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Does AIDS-Related Mortality Reduce Per-Capita Household Income? Evidence from Rural Zambia*

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This paper evaluates the effect of AIDS-related mortality on per-capita incomes of surviving household members, using a large nationally representative sample of rural households from Zambia. To minimize selection bias that may arise because AIDS is likely to be the endogenous outcome of individual behavior, we employ a difference-in-difference propensity score matching estimator. We find that the death of a prime-age member has no significant impact on per-capita household income. This result continues to hold when we control for spillover effects by excluding households from the control group if members departed or joined for reasons related to AIDS. A likely explanation for this finding is that surviving household members pursue a mix of income and demographic coping strategies that prevents income losses in the short to medium run.

Keywords: HIV/AIDS, prime-age mortality, per adult equivalent income, difference-in-difference, propensity score matching, spillovers, Zambia

JEL classification: I31, J19, C14, C23

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1. Introduction

Within two decades, AIDS has become the leading cause of adult death on the African continent (UNAIDS 2007). In 2007 alone about 1.5 million Africans died due to AIDS and almost 2 million got infected with HIV (UNAIDS 2008a). With 22 million, or two thirds, of the global total of 33 million people infected with HIV, sub-Saharan Africa is the epicenter of the AIDS epidemic. The consequences of AIDS-related deaths are not comparable to those from other diseases because AIDS hits adults at the peak of their productivity and earning capacity. It disables and kills the people on whom families rely for their livelihood. Consequently, it has been widely conjectured that AIDS may constitute a severe impediment to Africa's socio-economic development. As a result, development agencies, international organizations and national governments are spending billions of dollars each year to respond to the epidemic.

Yet, the impact of AIDS-related mortality on household welfare as measured by per-capita income or expenditure is not clear a priori. While AIDS-related mortality may lower total household income through the loss of productive family members, the size of the household is also reduced by the death of a member. The direction of the effect depends on whether the deceased member was a net consumer or a net producer for the household. In addition, AIDS-afflicted households may cushion the adverse welfare effects through adjusting the income-generating activities of remaining members (income coping) or the composition and size of the household (demographic coping).

Income-coping strategies include changes in the supply and allocation of household labor. In rural areas, non-farm employment may be given up to fill labor shortages on the farm. Case studies for Rwanda (Donovan et al. 2003) and Kenya (Yamano and Jayne 2004) show for example that off-farm income of vulnerable and asset-poor households is at particular risk if an adult member dies. By contrast, for the Kagera region in Tanzania, Beegle (2005) finds no significant changes in labor supply of individuals in households having experienced a prime-age adult death. Another income-coping strategy is the sale of assets. Yamano and Jayne (2004) as well as Chapoto and Jayne (2008) find that prime-age mortality has only a minor impact on livestock, but greatly reduces the value of small animals such as goats and sheep in rural Kenya and Zambia, respectively.

Demographic coping may involve attempts to attract additional net producers, e.g. by letting migrants return to support the household, or to reduce the number of net consumers, e.g. by sending children away to members of the extended family. Demographic coping has been shown to exist in the African context, with a strong variation across countries. At one extreme, afflicted households in Kagera, Tanzania were able to maintain their household size (Ainsworth et al. 1995). In Uganda, by contrast, household size declined by about one person after a prime-age death, indicating that no new members could be attracted (Menon et al. 1998). Based on data from 21 countries across Africa, Beegle et al. (2009) also show that many single orphans do not stay with the remaining parent, but are rather sent away to live with other relatives.

Despite the theoretical ambiguity and the policy relevance of the topic, surprisingly little empirical knowledge exists about the effects of AIDS deaths on the welfare of African households (Beegle and De Weerdt 2008).[1] This paper aims to make a contribution to filling this gap by evaluating the changes in per-capita household incomes associated with AIDS-related mortality in rural Zambia, distinguishing between the deaths of household heads and spouses, who are likely to be net producers, as well as other adult household members, who might rather tend to be net consumers. Our methodological focus is on making causal inferences and we use a large nationally representative longitudinal survey that tracks about 5400 Zambian households over the period 2001-2004.

To minimize the selection bias that potentially arises because AIDS-related mortality is likely to be the endogenous outcome of individual behavior, we employ a difference-in-difference propensity score matching (DID-PSM) estimator. While PSM removes the selection bias due to observed differences between afflicted and non-afflicted households, the DID estimator differences out the time-invariant unobserved heterogeneity between the two groups.

To the extent that demographic coping plays a significant role, spillover effects may cause a further bias in the estimated impact of prime-age mortality. For instance, households without a deceased member may experience welfare losses when taking care of children sent away by AIDS-afflicted households. These effects have been neglected in the previous literature. We assess their relevance by testing whether non-afflicted households with AIDS-related demographic changes fared worse in per-capita income terms than their counterparts without such changes. In estimating the welfare effects of prime-age mortality, we exclude households from the control group if members departed or joined for reasons related to AIDS.

[1] Beegle et al. (2008) for Kagera, Tanzania, as well as Mather and Donovan (2008) for Mozambique are notable exceptions.

Our paper is closely related to two previous empirical studies by Beegle et al. (2008) and Chapoto and Jayne (2008). Beegle et al. (2008) share our objective of evaluating the impact of prime-age mortality on the per-capita welfare of surviving household members. Yet they do so for one specific region characterized by high HIV prevalence, the Kagera district in Tanzania, which makes it difficult to extrapolate their results to the national level. We use the same dataset as Chapoto and Jayne (2008), but their analytical focus is somewhat different from our study. Most notably, they look at how AIDS-related deaths affect various (farm and non-farm) income sources rather than household income per capita. Furthermore, Beegle et al. (2008) as well as Chapoto and Jayne (2008) rely on parametric approaches as compared to the non-parametric PSM applied here.

The remainder of the paper is structured as follows. Section II describes some key characteristics of the dataset, while Section III explains the method used to perform the estimations. The results of the empirical analysis are presented in Section IV. The paper closes with some concluding remarks.

2. Data and Setting

We use a nationally representative longitudinal data set of 5,420 rural farm households from Zambia surveyed in 2001 and 2004. As a very poor country with high HIV prevalence rates, Zambia makes an interesting case study. Three in four rural Zambians live below the national poverty line (World Bank 2007) and about 15 percent of adults aged between 15 and 49 are HIV-positive (UNAIDS 2008b). As a result, life expectancy at birth has fallen to less than 41 years, making it the second lowest in the world (UNDP 2007). This naturally raises the question to what extent AIDS has contributed to the low levels of socio-economic outcomes in Zambia.

The household survey was conducted by the Central Statistical Office in conjunction with the Ministry of Agriculture and Cooperatives and Michigan State University. In addition to standard socio-economic information of the household and its members, the survey paid particular attention to capturing a household's income generation process and its demographic development including the death of members. It not only recorded all deaths of household members after 2001, but also asked whether households had experienced the loss of a member between 1996 and 2001. To the best of our knowledge, the survey design makes it one of the most representative and comprehensive longitudinal household surveys available from Africa to examine the socio-economic effects of prime-age mortality. In particular, the combination of both the large sample size and relatively high HIV prevalence in Zambia provides a sufficiently large subsample of households afflicted by adult mortality. It thus allows examining the impact of a low-frequency event such as prime-age death with meaningful statistical power.

With a re-interview rate of 78 percent (6,922 households were interviewed in 2001), the rate of attrition in the sample turns out to be fairly high.[2] To the extent that it reflects household dissolution caused by the death of an adult member, attrition may lead to underreporting of prime-age mortality and bias estimations of the socio-economic consequences: households that dissolve are likely to be hit harder by adult mortality than those that remain intact. Using the same dataset, Chapoto and Jayne (2008) apply inverse probability weights to control for potential attrition bias. They find that this correction has little effect on the magnitude of the estimated impact of mortality, which suggests that attrition bias is not a major problem.

Although the surveys collected detailed information on mortality, epidemiological information on the cause of death is not available. For this reason, we follow the standard approach and use disease-related prime-age (15-59 years) mortality as a proxy for AIDS mortality. Comparing provincial HIV prevalence rates from antenatal clinics with adult mortality rates from the survey also used in this study, Chapoto and Jayne (2008) illustrate that the large majority of prime-age deaths are indeed likely to be AIDS-related.

Around ten percent of the households re-interviewed in 2004 experienced at least one prime-age adult death between the two survey years. We reduce this sample in various ways to arrive at treatment and control groups best suitable to identify the effects of adult mortality. First, households are only included if no member had died before the baseline survey was conducted, and if no chronically ill members were present in the household in 2001, because AIDS-related death is typically preceded by prolonged illness.[3] This correction is made to ensure that 2001 incomes are not contaminated in the sense of being lowered by AIDS-related adjustments. Second, we only consider the death of individuals who were recorded as household members in the baseline survey, eliminating from the sample the group of more than 150 households that experienced the death of a member who returned to the household to seek terminal care after the baseline survey. The main reason for doing so is that in households suffering the death of a long-

[2] It should be noted, however, that the re-interview rate rises to 89 percent if one excludes attrition caused by enumerators not returning to several enumeration areas (Chapoto and Jayne 2008).

[3] Overall, 548 households reported a prime-age death in the period 1996-2001. The total number of households with a chronically ill member was 580 in 2001.

term resident the deceased member contributed to initial income in 2001, whereas this was not the case for households incurring the death of a member who had not been a resident at the time of the baseline survey. As a consequence, the impact of prime-age mortality on welfare may be more severe for the former than for the latter (compare Chapoto and Jayne 2008). We end up with a treatment group of 221 households, who are fairly equally distributed across income quartiles (see Table 1). Among these households, 210 reported one dead member, nine reported two dead members, and two reported three dead members.

Another 465 households incurred the death of a prime-age adult between 1996 and 2001. They constitute the relevant sample for evaluating the medium-term impact of AIDS-related mortality. Again, afflicted households are fairly evenly spread over the income distribution.

Table 1: Number of households reporting prime-age mortality

	Total	Quartile 1	Quartile 2	Quartile 3	Quartile 4	Income change 2001-2004 (in ZMK)	
						Mean	SD
Period 2001-2004							
Any prime-age death	221	57	60	44	60	-26379	1059819
Death of head/spouse	124	28	35	28	33	-7852	1195724
Death of other members	105	30	26	18	31	-58859	833998
Death of male members	101	18	32	22	29	27930	866045
Death of female members	130	40	31	24	35	-75022	1157807
Period 1996-2001							
Any prime-age death	465	138	101	109	117	-36684	770837
Death of head/spouse	96	27	23	20	26	-140071	775322
Death of other members	375	112	82	89	92	-9133	764037
Death of male members	218	66	51	46	55	-90790	828569
Death of female members	277	77	61	69	70	1887	692275

Note: Mean 2001 per adult equivalent incomes of the four income quartiles are ZMK 91669, ZMK 217212, ZMK 422337 and ZMK 1347059, respectively.

When it comes to evaluating the impact of prime-age mortality on household welfare, either consumption or income can be employed as an indicator. While consumption is usually measured with a higher degree of precision, income changes may give a better indication of the household's future prospects and the sustainability of a household's adopted strategies to cope with the loss of an adult member. For instance, households may be able to temporarily smooth consumption by selling assets, but lower their income-generating capacity through such measures. In our case, consumption data are not available, but the survey offers detailed information on the income sources of a household. Total yearly income is computed as the sum of the value of agricultural production (including production for home consumption), livestock produce, and off-farm income including remittances. All monetary values are given in 2004 Zambian Kwacha (ZMK). Based on the total household income and the adult equivalent conversion factors provided by the Ministry of Planning, per-adult equivalent incomes are then computed. On average, yearly per-adult equivalent incomes in AIDS-afflicted households slightly fell over the period 2001-2004 (Table 1).[4]

As argued above, the income effect of a death depends on whether the deceased was a net consumer or a net producer for the household. However, the dataset does not allow identifying individual income and consumption levels. Instead, we try to account for the previous position of the deceased in the household by distinguishing between the deaths of a household head or his/her spouse and other adult members as well as between the deaths of male and female adults.[5] Note that the ratio of head's/spouse's and other members' death is different for the two periods because for the period 2001-2004 the reference was made to the head at the time of the death, while for the period 1996-2001 the reference was made to the head at the time of the interview in 2001.[6]

3. Methodology

In estimating the causal impact of prime-age mortality on household welfare two problems have to be taken into account. First, with sexual transmissions accounting for the vast bulk of HIV infections in Africa, AIDS is a behavioral disease. This may give rise to endogenous selection into treatment (prime-age mortality being the treatment). Second, to the extent that demographic coping leads to negative spillover effects on the control group of households without a deceased member, the causal effect of AIDS-related deaths will be underestimated.

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[4] At an exchange rate of roughly 5000 ZMK/US$, the drop in per-adult equivalent incomes amounts to about US$ 5 for households experiencing the death of a member between 2001 and 2004. This change in income, however, is not significantly different from zero.

[5] To keep the number of observations for each kind of prime-age death large enough, we abstain from further disaggregation.

[6] No deaths of heads should therefore be reported for the period 1996-2001, but some households nevertheless did so, probably confounding current with former (deceased) heads.

3.1 Estimation Method

As a solution to the problem of endogenous selection, we employ the DID-PSM estimator. While PSM removes the selection bias due to observed differences between afflicted and non-afflicted households, the DID estimator controls for time-invariant unobserved characteristics such as risk attitudes and sexual behavior. A number of different issues have previously been studied using the DID-PSM approach. Recent examples include the impact of foreign firm ownership on domestic wages (Görg et al. 2008) and the schooling effects of conditional cash transfers (Behrman et al. 2009).

The general idea of matching is to find a comparison group that shares the same characteristics as the treatment group, but did not receive treatment. However, there is a trade-off between the potentially wide range of characteristics one might want to match on and the chances of finding matches which would be identical across all characteristics. In addition, it is not clear how each characteristic should be weighted. Rosenbaum and Rubin (1983) suggest the use of PSM to solve this dimensionality problem. Performing PSM involves matching treated to non-treated households based on similarity of their predicted probability of receiving the treatment (their "propensity scores"):

(1) $P(X_i) = \Pr(D_i = 1 | X_i)$

where $D_i = 1$ refers to households i receiving a treatment, X_i is a vector of pre-exposure characteristics, and $0<P(X_i)<1$. PSM matches pairs on the basis of how close the propensity scores $P(X_i)$ are across the two samples of treated and non-treated observations. Through the use of the propensity scores, PSM balances the observable covariates X between the treatment and the control group and thus selects similar non-treated observations for each of the treated observations.

The assumption needed for the treatment effect to be identified is that selection into treatment is random conditional on the observable covariates X (conditional independence assumption). In other words, all factors which simultaneously determine treatment and outcomes of interest should be observed. If data quality is not sufficient to capture important components of X, then the presence of these unobserved characteristics implies that PSM will be biased.

To more convincingly argue that the assumption of conditional independence is satisfied, we combine PSM with the DID approach. Hence, we compare changes in the levels of the outcome variable rather than the levels themselves. This allows us to purge all time-invariant unobservables from the analysis. Yet, unobservable effects may still bias our results if they are time-varying and apply differently to treated and non-treated observations. This cannot be ruled out completely, but we are confident that our dataset is rich enough to control for important time-varying factors.

The resulting DID-PSM estimator for the average treatment effect on the treated households (ATT) can be expressed as

$$(2)\quad ATT = \sum_i \omega_i \left[(y_{i1} - y_{i0}) - \sum_{j \in c(p_i)} w_{ij} (y_{j1} - y_{j0}) \right]$$

where the ω_is are sample weights used to construct the mean impact PSM estimator for the afflicted households i, $(y_{i1} - y_{i0})$ is the difference in these households' income per adult equivalent before and after the death of a member, and $(y_{j1} - y_{j0})$ is the difference in income per adult equivalent for households j of the control group during the same period. The w_{ij}s are the weights applied in calculating the average outcome of the matched non-afflicted households. In our case, these weights are based on Kernel matching, which is a non-parametric approach that uses weighted averages of all households in the control group to construct the counterfactual outcome.[7] The average treatment effect is only defined in the region of common support $c(p_i)$, which implies that all observations whose propensity score is lower than the minimum and larger than the maximum of the opposite groups are excluded. Since our longitudinal dataset consists of just two waves, we can only evaluate absolute changes in per adult equivalent income between 2001 and 2004. Even in a very poor context like rural Zambia it is likely that these absolute changes depend on initial income levels. In absolute terms, richer households may incur higher fluctuations in income than their poorer counterparts. To avoid matching households with very different income levels, we enforce exact matches within income quartiles.

To estimate the propensity score for each observation in the treatment and control group we use the predicted values from a logit model. Only variables which have an effect on both treatment and outcome and are not affected by the treatment should be included. Building on the existing literature on the determinants of HIV infection, we identify a set of variables simultaneously affecting a household's probability to experience the death of a prime-age member and its income generation capacity. These variables are constructed using pre-treatment data from the baseline

[7] See Smith and Todd (2005) for a discussion of different matching methods.

survey. At the household level, we include variables capturing the demographic composition of the household, also accounting for the sex and educational levels of prime-age adults and the household's head (compare Hargreaves and Glynn 2002 and Chapoto and Jayne 2006). We also control for the importance of different income sources, as involvement in local non-farm activities or migration not only has an effect on income levels, but also increases the risk of infection with HIV (Ainsworth and Semali 1998 and Lurie et al. 2003). As a proxy for social capital we add a dummy indicating whether a household has a relation to the village head (compare Pronyk et al. 2008). The effect of household wealth is captured through land endowments and a broader asset index[8] (compare Gillespie et al. 2007). We also include a dummy for radio ownership to proxy access to information. At the community level, we control for the distance of the village to the district town and nearest tarred main road (compare Buvé et al. 2002 and Tanser et al. 2000). Besides, we add population density and rainfall levels and variability.[9] Finally, by introducing provincial dummies, we also control for the observed regional variety in HIV prevalence. Table A1 presents a complete list of the explanatory variables included in the logit model to estimate the propensity score.

A formal requirement for a reliable estimation with PSM is that, under the assumption of independence conditional on observables, the relevant covariates are balanced between the treatment group and the comparison group. Lack of balance points to a possible misspecification of the propensity score model. Hence, it is important to verify that the balancing condition is satisfied by the data. We perform three different balancing tests suggested in the literature.

The first balancing test (Sianesi 2004) re-estimates the propensity score on the matched sample, that is only on afflicted households and matched non-afflicted households, and compare the pseudo-R2 before and after matching. The pseudo-R2 indicates how well the regressors explain the probability of being affected by prime-age mortality. After matching there should be no systematic differences in the distribution of covariates between both groups and hence the pseudo-R2 should be fairly low. As shown in the upper part of Table A2, the pseudo-R2 indeed approaches zero after matching.

[8] The asset index is based on a principal component analysis of the following variables: Dummy for iron roof, dummy for brick walls, dummy for modern door, dummy for cement floor, and the value of agricultural productive assets.

[9] Community variables are taken from supplementary datasets: Distance and rainfall data for the period 1990-2004 were kindly provided by Michigan State University's Food Security Group. Data of population density come from the 2000 Census.

The second balancing test (Rosenbaum and Rubin 1985) calculates the standardized bias before and after matching. For each covariate it is defined as the difference in the means of the treated and (matched) control group as a percentage of the square root of the mean of variances in both groups. In the matched sample the standardized bias should be reduced to less than twenty percent. In our case, PSM reduces the mean bias by about 70 percent, bringing the remaining mean bias down to about five percent, which is far from the critical value of twenty percent (see lower part of Table A2).

The third balancing test (Dehejia and Wahba 2002) first divides the observations into strata based on the estimated propensity scores. These strata are chosen so that no statistically significant difference in the mean of the estimated propensity scores remains between the treatment and comparison group observations within each stratum. Then, t-tests are used to test within each stratum for mean differences in the regressors between the observations in the treatment and comparison group.[10] In our propensity score model, mean differences turn out to be insignificant across the board, suggesting that the balancing conditions are not violated.[11]

As a final point, it is illustrative to briefly compare PSM to other non-experimental methods of impact evaluation. Like PSM, OLS and IV estimations also rely on the conditional independence assumption. In the case of OLS, this is the assumption of exogeneity of treatment. The IV estimator in turn allows for the possibility of endogenous treatment, but rests on the challenging exclusion restriction. An advantage of PSM over these approaches is that it is a non-parametric method. Hence, it does not impose arbitrary assumptions of functional forms and error distributions. In addition, PSM also uses a different sample by confining matching to the region of common support thus dropping unmatched observations from the control group (compare Ravallion 2008).

3.2 Spillover Effects

A unique characteristic of the Zambian dataset is that it allows us to link demographic changes in the composition of households to AIDS-related mortality. The survey included specific questions on why a member (12 years or older) joined or left a household between the two waves, and on whether a household hosted orphans (11 years or younger). We then define the following three

[10] In case of remaining differences, Dehejia and Wahba (2002) suggest adding higher-order and interaction terms in the propensity score specification until such differences no longer emerge.
[11] Also compare Table A3, which shows simple t-tests for the matched sample within different income quartiles.

groups of individuals who may give rise to an AIDS-related demographic burden for households without a deceased member, thus reducing their per-adult equivalent income:

1. *New members* (12 years and older), who were not in the household in the first wave, but were so in the second wave; they joined the household for the following reasons: having lost parents/being fostered, having been widowed, returning to the household because of sickness[12], needing help without being sick, and marrying a household member[13]. Not included as AIDS-related cases are individuals who joined the household to help with activities, to work for the household, because of old age, and because of divorce or separation.

2. *Departed members* (12 years and older), who were a household member in the first wave, but no longer so in the second wave; they departed from the household to live with other relatives. Individuals who departed to find a job, to establish a new home, to enter marriage, and because of divorce or separation are not counted as AIDS-related cases.

3. *Orphans* (11 years and younger), who lost at least one parent and did not live with the remaining parent. Information for orphans is only available for the 2004 wave, i.e., we do not know whether they were already members of the household in the first (2001) wave.

Employing this demographic information, we check in various ways whether spillover effects can be observed in the data. First, based on Dzekedzeke and Fylkesnes (2006), who use DHS and antenatal care clinic data to estimate the HIV prevalence in 2001, we compare households without a deceased member in low-prevalence and high-prevalence provinces.[14] Specifically, we test whether the number of new and departed members is higher in regions with high HIV prevalence. Obtaining a significant difference across regions would not only point to spillovers but also suggest that the above definitions indeed provide a reliable proxy for AIDS-related demographic changes. Second, we examine whether households who are indirectly affected by

[12] Death due to AIDS is normally preceded by prolonged and severe illness. Given the transmission nature of HIV, it is likely that the partner of a deceased is also infected. Using the same dataset, Chapoto and Jayne (2006) provide evidence that this is the case in Zambia.

[13] Beegle and Krutikova (2008) show for Zambia that adult death affects the timing of marriage in affected households. In particular girls who lose their father marry at significantly younger ages. In addition, Ueyama and Yamauchi (2009) demonstrate for the case of Malawi that women's marriage age is lowered in general if the local marriage market is characterized by excess adult mortality.

[14] Low-prevalence provinces are Northern and Northwestern (8.3 and 9.0 percent, respectively), while the three provinces with the highest prevalence are Copperbelt, Lusaka, and Southern (19.9, 22.0, 17.6 percent, respectively).

AIDS through demographic changes fare worse in terms of income per capita than their unaffected counterparts. Finally, we control for spillover effects in the estimation of the impact of AIDS-related mortality on per adult equivalent incomes by excluding households from the control group if members departed or joined for AIDS-related reasons.

Of course, per adult equivalent incomes of households in the control group are not only subject to demographic spillovers. The level of total income of such households is also affected through economic spillovers. High adult mortality rates may alter communities' economic structure, e.g. through general equilibrium effects on local labor markets or the availability of land for cultivation. AIDS may also lower the resilience of communities' social networks to other shocks. However, the indirect nature of these effects renders them not only difficult to identify but also likely to be small. Using the same dataset, Jayne et al. (2006) find only minor effects of prime-age mortality on Zambian communities' crop output, mean income, and income per capita. We are thus confident that economic spillovers do not seriously impinge on the validity of our control group.

4. Results

4.1 Base Estimates

Before turning to the role of spillover effects, we first present PSM-DID estimates based on the unrefined control group (Table 2). This facilitates comparison with previous studies where spillovers were not taken into account. Note that the number of observations in Table 2 is lower than in Table 1 because a few cases are off the region of common support. Standard errors are computed using bootstrapping with 400 replications.[15] At the aggregate level, we find a positive but insignificant effect of prime-age mortality on household per adult equivalent income. Disaggregating impacts according to the gender and position of the deceased person leads to even higher standard errors of the estimated treatment effects, rendering it impossible to identify any pattern that would point to differences between net producers and net consumers.

[15] The reason for using bootstrapping is that conventional standard errors tend to be biased because the estimated variance of the treatment effect should also include the variance due to the estimated propensity score and the imputation of the common support. The validity of bootstrapped standard errors in matching estimators has been questioned by Abadie and Imbens (2008), but their critique does not apply to kernel-based matching employed here.

Table 2: Short-run income effects of recent prime-age mortality (2001-2004)

Treatment	Observations	ATT (in ZMK)	Standard error	p-value
Any prime-age death	217	90676	73840	0.22
Death of head/spouse	120	47278	112550	0.67
Death of other members	96	-32900	115592	0.78
Death of male members	98	64596	102804	0.53
Death of female members	125	-11234	107407	0.92

Chapoto and Jayne (2008) provide detailed evidence on the coping mechanisms that may lie behind this 'non-finding'. As concerns income coping, they show that the death of a prime-age male household head is associated with a modest fall in the value of cattle, while the death of other adult household members has no significant effect on cattle assets. After the death of male and female adults, the value of small animals declines by 36 percent and 18 percent, respectively. Selling small animals thus appears to be a quantitatively important coping strategy used by AIDS-afflicted households. In addition, demographic coping plays a role. While households experiencing the death of the head are partially able to replenish their household size by attracting additional members, the death of other members is associated with a more than one-person decline in household size due to a significant reduction in the numbers of boys and girls. According to Chapoto and Jayne (2008, p. 344), the latter suggests that "in many cases, the deceased adult's family lived with him or her while the sick person received terminal care at a relative's home (possibly the older parents) and then left after the person passed away." When re-estimating the relationship for the present sample that excludes sick persons seeking terminal care, we find a less than one-person decline in household size both for aggregate deaths (see first row in Table 3) and for all subgroups including other members. In combination, the attraction of new household members and the selling of productive assets may have prevented incomes from falling over the period 2001-2004.

Our core result is corroborated by Mather and Donovan (2008) for Mozambique, a neighboring country that provides a comparable setting of predominantly poor rural households. Mather and Donovan (2008) also find insignificant effects of prime-age mortality on rural households' per adult equivalent income irrespective of whether household heads or other members died, and identify a loss of assets and changes in household composition as major factors contributing to this outcome.

Two further studies investigating the welfare impact of prime-age mortality come to different conclusions. For the case of Indonesia, Grimm (forthcoming) shows that indirect coping effects more than offset the direct losses caused by the death of an adult household member, leading to overall gains in consumption per adult equivalent. Notably, surviving members of Indonesian households experiencing prime-age adult deaths are found to rely on increases in labor supply as one coping strategy, a scenario that is unlikely to happen in the rural African context. By contrast, Beegle et al. (2008) estimate for the Kagera region in Tanzania that within the first five years after the death of an adult per adult equivalent consumption of the surviving household members grew by about seven percent less than consumption levels of non-afflicted households. Nonetheless, on average consumption growth continued to be positive for AIDS-afflicted households for the period under consideration (2000-2004). Positive economic conditions may thus have enabled AIDS-afflicted households to uphold or even increase their consumption levels over time without resorting to desperation-led selling of assets.

4.2 Controlling for Spillover Effects

The demographic coping of AIDS-afflicted households may translate into significant spillover effects and thereby affect the welfare estimates reported above. We find that on average individuals (12 years and older) more frequently join and leave households without a deceased member when the households reside in regions with high HIV prevalence, but that these changes in the composition of households do not lead to significant welfare changes (Table 3). Having to care for orphans, by contrast, is estimated to lower household income per adult equivalent by roughly US$ 20 on average. Given the low income levels characterizing rural Zambia, this is a quantitatively important income drop of almost 20 percent. Overall, we thus obtain evidence pointing to the existence of considerable spillover effects.

Table 3: Spillover effects of recent prime-age mortality

a) Does the adult equivalent size of AIDS households change differently?

Treatment	Observations	ATT	Standard error	p-value
Any prime-age death	220	-0.43**	0.18	0.02

b) Do more persons (12 years and older) join households without a deceased member in high-prevalence regions?

Treatment	Observations	ATT	Standard error	p-value
High-prevalence region	500	0.08**	0.04	0.05

c) Do more persons (12 years and older) leave households without a deceased member in high-prevalence regions?

Treatment	Observations	ATT	Standard error	p-value
High-prevalence region	499	0.10*	0.05	0.06

d) Do households without a deceased member but with AIDS-related demographic changes fare worse in terms of income?

Treatment	Observations	ATT (in ZMK)	Standard error	p-value
New members (12 and older)	435	-43397	40850.84	0.29
Orphans (11 and younger)	248	-95182*	55014.26	0.08
Departed members (12 and older)	398	18210	50318.79	0.72

In a subsequent step, we re-run the DID-PSM regressions using only households without a joining or departing member as a control group. The results are shown in Table 4. For aggregate deaths, the estimated impact of AIDS-related mortality on household incomes turns negative when controlling for spillovers, but continues to be insignificant. Disaggregating households by the position or sex of the member who died again produces absolutely insignificant results.

Table 4: Short-run income effects of recent prime-age mortality (2001-2004) controlling for spillovers

Treatment	Observations	ATT (in ZMK)	Standard error	p-value
Any prime-age death	207	-67771	74522	0.36
Death of head/spouse	119	8376	115020	0.94
Death of other members	87	-80803	131160	0.54
Death of male members	93	-30852	100593	0.76
Death of female members	120	-52689	113779	0.64

Since we do not find evidence for significant short-run income effects, even after controlling for spillovers, we can also examine the medium-run income effects for the period 2001-2004 resulting from prime-age deaths that occurred before 2001. As noted above, the household survey also recorded the death of a member between 1996 and 2001. Our identifying assumption is that 2001 incomes are not contaminated by short-run AIDS-induced welfare losses. The results shown in Table 5 point to the absence of such effects, which is in line with what Beegle et al. (2008) found for the Kagera region in Tanzania. Beegle et al. (2008) suspect that over the medium run factors not related to AIDS increasingly blur any existing difference between afflicted and non-afflicted households.

Table 5: Medium-run income effects of less recent prime-age mortality (1996-2001) controlling for spillovers

Treatment	Observations	ATT (in ZMK)	Standard error	p-value
Any prime-age death	449	-29288	45954	0.52
Death of head/spouse	85	-122950	121992	0.31
Death of other members	359	5068	46239	0.91
Death of male members	203	-49586	61844	0.42
Death of female members	265	48548	52908	0.36

5. Conclusion

Finding no negative impact of prime-age mortality on the per-capita incomes of surviving household members may appear counterintuitive, in particular if it is the household head or the spouse who died. For the Zambian case, one might argue that by employing income as a welfare indicator the true impact is not easily identifiable due to possible measurement error, even though income generation was carefully captured in the household surveys. Yet, through income and demographic coping, which has been shown to exist on a significant scale, AIDS-afflicted households may well be able to preserve their per-capita income levels over the short to medium term as our analysis and a similar study for rural Mozambique suggest. This is not to deny the potentially negative impact of prime-age mortality on human capital formation and its transmission across generations (e.g. Bell et al. 2006). Besides, our analysis focuses on AIDS-related mortality but not morbidity. Yamano and Jayne (2005) and Linnemayr (forthcoming) present evidence that AIDS may already lower household welfare long before the infected household member dies. On a more speculative note, our finding could also reflect the specific context of a very poor rural economy with high subsistence shares, where in a Lewis-type fashion surplus labor on farms implies that no welfare losses occur when the household loses a member. In a similar vein, the distinction between net producers and net consumers may get blurred in such a setting because all household members tend to be involved in the same set of low-return activities.

As concerns policymaking, the finding that surviving household members are able to avoid a drop in per-capita incomes after a prime-age death does not render public support unnecessary if coping involves actions with severe long-run consequences, such as depleting the only assets a household owns or taking children out of school. Our analysis of spillover effects suggests that the targeting of AIDS-related public interventions should extend to households not experiencing a death but hosting orphans as they are shown to incur considerable reductions in per-capita incomes. Overall, since AIDS is unlikely to be a major contributor to low income levels in rural Zambia, efforts to tackle the HIV/AIDS epidemic should not play a major role in poverty-reduction strategies.

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Appendix

Table A1: Summary statistics

	Treated (n=221)		Control (n=3289)	
	Mean	SD	Mean	SD
No 0-14 years	3.11	2.36	2.96	2.02
No 15-59 male skilled	0.50	0.85	0.39	0.68
No 15-59 male unskilled	1.18	1.31	0.94	0.93
No 15-59 female skilled	0.38	0.79	0.21	0.58
No 15-59 female unskilled	1.51	1.23	1.17	0.84
No 60 years and older	0.40	0.68	0.24	0.52
No migrants	0.39	0.83	0.37	0.80
Male head	0.81	0.40	0.82	0.39
Skilled head	0.22	0.42	0.23	0.42
Relationship with village head	0.37	0.48	0.36	0.48
Crop income (%)	69.12	33.87	71.77	31.88
Self-employment income (%)	13.15	25.25	12.89	24.79
Remittances income (%)	3.74	11.08	3.15	9.72
Wage income (%)	10.58	24.75	9.14	23.56
Land per adult equivalent	0.72	0.89	0.83	1.46
Radio	0.38	0.49	0.37	0.48
Asset index	-0.13	1.45	-0.20	1.33
Distance to main road	26.83	39.80	24.89	35.30
Distance to district town	32.74	22.23	34.37	22.41
Population density	22.46	37.92	24.92	44.58
Mean rainfall	963.02	178.49	998.84	181.99
Rainfall variability	155.06	41.88	158.27	38.44
Central	0.15	0.36	0.10	0.30
Copperbelt	0.04	0.19	0.07	0.25
Eastern	0.21	0.41	0.22	0.42
Luapula	0.14	0.34	0.13	0.34
Lusaka	0.05	0.22	0.02	0.15
Northern	0.12	0.33	0.16	0.37
Northwestern	0.03	0.18	0.07	0.26
Southern	0.13	0.33	0.12	0.33
Western	0.14	0.34	0.10	0.30

Note: Treatment is any prime-age death between 2001 and 2004.

Table A2: Pseudo R2 before/after PSM and mean bias reduction

	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Pseudo R2 before	0.136	0.145	0.133	0.118
Pseudo R2 after	0.016	0.019	0.02	0.022
Mean bias reduction (%)	78.93	68.57	67.76	63.61
Mean bias after matching (%)	3.19	4.07	5.24	4.49

Table A3: T-tests for matched sample after PSM

2001 Characteristics	Quartile 1				Quartile 2				Quartile 3				Quartile 4			
	Mean (matched sample)		t-statistics	p-value	Mean (matched sample)		t-statistics	p-value	Mean (matched sample)		t-statistics	p-value	Mean (matched sample)		t-statistics	p-value
	Treated	Control			Treated	Control			Treated	Control			Treated	Control		
No 0-14 years	2.87	2.96	-0.24	0.82	2.97	3.02	-0.16	0.88	3.40	3.18	0.37	0.72	3.02	3.06	-0.10	0.92
No 15-59 male skilled	0.20	0.25	-0.52	0.60	0.36	0.34	0.10	0.92	0.53	0.52	0.09	0.93	0.82	0.83	-0.06	0.95
No 15-59 male unskilled	1.33	1.31	0.09	0.93	0.98	1.01	-0.14	0.89	1.33	1.05	0.93	0.35	0.93	1.02	-0.46	0.65
No 15-59 female skilled	0.18	0.16	0.27	0.79	0.44	0.40	0.25	0.80	0.37	0.35	0.10	0.92	0.43	0.58	-0.79	0.43
No 15-59 female unskilled	1.56	1.52	0.24	0.81	1.41	1.49	-0.49	0.63	1.58	1.33	0.80	0.43	1.33	1.40	-0.33	0.74
No 60 years and older	0.42	0.39	0.26	0.80	0.36	0.34	0.10	0.92	0.28	0.29	-0.08	0.94	0.45	0.40	0.37	0.72
No migrants	0.22	0.26	-0.35	0.73	0.36	0.36	-0.03	0.98	0.53	0.54	-0.02	0.99	0.48	0.49	-0.01	0.99
Male head	0.73	0.72	0.05	0.96	0.76	0.79	-0.35	0.73	0.88	0.87	0.15	0.88	0.85	0.87	-0.36	0.72
Skilled head	0.11	0.12	-0.14	0.89	0.20	0.18	0.34	0.73	0.23	0.25	-0.17	0.87	0.33	0.38	-0.48	0.63
Relationship with village head	0.44	0.43	0.10	0.92	0.36	0.39	-0.41	0.69	0.37	0.39	-0.17	0.87	0.30	0.30	0.05	0.96
Crop income (%)	81.05	81.94	-0.17	0.87	76.34	76.51	-0.03	0.97	71.63	70.23	0.20	0.84	48.10	48.01	0.01	0.99
Self-employment income (%)	3.38	3.81	-0.15	0.88	12.73	12.01	0.18	0.86	13.31	13.37	-0.01	0.99	22.52	24.38	-0.31	0.76
Remittances income (%)	7.34	7.13	0.06	0.95	2.74	3.02	-0.25	0.81	5.01	6.02	-0.36	0.72	0.77	0.94	-0.28	0.78
Wage income (%)	5.72	4.93	0.24	0.81	6.70	6.18	0.16	0.87	7.35	7.70	-0.08	0.94	21.86	21.29	0.09	0.93
Land per adult equivalent	0.36	0.38	-0.26	0.80	0.80	0.92	-0.42	0.68	0.76	0.79	-0.16	0.87	0.95	0.99	-0.16	0.88
Radio	0.13	0.14	-0.24	0.81	0.29	0.30	-0.15	0.88	0.47	0.42	0.43	0.67	0.63	0.64	-0.06	0.95
Asset index	0.63	-0.61	-0.14	0.89	-0.43	-0.41	-0.11	0.91	0.23	-0.28	0.19	0.85	0.69	0.85	-0.47	0.64
Distance to main road	30.70	29.72	0.12	0.90	23.64	24.15	-0.08	0.94	28.82	28.21	0.07	0.95	24.18	22.95	0.19	0.85
Distance to district town	32.68	32.87	-0.05	0.96	31.66	32.81	-0.27	0.79	35.46	34.40	0.23	0.82	32.02	31.64	0.10	0.92
Population density	18.75	19.67	-0.20	0.84	18.93	19.25	-0.06	0.95	20.21	21.18	-0.19	0.85	31.66	30.37	0.12	0.91
Mean rainfall	962.62	969.33	-0.20	0.84	1008.70	1000.80	0.23	0.82	968.56	978.38	-0.24	0.81	922.90	921.08	0.06	0.95
Rainfall variability	151.53	152.11	-0.07	0.94	150.67	149.79	0.12	0.90	150.24	151.91	-0.22	0.83	166.23	165.62	0.08	0.94
Central	0.05	0.06	-0.17	0.87	0.10	0.12	-0.24	0.81	0.21	0.19	0.26	0.80	0.23	0.19	0.52	0.61
Copperbelt	0.04	0.04	-0.18	0.86	0.02	0.02	-0.29	0.78	0.05	0.05	-0.03	0.98	0.05	0.05	-0.03	0.98
Eastern	0.20	0.21	-0.10	0.92	0.19	0.19	-0.04	0.97	0.21	0.21	-0.05	0.96	0.25	0.23	0.27	0.79
Luapula	0.16	0.17	-0.10	0.92	0.20	0.21	-0.07	0.94	0.14	0.14	0.04	0.97	0.05	0.06	-0.17	0.86
Lusaka	0.02	0.01	0.21	0.84	-	-	-	-	0.12	0.10	0.27	0.79	0.08	0.08	0.11	0.91
Northwestern	0.02	0.03	-0.44	0.66	0.03	0.04	-0.17	0.86	0.02	0.03	-0.28	0.78	0.05	0.04	0.24	0.81
Southern	0.16	0.16	0.05	0.96	0.07	0.09	-0.42	0.68	0.07	0.07	-0.06	0.95	0.18	0.21	-0.31	0.76
Western	0.20	0.18	0.23	0.82	0.19	0.15	0.55	0.58	0.07	0.07	-0.09	0.93	0.07	0.08	-0.36	0.72