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ESTIMATING THE INTRAHOUSEHOLD INCIDENCE OF ILLNESS: CHILD HEALTH AND GENDER-INEQUALITY IN THE ALLOCATION OF TIME

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In this paper we develop and implement a method for estimating the effects of infant morbidity on the differential allocation of time of family members based on discrete indicators of health and activity participation, commonly available in survey data, and within the context of a household model in which health is determined endogenously. Our estimates that take into account the "simultaneity" of health-activity associations indicate that increased levels of infant morbidity significantly exacerbate existing differentials in the accumulation of human capital across teenage boys and girls in Indonesia.

1. INTRODUCTION

In recent years, a number of studies have provided quantitative evidence on the impact of a person's health on his or her time allocation, and on productivity, farm profits and wage rates in both developing and developed countries (Behrman and Deolalikar 1988a; Bartel and Taubman 1979; Deolalikar 1988; Lee 1982; Sahn and Alderman 1988; Sickles and Taubman 1986; Strauss 1986; Pitt and Rosenzweig 1985). Not all of the costs of ill health, however, are borne by the individual whose health is temporarily or permanently impaired. Within a household, the ill health of one person is likely to evoke resource adjustments by other persons in the household in which the illness occurs. For example, family members may spend more time with children when they are ill. To the extent to which time in family-provided health "care" is transferred from activities that contribute to the long-term health or development of those persons caring for the family member in bad health, estimates of the direct effects of health initiatives on health understate the beneficial role of such programs. Few estimates exist, however, of the consequences of illness for the composition of household activities.

In many developing countries, older children appear to contribute importantly to child care along with the mother.[2] In such settings, among older children, schooling, employment in productive activities, and household care are competing activities. Table 1 provides a description of the principal activities of Indonesian children in age groups 10 to 13 and 14 to 18, of mothers and of household heads in households in which there is an infant aged less than two years, obtained from a

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[2] For example, in rural Philippines households (Evenson, et al. 1980), older siblings of children aged less than three contribute 15 percent of the total hours spent in child care by family members.

TABLE 1
DIVISION OF LABOR BY HOUSEHOLD MEMBERSHIP, SEX AND AGE IN HOUSEHOLDS WITH CHILDREN LESS THAN TWO YEARS OLD*

	Children					
	Age 10–13		Age 14–18			
Major Activity	Boys	Girls	Boys	Girls	Mothers	Male Heads
Labor force	3.8**	4.0	28.7	15.9	24.2	97.4
School	91.2	86.5	60.7	51.1	0.2	0.2
Household care	0.8	5.2	2.9	24.6	75.2	0.1
Other	4.2	4.3	7.7	8.3	0.3	2.3

* Total number of households = 10,231.
** Percent of group in activity.

1980 national probability survey (SUSENAS 1980). There are three striking features of Table 1. First, almost all male heads in such households report that their principal activity is participation in the labor force. Second, almost 25 percent of teenage girls aged 14 to 18 in households with infants have as their principal activity household care. Third, girls in the 14 to 18 year age group are 8 times more likely than similarly-aged boys to report that the household is the location of their principal activity, and are 16 percent less likely than boys to report their principal activity as schooling. While these figures cannot indicate differences in hours allocated to any activity, they are consistent with gender differences in the time-allocation patterns of teenagers observed in many Asian Societies.[3]

A natural question is whether changes in levels of infant morbidity would significantly affect the disparities in the intrafamily allocation of activities by sex observed in a country like Indonesia, and thus whether observed gender inequality in human capital could be reduced through improvements in child health. While there has been much attention paid to gender effects in the intrahousehold allocation of nutrients (Behrman 1988a), there is little evidence on the relationship between health levels and gender differences in the allocation of time. Yet how children spend their time may be as important as how well they are fed in determining their subsequent development even in low-income settings. Identification of the "third party" incidence of health—the effects of the health of person i on the behavior of person j—when the behavior of j may directly affect i's health (e.g., time in child care), however, is not straightforward. For example, it is difficult to find instruments that directly influence i's health but not that of j, net of the health of i. Perhaps for this reason, the few existing studies (e.g. Salkever 1982; Bartel and Taubman 1986) of intrafamily health effects have not attempted to discriminate between the time-allocative consequences and causes of ill health.

In this paper we demonstrate the difficulties of identifying both the own- and

[3] In the Philippines (Evenson, et al. 1980) among children aged 12–14, girls' hours in the labor force were 52 percent those of boys, while boys' hours in household activities were 44 percent those of girls. In an intensive anthropological study of a village in Bangladesh (Cain 1980), the number of hours per day in wage work spent by girls aged 13–15 was found to be 21 percent that of similarly aged boys; girls' hours in crop production was 41 percent that of boys, and the number of hours spent by girls in household work was almost 10 times that of boys in that age group.

cross-effects of health within a household, and develop and implement a method for estimating the effects of infant health on the differential allocation of time by other family members that is not inconsistent with models of household behavior. We are able, under some strong simplifying assumptions, to estimate, based on data from Indonesia, how mothers and teenage girls and boys reallocate their activities relative to each other in response to an infant's health. From such estimates we can thus test whether, in particular, the division of activities among teenagers by sex is influenced by the demand for child care responsibilities associated with infant morbidity. We also test whether the infant's gender alters the intrahousehold allocation of activities.[4] Section 2 contains a simple model used to illustrate the identification problem. Section 3 describes the data and the econometric methodology we use to identify the effects of child health on the family activity distribution in a context in which both the endogenous health and activity indicators are measured by discrete variables. Section 4 presents estimates, based on Indonesian household data, of the differential responses of family members to infant health. The results indicate that inattention to problems of the measurement and endogeny of health leads to misleading estimates of the effects of variations in child morbidity on the intrahousehold division of labor. Our estimates that take into account the "simultaneity" of health-activity associations indicate that increased levels of infant morbidity significantly exacerbate existing differentials in the accumulation of human capital across teenage boys and girls in Indonesia. Section 5 contains a summary and conclusion.

2. HOUSEHOLD ACTIVITIES AND THE EFFECTS OF HEALTH

Consider a set of households each consisting of n member "types" (defined, say, by sex, age, etc.), in which the total time Ω of each household member i in household t is allocated between two activities, work (for pay) and home time l_{it}. The home time of every member of household t influences jointly the healthiness of each member H_{it}, along with health goods X_{it} allocated to i. Each member's home time is thus a public good, while X_{it} is a "private" good. This private-public distinction is not essential to the model, as discussed below, but simplifies notation. The health technology is thus given by:

$$H_{it} = h(l_{1t}, \dots, l_{nt}, X_{it}, H_{1t}, \dots, H_{i-1t}, H_{i+1t}, \dots, H_{nt}), \tag{1}$$

where (1) also incorporates the possibility of intrafamily health externalities inclusive of contagion and/or health efficiency effects on home time. Assume also that the household welfare function contains the home time and health of each individual member and a jointly consumed commodity Z_t.[5] Households are

[4] In Evenson, et al. (1980), the total number of hours spent in child care by family members was 23 percent less when the young child was a girl than when the young child was a boy. Mothers spent 23 percent more hours with boy than with girl infants, while older siblings of the child spent 23 percent less hours caring for their infant sisters compared to their infant brothers.

[5] We have, for simplicity, excluded health endowments from the production function. The existence of unmeasured health endowments that vary across individual family members and households has

heterogeneous in their preferences for these "goods," as expressed by the parameter ε_t, such that:

$$U_t = U(l_{it}, \dots, l_{nt}, H_{1t}, \dots, H_{nt}, Z_t; \varepsilon_t). \tag{2}$$

The household budget constraint is

$$F_t + \Sigma_i(\Omega - l_{it})w_{it} = p_x \Sigma_i X_{it} + p_z Z_t, \tag{3}$$

where w_{it} = market wage rate of person-type i in household t, p_x = price of the health good, p_z = price of Z, and F_t = nonearnings income in household t.

Maximization of (1) subject to (2) and (3) yields n necessary first order conditions for the allocation of the home time of each household member:

$$\frac{\partial U}{\partial l_i} + \sum_k \frac{\partial U}{\partial H_k}\frac{\partial H_k}{\partial l_i} = \lambda w_i, \tag{4}$$

where it can be seen that i's time in the home l_i depends on the (marginal) effects of his (her) time on the healthiness of each family member. The n household demand equations for members' home time, solved in terms of all of the exogenous variables of the model, are given by

$$l_{it} = l(p_x, p_z, w_i, w_1, \dots, w_{i-1}, w_{i+1}, \dots, w_n, F_t, \varepsilon). \tag{5}$$

Note that if home time were a private good, allocatable to the production of health for each individual household member, there would be n^2 demand equations for the l_{ikt}, $i = 1, \dots, n$; $k = 1, \dots, n$, with $l_{it} = \Sigma_k l_{ikt}$.

Now consider an experiment in which the health of member i (and only member i) is exogenously increased and we wish to predict, from the model, the consequences for the allocation of the home time l_j of household member j. From Tobin and Houthakker (1951), we know that for a small change in h_i, at the equilibrium solution, the response is given by

$$\frac{dl_j}{dH_i} = \frac{(dl_j/dp_i^*)^c}{(dH_i/dp_i^*)^c}, \tag{6}$$

where p_i^* is the person-specific (shadow) price of a (notional) unique good allocated to the production of health for member i, and the superscript c denotes a compensated effect. Expression (6) indicates that the effect of a change in member i's health on member j's time allocation cannot be obtained by estimating the reduced-form equation (5) because there are no observed p_i^*'s—all of the n health goods allocated to each household member have exactly the same price (p_x).

Because the person-specific health good prices p_i^* are unobserved, conditional demand equations must be estimated in which a member type's health is the

important implications for the estimation of the health technology (Rosenzweig and Wolpin 1988). They are also a source of heterogeneity, which requires that some procedure be used for identifying the behavioral consequences of changes in health in the absence of experimental data, as elaborated below. The problem of the identification of health effects also arises if households differ in preferences.

conditioning variable to obtain directly the effects of a change in a specific person's health on household time allocations. An estimation problem arises, however, because the variation in the health of a particular member type across households is not exogenous, as indicated by the model, as long as households are heterogeneous in preferences. One procedure is to use instrumental variables. The question then becomes what is the appropriate instrument that yields an estimate of relation (6) from nonexperimental data.

Suppose that it is possible (as in the linear case) to solve out from the reduced-form relationships for the health of member i an expression for one of the observed exogenous prices. Substitution of that solution for the exogenous variable in the reduced-form relation for the home time of member j would yield an equation for the home time of member j in which the health of member i appears on the right-hand side and one exogenous variable is excluded. The solved-out excluded variable thus is eligible as an instrument. The problem with this procedure is that, while it eliminates the problem of the endogeny of health by allowing use of an instrumental variables procedure, it does not yield an estimate of (6) corresponding to the appropriate experiment. For example, if the solved-out and excluded "instrument" is p_x, the observed price of the health good allocated to all household members, then the estimated member-i health effect on the time allocation of member j is just

$$\frac{dl_j}{dh_i} = \frac{(dl_j/dp_x)}{(dh_i/dp_x)}. \tag{7}$$

Expression (7) yields no more information than is obtainable by estimating separately the health and time-allocation reduced forms for persons i and j, respectively. Moreover, the "solved-out" estimate (7) only corresponds exactly to expression (6) if there are no interdependencies among individuals, if all individuals in a population are independent. But in that case there are no "cross" health effects other than via health externalities (contagion). In an interdependent household, as depicted in (1) and (2), a change in p_x affects the allocation of time for person j conditional on person i's health; however, a change in the shadow price p_i^* corresponding to the notional health good specific to person i has no effect on person j's time allocation conditional on i's health. The cross effect of a change in p_x, or any other price common to all family members, on the time allocation of any household member is not the same as the cross effect of a change in a person-specific price p_i^*.

The absence of individual-specific health good prices makes it necessary to directly estimate person-specific health effects but also all but eliminates the possibility of using instruments both to deal with the endogeny of health and to achieve identification of the correct person-specific cross or own health effects.[6]

[6] One possible set of person-specific instruments would include the characteristics of a person's *nonresident* relatives; i.e., relatives who do not participate in household activities. For example, the healthiness or other traits of the mother's parents might influence the mother's propensity for illness but not, given her health, her own or other household member activity allocations. There is an important intergenerational asymmetry, however, as such variables would only permit identification of first

How has this identification problem been solved in prior work on the behavioral consequences of health? In Bartel and Taubman's (1986) study of mental health cross-effects between husbands and wives, healthiness is treated as an exogenous variable, uninfluenced by a spouse's work time or labor force participation; that is, the l_{kt}, $k = 1, \dots, n$, are absent in (1). Similarly, in Salkever (1982), the only study we know of to estimate the cross effects of child health, child health is assumed to be exogenous to mother's work time in the empirical analysis even though in the theoretical model child health is presumed to be influenced by mother's care (time at home). In Pitt and Rosenzweig (1986), the health of the male head of the household is treated as (and is confirmed to be) an endogenous variable affected by and affecting his own supply of labor, and variables corresponding to health programs and sanitation conditions are employed as instruments. However, what is estimated is clearly expression (7) and not (6) because the wife's (and children's) health is also affected by the health programs, which correspond to the variable p_x. The health effect estimated cannot therefore be interpreted as the own effect of the head's health on his labor supply.

To identify the consequences of changes in health in a household context in which health is influenced by the behavior of household members thus requires that additional model structure be imposed, in the absence of exogenous person-specific health-good prices. One strategy, for example, which readily permits identification of the *differential* health effects on family members' time allocation, employed below, is to impose equality restrictions (rather than zero restrictions inconsistent with a general *household* model) on the effects of subsets of exogenous variables on subsets of person-specific behaviors. For example, consider linearized versions of demand equations for the home time of two family members i and j conditional on the health of family member k:

$$(8) \qquad l_i = \alpha_{0i} + \alpha_1 w_i + \alpha_2 \psi_i(w_j, W_i) + \alpha_{3i} p_z + \alpha_{4i} p_x + \alpha_{5i} H_k + \alpha_{6i}\varepsilon$$

$$(9) \qquad l_j = \alpha_{0j} + \alpha_1 w_j + \alpha_2 \psi_j(w_i, W_i) + \alpha_{3j} p_z + \alpha_{4j} p_x + \alpha_{5j} H_k + \alpha_{6j}\varepsilon,$$

where $\psi(\ )$ is some summary statistic for the wage rates of all household members and W_i = vector of members' wages other than i. If, net of the health of family member k, the effects (α) of a change in the price of, say, p_x on the time allocation of i and j are the same (e.g., if the health production function for i and j is the same), then, with $\alpha_{4i} = \alpha_{4j}$ the ij difference equation would be

$$(10) \quad l_i - l_j = \alpha_{0i} - \alpha_{0j} + \alpha_1(w_i - w_j) + \alpha_2(\psi_i - \psi_j) + (\alpha_{3i} - \alpha_{3j})p_z + (\alpha_{5i} - \alpha_{5j})H_k + (\alpha_{6i} - \alpha_{6j})\varepsilon$$

and p_x, which affects the level of individual k's health, could serve as an identifying instrument yielding the appropriate estimate of the person-specific cross health effect (5). Restrictions based on the equality of parameters across household

generation (parents) health effects on their children, but not *vice versa* or among (second generation) children.

member types are thus one means of estimating how the healthiness of a family member influences the time allocation of the family members.

The plausibility of the equality of price effects across individuals conditional on one member's health status depends in part on the classifications of family members chosen. Almost all life-cycle models, with finite horizons, predict that behavior will be age-dependent; in such models price effects associated with individuals of vastly different ages are unlikely to be identical. Stratification by gender, within age groups, the strategy we employ below, may be more plausible. Yet any differential experiences across gender groups, arising for reasons of discrimination or biology, may result in differential gender-specific responses to price changes at a given point in time that arise for reasons other than differential responses to changes in the health status of other household members. Behrman and Deolalikar (1988b) have found, for example, based on household data from South India, some evidence that reduced-form price effects on health were different across girls and boys or men and women, consistent with evidence of significant bias towards males, with respect to nutrients, based on the same data (Behrman 1988b). However, such estimates do not necessarily imply that price effects differ by gender conditional on the health of infants. More generally, differences in unobservables across individuals may render procedures treating price effects as random coefficients rather than fixed more appropriate. We utilize methods that assume that all unobservables are impounded in an additive error, as is conventional in the literature.

3. DATA AND ESTIMATION PROCEDURE

Identifying the effects of child health on the intrafamily allocation of time based on cross-member and activity-specific equality restrictions imposes data requirements that are severe—information on child health, on the activities of all household members, on the prices of health goods, as well as a large sample size are required. Sample size is important since the existence of differences across teenage siblings and the mother *within* families is used to identify the time-allocative consequences of child morbidity.

The 1980 National Socioeconomic Survey (SUSENAS) of Indonesia provides information on household-level food consumption and on the incidence of illness during the past year for all children less than 10 years of age in approximately 60,000 households distributed throughout the country. All members of households in the survey also provide information on their principal activity in the last week, divided among labor force, school, household care, and other, and on their age, sex, relationship to head, educational attainment, employment time (if any) and wage rates.[7]

[7] Six mutually exclusive principal activities are distinguished in the SUSENAS survey; work, temporarily not at work, school, home-care, retired or disabled, and other. Temporarily not at work is defined as having a job but not working during the last week for reasons such as strike, flooded fields or holiday. These first two activities are aggregated into one category. The home-care category excludes those who do household chores for pay. The "other" category is defined to include those activities not

The 1980 SUSENAS also identifies the county (*kabupaten*) in which each household resides. We constructed from the household data, *kabupaten* price medians for 14 individual foods and price indices for 12 food groups based on *kabupaten* average consumption shares. Average *kabupaten* prices have been found to vary substantially in the cross-section even for homogeneous goods (such as kerosene and named-brand goods) due to the island geography of Indonesia and its relatively underdeveloped infrastructure (Pitt and Rosenzweig 1986). We also obtained information on village-level health programs, drinking water sources, and sanitation facilities, which were aggregated to *kabupaten* village frequencies.[8] There are thus 525 areas defined in the data set, based on rural-urban distinctions within the 300 *kabupatens*. All of these *kabupaten*-level variables are potential instruments in an analysis in which differential activity effects are estimated, since all may potentially affect the levels of infant illness (health), but may not *differentially* influence the time allocation of the family members net of their effects on infant health.

Because almost all children aged 6 to 10 are in school (or report that they are), to examine the impact of child health on the activities of older children requires that we look at the effects of the health of children less than 10 (since no health information is provided for children 10 years of age or older) on the time allocation of family members aged 10 and above. In particular, we will examine the effects of the health of infants aged three years or less on the activities of their mothers and their siblings aged 10 to 18.[9] We exclude male heads of households because, as Table 1 indicates, virtually all heads report their principal activity to be participation in the labor force. Without any sample variation in their reported activities, we cannot estimate any responses to changes in health levels, if any, by male heads of households. Our data set for analysis thus consists of 5831 sets of household triads—teenage daughter, son and mother activities in households with a child less than four years of age.

Two important features of the data are that (i) all of the activity variables and the measure of health (sick or not) are discrete and (ii) health or illness refers to the past year while the activities correspond to the last week. Our choice of econometric technique is influenced both by the nature of the data and by the restrictions implied as sufficient by the model to identify the within-family activity effects of infant illness.

falling into the first five and includes a "life of leisure" ("*hidup bermalas-malas*"). Households in which the mother of an infant or her teenagers were disabled were dropped from the sample.

[8] Information on village-level health, water and sanitation facilities come from the data tapes of the Village Potential (*Potensi Desa*) census carried out as part of the 1980 Population Census (*Sensus Penduduk*). Health facilities are measured by the proportion of villages in each rural/urban *kabupaten* with a maternity hospital, a family planning clinic, a doctor, and a polyclinic or a public health clinic (PUSKESMAS). Water sources are measured by the proportion of villages whose principal water source is from a pipe, a pump, a well, a spring, a river or solely from rainfall. Waste disposal methods are the proportion of villages in which waste is buried or burned. Sanitation facilities are village lavatories, distinguished by family/nonfamily and Presidential Decree (INPRES)/non-INPRES.

[9] Age stratification minimizes the possibility of age-dependent price effects. Within the age groups, we assume that price effects are independent of age. A larger sample size might allow a finer age stratification among the teenagers.

As noted above, our strategy, which permits identification of the consequences of health in a household context, is to impose equality restrictions on the effects of subsets of exogenous variables on subsets of person-specific behaviors. Consider linearized versions of the conditional activity equations for family members in a household containing a mother and her teenaged son and daughter:

$$I_{ij}^* = (\alpha_{iM} + \delta_{ij}^A D_j)' A_j + (\gamma_{iM} + \delta_{ij}^h D_j) h^* + (\beta_{iM} + \delta_{ij}^x D_j)' X + Z\lambda_{ij} + \varepsilon_{ij} \tag{11}$$

where I_{ij}^* is the level at which household member j undertakes activity i; D_j has the value of one in the equation for j and zero otherwise in the son and daughter equations; h^* is the endogenous health of the mother's infant child; A is a vector of member-specific exogenous variables; X and Z are vectors of household-specific exogenous variables, to be distinguished below; a, β, γ, δ and λ are vectors of coefficients, and ε_{ij} are error terms having a multivariate distribution with zero means and covariance matrix Σ. Furthermore, ε_{ij} is the sum of a household specific component ν_i and a person-specific component ψ_{ij}

$$\varepsilon_{ij} = \nu_i + \psi_{ij}. \tag{12}$$

The household-specific term ν_i reflects unobserved variation in the health environment of households, the infant health technology and household preferences. The person-specific component ψ_{ij} represents unobservable individual attributes such as ability and health endowments.

The reduced form equation for the healthiness of the mother's infant is

$$h^* = X\Pi_x + Z\Pi_z + A_j\Pi_a + \eta \tag{13}$$

where $\Pi = (\Pi_x, \Pi_z, \Pi_a)$ is a vector of reduced-form parameters and η is an error term. The vector of exogenous variables Z are those for which equality restrictions are imposed:

$$\lambda_{ij} = \lambda_{ik} \quad i \neq k;\ i, k = \text{member type}. \tag{14}$$

Identification requires that there are at least as many variables in Z as endogenous regressors.

Neither the health h^* nor the activity variable I_{ij}^* is observed in our data. What is observed is a set of three dichotomous indicators (I_{1j}, I_{2j}, I_{3j}) indicating whether labor force, school, household care or leisure is the primary activity of the household member, and a dichotomous indicator for h^*. Estimation of the parameters of the system (11) through (14) is thus complicated by the discrete nature of all of the endogenous variables, the endogeny and discreteness of one of the regressors, and the selection of households—those having the mother of an infant of age 3 or below and two siblings 10 to 18 years of age and of opposite sex resident in the household. One approach to estimating the system is to extend Mallar's (1977) simultaneous equations probit model to the multivariate case with sample selection. Even for the simple case combining the binary choice of time activities among two individuals with sample selection, however, the likelihood is computationally intractable, involving the evaluation of the trivariate normal cumulative distribution. In addition, this likelihood would need to simultaneously estimate a large number of parameters: the parameters of the system (11) through

(14) plus an additional set of parameters to account for the selection of the sample of households with teenagers and infants.

Using a fixed effects approach such as in Chamberlain (1980) eases the computational problem greatly. First, note that the identifying restrictions (14), the equality restrictions imposed on the effects of subsets of exogenous regressors (Z) on *person-specific* behaviors, imply that the set of regressors Z have zero coefficients in a differenced conditional time allocation equation. By differencing the conditional time allocation equations for any pair of household member types (say daughter and sons), we thus reduce the number of parameters we need to estimate. That we cannot identify the parameters λ_{ij} is not of great concern since it is maintained that they do not *differentially* affect time allocation and it is differential effects in which we are primarily interested. Likewise, with this technique only the differential effects δ on time allocation are identified for the regressors X and h^* that do not vary across member types. For example, we cannot determine whether a change in an element of X, X_n, increases or decreases the likelihood that any of the three person-types will be engaged in any of the alternative activities. We *will* be able to estimate how a change in X_n differentially affects the likelihood of engaging in any time-activity for any pair of person-types. Although identifying level effects on activities—that is the parameters β, γ, and λ—is more informative, the identification of the δ parameters is sufficient to test hypotheses about intra-household distributions.

A second advantage of a fixed effects procedure is that differencing eliminates the sample selection problem. Given a selection rule, the residuals in (11) can be rewritten as the sum of residuals having zero mean and another term which adjusts for the truncation of the distribution of behaviors. As is well known, in the case of normality, this term is proportional to the Mills ratio (Heckman 1974 and Lee 1976). An additive term analogous to the Mills ratio makes this adjustment for truncation if the error structure has a logistic rather than a normal distribution. The important point is that the term which adjusts the residual for its truncation is household-specific, that is, it is the same for all household-member types since it is households—rather than household members—that are selected into or out of the sample. These additive adjustment terms vanish when household member type equations are differenced as long as error covariances do not differ significantly across household members.

A third, related, advantage of the fixed-effects method is that, as long as heterogeneity across households is captured by an additive term, as we assume, it eliminates any bias in second-stage coefficients arising from nonrandom household or program placement or from households choosing their family size. That is, if the household-level unobservables influencing family structure (number of teenage sons and daughters), household location, or the geographical placement of programs influence only activity levels and not differences in gender-specific activities, the second-stage estimates will be consistent.[10] The same property does not apply

[10] Evidence on the endogeny of programs, induced by systematic location choices by households and/or by purposive governmental program placement, is presented in Rosenzweig and Wolpin (1986, 1989).

to the first-stage equation.

The fixed effects logit model, which conditions on sufficient statistics for the incidental parameters, can provide consistent estimates of the behavioral parameters while a model with normally distributed residuals cannot (Chamberlain 1980). Consider the case of two household member-types and one of the activity choices. Following Chamberlain, maximum likelihood estimation of a binary fixed effects logit model is accomplished as a standard logit problem with a rescaled dependent variable. This is equivalent to maximizing the conditional likelihood function, conditioning on a set of sufficient statistics for the incidental parameters ν_i. In the case of two persons ($j = 1, 2$) and an activity a, $\Sigma_j I_{aj}$ is a sufficient statistic for ν_i. If $\Sigma_j I_{aj} = 0$ or 2 then both I_{a1} and I_{2a} are determined given their sum. Thus the only case of interest is $\Sigma_j I_{aj} = 1$. The possibilities are ($I_{a1} = 1$, $I_{a2} = 0$) or ($I_{a1} = 0$, $I_{a2} = 1$), and thus the rescaled dependent variable y_{a12} has the value one if the first possibility is true and zero if the second is true. All observations for which $\Sigma I_{aj} = 0$ or 2 do not contribute to the likelihood and are dropped.

With more than two persons the fixed effects logit model becomes multinomial. Unlike the conventional use of fixed effects models, we do not assume that all of the parameters are identical across individuals. However, the model estimated restricts mother's choices to home and labor force, as they are rarely engaged primarily in leisure and schooling in our data. Thus, we identify daughter-son parameter differences for all pairs of choices and daughter-mother and son-mother differences for home care versus labor force only. Of the six sets of daughter-son activity choice parameter differences only three are stochastically independent and enter the likelihood. Of the two sets of teenage child-mother parameter differences only one is stochastically independent.

Households in which the mother and her teenaged siblings are all engaged in the same activity do not contribute information to the likelihood function. Thus, households in which the mother, teenaged daughter and son are at work or all are engaged in home care are excluded from the sample. Moreover, the largest activity subgroup, in which both of the sex-differentiated siblings are in school, also does not contribute to the likelihood because conditioning on all activity sets with two or three persons in school always corresponds to the siblings being in school (mothers are never in school). There are thus 1833 triads contributing to the fixed effects likelihood.

The two-person fixed effects probability model can equivalently be written in the common latent variable formulation:

$$I_{a1}^* = (\alpha_{aM} + \delta_{a1}^A)'A_1 + (\gamma_{aM} + \delta_{a1}^h)h^* + (\beta_{aM} + \delta_{a1}^x)'X + Z\lambda_{a1} + \varepsilon_{a1}, \tag{15}$$

$$I_{a1} = 1 \text{ if } I_{a1}^* > 0,\ I_{a1} = 0 \text{ otherwise;}$$

and

$$I_{a2}^* = (\alpha_{aM} + \delta_{a2}^A)'A_2 + (\gamma_{aM} + \delta_{a2}^h)h^* + (\beta_{aM} + \delta_{a2}^x)'X + Z\lambda_{a2} + \varepsilon_{a2}, \tag{16}$$

$$I_{a2} = 1 \text{ if } I_{a2}^* > 0,\ I_{a2} = 0 \text{ otherwise.}$$

The binary fixed effects logit model uses two cases.

Case 1: $I^*_{a1} > 0$ and $I^*_{a2} \leq 0$,

so that

$$y^*_{a12} = I^*_{a1} - I^*_{a2} = \alpha'_{aM}(A_1 - A_2) + \delta^{A'}_{a2}A_1 - \delta^{A'}_{a2}A_2 + (\delta^h_{a1} - \delta^h_{a2})h^* + (\delta'_{a1} - \delta'_{a2})X + (\psi_{a1} - \psi_{a2}) > 0, \tag{17}$$

and

Case 2: $I^*_{a1} \leq 0$ and $I^*_{a2} > 0$,

so that

$$y^*_{a12} = I^*_{a1} - I^*_{a2} = \alpha'_{aM}(A_1 - A_2) + \delta^{A'}_{a1}A_1 - \delta^{A'}_{a2}A_2 + (\delta^h_{a1} - \delta^h_{a2})h^* + (\delta'_{a1} - \delta'_{a2})X + (\psi_{a1} - \psi_{a2}) \leq 0. \tag{18}$$

Thus, y^*_{a12} is a latent variable which underlies the rescaled dichotomous indicator y_{a12} resulting in a linear latent variable model statistically identical to that of Mallar's except for the (yet unspecified) error distribution.[11] Extension of Mallar's results to the case of the logistic distribution and to polychotomous choice is relatively straightforward, requiring only that the second-stage compound error have a logistic cumulative distribution. Estimation of the covariance matrix Ω is not straightforward, since the endogeny and latent nature of one of the regressors (h^*) must be accounted for. The formula that we use for computing the asymptotic covariance matrix Σ is derived in the Appendix.

In our data the vector of individual-specific exogenous variables A consists only of age, in years. In our initial specifications the vector X contains the number of teenage daughters and sons and a dummy variable indicating whether the household resides in an urban area. We test for differential effects on activity allocations of additional household-level variables, including the educational attainment of the mother and the husband's wage, below.

The vector Z, consisting of exogenous regressors which do not differentially affect the allocation of time among household member-types, net of infant morbidity, contains two subsets of regressors: the 14 commodity prices and 12 price indices and the community characteristics, including programs and health facilities (4 variables), public waste facilities (7 variables) and drinking water sources (5 variables).[12]

[11] Note that the use of the two-stage procedure to predict (latent) infant illness eliminates measurement error in the sickness variable, even if the instruments are themselves error-ridden, as long as the measurement errors in the imperfect instruments are uncorrelated with those associated with the measurement of illness (Tukey 1951).

[12] We do not deflate prices by region-specific price indices, thus allowing for money illusion in the first-stage equation. Restricting price effects to be homogeneous of degree zero would add somewhat to efficiency, if true. Of course, the first-stage equation is not interpretable as a demand equation given sample selection. We present the first-stage estimates only to demonstrate that the instruments are important determinants of infant health. The consistency of our second-stage estimates only depends on

TABLE 2
SINGLE AND TWO-STAGE MAXIMUM LIKELIHOOD MULTINOMIAL FIXED EFFECTS LOGIT ESTIMATES: DIFFERENTIAL EFFECTS OF INFANT ILLNESS ON HOUSEHOLD ACTIVITIES OF DAUGHTERS, SONS, AND MOTHERS RELATIVE TO HOME CARE

Household Pair/Infant Illness:	Alternate Activity to Home Care					
	Labor Force		School		Leisure	
	Exogenous	Endogenous	Exogenous	Endogenous	Exogenous	Endogenous
Daughter vs. Son	2.84 (1.40)*	−1.25 (2.23)** [3.23]***	3.21 (1.58)*	−1.07 (2.03)** [2.85]	3.23 (1.58)*	−1.11 (1.88)** [2.57]
Daughter vs. Mother	−.348 (1.87)	.072 (0.49) [0.60]	—	—	—	—
Son vs. Mother	−3.19 (1.57)	1.32 (2.32) [3.20]	—	—	—	—

* Asymptotic t-ratios in parentheses in column.

** Asymptotic t-ratios corrected for use of stochastic regressors, estimated from first stage, in parentheses.

*** Uncorrected t-ratios computed directly from information matrix in brackets.

4. RESULTS

Table 2 reports the estimates of the differential activity effects, relative to the household care activity, of child illness (the δ_{ij}^{h}) and their associated asymptotic t-ratios obtained using (i) the maximum-likelihood multinomial fixed effects model, which does not take into account the endogeny of child health, and (ii) the two-stage multinomial fixed effects model. There are five sets of estimates reported for each procedure, corresponding to the three other activities engaged in by the teenaged siblings (labor force, school, and leisure) and the labor force activity of mothers. Table A in the Appendix reports the first-stage, maximum-likelihood logit estimates of the reduced-form determinants of child illness used in the two-stage model. In that equation, the sets of area-level food prices ($\chi^2(26) = 80.6$), programs and health facilities ($\chi^2(7) = 17.6$), and water sources ($\chi^2(5) = 18.4$) contribute significantly to the value of the likelihood—the environment in which each household resides thus significantly influences the probability that a young child will be ill.

The set of variables included in the initial specification contribute significantly to the likelihood for each of the activity differentials, whether or not the predicted measure or the actual dichotomous indicator of child morbidity is used. The two-stage and single-stage fixed effects logit estimates of differential illness effects are not directly comparable, since illness in the latter is measured by a dichotomous indicator of morbidity, while illness in the former is measured by the predicted latent measure of illness obtained from the first-stage logit illness equation. However, the logit model provides a mapping between a change in the latent index

the lack of a correlation between the instruments and differences in person-specific errors within each household.

of morbidity and the probability of illness. For example, a one standard deviation decrease in the latent illness variable (0.63) would be associated with a decline in the probability of illness, evaluated at its mean level (0.52), of 0.15, or 29 percent. Roughly speaking, the estimated single-stage coefficient for illness incidence would have to be approximately four times the magnitude of the coefficient estimated using the predicted latent index for the two estimates to yield similar quantitative effect. However, the signs of the single-stage and two-stage illness estimates differ in every case.[13] Moreover, the inconsistent single-stage multinominal logit estimates do not indicate any statistically significant differences in the activity responses of the teenage siblings to child illness. Not taking into account the possibility that differential activity allocations may influence child health thus results in misleading inferences about the effects of changes in child morbidity on the gender-based division of household activities.

The two-stage procedure that takes into account the possibility that child illness may also be influenced by differential activity allocations indicates that variation in the levels of child morbidity significantly alter the division of time across mothers and their older sons and across teenage sons and daughters with respect to their allocations of both time to the labor force and to school. In particular, the two-stage estimates indicate that mothers shift out of (into) the labor force to (from) home time less (more) strongly than do their teenage sons (but not less so than daughters) when their young children are ill and teenage daughters are more (less) likely to increase (decrease) their time in household care relative to both labor force employment and to school than are their brothers in response to their younger sibling's illness. These results thus suggest that reductions in child morbidity would diminish significantly existing sex-based inequalities among teenagers in their allocation of time between household activities and activities that contribute to the development of skills, via schooling and labor market experience, that augment earnings.

To assess the quantitative importance of the estimates of differential morbidity effects on cross-member activity distributions, it is necessary to impose additional restrictions. The effect of a change in child health h on the probability P_a^d of a teenage daughter, say, participating in one activity among the four possible activities, 1, 2, 3, 4 (labor force, schooling, leisure, and home care), is:

$$\frac{dp_1^d}{dh} = p_1^d(1 - p_1^d)[\gamma_{1s}^h + \delta_{1d}^h] - p_1^d p_2^d[\gamma_{2s}^h + \delta_{2d}^h] - p_1^d p_3^d[\gamma_{3s}^h + \delta_{3d}^h], \tag{19}$$

while for the mother, who participates in only two activities, home care and employment (see Table 1), the effect of a change in child health on the probability she participates in the labor force is given by:

[13] One possible reason why the inconsistent estimates yield positive signs on gender differentials in the activity choices of the teenage siblings is that teenage girls are better providers of child care than are teenage boys. Thus in households in which girls stay at home relative to boys, young children are less likely to be ill. It is this reverse causation that is eliminated by the use of instrumental variables.

$$\frac{dp_1^m}{dh} = p_1^m(1 - p_1^m)[\gamma_{1s}^h + \delta_{1m}^h]. \tag{20}$$

Although γ_{as}^h, the *level* effect of h on the latent activity index of the son for activity a is not identified, since for each activity a, P_a^i is known and the δ_{ad}^h and δ_{am}^h are estimated, comparisons of probability effects across family members only require knowledge of any one person-specific level effect. Thus, for example, if it is known or thought to be highly likely that teenage sons do not take on child care responsibilities, then it is easy to show that it is possible to identify, from our estimates of the δ_{ai}^h, the level effects of a change in child illness on the probabilities of mothers and daughters (but not sons) allocating their time to each of the activities.

In Indonesia, where our data suggest that less than three percent of male siblings aged 14 to 18 have as their principal activity household care, the assumption that teenage boys do not alter their time devoted to household activities in response to the illness of a young sibling appears not very strong, certainly less so than one which imposes an arbitrary value for the response of the mothers' (daughter's) time to her child's (younger sibling's) illness. Our two-stage estimates indicate, based on the activity participation rates of girls 14 to 18 and of the mother given in Table 1, that with no response of teenage boy's time in the home to infant illness, the probability that the mother devotes her time to the labor force decreases by 0.15 (63 percent) from its mean probability of 0.24 in response to a one standard deviation decrease in latent child health (a rise in the incidence of child illness by 29 percent). The daughter's likelihood of labor force participation also declines, by 0.04 (25 percent) from its mean probability of 0.16 as does the likelihood of her attending school, by 0.075 or by 15 percent, while her participation in home care rises by 0.13 or 53 percent. The daughter's likelihood of being at home but not engaging in household care drops by 0.014 or 17 percent.

As noted, because age differs across family members, in contrast to household-specific variables, the effects of own age on the levels of activities can be identified without further restrictions. Table 3 reports the two-stage fixed effects multinomial logit estimates of member-specific age effects for three principal activities relative to the household care activity and test statistics on differences in age effects across daughters, sons and mothers within activities. These estimates, which control for household "fixed effects," confirm what is evident in Table 1, that there are significant gender-related differentials in the age patterns of household activities of teenagers for all three activity allocations. Although there appears to be no significant age gradient for mothers with respect to their allocation of time between household care and labor force employment, both teenage daughters and sons significantly decrease their participation in school relative to household care activities as they age, but teenage girls' are significantly more likely to reduce their leisure time and significantly less likely to increase their participation in the labor force compared to teenage boys. That is, as they age boys leave school and enter the labor market; girls leave school, diminish their leisure and participate more in household activities.

Computations of quantitative age effects on the probability of participation in an

TABLE 3
TWO-STAGE MAXIMUM LIKELIHOOD FIXED EFFECTS LOGIT ESTIMATES OF AGE EFFECTS ON HOUSEHOLD ACTIVITY CHOICES RELATIVE TO HOME CARE, BY HOUSEHOLD MEMBER TYPE

	Activity		
Age of	Labor Force	Schooling	Leisure
Daughter	.0073	−.939	−.486
	(0.21)*	(13.6)	(6.01)
Son	.504	−.438	−.0162
	(4.71)	(4.48)	(0.15)
Mother	.010	—	—
	(0.74)		
Daughter-son differential			
t-statistic	4.47	4.48	3.96
Daughter-mother differential			
t-statistic	0.10	—	—
Son-mother differential			
t-statistic	4.61	—	—

* Asymptotic t-ratios in parentheses.

activity requires, as shown in (18), not only the estimated multinomial logit age coefficient displayed in Table 3, but also activity-specific probabilities. To assess the extent to which the gender-based division of activities evolves with age among teenagers, we computed the effects of age on activity probabilities assuming, counterfactually, that activity participation probabilities are the same for boys and girls at age 14 and used the average of those sex-specific probabilities at that age. The results indicated that both sons' and daughters' probabilities of participation in household care rise with age, but that even if the activity distribution at age 14 were equal across siblings, the probabilities of engaging in household care would rise significantly faster for teenage girls. At the sample means for teenagers aged 14, teenage girls' likelihood of participation in household care increases by 0.20 from age 15 to age 18, or by more than 100 percent (136 percent), while the probability of taking on household responsibilities increases by 0.063 or by 75 percent over the same life-cycle period for their teenage male siblings. Labor force participation rates would rise by 0.094 (69 percent) and by 0.10 (76 percent) per year, respectively, for girls and boys, while school rates would diminish by 0.17 (24 percent) for girls and by 0.13 (18 percent) per year for teenage boys.

In Table 4 we report Wald test results for a number of hypotheses that can be tested with our estimates, and tests of our initial specification. Our estimates indicate, not surprisingly, rejection of the hypotheses at the 0.01 level that (i) there are no differences across teenage daughters and sons in the age trajectories of their activity allocations and (ii) the responses of son and daughter allocations to own age, infant illness and the number and sex composition of household teenagers are identical. However, we cannot reject the hypothesis that mother's and teenaged daughters exhibit the same behavior with respect to their allocation of time between home and the labor market; that is all of the δ_{am} in the daughter-mother employment equation are jointly zero. In contrast, the behavior of mothers and teenaged sons are significantly different at the .01 level. The division of labor by

TABLE 4
CHI-SQUARE TEST STATISTICS: JOINT HYPOTHESIS TESTS

Null hypothesis	Chi-square Value	Degrees of Freedom
No age differentials in activities between teenaged daughters and sons	21.4	3
Teenaged daughters and mothers identical activity behavior	14.5	6
Teenaged sons and mothers identical activity behavior	69.3	6
Teenaged sons and daughters identical activity behavior	73.6	15
No influence of number of teenaged daughters and sons on activity differentials	19.6	8
No influence of father's wage on member-specific activity differentials	8.64	4
No influence of mother's schooling on member-specific activity differentials	7.50	4
No influence of infant's sex on member-specific activity differentials	5.00	4

sex, evident among husbands and wives in Indonesia, thus appears to begin prior to marriage and to result in differentiated skills, by sex, at the time of marriage.

We also tested whether the husband's (head's) wage, the mother's level of schooling, and the sex of the infant influenced activity patterns by adding these variables to our initial specification. While the number of sons and daughters, included in our initial specification, does significantly affect the household activity distribution, we could not reject the hypotheses of no influence of either the parental characteristics or the young child's gender. The latter result thus suggests that which member in the household takes on child care responsibilities does not depend on the gender of the child to be cared for, despite there being marked differentiation by activities among children by gender when children reach their teenage years.

5. CONCLUSION

In this paper we have considered the identification issues that arise in attempting to estimate from the perspective of an integrated model of intrahousehold activities the effects induced by changes in levels of child morbidity on the allocation of time among family members. The empirical analysis was applied to household and community level data from Indonesia, a country that exhibits a marked division of labor between market and household activities across men and women beginning at age 13. Our empirical results, obtained using an estimation procedure that takes into account the possibility that infant health is both influenced by and influences how a household distributes its activities across its members, indicate that existing sex-based differences in the division of time between household, labor-force and schooling activities in Indonesia are worsened among teenagers where child morbidity is at a higher level. In particular, we found that teenaged daughters were significantly more likely to increase their participation in household care activities, to decrease their participation in market activities and to drop out of school compared to teenaged sons in response to increases in infant morbidity.

Our results also suggest that estimates of the consequences of child health on the activities of family members need to consider both the endogeny of health and the integrated nature of households, as estimates of the effects of variations in child morbidity obtained without accounting for the simultaneity between the household activity distribution and child health differed markedly from those estimates which did. Attention to the determinants of health in a household context thus not only should result in better estimates of the specific consequences of interventions designed to improve health but in a greater awareness of the differentiated effects of such interventions.

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APPENDIX
THE TWO-STAGE FIXED EFFECTS MULTINOMIAL LOGIT LIKELIHOOD AND ASYMPTOTIC COVARIANCE MATRIX

The two-stage multinomial fixed effects logit model represented by equation (10) through (13) can be more compactly written as

$$h^* = X_1 \pi + \eta \tag{A.1}$$

where $X_1 = (X, Z, A_j)$ and $\pi = (\pi_x, \pi_z, \pi_a)$ and

$$y^*_{ijk} = I^*_{ij} - I^*_{ik} = a_{ij}(A_j - A_k) + (a_{ij} - a_{ik})A_k + \beta_{ijk} X_2 + \gamma_{ijk} h^* + \varepsilon_{ijk}, \tag{A.2}$$

$$\begin{Bmatrix} i = \text{home, school, work, leisure} \\ j, k = \text{daughter, son} \end{Bmatrix} \quad \text{and} \quad \begin{Bmatrix} i = \text{home, work} \\ j, k = \text{daughter, son, mother} \end{Bmatrix}$$

where I^*_{ij} is a latent indicator of time allocated to activity i by member j, A_j is the age (in years) of household member j, X_2 is a set of household specific exogenous regressors, a_{ij}, β_{ijk} and γ_{ijk} are parameters and ε_{ijk} is an error term. The equations A.2 are differenced activity choice equations and thus the parameters β_{ijk} and γ_{ijk} represent the parameter differences between member types j and k in the activity equations for activity i.

Substituting (A.1) into (A.2) yields

$$y^*_{ijk} = a_{ij}(A_j - A_k) + (a_{ij} - a_{ik})A_k + \beta_{ijk} X_2 + \gamma_{ijk}(X_1 \pi) - u_{ijk}, \tag{A.3}$$

$$\begin{Bmatrix} i = \text{home, school, work, leisure} \\ j, k = \text{daughter, son} \end{Bmatrix} \quad \text{and} \quad \begin{Bmatrix} i = \text{home, work} \\ j, k = \text{daughter, son, mother} \end{Bmatrix}$$

where $u_{ijk} = \gamma\eta + \varepsilon_{ijk}$ and η and ε_{ijk} are logistic.

The maximum likelihood estimates of π are obtained from maximizing the standard logit likelihood

$$L_1 = \Pi_{i=1}^{N} \frac{e^{hX_1 \pi}}{1 + e^{X_1 \pi}} \tag{A.4}$$

where h is the observed dichotomous indicator of h^*.

Only four of the equations (A.3) are stochastically independent. For example, for the activities home and work, son-mother parameters are equal to the daughter-mother minus the daughter-son parameters. There are additional restrictions on the parameters on individual specific ages a_{ij}. For example, the parameter a_{ij} appears in both the equation for y^*_{ijk} and y^*_{ijm}, $m = k$.

Following Chamberlain (1980), the parameters of the second stage equations (A.3) are estimated by maximizing a likelihood which is the sum of a set of multinomial likelihoods corresponding to each of nine alternative sets. An alternative set is defined by the frequency of each activity among the three member types and its elements are every combination of member types with that frequency of activities. For example, the alternative set of (home, school, work) has as elements all combinations of triads for which one and only one member type undertakes each of the activities of the set. Some elements are not observed in our data because mothers do not have school or leisure as a primary activity. The number of elements in an alternative set determines the dimension of the multinomial likelihood corresponding to it. Alternative sets having only one element, such as {school, school, home}, do not contribute to the likelihood and are dropped. The nine multinomial likelihoods corresponding to each alternative set have in common four sets of independent parameters and are thus summed to form a single likelihood for which the implied parametric restrictions are imposed.

The covariance matrix Σ of the parameters $\theta = \{a_{ij}, \beta_{ijk}, \gamma_{ijk}\}$ of (A.3) are estimated as

$$\Sigma = \frac{1}{N} JB\left(\frac{1}{N} \Sigma_{i=1}^{N} D_i D_i'\right) B'J' \tag{A.5}$$

where

$$J = -\left(\frac{\partial^2 \ln L_2(\theta, \hat{\pi})}{N \partial\theta \partial\theta'}\right)^{-1}$$

$$B = \left(I \middle| - \left(\frac{\partial^2 \ln L_2(\theta, \hat{\pi})}{N \partial\theta \partial\pi'}\right)\left(\frac{\partial^2 \ln L_1(\pi)}{N \partial\pi \partial\pi'}\right)^{-1}\right)$$

and

$$D_i = \begin{pmatrix} \left.\dfrac{\partial \ln L_1(\pi)}{\partial \pi}\right|_{X_{1i}} \\ \left.\dfrac{\partial \ln L_2(\theta, \pi)}{\partial \theta}\right|_{A_i, X_{1i}, X_{2i}} \end{pmatrix}$$

TABLE A

MAXIMUM LIKELIHOOD LOGIT ESTIMATES: FOOD PRICE, PROGRAM AND WATER SOURCE EFFECTS ON THE PROBABILITY OF ILLNESS FOR CHILDREN AGED 0–3*

Variable	Coefficient	Asymptotic t-ratio
Food and food-group prices ($\chi^2(26) = 80.6$)		
Rice	−0.231	0.68
Saltfish	0.0547	1.85
Stringbeans	0.109	0.91
Eggplant	−0.0436	0.23
Banana	−0.0302	0.20
Salt	2.34	1.04
Coconuts	−0.354	1.07
Cooking oil	0.121	1.11
Sugar	−8.53	3.05
Tea	0.188	1.03
Coffee	0.112	0.88
Kerosene	1.36	2.01
Eggs	−0.371	0.55
Spinach	0.500	0.26
Meat	−0.463	1.66
Milk products	0.846	1.80
Vegetables	−1.07	3.73
Fruits	−0.401	0.16
Spices	−0.147	0.56
Tobacco	−0.516	1.75
Fish	−0.514	2.31
Peanuts	−0.0169	0.38
Noodles	0.280	1.22
Grains	−0.883	0.21
Legumes	0.469	1.54
Tubers	0.367	1.44
Health Facilities** ($\chi^2(7) = 17.6$)		
Puskesmas or polyclinic	−0.109	0.31
Maternity hospital	−1.17	2.07
Family planning clinic	−0.240	0.85
Doctor	−0.115	0.20
INPRES public toilets	−1.24	1.04
INPRES family toilets	−15.8	1.56
Non-INPRES public toilets	0.565	0.42
Drinking water sources ($\chi^2(5) = 18.4$)		
Pipe	1.26	1.97
Pump	−0.926	0.70
Well	0.288	0.48
Spring	−0.533	0.69
River	−0.268	0.39
Number of observations	1833	

* Specification also includes number of teenage children and their ages, mother's age and schooling attainment, father's wage, and urban residence indicator variable.

** Proportion of villages in *kabupaten* with facility.

where $L_1(\pi)$ and $L_2(\theta, \hat{\pi})$ are the first- and second-stage likelihoods, respectively, I is an identity matrix conformable with J and i indexes the N triads. Table 2 reports t-ratio's derived from both the information matrix J and the correct covariance matrix Σ.

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