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Adult Mortality and Consumption Growth in the Age of HIV/AIDS[*]

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Abstract

This paper uses a 13-year panel of individuals in Tanzania to assess how adult mortality shocks affect both short and long-run consumption growth of surviving household members. Using unique data which tracks individuals from 1991 to 2004, we examine consumption growth, controlling for a set of initial community, household and individual characteristics; the effect is identified using the sample of households in 2004 which grew out of baseline households. We find robust evidence that an affected household will see consumption drop 7 percent within the first five years after the adult death. With high growth in the sample over this time period, this creates a 19 percentage point growth gap with the average household. There is some evidence of persistent effects of these shocks for up to 13 years, but these effects are imprecisely estimated and not significantly different from zero. The impact of female adult death is found to be particularly severe.

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Introduction

While there are other more prevalent diseases in Africa, the characteristics of HIV/AIDS suggest that its economic and demographic impact will be profound. In the absence of the AIDS epidemic, prime-age deaths would be relatively rare in Africa. Because HIV in Africa is transmitted primarily through heterosexual contact, the epidemic is having a dramatic impact on the mortality of men and women in their prime childbearing and earning years; consequently, the mortality rates of adults 15–50 increase dramatically in areas affected by the epidemic (Ngom and Clark, 2003). Moreover, HIV/AIDS is not restricted to poorer populations. Unlike other major diseases in Africa, HIV/AIDS is prevalent among the better educated and higher income Africans in urban areas, with mixed evidence about the correlation between socio-economic status and HIV. For example, earlier evidence (World Bank, 1999) indicated higher rates among better education/higher income groups, whereas more recent population survey data analyzed by De Walque (2006) does not indicate a correlation between education level and HIV status.

The implications of the epidemic, increased rates of severe illness and prime-age adult mortality, suggests that the disease will have consequences for a host of socio-economic indicators. This paper adds to the literature by addressing an empirical question which to-date has been under-explored: the long-run implications on living standards of adult mortality shocks on surviving family members. The study focuses on the Kagera Region in the North-West of Tanzania, an area deeply affected by HIV/AIDS.

Research on the socioeconomic impact of AIDS is wide and varied, and can be partitioned into estimates of macroeconomic costs and analysis of household (microeconomic) impacts. Empirical analysis of the economic impact of high prime-age adult mortality due to HIV/AIDS and other fatal illnesses at the macroeconomic level draws mixed conclusions. Early

examples of macroeconomic studies in this area include Arndt and Lewis (2000), Bloom and Mahal (1997), Cuddington (1993a), Cuddington (1993b), Cuddington and Hancock (1994), Over (1992), and Quattek (2000). These macroeconomic studies have been critiqued for underestimating the impact of HIV/AIDS because they do not take into account the potential impact on human capital formation and its transmission between generations. Bell, Devarajan and Gersbach (2006), McDonald and Roberts (2006) and Corrigan, Gloom and Mendez (2006) extend these models in varies ways to include the intergenerational effects on human capital accumulation and find large effects on national income per capita. On the other hand, Young (2005) models this orphan effect and finds that the reduction in fertility dominates and results in *higher* per capita consumption possibilities.

The underpinnings of the macroeconomic studies are the behaviours and outcomes for individuals affected by HIV/AIDS: individuals who are infected as well as people with socio-economic ties to infected persons. There are several pathways through which an adult mortality can affect consumption or income levels among surviving household members. The direct costs of these events include medical expenses as well as funeral costs (which can be larger than the medical costs in areas with low health care provision). Illnesses are associated with the loss of earnings for both the sick household members and care-givers in the household. Un-earned income for households may also suffer if remittances are curtailed due to illnesses and mortality. Deaths can result in asset losses due to disinheritance (for example, land grabbing). Finally, there may be significant intergenerational effects if illness and mortalities result in lower health and education investments in children (say due to costs of illness and liquidity constraints), compromising the future income of these children when they reach adulthood. On the other hand, the *per capita* income standing of surviving households could theoretically *rise* through increases

in labor scarcity which increase the value of value time. This factor explains the results in the simulations by Young (2005) where the AIDS epidemic results in *higher* per capita consumption in the South African economy.

While there is increasing evidence of short-run income impacts on households due to shocks such as large-scale fiscal crises (see works cited in Fallon and Lucas, 2002), morbidity shocks (Kochar, 1995), and weather variation (Paxson, 1992; Kinsey, Burger and Gunning, 1998), there are fewer studies on the income effects of deaths of household members. Grimm (2006) examines the consumption of survivors' after the death of a household member in Indonesia, including deaths of children and elderly household members. In this setting, given low prevalence levels, deaths are unlikely to be primarily due to HIV/AIDS. Christiaensen, Hoffmann and Sarris (2006) and Chapoto and Jayne (2006) study the relatively short-run impact of deaths on consumption levels (for deaths in the last 2 years in Tanzania and Zambia, respectively), while Yamano and Jayne (2004) study the impact of adult deaths on activities and particular sources of income. Consumption losses from health shocks can extend beyond the short-run impacts as shown in the study of panel data from 1999-2004 from Ethiopia in Dercon, Hoddinott and Woldehanna (2005).

In their recent review of household studies of the impact of HIV/AIDS morbidity and mortality on household income and expenditure, Naidu and Harris (2005) emphasize the gaps in this literature including comprehensive measures of household expenditure (or income) and longitudinal data with which to track impacts over time. The lack of quantitative studies of the impact of an adult death at the household or individual level perhaps stems in large part from the difficulty in collecting data with the appropriate information to analyze the impact of adult mortality within households. To this end, this project follows on the original panel data collected

in the Kagera region of Tanzania (Kagera Health and Development Survey – KHDS) originally designed to assess the short-run impact of an adult death.

Studies of the baseline KHDS panel found that many households seem to recover from the shock in terms of consumption expenditure (see World Bank, 1999, Chapter 4). Lundberg, over and Mujinja (2000) study the various coping strategies that household employ, including private transfers, credit, and public assistance. As this is based on an 18-month panel and up to 2 years after the event, it is unclear if these outcomes reflect the long-term impacts. If households have short-run coping strategies (such as selling off assets, borrowing, increased remittances from relatives) that are not sustainable in the long-run, the true impact of these shocks may be quite different from the short-run outcomes. Long-run impacts could evolve if this shock results in changes in income or asset strategies, such as reluctance to engage in high-risk/high-return activities, holding more liquid and less productive assets, or lower investment due to lower access to credit.

This paper uses a 13-year panel of individuals to assess how adult mortality shocks affect both short and long run consumption growth of surviving household members. For a sample of households interviewed in 1991, we traced these members in 2004, including those who had relocated or formed their own household. For each baseline household member that died a special mortality questionnaire was administered, recording the year the person died, the reasons for their death, as well as who of the surviving baseline household members they were living with at the time of their death. Our analysis will focus on the effect of the death of these baseline household members on the households they were living in at the time of their death. We do this by regressing consumption growth between 1991 and 2004 on an indicator variable for whether any previous household member died while residing in the household. The nature of our data

allows us to control for baseline household fixed effects, offering identification of the effects of adult mortality via the different split-off households stemming from the same initial household. By thus exploiting the variation in outcomes across households whose members were all living together at baseline not at the time of the adult mortality event, we control more fully for heterogeneity in initial socio-economic conditions that may be correlated with subsequent adult mortality and the path of consumption growth. We also control for the characteristics of the 2004 households including household age fixed effects to account for the strong life-cycle patterns one may expect to observe in consumption.

The following pattern emerges: an affected household will see consumption drop by 7% within the first five years after the adult death. Meanwhile, as this is a period of rather high consumption growth, the average household continues to grow, at a rate that can be estimated to be about 13 percent over the same five year period, creating a 19 percentage point growth gap. For deaths that occurred 6-13 years ago, we consistently find negative but insignificant effects. This may be suggestive of no persistence in the impact of adult death and a full recovery. However it may well be that other events blur the difference between affected and non-affected households. A 'casual observer', and even our relatively detailed data set, may then only imprecisely measure differences in consumption between them, but a persistent effect, at least up to 13 years, seems plausible if unproven given our regression results. Further testing fails to find any evidence of risk-sharing for adult mortality shocks among the 2004 households which originated from the sample of initial households.

Setting and Data

The data for this study are from the Kagera Region, an area far from the capital and coast, bordering Lake Victoria, Rwanda, Burundi and Uganda. It is overwhelmingly rural and primarily engaged in producing bananas and coffee in the north and rain-fed annual crops (maize, sorghum, cotton) in the south. Relatively low-quality coffee exports and agricultural produce are its main source of income. It is not one the poorest areas of Tanzania, with mean per capita consumption near the mean of mainland Tanzania in 2000. Growth and poverty reduction appears to mirror the rest of Tanzania: real GDP growth was just over 4 percent per year between 1994 and 2004, while poverty in Kagera is estimated to have changed very slightly, falling from 31 percent to 29 percent between 1991 and 2000/01 using the national data.

It is nevertheless an area of early and high HIV-prevalence. Kwesigabo *et al*. (2005) reported on three population samples in 1987 in districts of contrasting exposure in Kagera, finding in 1987 overall age-adjusted HIV-prevalence in urban Bukoba district of 24.2%, of 10.0% in Muleba district, a medium-prevalence area, and of 4.5% in Karagwe district, a low-prevalence area. Subsequently, this baseline sample was followed further, and prevalence rates appear to have been coming down, both due to mortality and lower incidence. In urban Bukoba, prevalence went down to 18.2% in 1993 and 13.3% in 1996. In the other areas studied, prevalence also declined considerably, to 4.3% and 2.6% in Muleba and Karagwe respectively. Kwesigabo *et al*. (2005) note that the decline in these areas of different initial HIV-exposure suggests that the epidemic may have been arrested early without necessarily peaking to saturation levels. Nevertheless, and relevant for our study, a rapid decline in prevalence, even without any new incidence of HIV, is only possible due to large mortality in this period.

The Kagera Health and Development Survey (KHDS) was originally conducted by the World Bank and Muhimbili University College of Health Sciences (MUCHS), and consisted of 915 households interviewed up to four times from fall 1991 to January 1994 (at 6-7 month intervals). In addition to the household survey, the KHDS included surveys of communities, prices and facilities.[1] The KHDS 1991-1994 serves as the baseline data for this paper. The household questionnaire was a Living Standards Measurement Study survey instrument which contained numerous indicators of well-being, such as consumption, expenditure, asset holdings, morbidity, health, nutrition, and education. Even though the sample was not specifically designed to be self-weighting, a comparison with the 1991 Household Budget Survey suggests that in terms of basic welfare and other indicators, the baseline data are similar to a representative sample for this period from the Kagera region.

The KHDS 2004 was conducted in the first half of 2004 (Beegle, De Weerdt and Dercon, 2006a). The objective of the KHDS 2004 survey was to reinterview all individuals who were household members in any round of the KHDS 1991-1994 and who were alive at the last interview. This effectively meant turning the original household survey into an individual longitudinal survey. When a panel respondent was located, the household in which these individuals lived in 2004 was administered the full household questionnaire. The KHDS 2004 used the original questionnaire as the foundation of the survey instrument, to ensure comparability of all main indicators and variables from the earlier survey. For all panel respondents, there is a module on the incidence of economic shocks (both positive and negative) in the last 10 years.

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[1] Information, documentation and the full data set of the KHDS (1991-1994 and 2004) can be found on the Living Standards Measurement Study website: http://www.worldbank.org/lsms/. For further description of the original project and 1991-1994 data see Ainsworth *et al*. (1992) and World Bank (1993).

Although the KHDS is a panel of respondents and the concept of a 'household' after 10-13 years is a vague notion. It is common in panel surveys to consider recontact rates in terms of households. Excluding households in which all previous members are deceased (17 households with 27 people), the field team managed to recontact 93% of the baseline households (that is, at least one previous household member was reinterviewed in 2004). This is an excellent rate of recontact compared to panel surveys in low-income countries *and* high-income countries. The KHDS panel has an attrition rate that is much lower than that of other well-known panel survey summarized in Alderman *et al*. (2001) in which the rates ranged from 17.5% attrition *per year* to the lowest rate of 1.5% per year. Most of these surveys in Alderman *et al.* (2001) covered considerably shorter time periods (two to five years). From an individual respondent perspective, excluding people who died, 82% of all respondents (5,404 total) were located and reinterviewed.

Because people have moved out of their original household, the new sample in KHDS 2004 consists of about 2,700 households which were recontacted from the 832 baseline households. Much of the success in recontacting respondents was due to the effort to track people who had moved out of the 51 baseline communities (mostly villages). One-half of all households interviewed in 2004 were tracking cases, meaning they did not reside in the baseline communities. Of those households tracked, only 38% were located nearby the baseline community. Overall, 32% of all households were not located in or relatively nearby the baseline communities. While tracking is costly, it is an important exercise because migration and dissolution of households are often hypothesized to be important responses to hardship and a strategy to escape poverty. Excluding these households in the sample raises obvious concerns regarding the selectivity of attrition. In particular out-migration from the village, dissolving of

households, and even marriage, may be responses to changing economic or family circumstances. Thus, this relocation may be correlated with differential consumption growth paths. This is what is observed in these data: income outcomes for individuals who migrated are significantly different than that experienced by non-migrants (see Beegle, De Weerdt and Dercon, 2006b). Moreover, respondents who moved farther away experienced the most income growth (where the greatest distance tends to be those who were residing in Dar es Salaam in 2004).

The main focus of this study is the impact of adult mortality on subsequent consumption (or poverty) of survivors as much as 12 years later. Adult mortality is but one of many events that can have serious negative effects on future consumption outcomes. To get a sense of the overall scope of shocks, as perceived by panel respondents, Table 1 gives an overview of the most important shocks mentioned by the panel respondents of age 20 or older in 2004. Each panel respondent above the age of 20 reported, on a scale of 1 to 5, how their wealth and living conditions were in each year between 1994 and 2003. For each year labelled 'very bad' (5), the most important reason for this was asked. Table 1 shows that about two-thirds of respondents reported at least one of these years to be very bad. About 20% of all respondents reported that their living conditions and wealth were 'very bad' due to the death of a relative or other person; this is the most frequently mentioned reason. Serious illness and adverse weather condition leading to harvest failure score second and third place at 13% and 12% of panel respondents respectively. In total 16% of the panel respondents above 20 years old report at least one year to have been very bad because of agricultural problems (including negative effects of weather problems which affected prices of output and crop or livestock pests and diseases). Loss of assets due to crime, violence, eviction, fire etc. and problems related to wage or other off-farm

employment were the next major category of shocks. About 2% of the respondents reported very bad living conditions because of family problems and 1% because of jail sentences or lawsuits.

One of the difficulties with measuring the events that have happened in order to assess the impact of the shock is that these events are usually self-reported by respondents. The problem, then, is that we may not have a measure of the event itself but rather a report of the event *conditional* on being a significant negative economic shock from the perspective of the household. Thus, shocks reported by households will be endogenous, resulting in an upward bias in the impact of the event if more salient events are those whose socio-economic impact is larger.

Using information on the survival status of our sample of baseline respondents, as well as the panel respondents with whom they resided when they died between the baseline and 2004 rounds, we will measure deaths not based on the panel respondent identification of the shock but from the actual event itself.[2] However, for understanding how an adult death may be correlated with other negative events, we will also use the household reports of agricultural shocks with the caveat above.

Table 2 shows the distribution of the occurrence of a death in the household (categorized by year and sex of the deceased) for the 2,611 households with full information, used in the analysis. These are the households interviewed in 2004 with at least one member who was included in the baseline data for 1991-94. In total 961 individuals we had interviewed in 1991 died before 2004. This study restricts the deaths considered to those individuals who were aged between 20 and 55 at the time of their death, included in the baseline data, and were living with at least one other panel respondent at the time of their death. The data show that these

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[2] Household questionnaires that measure adult death through explicit questions on recent mortality episodes rather than a "shock" module in the household questionnaire will also avoid this issue. Other data which can also be used include information on events from a community questionnaire or rainfall data, both of which result in less variation as they measure covariate events.

deaths affected 644 panel households (in the sense that a member of the household suffered the loss of an adult living with them), on which we have full baseline and follow-up data, constituting nearly one quarter of the total sample of panel households.

Methodology and empirical specification

Using the sample of respondents interviewed in 2004, the objective in this study is to evaluate the impact of adult mortality on consumption for surviving respondents, focusing on the growth in consumption between the baseline and follow-up survey. We use as the unit of observation the surviving panel household i, defined as a household that includes at least one survey respondent who was interviewed in the baseline. The treatment of interest is the adult mortality shock D. The relationship of interest used in the evaluation can be written as the following general specification:

$$(\ln C_{i1} - \ln C_{i0}) = \beta X_{i0} + \gamma D_i + \varepsilon_{i1} \qquad (1)$$

Consumption (C) is measured in per capita terms at baseline, (period 0, which is the first KHDS 1991-1994 interview and for most households refers to an interview in late 1991 or early 1992) and in 2004 (period 1).[3] Since consumption growth may be characterised by substantial heterogeneity among households, vector X is a set of control variables, such as household characteristics of the respondents in the panel households, measured at baseline, including age, sex, wealth, education and other initial conditions. Death (D) captures the death of a previous

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[3] Consumption per capita is the annual per capita Tanzania shilling value of total household consumption, including actual consumer expenditures on food and non-food items as well as the valuation of foods grown by farm households or received as gifts. The consumption values are in terms of average price level of the KDHS villages from January-June 2004. The average exchange rate during this period was 1,107 Tanzania Shillings to $1 USD. We compute baseline per capita consumption based on the first interview of the 1991-1994 panel as the questionnaire in this round is consistent with the 2004 questionnaire. For the second-fourth rounds of 1991-1994 the consumption modules are not identical. Specifically, the recall periods of several major subcomponents of the consumption aggregate refer to the 6-month intervals between survey rounds rather than a 12-month recall.

household member living in household i at time of death. Thus, these deaths occur sometime between period 0 (October 1991- January 1994) and period 1 (2004).

Estimated using OLS, equation (1) is equivalent to a difference-in-difference specification with a set of controls, and the parameter γ offers the average treatment effect. There are important econometric difficulties with estimating equation (1). First, while the difference-in-difference specification controls for unobserved (time-invariant) heterogeneity affecting *levels* of consumption, and X_{i0} captures observed heterogeneity affecting *changes* in consumption, there could be unobserved heterogeneity affecting consumption changes, correlated with adult death. For example, deaths may occur with higher frequency in poor and vulnerable households with limited growth potential. The controls at baseline may not limit this problem sufficiently. Also, death could be endogenous to consumption since consumption expenditures can influence health and mortality.[4]

Our (drastic) solution is to estimate a model with initial household fixed effects (IHHFE) whereby individuals are compared to others who were living with them in the same household in 1991.[5] Having data which follows individuals who have moved from the original household allows us to exploit the following variation: some baseline household members may be living with the deceased individual at the time of the death (between 1991 and 2004), while others will have moved into different households prior to the death (see Appendix 1 for further discussion). Defining the initial household for panel household i as household j, we estimate

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[4] Grimm (2006), for example, addresses this by instrumenting deaths. In our context, some of this concern is mitigated because the deaths are primarily caused by a disease which is almost always undiagnosed and fatal. Moreover, treatment is extremely limited; ARV treatment has only recently been available for a small number of HIV-infected persons through the Kagera's Regional Hospital in Bukoba.

[5] Witoelar (2005) uses a similar approach which he calls extended-family fixed effects. He studies consumption smoothing and income pooling among split-off households using data from the Indonesia Family Life Survey panel. He finds evidence against income pooling within the set of split-off households, although he does conclude that to some degree households within the extended family pool resources.

$$(\ln C_{i1} - \ln C_{i0}) = \beta X_{i0} + \gamma D_i + \nu_j + \varepsilon_{i1} \tag{2}$$

in which ν_j captures all (unobserved and observed) heterogeneity associated to belonging to initial household j and X_{i0} is now restricted to the initial individual characteristics of panel individuals now residing in a particular panel household.

Nevertheless, more potential problems remain. First, adult mortality may be correlated with other shocks that occur. For example the adult death may be correlated with agricultural shocks. To investigate this, we address this by including information on agricultural shocks for the sample of people age 20 or older for whom we have this information. Secondly, the IHHFE framework implies that the impact of an adult death is identified based on the sample of households that have since separated and reside in different households by 2004. It is possible that the multiple households in 2004 which stem from the same origin household are rather different from each other, for example some consisting of close relatives of each other, while others only consist of initial household members that are otherwise unrelated to other households in the dynasty, making the treatment group of household members (those who remained living with the person who will later die) incomparable to the comparison group of household members (those who will have split-off before the person in question dies). To investigate this further, we re-run the regressions focusing on a much narrower control and treatment group, defined on the basis of a sub-sample of the data set, in which we only consider initial households that include individuals with the same blood relationship to the adults that subsequently died. We report on three sub-samples: children of the deceased, parents and children of the deceased, and spouses, parents and children of the deceased. The variation used to identify the impact of the adult mortality shock is then based on those households splitting so that these different types of blood relatives are spread across different households. In all three sub-samples, the control and

treatment groups are then defined on the basis of a particular close blood-relationship to the deceased.

Finally, the use of the IHHFE framework creates also a problem of interpretation. Since we only have one observation of changes in consumption, the use of the initial household fixed effect implies that we are jointly testing whether there is any impact of an adult mortality shock *and* whether there is risk-sharing within all 2004 households from the same initial household. For example, it could be that a mortality death is very costly, but that these costs are effectively insured by the other members of the same initial household, so the effect shows up as insignificantly different from zero on the mortality term, since the entire effect is shared by the same 'extended' family and picked up by the initial household fixed effect. To disentangle these effects, we drop the initial household fixed effects, and revert to a specification as in (1), include specific household baseline characteristics in X_{i0}, and augment the regression with a term D_j capturing the adult mortality shock of *any* initial panel respondent (which is the same for all split-offs from the same initial household). The regression then becomes:

$$(\ln C_{i1} - \ln C_{i0}) = \beta X_{i0} + \gamma D_i + \varphi D_j + v_j + \varepsilon_{i1} \qquad (3)$$

If risk-sharing of shocks were to take place, then the φ should be significantly different from zero, controlling for a shock faced only by a particular panel household. Full risk-sharing in the extended family would be implied if $\gamma = 0$ as well.

Regression Results

We start by estimating Equation (2), using IHHFE. Table 3 columns (1) and (2) offer results excluding crop shocks; in columns (3) and (4) these shocks have been added. Since the shocks were only collected for panel respondents above 20 years old, the sample is slightly

smaller. We first offer a test of the impact of any adult mortality, and whether we control for crop shocks or not, we find a strongly significant impact of a prime-age death on the household of the deceased. We find that the deaths that occurred between 2000 and 2004 reduce consumption growth by 30 percentage points (30% of the standard deviation of the 13-year average of the regression sample). With average consumption growth in the sample used in column (3) at 45% over 13 years (about 3% per annum), this means that these households grew with 15% over the same 13 year period. [6] Assuming growth was equal across the years, a household experiencing an adult death between 2000-2004 would already have grown with 24% between 1991-1999 and subsequently experiences a consumption downfall of 8 percentage points, or 7 percent.

For the death occurring earlier, the coefficients are negative, smaller in absolute value and less significant. This pattern emerges in further specifications as well. *Prime facie*, this would be evidence that adult mortality shocks are not persistent at all: adult mortality shocks have no effects after five years, so that there is a strong recovery. However, it would also be consistent with our inability to control fully for other shocks and events in this 13 year period: even if these shocks are not correlated with adult mortality shocks, and therefore even if they do not affect the point estimates in the regression, they create noise, confounding the difference between the treatment and control group. In other words, we may be facing a problem of increasing imprecision in our ability to identify the impact of the shocks of a long time ago. While we may not be able to identify persistence, this is not strong evidence against persistence either.

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[6] For a more detailed discussion of the factors driving consumption growth in this period in these data, see Beegle *et al*. (2006b).

Splitting the mortality variable up by the gender of the deceased and focusing on the specification controlling for crop shocks (Table 3 column 4) shows that this affect is driven by a large and relatively precisely estimated impact of female deaths from 2000-2004, and no significant effect can be found for male deaths. The results in (4) are consistent with (1) and (3), and once we control for agricultural crop shocks there are no *significant* persistent effects for mortality shocks that occurred more than five years ago. It is interesting to note that crop shocks display a more persistent negative effect than mortality shocks: crop shocks going back to 1991 are significant and persistent for a long time, leading to growth losses with point estimates of between 17 and 40 percent.[7]

Initial household conditions may play an important role in the magnitude of the effect of deaths on income growth. We explore this potential heterogeneity by interacting adult death with baseline wealth of the household. Specifically, interaction terms are included for the mortality shock and whether the household belongs to the top quartile of household consumption in 1991 ('rich'), as well as having household consumption below the median ('poor'). Table 4 shows some of the interactions that were investigated. In general, we do not find a significantly different impact according to wealth or poverty. There was similarly no evidence of that crop shocks had different effects for rich or poor households either. In all regressions explored, the adult death effect remains strongly significant and relatively stable.

The magnitude of the effect of adult deaths might also vary depending on the length of illness preceding the death, in addition to the realization of the death itself. Spells of illness prior to death are hypothesised to constitute periods of severe financial stress to households (for

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[7] In 1996-99, the crop shock variable is only significant at 11%, but almost the same value and same standard error as in the more parsimonious equation (3)). Testing a linear restriction on the three crop shocks suggests that they are not significantly different from each other ($F(2, 1416)=0.43$), and jointly significantly different from zero ($F(3,1416)=4.11$, or significant at 1%). Crop shocks, even those more than 10 years ago, appear to have an impact.

reasons noted above). The inclusion of a measure of length of illness (the number of months the person was ill for before he or she died), however, did not have any additional explanatory power over the mortality variables (Table 4, column 3).[8]

As discussed in the previous section, the tests in Tables 3 and 4 are a joint test of the impact of an adult mortality shocks whether there is any impact of an adult mortality death *and* whether there is risk-sharing within all split-offs of the same initial household. As in equation (3), we over-specify the earlier regressions with a variable describing the mortality of any prime age adult in the baseline survey belonging to the initial household. Under the null of full risk-sharing, consumption changes should not be affected by mortality shocks of a baseline member residing with the particular household, but should be significant for any mortality shock of a baseline member of the initial household. Table 5 presents the results.

To implement the test, the otherwise perfectly collinear initial household fixed effect will have to dropped, and replaced by a vector also including a series of baseline household characteristics. We include initial asset values, household demographics (males/females aged 0-5, 6-15, 16-65 and 66+), characteristics of the head at baseline (number of years of education, sex, age and age squared) and the number of children of the head living outside the household. The regression also includes a full set of cluster fixed effects, (mean) age fixed effects, the mean individual baseline characteristics of those residing together in 2004 and agricultural shocks. Finally, to capture initial household conditions more fully, we also include household consumption at baseline. Since this variable is most obviously likely to be endogenous (including but not exclusively due to measurement error, thereby affecting both the dependent variable and the initial level of consumption), it is necessary to explore the use of IV estimation. Finding

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[8] The information on months ill before death was collected using a specific instrument probing for details on each mortality case.

credible instruments for initial consumption is not self-evident, but we have access to rainfall data preceding the baseline survey. In the Kagera farming system, droughts and generally poor rainfall can present serious problems to crop income, so we use z-scores of rainfall deviations in the year preceding the baseline, with positive rainfall deviations truncated to zero. To allow for household level variation, we use as identifying instruments all the RHS variables interacted with this truncated z-score. The full first stage regression is reported in Appendix 2. The diagnostics for the instruments are encouraging: for example, the Cragg-Donald weak instrument F-stat is 8.06, while the Sargan over-identification statistic is 14.66 with a p-value of 0.55.

Table 5 offers the results. First, column (1) offers the IHHFE findings for the particular (somewhat smaller) sub-sample for whom we have full information on all the controls used. As before, a clear negative effect of a prime age death can be found. Column (2) uses a specification as in equation (3), using all the controls, including initial consumption, but the latter treated as exogenous. The impact of an adult mortality shock is again negative and significant at 5 percent: perfect risk-sharing is not taking place. The impact of any prime age death of a respondent in the initial household, our means of assessing the presence of at least some risk-sharing, is not significantly different from zero, implying that there is no evidence suggestive risk-sharing between the split-offs of the same initial household. However, this may a consequence of the presence of lagged consumption, a likely endogenous variable, as an explanatory variable. Column (3) therefore offers the same regression, this time using as discussed above the interaction of household characteristics with the truncated z-score of rainfall in the year preceding the baseline. The coefficient and standard error on deaths in 2000-2004 are virtually exactly the same as without instrumenting, suggesting that the likely presence of endogeneity

from including an initial value of consumption as a control variable did not affect our findings regarding the impact of a prime age death in the household.

A final concern with the results presented in Tables 3 and 4 using IHHFE is that the treatment and comparison groups may not be comparable: even though split-offs originate from the same initial household, there may be some non-random sorting into particular groups, making the evaluation of the impact of adult mortality flawed. For example, it may be that baseline individuals in a household were split in a group of relatives and non-relatives of the deceased before the mortality shock. This could be either in anticipation of a period of hardship in the household or unrelated to it. For example, in anticipation of an adult dying, household splits may also involve the placement of the ill person with relatives only, and not randomly across the split-offs. Our regressions already control for a range of variables, like age fixed effects, sex, years of schooling and number of biological children of the respondent living outside the household at baseline. In order to specifically accommodate these concerns, we restricted our sample to create more directly comparable control and treatment groups. We report on three restricted sub-samples: children of the deceased, parents and children of the deceased, and spouses, parents and children of the deceased. The variation used to identify the impact of the adult mortality shock is then based on those households splitting so that these different types of blood relatives are spread across different households. While this may not solve all problems of (endogenous) placement across split-off households, it forces the comparison to be done between 2004 households with similar blood relatives. Table 6 reports the results, while Table 7 shows the number of 'useful' treatment observations from which the results are derived. In all specifications, the impact of adult death is strongly significant, and larger in size compared to the earlier results in Table 3. It would appear that households of children living with a parent that

dies are more strongly affected than other households, and the effect declines somewhat when broader categories of relatives are added. In view of the results, this would also suggest that living in a household in which a non-relative dies has less of an impact on consumption changes. Although not reported, this is confirmed in the data. Again, female deaths dominate the results and male deaths do not appear to have a significant impact.

Conclusions

More than 20 years after the AIDS epidemic surfaced in Africa, it is perhaps surprising that there are significant gaps in empirical evidence of the impact of HIV/AIDS in regards to the income of surviving household members. This paper uses unique longitudinal data designed to measure this impact. In the broader context, these results can be placed in the context of macroeconomic studies which draw competing conclusions about the implications of the AIDS epidemic for national levels of income per capita. We provide evidence that adult mortality shocks, especially of females, have a large impact on the growth in consumption of surviving household members in a region of Tanzania that has been seriously afflicted by the HIV/AIDS epidemic

In our sample, 22% of the households experienced an adult death between 1991 and 2004. We find significant and robust evidence that the impact of a prime-age death results in a 7% drop in consumption in the first five years after the death. After five years the effect remains negative, but becomes smaller and more imprecise. Statistically, the effects are not significant, so there is no evidence of a persistent impact of shocks after five years, suggesting a strong recovery. Nevertheless, we should be cautious with this interpretation: Is there really no persistence to these serious shocks? The regressions are not be able to control for all other shocks and events, moving households up and down the consumption distribution, creating noise and

potentially contributing to the imprecision of the estimates for mortality shocks that have occurred in the early part of the 13-year period. Given the point estimates, the lack of persistence is not easily proven either, although they are becoming smaller in absolute value for shocks that occurred a long time ago, so the effect may well be fading. This would suggest that a 'casual observer' should not expect to see distinguishable differences between households who did or did not experience a prime-aged adult death as long as 13 years ago, but that the isolated effect of the death may well be persistent even to date and could have important economy-wide influence in high mortality areas. Of course, we are unable to confirm that this result reflects the actual time path of the impact of adult mortality on income, or changes in regards to the income effects of adult mortality over this time period (1991-2004) as the scope of epidemic changes in Tanzania.

The unique feature of our data set, that is follows individuals belonging to a baseline set of households re-interviewed more than 10 years later, allows us to show that these results are robust to household heterogeneity. The impact of death does not appear to be different among households that are richer or poorer at baseline. The effect on income growth appears largely linked to the mortality event; the impact does not vary with the length of the illness. We also did not find any evidence of risk-sharing between different households split off from the original households at baseline, while the impact of adult death is higher when the death involves a close blood relative.

How households may have been able to cope in the long run is to be assessed further in these data. For example, it may be possible that it is achieved via further re-organization of the household, including via migration and household re-formation. In Beegle and Krutikova (2006), it is found that orphans are more likely to marry young, while migration of some individuals also

can re-align the returns to labor within the family. More research is needed to assess these issues

more comprehensively.

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Table 1: Shocks experienced from 1994-2003

Type of shock	Frequency	Percent
Death of family member	629	20%
Serious illness	399	13%
Poor harvest due to adverse weather	369	12%
Poor harvest due to pests or crop diseases	75	2%
Poor crop prices	71	2%
Problems with livestock	3	0%
Loss of assets due to crime, violence, eviction, fire, etc.	196	6%
Loss in wage employment	127	4%
Loss in off-farm employment	40	1%
Family problems	49	2%
Lawsuits and imprisonment	39	1%
Other reasons	83	3%
Total	2,080	67%

Note: Based on the sample is 3,017 panel respondents 20 years and older in 2004 who reported that any of these years was "very bad".

**Table 2: Panel households experiencing the death of
a prime-age baseline respondent**

| Year of death | (1) Sample of Panel Households used in Main Regressions (*N*=2,611) | |
	Number	Percent
1991-1995	254	9.7
Male death	134	5.1
Female death	171	6.5
1996-1999	198	7.6
Male death	76	2.9
Female death	159	6.1
2000-2004	192	7.4
Male death	76	2.9
Female death	149	5.7

Table 3: Consumption growth 1991-2004: Effect of a prime-aged death (20-55 yrs.)
of a baseline respondent on survivors (Initial Household Fixed Effects)

	(1)	(2)	(3)	(4)
Death 1991-1995	-0.180		-0.111	
	[0.156]		[0.171]	
Death 1996-1999	-0.070		-0.149	
	[0.145]		[0.157]	
Death 2000-2004	-0.258		-0.298	
	[0.117]**		[0.130]**	
Male Death 1991-1995		-0.126		-0.019
		[0.223]		[0.245]
Male Death 1996-1999		0.055		0.081
		[0.239]		[0.269]
Male Death 2000-2004		-0.172		-0.057
		[0.193]		[0.205]
Female Death 1991-1995		-0.388		-0.184
		[0.215]*		[0.236]
Female Death 1996-1999		-0.139		-0.059
		[0.173]		[0.182]
Female Death 2000-2004		-0.217		-0.385
		[0.140]		[0.156]**
Crop Shock 1991-1995			-0.398	-0.400
			[0.223]*	[0.224]*
Crop Shock 1996-1999			-0.191	-0.184
			[0.114]*	[0.114]
Crop Shock 2000-2004			-0.168	-0.174
			[0.088]*	[0.088]**
Additional controls				
Average # of biological children living elsewhere	-0.061	-0.061	-0.060	-0.061
	[0.026]**	[0.026]**	[0.026]**	[0.026]**
Avg. years of schooling	0.017	0.017	0.021	0.021
	[0.011]	[0.011]	[0.011]*	[0.011]**
Share of males in initial HH	0.227	0.227	0.296	0.299
	[0.049]***	[0.049]***	[0.052]***	[0.052]***
Share of chronically ill HH members	-0.032	-0.037	-0.048	-0.048
	[0.093]	[0.093]	[0.095]	[0.096]
Observations	2,611	2,611	2,281	2,281

Notes: Unit of observation are households in 2004. Regressions include age fixed effects (i.e. a spline function allowing for different slop for each year). Regressions (3) and (4) have a smaller sample because the crop shock variable was only collected for panel respondents above 20 years old. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 4: Consumption growth 1991-2004: interaction effects
with prime-aged death (20-55 yrs.) of a baseline respondent on survivors
(Initial Household Fixed Effects)

	(1)	(2)	(3)
Death 1991-1995	-0.058	-0.323	-0.145
	[0.190]	[0.290]	[0.180]
Death 1996-1999	-0.176	0.017	-0.211
	[0.180]	[0.226]	[0.175]
Death 2000-2004	-0.347	-0.438	-0.318
	[0.151]**	[0.180]**	[0.134]**
Baseline wealth interactions			
Rich Household & Death 1991-1995	-0.258		
	[0.436]		
Rich Household & Death 1996-1999	0.145		
	[0.370]		
Rich Household & Death 2000-2004	0.214		
	[0.298]		
Poor Household & Death 1991-1995		0.337	
		[0.358]	
Poor Household & Death 1996-1999		-0.315	
		[0.313]	
Poor Household & Death 2000-2004		0.269	
		[0.258]	
Length of illness			
Months ill before death 1991-1995			0.009
			[0.013]
Months ill before death 1996-1999			0.005
			[0.006]
Months ill before death 2000-2004			0.0002
			[0.001]
Crop shock and interactions			
Crop Shock 1991-1996	-0.331	-0.524	-0.398
	[0.242]	[0.364]	[0.223]*
Crop Shock 1996-1999	-0.231	-0.286	-0.189
	[0.125]*	[0.163]*	[0.114]*
Crop Shock 2000-2004	-0.160	-0.120	-0.169
	[0.098]	[0.133]	[0.088]*
Rich Household & Crop Shock 1991-1996	-0.431		
	[0.654]		
Rich Household & Crop Shock 1996-1999	0.244		
	[0.303]		
Rich Household & Crop Shock 2000-2004	-0.050		

Table 4: Consumption growth 1991-2004: interaction effects
with prime-aged death (20-55 yrs.) of a baseline respondent on survivors
(Initial Household Fixed Effects)

	(1)	(2)	(3)
	[0.226]		
Poor Household & Crop Shock 1991-1996		0.181	
		[0.466]	
Poor Household & Crop Shock 1996-1999		0.191	
		[0.231]	
Poor Household & Crop Shock 2000-2004		-0.077	
		[0.177]	
Additional controls			
Average # of biological children of PHHM living elsewhere	-0.061	-0.061	-0.060
	[0.026]**	[0.026]**	[0.026]**
Avg. years of schooling	0.020	0.021	0.021
	[0.011]*	[0.011]*	[0.011]*
Share of males in initial HH	0.295	0.297	0.298
	[0.052]***	[0.052]***	[0.052]***
Share of chronically ill HH members	-0.044	-0.048	-0.046
	[0.096]	[0.096]	[0.096]
Observations	2281	2281	2281

Notes: 'Poor' is defined as below median household consumption in 1991; 'rich' is defined as top quartile household consumption in 1991. Length of illness is the number of months the deceased was ill before death. Regressions include age fixed effects (i.e. a spline function allowing for different slop for each year). Regressions include age fixed effects (i.e. a spline function allowing for different slop for each year). * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 5: Consumption growth 1991-2004:
Distinguishing between Income Pooling and Shocks

	(1) IHHFE	(2) non-IV cluster and age FE	(3) IV cluster and age FE
Death 1991-1995	-0.092	-0.018	-0.031
	[0.177]	[0.136]	[0.138]
Death 1996-1999	-0.241	-0.087	-0.083
	[0.166]	[0.130]	[0.131]
Death 2000-2004	-0.324	-0.210	-0.210
	[0.137]**	[0.106]**	[0.106]**
Any Death 1991-1995 from Initial HH		-0.019	-0.009
		[0.124]	[0.125]
Any Death 1996-1999 from Initial HH		-0.063	-0.067
		[0.113]	[0.113]
Any Death 2000-2004 from Initial HH		0.112	0.121
		[0.079]	[0.081]
Crop Shock 1991-1996		-0.137	-0.137
		[0.180]	[0.180]
Crop Shock 1996-1999		-0.130	-0.130
		[0.095]	[0.096]
Crop Shock 2000-2004		-0.245	-0.245
		[0.071]***	[0.071]***
Additional controls: 2004 household			
Avg. years of schooling of PHHM	0.021	0.026	0.024
	[0.011]*	[0.009]***	[0.009]***
Share of male PHHM	0.304	0.293	0.292
	[0.053]***	[0.048]***	[0.048]***
Share of chronically ill PHHM	-0.074	-0.045	-0.042
	[0.098]	[0.076]	[0.077]
Avg. #of biological children of PHHM	-0.07	-0.057	-0.059
living elsewhere	[0.027]**	[0.021]***	[0.021]***
Additional controls: baseline household			
Years of education head		0.006	0.003
		[0.009]	[0.010]
Sex of head		-0.128	-0.133
		[0.061]**	[0.062]**
Age of head		-0.011	-0.011
		[0.007]	[0.007]
Squared age of head		0.0001	0.0001
		[0.00007]	[0.00007]
No. of males 0-5 years		-0.068	-0.061
		[0.026]**	[0.029]**

Table 5: Consumption growth 1991-2004:
Distinguishing between Income Pooling and Shocks

	(1) IHHFE	(2) non-IV cluster and age FE	(3) IV cluster and age FE
No. of males 6-15 years		0.075	0.085
		[0.020]***	[0.026]***
No. of males 16-60 years		-0.019	-0.014
		[0.022]	[0.024]
No. of males 61+ years		-0.013	0.012
		[0.079]	[0.089]
No. of females 0-5 years		-0.053	-0.048
		[0.027]**	[0.028]*
No. of females 6-15 years		0.026	0.032
		[0.018]	[0.021]
No. of females 16-60 years		0.045	0.050
		[0.020]**	[0.022]**
No. of females 61+ years		-0.053	-0.045
		[0.050]	[0.052]
log of value of all assets		0.064	0.050
		[0.026]**	[0.034]
log of consumption (instrumented in (3))		-0.923	-0.817
		[0.046]***	[0.182]***
Observations	2150	2150	2150

Notes: Individual level variables are means across all split-off panel respondents living together. Regressions include age fixed effects (i.e. a spline function allowing for different slop for each year). Initial consumption is included as an endogenous variable instrumented by all RHS variables interacted with z-scores of rainfall deviations in the year preceding the baseline, with positive rainfall deviations truncated to 0. Diagnostics for the IV regression are as follows: Cragg-Donald weak instrument F-stat = 8.06; Sargan Statistic 14.66 (*p*-value 0.55). (2) same as (1) without instrumented baseline consumption. (3) preferred FE regression model on IV-model sample. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 6: Consumption growth 1991-2004, effect of a prime-aged death (20-55 yrs.) of a baseline respondent on survivors: initial household fixed effects on restricted sample of survivors having the same biological relation to the deceased.

	(1)	(2)	(3)	(4)	(5)	(6)
	Sample of children of deceased	Sample of children or parents of deceased	Sample of children, parents or spouses of deceased	Sample of children of deceased	Sample of children or parents of deceased	Sample of children, parents or spouses of deceased
Death 1991-1995	-0.279	-0.297	-0.142			
	[0.323]	[0.326]	[0.298]			
Death 1996-1999	-0.106	-0.312	-0.275			
	[0.271]	[0.270]	[0.257]			
Death 2000-2004	-0.736	-0.649	-0.587			
	[0.233]***	[0.223]***	[0.207]***			
Male Death 1991-1995				-0.018	0.024	0.059
				[0.464]	[0.479]	[0.412]
Male Death 1996-1999				0.133	0.028	0.15
				[0.516]	[0.434]	[0.403]
Male Death 2000-2004				-0.237	-0.461	-0.335
				[0.387]	[0.360]	[0.307]
Female Death 1991-1995				-0.263	-0.333	-0.117
				[0.443]	[0.434]	[0.415]
Female Death 1996-1999				-0.384	-0.063	-0.100
				[0.377]	[0.370]	[0.344]
Female Death 2000-2004				-0.838	-0.805	-0.631
				[0.302]***	[0.279]***	[0.257]**
Crop Shock 1991-1996	0.213	0.299	0.258	0.218	0.342	0.323
	[0.623]	[0.617]	[0.612]	[0.654]	[0.647]	[0.634]
Crop Shock 1996-1999	-0.442	-0.479	-0.346	-0.443	-0.480	-0.338
	[0.335]	[0.335]	[0.299]	[0.340]	[0.338]	[0.303]
Crop Shock 2000-2004	-0.082	-0.097	-0.133	-0.088	-0.103	-0.130
	[0.258]	[0.247]	[0.232]	[0.261]	[0.249]	[0.235]
Additional controls						
Avg. # of biological children living elsewhere	-0.194	0.189	0.111	-0.211	0.200	0.107
	[0.126]	[0.092]**	[0.081]	[0.128]	[0.093]**	[0.081]
Avg. years of schooling	0.012	0.045	0.039	0.019	0.054	0.044
	[0.028]	[0.026]*	[0.025]	[0.029]	[0.027]**	[0.025]*
Share of males in initial household	0.237	0.252	0.195	0.264	0.260	0.197
	[0.127]*	[0.120]**	[0.116]*	[0.128]**	[0.121]**	[0.117]*
Share of chronically ill household members	-0.102	-0.187	-0.251	-0.057	-0.166	-0.243
	[0.225]	[0.220]	[0.204]	[0.229]	[0.223]	[0.206]
Observations	527	665	722	527	665	722

Notes: Initial household fixed effects regressions as in Table 3, but with samples restricted to biological children of deceased in regressions (1) and (4); restricted to biological children or parents of the deceased in regressions (2) and (5) and restricted to biological children, parents or spouses of the deceased in (3) and (6). Standard errors in square brackets. * significant at 10%; ** significant at 5%; *** significant at 1%.
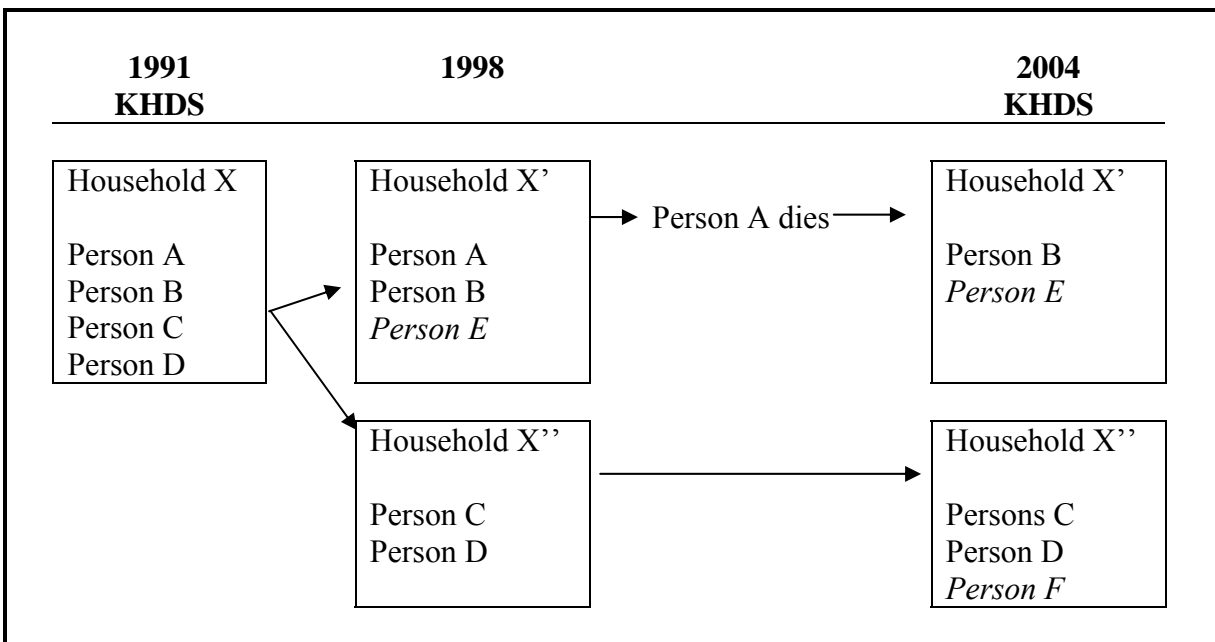
Table 7: Number of useful treatment observations in restricted sample of Table 6

	(1) Sample of children of deceased	(2) Sample of children or parents of deceased	(3) Sample of children, parents or spouses of deceased	(4) Sample of children of deceased	(5) Sample of children or parents of deceased	(6) Sample of children, parents or spouses of deceased
Death 1991-1995	35	51	56			
Death 1996-1999	50	65	68			
Death 2000-2004	46	66	68			
Male Death 1991-1995				20	31	34
Male Death 1996-1999				14	22	22
Male Death 2000-2004				18	30	31
Female Death 1991-1995				23	28	28
Female Death 1996-1999				32	41	45
Female Death 2000-2004				33	43	46

Notes: The number of treatment observations in the sample from initial households with at least one non-treatment observation included in the sample.

Appendix 1: Illustration of KHDS sample

As described in the text, the set of baseline households in 1991 grew into more than 2,700 households in 2004. The flowchart below illustrates the potential configuration of a household in the KHDS sample and how it relates to our identification strategy using the Initial Household Fixed Effects (IHHFE) estimator. In the example below, in 1991 household X consists of four individuals A, B, C and D. In 1998 household X has split into two households: household X' consisting of individuals A and B and a new individual E, and household X'' consisting of individuals C and D. Between 1998 and 2004, person A dies, while household X'' is joined by individual F. Using an IHHFE estimator, the effect of adult mortality will be identified from the comparison between household X' and X'' controlling for the otherwise shared history of both households X' and X''.

1991 KHDS	1998	2004 KHDS
Household X Person A Person B Person C Person D	Household X' Person A Person B *Person E* → Person A dies →	Household X' Person B *Person E*
	Household X'' Person C Person D →	Household X'' Persons C Person D *Person F*

Appendix 2: First Stage Regression from Table 5 of Consumption (log per capita) 1991

	Coef.	Std. Err.	t	P>|t|
Included Instruments				
Death 1991-1995	0.106	0.064	1.640	0.10
Death 1996-1999	-0.038	0.062	-0.610	0.54
Death 2000-2004	0.011	0.050	0.220	0.82
Any Death 1991-1995 from Initial HH	-0.087	0.059	-1.480	0.14
Any Death 1996-1999 from Initial HH	0.027	0.054	0.500	0.62
Any Death 2000-2004 from Initial HH	-0.064	0.038	-1.700	0.09
Crop Shock 1991-1996	-0.018	0.085	-0.210	0.83
Crop Shock 1996-1999	0.004	0.045	0.090	0.93
Crop Shock 2000-2004	-0.016	0.034	-0.470	0.64
2004 household characteristics				
Avg. # of biological children of PHHM living elsewhere	0.003	0.015	0.190	0.85
Avg. years of schooling of PHHM	0.014	0.007	1.880	0.06
Share of male PHHM	-0.033	0.044	-0.760	0.44
Share of chronically ill PHHM	0.012	0.069	0.170	0.86
Baseline household characteristics				
Years of education head	0.044	0.008	5.420	0.00
Sex of head	0.213	0.061	3.470	0.00
Age of head	-0.021	0.007	-2.910	0.00
Squared age of head	0.000	0.000	3.870	0.00
No. of males 0-5 years	-0.118	0.025	-4.700	0.00
No. of males 6-15 years	-0.059	0.018	-3.310	0.00
No. of males 16-60 years	-0.057	0.020	-2.890	0.00
No. of males 61+ years	-0.533	0.080	-6.630	0.00
No. of females 0-5 years	-0.101	0.024	-4.250	0.00
No. of females 6-15 years	-0.004	0.015	-0.260	0.80
No. of females 16-60 years	-0.053	0.018	-3.040	0.00
No. of females 61+ years	0.021	0.048	0.430	0.66
log of baseline value of all assets	0.201	0.026	7.750	0.00

Appendix 2: First Stage Regression from Table 5 of Consumption (log per capita) 1991

	Coef.	Std. Err.	t	P>|t|
Excluded Instruments				
Interaction terms of negative rainfall deviation in 1991				
(calculated as a truncated z-score out of a 25 year distribution) with:				
2004 household characteristics				
Avg. # of biological children of PHHM living elsewhere	-0.046	0.038	-1.200	0.229
Avg. years of schooling of PHHM	-0.001	0.020	-0.030	0.978
Share of male PHHM	-0.072	0.121	-0.600	0.550
Share of chronically ill PHHM	0.124	0.194	0.640	0.524
Baseline household characteristics				
Years of education head	0.062	0.024	2.570	0.010
Sex of head	0.484	0.171	2.820	0.005
Age of head	-0.073	0.018	-4.090	0.000
Age of head squared	0.001	0.000	4.690	0.000
No. of males 0-5 years	-0.069	0.068	-1.020	0.310
No. of males 6-15 years	0.133	0.051	2.590	0.010
No. of males 16-60 years	-0.004	0.053	-0.080	0.939
No. of males 61+ years	-0.905	0.208	-4.360	0.000
No. of females 0-5 years	-0.144	0.070	-2.060	0.039
No. of females 6-15 years	0.253	0.046	5.540	0.000
No. of females 16-60 years	0.001	0.048	0.030	0.980
No. of females 61+ years	0.261	0.123	2.120	0.034
Log of value of all assets	0.219	0.074	2.970	0.003