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Research Article

**Does biological relatedness affect
survival?**

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ABSTRACT

Objective: We studied child survival in Rakai, Uganda where many children are fostered out or orphaned.

Methods: Biological relatedness is measured as the average of the Wright's coefficients between each household member and the child. Instrumental variables for fostering include proportion of adult males in household, age and gender of household head. Control variables include socioeconomic status (SES), religion, polygyny, household size, child age, child birth size, and child HIV status.

Results: Presence of both parents in the household increased the odds of survival by 28%. After controlling for the endogeneity of child placement decisions in a multivariate model we found that lower biological relatedness of a child was associated with statistically significant reductions in child survival. The effects of biological relatedness on child survival tend to be stronger for both HIV- and HIV+ children of HIV+ mothers.

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Conclusions: Reductions in the numbers of close relatives caring for children of HIV+ mothers reduce child survival.

1. Introduction

There are now an estimated 11 million AIDS orphans living in sub Saharan Africa (UNAIDS 2002). (The conventional definition of “orphan” in most studies is a child below the age of 15 who has lost one or both parents.) Consideration of the impact of orphanhood typically emphasizes the orphan’s loss of the dead parent’s *productive capacity* (Over, Ellis, Huber, and Solon 1992) and the need to provide orphans financial security despite the loss of a parent. However there is a growing realization that the prevalent African tradition of child fostering might be sufficient to offset the financial predicament of orphanhood (Foster, Shakespeare, Chinemana, Jackson, Gregson, Marange, and Mashumba 1995; Kamali, Seeley, Nunn, Kengeya-Kayondo, Ruberantwari, and Mulder 1996; Urassa, Boerma, Ng’weshemi, Isingo, Schapink, and Kumogola 1997). Earlier observations on the wide prevalence of child fostering in West Africa (Bledsoe and Brandon 1987; Isiugo-Abanihe 1985) were reinforced by data showing that 14% of Northwestern Tanzanian households (Urassa et al. 1997) and 19% of Southern Ugandan households fostered orphan children (Nalugoda, Wawer, Konde-Lula, Menon, Gray, Serwadda, Sewankambo, and Li 1997). The extended family has been a traditional source of financial security and could be a very successful coping strategy. Yet financial security and caring by foster families may not be able to replace lost parents.

The objective of this paper is to develop a measure of the biological relatedness to household members and to ask whether biological relatedness affects the survival of children. We will show that biological relatedness can be measured, and that it has independent effects on child survival. We suggest that biological relatedness could be a useful proxy measure of the caregiver concern that may be lost as a consequence of adult mortality.

2. Background

The debate in policy circles about what to do about the orphan crisis can be characterized as a debate over the adequacy of foster families. Caldwell and Caldwell (1990) have expressed immense respect for the capacity of the extended African family, writing,

"It is unwise in any economic model to assume a relationship between biological parenthood and economic responsibility for bringing up children. ... costs are widely shared, and few Africans feel significantly less responsible for the schooling costs of their nephews and nieces than for those of their own children."

Optimists who say that no assistance is needed, point to data showing no significant difference in mortality between orphans and non-orphans (Urassa et al. 1997) and minor differences in schooling rates. Nalugoda et al. (1997) found in Rakai that 56% of orphans and 64% of non-orphans were enrolled in school. Ainsworth and Rwegarulira (1992) found that in Kagera roughly 65% of teenage orphans were enrolled in school and 75% of corresponding non-orphans.

In contrast to these optimistic views, Sengendi and Nambi (1997) noted severe psychological effects related to bereavement in both orphaned children and their foster parents in Rakai. In focus groups conducted in Uganda, Ntozi and Mukiza-Gapere (1995) uncovered widespread reports of orphans being dispossessed and stigmatized for fear of AIDS. Lack of money affected 58% of Ugandan orphans (Ntozi 1997). The Kagera data show that mother's deaths (but not father's deaths) are followed by child stunting (Ainsworth and Semali 1998). This would indicate that while widowed and foster families are able to buffer the child's health from the devastating loss of a father's resources and protection, they cannot insulate the child from the loss of a mother. Ntozi (1997) concludes that,

"...the extended family has tried its best to cope with the orphan problem without much external assistance. It is therefore recommended that the family's weak financial capacity to cope with the orphan problem should be countered by external assistance from government and international and local AIDS groups."

3. A sociobiological perspective

There is some evidence from sub Saharan Africa to support the interplay of household kinship with child welfare. In much of Sub-Saharan Africa, school enrollment is lower for orphans relative to children living with parents (Ainsworth and Filmer 2002). More specifically, in each of 10 countries studied, African orphans fostered to non relatives were less likely to attend school (Case, Paxson, and Ableidinger 2002). One reason to suspect that foster families will not make up for the losses undergone by orphans is a belief that foster parents being less biologically related to a child will be less solicitous of a foster child's welfare. The sociobiological perspective assumes that natural selection would ordinarily favor selfish over altruistic behavior. Sociobiologists have modified the definition of "selfish behavior" to include activities which benefit one's

kin and which increase the reproduction of whatever genes are held in common among kin. This has been stated as Hamilton's Rule:

"The social behavior of a species evolves in such a way that in each distinct behavior-evoking situation the individual will seem to value his neighbors' fitness against his own according to the coefficients of relationship appropriate to that situation." (Hamilton 1964)

The coefficient of relationship (ω) is derived with reference to Mendelian inheritance. For example, ω is $\frac{1}{2}$ for parents, $\frac{1}{4}$ for grandparents etc. In its simplest form Hamilton's rule can be expressed as: $\omega b - c > 0$ where b is the fitness benefit to the beneficiary and c is the fitness cost to the altruist. The case where $\omega = 1$ suggests a case where the giver and receiver are the same organism. Here, Hamilton's rule reduces to the basic principle of rational choice theory: the benefit of any action must exceed its cost.

One reaction to the proposition that Hamilton's rule might apply to human behavior would be to reject it a priori by appealing to the countless cases of altruism among unrelated persons and the superb outcomes realized by adopted children around the world. However Hamilton's rule should not be interpreted as a natural law that can never be violated. Instead, Hamilton's rule refers to an equilibrium that the forces of natural selection would bring about if left unchallenged. A priori, Hamilton's rule may or may not be a central tendency for humans at present. The best test of whether Hamilton's rule applies to humans would use data on intrahousehold transfers describing their fitness benefit and fitness cost. Such a study would leave open the question of how important these statistical effects are for child survival. In this paper we will look at the reduced form of the impact of biological relatedness on child survival. If biological relatedness turns out to be important for survival, subsequent studies would be warranted to establish a detailed causal pathway. In this paper we simply ask whether household relatedness coefficients are correlated to child survival.

4. Methods

4.1. Data

Our data come from the rural Rakai district of Uganda, which has a year 2000 population of 425,000 residing about two hours by road from Kampala (Nalugoda et al. 1997; Wawer, Serwadda, Musgrave, Konde-Lule, Musagara, and Sewankambo 1991). Individuals under age 15 constitute 49.5% of the population. In surveys between 1989 and 1992 HIV prevalence among persons aged 15 and over was 35% in trading centers on main roads, 23.1% in trading villages on secondary roads, and 11.8 % in rural

villages (Nalugoda et al. 1997). Latest estimates suggest that approximately 29,000 children in the district have lost at least one parent; 4500-5000 of these have lost both parents (Nalugoda et al. 1997).

The Rakai Project has been studying the population of Rakai District and its epidemiological experience since 1988 (Gray, Wawer, Serwadda, Sewankambo, Li, Wabwire-Mangen, Paxton, Kiwanuka, Kigozi, Konde-Lule, Quinn, Gaydos, and McNairn 1998). Among other activities this project has followed the populations of 56 communities located on secondary roads between 1994 and 2001. The original purpose of the population cohort was to evaluate the impact of preventive interventions against sexually transmitted diseases (STDs) and AIDS by a community randomized clinical trial. This paper uses data from the Maternal Infant Supplementary Study (MISS) cohort which was comprised of pregnant mothers and their children identified during the STD trial (1994-1999).

4.2. Sampling Frame For Maternal Infant Supplementary Study (MISS)

A baseline cohort composed of 6216 women aged 15-59 entered surveillance in 1994. Women in this cohort who became pregnant while under surveillance between 1994 and 1999 were prospectively enrolled. Participation exceeded 90% of all who were approached. The women were given an extensive pre-partum physical exam, lab tests, and interviews. Mothers and surviving newborns were visited postpartum and both were examined. Data collection included prenatal STDs and HIV, pregnancy outcomes including anthropometry, prenatal and neonatal demise and subsequent determination of child HIV infection and long-term survival. As of late 2000, child vital status had been determined for 3635 children based on census and follow up data collected from 1998 to 2000. Tests for HIV used two enzyme immunoassays with Western blot confirmation of discordant assays. Infant HIV infection was diagnosed by RNA RT-PCR (Roche Amplicor 1.5).

4.3. Analytic Sample

The relationship of a child to the head of household was determined from the census records. Biological relatedness to all household members could be unambiguously determined for 2322 of the children whose vital status was known. The most common reason for ambiguity was children whose relationship to the household head was coded as “other” (as opposed to “unrelated” or “foster”). These 2322 children compose the analytic sample for the bivariate analyses. Multivariate regression models requirements

for complete data on a number of sociodemographic factors and HIV status further reduced the eligible sample to 2208 children.

5. Assessing Biological Relatedness

In a context with prevalent fostering of children and frequent adult migration, orphanhood is the most stressful of several ways in which children's kinship ties to household members are diluted. In order to assess all processes diluting child's kinship we sought to parameterize the biological relatedness of children to other household members.

During the annual census conducted by the Rakai Project a household roster is completed including "relationship to household head" for every household member. We used the algorithm depicted in Table 1 to assign a coefficient of biological relatedness between each household member and the child of interest based on that child's own relationship to household head, and the relationship of each household member to the household head. In addition one of the investigators (ES) visually inspected the data for all cases where a child's relationship to household head was "other" or "grandchild" (Note 1).

We calculated the unweighted sum of all of biological relatedness (Wright's) coefficients $\times 100$ and divided it by the number of non-self household members to form an index of biological relatedness (IBR). Had we not adjusted by household size, the sum of Wright's coefficients would have been itself reflective of household size rather than kinship. The natural interpretation of IBR is the percentage match of the propositus's genes to a simple random sample of the gene pool of all other household members. Such an index would be 100% for each member of a household made up of clones (or of 2 identical twins), 50% for each sibling in a household of only full siblings and 0% for an adopted unrelated child in any household.

5.1. Statistical Analysis

As a simple means of assessing the relationship of household structure to child survival we used 2 by 2 tables to calculate the unadjusted odds ratios of survival for children with various combinations of parental absenteeism and parental demise. We stratified the analysis by maternal HIV status in order to isolate effects that may differ for children of infected and uninfected mothers.

Multivariate models were estimated by stratifying on woman's HIV status, and infant HIV status controlling for other socio-demographic variables. Covariates

included in the models included mother's age (categorical: 15-19 or >30yrs; reference group was 20-30), mother's education (any versus none), low socioeconomic status (owning less than the median number of assets), religion (Muslim versus non-Muslim), and infant chest circumference (low versus normal, >30cms), which is a proxy measure of low birth weight (WHO 1993). These multivariate models were adjusted to control for endogenous child placement decisions as discussed below.

5.2. Controlling for the Endogeneity of Biological Relatedness

Intuition and economic theory suggest that the biological relatedness of a child could be heavily influenced by household choices related to fostering, the survival of other household members, and economic resources. There might be reverse causation in which a child's survival propensity affects the household structure for that child. For instance, a child's endowment of poor health might influence decisions to foster out that child or decisions to have extended family move in or out. In order to account for the potential endogeneity of IBR we instrument it using as instruments: proportion of adult males in household, age of household head, and gender of household head. These instruments are assumed to affect child survival only through their effect on the biological relatedness of the child to other household members. Objections that these instrumental variables have their own "independent" effects on survival might be launched on the grounds that households with older heads and more male members may have a lower preference for child well-being than a younger and more female dominated household. However, biologists explain a lower male preference for the survival of offspring on the basis of lower male certainty of paternity and a perception of potentially lower biological relatedness by males. Such a putative "independent" effect of maleness on preferences would actually be mediated by biological relatedness after all. Another criticism may be that the instrumental variables are predictive of unmeasured components of SES and hence may directly influence child survival. Later we offer empirical evidence that independent effects of household structure via SES are not occurring. Ultimately an analysis based on instrumental variables requires cautious interpretation and an evaluation of the plausibility of the identifying assumptions. The required assumption here is that child frailty causes few if any Ugandan households to make significant shifts in the age and sex of the household head or the proportion of male household members.

The socioeconomic variable (SES) used in the model was created using a simple additive scale of household modern possessions: vehicles, radios, household construction materials, electricity, and latrines. Households whose asset score was less than the median were coded as low SES. Sensitivity tests confirmed that the effects of

biological relatedness markers on survival were not significantly affected by alternative SES constructs based on principal components analysis following Filmer and Pritchett (Filmer and Pritchett 2001).

We use a full information maximum likelihood (FIML) approach. The FIML system is composed of a probit equation predicting the probability of child death as a function of covariates including the index of biological relatedness. The other equation in the FIML system is the maximum likelihood equivalent of an ordinary least squares equation predicting the index of biological relatedness from the same covariates and, in addition, the three instruments: proportion of adult males in household, age of household head, and gender of household head. Hausman tests are used to test the hypothesis that the biological relatedness is endogenously tied to child survival.

6. Results

Table 2 shows the means, proportions and standard deviations of the variables used for the analysis. One can gain a preliminary impression that children who died had a higher percentage of mothers who were absent or deceased and a lower index of biological relatedness.

Table 3 confirms the general impressions gleaned in Table 2—indicating that the risk ratio of child death is statistically significantly lower for children with both parents present. The effects of parental presence on survival is stronger among HIV positive mothers.

Table 4 explores the determinants of household structure. The purpose of this exercise is to assess whether the instrumental variables are strong or weak predictors of biological relatedness. Column [1] and column [2] show the results of probit models of the probability of mother absence, and indicates that mother's absence is more likely for children with HIV positive mothers. Maternal absence appears more common among children with younger mothers. The household structure instruments—age of head, sex of head, and percent of adults who are male—are correlated with maternal absence. Column [3] indicates that the household structure instruments have little direct relationship with child mortality, and would be appropriate as instruments if they were well correlated with biological relatedness. Column [5] shows the parameters of the regression used to instrument the index of biological relatedness. Comparing columns [4] and [5] one can see that the incremental R^2 of the three instruments was $0.2662 - 0.0095 = 0.25$ suggesting that instruments are well-correlated with the index of biological relatedness (Shea 1996). We find in column [5] that biological relatedness is higher in male headed households and decreases as more males are added to the household. Biological relatedness first decreases then increases with the age of the

mother and decreases for mothers who are HIV positive. Relatedness appears to be increased in polygynous households even though the algorithm which computed biological relatedness coded as zero the Wright's coefficient of head's co-wives. The potentially higher number of half-siblings (with Wright's coefficients of 0.25) that children have in polygynous households may be sufficient to raise the IBR.

In Table 5 we present probit and instrumental variables (IV) probit estimates of the odds ratios of survival based on the instrumented IBR. The elasticity of the relationship between IBR and the probability of child death based on column 1 was calculated as -0.424 (95% CI: -0.953; 0.105). In concrete terms this means that a 50% increase in the IBR (equivalent to replacing 1 stepparent with 1 biological parent of a two parent-one child family) would lower the probability of child death by 21% ($= 0.5 \times -0.42$). A 5% increase in IBR would lower the probability of death by 2.1%. More familiar factors associated with higher odds of child death in the full sample included low SES, no maternal education, and inadequate birthweight. In stratified analyses it appeared that the effects of IBR were similar in "mothers present" households (column [3]) where none of the variation in IBR could be due to absent mothers. The P-value for IBR in column [3] is $P=0.12$. The coefficient on IBR is larger in "fathers present" households where reductions in biological relatedness are more likely to reflect the absence of mothers. This asymmetry is consistent with the common sense notion that not all reductions in biological relatedness are the same—mothers are likely to count more.

The Hausman test statistics at the bottom of Table 5 are all negative. While it is impossible to directly interpret these results using a chi-squared distribution, one author suggests interpreting below zero Hausman tests results as zero and to thus accept the null (Greene 2000). In this case the null hypothesis is that the index of biological relatedness is exogenous to child survival.

7. Discussion

Past studies of the effects of household structure have verified the commonsense notion that children's welfare depends on who is present and absent from the household (Sear, Steele, McGregor, and Mace 2002). Rather than continuing to study household structure in terms of who is present/absent, this study operationalizes household structure as a single continuous variable reflecting biological relatedness.

We have measured biological relatedness surrounding children in Ugandan households based on widely available household rosters. One limitation of this approach is that typical questions on relatedness to household head can leave the child's relatedness to other household members ambiguous in non-nuclear households.

Because of this, the sample we studied is relatively enriched with nuclear household structures and is not representative of all of households in the rural district under study. Another potential limitation of our approach is the possibility that household informants feel so attached to their foster children that they report them on the roster as simply children of the head. All of the interviewers were trained to emphasize to informants that the rosters required *biological* relationships to household head. If foster children were reported as biologically related this would be a conservative error—biasing the effects of biological relatedness towards zero. A further limitation is that children with reduced biological relatedness (e.g. due to parental demise) are among the most difficult to follow up. This leads to a degree of sample selection bias in our estimates. Intuitively, we believe that survival outcomes are likely to be worse for children in the households where parental demise or departure uprooted the entire household. Thus the sample selection bias is also likely to bias our estimates towards zero.

Despite these limitations, we do find that the impact of biological relatedness on child survival is statistically significant, even adjusting for the endogeneity of child placement. The effects of biological relatedness are not negligible, although the direct effects of HIV on survival overwhelm other determinants.

8. Conclusion

Our results indicate that households coping with HIV face something more than a cash shortage. They face a shortage of related adults. Children who lose close proximity to kin require special attention, not only financially but also in the form of advocacy, quality time, and mentoring.

Reduced biological relatedness is caused partly by adult death and partly by decisions on child placement. Children of HIV infected parents are more likely to have reduced biological relatedness to adults in their household, even before the death of their mothers. Reduced biological relatedness is associated with reduced child survival.

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Table 1: *Coefficients of biological relationship as a function of relationship to head*

	H	W	S	POH	COH	GCH	GNH	N	A
Head (H)	1								
Wife Of head (W)	0	1							
Sibling of head (S)	.5	0	1						
Parent of head (POH)	.5	0	.5	1					
Child of head (COH)	.5	.5*	.25	.25	1				
Grandchild of head (GCH)	.25	.25*	.12	.12	.5 or .25‡	1			
Grandniece of head (GNH)	.12	.12*	.12‡‡	.06	.06	.12	1		
Niece/neph of head (N)	.25	.25*	.25‡‡	.25	.12	.25	.5‡	1	
Adopted child of head (A)	0	0	0	0	0	0	0	0	1

Notes

. *Possibly 0 if head is polygynous.

‡Possibly 0 if child through spouse's lineage.

‡‡Possible that child is direct descendent

Table 2: *Distribution of children under 5 years of age by own characteristics and Mother's characteristics*

Characteristics of Mothers and Children	Living Children			Dead Children		
	Number Nonmissing	Mean or %	SD	Number Nonmissing	Mean or %	SD
Asset Ownership (SES)						
Low Asset Ownership	2146	55%		315	63%	
Other than Low Asset Ownership	2146	45%		315	37%	
Mother's Education						
Mothers with no education	2146	10%		315	16%	
Mothers with any education	2146	90%		325	84%	
Mother's Age						
<20	2132	16%		313	19%	
age ≥20 and age <30	2132	61%		313	59%	
30+	2132	23%		313	22%	
Age of Head of Household	2141	34.69	2.69	315	35.74	14.40
Sex of Head of Household						
% where head is male	2141	87%		315	85%	
% where head is female	2141	13%		315	15%	
Household # of Males	2146	2.79	.48	315	2.98	1.83
HIV Status						
HIV Positive Mother	2090	15%		309	27%	
HIV Negative Mother	2090	85%		309	63%	
LBW						
Chest <30 cm	2104	8%		264	13%	
Chest ≥30 cm	2104	92%		264	87%	
Polygyny						
Polygyny (>=2 wives)	1470	20%		199	21%	
Monogamous	1470	80%		199	79%	
Religion						
Muslim	2146	16%		315	15%	
Catholic	2146	62%		315	59%	
Protestant or Pentecostal	2146	21%		315	24%	
Orphanhood/Absent Mother						
Mother Dead	2146	1%		315	2%	
Mother not in HH	2146	3%		315	5%	
Mother Alive and Present	2146	96%		315	83%	
Index of Biological Relatedness	2047	40.91	8.51	264	40.09	8.55

Table 3: *Bivariate Analysis of Household Structures and Child Survival*

MOTHER	FATHER	All			Mother HIV Positive			Mother HIV Positive Child HIV Negative		
		N	% DEAD	Risk Ratio †	N	% DEAD	Risk Ratio	N	% DEAD	Risk Ratio
Present	Present	2027	11%	1.000	275	16%	1.000	241	14%	1.000
Away	Present	44	20%	1.859 (1.025: 3.373) **	12	42%	2.546 (1.238: 5.235) **	11	36%	2.655 (1.142: 6.173) ‡
Dead	Present	12	33%	3.029 (1.348: 6.809) ‡	6	50%	3.055 (1.314: 7.103) ‡	5	40%	2.921 (0.954: 8.947) ‡
Present	Away	204	12%	1.113 (0.756: 1.641)	68	26%	1.617 (1.003: 2.609) *	57	18%	1.281 (0.672: 2.444)
Present	Dead	46	15%	1.383 (0.692: 2.767)	20	35%	2.138 (1.112: 4.115) **	20	35%	2.556 (1.300: 5.026)
Away	Away	47	19%	1.741 (0.955: 3.173) *	8	63%	3.819 (2.097: 6.957) ‡	3	33%	2.434 (0.476: 12.442) ‡

Notes

† Denominator for risk ratio is always mother present, father present population

Confidence intervals are shown in parentheses *p<0.10 **p<0.05 ***p<0.10

‡ Sample size less than 20, confidence intervals unreliable

Table 4: *Assessing the Impact of Household Structure on Biological Relatedness and Child Mortality*

Dependent Variable:	Probit* [1] Whether Mother is Absent	Probit* [2] Whether Mother is Absent	Probit [3] Child Death	OLS [4] Index of Biological Relatedness	OLS [5] Index of Biological Relatedness
N	2,458	2,458	2317	2,450	2,446
Pseudo R 2 (probit) or R 2 (OLS)	0.019	0.0357	0.0137	0.0095	0.2662
Constant	-0.4868 (-2.8) **	-0.3806 (-1.68) *	-1.24 (-9.5) **	42.0067 (70.82) ***	45.6953 (46.90) ***
Household Head Gender (1=Male)		-0.3802 (-3.32) ***	-0.119 (-1.19)		4.9907 (7.43) ***
% of adults who are male		0.0429 (1.6) *	0.026 (-1.44)		-2.4010 (-10.33) ***
Household Head Age		0.0048 (1.88) *	0.002 (-0.79)		-0.0751 (-3.8) **
SES index	0.2922 (3.64) ***	0.2740 (3.30) ***		0.3418 (0.76)	0.2861 (0.8)
Mother Under Age 20	0.2304 (2.84) ***	0.1967 (2.47)		-1.3064 (-2.22) **	0.0408 (0.09) ***
Mother Over Age 29	-0.1668 (-2.12) **	-0.2495 (-2.87) **		-0.7009 (-1.48)	1.8210 (3.33) ***
Mother with No Schooling	0.1458 (1.1)	0.1414 (1.05)		-0.2532 (-0.43)	-0.3856 (-0.7)
Muslim	0.0997 (0.62)	0.0852 (0.53)		-1.1786 (-1.61)	-0.4424 (-0.66)
Catholic	0.0785 (.57)	0.0504 (.37)		-0.6942 (-1.29)	-0.2542 (-.49)
Polygyny (1=Polgynous, 0=No)	0.1408 (1.59)	0.1130 (1.3)		0.1626 (.29)	0.9186 (1.78)
Low Birthweight (1=Chest<30 cm)	-0.1301 (-1.14)	-0.1267 (-1.1)		0.4601 (0.58)	0.3303 (0.55)
Mother's HIV status (1=HIV+, 0=HIV-)	0.2947 (3.11) ***	0.2347 (2.5)		-1.4272 (-2.45) ***	-0.8839 (-1.82) *

Notes

*p<0.10 **p<0.05 ***p<0.01

z-statistics (probit) and t-statistics (OLS) in parentheses are adjusted using robust standard errors

Table 5: Probit Regression of the Probability of Child Death

	Probit All Children [1]	IV Probit All Children [2]	IV Probit Mothers All Present [3]	IV Probit Fathers All Present [4]	IV Probit Both Mothers and Fathers Present [5]
N	2,208	2,208	2,127	1,974	1,936
Log Likelihood	-8,146	-8,146	-7,815	-7,213	-7,071
Constant	-1.231 (-5.45) ***	-1.022 (-3.63) ***	-1.076 (-3.45) ***	-0.433 (-1.2)	-0.592 (-1.71) *
Index of Biological Relatedness	-0.006 (-1.58)	-0.011 (-1.8) *	-0.011 (-1.57)	-0.025 (-2.99) ***	-0.022 (-2.7) ***
SES index	0.198 (2.37) **	0.196 (2.36) **	0.212 (2.57) **	0.201 (2.36) **	0.217 (2.51) **
Mother Under Age 20	0.150 (1.43)	0.140 (1.35)	0.156 (1.42)	0.112 (1.07)	0.123 (1.12)
Mother Over Age 29	-0.043 (-0.46)	-0.049 (-0.53)	-0.051 (-0.53)	-0.078 (-0.85)	-0.086 (-0.91)
Mother's Education (1=No education)	0.364 (2.46) **	0.365 (2.47) **	0.394 (2.68) ***	0.351 (2.41) **	0.376 (2.57) **
Religion (1=Muslim, 0=Non- Muslim)	-0.082 (-0.65)	-0.085 (-0.68)	-0.050 (-0.38)	-0.051 (-0.41)	-0.042 (-0.33)
Religion (1=Catholic, 0=Non- Catholic)	-0.101 (-1.08)	-0.101 (-1.08)	-0.055 (-0.57)	-0.060 (-0.63)	-0.032 (-0.34)
Low Birthweight (1=Chest<30 cm)	0.208 (1.79) *	0.211 (1.82) **	0.230 (1.95) **	0.241 (1.93) **	0.253 (1.97)
Child is Male	-0.049 (-0.76)	-0.052 (-0.78)	-0.045 (-0.65)	-0.039 (-0.53)	-0.024 (-0.33)
Child HIV Infected	1.310 (7.19) ***	1.296 (7.12) ***	1.181 (5.86) ***	0.949 (3.9) ***	0.903 (3.62) **
Hausman Statistic		(-0.56)	(-0.01)	(-0.43)	(-0.19)

Notes

Z-statistics in parentheses are adjusted for clustering using robust standard errors

Index of biological relatedness=100x(Household sum of relatedness coefficients/Count of All Members Except Child)

*p<0.10 **p<0.05 ***p<0.01

Notes

1. In cases where the child was the grandchild of the head, children of the head could possibly be aunts, uncles or parents of the child. Visual inspection of the ages and relationships of the adults could determine these relationships in the majority of cases.

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