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Child Health and Mortality: Does Health Knowledge Matter?[1]

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This paper studies factors that influence child health in Bissau, the capital of Guinea-Bissau. This environment is characterised by high infant mortality, but not by malnutrition. We show that although maternal education is important in determining child health and mortality this effect diminishes or disappears when health knowledge is introduced as an explanatory variable. It emerges that health knowledge has large and positive effects on both child mortality and health when instrumented for to capture endogeneity.

1. Introduction

Most economic and demographic studies of the determinants of child health find a positive effect of parental, especially maternal, educational attainment on child health.[2] However, so far little effort has been directed at understanding exactly how education affects child health. Two notable exceptions are Thomas *et al.* (1991) and Glewwe (1999). Although their analytical approaches differ and data come from respectively Brazil and Morocco, the same overall conclusion is reached. The impact of maternal education can be explained by other indicators; in Thomas *et al.* (1991) by access to information through

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[2] See Strauss and Thomas (1995) for a review and Lam and Duryea (1999) for a recent paper.

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different channels and in Glewwe (1999) by the health knowledge of the mother.[3]

What is common to almost all studies of child health, including the two just referred to, is their use of data from areas where malnutrition is widespread. It is, however, far from evident that conclusions and policy implications drawn from these studies are applicable in areas where the prevalence of malnutrition is low. On this background, the purpose of this paper is twofold. First, to analyse whether taking account of parental knowledge about determinants of child health affects the estimates of the effects of parental education. Secondly, to determine which factors influence child health and mortality in an environment characterised by high infant mortality, but not by malnutrition.

The area studied is the Bandim 1 district in Bissau, the capital of Guinea-Bissau. Three reasons motivate this choice. First, very few studies of child health and mortality in West Africa exist in spite of relatively high mortality rates. Thus, economic analyses of child health and mortality in West Africa are clearly needed. Secondly, the West African context differs markedly from that of the Indian sub-continent and South East Asia, which has played an influential role in the literature on child health and mortality. Finally, a comprehensive health project (The Bandim Health Project) has been active in the study area for more than 20 years. Consequently, registration information is available together with background information on the households in the area.

The structure of the paper is as follows. After this introduction, we present the theoretical framework in Section 2, and in Section 3 we discuss the estimation strategy. Background information about Guinea-Bissau, the Bandim 1 district and the data is presented in Section 4 together with descriptive statistics, while Section 5 examines factors affecting child mortality. Section 6 does the same for child health. Finally, Section 7 concludes and identifies areas for future research.

2. Model

Following Rosenzweig and Schultz (1983), we assume that the preference ordering of a household j over a composite consumption

[3] See Kenkel (1991) for an examination of the effect of health knowledge on health in a developed country, i.e., USA.

good X_j, a composite health environment good Y_j and a vector $\mathbf{H_j}$ (= (H_{1j}, H_{2j}, . . ., H_{ij}, . . ., H_{nj})) over the health of the n children in the household can be represented by a utility function

$$U_j = U(X_j, Y_j, \mathbf{H_j}). \tag{1}$$

In Rosenzweig and Schultz (1983) child health is determined by the health environment good Y_j, a child-specific health input I_{ij}, which does not affect parental utility directly, and a child health endowment μ_{ij}.[4] We assume that the health knowledge of the household κ_j also influences child health. In this case, the child health production function is

$$H_{ij} = \Gamma(Y_j, I_{ij}, \kappa_j, \mu_{ij}) \tag{2}$$

where Y_j, I_{ij} and κ_j are likely to be endogenous, as discussed below.

The budget constraint of the household is given by

$$M_j = X_j + p_Y Y_j + p_I \mathbf{I_j} \tag{3}$$

where M_j is household income, p_Y and p_I are the prices of the health environment good and the child specific health inputs relative to the price of the non-health consumption goods, and $\mathbf{I_j}$ is a vector of the child-specific health inputs. Both household income and input prices are assumed to be exogenous to the household.[5]

From maximization of (1) subject to the health production function (2) and the budget constraint (3), the reduced form household demand functions for the composite goods X, Y and I are

$$D_c = D(p_Y, p_I, \kappa_j, M_j, \mu_j) \qquad c = X, Y, I. \tag{4}$$

Reduced form household demand functions for health inputs were among the principal objects of study in empirical analyses of household health in the 1970s. Following Rosenzweig and Schultz (1983), more recent studies have concentrated on estimating the underlying technical relationships embedded in the health production function (2).

[4] The health environment composite good Y_j consists of goods which affect both utility and child health directly. In developed countries this could, for example, be smoking, while the number of children in the household, household sanitation or the source of water are examples relevant to developing countries. Child specific health inputs include intake and individual care.

[5] The validity of these assumptions and the constraints imposed by data quality are discussed in Section 4.

3. Estimation Strategy

Inserting (4) for Y and I into (2) results in

$$H_{ij} = \Gamma(D_Y(p_Y, p_I, \kappa_j, M_j, \mu_j), D_I(p_Y, p_I, \kappa_j, M_j, \mu_j)\ \kappa_j, \mu_{ij}) \quad (5)$$

$$= F(p_Y, p_I, \kappa_j, M_j, \mu_j).$$

This indicates that child health can be described by some function F of relative prices (p_Y and p_I), household income M_j, household health knowledge κ_j and unobservable child health endowments for all children in the household μ_j (including those of the individual child μ_{ij}). Since all children in our cohort were living within a relatively small geographical area and since no price information is available in the data, we assume that relative prices are identical for all households.[6] Hence, child health can be estimated as a function of household income M_j, health knowledge κ_j, total household stock of unobservable health endowments μ_j and the individual unobservable child health endowment. The above model is formulated with reference to health. In this paper, we are, however, also interested in the determinants of mortality. Yet, since it is reasonable to expect that health and mortality outcomes are realisations of the same underlying process, both can be described by (3).

3.1 Econometric Issues

One problem estimating child health as a function of household income M_j, health knowledge κ_j, total household stock of unobservable health endowments μ_j and the individual unobservable child health endowment is the inclusion of variables that may be endogenous. This could arise from child health heterogeneity observable to the parents, but not to the researcher. If parents adjust their behaviour depending on this heterogeneity, the explanatory variables and the error term, which includes the heterogeneity, will no longer be uncorrelated. Consequently, the estimated parameters for all variables, not just the endogenous variable, may end up being biased. As an example, families with children who are more prone to being sick may seek out relevant health knowledge.[7] Hence, we expect that if we do not take

[6] This is supported by data. See Section 4 for further background on the the Bandim 1 area.

[7] It may be that parents, even if they do care about the health of their children, will

account of endogeneity the estimated effect of health knowledge will be biased downwards, which is exactly what Glewwe (1999) found.

Two additional problems, both related to the non-random selection of survey cohorts, must also be considered. The first is the possibility of mortality selection. This problem can arise when estimating health outcomes because the children, who enter the health survey, are surviving children and therefore may not be a random sample of the children born. The second potential problem is the possibility of fertility selection. It works along the same lines as mortality selection in that the children born are unlikely to be a random sample of those potentially born if all parents care equally about the health outcome of their children. The problems of fertility and mortality selection are discussed in Pitt (1997) and in relation to our data in Section 4.

3.2 Econometric Models Estimated

In accordance with the above discussion, we use the following estimation strategy.[8] First, we ignore health knowledge and use the sex of the child, the age of the mother and the father's and the mother's education as explanatory variables, all of which we consider exogenous.[9] This allows us to determine the total effect of parent education on child health and mortality.

The vital registration dimension of the Bandim project allows us to follow the children from birth until they die, leave the sample or reach 5 years of age. We can therefore estimate the effects of health knowledge and the other explanatory variables on child survival using a piecewise-linear log-hazard model. Let $m_i(t)$ be the mortality hazard of child i at time t, $T(t)$ one or more duration dependencies (e.g. the age of the child) and X_i the exogenous explanatory variables presented above. On this basis, the first hazard model, model I, of the mortality of the children can be formulated as

$$\ln m_i(t) = T(t)\gamma + X_i\beta_m. \tag{6}$$

The health of children is measured by height for age as a percentage of the reference median.[10] Hence, we can estimate the determinants of

acquire the 'wrong' health knowledge. That does not, however, affect our argument as long as at least some of the parents get the correct knowledge.

[8] All estimations are carried out using aML developed by Lee A. Lillard and Constantijn W.A. Panis.

[9] In the child health estimations the age of the child is also included.

[10] Section 4 has more information on this.

child health using a continuous model. Let h_i be the health measure of child i and ε an i.i.d. residual, with $\varepsilon \sim N(0, \sigma_\varepsilon^2)$. The estimated model I of the health of the children is therefore

$$h_i = X_i\beta_h + \varepsilon_i. \tag{7}$$

In model II we include the health knowledge of the mother without adjusting for possible endogeneity. We use a dummy K_i to indicate whether the mother of child i knows the reason for malaria or not. The hazard model of child mortality becomes

$$\ln m_i(t) = T(t)\gamma + X_i\beta_m + K_i\alpha_m \tag{8}$$

and the continuous model for child health is

$$h_i = X_i\beta_h + K_i\alpha_h + \varepsilon_i. \tag{9}$$

Finally, we take account of health knowledge endogeneity. This is done by estimating a full-information maximum likelihood (FIML) model, where the outcome for the child and the mother's health knowledge are estimated jointly, with the error terms of the two equations correlated, and where health knowledge enters into the explanatory variables for the child outcome. The equation for mothers' health knowledge is estimated using a probit model. Let Z be a suitable instrument, and let v_j and v_K be jointly distributed with $N_2(0, 0, \sigma_j^2, \sigma_K^2, \rho)$ and $j = m, h$, then the estimated system for the hazard model of child mortality is

$$\ln m_i(t) = T(t)\gamma + X_i\beta_m + K_i\alpha_m + v_{m,i} \tag{10}$$

$$K_i = X_i\beta_K + Z_i\theta + v_{K,i} + \varepsilon_i \tag{11}$$

and for the continuous model of child health it is

$$h_i(t) = X_i\beta_h + K_i\alpha_h + v_{h,i} \tag{12}$$

$$K_i = X_i\beta_K + Z_i\theta + v_{K,i} + \varepsilon_i. \tag{13}$$

The residual for the probit model is $\varepsilon \sim N(0, 1)$.The equations are presented as model III.

Prior to reviewing the data, we stress that exogeneity of all variables in X is a maintained assumption from model I onwards. We recognise that parental education is not necessarily exogenous if there are intergenerational correlations in endowments. If this is, indeed, the case, the estimated parameters are potentially biased. If we had information on, for example, the educational attainment or height (or

some other measure of health) of the grandparents we could use these indicators as instruments for parental education. Nevertheless, the underlying problem of correlation would in principle remain.[11] A better instrument would be access to schooling for the parents when they grew up. Information like this is unfortunately not available here, so we are forced to ignore this potential problem.[12] Finally, the age of the mother is also likely to be endogenous as argued by Rosenzweig and Schultz (1983). Here we encounter the same problem of missing data as with parental education.

4. Data

Guinea-Bissau is a small West African republic covering an area of 36,000 square kilometers. The size of the population is approximately 1.1 million, and population growth was 2.9% per annum during 1970–95. The country has as many as 33 different ethnic groups. The dominant religious affiliations are animist beliefs (63%), Islam (33%) and Christianity (4%) (see Oosterbaan and Barreto da Costa, 1995).

The 1995 GNP per capita was estimated at US$250, placing Guinea-Bissau among the ten poorest countries in the world. Social indicators paint a bleak picture. In 1996, estimated infant mortality was 132/1000 and life expectancy at birth was 43.4 years. UNDP estimates that only 41% had access to health services and 60% to safe water. The male and female adult literacy rates were approximately 68 and 42.5% respectively. The dismal economic and social indicators are mirrored in Guinea-Bissau's ranking in the Human Development Index, 164th out of 174 nations.

From colonial times the capital of Guinea-Bissau, Bissau has been divided into barrios (districts). Since independence in 1974 the area of origin for our cohort, Bandim 1, has changed from being a semi-rural area to an integrated part of Bissau. The majority of houses are built of sun-dried mud-bricks (some with a small addition of cement). Most households are served by pit latrines and hand-dug wells. There is no sewerage system in Bandim 1 and waste is left to rot among the houses. Residents in Bandim 1 differ from the national average with respect to religious affiliation. The predominant local belief is animism

[11] If there is correlation between the child and parent endowments, there must also be correlation between the child and the endowments of its grandparents.
[12] There is also a potential bias in the way marriages are formed. This is discussed in Foster (1996).

(approximately 60%), but there are also considerable Catholic and Protestant communities (approximately 25 and 10% respectively).

The Bandim Health Project was initiated in 1978 as a collaborative research project between the Ministry of Public Health in Guinea-Bissau (MINSAP) and the Swedish Agency for Research Cooperation with Developing Countries (SAREC). The objective was to examine and compare the nutritional status of major ethnic groups living in different areas of Guinea-Bissau. As part of the project, all pregnancies, births and child deaths in the Bandim 1 district have been registered since January 1979.

The initial Bandim project progressed in four stages between November 1978 and January 1980. In November 1978 a census of Bandim 1 was carried out, which was followed by more detailed registration and examination of all children in Bandim 1 under the age of six in December 1978. From January 1979 all new pregnancies were registered together with background characteristics of the household. Finally, in December 1979 and January 1980 all children born in 1979 and the children registered in December 1978 were called in for re-examination. This included collection of anthropometric measurements, breast-feeding status and additional information about the household.[13]

In this analysis, we use the children born between January 1979 and December 1979.[14] Since the Bandim project has in effect acted as a vital registration system from January 1979 to the outbreak of the civil war in 1998, it is possible to follow the children from birth until they die, move out of the area or the registration ends. This vital registration system, together with the re-examinations in December 1979 and January 1980, allows us to look at both survival and health of the children.[15]

Children born prematurely and twins, which are both groups that exhibit excess mortality, are excluded from the sample. The same is the case for children without information about their sex and those with no information about why they left the sample or with inconsistency

[13] A detailed description of data and further information on the data collection process is available from the authors upon request.
[14] The first birth in our sample took place on 3 January 1979 and the last on 23 December 1979.
[15] Since the re-examination happened between 13 December 1979 and 11 January 1980, the children were between eight and 363 days old when they were examined.

among different variables.[16] Finally, children for whom important information about their household is missing are also excluded.[17] This leaves us with a sample of 308 children, for 221 of whom we have anthropometric measures.

4.1 Variables

Since our data were not originally collected with economic analysis in mind, we are limited in the number of relevant variables available for use in the present analysis. The variables we rely on to estimate the determinants of child health and mortality are the sex of the child, the educational attainment of the father and the mother, the mother's age, and whether the mother knows the reason for malaria. For the child health equation we also include the age of the child to account for the fact that parental behaviour only affects child health with a lag. Table 1 presents the descriptive statistics for the variables used.

As alluded to in the introduction, there is significant mortality risk in the area. Infant mortality was 0.1161 and under-5 mortality was 0.2167. Hence, more than 10% of the children in the area were likely to die before reaching 1 year and more than 20% of those born did not survive beyond 5 years of age. Attrition is also important. More than half of the children left the area before they turned five.

The health of the children is measured by height for age (HAM), calculated as a percentage of the reference median. This is the international growth reference population recommended by the World Health Organisation (WHO, 1986). Height for age is an indicator of stunting and is a measure of long-term health.[18] The prevalence of HAM Z-scores two standard deviation units below the reference median is 7.6%, and, compared with what has been reported by Gorstein *et al.* (1994) for nutritionally constrained populations, this is a very low relative prevalence. Thus, we reiterate that the Bandim 1

[16] One example is children for whom the re-examination date is after they have left the sample either due to death or migration. Another example is children, who have left the sample before being five years of age, and nevertheless are marked as being in the sample.

[17] The key variables are education of the father and the mother, the age of the mother, the religion of the household and knowledge of the transmission mechanism of malaria. If these are all missing the observation is excluded.

[18] Weight for height, which is a measure of wasting, is a good indicator of current health.

Table 1: *Variables*

Variable name	Mean	Standard deviation	N	Variable definition
Child health and mortality				
Infant mortality[a]	0.1161	0.0185	308	Died before 365 days
Child mortality[a]	0.2167	0.0252	308	Died before 1825 days
Height for age	97.460	4.047	221	Per cent of reference median
Independent variables				
Sex of child	0.474	0.500	308	Male: 0; female: 1
Age of child	181.100	103.277	221	Age in days at measurement
Age of mother	24.626	5.960	286	Age of mother in years
Father's education	2.464	2.384	138	Years
Mother's education	0.798	1.633	168	Years
Knows reason for malaria	0.427	0.496	213	Do not: 0; know: 1
Instrumental variables				
Religion	0.378	0.486	217	Animist: 0; other: 1

[a]Based on life table calculations with correction for attrition.

area is characterised by high mortality, but not by any significant malnutrition.

The inclusion of the sex of the child provides a test for whether there exists differential treatment of boys and girls. The age of the mother has been included because it may affect the child's health and survival through either biological factors, such as decreased birth weight with rising maternal age at delivery, or experience with child care, which increases with age.

In the model presented in Section 2 income affects child health. While short-term fluctuations in income may have an effect on child health, it is likely that permanent income is the best predictor of child health. The only variable available in the sample, which is likely to capture permanent household income is the education of the father. Furthermore, since total income of the household is likely to be endogenous to the health decisions of the family, the education of the father or the land holdings of the family are often used as proxies

or instruments for income. Since we focus on an urban sample, we cannot use land holdings. Hence, the education of the father enters directly.[19]

The final two variables included are the educational attainment of the mother and her health knowledge. The average education of mothers is very low, less than a year. This is significantly less than the duration of male education. During re-examination of the child the mother was asked what she thought the reason for malaria is. This is the proxy for health knowledge used here, with 1 indicating a correct answer and 0 a wrong answer. As Table 1 shows, less than half of those who answered had the right answer. One can, of course, think of other questions which may be relevant indicators of the health knowledge of the mother. In fact, the survey used by Glewwe (1999) in his analysis of the effect of health knowledge in Morocco contains five different questions about various aspects of health care. Answers to these questions were, however, highly correlated, and all but one question have a significant effect on child health.[20] Accordingly, while having only one question may appear to be a limitation, this result indicates that we should be able to capture at least part of the impact of basic health knowledge using the question about malaria.

Since the health knowledge of the mother is likely to be endogenous, as discussed in Section 3, we need an instrument that is correlated with health knowledge but not with the unobservable health endowment of the child. The instrument used here is the religion of the household. The underlying idea is that belonging to an organised religion, i.e. a religion other than the animist variant, is likely to increase a person's health knowledge. Local animist beliefs to a high degree dictate the actions taken and the treatment parents seek when a child is sick. However, most of these (e.g. changing the name of the child to appease the spirits of the ancestors or withholding liquids in the case of fever) will have no effect or actually worsen the condition of the child. In addition, one could envision correct health knowledge being disseminated through church gatherings, sermons or from direct information provided by the church or its members. As will be discussed below,

[19] As an alternative to income one might use measures of household wealth. The available variables, which could reflect wealth, all show relatively little variation and none of the ones tried were significant.

[20] The one question, which does not have an effect, is one on vaccination, and 95% answered it correctly, while for the others only between 44 and 54% had the correct answers.

there is a significant effect of religion on health knowledge even after controlling for the education of the mother.

In the model discussed above, prices influence household decisions. As mentioned, we assume that relative prices are the same for all households living in the area. While there is no price information in the data, we could use dummies to divide the area. The area is, however, relatively small and none of the dummies were significant. We take this as an indication that our assumption about relative prices is valid.

As mentioned in Section 3, we also have to consider the possibility of mortality and/or fertility selection leading to biased results. With respect to the potential mortality selection, many of the children are measured at such an early age that a significant proportion of the deaths occur after they are being measured. This indicates that the results are less likely to be subject a severe mortality bias. It does, however, also imply that parental action has less of an effect than would be the case if all children were measured after, say, their first (1 year) birthday. Given that data requirements to properly assess (let alone address) the issue of fertility selection far exceed what are available to us, we can only acknowledge the potential existence of this problem.

As in many data sets from developing countries, a substantial number of observations have information missing. Here, we solve this problem by substituting in means for the missing values and including a dummy for the information being missing. Including a dummy for missing information provides an indication of whether the information is missing randomly or not. Since some of the information on health knowledge is missing, we have also included the mean (the percentage that knows the correct answer) plus a dummy.

5. Child Mortality

In this section, we review the results for child mortality. Table 2 presents the outcome of the three models discussed in Section 2. The nodes for the duration dependence are set at 1, 6, 12 and 36 months.[21] As expected, all models show that the decline in the hazard rate is sizeable and significant during the first month of life. After 1 month there is a small, but nevertheless significant, increase until children are

[21] All estimations are done in months, calculated as days/(365.25/12). The scale chosen has no effect on the results.

Table 2: *Child Mortality*

	I Piecewise-linear	II Piecewise-linear	III FIML w. knowledge
Sex	0.1369	0.3490	2.8294***
	(0.2695)	(0.3094)	(0.6907)
Mother's age	–0.0310	–0.0330	0.0407
	(0.0218)	(0.0246)	(0.0456)
Mother's age missing	–1.3003	0.0893	2.1893**
	(1.0039)	(1.0704)	(1.0588)
Father's education	–0.2273**	–0.2101*	–0.5940***
	(0.0909)	(0.1183)	(0.1674)
Father's education missing	0.7109*	0.5214	0.3729
	(0.4032)	(0.4347)	(0.4968)
Mother's education	–0.3899**	–0.1659	0.0206
	(0.1642)	(0.1633)	(0.1747)
Mother's education missing	–0.9045**	–2.1483***	–8.2687***
	(0.3789)	(0.4444)	(1.5885)
Knows reason for malaria		–0.8598*	–10.9903***
		(0.4883)	(1.6722)
Knows reason missing		2.5318***	10.4527***
		(0.4002)	(1.7128)
		Duration dependence	
0–1 month	–5.1349***	–5.0761***	–2.4724**
	(1.0923)	(1.0886)	(0.9724)
1–6 months	0.4765**	0.5295***	0.7936***
	(0.1999)	(0.2052)	(0.2611)
6–12 months	–0.1882*	–0.1582*	0.4127**
	(0.0988)	(0.0957)	(0.2076)
12–36 months	–0.0331	–0.0334	0.0599**
	(0.0293)	(0.0293)	(0.0269)
36– months	–0.0030	–0.0031	0.0444
	(0.0386)	(0.0389)	(0.0386)
Constant	–0.0388	–0.7877	–13.5140***
	(0.7186)	(0.7783)	(3.3256)
σ	3.7954	3.7810	4.2530
ρ			–0.6141**
			(0.2367)
ln L	–537.12	–506.63	–631.45
Observations	308	308	308

Asymptotic Huber-corrected standard errors in parentheses. Statistically significant at the ***1%, **5% and *10% levels.

half a year old. From 6 months onwards the picture becomes somewhat unclear. Models I and II show small, but significant, decreases until 1 year and then non-significant decreases afterwards. In contrast, model III shows significant increases until 36 months of age, after which the increase is non-significant.

In model I the education of the father and the mother both lead to a significant decrease in the hazard rate. The higher education the parents have, the longer the child is likely to live. This is in line with earlier literature on the relationship between maternal education and child health. Interestingly, the two dummy variables for missing education information have opposite signs. The dummy for the missing education of the father is positive, whereas it is negative when the mother's education is missing. That these dummies are significant indicate that information is not missing randomly. Most likely, those households which did not supply any information on the education of the father are also those households that do not have a father present. This tends to support our use of father education as a sensible proxy for household income, since households without a father present are likely to have lower income. Neither the sex of the child nor the age of the mother have any significant impact on its survival prospects in this specification.

Including the health knowledge of the mother and a dummy for whether this variable is missing in model II leads to a number of noticeable changes. The effect of maternal education becomes insignificant, while the health knowledge and the dummy are significant. The health knowledge has the expected effect in that it decreases the risk of dying for the child. In contrast, the children for whom no information on the health knowledge of the mother is available are at a greater risk of dying.[22]

Model III takes account of endogeneity by estimating the hazard model jointly with the health knowledge of the mother. This reinforces the observed changes from model I to model II. In addition to the variables that are significant in model II, the sex of the child is now also significant, and the other variables become more significant. After considering endogeneity, girls are significantly more at risk of dying than boys, and the effect of the education of the mother is far from significant. This indicates that the health knowledge of the mother is a

[22] This is hardly surprising since information on the mother's health knowledge is not available for those children who did not survive until the re-examination date.

more important determinant of the chance of child survival than education *per se*. An important consideration related to model III is how well the instrument performs. Table 3 shows the results for the estimations of the mother's health knowledge. The effect of religion on health knowledge is significant and has the expected sign. Furthermore, the significance of the correlation coefficient indicates that health knowledge is indeed endogenous and the positive coefficient

Table 3: *Health Knowledge*

	III FIML w. mortality	III FIML w. health
Constant	–1.6009**	–1.1503**
	(0.6335)	(0.5136)
Sex	0.0634	0.0506
	(0.2450)	(0.1769)
Mother's age	0.0340	0.0177
	(0.0211)	(0.0153)
Mother's age missing	0.3617	0.1078
	(0.4384)	(0.3289)
Father's education	0.0513	0.0506
	(0.0714)	(0.0532)
Father's education missing	0.2140	0.2445
	(0.3798)	(0.2782)
Mother's education	0.1702*	0.1441*
	(0.0984)	(0.0781)
Mother's education missing	–0.6894*	–0.3621
	(0.4042)	(0.2900)
Age		0.0005
		(0.0010)
Religion	0.6098**	0.3501*
	(0.2771)	(0.1817)
σ	1	1
ρ	0.7814***	–0.6141***
	(0.1394)	(0.2367)
ln L	–631.45	–742.32
Observations	212	212

Asymptotic Huber-corrected standard errors in parentheses. Statistically significant at the ***1%, **5% and *10% levels.

shows that parents with a child who is more at risk and thus have a higher hazard rate are more likely to have the correct health knowledge.

6. Child Health

The results of the child health models are shown in Table 4. Generally, the results are less clear than in the case of mortality. For all models the health of the child is decreasing with age relative to the reference population and there does not appear to be any effect of the sex of the child on its health. The age of the mother and the dummy for whether this information is missing are significant and positive in all models, except that mother age is insignificant in model III. The education of the father has a positive and significant effect except in model III, where the effect is insignificant.

Of particular interest is the effect of maternal education. It is not significant in any of the models, although it does have the expected sign in models I and II. The health knowledge of the mother only has a significant effect in model III when it is treated as an endogenous variable. In this case the effect becomes very large and significant. The correlation coefficient is negative and significant, indicating again that health knowledge is endogenous and that parents with less healthy children are more likely to collect the correct health knowledge.

It may not be surprising that we find very little effect on child health of the variables examined. As discussed above, the nutritional standard is generally good and malnutrition is limited. Two reasons for this could be a climate favourable to farming and the widespread and prolonged breast-feeding practice in the area.[23]

7. Conclusion

This paper has identified health knowledge, measured by the knowledge of the mother about the cause of malaria, as a key determinant of child mortality and morbidity in the Bandim 1 district in Bissau, the capital of Guinea-Bissau. Distinct characteristics of the area analysed include a very low prevalence of malnutrition and a limited degree of gender discrimination. These are significant departures from conditions typical in the existing analyses of child morbidity and mortality in South East Asia and on the Indian subcontinent. The

[23] See Mølbak *et al.* (1994) for a discussion of breastfeeding in the Bandim area.

Table 4: *Height for Age*[a]

	I continuous	II continuous	III FIML w. knowledge
Age	–0.0047*	–0.0047*	–0.0055*
	(0.0027)	(0.0028)	(0.0032)
Sex	0.3408	0.3162	0.2765
	(0.5263)	(0.5231)	(0.5836)
Mother's age	0.1167**	0.1107**	0.0787
	(0.0472)	(0.0473)	(0.0504)
Mother's age missing	3.5295***	3.4992***	3.3986***
	(0.9767)	(1.0064)	(1.1996)
Father's education	0.3119**	0.2902*	0.1864
	(0.1518)	(0.1513)	(0.1708)
Father's education missing	0.4625	0.4059	0.1449
	(0.7592)	(0.7627)	(0.9614)
Mother's education	0.2728	0.2182	–0.0575
	(0.2128)	(0.2148)	(0.3167)
Mother's education missing	–1.5247*	–1.3867*	–0.8676
	(0.8090)	(0.8147)	(1.0375)
Knows reason for malaria		0.7849	4.9711**
		(0.5150)	(2.1928)
Knows reason missing		–0.2962	–0.5505
		(0.7461)	(0.7569)
Constant	94.1216***	94.0548***	93.7103***
	(1.6425)	(1.6303)	(1.8022)
σ	3.7954	3.7810	4.2530
ρ			–0.6141***
			(0.2367)
ln L	–608.56	–607.49	–742.32
Observations	221	221	221

Asymptotic Huber-corrected standard errors in parentheses. Statistically significant at the ***1%, **5% and *10% levels.
[a]Measured as a percentage of the reference population median.

mortality of the children in the Bandim 1 area does, however, compare to child mortality in these areas. The fact that health knowledge appears to have a positive effect and, in fact, 'crowds out' the effects of the education of the mother is in line with the findings of Thomas *et al.*

(1991) and Glewwe (1999). As in Glewwe (1999), not taking account of the endogeneity of health knowledge leads to a large downward bias in the effect of health knowledge.

Our results have two interesting policy implications. First, since health knowledge need not be closely associated with the income or education of the parents, a focused transfer of knowledge may be beneficial to child health. This is especially relevant for women older than, say, 15 years, who are no longer in school. Secondly, it appears that teaching health knowledge in school, and particularly at an early age, may well be a way to improve the survival chance of future generations of children, especially since so many girls leave school early.[24]

As shown by Glewwe (1999), different types of health knowledge were closely correlated in Morocco and they were all important in explaining child health. It is likely that something similar is the case in Guinea-Bissau. Thus, even though we use the knowledge of the reason for transmission of malaria as a single measure of health knowledge, a health education programme should no doubt have a wider focus.

Finally, it is clear that while many studies have identified a positive effect of the education of the mother on child health, it is an important challenge for future research to examine in more detail exactly how this connection comes about.[25] This study has, in line with Thomas *et al.* (1991) and Glewwe (1999), shown that important and policy relevant information does emerge when the effect of education on health is 'disaggregated'.

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[24] Table 1 depicts the significant differences in male and female education of the parents in our cohort.

[25] Related to this is that closer attention also needs to be paid to the quality of the schools and what is actually taught.

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