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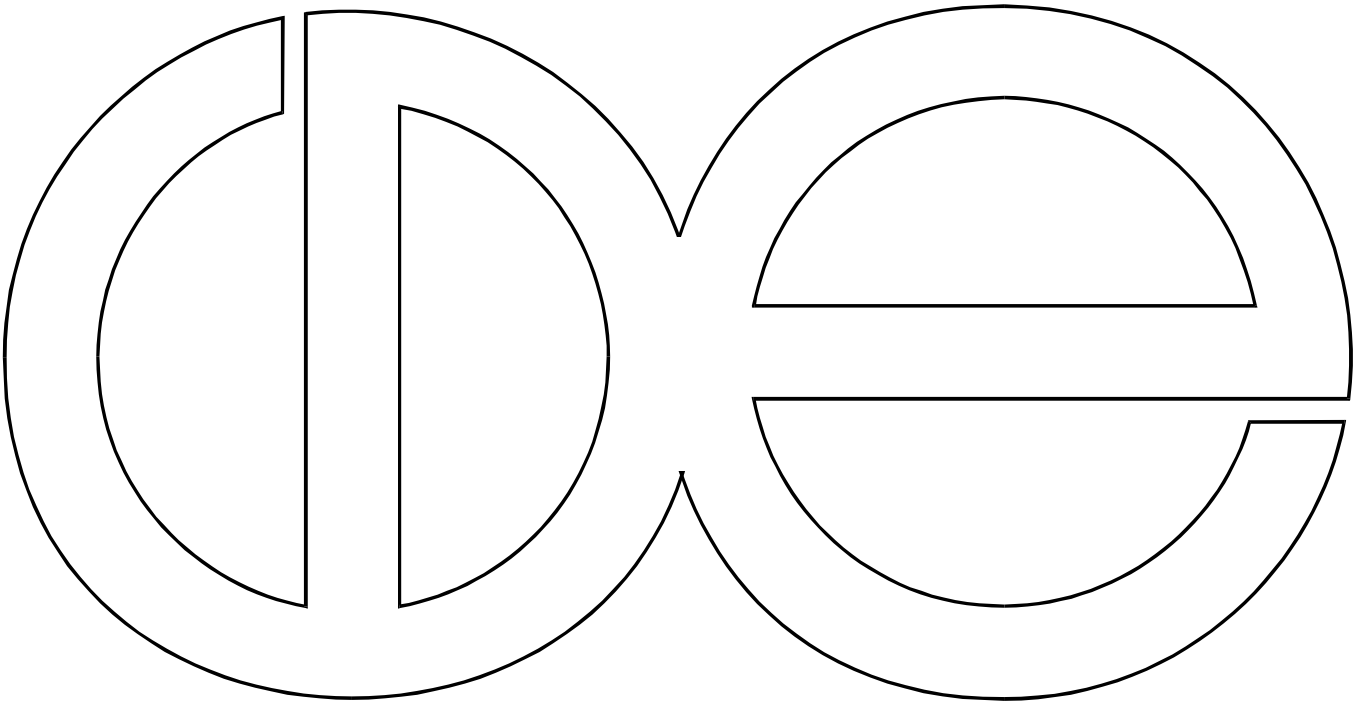

A BIVARIATE PROBIT ANALYSIS OF SOCIAL INTERACTION AND TREATMENT EFFECTS

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Introduction and problem statement

In this study I explore household and social determinants of child immunization and prenatal and delivery care. These and a handful of other health behaviors can reduce maternal and infant and child mortality by as much as 80 percent (WHO 1978; Walsh and Warren 1979). Most demographers agree that primary health care programs are an increasingly important cause of contemporary mortality and fertility transitions (Palloni 1990; Pebley 1993), and much research has been directed at understanding the factors that encourage and hinder PHC utilization.

Like demographic transitions generally, the development of national health care systems was originally conceptualized in terms of modernization theory. As countries develop, governments would supply basic health services. Increased income and exposure to new ideas would induce people to demand modern contraceptives, immunizations and other "personal" health services (Birdsall 1989). Subsequent events have shown that demographic and health transitions are far more complex. Contemporary transitions are proceeding apace even in the poorest countries (Bongaarts and Watkins 1996), and they turn out be robust to economic crises and prolonged recessions (Palloni 1981; Preston 1986; Hill and Pebley 1989). At the aggregate level PHC utilization does not seem to correlate with economic conditions (Caldwell 1986, Peabody 1996). Demographers and medical sociologists alike have noted that health behaviors and health outcomes frequently cluster at the community level, even when income, health risks, medical care availability, cultural context and other variables are controlled (Noack 1988; Mechanic 1994; Steele, Diamond and Amin 1995; Pebley, Goldman and Rodriguez 1996; Matthews and Diamond 1997). Such clustering may be due to some combination of local economic constraints, health service performance, social interaction effects or other unmeasured contextual causes. If

they could differentiate and predict these different effects, policymakers and practitioners would be in better positions to allocate scant health resources.

Similarly, there is a need to reconceptualize the processes through which individuals and households choose to avail themselves of health services. Most household-level research to date has employed a "proximate determinants" approach, (Bongaarts and Potter 1983; Mosley and Chen 1984), which imposes the assumption that agents act independently in choosing their health behaviors. Related to this is the household health production model (Schultz 1984; DaVanzo and Gertler 1990), which emphasizes individual health status and how individuals attain health, given their information and economic constraints. Although results have been generally consistent, neither approach accounts for much of the observed variance in health behaviors within or across households (Behrman 1990). Apparently, a household's decision to adopt a particular health behavior involves more than simple cost-benefit calculations.

The lack of household-level data and suitable analytic methods has stymied the development of new theoretical approaches, but this is changing. Data constraints have been dramatically lessened, thanks to the World Fertility and Demographic and Health (DHS) surveys. These multi-stage sample surveys have generated detailed household- and neighborhood-level health behavioral data for scores of countries dating back to the late 1970s. Meanwhile, administrative data on the technical quality and utilization patterns of PHC programs- the "supply side" of health- are increasingly available as ministries of health develop better management and technical capabilities. On the methodological front, new statistical approaches increasingly allow researchers to parse individual and contextual health behavioral determinants using cross-

sectional data.

In this paper I review some of these developments. I begin by describing the household health production model. I then elaborate the model to account for mortality selection, treatment effects and social interaction effects, and use the expanded model to analyze two pairs of health behaviors and outcomes from two countries.

Empirical considerations

Study countries

I examine data from two Latin American countries, Colombia and Paraguay. The countries contrast sharply in their geographies, histories, ethnic makeup and development paths. Paraguay is landlocked, relatively remote and little industrialized. Colombia's location is more central and its economy more differentiated. These differences aside, both countries are experiencing rapid urbanization, increasingly complex migration flows and mortality and fertility levels that have been falling for decades (Table 1). Mortality has historically been higher in Colombia but by the early 1980s mortality rates were about equal in the two countries. The declines began earlier in Paraguay but the rates of decline have been faster in Colombia. Since then Colombia's mortality and fertility have fallen below Paraguay's. On balance, the two countries trace different trajectories on Latin America's demographic spectrum.

I expect the links between health behaviors and demographic trends to be strongest in Latin America. By the early 1970s an estimated 63% of the Region had access to basic health care, including hospital care, household sanitary inspection and vector control measures,

immunizations, perinatal care and routine disease reporting (Pan American Health Organization 1992). No other developing region has such an extensive health sector. By and large, the Latin American PHC effort has been successful. For the Region as a whole, the percentage of children fully immunized before two years of age rose from 50 percent in 1983 to over 75 percent in 1990 (de Quadros et al. 1991); the percentage of mothers who had ever used oral rehydration therapy in the home to treat childhood diarrhea rose from 12 percent in 1984 to 56 percent in 1992 (WHO 1992); the percentage of reproductive-age women in unions who were using modern contraceptives rose from around 37 percent in 1986-89 (Rutenberg et al. 1991) to over 50 percent by 1990 (United Nations 1992); and the percentage of mothers using prenatal services rose from 56 percent in 1980 to 71 percent in 1990 (PAHO 1993). Table 2 shows selected PHC indicators for the two study countries over this period.

Health behaviors

I examine two qualitatively different yet demographically influential health behaviors. There is considerable empirical evidence that increased use of immunizations and prenatal and delivery services significantly altered the Latin American mortality pattern during the 1980s (PAHO 1992; Bahr and Wehrhahn 1993; Alvarez- Laurrauri et al. 1994; Blaney 1994; Nino et al. 1994; Rivera-Lopez et al. 1994; Matthews and Diamond 1999). Several simulation studies have estimated their mortality impacts. According to the World Health Organization (WHO), the WHO Global Expanded Programme on Immunization (EPI) has averted on the order of 3 million childhood deaths annually since 1991 (Grant 1991). In a recent paper Matthews and Diamond (1999) project that, by the year 2025, full coverage with all six EPI antigens would decrease underfive mortality ($_5q_0$) by 20 per 1000, which would add 1.2 years to the life expectancy of

Colombian and Paraguayan children under age five. Similar studies for maternal health services are lacking. However, some extrapolations are easily made. Professional delivery management reduces perinatal infant mortality by about 50 percent (Becker and Black 1996) and maternal mortality by about 70 percent (Pan American Health Organization 1993). In 1991 there were an estimated 7 million perinatal deaths and 450,000 maternal deaths in the developing world (WHO 1996). These perinatal deaths represent about one-half of all deaths below age five (Tsui, Wasserheit and Haaga 1997). Compared to immunization, attended delivery coverage in developing countries has increased rather modestly. As late as the mid-nineties only 37 percent of these births were professionally attended (WHO 1993). Elsewhere I examine the demographic impacts of these two health behaviors in the study countries in greater detail (McQuestion 2000).

Theoretical and methodological considerations

The household health production model

From an economic standpoint a household can be viewed as an organization concerned with maximizing utility for its members. Altruistic parents derive some utility from producing healthy children and they combine their knowledge, time and skills to do so. Economists have conceptualized these processes using a specific model termed the household health production model. Health is the output and nutrition, childcare and specific health behaviors are some of the inputs to this household health production function. Health, however, is just one source of utility. Household members also allocate scarce resources to consumption, saving and to other forms of investment. At the aggregate level, health status is the sum of health production across individual households. Fostering economic growth, increasing PHC program efficiency and accessibility, educating parents and controlling public health risks are some of the ways governments can help

households produce more health and thus improve population health status. In sum, the HHP model posits individual endowments and market forces as the determinants of household health production. Social norms are generally considered exogenous, that is, households view them as fixed constraints (Behrman and Deolalikar 1990).

Formally, the HHP model specifies health behaviors- a class of proximate mortality determinants- for household *i* as one set of endogenous inputs (I_i) to a child health production function, the output of which is nutritional status, survival or morbidity status or some other individual health outcome (Y_i). The other production function inputs are biological endowments (B_i) and the effects of omitted or unobservable variables (e_{1i}). The likelihood a given health behavior will occur depends on preferences (P_i), economic endowments (E_i), local prices and health program characteristics (X_r) and other unobservables (e_{2i}). The behaviors and health outcome occur together and are presumably influenced by some of the same variables. For example, a frail child with inferior health endowments (B_i) may use more health services yet still face a higher probability of dying (Schultz 1984:218). An estimate of the behavior's effect on the demographic outcome will therefore be biased because unmeasured variables simultaneously cause both behavioral input and health output. This potential endogeneity bias is purged using simultaneous equations of the form:

$$Y_i = c_o + c_i I_i + c_2 B_i + e_{1i} \qquad (1)$$

$$I_i = a_o + a_1 E_i + a_2 B_i + a_3 X_r + a_4 P_i + e_{2i}$$

where the c's are linear parameters of the production function and the a's are parameters of the

behavioral (demand) function. Figure 1 shows these relations in graphical form. Note that the exogenous observables E_i and X_r serve as instruments, allowing **I** to be identified.

A bivariate probit HHP model

Most household health production studies model continuous outcomes such weight for age (Blau 1986) or height for age (Barrera 1990). A handful of HHP studies have modeled discrete outcomes like the probability of a recent illness episode (Pitt and Rosenzweig 1989; Cebu Study Team 1992), or a birth or death (Pitt 1995). These studies, however, used panel data and fixed or random effects specifications to generate unbiased parameter estimates. In the present study, the data are cross-sectional and the health behaviors and outcomes I model are both discrete. The appropriate specification for this type of model is the bivariate probit, a simultaneous equations model that controls for the endogeneity of two related choices (Ashford and Snowden 1970; Greene 1993). The bivariate probit estimator is usually expressed in terms of a continuous latent variable representing propensity, utility or preferences. Let Y_{i1}* be the difference between benefits and costs of a given health status for individual *i*, conditional on health behavior *Ii*, a set of household characteristics X_{i1}, and other unobserved variables ε_{i1}. What is observed is the individual's health status choice, represented as $Y_{i1.}$ The relation can be written as:

$$Y_{i1}^* = X_{i1}\beta + \varepsilon_{i1} \qquad (2)$$

Y_{i1} = 1 if $Y_{i1}^* > 0$, 0 otherwise

Similarly, let Y_{i2}* represent the propensity or benefit-cost difference perceived by individual i with respect to health behavior Y_2, conditional on a set of household characteristics X_{i2} and

unobserved variables $\xi_{i2.}$ The behavioral choice is again an observed binary outcome as shown in Equation (3):

$$Y_{i2}^* = X_{i2}\beta + \xi_{i2} \qquad (3)$$

$$Y_{i2} = 1 \text{ if } Y_{i2}^* > 0, \text{ 0 otherwise}$$

In this seemingly unrelated bivariate probit specification all variables (**Y_1, Y_2, X_1, X_2**) are observed for each individual. The model is just identified when the same vector of covariates appears in each equation (McLanahan and Sandefur 1994:166; Greene 1995:458). The random error terms, ε_{i1} and ξ_{i2}, are dependent and normally distributed, such that $E[\varepsilon_{i1}] = E[\xi_{i2}] = 0$, $var[\varepsilon_{i1}] = var[\xi_{i2}] = 1$ and $cov[\varepsilon_{i1}, \xi_{i2}] = \rho$. If a Wald Test shows ρ is insignificant then no endogeneity bias is present and the two models can be estimated separately as binomial probits. If however ρ is significant and the log-likelihood of the bivariate estimate is significantly less than the joint binomial probit log-likelihoods, then indeed Y_{i1} and Y_{i2} are endogenous processes (Bertaut 1998; StataCorp 1999:137). The log-likelihood for the bivariate probit is:

$$L = P_{y1=0}\, F(-X_{y1i}\beta_{y1})\, P_{y1=1, y2=1}\, F_2(X_{y1i}\beta_{y1}, X_{y2i}\beta_{y2}, \rho)\; P_{y1=1, Y2=0}\, F_2(X_{y1i}\beta_{y1}, -X_{y2i}\beta_{y2}, -\rho) \qquad (4)$$

where F is the standard univariate normal cumulative distribution and F_2 is the standard bivariate normal cumulative distribution with correlation ρ. Equations (2) and (3) are simultaneously estimated using maximum likelihood, producing unbiased estimates of parameter coefficients b and ρ. The seemingly unrelated bivariate probit estimator thus achieves the main

desideratum of the HHP model. It also provides a general test of whether health behaviors and outcomes are related through unmeasured variable effects.

In recent work the bivariate probit has been used to measure the effects of workplace smoking bans on workers' decision to smoke (Evans, Farrelly and Montgomery 1996); to examine the relationship between welfare and labor force participation decisions (Christofides, Stengos and Swidinsky 1997); and to measure the effects of Catholic schools on high school graduation and college matriculation (Evans and Schwab 1995). In the health literature Ettner (1996) used a bivariate probit to estimate the effects of having a regular medical provider on the use of specific preventive health services. In another study Hisnanick and Surinder (1996) showed that low-income veterans with relatively greater disabilities were less likely to obtain private insurance and more likely to use VA hospitals.

Mortality selection

The DHS data and bivariate probit models I use contain information only on surviving children. It is likely that mortality risk and health behaviors are correlated through other, unmeasured variables. The distributions of these unmeasured variables are conceivably different among households that have and have not lost a child. Risk factor parameter estimates are likely to be biased to the extent that their effects are correlated with unmeasured mortality risk. Investigators have taken a number of approaches to correcting mortality sample selection bias (for a review see Pitt 1995). My approach is to impose an exclusion restriction and to estimate a series of simultaneous Heckman (1979) selection models of the form:

$$y_j = x_j b + u_{1ij} \qquad (5)$$

$$z_j^* = w_j\, g + u_{2ij}$$

$$z_i = 1 \quad \text{if } z_j^* > 0$$
$$z_i = 0 \quad \text{if } z_j^* \leq 0$$

where the dependent variable in the structural equation, y_j, is observed only if $w_j\, g > 0$. Additionally, it is assumed that $u_1 \sim N(0,\sigma_u)$, $u_2 \sim N(0,1)$ and $\text{corr}(u_1,u_2) = r$. Here, u_1 and u_2 are random error terms, z_j is a binary selection indicator variable coded "1" for those cases observed, x_j is a vector of individual and household covariates, w_j is a second vector of fixed covariates one of which is not correlated with u_1; and b and g are vectors of parameters. As y_j and z_i I use, respectively, number of live births and an indicator for whether or not the parents have lost a child. I obtained the instrumental variables in w_j from local census data, a process I describe more fully below. The selectivity effect is measured by the inverse Mills ratio (Heckman 1979), a random variable expressed as:

$$imr_i = \phi\,(z_i g\text{-hat})/\Phi(z_i g\text{-hat}) \qquad (6)$$

where ϕ is the normal probability density function and Φ is the normal cumulative distribution. I recover the *imr* terms from the selection models and include them as measures of mortality selectivity in bivariate probit models.

Social effects

A finding that social forces affect health behaviors over and above economic and other forces would explain how people can be getting healthier even as they are becoming poorer. In a recent

review, Bongaarts and Watkins (1996) postulated that increasing social interactions at all levels explain why the latest transitions have begun in unexpected places and proceeded faster than earlier transitions. Their causal argument is that, as heterogenous actors increasingly interact, their thresholds for behavioral change fall (Bongaarts and Watkins 1996:46).

The social interaction framework outlined by Bongaarts and Watkins allows actors' behavioral propensities to be differentially influenced by the behaviors of others in their reference group (Case, Hines and Rosen 1989; Case 1991). In the econometrics literature a number of recent studies incorporate social effects[1] on individual outcomes. The models attribute dependent outcomes to such causes as social norms (Cole, Mailath and Postlewaite 1992; Bernheim 1994) and peer group influence (Case and Katz 1991; Glaeser, Sacerdote and Scheinkman 1996), contagion or mimicry (Erbring and Young 1979; Case 1991; Crane 1991). The models share the hypothesis that group-level processes have independent effects on individual outcomes, over and above those of individual-level variables. One can divide social interactions models into two causally distinct categories. Normative models focus on an actor's preferences or subjective expectations about how others will perceive and evaluate a future behavior. At play is one's social status, which determines the allocation of nonmarket goods such as mating opportunities and membership in exclusive institutions. An agent's utility therefore depends in part on the status she is accorded by meeting normative expectations (Cole, Mailath and Postlewaite 1992). The desire of agents to conform and to avoid social sanctions- both unobservable, individual-level processes- explains why behavioral outcomes are correlated. Because normative processes

[1] For useful reviews see Bikhchandani, Hirshleifer and Welch 1992, and Brock and Durlauf 2000.

are unobservable, inferences in such a model must be made on contextual grounds. Researchers positing normative effects have shown that the higher the concentration of black adult role models in a given neighborhood, the more likely young, black women will be sexually active (Brewster 1994), and experience premarital births (Sucoff and Upchurch 1998). Similarly, a Scottish study attributed the clustering of low educational attainment scores to a lack of individual competitiveness in socioeconomically deprived neighborhoods (Garner and Raudenbush 1991). Although mean neighborhood attainment scores were positively correlated with individual scores, this contextual effect disappeared when the neighborhood deprivation measure was added to the model. In another study, the probability a Bangladeshi woman would accept modern family planning methods was shown to depend negatively on the religiosity of household heads in her extended family compound (Kamal, Sloggett and Cleland 1999). Peer influence and contagion models take a conceptually distinct approach. In these models an actor's behavioral choice, whether conformist or deviant, is conditional on the past performances and outcomes she has perceived among significant or adjacent others. Erbring and Young (1979) term this mechanism *endogenous feedback.* The assertion that actors evaluate others' past behaviors, as opposed to anticipate others' reactions to a future behavior, makes it straightforward to operationalize endogenous feedback models. A recent example of a contagion model is a study by Case and Katz (1991). The authors used observational data to demonstrate that such youthful behaviors as crime, drug abuse, idleness and church attendance are positively correlated with the proportions of youths in adjacent neighborhoods engaging in those behaviors.

In practice there is no way to identify a social effect because such processes are inherently unobservable. The conventional approach has been to represent a social effect as the mean

behavioral outcome of adjacent others. Merely adding such a "Y-bar" contextual variable to a classic regression model, however, incurs intractable identification problems which lead to inconsistent and biased parameter estimates (Hauser 1970, Blalock 1984, Manski 1995). The development of the bivariate probit and other nonlinear random effects models greatly alleviates these problems and provides new approaches to representing social effects. Brock and Durlauf (2000) recently proposed a series of binary choice models which can be used to estimate social interaction effects. Their departure point is a simple model in which individual *i* chooses behavior ? $_i$ in order to maximize the payoff function V, conditional on an observable vector of characteristics Z_i and two unobservable random shocks w_- (1) and $\varepsilon_i(-1)$. The random shocks represent personal idiosyncracies relevant to each choice. They are assumed to be independently and identically extreme-value distributed. The relation is expressed as: $\max_{\omega i \in \{-1,1\}} V(\omega_i, Z_i, \varepsilon_i(\omega_i))$. The authors expand this standard formulation to include social utility. Specifically, the utility agent *i* assigns to a given behavioral choice w_i is also determined by her beliefs about the choices made by other agents *j* in her neighborhood, $m^e_i(w_{-i})$. This expanded payoff function is represented as:

$$V(\omega_i, Z_i, \mu^e_i(\omega_{-i}), \varepsilon_i(\omega_i)) = u(\omega_i, Z_i) + S(\omega_i, Z_i, \mu^e_i(\omega_{-i})) + \varepsilon_i(\omega_i) \qquad (7)$$

Social utility, then, captures an agent's evaluation of other agents' choices regarding behavior w_- It is independent of the agent's own tastes or idiosyncracies $\varepsilon_i(\omega_i)$. Assuming that the agents in a particular neighborhood *n(i)* equally influence one another's behaviors, the proportion of agents *n* choosing behavior *w* measures the social utility *S* to agent *i* of choosing w_i. With the further

assumptions that agents are rational and self-consistent, Brock and Durlauf demonstrate that there is a unique average choice level equilibrium point in any neighborhood. Given this, social utility can be expressed as the expected mean behavioral choice ϖ:

$$S(\omega_i, Z_i, \mu^e_i(\omega_{-i})) = \varpi = J_{i,j}\ \omega_{-i}\ \Sigma_{j \in Ni}\ E_i\ (\omega_j) \qquad (8)$$

$$\text{cov}(\mu^e_i(\omega_{-i}), \varepsilon_i(\omega_i)) = 0$$

where J is a weight assigned to all pairs of members of community *n(i)* such that $J_{i,j} = 0$ if $j \notin n(i)$. This contextual variable is identified, the authors argue, provided Z_i includes at least one additional, exogenous neighborhood-level variable (Brock and Durlauf 2000:33). This neighborhood-bounded social utility formulation recalls Erbring and Young's conceptualization of social contagion as an equilibrium point that is reached in the presence of endogenous feedback (Erbring and Young 1979:412-3). That point is the sum of individual outcomes y_i plus the share of individual outcomes that is determined by contextual mechanisms ($a y_i$'). Following Brock and Durlauf, I include social interaction terms in each of the four bivariate probits I estimate.

Treatment effects

The explanatory power of the HHP model can be extended in yet another way. When the behavior in question is participation in a particular health program, the HHP model essentially becomes a treatment effects model. To infer treatment effects is to assume that health behaviors change through individual cognitive pathways attributable to participation in health programs. A

treatment effect is thus distinct from economic or social effects. The treatment effects model assumes that the health outcomes were attained after the behaviors were performed. *Temporal order* is therefore established even when the data are cross-sectional. Given this, average program treatment effects can be estimated at the group level. An average treatment effect can be defined as the difference in mean outcomes between treatment and control groups, provided: (a) assignment to the treatment is random, exogenous or "strongly ignorable," and; (b) alternatives of treatment or no treatment are plausible for each individual, even though only one of these can be observed[2] (Rubin 1974). High health-seeking propensities and/or susceptibility to social effects might have caused certain individuals to self-select into treatment groups. In a single-equation model this would upwardly bias the estimated covariate effects on outcomes. However, assignment can be considered "conditionally random" (Sobel 1996:369) or strongly ignorable if a set of instrumental variables Z can be identified and shown to cause assignment to the treatments (behaviors) but not the outcomes (Angrist 1991). On this basis, Angrist, Imbens and Rubin (1993:8) argue, instrumental variables can be used to estimate a "local average treatment effect." Evans, Farrelly and Montgomery (1996) used this approach to demonstrate that workplace smoking bans significantly reduce the probability of workers smoking. Similarly, Zweimuller and Winter-Ebmer (1996) found that Austrian manpower training programs both enlist less motivated participants and increase their subsequent employment chances. In a follow-up study to that of Coleman, Hoffer and Kilgore (1982), Evans and Schwab (1995) fitted bivariate probits which showed that attending a Catholic school conferred an average treatment effect ranging from 10 to 13 percent on the probability of completing high school and enrolling

[2] The fact that only one of the potential outcomes is observable for any individual has been termed the Fundamental Problem of Causal Inference (Holland 1986).

in college. They found significant treatment effects in ten differently specified bivariate models; disturbance correlations were insignificant in all but two of them. A particular Catholic school treatment effect and not sample selection bias, they concluded, explains why students from such schools more likely graduate and go on to college.

Following Angrist (1991), I model the bivariate probits with an aggregate-level instrumental variable in each behavioral equation. This exclusion restriction both satisfies the identification condition in Brock and Durlauf's (2000) social interactions model and allows me to calculate treatment effects after model estimation. With this I can write the full bivariate probit treatment effects HHP model as follows:

$$Y_i = 1 \text{ if } Y^*_i - \beta_0 + \beta_1 X_{ij} + \beta_2 imr_j + \beta_3 \varpi_k + \beta_4 S_i - [\eta_{1i} - u_i \lambda] > 0 \qquad (9)$$

$$= 0 \text{ otherwise}$$

$$S_i = 1 \text{ if } S^*_i - \gamma_0 + \gamma_1 X_{ij} + \gamma_2 imr_j + \gamma_3 \varpi_k + \gamma_4 Z_k - [\eta_{2i} - u_i] > 0 \qquad (10)$$

$$= 0 \text{ otherwise.}$$

where Y^*_i is a latent dependent variable (propensity to fully immunize a child or seek professional delivery assistance), X_{ij} is a vector of individual and household characteristics, imr_j is the inverse Mills ratio from the Heckman selection model, ϖ_k is the mean outcome for neighborhood k, S^*_i is a latent variable representing propensity to participate in the program, S_i is a treatment indicator (i.e., cardholdership or prenatal use), Z_k is an instrumental variable, β_0 - β_4 and γ_0 - γ_4 are vectors of parameter effects, η_{1i} and η_{2i} and u are independent, normally

distributed errors. The complex error term gives the following covariance matrix:

$$\begin{bmatrix} \sigma_1^2 + \sigma_u^2\lambda^2 & \sigma_u^2\lambda \\ \sigma_u^2\lambda & \sigma_2^2 + \sigma_u^2 \end{bmatrix}$$

With the restriction that ($\sigma_1^2 = \sigma_2^2 = \sigma_u^2$), λ represents the correlation between η_{1i} and η_{2i} in:

$$\rho = (1/\sqrt{2}) * [\lambda/\sqrt{(1 + \lambda^2)}] \qquad (11)$$

I then compute the treatment effects using:

$$(1/n)\,\Sigma(\Phi\,[(\beta_0 + \beta_1 X_{ij} + \beta_2 imr_j + \beta_3 \varpi_k + \beta_4 S_i + u_i\,\lambda)/\sigma_1] - \Phi[(\beta_0 + \beta_1 X_{ij} + \beta_2 imr_j + \beta_3 \varpi_k + u_i\,\lambda)/\sigma_1]) \qquad (12)$$

Data and methods

Datasets

I obtained detailed information on individual health behaviors and household structural and demographic characteristics from Colombia's and Paraguay's 1990 Demographic and Health (DHSII) surveys. The sample for Colombia's 1990 DHSII was itself a subsample of the Departamento Nacional de Estadistica y Censo (DANE) "national master sample." The latter is based on the 1985 census and had been last updated in late 1989. A total of 713 DHS segments were surveyed, consisting of the smallest censal units- census tracts- drawn from 120 *municipios.* The sampling frame was constrained to include the country's three largest cities: Bogota,

Medellin and Cali. The Paraguayan DHS sample was drawn from 232 primary sampling units. The units were randomly sampled from lists of enumeration areas used in the country's 1982 census. In each unit an average of 25 households were interviewed. Thus, the Paraguayan DHS segments were roughly twice as large as the Colombian segments.

During visits to Bogotá and Asunción I had obtained lists of the particular census tracts and sectors from which the DHS II sample segments were selected. The lists allowed me to add the 1982 (Paraguay) and 1985 (Colombia) census identifiers (segment number) to each child record for later linkage to ministry of health and census data. Matching the data however was problematic. On the first attempt I was able to match 2674 of the Colombian child records (71%), leaving 1077 unmatched records distributed across 199 segments in 132 census sectors in 13 *municipios* and 12 *departmentos*. Correspondence with counterparts in PROFAMILIA, the executing agency for the DHS II/Colombia survey, established that the unmatched 199 segments were drawn not from the 1985 census but from a 1989 recensus of urban areas carried out by DANE (Correal 1990). PROFAMILIA counterparts eventually supplied 186 of the missing 199 census sector identifiers. Matching the Paraguayan DHS records with census identifiers was comparatively easier due to the higher level of aggregation and lack of recensus. Only 14 Paraguayan DHS clusters failed to match census sectors.

To obtain the instrumental variables I need for the Heckman and expanded HHP models I computed a series of contextual variables from 5% samples of Colombia's 1985 and Paraguay's 1982 national population censuses. Expressed as percentages, the variables aggregated the demographic and socioeconomic characteristics of mothers, households and working adults at

cluster, *municipio* and *departmento* levels. Included were measures of fertility and child mortality, migration patterns, household structure, marital status, quality of housing, home ownership, educational attainment, school enrollment, labor force participation and occupations. I used these primary data to construct a series of social structural measures, including socioeconomic level, inequality and social diversity (heterogeneity) indices. To obtain inequality measures I used educational attainment levels and housing quality to compute Gini indexes for each level of aggregation. I use the following formula (Theil 1972): $G= 1/2\ \Sigma\Sigma\ |(1/k)P_i - (1/k)P_j|$ where P_i and P_j are the weighted housing and educational scores for each of k=5 strata. The housing index consisted of a score based on four key housing characteristics: wall construction, floors, location of the kitchen and electricity. To measure social heterogeneity, in the sense of the distribution of nominally distinct groups within communities and municipalities, I use both diversity and entropy indexes. For the former I use Lieberson's (1969) formula: $D=(1-\Sigma P_i^2)/(1-1/k)$; after Theil (1967), the entropy index is expressed as: $H_{rel}= (-\Sigma P_i \log 2P_i)/\log 2K$, where K equals the number possible categories. Using this index, maximum diversity occurs when all nominal groups are equally represented (White 1986). I computed separate entropy indices for child mortality; adult literacy; female labor force participation; male and female occupations; and gender, marital status and nativity of household heads. Information on language spoken in the household was available for Paraguay but not Colombia. I used this variable to compute ethnic entropy and diversity indexes. I computed a third series of structural measures consisting of the simple Pearson correlations for each pair of inequality and heterogeneity measures on *municipio* and *departmento* levels. I then merged these recoded census files with the DHS files. The census files, however, failed to match 356 of the 3751

(10%) Colombian and 14 of 4698 Paraguayan child records. These unmatched Colombian cases were from 61 of the 713 DHS segments. Eight of the 61 unmatched segments were distributed across seven *municipios* in six *departmentos*. The census sector codes for these were simply missing from the DHS sample list. Of the remaining unmatched segments, 12 were from Medellin and 41 were from Barranquilla. Each of these segments had identifiable census sector codes, however those codes were not included in the 1985 census. I concluded that these represented newer settlements, which did not exist in 1985 but which DANE had subsequently added to the "national master sample". Unable to impute any census-based values for any of these 61 segments, I dropped them from the dataset. The final hierarchical datasets were structured as follows:

	Colombia	Paraguay
departmentos	24	31
municipios/distritos	120	156
clusters	713	254
households	8715	6348
women 15-45	5367	6262*
children <5 years	3751	4698
children 9-68 months	2912	3424

*includes women 15-49

Household-level variables

I model the associations between health behaviors and outcomes conditional on a set of well-known risk factors shown elsewhere to affect household health production. Based on exploratory multivariate logistic regressions, I reduced the household constraints on full immunization and attended delivery to four essentially orthogonal variables: the gender of the household head, number of live births, maternal educational attainment and spouse’s occupation. Female

headship is a constraint on household health production because such households have lower income and fewer social ties (Desai and Ahmad 1998). Exploration revealed that the number of live births is positively correlated with past child mortality and negatively correlated with both behavioral outcomes in both countries. The positive correlation of maternal education with household health production is well known (Boerma et al. 1990; Rutenberg et al. 1991). Possible mechanisms for this relationship include better cognitive skills, information and/or the superior wages better-educated mothers command in the labor market. Maternal education and maternal employment are indeed correlated in both countries. The relationship however is U-shaped rather than linear, suggesting that poorly educated and better-educated mothers are both more likely to be working, presumably for different reasons. I found that a second labor force measure, spouse's occupational prestige, explained a significant portion of variation in the exploratory multivariate models. In particular, a dummy for whether the spouse is engaged in agriculture correlated negatively with both health behavioral outcomes. In Paraguay a fifth household covariate, language spoken in the household, emerged as a strongly negative predictor of both outcomes. I interpret language as an indicator of ethnicity, which has been shown elsewhere to predict health behaviors and outcomes (Pebley, Goldman and Rodriguez 1996). Unfortunately, there are no ethnicity or language measures in the Colombian DHSII. Descriptive statistics for all household and community-level variables are shown in Table 3.

Instrumental variables

I used multivariate models to evaluate the census measures as potential instruments for the Heckman and bivariate probit models. For the Heckman selection models I fit simultaneous OLS and probit equations with the number of live births and an indicator for no child deaths as the

dependent variables. For the bivariate probit models I used binomial probits. In each equation I included the age and household risk factors along with the candidate instrumental variable. Descriptive statistics and actual parameter estimates for the instruments I selected are shown in Tables 4 and 5. For reliability, I select two instruments for each of the bivariate probit models.

An acceptable instrumental variable must be a significant predictor (at the 95% level) in a mortality or behavioral equation but an insignificant predictor in the corresponding live birth and outcomes equation. The instruments in two Colombian behavioral equations and in Paraguay's mortality selection equation fail to meet this criterion using the Huber/White/sandwich variance estimator. All, however, are significant at the 95% significance level when an uncorrected variance estimator is used. A second criterion is that there must be a nontrivial relationship between the instrument and endogenous dependent variable (Bound, Jaeger and Baker 1995). The sample means show that the instruments represent substantial population characteristics, suggesting that they meet this test from a statistical viewpoint.

Social interaction measures

To compute the social interaction measures I exploit the nested data structure of the multistage cluster DHS surveys (Institute for Resource Development/Westinghouse 1987). Within each randomly selected cluster, households were systematically sampled until a target number of reproductive-aged women was obtained. The respondents are thus neighbors, many of whom know and presumably influence one another. I use the proportions of sample children with cards and the proportion of sample mothers who used professional prenatal services in the index

municipio as proxies for the social utility of these behaviors. I compute these contextual variables prior to subsetting the dataset to only children eight months of age or older. To create the social interaction measures for each child in each dataset I used a series of N x (N-1) matrices. I operationalize adjacency in a geographic sense by assigning a weight of 1 to a child *i+1* living in the same *municipi*o as index child *i*; all others are weighted zero. I used a second N x (N-1) matrix containing age in months to generate a lag operator for each cell of the matrix. I constructed two more N x (N-1) matrices containing indicators for full immunization or censoring, and use or nonuse of professional delivery services at the last birth. I then computed the cross products of these matrices and summed the resulting matrices for each child (row). I computed the social interaction measures by dividing the numbers of older, fully immunized children and those whose mothers delivered under professional care by the total number of older children in the index *municipio*. The interaction measures are thus proportions between 0 and 1 appended to each child record.

Design effect

The fact I am not working with simple random samples presents another type of statistical problem. The DHS survey clusters are randomly sampled but households within clusters are systematically sampled until a requisite number of women ages 15-49 is interviewed. Health information is collected for all children under age five in each household. Observations are thus clustered within households and sample clusters. This design effect produces intra-class correlation, which artificially reduces the variance of the data collected. This in turn increases the probability of a Type I error (a false positive inference) because the standard errors of measured covariates are underestimated. This data artifact is unrelated to the population-level social

interaction effects I am modelling. To correct for this DHS survey design effect I use the Huber/White/sandwich variance estimator (StataCorp 1999: 256-60) in all models.

Model-building

I proceed in stepwise fashion, beginning with simple household-level models. I then add the selection correction and social interaction measures to each bivariate probit equation. If social interaction conditions individual health behaviors and outcomes then the rhos in the bivariate probits ought to be significant. The lagged mean behavioral measures ought to be positively correlated with all four dependent variables. If the social interaction coefficients are positive and the rhos and treatment effects decrease then I have circumstantial evidence that the behaviors of adjacent others affect individual behaviors and outcomes. If the treatment effects are substantial and remain so after adding the mean behavioral measures then I cannot rule out individual cognitive change as a cause of that outcome. Indeed, I expect that health choices are conditional on both social interaction and program treatment effects.

RESULTS

Baseline and selection models

Table 6 shows the results of four bivariate probits for cardholdership and full immunization, with and without adjustment for mortality selection. Looking first at the unadjusted baseline models, the rhos are significant and positive in each, suggesting that those with fully immunized children more closely resemble cardholders than non-cardholders. The net effect of these unmeasured factors, in other words, favors both choices. Among these unmeasured factors could be social interaction effects.

The adjusted models show that there is a significant mortality selection effect in all four equations. Interestingly, the inverse Mills ratios are negative in the Colombian equations and positive in the Paraguayan equations. In Colombia, mothers who have previously lost a child are less likely to have cards and fully immunized children than are those who have not lost a child. In Paraguay it is the reverse. In both immunization equations, adjusting for mortality selection clearly removes an upward bias on the child age dummies and household risk factor coefficients. If the immunization histories of dead children were included in the data then the risk effects on immunization would be that much more negative. Past child mortality, however, does not decisively affect the observed cardholdership predictors.

The baseline and selection model parameters are easier to interpret in Table 8, which shows the marginal effects of each observable with and without the inverse Mills ratio. Each coefficient can be interpreted as a probability conditional on the other observables weighted by their sample means. In Colombia, female headship has a borderline significant negative effect, reducing full immunization probability by 4 percent. Controlling for mortality selection makes this effect insignificant. On the other hand, the effect of birth order, poor maternal education and agricultural dependency all become considerably more negative in the adjusted Colombian immunization model. In Paraguay's unadjusted immunization equation, four of five unadjusted risk factors are significant in the expected negative direction; three become more negative with selection controlled. Of these, low or no maternal education is the most important, reducing immunization probability by 21 percent. This is more than double the effect of this variable in

Colombia. None of the risk factors is very important in any of the cardholdership models, suggesting that enrollment in both national EPI programs is relatively egalitarian.

Tables 7 and 9 show results for the unadjusted and adjusted maternal health models. Again the rhos are significantly positive, and they are much larger than the rhos in the immunization models. Those who choose professional delivery attendance have much more in common with prenatal versus non-prenatal users. As in the immunization models, part of this correlation could be due to social effects. The inverse Mills ratios are not significant in any of the adjusted equations but adding the selection correction nevertheless affects some parameter estimates. In Colombia, controlling for mortality selection moderates two risk factor effects on attended delivery. In Paraguay, however, the adjustment removes an upward bias on the same two delivery risk factors, revealing that those Paraguayan households that have lost a child are poorer than those that have not. Agrarian and non-Spanish speaking households emerge as particularly high-risk groups with respect to maternal health. Looking at the behavioral equations, risk factors are again seen to be less influential in the choice to enroll in a particular PHC program, with the exception of maternal education. Poorly educated mothers are less likely to enroll in prenatal services in both countries. In both equations, controlling for past mortality removes a spurious agrarian household effect on prenatal use. A possible explanation is that past mortality risk was higher in rural areas of both countries and the agrarian indicator actually measures this differential.

Summarizing the results so far, the HHP models show that socioeconomic differentials are modest with respect to PHC utilization. This is particularly clear for immunization

cardholdership. Prenatal use, on the other hand, is still conditioned by maternal education and, in Paraguay, ethnicity. Socioeconomic disparities are more evident with respect to health outcomes, particularly in Paraguay. Secondly, the results provide *prima facie* evidence that social interactions affect both outcomes. Thirdly, uncontrolled mortality selection subtly biases observed covariate effects and would have led to false inferences on several of the explanatory variables. These included female headship and maternal education in Colombia's immunization model, and maternal education and dependence on agriculture in both maternal health models. In light of these selection effects I retain the inverse Mills ratios in all subsequent models.

Treatment effects

Next, I extend the HHP models by estimating treatment effects. I calculate the treatment effects using predicted probabilities from the bivariate probits with instrumental variables added for identification. A comparison of Tables 10 and 11 to Tables 6 and 8 shows the seemingly unrelated model parameters are essentially unchanged by the addition of the instruments. The disturbance correlations are essentially identical to those reported in the adjusted baseline models, indicating that the instrumental variables are also uncorrelated with these unobservable effects. I conclude that the specification is robust.

The results show that treatment effects on immunization are rather modest in both counrties. Exposure to the routine EPI programs increases full immunization probabilities by 8-10 percent. Prenatal treatment effects are quite a bit larger, ranging from 25 percent in Colombia to 43 percent in Paraguay. It thus appears that household health production through maternal health is cognitively mediated while immunization is not. The weak immunization treatment effects could

reflect the fact that, during this period, many children were immunized through mass campaigns with little attendant health education from the routine EPI programs.

Social interactions

The treatment effects models in Tables 10 and 11 serve as baselines for the social interaction HHP models shown in Tables 12 and 14. In these models the proportions of older children with cards, and of older children whose mothers used prenatal services, have the expected positive effect on the respective outcomes. In Brock and Durlauf's (2000) terms, parents perceived significant social utility in choosing to immunize and professionally deliver their children. Comparison of the tables shows that the risk factor estimates in the outcome equations are again almost identical under both specifications. However, adding the mean behavioral estimates biases some of the coefficients in the behavioral equations, particularly maternal education, female headship and ethnicity. This is not surprising in that the mean behaviors and individual behaviors are likely affected by the same variables. The behavioral equations are clearly misspecified so further inferences on these parameters are risky.

The marginal effects on the outcomes are shown in Tables 13 and 15. The social interaction effects increase full immunization probabilities by 15 percent in Colombia and 28 percent in Paraguay. In each country the social interaction effect exceeds that of any of the household risk factors and, in both, they far exceed the routine immunization program treatment effects. This suggests that the immunization choices of parents were more strongly influenced by the observed immunization behaviors of others than by the knowledge they received through the PHC system. The pattern is rather different for maternal health. In Colombia, the observed proportion of local

mothers using prenatal services and actual enrollment in those services have roughly the same influence on the choice of attended delivery. In Paraguay, in contrast, the prenatal treatment effect is substantially larger than the social interaction effect. This weaker social effect may reflect generally weaker normative effects. Only about half of Paraguayan mothers seek professional delivery, compared to 82 percent of Colombian mothers.

The behavior of the unobservables in these social interaction models is also informative. In Paraguay's immunization model, rho falls from 0.16 to 0.11, a decrease of 31 percent. The rhos are little affected in any of the other models. For the case of immunization in Paraguay, then, the social interaction term is both significant as a predictor and explains a sizeable portion of the unobservable variables that affect both immunization choices. This is strong evidence that Paraguayan parents perceived considerable social utility in deciding to immunize their children.

DISCUSSION AND CONCLUSIONS

This exploration has confirmed some well-known relationships and revealed some new insights into household health production that help explain the robustness of contemporary mortality transitions. Comparing different countries increases external validity while comparing different behaviors and outcomes reveals the heterogeneity underlying household health production. Expanding the analytical model allows a direct comparison of the effects of traditional household constraints, PHC program participation and social interactions on health outcomes.

I conclude that considerable health disparities still characterized both Colombia and Paraguay during the 1985-90 period. The disparities, reflected in the persistently significant household risk factors, were generally greater in Paraguay and they affected maternal health more than immunization. By controlling for sample selection on past mortality I avoided Type I inferential errors on observable risk factors in three of the four models.

The treatment effects models show that exposure to PHC programs increases the likelihood of health behavioral change, over and above household risk factor effects. Treatment effects appear to be much more important to maternal health than to childhood immunization. The treatment effects, however, do not fully offset the negative risk factor effects for either outcome.

I find consistent evidence that social interactions constitute a third causal mechanism of health behavioral change, separate and distinct from individual constraints and treatment effects. In all four bivariate probit models the social interaction effects rivaled or surpassed those of observed risk factors and of treatment effects. The social interaction effects are strongest for immunization in Paraguay, and for maternal health in both countries. Reliance on mass campaigns may explain Paraguay's large social immunization effect. When immunization occurs through popular mobilization actors can more intensively evaluate others and this causes them to accord higher social utility to their behavioral choices. On the other hand, no analogous large-scale interventions were mounted for maternal health over this period in either country, yet the social interaction effects are large and positive. One might speculate that the routine, normative social effects attending childbirth are relatively stronger than the campaign-influenced immunization interaction effects.

Assuming that household risk factor, treatment and social interaction effects are additive, a high-risk Colombian mother exposed to the mean social interaction effect and participating in the routine immunization program would be about as likely to immunize her child as would a low-risk, non-participating Colombian mother in an anomic setting. For delivery, either prenatal program participation or social interaction would be sufficient to offset her household risk factor effects. For a high-risk Paraguayan mother, the combined program treatment and social interaction effects would not be sufficient to offset the household risk factor effects on immunization but the social interaction effect alone would be sufficient in the case of attended delivery. These results, I conclude, support the hypothesis that Latin America's mortality transition continued apace through the 1980s due to a combination of PHC program treatment and social interaction effects.

(end text)

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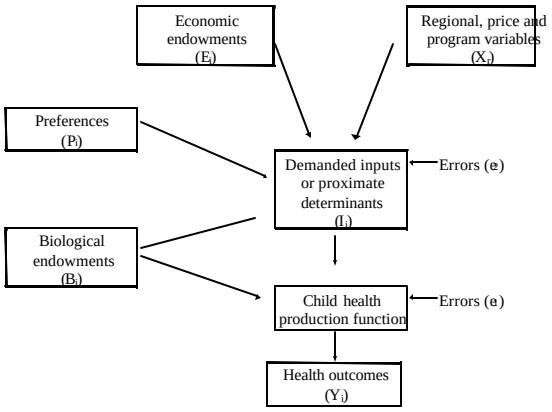
Figure 1. Household health production model



source: Schultz 1984.

Table 1. Selected demographic indicators, Colombia and Paraguay, 1950-1995.

	50-55	55-60	60-65	65-70	70-75	75-80	80-85	85-90	90-95
Colombia									
Life expectancy at birth	50.6	55.2	57.9	60.1	61.7	64	67.2	68.3	69.3
Deaths/1000 pop.	16.7	13.4	11.5	10.1	8.6	7.6	6.4	6.1	6
Infant mortality rate	123	105	92	82	73	59	41	40	37
Maternal deaths/100,000 births[1]						126	86.1	107	
Births/woman age 49[2]	6.76		6.76		4.66		3.51		2.67
Births/1000 pop.	47.3	45.4	44.2	41.6	32.6	31.7	29.4	25.9	24
Migrants/1000 pop.[3]	-2.38	-2.74	-2.94	-2.94	-2.58	-2.23	-1.93	-1.58	-1.37
Population growth rate	2.82	2.94	2.98	2.86	2.32	2.29	2.09	1.97	1.85
Paraguay									
Life expectancy at birth	62.6	63.2	64.4	65	65.6	66	67.4	68.7	70
Deaths/1000 pop.	9.3	8.9	8.1	7.6	7.2	6.8	6.4	6	5.5
Infant mortality rate	159	148	136	126	110	100	82	70	64
Maternal deaths/100,000 births[1]						469	365	150	
Births/woman age 49[2]	6.8		6.8		5.65		4.82		4.34
Births/1000 pop.	47.3	44.5	42.3	39.5	36.6	34.6	36.1	36.1	33
Migrants/1000 pop.[3]	-10.29	-8.97	-5.27	-4.58	-3.2	3.47	2.96	1.01	.22
Population growth rate	2.78	2.67	2.9	2.7	2.6	3.19	3.2	2.91	2.69

sources: [1]United Nations Population Division. 1994. Urban and rural areas 1950-2025 (The 1994 revision). New York: United Nations.

[2] a) World Health Organization 1996. b) United Nations 1993. c) UCLA 1996, P. 147

[3] CELADE. 1997.

Table 2. Selected primary health care indicators, 1980-87

Indicator/Country	Year	Colombia	Paraguay
infant mortality rate[1]	80-82	46	46
	86-87	40	43
access to safe water[2] (%)	80-82	-	25
	86-87	92*	33
immunization: DPT3,0-11m (%)	80-82	21	26
	86-87	57	55
immunization: measles <12m (%)	80-82	22	20
	86-87	58	51
access to PHC services[3] (%)	80-82	88	-
	86-87	87	-
prenatal care use (%)	80-82	65*	65
	86-87	65	57
delivery attendance (%)	80-82	51*	22
	86-87	51	30

Source: WHO. 1991.

*1983-85

Notes: [1] deaths below 12 months of age/1000 live births.

[2] households with water piped in home or within 15 min. walk

Table 3. Means and standard deviations of individual and community-level variables, Colombia and Paraguay DHSII, 1990

	Colombia		Paraguay	
Variable	**Mean**	**Std. Dev.**	**Mean**	**Std. Dev.**
Individual-level variables				
child age 18-23m	0.120	0.325	0.105	0.307
child age 24-29m	0.122	0.327	0.107	0.309
child age 30-35m	0.118	0.323	0.111	0.314
child age 36-41m	0.114	0.318	0.104	0.305
child age 42-47m	0.114	0.318	0.097	0.296
child age 48-53m	0.110	0.313	0.102	0.303
child age 54m+	0.115	0.320	0.221	0.415
maternal age <20 y	0.042	0.200	0.041	0.198
maternal age 30-39 y	0.336	0.472	0.371	0.483
maternal age 40-45 y	0.049	0.215	0.124	0.330
female hh head	0.160	0.366	0.086	0.281
birth order	2.592	1.950	3.977	2.984
live births	2.924	2.025	4.588	3.083
mother primary or no ed	0.494	0.500	0.783	0.412
spouse farmer	0.060	0.238	0.487	0.500
no Spanish			0.478	0.500
fully immunized	0.707	0.455	0.489	0.500
has card	0.434	0.496	0.355	0.478
attended delivery	0.833	0.373	0.533	0.499
prenatal use	0.834	0.372	0.753	0.431
Community-level variables				
vaccine coverage 1986[a]	56.60	32.09	30.00	27.75
vaccine coverage 1988-89[b]	86.00	15.12	46.49	35.28
proportion older children fully immunized	.732	.159	.509	.262
proportion older children professionally delivered	.824	.223	.510	.282
n	2912		3424	

[a] Colombia: percent children below age 1 in municipio who received OPV3; Paraguay: percent children below age 2 in cluster who received DPT3.
[b] Colombia 1988, Paraguay 1989.

Table 4. Probit estimates, instrumental variables, Colombia 1990

Instrument	Sample mean	Std. Dev.	Coefficient	S.E.	t-ratio	Coefficient	S.E.	t-ratio
Model[a]			*no child death*			*live births*		
Municipal occupational concentration index	0.331	0.057	-1.069	0.531	-2.012	-0.280	0.207	-1.354
Model[b]			*cardholdership*			*immunization*		
Proportion dept. female lf self-employed	0.312	0.058	-1.091	0.522	-2.089	-0.573	0.498	-1.149
Prop municipal female lf laborers	0.086	0.086	0.606	0.349	1.736	0.339	0.423	0.801
Model[b]			*prenatal*			*delivery*		
Tract female labor force diversity index	0.614	0.371	-0.184	0.101	-1.826	-0.031	0.123	-0.249
Municipal hh union status diversity index	0.836	0.119	-0.710	0.313	-2.270	-0.284	0.358	-0.792

[a] live births estimated by OLS, other independent variables are those shown in Table 1.1.

[b] Other independent variables modeled are shown in Table 1.1.

Table 5. Probit estimates, instrumental variables, Paraguay 1990

Instrument	Sample mean	Std. Dev.	Coefficient	S.E.	t-ratio	Coefficient	S.E.	t-ratio
Model[a]			*no child deaths*			*live births*		
Tract female labor force participation entropy	0.624	0.122	-0.457	0.293	-1.557	0.431	0.386	1.118
Model[b]			*cardholdership*			*immunization*		
Prop tract households non-Spanish speaking	0.036	0.121	0.482	0.219	2.205	0.380	0.288	1.322
corr munic housing index x munic language entropy	-0.299	0.304	0.255	0.110	2.318	0.047	0.110	0.423
Model[b]			*prenatal*			*delivery*		
Prop municipal households rural	0.551	0.497	0.164	0.082	1.994	-0.004	0.071	-0.05
Municipal occ prestige score	0.330	0.235	-0.331	0.168	-1.969	0.011	0.149	0.075

[a] live births estimated by OLS, other independent variables are those shown in Table 1.1

[b] Other independent variables modeled are shown in Table 1.1.

Table 6. Bivariate probit estimates of full immunization, adjusted for mortality selection

	Colombia (n=2912)						Paraguay (n=3424)					
	unadjusted			adjusted			unadjusted			adjusted		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Immunization												
Constant	0.283	0.072	3.943	0.830	0.152	5.473	0.248	0.077	3.232	0.206	0.077	2.680
child age 18-23m	0.385	0.090	4.252	0.363	0.090	4.027	0.416	0.091	4.576	0.406	0.092	4.414
child age 24-29m	0.586	0.098	5.986	0.558	0.098	5.691	0.438	0.099	4.427	0.429	0.099	4.345
child age 30-35m	0.445	0.093	4.799	0.405	0.094	4.300	0.493	0.085	5.830	0.476	0.085	5.605
child age 36-41m	0.592	0.094	6.313	0.549	0.095	5.807	0.625	0.084	7.431	0.602	0.085	7.060
child age 42-47m	0.541	0.092	5.845	0.501	0.094	5.343	0.684	0.093	7.359	0.648	0.094	6.896
child age 48-53m	0.640	0.095	6.723	0.577	0.098	5.905	0.628	0.086	7.323	0.585	0.088	6.682
child age 54m+	0.682	0.093	7.334	0.615	0.095	6.476	0.662	0.075	8.798	0.606	0.076	7.927
female hh head	-0.118	0.070	-1.696	-0.042	0.072	-0.578	-0.078	0.098	-0.798	-0.093	0.098	-0.942
birth order	-0.027	0.014	-1.936	-0.059	0.016	-3.650	-0.024	0.010	-2.416	-0.063	0.016	-4.042
mother primary or no ed	-0.134	0.058	-2.303	-0.261	0.066	-3.943	-0.410	0.069	-5.971	-0.567	0.081	-7.018
no Spanish							-0.365	0.074	-4.949	-0.357	0.074	-4.811
spouse farmer	-0.254	0.109	-2.337	-0.348	0.110	-3.153	-0.335	0.073	-4.589	-0.420	0.075	-5.604
inverse Mills ratio				-0.968	0.234	-4.145				1.062	0.292	3.639
Cardholdership												
Constant	0.213	0.070	3.040	0.628	0.144	4.362	-0.255	0.081	-3.150	-0.277	0.080	-3.457
child age 18-23m	-0.069	0.090	-0.766	-0.086	0.090	-0.955	0.099	0.085	1.163	0.091	0.085	1.072
child age 24-29m	-0.247	0.092	-2.679	-0.271	0.093	-2.930	0.012	0.085	0.138	0.004	0.085	0.053
child age 30-35m	-0.364	0.088	-4.142	-0.399	0.089	-4.482	-0.023	0.089	-0.258	-0.036	0.090	-0.396
child age 36-41m	-0.374	0.086	-4.343	-0.411	0.087	-4.709	0.073	0.090	0.812	0.058	0.091	0.635
child age 42-47m	-0.356	0.089	-3.996	-0.389	0.090	-4.331	-0.033	0.089	-0.375	-0.055	0.091	-0.612
child age 48-53m	-0.413	0.090	-4.612	-0.465	0.092	-5.076	-0.082	0.090	-0.910	-0.108	0.091	-1.183
child age 54m+	-0.446	0.090	-4.973	-0.502	0.092	-5.467	-0.242	0.074	-3.251	-0.276	0.077	-3.592
female hh head	-0.240	0.072	-3.342	-0.182	0.074	-2.462	-0.156	0.106	-1.464	-0.164	0.106	-1.551
birth order	-0.050	0.015	-3.356	-0.075	0.017	-4.374	0.001	0.009	0.164	-0.020	0.014	-1.436
mother primary or no ed	0.099	0.057	1.731	0.004	0.063	0.059	-0.042	0.075	-0.563	-0.128	0.091	-1.412
no Spanish							-0.079	0.074	-1.072	-0.073	0.073	-0.998

Table 6, continued. Bivariate probit estimates of full immunization, adjusted for mortality selection

	Colombia (n=2912)						Paraguay (n=3424)					
	unadjusted			adjusted			unadjusted			adjusted		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
spouse farmer	-0.044	0.135	-0.328	-0.114	0.137	-0.829	0.011	0.082	0.135	-0.034	0.082	-0.415
inverse Mills ratio				-0.733	0.220	-3.330				0.582	0.274	2.121
rho[a]	0.163	0.034	4.734	0.154	0.034	4.469	0.171	0.037	4.615	0.165	0.037	4.427
log-likelihood	-3642.3			-3626.7			-4322.5			-4308.5		

[a] rho is the disturbance correlation.

Table 7. Bivariate probit estimates of attended delivery, adjusted for mortality selection

	Colombia (n=2912)						Paraguay (n=3424)					
	unadjusted			adjusted			unadjusted			adjusted		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Delivery												
Constant	1.792	0.083	21.511	1.301	0.938	1.387	1.027	0.079	12.991	0.958	0.106	9.013
maternal age < 20y	-0.152	0.161	-0.944	-0.424	0.525	-0.808	-0.108	0.135	-0.799	-0.014	0.163	-0.083
maternal age 30-39	0.463	0.091	5.104	0.649	0.368	1.764	0.171	0.071	2.402	0.052	0.126	0.413
maternal age 40-45	0.655	0.170	3.850	0.928	0.537	1.728	0.382	0.118	3.239	-0.041	0.397	-0.104
female hh head	0.068	0.095	0.717	-0.018	0.189	-0.096	-0.320	0.096	-3.326	-0.329	0.096	-3.421
live births	-0.151	0.023	-6.569	-0.152	0.023	-6.603	-0.082	0.013	-6.237	-0.084	0.014	-6.186
mother primary or no ed	-0.789	0.084	-9.424	-0.627	0.323	-1.942	-0.255	0.082	-3.127	-0.438	0.183	-2.390
no Spanish							-0.453	0.076	-5.990	-0.456	0.076	-6.012
spouse farmer	-0.383	0.150	-2.549	-0.289	0.244	-1.184	-0.445	0.069	-6.485	-0.540	0.100	-5.424
inverse Mills ratio				0.935	1.768	0.529				1.022	0.958	1.067
Prenatal												
Constant	1.845	0.073	25.368	1.615	0.919	1.756	2.010	0.103	19.598	2.093	0.137	15.260
maternal age < 20y	-0.347	0.136	-2.555	-0.476	0.523	-0.911	-0.305	0.150	-2.032	-0.425	0.201	-2.114
maternal age 30-39	0.414	0.091	4.566	0.503	0.370	1.358	0.155	0.084	1.843	0.325	0.174	1.862
maternal age 40-45	0.523	0.174	3.000	0.653	0.529	1.236	0.249	0.136	1.835	0.814	0.592	1.376
female hh head	-0.091	0.098	-0.927	-0.132	0.197	-0.668	-0.258	0.110	-2.346	-0.249	0.111	-2.248
live births	-0.158	0.021	-7.520	-0.158	0.021	-7.453	-0.077	0.016	-4.942	-0.075	0.016	-4.699
mother primary or no ed	-0.753	0.080	-9.442	-0.676	0.319	-2.122	-0.689	0.118	-5.844	-0.457	0.263	-1.738
no Spanish							-0.482	0.086	-5.582	-0.479	0.087	-5.475
spouse farmer	-0.338	0.139	-2.435	-0.293	0.233	-1.259	-0.244	0.097	-2.532	-0.124	0.150	-0.828
inverse Mills ratio				0.439	1.731	0.254				-1.273	1.355	-0.939
rho[a]	0.499	0.040	12.453	0.499	0.040	12.463	0.611	0.032	19.130	0.612	0.032	18.992
log-likelihood	-2157.3			-2157.1			-3516.3			-3512.9		

[a] rho is the disturbance correlation.

Note: Huber/White/sandwich variance estimators used in all models.

Table 8. Bivariate probit marginal effects on child immunization, adjusted for mortality selection

	Colombia (n=2912)				Paraguay (n=3424)			
	unadjusted		adjusted		unadjusted		adjusted	
Independent variable	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**
Immunization								
child age 18-23m[a]	0.116	4.865	0.110	4.536	0.144	4.736	0.139	4.604
child age 24-29m	0.168	7.884	0.161	7.375	0.151	5.035	0.147	4.894
child age 30-35m	0.132	5.714	0.121	5.06	0.169	5.815	0.163	5.555
child age 36-41m	0.168	7.803	0.157	7.022	0.214	7.495	0.205	7.121
child age 42-47m	0.157	7.076	0.146	6.384	0.232	8.136	0.220	7.564
child age 48-53m	0.180	8.567	0.165	7.399	0.213	7.409	0.199	6.767
child age 54m+	0.189	9.235	0.173	7.931	0.227	9.507	0.208	8.411
female hh head	-0.040	-1.741	-0.014	-0.585	-0.027	-0.955	-0.032	-1.122
birth order	-0.009	-2.133	-0.018	-4.309	-0.009	-2.956	-0.022	-5.639
mother primary or no ed	-0.046	-2.585	-0.088	-4.434	-0.151	-6.465	-0.206	-8.120
no Spanish					-0.135	-6.806	-0.131	-6.638
spouse farmer	-0.088	-2.359	-0.123	-3.155	-0.123	-5.872	-0.155	-7.055
inverse Mills ratio			-0.328	-4.706			0.344	5.559
Cardholdership								
child age 18-23m	-0.026	-0.796	-0.033	-1.001	0.037	1.121	0.034	1.030
child age 24-29m	-0.093	-2.963	-0.101	-3.259	0.004	0.119	0.001	0.035
child age 30-35m	-0.135	-4.420	-0.146	-4.848	-0.009	-0.281	-0.014	-0.429
child age 36-41m	-0.138	-4.474	-0.150	-4.937	0.026	0.798	0.020	0.619
child age 42-47m	-0.132	-4.284	-0.143	-4.692	-0.013	-0.392	-0.021	-0.641
child age 48-53m	-0.153	-4.968	-0.169	-5.595	-0.030	-0.942	-0.039	-1.232
child age 54m+	-0.163	-5.418	-0.181	-6.111	-0.087	-3.389	-0.099	-3.842
female hh head	-0.091	-3.763	-0.069	-2.748	-0.056	-1.976	-0.059	-2.076
birth order	-0.020	-3.781	-0.029	-5.042	0.000	0.150	-0.008	-1.739
mother primary or no ed	0.038	1.983	0.002	0.082	-0.015	-0.654	-0.048	-1.816
no Spanish					-0.029	-1.501	-0.027	-1.391

Table 8., continued. Bivariate probit marginal effects on child immunization, adjusted for mortality selection

	Colombia (n=2912)				Paraguay (n=3424)			
	unadjusted		adjusted		unadjusted		adjusted	
Independent variable	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**
spouse farmer	-0.017	-0.444	-0.043	-1.114	0.004	0.206	-0.013	-0.586
inverse Mills ratio			-0.269	-3.884			0.219	2.746

[a] These coefficients are normalized. For the j'th variable, B_j phi(z) represents the change in probability of full immunization for a unit change in j where z= $phi^{-1}(p)$, p is the sample mean of the response variable and Bj is the probit coefficient for the variable.

Note: Huber/White/sandwich variance estimators used in all models.

Table 9. Bivariate probit marginal effects on maternal health, adjusted for mortality selection

	Colombia (n=2912)				Paraguay (n=3424)			
	unadjusted		adjusted		unadjusted		adjusted	
Independent variable	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**	**Coefficient**	**t-ratio**
Delivery								
maternal age < 20y[a]	-0.030	-0.907	-0.072	-0.614	-0.039	-0.955	-0.005	-0.118
maternal age 30-39	0.091	6.670	0.111	2.077	0.058	3.060	0.017	0.498
maternal age 40-45	0.107	6.175	0.124	2.913	0.125	4.272	-0.017	-0.169
female hh head	0.018	1.053	0.007	0.203	-0.110	-4.013	-0.113	-4.110
live births	-0.032	-9.410	-0.032	-9.395	-0.028	-7.645	-0.029	-7.806
mother primary or no ed	-0.171	-12.141	-0.149	-2.618	-0.089	-3.796	-0.154	-3.148
no Spanish					-0.165	-8.337	-0.166	-8.380
spouse farmer	-0.091	-3.198	-0.075	-1.573	-0.162	-7.629	-0.199	-6.173
inverse Mills ratio			0.118	0.419			0.318	1.747
Prenatal								
maternal age < 20y	-0.079	-2.216	-0.089	-0.759	-0.085	-2.291	-0.130	-2.861
maternal age 30-39	0.082	5.964	0.087	1.614	0.049	2.903	0.101	3.242
maternal age 40-45	0.089	4.570	0.094	1.680	0.074	3.130	0.194	4.078
female hh head	-0.018	-0.988	-0.020	-0.595	-0.077	-2.830	-0.073	-2.699
live births	-0.033	-9.762	-0.033	-9.705	-0.022	-7.069	-0.021	-6.826
Mother primary or no ed	-0.163	-11.622	-0.159	-2.894	-0.168	-9.428	-0.110	-2.812
no Spanish					-0.134	-8.370	-0.133	-8.310
Spouse farmer	-0.078	-2.792	-0.075	-1.615	-0.066	-3.876	-0.026	-0.943
Inverse Mills ratio			0.026	0.089			-0.451	-1.905

[a] These coefficients are normalized. For the j'th variable, B_j phi(z) represents the change in probability of full immunization for a unit change in j where z= $phi^{-1}(p)$, p is the sample Mean of the response variable and Bj is the probit coefficient for the variable.
Note: Huber/White/sandwich variance estimators used in all models.

Table 10. Baseline bivariate probit treatment effects immunization models, DHSII 1990

	Colombia (n=2912)						Paraguay (n= 3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Immunization												
Constant	0.830	0.152	5.474	0.821	0.153	5.373	0.207	0.077	2.681	0.206	0.077	2.679
child age 18-23m	0.363	0.090	4.027	0.372	0.092	4.057	0.406	0.092	4.412	0.406	0.092	4.413
child age 24-29m	0.558	0.098	5.691	0.556	0.099	5.614	0.429	0.099	4.346	0.429	0.099	4.346
child age 30-35m	0.405	0.094	4.299	0.403	0.095	4.236	0.476	0.085	5.605	0.476	0.085	5.605
child age 36-41m	0.549	0.095	5.807	0.530	0.095	5.552	0.602	0.085	7.060	0.602	0.085	7.061
child age 42-47m	0.501	0.094	5.344	0.509	0.094	5.408	0.648	0.094	6.897	0.648	0.094	6.896
child age 48-53m	0.577	0.098	5.906	0.577	0.099	5.837	0.585	0.088	6.682	0.585	0.088	6.683
child age 54m+	0.615	0.095	6.475	0.609	0.096	6.369	0.606	0.076	7.927	0.606	0.076	7.926
female hh head	-0.042	0.072	-0.578	-0.040	0.072	-0.550	-0.093	0.098	-0.942	-0.092	0.098	-0.940
birth order	-0.059	0.016	-3.652	-0.059	0.016	-3.621	-0.063	0.016	-4.041	-0.063	0.016	-4.041
mother primary or no ed	-0.261	0.066	-3.943	-0.255	0.066	-3.850	-0.567	0.081	-7.017	-0.567	0.081	-7.017
no Spanish							-0.357	0.074	-4.811	-0.357	0.074	-4.811
spouse farmer	-0.348	0.110	-3.152	-0.345	0.112	-3.089	-0.420	0.075	-5.604	-0.420	0.075	-5.605
Inverse Mills ratio	-0.968	0.234	-4.147	-0.947	0.235	-4.028	1.062	0.292	3.638	1.063	0.292	3.639
Cardholdership												
Constant	0.878	0.207	4.239	0.569	0.150	3.805	-0.290	0.080	-3.621	-0.203	0.086	-2.361
child age 18-23m	-0.080	0.090	-0.885	-0.107	0.092	-1.171	0.095	0.085	1.112	0.099	0.086	1.163
child age 24-29m	-0.268	0.092	-2.900	-0.279	0.094	-2.971	0.007	0.085	0.085	0.007	0.085	0.087
child age 30-35m	-0.392	0.089	-4.405	-0.389	0.090	-4.342	-0.034	0.090	-0.380	-0.030	0.090	-0.339
child age 36-41m	-0.407	0.087	-4.678	-0.416	0.089	-4.694	0.061	0.091	0.672	0.051	0.090	0.570
child age 42-47m	-0.375	0.090	-4.186	-0.399	0.090	-4.413	-0.052	0.091	-0.575	-0.059	0.091	-0.649
child age 48-53m	-0.459	0.091	-5.029	-0.459	0.093	-4.947	-0.102	0.091	-1.121	-0.105	0.090	-1.164
child age 54m+	-0.496	0.092	-5.391	-0.518	0.093	-5.593	-0.273	0.077	-3.552	-0.279	0.077	-3.629
female hh head	-0.187	0.074	-2.542	-0.175	0.075	-2.354	-0.160	0.106	-1.511	-0.152	0.107	-1.423
birth order	-0.072	0.017	-4.211	-0.072	0.017	-4.112	-0.019	0.014	-1.394	-0.021	0.013	-1.571
mother primary or no ed	0.016	0.064	0.253	0.007	0.064	0.115	-0.129	0.091	-1.422	-0.155	0.091	-1.693
no Spanish							-0.085	0.074	-1.154	-0.049	0.073	-0.674

Table 10., continued. Baseline bivariate probit treatment effects immunization models, DHSII 1990

	Colombia (n=2912)						Paraguay (n= 3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
spouse farmer	-0.098	0.138	-0.705	-0.128	0.140	-0.917	-0.033	0.082	-0.405	-0.016	0.082	-0.191
Proportion departmento female lf self-employed	-0.912	0.519	-1.759									
Proportion municipio female lf laborers				0.569	0.346	1.646						
Proportion of tract households non-Spanish							0.438	0.204	2.143			
Municipio housing gini, language entropy corr										0.254	0.106	2.392
Inverse Mills ratio	-0.691	0.220	-3.144	-0.709	0.223	-3.186	0.573	0.275	2.087	0.598	0.275	2.176
rho[a]	0.153	0.034	4.438	0.146	0.035	4.186	0.163	0.037	4.391	0.164	0.038	4.376
treatment effect	0.081	0.000		0.077	0.000		0.094	0.000		0.095	0.000	
log-likelihood	-3624.2			-3557.4			-4305.6			-4302.6		

[a] rho is the disturbance correlation.

Note: Huber/White/sandwich variance estimators used in all models.

Table 11. Baseline bivariate probit treatment effects maternal health models, DHSII 1990

	Colombia (n=2912)						Paraguay (n=3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Delivery												
Constant	1.302	0.938	1.388	1.298	0.938	1.384	0.944	0.076	12.390	0.943	0.076	12.376
maternal age < 20y	-0.424	0.525	-0.808	-0.426	0.525	-0.811	0.017	0.129	0.130	0.016	0.129	0.127
maternal age 30-39	0.649	0.368	1.764	0.650	0.368	1.767	0.019	0.101	0.190	0.019	0.101	0.190
maternal age 40-45	0.926	0.536	1.727	0.928	0.537	1.729	-0.149	0.307	-0.487	-0.151	0.307	-0.492
female hh head	-0.019	0.189	-0.100	-0.018	0.189	-0.096	-0.331	0.082	-4.051	-0.329	0.082	-4.032
live births	-0.152	0.023	-6.608	-0.152	0.023	-6.590	-0.085	0.011	-7.659	-0.085	0.011	-7.656
mother primary or no ed	-0.626	0.323	-1.942	-0.626	0.323	-1.939	-0.473	0.135	-3.497	-0.474	0.135	-3.503
No Spanish							-0.501	0.059	-8.449	-0.502	0.059	-8.460
spouse farmer	-0.289	0.244	-1.185	-0.289	0.244	-1.182	-0.546	0.079	-6.898	-0.546	0.079	-6.897
inverse Mills ratio	0.934	1.767	0.528	0.940	1.768	0.531	1.282	0.703	1.823	1.287	0.703	1.830
Prenatal												
Constant	1.725	0.920	1.875	2.283	0.952	2.397	2.008	0.108	18.602	1.613	0.225	7.159
maternal age < 20y	-0.462	0.522	-0.886	-0.397	0.522	-0.761	-0.428	0.147	-2.904	-0.396	0.145	-2.730
maternal age 30-39	0.503	0.370	1.360	0.450	0.369	1.218	0.335	0.129	2.598	0.288	0.125	2.303
maternal age 40-45	0.655	0.526	1.245	0.580	0.526	1.102	0.826	0.400	2.066	0.668	0.387	1.727
female hh head	-0.122	0.197	-0.619	-0.108	0.197	-0.552	-0.262	0.090	-2.911	-0.251	0.090	-2.795
live births	-0.158	0.021	-7.406	-0.158	0.021	-7.405	-0.076	0.012	-6.604	-0.077	0.012	-6.700
mother primary or no ed	-0.686	0.318	-2.153	-0.728	0.319	-2.285	-0.480	0.172	-2.796	-0.552	0.171	-3.230
No Spanish							-0.433	0.067	-6.485	-0.477	0.067	-7.103
spouse farmer	-0.285	0.233	-1.225	-0.349	0.233	-1.495	-0.156	0.095	-1.639	-0.225	0.098	-2.307
Tract female lf diversity index	-0.174	0.088	-1.972									
Municipio union status diversity index				-0.628	0.296	-2.125						
Proportion municipio households rural							0.178	0.047	3.745			

Table 11, continued. Baseline bivariate probit treatment effects maternal health models, DHSII 1990

	Colombia (n=2912)						Paraguay (n=3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Municipal occupational prestige score										0.121	0.056	2.166
treatment effect	0.255	0.001		0.255	0.001		0.433	0.001		0.434	0.001	
inverse Mills ratio	0.434	1.731	0.251	0.184	1.731	0.106	-1.268	0.877	-1.446	-0.900	0.851	-1.058
rho[a]	0.499	0.040	12.601	0.498	0.040	12.450	0.614	0.024	25.903	0.616	0.024	26.036
log-likelihood	-2154.3			-2153.5			.3505.9			-3510.6		

[a] rho is the disturbance correlation.

Note: Huber/White/sandwich variance estimators used in all models.

Table 12. Bivariate probit social interaction immunization models, DHSII 1990

	Colombia (n=2912)						Paraguay (n= 3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Immunization												
Constant	0.653	0.149	4.373	0.653	0.149	4.372	-0.013	0.083	-0.150	-0.013	0.083	-0.152
child age 18-23m	0.414	0.091	4.550	0.414	0.091	4.550	0.457	0.092	4.991	0.457	0.092	4.991
child age 24-29m	0.631	0.095	6.670	0.631	0.095	6.669	0.496	0.092	5.376	0.496	0.092	5.376
child age 30-35m	0.497	0.095	5.224	0.497	0.095	5.223	0.562	0.091	6.166	0.562	0.091	6.167
child age 36-41m	0.647	0.098	6.592	0.647	0.098	6.590	0.700	0.093	7.489	0.700	0.093	7.490
child age 42-47m	0.601	0.100	6.035	0.601	0.100	6.034	0.767	0.095	8.086	0.767	0.095	8.086
child age 48-53m	0.686	0.102	6.692	0.686	0.102	6.691	0.731	0.094	7.793	0.731	0.094	7.794
child age 54m+	0.717	0.102	7.023	0.717	0.102	7.023	0.771	0.082	9.371	0.771	0.082	9.371
female hh head	-0.029	0.070	-0.416	-0.029	0.070	-0.416	-0.096	0.080	-1.200	-0.096	0.080	-1.199
birth order	-0.056	0.015	-3.722	-0.056	0.015	-3.722	-0.063	0.012	-5.413	-0.063	0.012	-5.413
mother primary or no ed	-0.269	0.062	-4.337	-0.269	0.062	-4.336	-0.572	0.070	-8.116	-0.572	0.070	-8.116
no Spanish							-0.403	0.053	-7.562	-0.403	0.053	-7.562
spouse farmer	-0.344	0.106	-3.252	-0.344	0.106	-3.251	-0.424	0.058	-7.255	-0.424	0.058	-7.255
Proportion older children in municipio with cards	0.469	0.147	3.191	0.469	0.147	3.189	0.806	0.130	6.219	0.806	0.130	6.219
inverse Mills ratio	-0.937	0.216	-4.329	-0.937	0.216	-4.329	1.049	0.222	4.730	1.049	0.222	4.730
Cardholdership												
Constant	-0.868	0.242	-3.586	-1.001	0.200	-5.003	-1.084	0.092	-11.820	-1.036	0.096	-10.800
child age 18-23m	0.474	0.114	4.179	0.480	0.115	4.170	0.256	0.091	2.819	0.257	0.091	2.834
child age 24-29m	0.455	0.114	3.993	0.468	0.116	4.046	0.220	0.094	2.335	0.220	0.094	2.325
child age 30-35m	0.524	0.124	4.237	0.534	0.125	4.266	0.252	0.095	2.657	0.253	0.095	2.667
child age 36-41m	0.563	0.122	4.625	0.579	0.122	4.728	0.380	0.095	3.981	0.374	0.096	3.912
child age 42-47m	0.600	0.135	4.451	0.615	0.136	4.515	0.331	0.101	3.267	0.326	0.101	3.222
child age 48-53m	0.611	0.134	4.545	0.627	0.135	4.632	0.374	0.098	3.811	0.370	0.098	3.778
child age 54m+	0.521	0.128	4.079	0.536	0.128	4.195	0.265	0.088	3.001	0.260	0.088	2.945
female hh head	-0.091	0.074	-1.222	-0.087	0.075	-1.168	-0.194	0.087	-2.231	-0.190	0.087	-2.181

Table 12., continued. Bivariate probit social interaction immunization models, DHSII 1990

	Colombia (n=2912)						Paraguay (n= 3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
birth order	-0.060	0.018	-3.358	-0.064	0.018	-3.580	-0.017	0.012	-1.436	-0.018	0.012	-1.549
mother primary or no ed	-0.058	0.064	-0.901	-0.058	0.064	-0.912	-0.175	0.071	-2.467	-0.187	0.072	-2.615
no Spanish							-0.134	0.057	-2.341	-0.114	0.058	-1.984
spouse farmer	-0.035	0.135	-0.263	-0.024	0.135	-0.174	-0.079	0.062	-1.279	-0.070	0.062	-1.133
Proportion older children in municipio with cards	4.952	0.344	14.399	5.050	0.356	14.193	2.862	0.178	16.083	2.855	0.178	16.022
Proportion departmento female lf self-employed	-0.680	0.466	-1.459									
Proportion municipio female lf laborers				-0.963	0.384	-2.504						
Proportion of tract households non-Spanish							0.276	0.192	1.435			
Municipio housing gini, language entropy corr										0.129	0.078	1.649
inverse Mills ratio	-0.581	0.230	-2.526	-0.621	0.231	-2.681	0.540	0.231	2.333	0.555	0.232	2.396
rho[a]	0.139	0.036	3.914	0.141	0.035	3.985	0.109	0.030	3.615	0.110	0.030	3.654
treatment effect	0.081	0.000	0.000	0.082	0.000	0.000	0.065	0.000	0.000	0.066	0.000	0.000
log-likelihood	-3117.3			-3113.7			-4052.4			-4052.1		

[a] rho is the disturbance correlation. Note: Huber/White/sandwich variance estimators used in all models

Table 13. Bivariate probit marginal effects on immunization, DHSII 1990

	Colombia (n=2912)			Paraguay (n= 3424)		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Immunization						
child age 18-23m	0.122	0.024	5.157	0.155	0.030	5.205
child age 24-29m	0.176	0.021	8.280	0.168	0.029	5.713
child age 30-35m	0.143	0.024	6.087	0.189	0.029	6.622
child age 36-41m	0.178	0.022	8.153	0.233	0.028	8.377
child age 42-47m	0.168	0.022	7.503	0.253	0.028	9.075
child age 48-53m	0.188	0.022	8.631	0.242	0.028	8.550
child age 54m+	0.194	0.021	9.139	0.257	0.024	10.601
female hh head	-0.010	0.023	-0.422	-0.033	0.029	-1.163
birth order	-0.017	0.004	-4.007	-0.022	0.004	-5.573
mother primary or no ed	-0.090	0.020	-4.546	-0.206	0.025	-8.245
no Spanish				-0.146	0.020	-7.423
spouse farmer	-0.120	0.039	-3.101	-0.154	0.022	-7.175
Proportion older children in municipio with cards	0.146	0.046	3.152	0.280	0.044	6.398
inverse Mills ratio	-0.310	0.070	-4.458	0.368	0.077	4.761

NOTE: These marginal effects refer to Models 1, Table 4.10.

[a] These coefficients are normalized. For the j'th variable, B_j phi(z) represents the change in probability of full immunization for a unit change in j where z= $phi^{-1}(p)$, p is the sample mean of the response variable and Bj is the probit coefficient for the variable.

Note: Huber/White/sandwich variance estimators used in all models.

Table 14. Bivariate probit social interaction maternal health models, DHSII 1990

	Colombia (n=2912)						Paraguay (n=3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Delivery												
Constant	0.632	0.816	0.775	-0.255	0.629	0.8163	0.176	0.130	1.356	0.177	0.129	1.373
maternal age < 20y	-0.184	0.466	-0.396	-0.186	0.466	-0.399	0.104	0.132	0.787	0.103	0.132	0.780
maternal age 30-39	0.482	0.314	1.535	0.484	0.314	1.539	-0.035	0.102	-0.347	-0.036	0.102	-0.349
maternal age 40-45	0.606	0.467	1.298	0.609	0.467	1.302	-0.353	0.308	-1.148	-0.355	0.308	-1.153
female hh head	0.052	0.162	0.319	0.052	0.162	0.321	-0.336	0.082	-4.103	-0.334	0.082	-4.087
live births	-0.132	0.017	-7.617	-0.132	0.017	-7.616	-0.084	0.011	-7.492	-0.084	0.011	-7.489
mother primary or no ed	-0.672	0.277	-2.423	-0.671	0.277	-2.419	-0.530	0.136	-3.894	-0.531	0.136	-3.902
no Spanish							-0.447	0.060	-7.458	-0.447	0.060	-7.471
spouse farmer	-0.274	0.190	-1.437	-0.273	0.191	-1.433	-0.470	0.080	-5.872	-0.470	0.080	-5.876
Proportion older children in municipio with professional prenatal	1.155	0.132	8.766	1.156	0.132	8.776	0.849	0.116	7.343	0.845	0.115	7.342
inverse Mills ratio	0.308	1.540	0.2	0.315	1.542	0.204	1.744	0.706	2.470	1.748	0.705	2.478
Prenatal												
Constant	1.089	0.823	1.324	-1.684	1.015	-1.660	1.215	0.150	8.078	0.664	0.259	2.561
maternal age < 20y	-0.234	0.462	-0.507	-0.199	0.461	-0.432	-0.334	0.149	-2.239	-0.299	0.147	-2.029
maternal age 30-39	0.331	0.320	1.034	0.298	0.319	0.935	0.254	0.129	1.963	0.209	0.126	1.668
maternal age 40-45	0.330	0.463	0.712	0.286	0.462	0.618	0.552	0.399	1.384	0.396	0.386	1.025
female hh head	-0.054	0.170	-0.319	-0.050	0.170	-0.292	-0.269	0.090	-2.981	-0.260	0.090	-2.894
live births	-0.138	0.017	-7.95	-0.138	0.017	-8.016	-0.075	0.012	-6.463	-0.076	0.012	-6.569
mother primary or no ed	-0.735	0.280	-2.63	-0.759	0.278	-2.726	-0.561	0.174	-3.228	-0.639	0.173	-3.695
no Spanish							-0.378	0.067	-5.631	-0.424	0.068	-6.267
spouse farmer	-0.268	0.191	-1.409	-0.313	0.191	-1.636	-0.080	0.096	-0.837	-0.153	0.098	-1.566

Table 14., continued. Bivariate probit social interaction maternal health models, DHSII 1990

	Colombia (n=2912)						Paraguay (n=3424)					
	Model 1			Model 2			Model 1			Model 2		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Proportion older children in municipio with professional prenatal	1.123	0.132	8.542	1.093	0.133	8.216	0.896	0.122	7.332	0.961	0.122	7.876
Tract female lf diversity index	-0.167	0.075	-2.223									
Municipio union status diversity index				-0.392	0.248	-1.577						
Proportion municipio households rural							0.151	0.048	3.148			
Municipal occupational prestige score										0.145	0.057	2.550
treatment effect	0.226	0.001		0.227	0.001		0.421	0.001		0.422	0.002	
inverse Mills ratio	-0.203	1.543	-0.131	-0.354	1.538	-0.23	-0.659	0.874	-0.754	-0.285	0.849	-0.336
rho[a]	0.462	0.036	12.944	0.463	0.036	12.981	0.601	0.024	24.584	0.603	0.024	24.701
log-likelihood	-2097.8			-2099.0			-3465.4			-3467		

[a] rho is the disturbance correlation. Note: Huber/White/sandwich variance estimators used in all models

Table 15. Bivariate probit marginal effects on attended delivery, DHSII 1990

	Colombia (n=2912)			Paraguay (n=3424)		
Independent variable	**Coefficient**	**S.E.**	**t-ratio**	**Coefficient**	**S.E.**	**t-ratio**
Delivery						
maternal age < 20y	-0.021	0.100	-0.207	0.042	0.046	0.928
maternal age 30-39	0.089	0.055	1.616	-0.016	0.034	-0.464
maternal age 40-45	0.098	0.055	1.764	-0.117	0.102	-1.148
female hh head	0.017	0.032	0.522	-0.110	0.027	-4.024
live births	-0.021	0.002	-10.189	-0.024	0.004	-6.519
mother primary or no ed	-0.150	0.057	-2.650	-0.185	0.046	-3.979
no Spanish				-0.158	0.022	-7.136
spouse farmer	-0.067	0.046	-1.455	-0.178	0.030	-6.005
Proportion older children in municipio with professional prenatal	0.241	0.026	9.146	0.293	0.038	7.694
inverse Mills ratio	0.035	0.308	0.114	0.549	0.237	2.320

NOTE: These marginal effects refer to Models 1, Table 4.12.

[a] These coefficients are normalized. For the j'th variable, B_j phi(z) represents the change in probability of full immunization for a unit change in j where z= $phi^{-1}(p)$, p is the sample mean of the response variable and Bj is the probit coefficient for the variable.

Note: Huber/White/sandwich variance estimators used in all models.

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