The Impact of Adult Deaths on Children's Health in Northwestern Tanzania

by

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ABSTRACT

The AIDS epidemic is dramatically increasing mortality of prime-aged adults in many Subsaharan countries, with potentially severe consequences for surviving family members. Up to now, most of these impacts have not been quantified. Using a longitudinal household dataset from the Kagera region of Tanzania—an area hard-hit by the AIDS epidemic—we examine the impact of the mortality of prime-aged adults on the morbidity, height for age, and weight for height of children under five.

Controlling for individual characteristics, household assets, and community variables, orphanhood (from one or both parents) and recent deaths of adults in the household (who may or may not be parents) have independent impacts on raising morbidity and reducing height for age of children under five. Children from the poorest households, those whose parents are/were uneducated, and those with the least access to health care are the most severely affected. The impact of adult deaths on reducing height for age is delayed (not appearing until 4-6 months after an adult death) but substantial. There was no significant relation between adult mortality (or most other explanatory variables) and weight for height.

While an unknown share of excess morbidity and malnutrition due to adult deaths in this sample can be attributed to HIV infection in the children, we show that three important health interventions—measles immunization, oral rehydration salts, and access to health care—have important roles in mitigating the impact of adult deaths. Not only do these programs disproportionately improve health outcomes among the poorest children; among the poor they disproportionately benefit children affected by adult mortality.

I. Introduction

The health of children in developing countries has improved dramatically over the past decades. Between 1960 and 1990, infant mortality declined by half—from 226 to 106 per thousand—as a result of rising incomes, improved health care, increases in female education, and better nutrition (World Bank 1993). There is also evidence from many developing countries that children are taller and less wasted than in the past (Alderman 1990, Den Besten and others 1995). These trends represent not only improved welfare now, but greater productivity when these children reach adulthood. Malnutrition leads to delayed intellectual development (Brown and Pollitt 1996). Better nutrition in early childhood can improve cognitive achievement and affect subsequent schooling decisions (Behrman 1993). Better nourished children have higher educational achievement, even after controlling for the home environment and parents' tastes (Glewwe and others 1998). Taller adults earn higher wages, even after controlling for education, and this effect is particularly pronounced in low-income countries (Strauss and Thomas 1998).

The AIDS epidemic in Sub-Saharan Africa is already slowing if not reversing this trend (Timaeus 1998). In 15 countries, 5 percent or more of pregnant women attending prenatal clinics are infected with HIV, and in Southern Africa, maternal infection is as high as 30-40 percent (World Bank 1999). Children of HIV-positive mothers have higher prematurity, higher intra-uterine growth retardation, and higher incidence of low birthweight, compared to children of HIV-negative mothers (Taha et al 1995).[1] Mortality rates of children born to HIV-positive mothers are 3-10 times higher than for children born to HIV-negative mothers (Boerma et al 1998, Lallemont et al 1989, Taha et al 1995).[2]

Some of the increased mortality of children of HIV-positive mothers is due to mother-to-child transmission of HIV. Without any intervention, 13-48 percent of all newborns contract HIV from their HIV-infected mothers in utero, at birth, or via breastfeeding (World Bank 1997). There is no cure for HIV/AIDS once the child has acquired it. Stunting, nutritional wasting, acute, chronic and persistent diarrhea, failure to thrive, pneumonia, thrush, and neurological abnormalities are all associated with HIV

[1] These findings have not been universal, however. Lallemant et al (1989), for example, found no difference in the rate of stillbirths, gestational age, or birth weight among babies born to HIV-positive and HIV-negative mothers. Part of the reason for the diverse findings may be due to differences in methodology. Some are studies of HIV-positive and negative mothers that are "matched" on different characteristics, while others are prospective studies of HIV-positive and HIV-negative women and their children, in which socioeconomic and other characteristics are controlled for in multivariate analysis.

[2] In a prospective study of HIV-positive and HIV-negative mothers in urban Malawi, the mortality rate at 30 months was three times higher for the children born to HIV-positive mothers (36%) than for HIV-negative mothers (12%) (Taha et al 1995). In a study that matched HIV-positive and HIV-negative mothers on age, date of delivery, and place of residence in Brazzaville, Congo, the survival rate at 12.5 months was 97% for children of HIV-negative mothers but only 61% for children of HIV-positive mothers (Lallemant et al 1989).

infection in young children (Bailey et al 1999; Bobat et al 1998, 1999; Lepage et al 1996; Taha et al 1999;Thea et al 1993). Treatment with AZT before, during, and after birth, and substitution of bottled for breastmilk can reduce mother-to-child transmission rates by half to two-thirds in controlled clinical trials, but are still expensive to implement successfully in resource-scarce settings. More recently, a single dose of nevirapine for mothers during labor and children after birth has been shown equally if not more effective and far less costly (Marseille et al 1999). The only intervention that completely protects children from mother to child transmission is prevention of HIV in the mother. Clearly, public policy on AIDS prevention will have an important role in reducing the impact of the epidemic on malnutrition, morbidity, and mortality of young children.

The worsening of child health outcomes due to the AIDS epidemic reaches beyond the effects of mother-to-child transmission, however. Children not infected with HIV may have higher morbidity and lower nutritional status in a household with an AIDS patient because they are exposed to other infectious agents. Dayton (1999) finds a positive relation between parents' morbidity and low weight for height in children under 10 in the same sample of Tanzanian children studied here. The increased mortality of prime aged adults may also have important consequences for the health of orphaned children and other co-resident youngsters, through its economic impact. Producing healthy and well-nourished children requires key inputs, such as food and nutrients, health care, and the time of caretakers. The loss of productive adults reduces household income, indirectly reducing the ability to purchase or produce these inputs and directly reducing the adult time available to transform them into improved child health. In addition, if investments in child quality are linked to some anticipated future return in terms of old age security for parents, and if other adults do not expect these returns from children who are not their own, then the loss of a parent will lead to lower investments in health care and schooling for orphaned children.

To date, there is very little evidence about the magnitude of the impact of adult deaths on child health through channels other than mother-to-child transmission of HIV. In a study in Blantyre, Malawi, Taha et al (1996) found that, controlling for household socioeconomic characteristics, the child's gender, birthweight, first-born status, and the age and HIV status of the mother, young children whose mothers died were 3.3 times more likely to die themselves, compared to the children of mothers who didn't die. In contrast, in Zaire, Ryder et al (1994) found no difference in morbidity or indicators of social and economic well-being among HIV-negative AIDS orphans and age-matched children of HIV-positive and HIV-negative mothers alive at the time of the case mother's death. The authors concluded that "..the presence of a concerned extended family appeared to minimize any adverse health and socioeconomic effects experienced by orphan children". In a large study in Bangladesh that was not related to the AIDS

epidemic and in which it can be presumed from the timing of the study (1983-89) that HIV infection was not a factor, Strong (1998) found higher mortality for both sons and daughters who lost their mothers but not for those who lost their fathers. There was also higher mortality among girls when a woman who was not the mother died, but the death rates for boys and girls were not affected by other adult deaths (age 15-59). However, in a multivariate analysis of a subset of these children observed from age 12-83 months, deaths of other adults also significantly affected the child's survival probabilities.

This paper uses longitudinal socioeconomic data collected from households in the Kagera Region of Northwestern Tanzania in 1991-94 to address two questions. First, what will be the impact of the loss of adults and parents on the health of young children? We consider both the effect of orphanhood and the effect of the loss of other productive adults in the household on the morbidity, height, and weight of children under five years of age. We know of no other studies of child health that have been able to examine these impacts: prime-aged adult deaths are rare and studying their impact would require a very large sample. The survey we use took place in a region with elevated adult mortality due to the AIDS epidemic, and was conducted in a random sample of households stratified on indicators predictive of adult mortality, increasing the probability of observing an adult death. In addition, many studies of the health status of children exclude from the analysis orphans and children living away from their parents because the characteristics of their parents are unknown (e.g., Alderman 1990, Sahn 1990, Strauss 1988). However, these children may be at greatest risk of poor health outcomes and are the focus of this study.

The second issue we address is policies to reduce the impact of adult mortality on child health. There are many individual, household, community, and health-service factors that can affect child health outcomes. We identify the characteristics of children at greatest risk of poorer health as a result of adult deaths and the program inputs in the community and at the nearest health facilities that show the greatest positive relationship with child health. The next section discusses the economic model of the demand for child health that guides the choice of exogenous explanatory variables that are included as determinants of the reduced-form health demand equations. The third section describes the dataset and variables. The fourth section presents estimates of the reduced-form demand equations for current illness, height for age, and weight for height. In the final section we summarize the results and the main policy conclusions.

II. Analytic framework

Child health outcomes are the result of numerous decisions by the household concerning food consumption, the use of medical care, the amount of time spent caring for children,[3] and other "inputs" into childrearing. Exogenous factors also play a role—such as the child's (unobserved) frailty, disease vectors in the community, sanitation levels, food prices, the availability, quality and price of medical care, cultural practices, and social support structures. These affect child health either directly or indirectly by affecting household decisions on child health inputs. The characteristics of household members (such as their age, sex, or education)—particularly the mother's education—may affect the efficiency with which various inputs are used to produce child health and the quality of those inputs, like child care time. Finally, individual characteristics such as the child's age and gender affect growth through biological channels.

We posit that child health is produced at home according to equation (1), which expresses health outcomes of child i at time t as a function of three main household inputs: nutrient intake (N_{it}); medical care (M_{IT}); and the time input of adults (T^{a}_{it}). The efficiency of transforming these inputs into good health outcomes is mediated by parents' education (E^{fm}_{i}). Finally, the production of child health is conditioned on the child's individual endowments (C_{it}), household endowments (C_{ht}), and community characteristics (C_{jt}).

(1) $$H_{it} = H (N_{it}, M_{it}, T^{a}_{it}\, E^{fm}_{i}, C_{it}, C_{ht}, C_{jt}, e_{it})$$

where h denotes the household, j denotes the community, and e_{it} represents unobserved factors at the individual, household and community level that affect child health (see, for example, Behrman and Deolalikar 1988, Schultz 1984). Child endowments include age and gender, household endowments include physical assets and human capital of household members, and community characteristics include the availability and price of medical care, food prices, disease vectors, wages, rainfall, and other exogenous community attributes.

The death of a parent or adult in the household can be thought of as an exogenous shock to the child health production function. It will have a direct impact on household income, which conditions the purchase of child health services and food, as well as on the amount of adult time available to transform or

[3] Child care includes time spent feeding, bathing, seeking medical care, and stimulating a child's cognitive development (Engle 1995). We expect that not only the availability of time for child care but the type of interaction will have important effects on child health. However, information on these specific behaviors are not available.

process these inputs into better child health outcomes. However, household structure and composition in these households are dynamic (Ainsworth et al 1995, Ainsworth 1989). The impact of adult deaths through income and time shocks on child health is likely to be transitory as surviving household members remarry, join, or leave the household.

In much of Sub-Saharan Africa, children live in extended households that include multiple generations, aunts, uncles, and adult siblings. Thus, the death of an adult in the household does not necessarily imply that a child has lost his/her parent. The impact of these non-parental adult deaths may be thought of as transitory shocks to the production of child health. The death of a parent may have a more permanent effect, however. Other household members may not be good substitutes for a mother, for example, when children are very young and being breastfed. Parents, who have more intimate knowledge of their children's behavior and endowments, may be more efficient at transforming health and food inputs into child health. Further, the health of their children may hold greater weight in their own utility function than other children in the household. One's own children are a long-term source of income and old-age security in many societies; thus, a child's well-being is likely to be of concern to a parent, even if they must live apart. An adult in the household can be replaced, but the loss of the special attention given a child by a parent, especially early in life, is likely to have long-lasting implications.

The first three arguments in the production function—nutrient intake, medical care, and adult time—are endogenous, representing household decisions about the use of inputs. Estimating equation (1) would reveal important technical relationships between child health outcomes and these key inputs. However, in practice, these production functions are very difficult to estimate because the inputs and outcomes are jointly determined (Cebu study team 1991).

If we assume that households maximize the utility of their members over consumption, leisure, and child health, subject to a budget constraint and to the production function for child nutrition (1), we can solve for a reduced-form equation of the demand for child health, in which all of the explanatory variables are exogenous:

$$(2) \qquad H_{it} = H (E^{fm}_i, C_{it}, C_{ht}, C_{jt}, P_{jt}, W_{jt}, I_{ht}, D_{ht}, error_{it}),$$

where P_{jt} and W_{jt} are community-level prices and wages, I_{ht} is exogenous household unearned income, and D_{ht} is a measure of recent adult death in the household. A child's "orphan status" (the survival of his/her parents) is part of C_{it}, his/her individual endowment.

In this reduced form model, adult deaths and parents' deaths are expected to worsen child health through their negative impact on household income and the availability of adult time for production of child health. Educated parents should have healthier children by marshalling information and other inputs more efficiently to affect child health. Higher prices of medical care and food are expected to worsen child health by reducing the demand for health care and nutrients/purchased food, while better access to medical care and child health services in particular should be associated with better health outcomes. Higher wage rates for adults and children in the community are posited to raise the opportunity cost of time of care givers, prompting them to reduce the time spent producing child health. However, to the extent that these opportunities also raise income, higher wages could have a net positive effect on child health. Household wealth or assets should be associated with better health through the ability to buy more medical care and to purchase more and better quality food.

Health is multidimensional (Strauss and Thomas 1998). We estimate equation (2) for three related dimensions of child health: morbidity on the day of the interview; height for age; and weight for height. Low height in relation to a child's age is often referred to as "stunting" and low weight for height as "wasting". Both stunting and wasting reflect nutritional status, but there is no necessary relation between stunting and wasting at a given point in time: "... stunting is the product of a cumulative history of episodes of stress that led to reduced growth rates and that were not later made up by catch-up growth during more favorable periods. ... wasting reflects the presence or absence of stress at the time of measurement. These episodes of stress can be caused by chronic factors and/or by acute factors" (Martorell and Habicht 1986, p. 245). As noted by Waterlow (1992), weight deficits can be reversed quickly, while height deficits are slower to develop and slower to recover. Height for age should be thought of as reflecting a chronic process rather than chronic malnutrition. "…statistically, the two states of being wasted and stunted are not significantly associated" (Waterlow 1992, p. 195) Morbidity and malnutrition have a synergistic relationship. Illnesses such as tuberculosis, diarrhea, and measles, have well-documented biological effects on worsening children's nutritional status, while severely malnourished children have higher morbidity and mortality (Pelletier 1994, Waterlow 1992). Evidence of the impact of moderate malnutrition on the risk of morbidity and mortality of HIV-negative children is inconsistent and seems to depend on the setting (Chen et al 1980, Pelletier 1994, Schroeder and Brown 1994, Waterlow 1992). In the case of children with HIV/AIDS, there is a clear relation between morbidity and anthropometric measures: HIV-infected children are more likely to be stunted, underweight, and wasted compared to uninfected children of the same age, even when the mothers of the uninfected children are HIV-positive (Bailey et al 1999).

III. Data and descriptive statistics

The data for this analysis come from a longitudinal living standards survey of households conducted in the Kagera region of Northwestern Tanzania from 1991-94. Kagera is located to the west of Lake Victoria and borders the Rakai district of Uganda to the north, the countries of Rwanda and Burundi to the west, and Mwanza, Shinyanga, and Kigoma regions of Tanzania to the south. As of the 1988 census, about 1.3 million people lived in Kagera region. More than 80 percent of the population lives in rural areas, most of them involved in agriculture—tree crops in the north (bananas and coffee) and annual crops and livestock in the south. Because of its location next to Lake Victoria, which was settled by missionaries, the level of education in Kagera was one of the highest in Tanzania. Based on the dataset used for this analysis, household consumption expenditure per capita in the region was about $217 per capita in 1991, with a range of US$118 to $337 across the six districts.

The impact of the mortality of prime aged adults is difficult to measure because mortality of adults 15-50 is a rare event. In Kagera, however, adult mortality is higher than would be expected because of the early spread of HIV/AIDS in the area around Lake Victoria. The first case of AIDS in Tanzania was identified in Kagera in 1983, although HIV was probably present in the area at least a decade earlier. The area is at a crossroads for long-range commerce between the East African coast and central Africa, and was also heavily affected by the war between Tanzania and Uganda in 1978-79. More recently, it has been the site of refugee camps for those fleeing from conflicts in Rwanda and Burundi. Thus, it is not surprising that HIV spread early and quickly in Kagera region and the surrounding areas. A population-based survey in Kagera in 1987 found that roughly a quarter of prime-aged adults in the regional capital of Bukoba were infected with HIV, as were 10 percent of prime-aged adults in the surrounding rural areas in the northern and eastern part of the region near Lake Victoria (Killewo and others 1990). However, there was also variation in infection across the region, with a 5 percent infection rate among adults in the west and less than one percent infection rate in the south.

The dataset used here is the Kagera Health and Development Survey (KHDS), which interviewed 816 households over four passages at 7-month intervals between 1991 and 1994. The objective of the KHDS was to measure the economic impact of adult mortality on surviving household members. The household sample was random, stratified on geography, community adult mortality rates (as measured in the 1988 census and a subsequent enumeration for the survey), and indicators at the household level that were thought to be predictive of future adult deaths (see Over and Ainsworth 1989,

Ainsworth and others 1992, KHDS research team 1999). A household was defined as a group of persons living and sharing meals together in the same dwelling for at least 3 of the past 12 months. The KHDS collected extensive, detailed information on household income, consumption expenditure, and individual health status, including the height and weight of all household members. In addition, a great deal of detail was obtained on the mortality of household members. The KHDS did not perform any medical tests to determine the HIV status of respondents.

For the present analysis, we use health outcome measures from a maximum of 1,108 children under the age of 60 months, interviewed or measured from 1-4 times during the course of the survey, and for whom there were non-missing values of the dependent and explanatory variables.[4] Over the four passages of the longitudinal survey, we have 2,679 total observations to analyze from these children. Table 1 defines the variables used in the analysis and provides descriptive statistics on the sample.

Dependent variables

Our first measure of child health is whether the child was reported to be ill or injured on the day of the interview. By this measure, more than a quarter of the children in the sample (29.3 percent) were sick.[5] The most frequently reported symptoms were fever (26 percent) and diarrhea (15 percent). Parents or caregivers believed the children were suffering from: the common cold (32 percent); malaria (11 percent); diarrhea (5 percent); parasites (5 percent); and measles (1 percent). In 41 percent of the cases "other illnesses" were cited. These assessments were based on their own observation or that of a health professional, if one was consulted. None of the parents or caregivers reported that a child was suffering from AIDS. This is not surprising, however, since few parents would be aware of their children's HIV status, and AIDS presents itself as a series of common childhood illnesses and symptoms. Of the 784 children reported sick on the day of the interview, 20 were suffering from 3 common AIDS symptoms and 4 were suffering from all four symptoms, although these could also be attributed to other severe illness.[6]

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[4] 298 were interviewed four times, 208 three times, 261 two times, and 341 one time. Descriptive statistics in the text pertain to the pooled sample of children, which includes observations from more than one time period for many of them.

[5] There were several other measures of morbidity that could have been used, including: acute illness in the past 4 weeks; chronic illness lasting 6 months or more; chronic diarrhea or other symptoms of AIDS. We decided to use acute morbidity on the day of the interview as less subject to "telescoping" and recall biases than reported illness over a longer period (Behrman and Deolalikar 1988). Nevertheless, some bias in the reporting of child illness may remain and could be correlated with the socioeconomic characteristics of the household or the availability of health care (Sindelar and Thomas 1991, Strauss and Thomas 1998).

[6] The four main symptoms of AIDS about which all respondents were queried are: chronic diarrhea; severe weight

The second two indicators of child health are measurements of child height (or recumbent length) and weight. The nutrition indicators compare the weight and height of children in the KHDS sample with an international "reference" population of well-nourished children defined by the U.S. National Center for Health Statistics (NCHS) (WHO 1995). Researchers have found that well-nourished children in developing countries approach the mean nutritional status of the international reference population; environmental rather than genetic factors account for most differences in nutritional status (Habicht and others 1974, Martorell and Habicht 1986, Waterlow 1992).

Each child's nutritional status can be described by the number of standard deviations of his/her height for age and weight for height from the median of the reference population, also called the "z-score". For example, the height of a child with a height/age z-score of zero is the same as the height of the median child of the same age in the reference population. The height of a child with a height/age z-score of -1 is one standard deviation (SD) below the median height of children of the same age in the reference population.

The average child under five in the KHDS sample was short for his/her age but not thin. The mean height-for-age z-score was 1.7 standard deviations below the median of the reference population, while that for weight for height was only 0.26 standard deviations below the reference median. The common cut-off point for identifying severely malnourished children is a measurement more than 2 SD below the median of the reference population. Children 2 SD below the median in height/age are "stunted", while those 2 SD below the median in weight/height are "wasted". In the NCHS reference population, only 2.3 percent of children would be classified as stunted or wasted. According to this definition, 37 percent of the children in the Kagera sample were stunted, 2 percent were wasted, one percent were both stunted and wasted, and 59 percent were neither stunted nor wasted. These levels of malnutrition are lower than was found in the 1991-92 Tanzania Demographic and Health Survey.[7]

Explanatory variables

The explanatory variables in Table 1 are classified according to the factors in equation (2) that they represent.

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loss; chronic fever; and skin rash.

[7] The 1991-92 Tanzania Demographic and Health Survey found 47 percent of children under five stunted and 5.5

Individual endowments. These include the child's gender and age in months, his or her parents' education, and whether or not the child is an orphan. About half of the children were girls and the mean age was 29.8 months. Their mothers had 5 years of schooling, on average, and their fathers 6 years.[8] In very young children, it is relatively rare to have lost a parent, particularly the mother. Over all of the observations in the sample, 4 percent had lost their mother and 11 percent their father: 2.9 percent had lost their mother only, 10.0 percent had lost their father only, and 1.3 percent had lost both parents. The rate of orphanhood increases by age: maternal orphan rates rise from 0.8 percent among children under two to 6.4 percent among children aged 2-4 years (not shown). Paternal orphan rates are usually higher than maternal rates because fathers are generally older. They ranged from 5.9 percent to 14.8 percent for children under 2 and aged 2-4, respectively.

Adult time. The time spent producing child health and the quality of that time was not available from the survey and, in any event, would be endogenous. Thus, we use three household composition variables as indicative of the potential availability of adult time in the production of child health: the number of female adults aged 15-50; the number of girls aged 7-14; and whether or not the head of the household is female. Controlling for other factors, we expect that households with more female caregivers will have better health outcomes and that female-headed households may be more cognizant of or attentive to child health problems but perhaps more constrained for time inputs into child care. There were about 1.8 female adults and one girl aged 7-14 in the household of the average child; 23 percent lived in female-headed households.

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percent wasted, in a national sample (Bureau of Statistics 1993).

[8] Other analyses of the determinants of child health have dropped children whose parents are not in the household (including orphans) because they are missing variables like mother's height and age (Alderman 1990, Sahn 1990, Strauss 1988). Others have retained parentless children by including a dummy variable for the absence of the parent, but this doesn't solve the problem of not being able to control for these variables in the most affected children. In this analysis, we do not control for parents' height, age, or other variables not available for children with missing parents. Parents' schooling can be included, however, because it was collected for all children, even if the parent was deceased or non-resident.

Table 1. Definition of variables and descriptive statistics

(n=2,679 observations on 1,108 children)

Variable	Definition	Mean	Std. Dev.
Dependent variables			
ill now	Equals 1 if child was ill on the day of the survey, 0 otherwise.	.293	.455
ht_age	Height-for-age z-score[a]	-1.66	1.41
wt_ht	Weight-for-height z-score[b]	-.261	1.03
Explanatory variables			
Individual endowments			
girl	Equals 1 if child is female, 0 otherwise.	.478	.500
months	Child's age in months, entered in a quartic specification (months, months squared/10, months cubed/100, months4/1000).	29.8	17.4
momdead	Equals 1 if child's mother is dead, 0 otherwise.	.042	.201
daddead	Equals 1 if child's father is dead, 0 otherwise.	.113	.317
mgrade	Years of completed schooling of mother.[a]	4.94	2.95
fgrade	Years of completed schooling of father.[a]	5.91	2.88
Adult time			
female head	Equals 1 if household head is female, 0 otherwise.	.228	.420
female adults	Number of adult females 15-50 in the household	1.78	1.28
female teens	Number of females 7-14 in the household	.968	1.08
Household sanitation			
good water	Equals 1 if household drinking water source is piped or protected, 0 otherwise	.198	.399
latrine	Equals 1 if household has a pit latrine, 0 otherwise	.956	.205
no toilet	Equals 1 if household has no toilet or latrine, 0 otherwise	.037	.189
Household assets			
coffee	Equals 1 if household grows coffee, 0 otherwise	.776	.417
good floor	Equals 1 if floor of dwelling is other than dirt or mud, 0 otherwise.	.169	.375
durables/adult	Value of durable goods per adult/100 Tshs	165	946
Nearest health services			
distance	Kilometers to the nearest health facility.	2.82	2.93
malnutrition	Equals 1 if nearest health facility offers child malnutrition services, 0 otherwise[c]	.547	.498
ors	Equals 1 if oral rehydration salts (used in the treatment of diarrhea) were in stock at the nearest health facility on the day of the interview, 0 otherwise[c]	.739	.439
Community health indicators			
measles 35	Proportion of children 0-35 months who have been vaccinated against measles in other households of the same cluster.	.695	.206
epidemic	Equals 1 if community respondents reported an epidemic since the last interview or in the last year (for first interview), 0 otherwise	.176	.381
aids	Equals 1 if community respondents named AIDS as the	.487	.500

	#1 cause of adult death, 0 otherwise		
urban	Equals 1 if urban community, 0 otherwise	.264	.441
adult mortality rate	Number of deaths of adults >14 per 1000 total population in the primary sampling unit enumerated in 1991	14.8	8.01
Community economic variables			
more rain	Equals 1 if more rain this year than same time last year, 0 otherwise	.328	.470
less rain	Equals 1 if less rain this year than same time last year, 0 otherwise [a]	.114	.318
market distance	Distance to the nearest periodic market, in kilometers	3.92	5.79
road impassable	Equals 1 if road to cluster is ever impassable during the year, 0 otherwise	.462	.499
child labor	Equals 1 if child wage cited for the cluster, 0 otherwise	.483	.500
child wage	Daily child wage in Tshs (set to zero if no child labor)	70.9	88.9
male labor	Equals 1 if adult male wage cited for the cluster, 0 otherwise	.914	.281
male wage	Daily adult male wage in Tshs (set to zero if no adult male labor)	262	358
price index	Cluster-level, time-varying price index.	1.20	.264
Adult deaths in the household			
adult death	Equals 1 if an adult 15-50 died in the household since the last passage or (for the first passage) in the past 6 months, 0 otherwise	.038	.192
adult death 0-3	Equals 1 if an adult 15-50 died in the household 0-3 months before the interview, 0 otherwise	.017	.130
adult death 4-6	Equals 1 if an adult 15-50 died in the household 4-6 months before the interview, 0 otherwise	.019	.138
adult death 7-9	Equals 1 if an adult 15-50 died in the household 7-9 months before the interview, 0 otherwise	.015	.120

a. Based on a sample of 1,085 children, with 2,641 observations.

b. Based on a sample of 1,076 children, with 2,619 observations.

c. The mean for all observations. For the three percent of child observations that could not be linked to the nearest health facility, the value has been set equal to zero. Among those observations that could be matched, the mean for the malnutrition variable is 0.564 and for the ORS variable is .762.

Household sanitation. Safe drinking water and sanitary waste disposal are thought to be important determinants of child morbidity, especially diarrhea (Tonglet et al 1992). Nearly a fifth of the children lived in households that had piped or protected drinking water sources—that is, inside taps, outside private or public standpipes, protected wells (with pumps), or water tanker trucks. Almost all of the children (95.6%) lived in households with pit latrines, 3.7 % lived in households with no toilet, and the balance (<1%) had flush toilets. Perhaps because of the lack of variation in waste disposal among the households, the toilet and latrine variables were never statistically significant in any of the regressions; they were subsequently dropped.

Household assets. We use measures of assets instead of a measure of permanent household

income, such as consumption expenditure, because the latter would directly include endogenous spending on health care and food. Coffee is the major cash crop in Kagera region, grown by the households of more than three-quarters of the children. Thus, coffee as a perennial tree crop not only signals an enduring asset (unlikely to vary much in the short run), it is also a proxy for the availability of cash income. The type of flooring of the household's dwelling is another indicator of wealth—17 percent of the children lived in dwellings with a concrete, wooden, tile, or parquet floor, while the rest lived in houses with mud or dirt floors.[9] Finally, we include as a third asset variable the value of the household's durable goods (radios, bicycles, TV sets, and so forth) per adult. Nearly half (48%) of the children were in households that reported zero durable goods; the mean value of durable goods per adult across all children was 16,500 Tshs, or $49. Among those with any assets the mean was 31,800 Tshs, or $94.

Health services. The KHDS conducted interviews at the nearest health facilities to each cluster of households, from which information on the availability of services could be obtained—in this case, whether the facility offered nutritional rehabilitation services and whether oral rehydration salts (ORS, for treating dehydration linked to diarrhea) were in stock the day of the interview. The distance between survey clusters and the nearest facilities was measured by the research team in the field. Child health should be negatively affected by greater distance to a health facility, but positively affected by the availability of these services.

Community health indicators. Measles is a prime killer of children in Subsaharan Africa. It can reduce appetite for several weeks and lead to severe protein-energy malnutrition (Waterlow 1992). Measles vaccination among Bangladeshi children under five has been credited for a reduction in child mortality of 36-45 percent (Koenig et al 1990, 1991). The immunization rate for measles was computed from among children under three years of age in households of the same cluster, excluding the household of the reference child. The variables measuring whether or not there was a recent epidemic in the community and whether AIDS is reported as the prime cause of adult deaths come from a community questionnaire. Nearly half of the children were living in communities where AIDS was cited as the major cause of adult deaths, and one-fifth were in urban areas. The community adult mortality rate (age 15 and older) assigned to each child is based on the results of a house-to-house enumeration conducted for sampling purposes in 1991 in each primary sampling unit. The overall adult mortality rate of roughly 15/1000 is about three times higher than we would have expected in the absence of AIDS, although it is somewhat inflated by the inclusion of people over 50. We cannot predict *a priori* the relation between

[9] Aside from being a measure of low wealth, a dirt floor may also affect young children's exposure to bacteria, parasites, and infectious agents.

urban residence and morbidity; it may capture improved access to and quality of health and other services in urban areas. However, sanitation problems can also be more extreme in poor urban areas, with a higher concentration of people, and urban rates of HIV infection are higher.

Community economic variables. The dummy variables for more or less rain than the same period last year, for whether the road is impassable, and for child and adult wages come from responses to the community questionnaire completed every passage in each of the 51 survey clusters. Market distances were measured by the field teams. The price index is based on a separate survey of market prices conducted contemporaneously with the household interviews. The questionnaire included prices for 26 food and 5 non-food items. These results were synthesized into a cluster-level, spatial and time-varying price index.[10]

Recent adult deaths in the household. We used two different specifications of adult deaths in the household. The first is simply a dichotomous variable for whether an adult died in the household in the past 6 months (for the first interview) or since the last passage (roughly 6-7 months) for subsequent interviews. Nearly 4 percent of the children in the pooled sample were in households with an adult death in this time frame. In a second specification, we control for the time since the adult death by including dichotomous variables for deaths occurring 0-3 months, 4-6 months, and 7-9 months before the interview. The comparison group is children in households with no adult death in the past 9 months. Note that recent adult deaths and orphanhood are measured by different variables. For children whose co-resident parents died during the survey, the total impact will be the sum of coefficients on orphanhood and recent adult deaths.[11]

Interactions between orphanhood, adult deaths, and other variables. Interactions between the adult mortality variables (recent adult deaths and maternal and paternal orphanhood) and household assets are included to measure the extent to which better off households are more able to cope with the impact of deaths on child health.[12] The adult mortality and orphanhood variables were also interacted with

[10] We are grateful to Mead Over and Indrani Gupta for generating the price index. Their methodology is described in KHDS Research Team (1999). Ideally, we would have liked to enter a prices of a few specific foodstuffs in the regression estimates. However, none of the food items was observed in every passage in every cluster, and many are not grown in parts of the region.

[11] Among children living in households with a recent death, 37 percent are orphans. Ninety percent of orphaned children live in households that did not have a recent adult death (in the past 6-7 months).

[12] In previous work using this same data set, we found that male and female adults with more schooling and who had occupations other than farming were more likely to die of AIDS and other causes (Ainsworth and Semali 1998). The positive correlation between HIV infection and socioeconomic status has been found in many other studies (see Table 1 of Ainsworth and Semali 1998, and World Bank 1997). This being the case, we were concerned that the

the health service and measles immunization variables to assess whether children in hard-hit households are more likely than others to benefit. Most of these interactions were not statistically significant; three that were significant are reported in the regression results. Finally, we have interacted mothers' schooling with the distance to a health facility to test the hypothesis that more schooled mothers can compensate for the non-availability of health care in the production of child health. The interaction variables are reported in the regression results but not in Table 1, to conserve space.

IV. Estimation results

We estimate the determinants of child health (equation 2) in a specification that takes into account individual heterogeneity and the potential correlation in error terms for the same child over time. The model estimated is:

$$(3) \qquad y_{it} = a + \beta' x_{it} + \gamma' z_i + u_i + e_{it}$$

where y_{it} is a measure of health outcome for child i in time t, α is a randomly distributed constant, x_{it} are the time-varying exogenous variables, z_i are time-invariant exogenous variables, u_i is an unobserved child-specific effect that is persistent through time, and e_{it} is a random error term. In the case of a continuous dependent variable (the two measures of nutritional status), if the unobserved child-specific effects (u_i) are randomly distributed among the cross-section of children, they are constant over time for the same individual, and are uncorrelated with the other explanatory variables, the parameters β and γ can be estimated consistently and efficiently using generalized least squares (GLS) in a random effects model (Greene 1993, Hsiao 1986). For the height-for-age and weight-for-height regressions, results of a test developed by Breush and Pagan confirmed the existence of a child-specific effect (not shown).

For the regressions on morbidity (a dichotomous variable), we use a probit model in which the standard errors have been corrected for the serial correlation across time periods for observations on the same individual. Ignoring the correlation among the errors would produce consistent but inefficient

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positive correlation between adult deaths and socioeconomic status could confound the interpretation of results on the impact of deaths on child health: high economic status would be related to better health outcomes but also more deaths, which could potentially produce the result that adult deaths show no effect on child health. However, in this sample of young children, the correlation between variables measuring deaths and household assets is weak and to the extent it exists, in the opposite direction. There is no statistically significant correlation between maternal orphanhood and the asset variables nor between a recent adult death and the asset variables. Paternal orphanhood is *negatively* correlated with the value of durable goods and the flooring variable, and *positively* correlated with coffee.

estimates. An alternative model is a random-effects probit estimated by maximum likelihood techniques, which is consistent and efficient for panel data (Hsiao 1986, Maddala 1987). However, the random effects probit coefficients are difficult to interpret. Guilkey and Murphy (1993) show that when the standard errors of the probit are suitably corrected for autocorrelation across individuals over time, the estimator performs almost as well as the maximum likelihood random effects probit estimator. The morbidity results were generated both ways and the results were very similar. For ease of interpretation, we report for morbidity the effect of a one-unit change in the explanatory variables on the probability of being ill (dp/dx) holding all other regressors at their means (STATA Corporation 1999). For dichotomous explanatory variables, the results show the difference in probability of illness when the variable is evaluated at 0 and at 1. The T-statistics are calculated on the corrected standard errors of the underlying probit coefficients.

The consistency of the GLS random effects parameter estimates depends critically on the assumption of non-correlation between the individual effect (u_i) and the regressors (x_{it}). When this is violated, the parameter estimates are inconsistent. With or without this correlation, fixed effects estimates are consistent. For height-for-age and weight-for-height, we report the results of a Hausman specification test that compares the random and fixed effects coefficient estimates: If the random effects model is correctly specified (i.e., no correlation between the individual effect and the regressors), there will be no systematic differences in the coefficients. In the case of height-for-age, this test statistic was highly significant, which indicates that the random effects specification was a mis-specified model. Unfortunately, all time-invariant variables are dropped from the fixed-effects model, eliminating many variables of policy interest. For this reason, we present both random and fixed effects results for height for age. Probit results for regressions of morbidity and random effects results for height for age and weight for height are reported in Table 2; fixed effects results for height for age are in Table 3. To save space, coefficients on the polynomials in the child's age are not presented; they were highly significant in all three child health regressions.

Morbidity

The impact of adult mortality on reported morbidity is critically linked with the household's wealth (Table 2, column 1). Because of the many interaction terms, the coefficients on the non-interacted variables for maternal orphans, paternal orphans, and adult deaths should be interpreted as the impact in the households with the fewest assets—no durable goods, no coffee, and a dirt floor. The poorest children who are paternal orphans or who live in households with a recent adult death are significantly more likely to be reported ill, but the negative signs on most of the death-asset interactions indicate that the impact is

less severe among households with greater wealth.[13] Likewise, a recent adult death in households with poor housing (a dirt floor) is associated with substantially higher morbidity, while in households with better flooring this impact is reduced.

Reported morbidity is strongly affected by community disease vectors. Children were more likely to be ill in communities where there had been an epidemic and where AIDS was cited as the major cause of adult death.[14] Higher market prices and greater distance to the nearest market also are associated with higher child morbidity. Countering this is a very strong result for a key policy variable—oral rehydration salts. Holding all other variables at their means, the probability of being ill was nearly 8 percentage points lower when ORS were in stock at the nearest health facility. The reduction in morbidity associated with availability of ORS is even greater in households with a recent adult death, but the interaction is not statistically significant. A protected source of drinking water for the household is also associated with lower morbidity (4 percentage points).

[13] A specification with dummy variables for the time since an adult death at 0-3, 4-6, and 7-9 months before the interview did not yield statistically significant results of a lagged impact of adult deaths on morbidity.

[14] A dummy variable for communities in which malaria is the major cause of child death was insignificant in the child morbidity regression.

Table 2: Regression results

Variable	Morbidity n = 1108 obs = 2679		Height/Age n = 1085 obs = 2641		Weight/Height n = 1076 obs =2619	
	dp/dx	*T*	*Coeff.*	*T*	*Coeff.*	*T*
Individual endowments:						
Girl	.027	(1.43)	.225	(3.14)	.012	(0.23)
Mother's schooling			-.021	(-1.08)	.022	(1.51)
Father's schooling			.046	(3.33)	-.004	(-0.33)
Mom dead	.022	(0.20)	-.840	(-2.20)	.326	(0.87)
Dad dead	.161	(2.38)	-.333	(-1.99)	.166	(0.99)
Orphan interacted w/assets:						
Mom dead*schooling			.118	(2.44)	$-.015_{-5}$	(-0.34)
Mom dead*durables	-.002	(-2.47)	$-7.5x10^{-6}$	(-0.57)	$-2x10^{-5}$	(-0.16)
Dad dead*durables	-.001	(-1.77)	-.0003	(-1.18)	.0003	(0.93)
Dad dead*coffee	-.136	(-2.19)	.047	(0.28)	-.118	(-0.67)
Dad dead*good floor	.297	(2.43)	.612	(1.99)	-.434	(-1.47)
Adult time:						
Female head	.046	(1.92)	.093	(1.21)	-.018	(-0.27)
#Female adults	.001	(0.18)	-.009	(-0.41)	-.0007	(-0.03)
#Female teens	-.018	(-1.91)	.025	(0.96)	-.003	(-0.11)
Sanitation:						
Good water	-.039	(-1.65)	.012	(0.21)	-.026	(-0.45)
Household assets:						
Grows coffee	.021	(0.74)	.048	(0.71)	.087	(1.27)
Good floor	-.067	(-2.29)	.023	(0.28)	.176	(2.30)
Durables/adult	$-2x10^{-5}$	(-1.86)	.0001	(2.98)	$4x10^{-5}$	(1.35)
Health services:						
Distance	-.003	(-0.86)	-.055	(-2.18)	-.012	(-0.65)
Distance*momdead	-.010	(-0.73)	-.086	(-1.88)	-.059	(-1.50)
Distance*adult death	-.031	(-2.12)	-.030	(-1.33)	-.023	(-0.88)
Mother's schooling*distance			.008	(1.87)	.002	(0.71)
Malnutrition services	-.014	(-0.63)	.022	(0.66)	.046	(1.18)
ORS in stock	-.066	(-2.85)	-.006	(-0.17)	-.064	(-1.46)
ORS*adult death	-.097	(-0.88)	.372	(1.92)	.024	(0.11)
Community health:						
Measles vaccine<35 mo	.030	(0.67)	.209	(2.79)	.101	(1.18)
Epidemic	.051	(2.10)	-.006	(-0.15)	.074	(1.67)
Aids#1 cause of adult death	.050	(2.32)	-.010	(-0.30)	.020	(0.49)
Urban	-.045	(-1.67)	.380	(3.92)	-.204	(-2.62)
Adult mortality rate	-.002	(-1.62)	.020	(3.87)	-.004	(-1.01)
Community economy:						
More rain	-.031	(-1.40)			.150	(3.54)
Less rain			-.081	(-1.74)		
Distance to market	.004	(2.83)	-.001	(-0.21)	.002	(0.36)
Road impassable	.002	(0.12)	-.056	(-0.70)	-.125	(-2.05)
Price index	.157	(3.63)	-.294	(-3.70)	.066	(0.66)
Recent adult deaths						
Adult death/6 months	.274	(1.94)	-.216	(-1.11)	.055	(0.25)
Adult death*floor	-.234	(-2.86)	-.376	(-2.01)	.317	(1.49)
R-square	.0746		.2650		.0910	
Hausman spec. test for RE (p)			.0000		.1540	
Joint tests						
Orphanhood w/o interactions	.0586		.0119		.4181	
Orphanhood w/asset interactions	.0046		.0275		.8631	
distance w/death interactions	.0631		.0083		.2761	
ORS w/interactions	.0059		.1593		.3460	
adult death, adult death*floor	.0037		.0384		.2760	

Notes: A T-statistic of 2.326 or larger indicates statistical significance at p<=.01, 1.960 at p<=.05, and 1.645 at p<=.10. All regressions controlled for the child's age in months in a quartic specification, which was highly significant in all cases. Also included but not significant in any of the regressions were momdead*coffee, momdead*goodfloor, male labor, male wage, child labor, and child wage. These are not shown to save space. In addition, the morbidity regression included a dummy variable control for the first interview and the weight for height regression included dummy variable for the third and fourth passages, to take into account a change in weighing equipment. All three regressions included a dummy variable for the roughly 3 percent of observations that could not be matched with the characteristics of the nearest health facility.

Reporting bias may be influencing these results, even though we've attempted to minimize one source of bias—recall error—by using illness on the day of the interview as a dependent variable. Reported illness among children who are maternal orphans is not statistically different from the morbidity of non-orphans and there's a significant *positive* association between female headship and morbidity. In the latter case, it may be that controlling for the number of female adults, if one of them is also the head, there may be less time available for child care. However, whether the respondent was the mother and, if not, whether the adult respondent was male or female, could affect the accuracy of reported illness. Female household heads may be more aware than male heads of child illness, thus more likely to report it. The person who responded for a maternal orphan is more likely to be a male and in any event cannot be the mother, leading to under-reporting or less accurate reporting for maternal orphans relative to other children (Belcher et al 1976). The results for distance to a health facility suggest that the probability of reporting an illness may be related to the proximity of health services: in households with a recent adult death, the more distant is health care the *less* likely are children to be reported ill. Maternal education (which was dropped in the final specification to conserve the sample size) does not have a statistically significant relation with reported morbidity, even when we interact maternal education and maternal survival. Sindelar and Thomas (1991) also found reported morbidity to be independent of mother's education in Peru, and suggest that this could be related to positive and negative reporting biases associated with different types of health problem among educated mothers.

Height for age

While reporting errors for morbidity may be correlated with the characteristics of the respondents or availability of health care, complicating the interpretation of results, this problem does not arise for the anthropometric measurements made by the interviewers. The coefficients for height for age (Table 2, column 2) can be interpreted as the marginal effect of a one-unit increase in the explanatory variable on the height-for-age z-score, holding other explanatory variables at their means.

Both parents' survival and recent adult deaths are associated with large deficits in height for age among children in the poorest households—a deficit of nearly one standard deviation for maternal orphans (-.84) and a third of a standard deviation for paternal orphans (-.33). The impact of maternal orphanhood is equally severe regardless of the household's assets, while the impact for paternal orphans is felt only among those in poorer households. A recent adult death is associated with lower height for age in the

poorest households (with a dirt floor) and—surprisingly—the effect is even *greater* in households with better housing.

Orphans whose mothers were unschooled are the hardest-hit in terms of reduced height for age. Mother's schooling has no relation with height for age among children who are not orphaned, but among children who are, every year of schooling of the deceased mother reduces the impact of maternal orphanhood by 0.12 SD.[15] Thus, maternal orphans whose mothers had 8 years of schooling have similar height-for-age to non-orphans. Since the mothers are not alive, mother's education in this case is probably not reflecting improved technical efficiency in producing child health. More likely, it is correlated with other aspects of the child's endowment that we have been unable to control for or the quality of child care before the mother's death.

The availability of health inputs and community disease vectors show strong effects on height for age. The farther away is the nearest health facility, the lower is height, and the negative impact of distance on height for age is even greater for maternal orphans and children in households with recent adult deaths. However, maternal education and availability of health care are substitutes—the more educated was or is the child's mother, the less important is the distance to a health facility in determining height.[16] The availability of ORS at the nearest health facility has a positive relation with height, but only for children in households with a recent death. Height for age is considerably higher in communities with high measles vaccination coverage, but it does not seem to be affected by epidemics and adult AIDS deaths in the community. Curiously, the higher is the community adult death rate, the taller are children. Since adult deaths are associated with higher socioeconomic status in this region, the adult death rate may simply be proxying community wealth. Both drought and high market prices are associated with lower height for age, as expected.

The highly significant Hausman test statistic at the bottom of Table 2 for height for age indicates that the random effects model is misspecified—possibly due to correlation between the individual effect and the regressors. The estimates are therefore inconsistent. Table 3 presents the estimation results for the fixed effects model, which automatically drops time-invariant variables like the child's sex, his/her parents' schooling, and the distance to a health facility. In the fixed effects estimates, only four of the

[15] While much of the early literature found a positive relation between mother's education and child anthropometric status, subsequent studies that have controlled for household income, assets and/or mother's height have often found weak or nonexistent effects (Alderman 1990, Handa 1999, Sahn 1990, Strauss 1988).

remaining time-varying variables remain statistically significant: the positive relation between measles vaccination coverage and height for age; the positive relation between the availability of ORS and height in households with a recent adult death; and the negative interactions between paternal death and durable goods and between adult death and flooring.

Can we conclude that orphanhood has no effect on height for age when the coefficients are estimated consistently? It isn't clear. Since this model "differences" observations from two periods in time, in the fixed effects model the coefficients on the orphan variables should be interpreted as the effect of *becoming* an orphan. However, very few of these children became orphans during the survey. Furthermore, we expect that the impact of being an orphan on a measure of chronic health like height for age will be seen only over the longer term. For children who were already orphans (who didn't lose their parents since the last passage), orphanhood is a time-invariant variable and is dropped from the fixed effects model. So although the random effects coefficients on the orphan variables are most likely inconsistent, it isn't possible to conclude anything about the enduring effect of being an orphan on height for age from a fixed-effect model.

Finally, a different specification of recent adult deaths in the household finds evidence that the impact of adult deaths on height for age rises over time, and both the random- and fixed-effects models are in agreement (see Table 4). There is no significant relation between height and an adult death within 3 months of the death. This is as might be expected for a health measure that reflects the cumulative impact of health problems over the longer term. However, by 4-6 months following an adult death children in these households have height-for-age z-scores nearly 0.4 SD lower than children in households without a death. This effect is somewhat diminished 7-9 months after the adult death, but still substantial—a 0.25 SD deficit in height compared to children in households without a death.

[16] Thomas et al (1991) also found that mother's education and health services were substitutes, in Brazil.

Table 3: Fixed effects regression results, height for age

n=1,085 children, 2,641 observations

Variable	***Coefficient***	*T-statistic*
Individual endowments:		
Mom dead	-.298	(-0.53)
Dad dead	-.050	(-0.24)
Interactions with assets:		
Mom dead*schooling	-.063	(-0.79)
Mom dead*durables	.0002	(1.38)
Dad dead*durables	-.0006	(-2.00)
Dad dead*coffee	.156	(0.76)
Dad dead*good floor	.489	(1.22)
Household time:		
Female head	-.028	(-0.23)
#Female adults	.014	(0.47)
#Female teens	.039	(1.04)
Household assets:		
Durables/adult	$1.4x10^{-5}$	(0.25)
Health services:		
Malnutrition services	.011	(0.30)
ORS in stock	.020	(0.50)
Adult death*ORS	.439	(2.16)
Adult death*distance to nearest facility	-.035	(-1.52)
Mom dead*distance to nearest facility	.046	(0.44)
Community health:		
Measles vaccine<35 mo	.161	(2.03)
Epidemic	.003	(0.08)
Aids#1 cause of adult death	-.003	(-0.07)
Community economy:		
Less rain	-.039	(-0.78)
Price index	-.055	(-0.48)
Recent adult deaths:		
Adult death/6 months	-.289	(-1.42)
Adult death*floor	-.449	(-2.26)
R-square	.1530	
***Joint tests*:**		
Orphanhood w/o interactions	.8502	
Orphanhood w/ asset interactions	.3904	
Momdead*distance, adult dth*distance	.2992	
Adult death, adult death*ORS	.0757	
Adult death, adult death*floor	.0126	

Table 4: Random and fixed effects coefficients for time since adult death, height for age regression

	Random effects		**Fixed effects**	
Variable	**Coefficient**	**T-statistic**	**Coefficient**	**T-statistic**
Adult death last 0-3 months	-.193	(-0.93)	-.291	(-1.28)
Adult death last 4-6 months	-.362	(-2.11)	-.394	(-2.19)
Adult death last 7-9 months	-.254	(-1.96)	-.263	(-1.90)
R-square	.2658		.1559	
Hausman test for RE (pr)	.0000			
Joint test of adult deaths, 0-9 months	.0410		.0547	

Note: Regression included all other variables in Tables 2 and 3.

Weight for height

The results of the Hausman specification test in Table 2 (column 3) indicate that the random effects model is both efficient and consistent for weight for height. However, there are very few statistically significant determinants of weight for height (other than the polynomial in the child's age, which is not shown). Neither orphanhood nor recent adult deaths have any statistically significant relation with weight for height.[17] Mother's schooling is correlated with higher weight for height, but not at conventional levels of statistical significance. Better housing—having a concrete, tiled, or wooden floor, compared to a dirt floor—is the only household-level socioeconomic variable with strongly significant positive relation to weight for height. Children in households with concrete, wood, or tile floors have weight-for-height z-scores 0.2 SD higher than children in households with dirt floors. We have been using this variable as a proxy for wealth, but it is important to keep in mind that a dirt floor is also likely to raise the likelihood that toddlers and young children ingest dirt. This could explain equally the strong results for flooring in both the morbidity and weight-for-height results.

In terms of community and health service variables, children in communities with more rainfall than the previous year have higher weight for height, and those in urban areas or in locations that are cut-off from transport at certain times of the year have lower weight for height. The negative effect of urban residence may seem counter-intuitive, since urban children had *lower*, not higher morbidity. However, Sahn (1990) also found that low weight for height was more prevalent in Abidjan than in other cities and in rural areas of Côte d'Ivoire. Alderman (1990) found a similar relationship in Ghana when income was not controlled for. The characteristics of the health services show no significant relation with wasting. It is surprising that a recent epidemic is associated with better weight for height—even though it was also associated with higher morbidity.

Results for poor and non-poor households

The results for the determinants of morbidity and height for age show that the impact of adult mortality on child health is critically linked to the household's wealth. In Table 5, we present results for morbidity and height for age for children in 'poor' and 'non-poor' households.[18] Using data from the first time that each child was observed, we divided the observations according to the value of the total assets of

[17] The specification with time elapsed since an adult death was also statistically insignificant.

[18] In the previous section, we predicted levels of health for households with different levels of assets using a common set of regression coefficients. Here we allow the coefficients to vary according to whether the sample is from the top or bottom of the distribution of assets in the first interview. None of the adult mortality or policy

the child's household—including physical and financial assets and durable goods. The children whose households fell below the median are labeled as "poor" and those above the median as "nonpoor". All variables from Table 2 are included in the regressions, but the interactions between assets and orphanhood have been suppressed to conserve space.

Morbidity. The probability of being ill in poor households was 31 percent, compared to 27 percent among the nonpoor. Among children in poor households, orphanhood is not a good predictor of heightened morbidity. The statistically significant effect of paternal orphanhood in raising morbidity in the nonpoor households underscores the point that poor orphans are those most highly affected: Holding all explanatory variables at their means, paternal orphans in nonpoor households (and with no durable goods, no coffee, and a dirt floor)[19] are 32 percentage points more likely to be reported ill than non-orphans in the same low-asset households. Basically, this is reflecting the fact that some children in households with low assets in subsequent passages were assigned into the nonpoor group based on the their assets during the first interview. So it is again making the point that poor orphans have higher morbidity. When we take into consideration the asset interactions and predict morbidity using the values of all explanatory variables, a recent adult death is associated with similar, higher predicted morbidity in both poor and nonpoor households (.347 and .342, respectively). Safer water supply is associated with lower morbidity only in nonpoor households. ORS availability at the nearest health facility is significantly associated with lower morbidity in poor households and the impact of ORS in poor households with a recent adult death is several times greater (the sum of the two marginal effects). Greater distance to a health facility is associated with lower reported morbidity among poor households and among non-poor households with a recent adult death, likely a result of the reporting biases discussed earlier.

Height for age. Children in poor households have lower height for age (mean z-score of -1.78, 42.6% stunted) than do children in nonpoor households (mean z-score of -1.53, 34.2% stunted). The impact of orphanhood on height for age is greatest for children in poor households. Holding the values of all other variables at their means, in maternal orphans in poor households (and with no durable goods, no

variables were statistically significant for weight/height when the samples are divided into poor and non-poor.

[19] Recall that because of the multiple interaction terms with assets in these regressions, the coefficient on the orphan variables is interpreted as the relation for orphans in households with a dirt floor and no durable goods.

Table 5: Regression results for children in poor and nonpoor households

	Morbidity				*Height/Age*			
	Poor obs=1340		**Nonpoor obs=1339**		**Poor Obs=1323**		***Nonpoor* obs=1318**	
Variable	***dp/dx***	***T***	***dp/dx***	***T***	***Coeff.***	***T***	***Coeff.***	***T***
Individual endowments:								
Girl	.058	(2.10)	.003	(0.13)	.227	(2.16)	.242	(2.48)
Mother's schooling					-.034	(-1.24)	-.002	(-.08)
Father's schooling					.025	(1.32)	.070	(3.43)
Mom dead	-.032	(-0.26)	.032	(0.19)	-1.18	(-2.06)	-.625	(-1.17)
Dad dead	.086	(1.10)	.322	(1.99)	-.442	(-2.05)	-.371	(-1.20)
Mom dead*schooling					.145	(2.21)	.222	(2.16)
Adult time:								
Female head	.032	(0.94)	.099	(2.65)	-.037	(-0.33)	.203	(1.84)
#Female adults	-.021	(-1.42)	.016	(1.60)	.055	(1.33)	-.042	(-1.68)
#Female teens	-.020	(-1.34)	-.005	(-0.44)	-.055	(-1.23)	.071	(2.25)
Sanitation:								
Good water	-.001	(-0.03)	-.090	(-2.88)	.021	(0.25)	.004	(0.06)
Household assets:								
Grows coffee	-.039	(-0.89)	.112	(2.74)	-.055	(-0.59)	.163	(1.53)
Good floor	-.163	(-2.71)	-.033	(-1.01)	-.241	(-1.46)	.128	(1.41)
Durables/adult	$-1.3x10^{-5}$	(-0.21)	$-1.4x10^{-5}$	(-1.07)	.0001	(0.81)	$9x10^{-5}$	(2.69)
Health services:								
Distance	-.009	(-1.55)	.003	(0.57)	-.094	(-2.84)	.033	(0.81)
Distance*momdead	.009	(0.50)	-.013	(-0.57)	-.089	(-1.35)	-.212	(-2.26)
Distance*adult death	.005	(0.22)	-.045	(-2.16)	-.076	(-2.06)	.001	(0.05)
Mother's schooling*distance					.011	(1.74)	-.002	(-.24)
Malnutrition services	-.045	(-1.41)	.014	(0.47)	.062	(1.22)	-.013	(-0.28)
ORS in stock	-.096	(-2.83)	-.037	(-1.19)	-.051	(-0.92)	.051	(.98)
ORS*adult death	-.223	(-1.47)	.015	(0.09)	.367	(1.00)	.468	(2.00)
Community health:								
Measles vaccine<35 mo	-.023	(-0.35)	.075	(1.21)	.348	(3.19)	.063	(0.60)
Epidemic	.076	(2.07)	.022	(0.66)	-.008	(-0.14)	-.020	(-0.42)
Aids#1 cause of adult death	.070	(2.13)	.051	(1.81)	.021	(0.39)	-.047	(-0.97)
Urban	.069	(1.30)	-.103	(-3.09)	.442	(2.56)	.411	(3.44)
Adult mortality rate	-.002	(-0.94)	-.002	(-1.16)	.036	(4.28)	.009	(1.31)
Community economy:								
More rain	.0004	(0.01)	-.044	(-1.51)				
Less rain					-.084	(-1.27)	-.070	(-1.04)
Distance to market	.001	(0.56)	.006	(3.48)	.011	(1.18)	-.010	(-1.18)
Road impassable	.049	(1.62)	-.046	(-1.73)	-.067	(-0.58)	-.067	(-0.62)
Price index	.123	(1.82)	.213	(3.79)	-.424	(-3.27)	-.173	(-1.71)
Recent adult deaths:								
Adult death/6 months	.240	(1.14)	.290	(1.36)	.014	(0.04)	-.455	(-1.88)
Adult death*floor			-.222	(-3.18)	.007	(0.01)	-.402	(-2.01)
R-square	.0738		.1212		.3097		.2738	
Hausman test for RE (p)					.5383		.0000	
Joint tests								
Momdead, daddead	.5137		.1336		.0131		.2475	
Orphanhood w/asset int'ns	.3434		.0005		.0043		.1968	
Distance w/death interactions	.4911		.1538		.0009		.1288	
ORS, adult death*ORS	.0026		.4903		.4345		.0649	
adult death, adult death*floor			.0031		.9999		.0066	

Notes: See the notes for Table 2. Results for some variables are not shown to save space. Momdead*floor and adult death*floor had to be dropped from the morbidity regression for the poor, and momdead*coffee in that for the nonpoor due to collinearity problems.

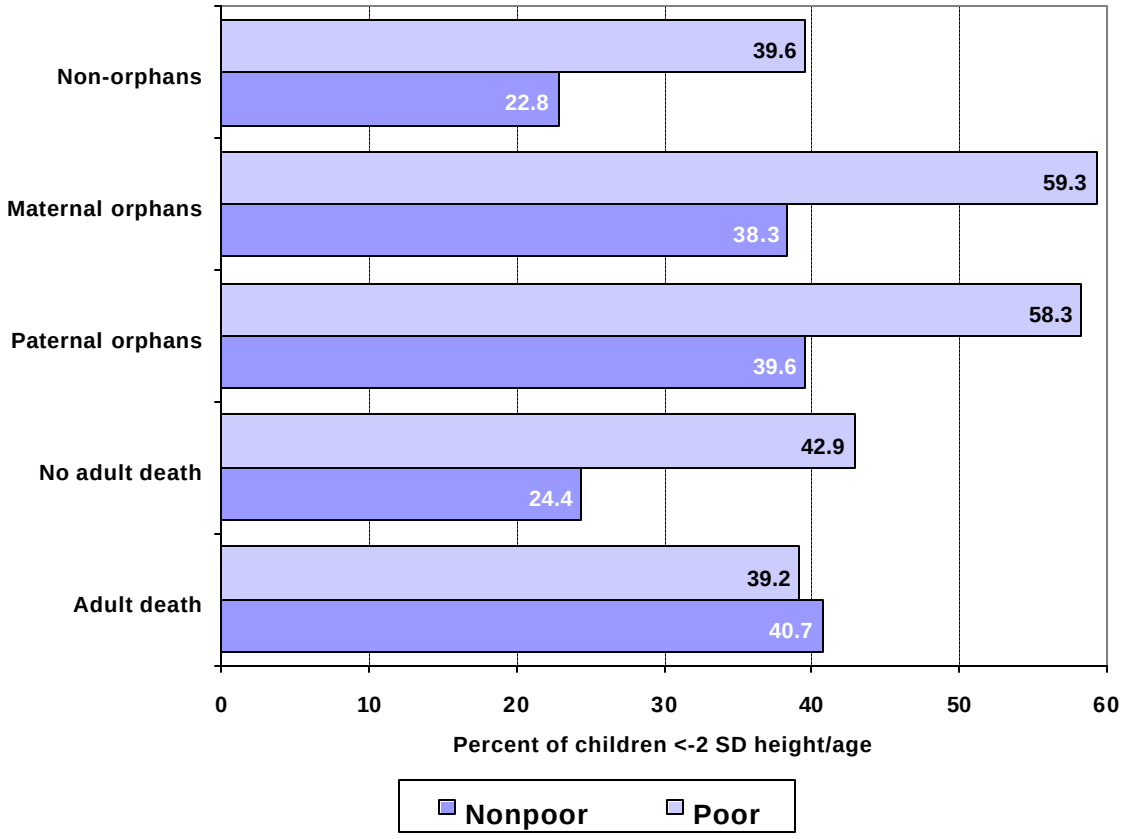
coffee, and a dirt floor) have dramatically lower height for age (1.18 SD lower) than children in the same type of households with living mothers, while paternal orphans also show large negative effects (0.4 SD

lower). Taking into account the various interaction terms between orphanhood and assets, predicted stunting among poor maternal and paternal orphans is 59.3% and 58.3%, respectively, compared to a rate of 39.6% among poor non-orphans (Figure 1). Among children in non-poor households, the effects of orphanhood are also negative but smaller and not statistically significant. In both poor and nonpoor households, mother's schooling offsets the negative impact of maternal orphanhood on height for age at .145 SD and .222 SD, respectively, for every year of schooling. However there is no direct impact of mother's schooling for children whose mothers are alive. Father's schooling is associated with higher height for age only in nonpoor households. Recent adult deaths affect height for age only in nonpoor households, and the impact is *greater* in households with better flooring.

The availability of health care is a key policy variable for height for age, particularly in the poorest households: every extra kilometer to the nearest health facility is associated with a decrease of .09 in height-for-age z-score; the effect is roughly doubled for poor maternal orphans and for poor households with a recent adult death. Among nonpoor households, only maternal orphans suffer from lower height as the distance to a health facility increases, but the effect is quite large (-.2 SD per km). Mother's schooling is a substitute for the availability of health care in poor but not nonpoor households. ORS availability at the nearest health facility has a large positive impact (.5 SD) on the height for age of children in nonpoor households, while the measles immunization rate has a large positive impact (.35 SD) on the height of the nonpoor. [20] The Hausman specification test for random effects passes for the subsample of children from poor households, but not among the nonpoor.

[20] Thomas et al (1996) found similar significant effects of the availability of measles vaccine on the height for age of poor children in Côte d'Ivoire.

Fig. 1: Predicted stunting among children under five by household assets



V. Summary and policy implications

At the beginning of this paper we posed two questions. First, what will be the impact of the death of adults and parents on the health of young children in areas hard-hit by the AIDS epidemic? Child health will worsen through two channels—via vertical HIV transmission from mother to child and via the economic impact of adult deaths on both infected and uninfected children. Using data from Kagera region of Tanzania and controlling for individual characteristics, household and community economic variables, we've found that adult deaths (recent adult deaths and paternal orphanhood) have an independent relation with two measures of child health--reported morbidity and height for age. Paternal orphanhood and other adult deaths will raise reported morbidity; no relation was found between maternal orphanhood and

reported morbidity. However, because the morbidity variable is "reported" rather than an objective measure, the impact of maternal orphanhood could be understated and that of paternal orphanhood overstated if mothers are most knowledgeable about their children's health and if women are better informed than men and thus more likely to report a child's illness. On the other hand, height for age is an objective measure that is not subject to reporting bias. Here the results were very clear. Both the loss of either parent and the deaths of other adults in the household will worsen height for age and raise stunting of children. Controlling for recent deaths, both maternal and paternal orphans are substantially more likely to be short for their age: the loss of a parent raises stunting among the nonpoor to levels found among poor children with living parents; among the poor, orphanhood raises stunting even higher. Children in the poorest households, children whose parents are/were uneducated, and those with the least access to health care are the most severely affected. The impact of adult deaths on reducing height for age is delayed, not appearing until 4-6 months after the death. There are signs that the effect persists but is reduced over a slightly longer term (6-9 months). Adult deaths and loss of a parent have no apparent relation to weight for height.

What are the policy implications of these results? First, the results have important implications for the targeting of public health interventions to improve child health. Like many of the areas most affected by AIDS in Subsaharan Africa, the Kagera region of Tanzania has high levels of poverty and low levels of child health. Targeting health interventions solely to orphans or children in households with a recent adult death would miss many children with equally severe health problems because of widespread poverty and would benefit some children in households whose resources are adequate to cope without external help. Rather, to the extent possible, interventions should be targeted to the poorest households, among which the households hardest hit by adult mortality are most likely to be found. The recommended targeting strategy for middle income countries with severe AIDS epidemics, like Thailand, or countries with lower poverty and better overall child health indicators might be quite different, although this remains to be demonstrated.

Second, despite the fact that an unknown amount of child morbidity in this sample is related to HIV infection, we have identified several public policies that will mitigate the impact of higher adult mortality on child health. Most of the policy variables, even when applied evenly across the population, will have greater impact on children in poor households than in nonpoor households and greater impact on children in households with higher mortality. For example, universal availability of ORS at health facilities would lower morbidity among children in poor households by about 8 percent (from .311 to .290), but the impact on children in poor households with recent adult deaths would be even greater—a 32 percent reduction (from .347 to .235). In contrast, it will have no effect on the morbidity or height for age for

children in nonpoor households, except for children living in households with a recent adult death. For the latter, availability of ORS would raise the mean height for age z-score from –1.76 to –1.62 and reduce stunting by 10 percent (from 38.3% to 34.5%). Making ORS more widely available at public health facilities will thus disproportionately help the poor and most severely affected children and, to the extent that public services are more likely to be used by the poor, are "self-targeted".

Measles immunization and improved physical access to medical care have similar effects on the poorest children and on children most affected by adult mortality. The predicted z-scores for poor and nonpoor children are –1.80 and –1.57, respectively, with stunting rates of 42.9% and 25.0%, respectively. Raising measles immunization coverage to 100% would raise the mean z-score among poor children to –1.69 and lower stunting by 18 percent (to 35.1%), while having no discernable effect on the height for age of nonpoor children. Reducing the distance to medical care to 5 km for those who live farther away would raise the height for age z-score of the poorest children to –1.76 and reduce stunting by 5 percent (to 40.8%). It would have an even greater effect on poor children in households with an adult death, raising the z-score from –1.75 to –1.66 and lowering stunting by 9 percent, from 39.2% to 35.7%. Among poor maternal orphans, z-scores would increase from –2.89 to –2.79, and stunting would decline by 5 percent, from 59.3% to 56.2%. Among the nonpoor, improved access to medical care would raise z-scores from –1.68 to –1.60 and reduce stunting by 10 percent (from 38.3% to 34.5%), while having no impact on other nonpoor children.

There was no significant relation between the availability of malnutrition services and any of the indicators of child health, but this variable was not very precisely measured on the health facility questionnaire. We don't know exactly what specific services were provided and available on the day of the interview. Interpretation of the results for malnutrition services (available to slightly more than half of the children) may also be confounded by endogeneity of program placement. That is, nutrition rehabilitation may be made available to health facilities in areas with the largest malnutrition problems, creating a positive correlation between the availability of services and negative health outcomes, even if these services are effective. The other three program variables are less likely to suffer from this problem.

Finally, reported child morbidity was significantly higher in communities where AIDS was reported to be the primary cause of adult death, even while controlling for the adult death rate and reported epidemics. The relative impact and costs of HIV prevention (among mothers and prevention of mother-to-child transmission) compared to these other more conventional interventions (ORS, measles

immunization, and availability of health care) for improving aggregate child health is not known. The results of this study are gleaned from a longitudinal household survey in which the services have been linked to outcomes, while controlling for individual, household, and community characteristics. More precise measures of impact could be obtained from a controlled field trial of these different interventions in the same setting. Such a study would be very worthwhile in setting priorities for public policy on child health in the most severely affected countries.

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