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# Women's Health Choices and the Effects on Child Health

Therése Hindman Persson\*

## Abstract

This paper analyses, both theoretically and empirically, women's health choices and their effects on child health for a sample of rural households in Cebu, Philippines. The present study differs from other studies by analysing separately prenatal and postnatal determinants of child health both under certainty and uncertainty, hence introducing the possibility of different health production functions before and after birth. An approach that is well in line with recent research in nutrition and epidemiology. Theoretically, the model predicts that the larger the probability of survival the less is spent on child specific health inputs after birth. That is, the less money need to be spent on compensating the child for a "bad start" in life. Empirically, the results show that family specific health endowments may explain a large part of the child's health, that water and sanitation are important for child health, and that smoking has a significant and negative effect on the health of the child after birth.

JEL classification: I12, I31, O15

Keywords: Philippines, Child Health, Health Production Function, Uncertainty, Water and Sanitation

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# 1 Introduction

Determinants of child mortality and morbidity in developed and developing countries have been studied thoroughly, and the literature is full of examples and estimates of child health production functions (e.g. Guilkey et al., 1989; Cebu Study Team, 1992; Lee et al., 1997; Kovsted et al., 1999; Currie, 2000). Most of the earlier studies employ the production function approach to child health as it was formulated by Rosenzweig and Schultz (1983a,b) although there have been attempts to model the biology underlying child health as well (Strauss & Thomas, 1995). One reason for focusing on infant/child health is of course that children, by being the future labour force, provide the foundation for future development and growth of the economy. Today, there is growing evidence that in utero conditions have a significant impact on future health outcomes (e.g. Mongelli & Gardosi, 2000). This theory is based on the early statistical analysis carried out by Barker of low birthweight data collected in the early 1900's in the south east of England, and the theory has therefore been called the "fetal origins" or the "Barker Hypothesis" (Lucas et al., 1999). In addition, it is well known that individual health can have impacts on several economic outcomes since the health status of a labourer is an important labour characteristic thus affecting the wage rate. However, health is also an input in the household firm/farm production function (Behrman and Deolalikar, 1988) which implies that household income and welfare are both directly and indirectly affected by the positive effects of better health. In addition, a good health is of vital importance for human capital accumulation and it affects children's schooling productivity in much the same way as it affects labour productivity among adults (e.g. Moock & Leslie, 1986; Jamison, 1986; Currie, 2000).

In this paper, the aim is to analyse women's health choices and their effects on child health for a sample of rural households in Cebu, Philippines. The present approach differs from other studies by analysing separately those determinants of child health acting before the child is born from those determinants acting after the child is born, thus allowing

for different health production functions before and after the birth of a child.<sup>1</sup> In other words, it is assumed that a woman's health-related actions might differ before and after child birth, and that the delivery plays a central role for a child's health prospects. This assumption is not controversial since numerous gender-related factors affect health-status and health care practices in developing countries i.e., factors associated with different traditional roles played by men and women and/or factors related to access to resources and levels of income (Development Studies Unit, 1995). For example, a woman's health-related actions after the child is born might differ depending on the sex of the child.

In addition, special attention is paid to the role of water and sanitation related inputs (WSI) for the production of health. The World Health Organization (WHO, 1995) estimates that about 3,6 million people die annually and about 1,5 thousand million suffer at any one time from infections stemming from unsanitary excreta disposal and poor personal and domestic hygiene. Every year more than two million children die from diarrhoea that could have been prevented by good sanitation and many more suffer nutritional, educational and economic losses because of diseases that improved sanitation can prevent (SAN 1995).

”(...) and the provision of safe drinking water and adequate sanitation is arguably the most important single contributor to a population's improved health status.” (Traoré, 1992: p. 3.7)

*Table 1* presents a classification of water and sanitation associated diseases. The four categories contain diseases that are common in poor countries and that can be prevented or minimised by the introduction of safe water supplies and adequate sanitation facilities. However, one should also bear in mind the illnesses resulting from arsenic poisoning and the nitrates, heavy metals, and pesticides which are the side products of the industrialised societies, and often pollutants of drinking water sources. Earlier studies (e.g. Lee et al., 1997; Van Poppel & Van der Heijden, 1997; Jalan & Ravallion, 2001) have shown that

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<sup>1</sup>This separation is in line with recent research in nutrition and epidemiology, e.g. Lucas (1999) and Rasmussen (2001).

improved sanitation as well as water quality may be important determinants of children's health. However, since it has been difficult to establish significant and conclusive results in these earlier studies, it is important to further analyse the effects of improved water and sanitation on child health.

Table 1.

Classification of water and sanitation associated diseases	
<i>Disease</i>	<i>Description</i>
Water-borne diseases	These diseases are caused by organisms that can survive in water and that can be ingested when contaminated water is drunk. Examples are typhoid, cholera, hepatitis, and shigellosis.
Water-washed diseases	These diseases are caused by the scarcity of accessible water supplies and they can cause diarrhoea and contagious skin and eye infections.
Water-based diseases	Some parasitic worms pass part of their life cycle in intermediate host organisms that live in fresh water. An example is Guinea worm, and people are infected by drinking water containing contaminated water crustaceans called Cyclops. Most water-based diseases are due to trematodes or flukes that have developmental stages in aquatic snails, for example schistosomiasis.
Water-associated vector-borne diseases	In this case, water provides a habitat for insect vectors of disease. Mosquitoes, for example, need water for part of their life cycle.

Source: Traoré (1992)

## 2 Theoretical Framework

Like most of the earlier studies, the present study employs the production function approach to child health as proposed by Rosenzweig and Schultz (1983a,b). Some of the recent developments of this approach (e.g. Guilkey et al., 1989; Cebu Study Team, 1992; Lee et al., 1997; Kovsted et al., 1999; Currie, 2000) are incorporated and, in addition, some new elaborations are proposed. For example, separate analyses of prenatal and postnatal determinants of child health both under certainty and uncertainty, which is in line with recent research in nutrition and epidemiology, e.g. Lucas (1999) and Rasmussen (2001).

When analysing consumer behaviour, the main objective is to explain the level of

demand for the commodities that an individual consumes, given assets, prices, and individual- and community endowments. A traditional approach to consumer choice is adopted and child health is viewed as a consumption commodity from which the household derives utility. There is, however, no market for child health and, therefore, the household acts as a producer of this commodity as well. Health is analysed at the household level since the immediate determinants of an individual's health are decisions made by the individual or by the household in which he or she lives (Behrman & Deolalikar, 1988). For the present analysis a unitary household model is employed<sup>2</sup>, and this model assumes that the household maximises a single utility function subject to a set of constraints and that all income and factor supply is pooled in the household.

According to Bergstrom (1995), the unitary model can be theoretically justified in at least four cases:

- If preferences in a household are convex and if indirect utility is of the Gorman polar form<sup>3</sup>, i.e. if it can be expressed in the form  $V_j(p, M_j) = \alpha(p)M_j + \beta_j(p)$  for each individual/household  $j$  for some function  $\alpha(p)$  and functions  $\beta_j(p)$ , then an efficiently operating household will act as if all household decisions were made by a single utility maximising consumer.<sup>4</sup>  $M_j$  is individual/household income.
- If income distribution within the family is itself the result of an optimising choice, i.e. there is a benevolent dictator, then even for very general individual preferences, aggregate household demand will behave as if it is the demand of a single maximiser.
- Even if the family is *not* a dictatorship, it might be that preferences of different members are interrelated by a "consensus" or "social welfare function" which takes into account the consumption levels of each of the members. The family act as if

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<sup>2</sup>For a review of models see Doss (1996).

<sup>3</sup>If indirect utility has this form then the income-consumption paths of all consumers are parallel straight lines. This implies that everyone allocates a marginal dollar of income in the same way (Bergstrom, 1995).

<sup>4</sup>See also Mas-Colell et al. (1995):116-120.

it were maximising their joint welfare function, i.e. the Bergson-Samuelson social welfare function.

- If each family member always gets the same fraction of income and if all family members have homothetic, but not necessarily identical, preferences then family demand can be rationalised as the choice of a single individual.

Empirically, Bourguignon et al. (1993) and Thomas (1990) have challenged the assumption that only total income should matter in consumption decisions, i.e. the assumption that "who earns what" is irrelevant. However, according to Doss (1996) the finding that individual labour income affects household expenditures, holding total household income constant, is not enough to reject the unified model. The unified model only predicts that exogenous income, i.e. income that does not alter the marginal productivity of individuals within the household, should be spent in the same manner. In addition, Doss (1996) asserts that in some instances, e.g. the Philippines, the unified model and the co-operative bargaining model are observationally equivalent.

## 2.1 The Model

Following Kovsted et al. (1999), each household,  $j$ , has a preference ordering over a non-health composite consumption commodity,  $X_j$ , a composite health environment commodity<sup>5</sup>,  $Y_j$ , and a vector of child health,  $\mathbf{H}_j$ , that can be represented by<sup>6</sup>

$$U_j = U(X_j, Y_j, \mathbf{H}_j), \quad (1)$$

where the marginal utilities of all arguments in the utility function are assumed positive but decreasing.  $\mathbf{H}_j = (H_{01j}, H_{1j})$  where  $H_{01j}$  is the health of the child, i.e. child one (1), at birth and  $H_{1j}$  is the health of the child after birth. To shorten the notation it is assumed that the woman is giving birth to one child.<sup>7</sup> The prenatal child health

<sup>5</sup>This commodity can include measures of drinking water quality and sanitary facilities as well as smoking etc.

<sup>6</sup>The household utility function is defined over the commodity space.

<sup>7</sup>Results when the model allows for the possibility of multiple births are available upon request.

production function is given by

$$H_{01j} = \Psi(Y_j, \eta_j, \mu_{1j}), \quad (2)$$

that is, child health at birth is assumed determined by the composite health environment commodity,  $Y_j$ , which before birth can be represented by health habits of the mother, e.g. medical care, smoking, alcohol consumption etc., the health status/history of the mother,  $\eta_j$ , e.g. nutritional status, healthiness during pregnancy, number of past pregnancies etc., and  $\mu_{1j}$ , a family-specific child health endowment, including e.g. the genetic make-up, that is known, but not controlled, by the family. It is assumed that once the child is born,  $H_{01j}$  is taken as given. Following Rosenzweig & Schultz (1983a,b), the production of child health after birth is given by the health production function

$$H_{1j} = \Gamma(H_{01j}, Y_j, I_{1j}, \mu_{1j}), \quad (3)$$

that is, the production of child health is determined by the health at birth,  $H_{01j}$ , the composite health environment commodity<sup>8</sup>,  $Y_j$ , a child specific health input,  $I_{1j}$ , that does not augment utility other than through the effect on child health, and a family-specific child health endowment that is known, but not controlled, by the family,  $\mu_{1j}$ . The marginal product of  $I_{1j}$  is assumed positive but decreasing.

The functional forms of (2) and (3) have interesting implications for the model (e.g. Bergstrom, 1995; Rosenzweig & Schultz, 1983b; Pollak & Wachter, 1975). If constant returns to scale and no joint production is assumed, i.e. a linear boundary of the production possibility set, then the model allows for a separation of the production and consumption activities. If, however, the assumption of no joint production is relaxed, as it is in the present study, then it is possible to allow for the realistic case that the composite health environment commodity both affects child health and contributes to utility directly, e.g. smoking. In addition, Kovsted et al. (1999) have noted that if the health production function is additive in  $\mu_{1j}$  the interpretation is that parents can compensate a child with low endowments by increasing health inputs. If, on the other hand,

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<sup>8</sup> *Goods* are inputs into the production of *commodities*.

the production function is multiplicative in  $\mu_{1j}$  then children with higher endowments benefit more from a given level of health inputs. The prenatal budget constraint facing the household is expressed as

$$M_j = X_j + p_Y Y_j, \quad (4)$$

where  $p_Y$  is the price, or cost, of the mother's health habits, e.g. cost of cigarettes, alcohol, medical care etc., and  $X_j$  is the non-health consumption commodity. Consequently, the postnatal budget constraint facing the household is expressed as

$$M_j = X_j + p_Y Y_j + p_I I_j, \quad (5)$$

where  $M_j$  is household income and  $p_Y$  and  $p_I$  are the prices of the health environment commodity and the child specific health input relative to the price of  $X_j$  i.e. the non-health consumption commodity. Consequently, The maximisation of (1) subject to (2) or (3) and (4) or (5) gives the reduced form household demand for  $X$ ,  $Y$  and  $I$  as functions of prices, income and the family-specific child health endowment. Two illustrative examples of possible specifications of the child health production function when  $\mu_{1j}$  is multiplicative/additive follows below.

#### CASE 1 *Cobb-Douglas utility and multiplicative endowment*

Assume first that both the prenatal utility and health production function are of the Cobb-Douglas type

$$U_j = X_j^a Y_j^b H_{01j}^c, \quad (6)$$

$$H_{01j} = Y_j^d \eta_j^e \mu_{1j}^f, \quad (7)$$

where  $a, b, c, d, e$  and  $f$  are constants and  $d + e + f = 1$ . Hence, the production function exhibits constant returns to scale and joint production. Equation (6) can be rewritten as

$$U_j = X_j^a Y_j^b (Y_j^d \eta_j^e \mu_{1j}^f)^c = X_j^a Y_j^{b+dc} \eta_{1j}^{ec} \mu_{1j}^{fc}. \quad (8)$$

As before, the budget constraint facing the household is expressed as

$$M_j = X_j + p_Y Y_j. \quad (9)$$

Assume for simplicity that  $a + (b + dc) + ec + fc = 1$ , then maximisation of (8) subject to (9) gives us the following familiar reduced form demand functions:

$$X_j = \frac{a}{1 - fc} M_j, \quad (10)$$

$$Y_j = \frac{b + dc}{1 - fc} \frac{M_j}{p_Y}. \quad (11)$$

The postnatal case gives a similar result. Given the same conditions as stated above, the utility function can be described by  $U_j = X_j^a Y_j^b \left( H_{01j} Y_j^d I_{1j}^e \mu_{1j}^f \right)^c = X_j^a H_{01j}^c Y_j^{b+dc} I_{1j}^{ec} \mu_{1j}^{fc}$  and the budget constraint by  $M_j = X_j + p_Y Y_j + p_I I_j$ . Since  $\mu_{1j}$  is multiplicative, children with higher endowments benefit more from a given level of health inputs.<sup>9</sup> In addition, since  $H_{01j}$  is given once the child is born it is introduced as a scale parameter like the technical coefficient in a standard Cobb-Douglas production function. The reduced form demand functions for  $X_j$ ,  $Y_j$ , and  $I_{1j}$  are thus given by:

$$X_j = \frac{a}{1 - fc} M_j, \quad (12)$$

$$Y_j = \frac{b + dc}{1 - fc} \frac{M_j}{p_Y}, \quad (13)$$

$$I_{1j} = \frac{ec}{1 - fc} \frac{M_j}{p_I}. \quad (14)$$

#### CASE 2 *Cobb-Douglas utility and additive endowment*

Assume now that the prenatal utility function is of the Cobb-Douglas type but that the health production function is additive in  $\mu_{1j}$ :

$$U_j = X_j^a Y_j^b \mathbf{H}_j, \quad (15)$$

$$H_{01j} = Y_j + \eta_j + \mu_{1j}, \quad (16)$$

where  $a$  and  $b$  are constants and  $a + b = 1$ . Hence, the production function does not exhibit constant returns to scale although there is still joint production. Equation (15) can be rewritten as:

$$U_j = X_j^a Y_j^b (Y_j + \eta_j + \mu_{1j}). \quad (17)$$

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<sup>9</sup>  $\mu_{1j}$  affects the marginal productivity of  $I_{1j}$ :  $MP_{I_{1j}} = \frac{\partial H_{1j}}{\partial I_{1j}} = e H_{01j} Y_j^d I_{1j}^{e-1} \mu_{1j}^f$

As before, the budget constraint facing the household is expressed as:

$$M_j = X_j + p_Y Y_j. \quad (18)$$

Maximisation of (17) subject to (18) gives us the following reduced form demand functions:

$$X_j = (1 - b)M_j, \quad (19)$$

$$Y_j = b \frac{M_j}{p_Y}. \quad (20)$$

Given the same conditions as stated above, the postnatal utility function can be described by  $U_j = X_j^a Y_j^b H_{01j}(Y_j + I_{1j} + \mu_{1j})$  and the budget constraint by  $M_j = X_j + p_Y Y_j + p_I I_j$ . Since  $\mu_{1j}$  is additive, children with low endowments can now be compensated by their parents through the increase of health inputs. The reduced form demand functions for  $X_j$ ,  $Y_j$  and  $I_{1j}$  are thus given by:

$$X_j = -a \frac{M_j + p_I \mu_{1j}}{p_I b - 2p_Y} p_Y, \quad (21)$$

$$Y_j = -b \frac{M_j + p_I \mu_{1j}}{p_I b - 2p_Y}, \quad (22)$$

$$I_{1j} = -\frac{p_I b Y_j + p_I b \mu_{1j} - p_Y Y_j}{p_I b} \rightarrow$$

$$I_{1j} = \frac{M_j + p_I \mu_{1j}}{p_I b - 2p_Y} \left(b - \frac{p_Y}{p_I}\right) - \mu_{1j}, \quad (23)$$

where  $b < \frac{p_Y}{p_I}$ . In the case of an additive endowment  $\mu_{1j}$  explicitly enters each reduced form demand function whereas in the case of a multiplicative endowment the reduced form demand relations were functions of only prices and income.

## 2.2 Adding Uncertainty to the Model

The model as described above embeds the assumption of certainty regarding the child's status at birth. However, it might be more plausible to assume that there is a degree of uncertainty regarding whether or not the child will die at or in immediate connection to birth. This uncertainty is likely to influence the mother's health habits.

Consider a pregnant woman at time  $t$  and assume that the household looks forward two periods, i.e. one period beyond the delivery at time  $t+1$ , to  $t+2$ . Now, the household must form an expectation at time  $t$  regarding the utility at time  $t+2$ . This expected utility is a weighted average of the utilities it receives if the child survives the delivery and if the child dies. Thus, the expected postnatal utility can be expressed as:

$$E_t[U_{j,t+2}] = \rho U_{j,t+2}^S + (1 - \rho) U_{j,t+2}^D, \quad (24)$$

where  $U_{j,t+2}^S = U^S(X_{j,t+2}, Y_{j,t+2}, H_{1j,t+2})$  if the child survives or  $U_{j,t+2}^D = U^D(X_{j,t+2}, Y_{j,t+2})$  if the child dies, and  $\rho$  is the associated probability of survival until  $t+2$ .  $H_{1j,t+2}$  is the health after birth and the postnatal health production function if the child survives the delivery is given by:

$$H_{1j,t+2} = \Gamma(H_{01j,t+1}, Y_{j,t+2}, I_{1j,t+2}, \mu_{1j}). \quad (25)$$

Many different factors affect the health of the child at the delivery. Some of these factors can be affected by the mother during the pregnancy, some factors have to do with the mother's health status, some factors have to do with family-specific health endowments, and some factors are exogenous and beyond the control of the mother. Therefore  $\varepsilon_{t+1}$  is included as an exogenous stochastic variable that takes the value 1 if the child survives the delivery and 0 if the child dies at birth. Therefore,  $\rho$  is assumed a function of the health of the child at birth,  $H_{01j,t+1}$ , which in turn is a function of the mother's health status/history,  $\eta_{j,t}$ , as well as of the "shock variable"  $\varepsilon_{t+1}$ . The expected utility can be described by:

$$E_t[U_{j,t+2}] = \rho(H_{01j,t+1}, \varepsilon_{t+1}) U_{j,t+2}^S + (1 - \rho(H_{01j,t+1}, \varepsilon_{t+1})) U_{j,t+2}^D. \quad (26)$$

The associated budget constraint facing the household is:

$$M_{j,t+2} = X_{j,t+2} + p_{Y,t+2} Y_{j,t+2} + \rho(H_{01j,t+1}, \varepsilon_{t+1}) p_{I,t+2} I_{j,t+2}. \quad (27)$$

Thus, the household's optimisation problem can be expressed as an optimisation in two steps; first choosing the optimal  $\rho$ ,  $\rho^*$ , i.e. the highest possible  $\rho$  given the restrictions,

and then maximising  $E_t[U_{j,t+2}]$  given  $\rho^*$ :

$$\max \rho(H_{01j,t+1}, \varepsilon_{t+1}), \quad (28)$$

$$\text{subject to } H_{01j,t+1} = Y_{j,t}\eta_{j,t}\mu_{1j}, \quad (29)$$

$$\text{and } M_{j,t} = X_{j,t} + p_{Y,t}Y_{j,t}. \quad (30)$$

$$\max_{X_{j,t+2}, Y_{j,t+2}, I_{1j,t+2}} E_t[U_{j,t+2}], \quad (31)$$

$$\text{where } E_t[U_{j,t+2}] = \rho^*(H_{01j,t+1}, \varepsilon_{t+1})U_{j,t+2}^S + (1 - \rho^*(H_{01j,t+1}, \varepsilon_{t+1}))U_{j,t+2}^D, \quad (32)$$

$$\text{subject to } M_{j,t+2} = X_{j,t+2} + p_{Y,t+2}Y_{j,t+2} + \rho^*(H_{01j,t+1}, \varepsilon_{t+1})p_{I,t+2}I_{1j,t+2}. \quad (33)$$

### CASE 3 *Optimisation under uncertainty*

Thus, given the probability distribution of  $\rho$ , the household chooses the period  $t$  consumption that is consistent with  $\rho^*$ .  $\rho^*$  is then used as an input in the second step of the optimisation. Assume the following functional forms for the postnatal utilities:

$$U_{j,t+2}^S = X_{j,t+2}Y_{j,t+2}H_{1j,t+2} \text{ where } H_{1j,t+2} = H_{01j,t+1}Y_{j,t+2}I_{1j,t+2}\mu_{1j,t+2}.$$

$$U_{j,t+2}^S = X_{j,t+2}Y_{j,t+2}^2H_{01j,t+1}I_{1j,t+2}\mu_{1j,t+2}, \text{ and} \quad (34)$$

$$U_{j,t+1}^D = X_{j,t+2} + Y_{j,t+2}. \quad (35)$$

$$E_t[U_{j,t+2}] = \rho^*[X_{j,t+2}Y_{j,t+2}^2H_{01j,t+1}I_{1j,t+2}\mu_{1j,t+2}] + (1 - \rho^*)[X_{j,t+2} + Y_{j,t+2}], \text{ and} \quad (36)$$

$$M_{j,t+2} = X_{j,t+2} + p_{Y,t+2}Y_{j,t+2} + \rho^*p_{I,t+2}I_{1j,t+2}. \quad (37)$$

$U_{j,t+2}^S$  has a multiplicative form and thus it is increasing in the health of the child.  $U_{j,t+2}^D$  is additive for simplicity. Maximisation of (36) subject to (37) gives the following reduced form demand equations for the child specific health input  $I_{1j,t+2}$ , the composite health environment commodity  $Y_{j,t+2}$ , and the non-health consumption commodity  $X_{j,t+2}$ :

$$I_{1j,t+2} = \frac{1}{4} \frac{M_{j,t+2}}{\rho^*p_{I,t+2}} - \frac{1}{2} (1 - \rho^*), \quad (38)$$

$$Y_{j,t+2} = \frac{1}{2} \frac{M_{j,t+2}}{p_{Y,t+2}} - \frac{1}{3} (1 - \rho^*) \left( \frac{1 + p_{Y,t+2} + \rho^*p_{I,t+2}}{p_{Y,t+2}} \right), \quad (39)$$

$$X_{j,t+2} = \frac{1}{4} M_{j,t+2} - \frac{1}{3} (1 - \rho^*) \left( 1 + p_{Y,t+2} + \frac{1}{2} \rho^* p_{I,t+2} \right). \quad (40)$$

These demand functions imply that the lower the probability of survival the higher is the postnatal consumption of child specific health inputs  $I_{1j,t+2}$ . In addition, the model implies that the consumption of child specific health inputs will fall as the price of these inputs rises, a result that is in line with Currie (2000) and that has interesting implications when it comes to "access" problems.

### 3 Determinants and Measurement of Child Health

Several attempts have been made to find the right way in which to measure health and its determinants. Usually the representation of health status in micro empirical studies is by: (i) clinical measures of bodily attributes; (ii) anthropometric measures of height, weight, triceps skinfold thickness, arm circumference, etc.; (iii) respondent-reported disease symptoms, mortality histories, and general health evaluation; and (iv) reports on incapacity for undertaking normal respondent activities (Behrman and Deolalikar, 1988). However, there are problems with self-reports and reports on other individuals because of potentially severe reporting biases when the information is self- or proxy-reported.

The dataset used in the present study, the Cebu Longitudinal Health and Nutrition Survey, contains detailed information on anthropometric measures for the woman giving birth as well as for the child being born. When analysing health and its determinants the question of how to best represent a person's health must be addressed. There are two obvious approaches: to analyse each identified health indicator separately or to construct a composite measure of health. However, it has been noted that a separate analysis of individual health indicators is preferred to the construction of a composite measure of health (Kovsted et al., 1999) and therefore the present study will conduct separate analyses of child anthropometric measures. However, the determinants of child health are divided into two groups: prenatal and postnatal determinants.

### 3.1 Prenatal Determinants of Child Health

- Household income

Several studies have attempted to measure the effect of income on health but there have been problems due to reverse causality (Ettner, 1996). In a study, Ettner (1996) derives consistent estimates of the structural effect of income on health using instrumental variables techniques, and finds strong empirical evidence that income has a large, beneficial impact on mental and physical health.

- Mother's (and household's) health knowledge.

Health knowledge naturally affects childrens' health since it affects the mother's and household's health habits. Kovsted et al. (1999) incorporate health knowledge<sup>10</sup> in their analysis of the determinants of child health and mortality in Guinea-Bissau, although they do not distinguish between prenatal and postnatal determinants of child health.

- Mother's health habits e.g. smoking, alcohol consumption, food consumption, health care visits, vitamin intake.

The relationship between smoking and birthweight has been thoroughly investigated, with studies involving over 500 000 births. By 1990, the evidence was strong enough for the US Surgeon General to conclude that maternal smoking definitely retarded fetal growth, causing an average reduction in birthweight of 200 g and doubling the risk of having a low-birthweight baby (Walsh et al., 2001). Drinking during pregnancy can cause damage to the unborn child, a condition known as Fetal Alcohol Syndrome (FAS). FAS refers to a specific cluster of anomalies associated with the use of alcohol during pregnancy, for example mental retardation, growth deficiencies, central nervous system dysfunction, craniofacial abnormalities and behavioral maladjustment's (Brooks, 1997).

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<sup>10</sup>Actually, they use maternal educational attainment (years of completed schooling), knowledge of what causes malaria, and the age of the mother as proxies for health knowledge.

In addition, it has been shown that prenatal care can have a significant impact on the incidence of low birthweight (e.g. Guilkey et al., 1989; Currie, 2000).

The present study regards the observable health habits as the result of the unobservable health knowledge thereby implicitly accounting for health knowledge. It is assumed that the mother's health habits affect a child's health even before it is born, hence, smoking, alcohol intake, vitamin intake and health care visits are included in the analysis.

- Mother's health history e.g. pregnancy history, nutritional status, illnesses during the pregnancy.

Numerous empirical evidence have shown that the health status of the mother affects the child's health, e.g. Bhargava (2000). Therefore, the present study analyses the effects of the mother's nutritional status during pregnancy as a potential prenatal determinant of a child's health.

### **3.2 Postnatal Determinants of Child Health**

- Household income (see motivation above).
- Gender.

A gender dummy is included in the empirical analysis to control for potential differences in health between children of different sex.

- Breast-feeding patterns

There is a widespread consensus that breast feeding has a positive effect on child health. Unfortunately, only eleven women have reported that they have attempted to breast-feed their children, in the sample used in the present study, and therefore, the lack of data makes it impossible to include this variable. However, the Cebu Study Team (1992), finds, using both the rural and urban sample (approximately 3300 households), that breast-feeding tends to enhance infant growth.

- General health environment.

Variables capturing the status of the general health environment the child is exposed to are also included. Such variables can for example include measures of household choice of drinking water source and toilet facility, as well as smoking, alcohol consumption and variables capturing whether the household has insurance or not.

## 4 Data

The dataset used in the present study is the Cebu Longitudinal Health and Nutrition Survey (CLHNS).<sup>11</sup> This survey, is part of an ongoing study of a cohort of Filipino women who gave birth between May 1, 1983 and April 30, 1984. The sampling frame was the 1980 Population Census, and at that time the Metro Cebu Area included 243 barangays<sup>12</sup> of which 155 was classified<sup>13</sup> as urban and 88 as rural. However, this classification was modified, and according to the modified classification there are 95 urban barangays and 148 rural. There is no universal definition of the concept rural but it is usually considered to be any locality that exists primarily to service an agricultural hinterland. This definition implies that rural areas can include towns of considerable size.

A two-stage stratified cluster sampling procedure was used to randomly select 33 barangays, containing approximately 20 000 households. Of the 33 sample barangays, 17 are located in urban, and 16 in rural areas. The project team had decided at the outset of the survey that three fourth of the sample was to be allocated to urban and only one fourth to rural respondents. From the 33 barangays a random sample of 3327 women were selected and a baseline survey of the selected women and their households were un-

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<sup>11</sup>The Cebu Longitudinal Health and Nutrition project is a joint endeavour of the Carolina Population Center, University of North Carolina at Chapel Hill, the Nutrition Center of the Philippines, Manila, and the Office of Population Studies, University of San Carlos, Cebu City.

<sup>12</sup>Barangay = community or village

<sup>13</sup>The Census Bureau had classified the barangays on the basis of population size and density, physical characteristics, and administrative functions.

dertaken. From these approximately 3300 households, 560 (prenatal estimations) and 434 (postnatal estimations) out of approximately 770 possible rural households (one fourth of the sample) have been used in the present study. One of the objectives of the present study is to explicitly incorporate water and sanitation variables. Since there is almost no variation in such variables among urban households only rural households have been included in the analysis. In addition, households for which there are no recorded answers regarding child health or the water and sanitation variables used in the present study have been excluded. The CLHNS data includes, among other things, information on socio-economic characteristics of the household, intra- household time allocation, maternal health behaviour, health status, health practices, child anthropometry and morbidity, family planning, community characteristics, and food/non-food prices.

## 4.1 Sample Descriptive Statistics

**Household Characteristics** There are five variables that are used to describe the household: the number of people in the household (*NOPERS*), the number of rooms in the house (*ROOMS*), household income (*YTOT*), see *Table 2*, the type of household and the type of construction material used for building the house, see *Table 3*, and *Table 4*.

Table 2.  
The number of people and rooms in the household and house

	Prenatal			Postnatal		
	<i>NOPERS</i>	<i>ROOMS</i>	<i>YTOT</i>	<i>NOPERS</i>	<i>ROOMS</i>	<i>YTOT</i>
Maximum	16	8	371278,96	16	8	99476
Minimum	2	1	0	2	1	0
Mean	5,43	2,40	10135,11	5,40	2,42	8648,25
Median	5	2	6342,50	5	2	6260
St.dev.	2,52	1,11	20207	2,48	1,08	10413,47
No. of obs.	560	560	560	434	434	434

Table 3.  
Types of households

		Prenatal	Postnatal
<i>Type</i>	<i>Description</i>	<i>Frequency</i>	<i>Frequency</i>
1	single-person HH	0	0
2	1 nuclear family HH	409	328
3	horizontally extended nuclear family	25	17
4	vertically extended nuclear family	34	31
5	hor. and ver. extended nuclear family	4	3
6	multi-nuclear family HH	88	55

Note: HH = household

A horizontally extended family means that it is extended across cohorts in contrast to a vertically extended family that is extended across generations.

Table 4.  
Types of construction materials

		Prenatal	Postnatal
<i>Type</i>	<i>Description</i>	<i>Frequency</i>	<i>Frequency</i>
1	light (nipa and similar)	335	262
2	mixed (cement and/or wood with nipa)	196	148
3	strong (cement, wood and galvanised iron)	29	24

The traditional bahay kubo (home cube), also known as the nipa hut, is a single room structure raised on stilts. It is made of bamboo and other types of grasses, as well as nipa leaves.

**Mother’s Health Habits** The dataset includes information on a number of variables describing the health habits of the mother. The variables included in the present analysis are whether or not the mother takes vitamins, has consulted a health practitioner (*PRENA*), has been on a prenatal visit at a government hospital (*GHOS*), see *Table 5*, the number of cigarettes smoked per day (*SMOKE*) and the amount of alcohol, in millilitres, the woman drinks each day (*ALCOML*), see *Table 6*.

Table 5.

Vitamin intake and prenatal health visits		
	0 = No	1 = Yes
Takes vitamins (prenatal)	310	226
<i>PRENA</i>	89	471
<i>GHOS</i>	527	33

Table 6.

Daily alcohol (in ml) and cigarette intake by the mother

	Prenatal		Postnatal	
	<i>SMOKE</i>	<i>ALCOML</i>	<i>SMOKE</i>	<i>ALCOML</i>
Maximum	24	720	24	240
Minimum	0	0	0	0
Mean	0,77	7,80	0,84	7,75
Median	0	0	0	0
St.dev.	2,30	40,71	2,50	30,38
No. of obs.	560	560	434	434

In the estimations the constructed interaction variable *SMOKE\*ALCOML* has been included to capture a possible behavioural effect.

**Mother's Health History** The mother's pregnancy history, see *Table 7*, can be an indicator of the health status of the mother and as such it can be regarded as a potential prenatal determinant of a child's health.

Table 7.

Mother's pregnancy history

	<i>PPREG</i>	<i>LBIRTH</i>	<i>ABABY</i>	<i>STPREG</i>	<i>MPREG</i>
Maximum	15	11	10	2	4
Minimum	0	0	0	0	0
Mean	2,94	2,69	2,47	0,03	0,23
Median	2	2	2	0	0
St.dev.	2,68	2,48	2,26	0,19	0,52
No. of obs.	560	560	560	560	560

Note: *PPREG* = No. of past pregnancies, *LBIRTH* = No. of past live births, *ABABY* = No. of living children, *STPREG* = No. of stillbirth pregnancies and *MPREG* = No. of miscarriage/abortion pregnancies.

However, looking at correlations between the variables reveal that *PPREG* is highly correlated with *LBIRTH* and *ABABY*, and therefore only *PPREG* has been included in the estimations. In addition to the mother's pregnancy history, data on the mother's anthropometry is used as a determinant of child health, see *Table 8*.

Table 8.  
Mother's anthropometry

	<i>Weight</i> <i>in kg</i>	<i>Height</i> <i>in cm</i>	<i>Arm circumf.</i> <i>in cm</i>
Maximum	78	165	36,6
Minimum	35	136,6	18,9
Mean	49,42	150,5	24,22
Median	49	150,3	24
St.dev.	6,27	5,02	2,17
No. of obs.	560	560	560

As noted by Kovsted et al. (1999), mid-upper-arm circumference (*ARMCIRCU*) can be used as a proxy for the nutritional status and income situation of the woman. The argument is that a woman with a large mid-upper-arm circumference is more likely to do less hard work and have more ample supplies of food since women rarely develop large muscle mass as a result of hard physical work.<sup>14</sup> However, *ARMCIRCU* and the mother's weight are highly correlated, which means that only one of these variables has been included in the estimations. *ARMCIRCU*<sup>2</sup> has been included to analyse if the results differ when the variable is squared.

**General Health Environment** To capture the general health environment, a water and sanitation measure has been constructed. Each household in the sample has chosen one drinking water source and one toilet facility, see *Table 9* for the available alternatives. The water and sanitation measure is simply the sum of the alternatives a household has chosen, i.e. if a household has chosen *Pump, in house* and *Open pit* the number in the measure equals nine, see *Table 10*. Constructed in this way, a higher number implies a less modern combination of water and sanitation facilities.

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<sup>14</sup>See also Ferro-Luzzi & James (1996) and James et al. (1994).

Table 9.

Available water and sanitation alternatives		
<i>Choice code</i>	<i>Drinking water source</i>	<i>Toilet facility</i>
1	Piped, in house	Flush, inside the house
2	Pump, in house	Flush, outside house
3	Pump, in yard	Water sealed, inside house
4	Rainwater	Water sealed, outside, private
5	Pump or piped, not in house/yard	Water sealed, outside, public
6	Open well	Antipolo
7	Spring, river, lake	Open pit
8	Purchased	None (go to field, river, etc)

Table 10.

Water and sanitation measure		
	Prenatal	Postnatal
	<i>WATSAN</i>	<i>WATSAN</i>
Maximum	16	16
Minimum	6	7
Mean	12,61	12,83
Median	13	13
St.dev.	2,22	2,14
No. of obs.	560	434

**Indicators of Child Health** As indicators of child health, the child's weight and height are used, see *Table 11*.

Table 11.

Indicators of child health					
	Prenatal		Postnatal		
	<i>Weight (gr)</i> <i>at birth</i>	<i>Height (cm)</i> <i>at birth</i>	<i>Weight (gr)</i> <i>after a year</i>	<i>Height (cm)</i> <i>after a year</i>	$\frac{\text{Weight (gr)}}{\text{Height (cm)}}$ <i>after a year</i> (i.e. wasting)
Max	5000	56	13000	87,5	154,95
Min	1500	41,5	6500	67,2	94,48
Mean	2962,05	48,63	9642,86	78,62	122,42
Median	3000	48,8	9600	78,9	122,39
St.dev.	456,52	2,93	1116,02	3,44	11
No. of obs.	560	560	434	434	434

Birthweight has been recognised as the single most important indicator of infant health since children of low birthweight (less than 2500 grams) experience post-neonatal mortality rates 10 to 15 times those found among infants of normal birthweight (Currie, 2000).

## 5 Estimations

In both the prenatal and postnatal case, a weighted ordinary least squares (OLS) procedure was employed since this procedure significantly improved the model fit. The weighting variable is the number of people in the household (*NOBERS*) and the individual weights,  $w_h$ , i.e. one for each household,  $h$ , are calculated as:

$$w_h = \frac{560}{\sum_{h=1}^{H=560} NOBERS_h} * NOBERS_h.$$

### 5.1 Prenatal Child Health Production Function

From (2), the prenatal child health production function is given by:

$$H_{01j} = \Psi(Y_j, \eta_j, \mu_{1j}), \quad (41)$$

that is, child health at birth is assumed to be determined by the health habits of the mother,  $Y_j$ , e.g. medical care, smoking, alcohol consumption etc., the health status of the mother,  $\eta_j$ , e.g. nutritional status and healthiness during pregnancy, and  $\mu_{1j}$ , a family-specific child health endowment that is known, but not controlled, by the family.

The results from regressing the child's birthweight on the different possible and available variables (see *Sections* 3.1 and 4.1 for descriptions and summary statistics of the variables) are presented in *Table 12*. Due to the relatively large number of potential explanatory variables, the results of three selection criteria (Amemiya, 1980), i.e. Theils  $\bar{R}^2$ , Akaike's Information Criterion, and Amemiyas Prediction Criterion, are reported in *Tables 12* and *13*. Based on these selection criteria, *Model 2* seems to be the "best" model. The results reveal, *ceteris paribus*, that the constant is highly significant which implies that the family-specific health endowment  $\mu_{1j}$  (together with variables potentially important but not controlled for due to lack of data) results on average in a birthweight of approximately 2300 grams. Smoking and drinking have no significant effects on birthweight, when analysed separately, which is somewhat surprising given that it is commonly argued that such activities reduce birthweight.

Table 12.  
OLS Prenatal regression results

Dependent variable: birthweight	<i>Model 1</i>		<i>Model 2</i>		<i>Model 3</i>	
Independent variables:	Coeff.	P-val.	Coeff.	P-val.	Coeff.	P-val.
<i>Constant</i>	2289,57	0,00	2266,67	0,00	2275,92	0,00
<i>YTOT</i>	0,0005	0,95	-	-	-	-
<i>PRENA</i>	109,80	0,03	120,33	0,01	118,37	0,02
<i>SMOKE</i>	-11,16	0,17	-	-	-	-
<i>ALCOML</i>	-0,34	0,48	-	-	-	-
<i>SMOKE*ALCOML</i>	0,45	0,02	0,33	0,05	0,36	0,03
<i>PPREG</i>	-2,72	0,72	-	-	-	-
<i>STPREG</i>	194,15	0,05	179,84	0,07	-	-
<i>MPREG</i>	45,46	0,24	-	-	-	-
<i>ARMCIRCU</i> <sup>2</sup>	0,98	0,00	0,99	0,00	0,98	0,00
No. of obs.	560	560	560	560	560	560
Selection criterion						
Theils $R^2$ (R-sq. adj.)	0,431		0,428		0,424	
Akaike's (AIC)	15,063		15,050		15,053	
Amemiya's PC	12,207		12,204		12,208	

However, the smoking and alcohol interaction variable has a significant *positive* effect on birthweight which seems awkward. In addition, these results seem to contradict the findings for Russia reported by Jensen & Richter (2001) who find that smoking and alcohol have significant negative effects on child health as measured by the stunting measure height-for-age. These differences may be a result of the fact that the present study explicitly separates the prenatal period from the postnatal period thus finding that smoking has ambiguous effects on birthweight but that smoking by the mother, i.e. passive smoking by the child, has indeed effects on the child's postnatal health. Prenatal care and arm circumference of the mother have significant and positive effects on birthweight which is as expected and in line with earlier results e.g. Jensen & Richter (2001), Kovsted et al. (1999) and Guilkey et al. (1989). In addition, the variable capturing the number of previous stillbirth pregnancies, *STPREG*, has a significant positive effect on birthweight, which at a first glance seems odd. However, it might be that the mother "improves" her way of life after having experienced stillbirth pregnancies, and therefore there is a positive effect of this variable on birthweight. The income variable is, however, insignificant.

## 5.2 Postnatal Child Health Production Function

When estimating the postnatal health production function, the dependent variable is the child's weight for height, i.e. the wasting measure. The possible explanatory variables are: household income, *YTOT*, the gender of the child, *SEXCHILD* (the number of male children in the sample is 222 and the number of female children is 212), general health environment measures, i.e. *WATSAN*, *SMOKE*, *ALCOML*, *SMOKE\*ALCOML*, and *ARMCIRCU*<sup>2</sup> as a measure of the nutritional status/income situation of the woman. Regression results are reported in *Table 13*.

Table 13.  
OLS postnatal regression results<sup>15</sup>

Dependent variable: $\frac{Weight}{Height}$ (i.e. wasting)	<i>Model 1</i>		<i>Model 2</i>		<i>Model 3</i>	
Independent variables:	Coeff.	P-val.	Coeff.	P-val.	Coeff.	P-val.
<i>Constant</i>	113,55	0,00	112,87	0,00	114,70	0,00
<i>YTOT</i>	0,0006	0,14	0,0006	0,14	-	-
<i>SEXCHILD</i>	6,47	0,00	6,49	0,00	6,43	0,00
<i>WATSAN</i>	0,45	0,07	0,46	0,06	0,37	0,12
<i>SMOKE</i>	-0,42	0,02	-0,42	0,02	-0,41	0,02
<i>ALCOML</i>	-0,08	0,70	-	-	-	-
<i>SMOKE*ALCOML</i>	0,01	0,84	-	-	-	-
<i>ARMCIRCU</i> <sup>2</sup>	-0,009	0,85	-	-	-	-
No. of obs.	434	434	434	434	434	434
Selection criterion						
Theils $R^2$ (R-sq. adj.)	0,468		0,467		0,464	
Akaike's (AIC)	7,552		7,538		7,539	
Amemiya's PC	4,695		4,689		4,692	

Based on the three selection criteria, *Model 2* seems to be the "best" model. *Ceteris paribus*, the results reveal that the constant, which captures the family-specific health endowments as well as variables potentially important but not controlled for due to lack of data, has a significant and positive effect on the child's weight for height. In addition, the results reveal that although alcohol intake does not seem to matter for child health, the smoking behaviour does. Contrary to the prenatal case, smoking has a significant and negative effect on the child's weight for height. That is, although smoking does

<sup>15</sup>There were no gains to be made, in terms of model fit and significance levels, from employing different specifications and estimation techniques. The results are available upon request.

not seem to affect birthweight it does affect the child's postnatal health. Thus, this result for smoking is in line with the findings for Russia as reported by Jensen & Richter (2001). Not surprisingly, the results show that boys, *ceteris paribus*, have a higher weight for height than girls do, i.e. the sex of the child has a significant and positive effect on weight for height. In addition, the health environment variable, *WATSAN* was significant, indicating that the choice of drinking water source and toilet facility has an effect on child health. However, since *WATSAN* is constructed in a such a way that an increase in the variable indicates a less modern combination of toilet facility and drinking water source, the significant positive result might indicate that a households that lives in a "bad" environment compensates the child thus improving the child's health.

However, contrary to the prenatal case, *ARMCIRCU*<sup>2</sup>, which was included since the nutritional/income status of the mother can be regarded as a measure of the general health environment in which the child lives, was insignificant, as was the income variable, *YTOT*.

### 5.3 Robustness of the Results

The data used in the present study has been collected using a two-stage stratified cluster sampling procedure. This two-stage design, in which primary sampling units or clusters (often villages) are drawn first and then households from within each cluster, is very common for household surveys in developing countries. However, as Deaton (1997) points out, treating a two-stage sample as if it was a simple random sample can have serious implications since the sampling variability of the estimates can be affected by the design. It is often the case that clustering increases the inter-cluster variability since households within clusters are frequently similar to one another in their relevant characteristics. Therefore, ignoring the cluster design can lead to standard errors that are too small and t-values that are too large, thereby overstating the precision of the estimates.

In order to check whether the results obtained in the present study are sensitive for the survey design a bootstrap approach proposed by Deaton (1997) is employed. Deaton's

approach implies bootstrapping the clusters rather than the households. A list of the  $n$  sample clusters is made, a bootstrap sample of size  $n$  is drawn with replacement, and the individual cluster-level data merged in. The bootstrap results indicate that clustering is not a problem and hence the OLS regression results seem to be robust to the design.<sup>16</sup>

## 6 Summary and Conclusions

In this paper, different aspects of child health and women's health choices for a sample of rural households in Cebu, Philippines have been analysed both theoretically and empirically. However, the present approach differs from other studies by analysing separately prenatal and postnatal determinants of child health both under certainty and uncertainty. Hence, the present analysis introduces the possibility of different health production functions before and after birth, thus letting the delivery play a central role for a child's health prospects. An approach that is well in line with recent research in nutrition and epidemiology. In addition, special attention is paid to the role of water and sanitation related inputs for the production of child health.

The theoretical model predicts a positive relationship between the mothers health habits during pregnancy and the expected survivability of the child at birth. That is, if the child is expected to survive delivery then the mother spends a larger amount of her income on the composite health commodity/her own health habits than if the child is not expected to survive. In the postnatal case, the model predicts that the larger the probability of survival the less is spent on child specific health inputs. It is reasonable to assume that if the probability of survival is low, as it usually is in high mortality/morbidity populations, then more money has to be spent on child specific health inputs in order to compensate the child for the "bad start" in life.

Empirically, the results show that family-specific health endowments may explain a large part of the child's health, and prenatal care and the mother's income/nutritional

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<sup>16</sup>The bootstrap results are available upon request.

status have significant effects on birthweight. Income has no significant effect on child health in any of the two periods. The number of previous stillbirth pregnancies has a significant positive effect on birthweight, which at a first glance seems odd. However, it might be that the mother "improves" her way of life after having experienced stillbirth pregnancies, and therefore there is a positive effect of this variable on birthweight. In the prenatal case smoking and alcohol consumption have no effects on child health when analysed separately. However, when letting smoking and alcohol consumption interact, the result is a *positive* effect on birthweight, which seems awkward. In addition, the results suggest that smoking by the mother has a significant and negative effect on the health of the child after birth thus implying that passive smoking by the child has important health effects. In addition, the results indicate that the choice of drinking water source and toilet facility has an effect on postnatal child health. The significant positive result of the water and sanitation variable might indicate that a households that lives in a "bad" environment compensates the child thus improving the child's health.

To some extent, the results obtained in the present study seem to contradict the findings for Russia reported by Jensen & Richter (2001) who find that smoking and alcohol have significant negative effects on child health as measured by the stunting measure height-for-age. However, some of the differences may be a result of the fact that Jensen & Richter explores a panel data set, and hence they do not explicitly separate the prenatal and postnatal periods, thus allowing for different health production functions as the present study does. Thus, it is possible to conclude that there can be important insights to be made from the explicit separation of the prenatal and postnatal periods as regards the determinants of child health.

A bootstrap approach has been employed to check whether the results obtained in the present study are sensitive for the survey design, i.e. the two-stage stratified cluster sampling procedure. The results indicate that clustering is not a problem and hence the OLS regression results seem to be robust to the design.

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