha^D e_{ij} + X_{ij}{}'\beta + \mu_{ij}\right) \qquad [7]$$

Interpretation of α^D is similar to that of α^I in [6]

4.2 Matching Specifications

We employ a linear matching estimator for binary treatment or exposure[2]. We use the GH indicator as an example of the health outcome of interest. For child k, $k = 1...N$, GH_{k1} denotes the good health indicator if child k was exposed, and GH_{k0} denotes the good health indicator if child k was not exposed. Let X denote a vector of characteristics or health determinants other than pesticide exposure E_k.

We are interested in the sample average treatment (or exposure) effect for the treated τ_T, conditional on X:

$$\tau_T = \tfrac{1}{N_T}\sum\nolimits_{k:E_k=1}\left(GH_{k1} - GH_{k0}\right) \qquad [8]$$

Where N_T is the number of exposed children, $E_k = 1$ indicates child k was exposed and $E_k = 0$ indicates child k was unexposed.

However, we cannot observe both GH_{k1} and GH_{k0} at the same time for child k because he or she was either exposed or unexposed. What a matching estimator does is to impute GH_{k0} for exposed child k. A matching estimator finds some unexposed children whose characteristics are identical or closest to those of the exposed child k. The closeness is determined by a metric.

Formally[3], let $\left\|x\right\|_V = (x'Vx)^{\frac{1}{2}}$ be the vector norm with positive definite matrix V. We define $||z\text{-}x||_V$ to be 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. 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 $d_M(k)$. Every match of X_k must have the opposite treatment than unit k; if k is exposed, the matches are unexposed and vice versa.

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[2] See Imbens 2003 for a review on semiparametric estimation of average treatment effects, and Abadie and Imbens 2002 for a detailed discussion on the properties on the matching estimators we use in this paper.
[3] The following description draws from Abadie, Drukker, Herr and Imbens 2002.

Let $J_M(k)$ denote the set of indices for the matches for unit k that are as close as the M th match, 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 as follows:

$$\tau_M^t = \frac{1}{N_1} \sum_{k:E_k=1} \left(GH_k - \frac{1}{\#J_M(k)} \sum_{l \in J_M(k)} GH_l \right) \quad [9]$$

4.3 Health Determinants

Following the discussion on the choice of health determinants implied by the theoretical mode, we categorize the child health determinants into five categories in addition to pesticide exposure: birth health endowment -- whether the child had birth health problem; family resources -- parental education and family income; parental health-related behaviors; other environmental toxins including lead, and demographics including age, gender, nutritional adequacy, and health-related behavior such as smoking.

<u>Health Endowment</u>

We observe birth related data for children under age 11. We construct an indicator whether the child is endowed with poor health at birth, and set it to one if one of the following three cases was true: a) the child was born with physical or mental problems or defects, b) the child received newborn care in an intensive unit care, premature nursery or any other type of spherical care facility, or c) the child stayed in a hospital continuously after birth for one week or longer.

It is to note that birth health endowment may be correlated with parents' own exposure. If there is a positive effect of maternal exposure during pregnancy on a child's poor health endowment, and the mother was exposed directly or indirectly during pregnancy, then our estimates of the effect of parental exposure on a child's health after birth may be biased downward.

An alternative is to use parents' health at the birth of the child as proxy for a child's health endowment, because there is an intergenerational transmission of health stock where healthy parents are more likely to give birth to healthy children than unhealthy parents. But we only observe parents' current health not at the birth of the child.

Controlling for parents' current health can be problematic; for example, it may dilute the effect of their exposure on their children's health, since their health is determined by many factors including their own exposure to pesticides.

Ethnicity provides another alternative. The Hispanics in this survey represent three ethnicity groups from three regions: 1) Mexican Americans residing in selected counties of Texas, Colorado, New Mexico, Arizona and California; 2) Cuban Americans residing in Dade County in Miami, Florida; and 3) Puerto Ricans residing in the New York City area including parts of New Jersey and Connecticut. Mexican Americans may be systematically more or less healthy than Cuban Americans. We control for the ethnicity of the child.

Health-Related Behavior

We observe several behavioral indicators that may serve as materials or time inputs to the health production, such as whether mother or father smokes, uses drugs or is a heavy drinker, whether the child or parents are well-nourished, whether the child or parents have a regular place for checkups or emergence care, and how recent was the last routine checkup.

However, children's health service utilization may be endogenous. For example, a child with poor health or chronic conditions may be more likely to have a place for routine checkups or illness, or have a more recent routine checkup, than a child with good health or without chronic conditions. Inclusion of these health service utilization data would cause upward bias for the exposure effect and hence we exclude them from the regressions.

Other Environmental Toxins

Other environmental toxins besides pesticides may have adverse effects on their health. Our estimates would be biased upward if we do not control for a child's exposure to other toxins, which is positively correlated with their pesticide exposure.

We address this omitted variable issue using two sets of control variables. The first is the child's serum level of lead. Studies show that high serum level of lead adversely affects a

child's cognitive development. Few studies have examined whether higher serum lead level is associated with poor physical health, and furthermore, what is the relative strength of lead and pesticides exposure that contribute to a child's health outcomes. Previous studies show that lead exposure is associated with family income, and hence inclusion of lead may dilute the effect of family income. This is not a problem to us because our focus is to obtain unbiased estimate of the effect of environmental toxins, pesticides in particular.

Health shocks including other environmental toxins may vary in types and concentrations across geographical regions. Our second set of control variables intends to capture the heterogeneity of other environmental toxins using geographical data. We control for a) population density of where the child lives, since it is likely to be positively correlated with many other environmental toxins; b) whether the family lives in a standard metropolitan statistical area (MSA) and whether or not they live in central city, since health shocks in a central city may differ systematically from those in a non-central city; and c) ethnicity which also capture the major regions, since health shocks in California or Texas may differ from those in New York City.

4.4 Econometric Issues

Four econometric issues are of special concern. First, the parameter estimates of $\hat{\alpha}^D$ or $\hat{\alpha}^I$ would be biased if the child's exposure, direct or indirect, were endogenous. For example, children with poorer health may be less likely to help their parents on farms, and this would lead to a downward bias of $\hat{\alpha}^D$. On the other hand, sick children may be more likely to spend more time in the contaminated house or have a greater contact with exposed parents, which subject them to a greater level of indirect exposure; this would cause an upward bias of $\hat{\alpha}^I$. However, such variations are not reported in the pesticide exposure history data.

Second, the parameter estimates of $\hat{\alpha}^D$ or $\hat{\alpha}^I$ would be biased if the family fixed effect varies among the siblings. It may be plausible that parents value their children's health differently depending upon their health or age or other characteristics. For example, parents may have greater preferences for the younger child than for the older child or vice

versa, or parents may value the health of the sick child more than that of the healthy one. The potential heterogeneity of how children's health stock enters parents' utility function would lead to the heterogeneity in unobserved consumption or investment in health. However, the survey provides no additional information on such potential heterogeneity.

Third, the parameter estimates of $\hat{\alpha}^I$ in [6] would be biased if the family unobservable is correlated with some regressors, such as family income or parental education. Lastly, our estimates $\hat{\alpha}^D$ and/or $\hat{\alpha}^I$ are subject to omitted variable bias, that is, when the error terms are correlated with the regressors. There may be unobserved child-specific individual heterogeneity, such as personality traits, that result in both health and pesticide exposure. For example, sick children may be more likely to have poor eating habits or hygiene than the healthy children. We will assess the direction and magnitude of the omitted variable bias following the baseline estimates.

4.5 The Applicability of the Matching Estimator

There are two assumptions for the identification in the matching analysis (Imbens 2003): whether to be treated is independent of the outcomes and the probability of being treated is bounded away from 0 and 1, conditional on covariates *X*. We discuss how plausible these assumptions hold in this study.

The first assumption states that whether or not parents are occupationally exposed to pesticide is independent of their children's health. This assumption is not testable empirically. We cannot rule out the possibility that parents who had not been occupationally exposed to pesticides choose not engage in farming or other pesticide-related activities because their children are sick or having chronic conditions. Given that there is virtually no study on the adverse health effect of parental exposure to children, we think such a correlation is unlikely at least for the parents in the survey.

The second assumption states that the probability of being exposed is bounded away 0 and 1 conditional on covariates *X*. Equivalently, it requires an overlap in characteristics between the exposed and the unexposed. This assumption is testable by examining the distributions of covariates *X* for the exposed and unexposed groups. 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, though the variables may differ in the sample means. For example, parents of exposed children have on average significantly more years of schooling than parents of unexposed children as indicated by the t-stats in Table 1; the distribution of maternal education for exposed children resembles that for unexposed children, both range from 0 to17 and both have a mass point at 0.

5. The Results

We first examine whether there is an externality from parental occupational pesticide exposure, and whether the externality is a causal one by employing a variety of robustness analysis. Then we examine whether the burden of the externality is borne asymmetrically, in another word, whether some children are more vulnerable than others, where we define vulnerable as prohibitively high cost of mitigating the externality.

5.1 Does the Externality Exist?

We look at the relationship between household pesticide exposure and children's health by running two health indicators – good health and chronic conditions, using the random effect logit model [6] on all children. Table 2 reports the coefficient estimates and Table 3 contains the marginal effects of the logit estimates as well as the matching estimates. In all estimates, we control for child characteristics, family resources, parental health behaviors, ethnicity, and geographic characteristics of the household.

We find that indirect exposure has a large and significant effect on a child's chance of attaining good health and developing chronic conditions. The logit estimates indicate that exposed children are 5.8% less likely to attain good health than unexposed children, and the coefficient estimate is significant at 5% level. Exposed children are 4.8 % more likely to develop chronic conditions than unexposed children, and the coefficient estimate is significant at 1% level.

We use the matching estimator developed by Abadie et al. (2002), adjusting for biases arising from matching continuous covariates and accounting for heteroskedasticity. The matching estimates in Table 3 show that exposure reduces a child's' chance of attaining

good health by 6.4%, and increases a child's chance of developing chronic conditions by 7.1%; both effects are significant at 1% level.

The high significance levels of the estimates indicate there is a strong statistical association. Is 5.8% of economic importance, in the case of the attainment of good health, for example? Under our specification that health determinants are linearly and additively separable, exposure has the similar magnitude of the effect as having additional 10 ug/dl in serum, namely, changes from 10 ug/dl (approximately the sample average) to 20 ug/dl; exposure has the similar magnitude of the effect as having one fewer year of education for both mother and father.

5.2 Is the Externality Causal?

A randomized trial would be an ideal research design to assess the causality. Given the observational data, we employ a number of econometric techniques to assess whether the link is a causal one with a focus on three issues: functional form, unobservable, and omitted variables.

First, we use matching analysis to mitigate the misspecification of the functional form of the determinants of a child's health. A matching estimator allows for any interaction or high order terms of the original covariates.

Fixed effect logit estimates

Second, we employ a fixed effect logit model to address the issue of family unobservable and present the results in Table 4. These estimates are on older children between age 12 and 17, since we observe their own direct exposure.

Not controlling for the family unobservable increases the marginal effect of exposure by 30% on the probability of having chronic conditions – 7.2% by the random effect estimates versus 5.1% by the fixed effect estimates assuming zero family effect. Both estimates are significant at 5% level.

In general, marginal effects in a fixed effect logit model depend on the unobservable family effect. We do not observe the family effect and neither do we know its

distribution. We simulate by enumerating the sample average of the family effect and plot the results in Figure 2.

To determine what range of the family effect is relevant, we use the intercept estimate from the random effect logit model[4] as a proxy for the distribution of the fixed effect. We overlay the 95 percent confidence interval of the random effect intercept. For this interval, exposure effect is monotonically decreasing with family effect, ranging from 45% to 1%.

<u>Omitted variables biases</u>

Third, we wish to assess the direction and possibly the size of potential bias resulting from omitted variables that are correlated with the observable. We take the following approach. Suppose that an omitted variable *v* serves as a child's health determinant, and *v* is correlated with family income, for example. We wish to assess whether the exclusion of *v* would lead to an upward or downward bias in our estimate of the exposure effect.

Let $\hat{\alpha}^1$ and $\hat{\hat{\alpha}}^1$ denote the marginal effect estimates of exposure in the regressions with and without family income, respectively. If $|\hat{\hat{\alpha}}^1| \gg |\hat{\alpha}^1|$, it suggests that omitted variables correlated with family income would cause an upward bias, and that the difference between the magnitudes of $\hat{\alpha}^1$ and $\hat{\hat{\alpha}}^1$ may be indicative of the size of the bias.

Child's own health behavior

We observe whether older children ever smoked. In assessing the potential biases from omitting health behavior variables, we use three estimators: random effect logit, fixed effect logit and matching; and present the results in Table 4. The random effect logit estimates indicate that omitting bad health behavior would lead to a downward bias for the exposure effect, and that the size of bias may not be negligible. Inclusion of child's smoking increases the exposure effect by 30% from 7.2% to 8.4%, and the effect of smoking is large and highly significant (12.9%).

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[4] The intercept estimate from the random effect logit model is -.93 with robust standard error 1.26; the 95 percent confidence interval is (-3.40, 1.53).

Inclusion of child's smoking in the fixed effect model shifts the distribution of the exposure effect to the right (Figure 2). Using the intercept estimate from the random effect logit with child's smoking[5], we obtain the same qualitative results; that is, the marginal effect is monotonically decreasing over the 95 percent interval of the intercept estimate. Except for a small range of large negative family unobservable, omitting bad health behavior leads to a downward bias for the most parts of the range, and the magnitude of bias decrease as the unobservable increases. At zero unit of family unobservable, there is a considerable downward bias, 5.1% versus 23.5%.

The matching estimates, which assume that the family unobservable is uncorrelated with the observable, show little differences in the exposure effect. Inclusion of child's smoking reduces the exposure effect slightly, from 7.9% to 7.1%.

Overall, omitting child's own risky behavior is likely to cause a bias, and the bias is likely to be a downward bias, especially for those children with less favorable health environment.

Birth health endowment

We observe whether younger children under age 11 had a birth health problem. We run the random effect logit model [6] and matching specification [8] on the younger children, and present the results in Table 5.

On the effect on the chronic conditions indicator, the matching estimates give identical results with and without controlling for poor birth health. The logit estimates show a small upward bias for the exposure effect, 6.2% versus 5.7%. Poor birth health has a strong and significant effect of 7.4%. We see a mixed result on the good health indicator where the logit estimates show a positive bias and the matching estimates show a negative bias from omitting the poor birth health variable, and the differences are very small in both estimators.

Our results indicate that there may be a bias from omitting poor birth health, the bias may be negative, and the size of bias is likely to be negligible.

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[5] The intercept estimate from the random effect logit model with child's smoking is 1.22 with robust standard error 1.45; the 95 percent confidence interval is (-1.62, 4.06).

Family resources and parental behavior

The unobservable health determinants are likely to be correlated with family income, parents' education and health behavior. Parents' education and family income are important health determinants, evident in the SES-health gradient literature. Table 6 reports our analysis similar to that of birth health.

For the good health indicator, omitting either family resources or parental health behaviors would lead to an upward bias for the exposure effect. The logit estimates indicate omitting parental behavior would lead to a bigger bias than omitting family income (from −5.8% to −10.3% versus from −5.8% to −6.%). The matching estimates show an upward bias from omitting family income (from −6.4% to −8.7%) and no bias from omitting parental behaviors.

On the contrary, for the chronic conditions indicator, omitting either family resources or parental health behaviors would lead to a downward bias for the exposure effect. The logit estimates show small changes in exposure effect. The matching estimates show that the downward bias can be substantial. In particularly, omitting parental health behaviors would cause a larger bias than omitting family resources (from 7.1% to 3.7% versus from 7.1% to 5.7%).

We find that the health effect of serum lead is robust to omitting (current) family resource and parental behaviors in both health indicators. For every additional microgram per deciliter of lead in blood, a child is .5% less likely to attain good health and .2% more likely to develop chronic conditions.

Our analysis suggests that omitting important health important such as family resources and parental behaviors is likely to cause a bias, and size of the bias is not negligible in some cases. The exposure effect remains 10 times to 20 times of the serum lead effect, and the latter is robust to family resources and parental behaviors. We conclude that pesticide exposure has a substantial adverse health effect, and the effect is robust to omitted variables related to family resources and parental behaviors.

5.3 Is the Burden of the Externality Asymmetric?

Our second question asks whether some groups of children are more severely affected by the externality than others, such that public policy may target those most vulnerable children. We run the same matching estimations on subgroups by demographics and socioeconomic characteristics, and present the results in Table 7.

Differences by age, ethnicity and geographic locations

We first estimate by two age groups – younger children under age 11 and older children above age 11. We find that exposure effect is larger for older children than younger children. Exposure increases the chance of having chronic conditions by 7.9% for the older children and 5.8% for the younger children, and both estimates are significant at one-percent levels. Exposure reduces the chance of attaining good health by 10% for the older children and 7% for the younger children, and both estimates are significant at five-percent levels.

There may be several explanations to the difference by age. One is that pesticide exposure has an accumulative effect on children's heath. Another explanation could be that the health effect of pesticide exposure is delayed, such that the effect is subtle and parents do not observe the symptoms at the early stage. A third one could be omitted variable bias; for example, older children spend more time helping their parents on the pesticide-related activities and thus are more intensely exposed. In this case, the observed difference by age reflects the difference in intensity of exposure. Another alternative is reporting errors where the parents reported younger children's health outcomes and older children reported their own.

Next we estimate by three ethnicity groups: Mexican Hispanics, Cuban Hispanics and Puerto Rican Hispanics. We find that the effect of exposure is most evident on the Mexican Hispanic children. Exposure reduces the chance of having good health by 7.4% and increases the chance of having chronic conditions by 7.4% for the Mexican Hispanics, and both estimates are highly significant; while the estimates are smaller and insignificant at the conventional significant levels for the other ethnicity groups.

There are several explanations to the difference by ethnicity. One is that Mexican children experience more intense exposure than Puerto Rican Hispanic children in the New York City or Cuban Hispanic children in Florida, because farming is more intense in where they live – California, Texas, Colorado, New Mexico and Arizona. In this case, the difference in ethnicity reflects the difference in the intensity of the exposure. Another explanation is the geographic variation in health determinants other than pesticide exposure, such as other environmental toxins, cultural preferences for children's health, and health care systems. Another alternative is the genetic susceptibility where the Mexican Hispanic children are more susceptible to the toxicity of pesticide than other Hispanic children. The difference by ethnicity is unlikely due to the symmetric reporting errors since the survey is a nationwide probabilistic sample.

Differences by income and education

We examine whether children from different socioeconomic status bear different degree of the health externality of pesticide-related activities. We focus on family income and parents' education. We estimate two groups of children by whether their (log) per capita income exceeds the sample average. We find that the effect is concentrated on poor children. Exposure increases the chance of chronic conditions by 8.4% and reduces the chance of attaining good health by 10% for the poor children. Exposure effect is of one-fifth magnitude and statistically insignificant for non-poor children for the good health indicator, and of two-thirds magnitude for the chronic conditions indicator.

Next we divide the sample by whether their fathers' education exceeds the sample average. We find that the heterogeneity of exposure effect by father's education is more evident in chronic conditions indicator than in good health indicator. Exposure increases the chance of developing chronic conditions by 10.4% for children whose fathers have below (sample) average education, and by 7.4% for children whose fathers have above average education. Both estimates are highly significant.

The asymmetry is more evident when we estimate two groups of children by whether their mothers' education exceeds the sample average. Exposure effect for children with less educated mothers is more than twice in magnitude the effect for children whose

mothers are better educated – 12.2% versus 5.5%; both estimates are significant at one-percent levels.

Our estimates by socioeconomic status show that poor children and children with less educated parents are more vulnerable to health shocks resulting from environmental toxins such as pesticide than non-poor children and children with better educated parents, to the extent that family income and parental education fail to provide protection for those health shocks.

Are the differences by socioeconomic status largely due the unobservable?

In answering our first question we examine our findings are robust to the unobservable, and especially the unobservable that is correlated with socioeconomic characteristics. We perform the same omitted variable analysis for subgroups partitioned by socioeconomic status, and results are shown in column (2) and (4) in Table 7.

Comparing the results with and without income in the first two columns, we find that income related family unobservable has a more pronounced effect on poor children than on non-poor children in attaining good health. Omitting income related unobservable would bias the estimate of exposure for poor children by approximately one quarter. We find virtually no effect from omitting income related unobservable on a child's chance of having chronic conditions, once their income is accounted for separately by the sample average. Taken together, the disparate vulnerability by income is robust to income related family unobservable.

We see similar or even stronger evidence when we examine omitted variables bias related to parents' education. We focus our discussion of the effect on chronic conditions. Omitting parents' education leads to a *downward* bias once their fathers' education is accounted for separately by the sample average. The downward bias is greater for children with less educated fathers (nearly 14%) than children with better-educated fathers (nearly 8%).

This counter-intuitive finding has two implications. First, better education by fathers provides no additional protection in mitigating the health risk resulting from pesticide exposure, particularly, on their children's developing chronic conditions; furthermore, the

unobservable correlated with better education by fathers increases the health risk by pesticide exposure. One explanation is that father's education may be associated with farming and other pesticide related jobs, for example, educated fathers may be more likely to be employed on farm than non-educated fathers; consequently, they are more exposed and bring home more pesticide to their children. Second, the family unobservable that is correlated with father's education has an adverse effect on a child's developing chronic conditions, and furthermore, the adverse effect is more pronounced for children with less educated fathers than children with better educated fathers.

Repeating the analysis on maternal education, we find that the disparity of vulnerability exists and widens considerably when we account for the family unobservable related to maternal education. Omitting maternal education related variables leads to an 8% downward bias for children with less educated mothers, and a 25% upward bias for children with better educated mothers. This result suggests that maternal education may have a threshold effect. When mothers attain certain level of schooling, their human capital and associated unobservable mitigate children's health risks resulting from environmental toxins. When mothers fail to attain certain levels of schooling, the unobservable associated with their human capital exacerbate the health risks caused by pesticide exposure.

Alternative explanation to the SES-gradient

We find strong evidence that children from low socioeconomic status are more severely affected by health shocks such as pesticide related environmental toxins than non-poor children. Our findings provide an alternative explanation on the SES-gradient, which states that the inverse relationship between income and children's health is more pronounced with age. Currie and Stablie (2003) attributes the SES-gradient to that poor children experience more health shocks. Our analysis suggests that the observed SES-gradient may be attributable to the heterogeneity in the severity of effects from adverse health shocks, as well as the heterogeneity in the degree that the family unobservable that is correlated with socioeconomic status mitigates or exacerbates the health shocks.

The externality and poverty

Case, Fertig and Paxson (2003) calls for more attention to be paid to health as a potential mechanism through which intergenerational transmission of poverty takes place: cohort members born into poor families experienced poor childhood health, lowered investment in human capital and lowered earnings at middle age when they become parents themselves. Our findings suggest that sources of health shocks such as pesticides and other toxins may be contributing to the increasing poverty at the very poor. Poor individuals are more severely affected by adverse health shocks at childhood than non-poor individuals. The greater vulnerability to childhood health shocks is associated with greater reduction in their earnings as parents, which makes their children poorer compared to the children of those who were not poor in their childhoods. Reducing sources of health shocks may help reduce the increasing income inequality.

5.4 Policy Implications

The economic cost of poor childhood health is substantial. Case, Fertig and Paxson (2003) find that poor childhood health persists over the life span, 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 poor health status at age 23 reduces log earnings by 50% at age 33 and 14.3% at age 42. They find each childhood chronic condition reduces log earnings by 7%.

For the sample children, we construct an estimate of the cost of childhood pesticide exposure as follows. We use 7% as the effect on a child's increased chance of developing chronic conditions, and 7% as the reduction in earnings due to poor health or chronic conditions in childhood. The average Hispanic annual income is approximately $30,000. The economic cost of childhood pesticide exposure is $20,000 per child, and $76,000 per family with an average 3.82 children per Hispanic family with 5% discounting rate.

These benefit estimates do not include medical expenditures or loss of income of parents who need to take care of sick children, which can be equally or more substantial than those from the lasting effect on earnings. In addition, children from low socioeconomic

status are more severely affected, and consequently, they incur higher costs from the adverse health shocks.

There are several policy instruments: income transfer, terminating secondary exposure pathway, 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 or other pesticide-related jobsites provide sanitary facilities such that farmers who have children at home must be cleaned before leaving the field contaminated with pesticide and other related toxins. Frisvold, Mines and Perloff (1988) report that lack of field sanitation on agricultural job sites increases the probability of agricultural workers reporting gastrointestinal disorders by 60%. The marginal cost of providing such a facility is of several magnitudes smaller of the benefit of correcting the externality on per farm worker basis.

6. Conclusion

In this paper, we estimate the health effect of children's chronic exposure to pesticide and related environmental toxins, which are brought home from their parents who engage in pesticide related activities. We find strong empirical evidence that the externality of parental occupational pesticide exposure exists, and that the externality is a causal one. We find that poor children and children with less educated parents bear disproportionably more burden of the externality. Our cost benefit analysis suggests that terminating the pathway of parental exposure can be an effective mechanism to correct the externality. Our findings have a broader implication on reducing the increasing poverty of the poor: poverty makes individuals more susceptible to health shocks at childhood, which is associated with increasing poverty for their children.

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Table 1. Summary Statistics

	All (1)	Unexposed (2)	Exposed (3)	t-stat (4)
Good health	.488 (.500)	.518 (.500)	.459 (.498)	3.04
Chronic conditions	.148 (.355)	.139 (.346)	.159 (.366)	1.44
Serum lead	10.427 (5.453)	10.433 (5.958)	10.420 (4.725)	-.08
Adequate nutrition	.99 (.07)	.99 (.05)	.99 (.09)	.00
Age	8.800 (4.84)	8.301 (4.752)	9.446 (4.883)	6.10
Female	.492 (.50)	.502 (.500)	.482 (.500)	-22.22
Mother education	5.607 (5.32)	4.430 (5.313)	7.127 (4.67)	13.99
Father education	3.994 (5.47)	2.589 (4.839)	5.808 (5.59)	18.74
log (per capita income)	8.217 (1.204)	8.266 (1.232)	8.154 (1.164)	-2.41
Parents have a medical place	.558 (.497)	.419 (.493)	.739 (.440)	17.75
Parents' last routine checkup	.685 (.464)	.514 (.500)	.906 (.291)	25.47
Parents' nutritional adequacy	1.018 (.817)	.719 (.797)	1.404 (.669)	24.22
Mexican Hispanic	.652 (.476)	.617 (.486)	.697 (.460)	4.36
Cuban Hispanic	.127 (.333)	.094 (.292)	.169 (.375)	5.65
Population density	4.247 (2.389)	4.068 (2.342)	4.479 (2.430)	4.42
Sample Size	2692	1517	1175	

Notes: Un-weighted sample means are shown in the table with 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 reported by the child. Parents' education is measured in years of completed schooling. Serum level of lead is measured in microgram per deciliter. Adequate nutrition is an indicator assessed by physicians. Last routine checkup is a categorical variable where a smaller value indicates a most recent utilization. Column (4) is t-stat under the null that the sample average of exposed children equals to the sample average of unexposed children.

Table 2. Logit estimates

Dependent Variable	Good health	Chronic conditions
Exposure	**-.233** (.111)**	**.397*** (.146)**
Serum lead	**-.020** (.010)**	**.015 (.009)**
Mother education	.097*** (.015)	-.020 (.019)
Father education	.089*** (.019)	.005 (.022)
log (per cap income)	-.470 (.570)	-.069 (.055)
Being a Mexican	-.055 (.123)	-.870 *** (.148)
Being a Cuban	.594*** (.190)	-.267 (.219)
MSA central	-.260 (.363)	-.460 (.415)
MSA not central	-.539* (.301)	-.386 (.336)
Pop Density	.071* (.038)	-.039 (.047)
Father smoked	-.186 (.148)	.286 (.212)
Mother smoked	.008 (.102)	.217* (.122)
Father drank	.062 (.108)	.031 (.145)
Mother drank	-.185** (.086)	-.090 (.101)
Parents have a medical place	.195 (.149)	.193 (.203)
Parents routine checks	-.686*** (.254)	.055 (.296)
Parents nutrition	-.702*** (.206)	.040 (.212)
Nutrition	-.470 (.570)	.309 (.845)
Age	.029*** (.009)	.022* (.013)
Wald stat (20)	117.29	65.01
Pseudo R^2	.04	.07

Notes: Estimates are on all children under age 17 using logit specification [6] using random effect estimators with the identical set of regressors. Robust errors are in parentheses. Sample size is 2692.

The exposure variable is an indicator whether anyone in the household is exposed. Regressors include child's age, sex, serum level of lead in ug/dl, child's nutrition status assessed by the physician, log of per cap family income, mother's and father's education in years of schools completed, whether the parents have place for regular medical checkups and emergence, and how recently the parents had routine checkups, whether mother (father) ever smoked, whether mother (father) was ever a heavy drinker, population density of where the family lives, ethnicity indicator whether the child is a Mexican-American, or a Cuban-American (indicator Puerto Rican is omitted), whether the family lives in a SAMA and/or central city.

*** Indicates 1% significance level, ** indicates 5% and * indicates 10% significance level.

Table 3.Marginal effects of logit and matching estimates

Dep. Var	Good health		Chronic conditions	
	logit	Matching	logit	Matching
Exposure	-.058**	-.064*** (.027)	.048***	.071*** (.019)
Serum lead	-.005**		.002	
Mother education	.024***		-.002	
Father education	.022***		.000	

Notes: Marginal effects of the logit estimates are calculated at the sample averages for one unit increase using the estimates in Table 3. Standard errors of the logit marginal effects are not presented. Matching estimates are adjusted for biases and heteroskedasticity (Abadie et al. 2002). Robust errors of the matching estimates are in parentheses. ** Indicates the coefficient estimate is significant at 5%, *** indicates the coefficient estimate is significant at 1%.

Table 4. Fixed effect logit and role of child's own health behavior

Chronic	Random effect		Fixed effect		Matching	
Exposure	.072**	.084**	.051**	.235**	.079*** (.032)	.071*** (.031)
Include Smoke?	No	Yes .129***	No	Yes	No	Yes

Notes: Presented are marginal effects. Dependent variable is chronic condition indicator. Random effect logit refers [6] and fixed effect logit refers to [7]. Marginal effects for fixed effect logit are calculated at the sample averages with sample average of family effect being zero. All estimates are on older children aged between 12 and 17. Sample size for random effect logit and matching estimates is 899. Sample size for fixed effect logit estimates is 112. Smoke is an indicator whether the child ever smoked. Coefficient estimate of smoking is significant at 1% in random effect and insignificant in fixed effect model.

Table 5. Role of birth health endowment

Dependent variables	Good health				Chronic conditions			
Estimators	logit		Matching		logit		matching	
Exposure	**-.077****	**-.073****	**-.070***** (.035)	**-.079***** (.036)	**.062*****	**.057*****	**.059***** (.023)	**.059***** (.023)
Serum lead	-.003	-.003			.001	.001*		
Mother education	.026***	.010***			-.005	-.014		
Father education	.027***	.025***			.000	-.005		
Include Poor birth health?	No	Yes, -.045	No	Yes	No	Yes, .074***	No	Yes

Notes: Presented are the marginal effects calculated at the sample averages using random effect logit model [6] on younger children under age 11. Sample size is 1775. All estimates use the identical set of regressors as in Table 3 with the exception of poor birth health variable, as indicated. Standard errors of the logit marginal effects are not presented. Robust errors of the matching estimates are in parentheses. ** indicates the coefficient estimate is significant at 5%, *** indicates the coefficient estimate is significant at 1%.

Table 6. Role of family resources and parental behavior

Dep. Variables	Good health						Chronic conditions					
Estimators	logit			Matching			logit			matching		
Exposure	**-.058****	**-.069*****	**-.103*****	**-.064***** (.027)	**-.087***** (.028)	**-.064***** (.026)	**.048*****	**.049*****	**.044*****	**.071**** (.019)	**.057***** (.020)	**.037**** (.019)
Serum lead	-.005**	-.006**	-.005**				.002*	.002***	.002*			
Include resources	Yes	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes	No	Yes
Include behavior	Yes	Yes	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes	No

Notes: Presented are the marginal effects calculated at the sample averages using random effect logit model [6] on all children under age 17. Sample size is 2692. All estimates use the same set of regressors as in Table 4 unless specifically indicated otherwise. Resources include log per capita income, mother education and father education. Parental behavior contains seven regressors: parents' access to medical services and their preventive use of medical services, parents' nutritional adequacy, father (or mother) ever smoke or was a heavy drinker. Standard errors of the logit marginal effects are not presented. Robust errors of the matching estimates are in parentheses. ** Indicates the coefficient estimate is significant at 5%, *** indicates the coefficient estimate is significant at 1%.

Table 7: Matching estimates by subgroups

Dependent Variable	Good health		Chronic conditions	
	(1)	(2)	(3)	(4)
Age 1-11 [1780]	-.070** (.035)		.058*** (.023)	
Age 12-17 [908]	-.100** (.048)		.079*** (.033)	
Mexicans [1755]	-.074*** (.032)		.074*** (.022)	
Cubans [342]	-.050 (.070)		.020 (.053)	
Puerto Ricans [595]	-.053 (.065)		.069 (.057)	
Below average income [1530]	-.100*** (.035)	-.127*** (.037)	.084*** (.029)	.082*** (.029)
Above average income [1165]	-.022 (.038)	-.015 (.038)	.059** (.028)	.059** (.026)
Include income?	Yes	No	Yes	No
Below average father education [1684]	-.097*** (.036)	-.113*** (.037)	.104*** (.028)	.089*** (.028)
Above average father education [1008]	-.091*** (.038)	-.080*** (.042)	.074*** (.027)	.068*** (.027)
Include parents' education?	Yes	No	Yes	No
Below average mother education [1324]	-.053 (.043)	-.008 (.045)	.122*** (.027)	.112*** (.027)
Above average mother education [1371]	-.085*** (.031)	-.115*** (.032)	.055*** (.021)	.069*** (.022)
Include parents' education?	Yes	No	Yes	No

Notes: All estimates use an identical set of regressors as in Table 3 unless otherwise indicated. The square bracket next to the heading in each row is sample size. *** Indicates 1% significance level, ** indicates 5% and * indicates 10% significance level.
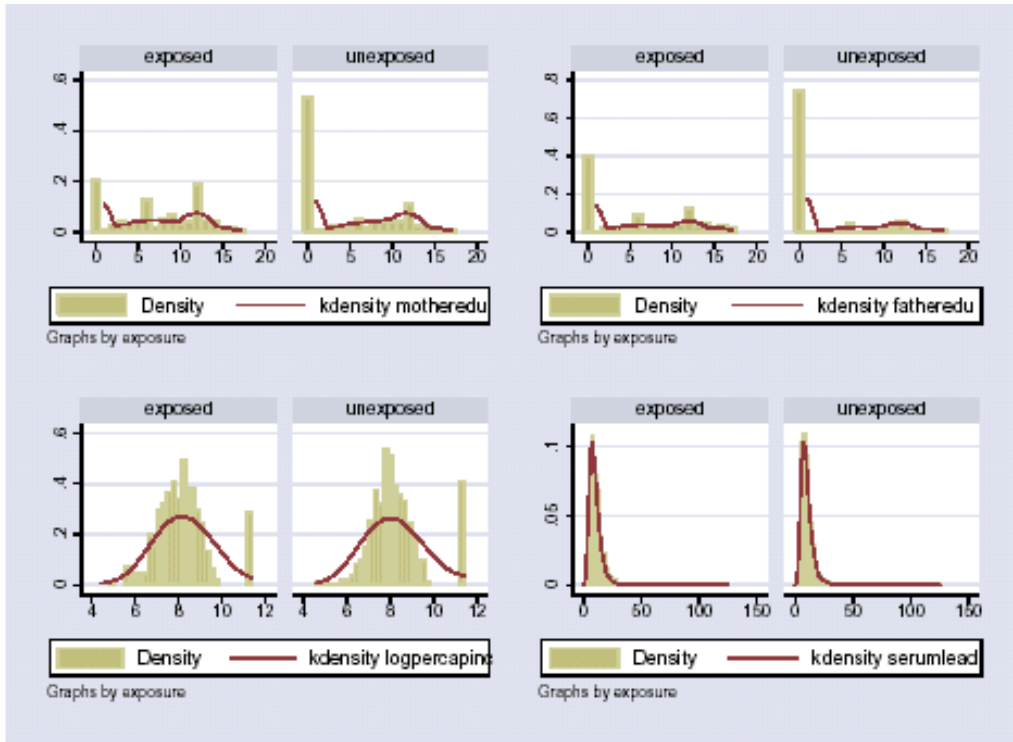
Figure 1: Distributions of key variables by exposure status

Figure 2: Marginal effects as a function of family effect

Exposure Effect on Chronic Conditions with & without Smoking