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Community-level effects on infant and child mortality in Zambia, with special attention to HIV prevalence

Short title: Childhood mortality in Zambia

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Abstract

We estimate the effect of HIV prevalence and other community characteristics on childhood mortality in Zambia. Community HIV rates significantly elevate risk of child death but not infant death, indicating that own or parental infection matters more than indirect mechanisms like a high dependency ratio. Other community characteristics also affect infant mortality and child mortality in distinct ways. The proportion of women in the community completing primary school significantly augments infant survival, an effect that is in addition to the positive individual-level effect of the child's own mother being educated. Full immunisation also improves infant survival chances, but beyond infancy material resources appear more crucial. Attainable health interventions could reduce childhood mortality even more than reducing the national-level HIV prevalence rate one to two percentage points. While profound improvement in childhood survivorship is unrealistic in the context of a generalised HIV epidemic like Zambia's, significant progress is nonetheless possible.

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Childhood mortality improved dramatically in sub-Saharan Africa in the decades following World War II, but since the 1980s, childhood mortality levels plateaued in the region as a whole, and even increased in some countries (United Nations 2002). While the HIV/AIDS epidemic is undoubtedly a major factor influencing recent mortality trends, other contextual factors would have made further improvements difficult even in the absence of the epidemic. During the period since the 1980s, many countries in the region have experienced fiscal austerity, structural adjustment, declining GNP per head, declining food production per head, and/or declining primary school enrolments (Awumbila 1997; Eloundou-Enyegue et al. 2000; Mason 1993, National Research Council 1993).

To date, efforts to estimate the relative contribution of these various factors to mortality levels have been hampered by a lack of nationally representative data on the prevalence of HIV. Previously available HIV data have come from non-representative samples (e.g., pregnant women, sex workers, those voluntarily seeking testing) or from sentinel sites that have limited geographic reach. However, the most recent round of Demographic and Health Surveys (DHS) included HIV testing for nationally representative samples of reproductive age men and women in eight African countries. The data for Zambia and Mali have been released, and this paper focuses on Zambia because there is little sub-national variation in HIV prevalence in Mali given the overall low levels of infection in the country (1.7 per cent of reproductive age adults (CPS/MS et al. 2002) versus Zambia's 15.3 per cent).

Zambia is an East African country where mortality improvement had been profound between 1950 and 1985, with the infant mortality rate (IMR, deaths per 1000 live births) dropping from 150 to 98 (United Nations Population Division 2002). However, between 1985 and 2000, the IMR climbed back to 110. The Zambia Demographic and Health Surveys of 1992, 1996, and 2001/02 show the same trend as the United Nations data, with increasing

mortality between 1992 and 1996. Both sources also report that infant mortality has begun to decline again in the most recent years (ORC Macro 2004; United Nations Population Division 2002).

Zambia is therefore a country showing signs of recent infant mortality improvement despite a diverse set of challenging circumstances. We seek to compare the magnitude of benefits to current survival probabilities conferred by positive community characteristics like high average levels maternal education or widespread use of antenatal health care with the risk associated with living in areas with high HIV infection rates. High HIV prevalence would obviously contribute directly to childhood death through vertical transmission, but the survival chances of uninfected children in high prevalence communities might also be compromised if health care resources are consumed by the epidemic. Generalised effects might also pertain because mortality of reproductive-aged adults increases the dependency ratio and decreases labour available to sustain the economy.

We estimate how much better mortality levels would be in the absence of the HIV epidemic, recognising that community HIV rates capture both these direct and indirect effects. We also compare the anticipated gains from socio-economic and health care improvements with those that would be anticipated if infection rates were lowered.

Data and Methods

The data are from the 2001-2002 Zambia Demographic and Health Survey (DHS). The sampling frame comprised a list of Standard Enumeration Areas from the 2000 Zambian Population Census. Within clusters, the demographic composition of each household was ascertained by a household questionnaire, and interviews were attempted with all women aged 15-49. In 7,126 households, 7,658 women were interviewed. We take information on childhood mortality from the birth histories collected during the women's interviews.

Children of women who have died are thus excluded from the analysis, and the impact of the epidemic on childhood mortality consequently underestimated.

Blood samples for HIV testing were collected at the time of the individual interviews. In one-third of the 7,126 households, questionnaires were administered to all men aged 15-59 as well as reproductive-aged women. In these same households, all eligible men and women were asked to consent to HIV and syphilis testing. To ensure confidentiality, dried blood samples for the HIV testing were labelled in such a way that they could not be linked to individual identifiers. The age, sex, and sero-status of the person contributing the blood sample are available in the HIV dataset, as is the location in which the blood sample was collected: both province within the country and whether a rural or an urban area. These data, though limited, nonetheless provide reliable estimates of the HIV rates in areas that respondents to the individual questionnaires (for whom a wide variety of information is available) are living.

Statistical model

We model the risk of childhood death as a function of the HIV prevalence in the child's community. Since other community and individual variables contribute to health outcomes (Andes 1989; Desai and Alva 1998; Kravdal 2004; Sastry 1996), We employ multi-level logistic regression models. The probability of infant, child, and under-five death are estimated in two-level models in HLM, using the robust option. The outcome variable is dichotomous—death of the individual child—and all of the level-one (individual) coefficients are log odds of death. The individual-level outcome is given by

$$\log(P/1\text{-}P)_{ij}=b_{0j} + b_{1j}x_{i1j} + \ldots + b_{bj}x_{ibj} \quad (1)$$

where $\log(P/1\text{-}P)_{ij}$ is the log odds of death for individual i in cluster j. The grand mean of the log odds for cluster j is represented by b_{0j}, and deviations from that grand mean according to

individual characteristics (x_{ibj}'s) are given by the rest of the equation. The level-two equation uses the sampling cluster as the unit of analysis and predicts the grand mean as

$$b_{0j}=m_0+m_1G_{1j}+ \ldots +m_kG_{kj} + U_{0j} \quad (2)$$

where the m_k's are ordinary least squares coefficients on community characteristics (G_k's), and the cluster-level errors are represented by theU_{0j}'s. The level-two (community) characteristics then affect the log odds of childhood death through the individual-level intercept.

Community characteristics

We calculate HIV prevalence rates (per cent of cases HIV positive) in 18 regions: the rural and urban areas of each of nine provinces using sample weights that differ by province and sex; the 2001-2002 Zambia DHS was not self-weighting (Dzekedzeke and Mulenga 2003). There were 3,950 blood samples from the approximately 2,375 households where the men's questionnaire was administered, and therefore an average of 219 cases per province/residence region. However, because rates of urbanisation differ markedly across provinces, sample sizes vary across province/residence regions as well. Therefore, we closely inspected the estimates where sample size was fewer than 50 cases: the urban areas of Eastern, Luapala, and Western Provinces. The original HIV prevalence rate for urban Eastern Province was 20.4 per cent, but that seemed too low both because it was only 7.8 percentage points above the rural rate (when on average urban rates were 12.4 percentage points higher than rural rates), and because estimates from sentinel surveillance (Zambia Central Board of Health 2002, as reported by the US Census Bureau 2003) also indicated 20.4 per cent was lower than might be expected for all adults based on data for pregnant women (see comparisons in Appendix A). We adjusted the estimate by averaging it with that for urban areas of Lusaka Province. Of Eastern Province's three neighbours, Lusaka had the most similar HIV

prevalence rate among pregnant women in the surveillance data. Moreover, the estimates for pregnant women were lower in Lusaka than in Chipata (the urban surveillance site in Eastern Province), and they therefore ease concern that we might be inflating the urban Eastern rate by averaging it with the urban Lusaka rate. We also weight Eastern and Lusaka equally when taking the average, even though Eastern had 49 cases and Lusaka 345; the purpose again is to be conservative in my adjustment of the estimate. My adjusted rate indicates that 21.8 per cent in urban Eastern Province are HIV positive.

For urban Luapala Province with 46 cases in the HIV data, the estimate accords well with surveillance data, and we therefore left it unadjusted. For urban Western Province, the original estimate was 33.3 per cent (11/33): we pooled urban areas of Western Province with urban areas of neighbouring Southern Province to produce the new estimate of 25.4 per cent. We used Southern Province to adapt the Western urban rate because the Zambia Central Board of Health estimates from sentinel surveillance cites in Mongu (Western Province) and Livingston (Southern Province) differed by only 0.2 percentage points (US Census Bureau 2003), and because North-Western Province does not have any major urban centres. These adjustments to the HIV prevalence rates matter little in the estimation of the final models. For instance, in the discrete-time model the coefficient on the adjusted HIV rate was 4.47 ($p<0.006$), while the coefficient on the unadjusted rate was 4.55 ($p<0.005$).

The set of province/residence specific rates given in Table 1 do not capture variation within smaller communities, but they nonetheless provide some indication of how much HIV rates vary within the country. They range from 4.7 per cent to 26.1 per cent, with a mean of 16.9 per cent and a median of 17.4 per cent. The national HIV prevalence rate from these data of 15.3 per cent is lower than the average of the province/residence rates because Zambia is still more rural than urban, and HIV prevalence is higher in urban areas.

Table 1 about here

We include a number of other community level variables that measure socio-economic context and health care context. These are all measured at the cluster level, and there were 320 sampling clusters in the 2001-2002 Zambia DHS. The number of observations per cluster depends on the variable in question. For instance, educational attainment was based on an average of 24 women per cluster while per cent immunised relied living children aged 12-36 months at interview, an average of eleven per cluster. For all of the socio-economic and health care variables, cluster estimates were stabilised using estimates from the 18 regions based on province and residence. The relative weight given to the province/residence estimates versus the cluster estimates was determined by the distance between the number of observations in the cluster and 50. Where the cluster had more than 50 observations, we simply used the cluster estimate. Kravdal (2004) gives evidence supporting the appropriateness of basing community estimates on the small samples available for DHS clusters, but we nonetheless used only the province/residence estimates for variables where the number of observations in a cluster was below five.

The health care variables include pregnancy health care use, maternal nutrition, children's growth, and children's immunisation. The pregnancy health care use measure is the percentage of births in the past four years where the mother made at least one antenatal visit, used a skilled birth attendant, or delivered in a medical facility.[1] We measure maternal nutrition as the proportion of women who have given birth in the four years before survey (but who are not currently pregnant or less than three months postpartum) with a Body Mass Index (BMI) of less than 18.5. Children's growth is indicated by the proportion more than two standard deviations below the reference median of height-for-age (i.e., stunted). Both maternal and child nutrition measures could be affected by HIV prevalence, but the multivariate analysis provides an estimate of how much nutrition matters net of the epidemic.

Finally, children's immunisation is the proportion of children aged 12-36 months who have received all eight of the immunisations that the World Health Organization recommends be administered in the first year of life. These variables approximate the health care context of the community. It is not possible to include individual-level indicators of variables like stunting and immunisation as predictors of mortality since values are missing for deceased children. We also do not include breastfeeding as a health input. While a community variable could have been created as it was for stunting and immunisation to avoid the individual-level measurement problems in the face of mortality, breastfeeding in the first year of life is uniformly high in Zambia: only eight out of the 1372 living children under twelve months at interview were weaned.

The socio-economic indicators at the community level are urban residence, women's education, and household wealth. Each sampling cluster contained only either rural or urban households, and therefore urban residence is included as a dummy variable. Community education is the proportion of women that have completed primary school. Primary school lasts for seven years in Zambia, and most that are going to complete it have done so by age 15. Areas where this assumption is in error are ones with high rates of grade repetition, and underestimating the eventual completion rates nonetheless captures a negative aspect related to educational attainment in those areas. Estimates of the effects of community education would undoubtedly be higher if health care variables were omitted since use of modern health care services is one of the pathways through which maternal education affects childhood mortality (see, e.g., Caldwell 1986). Nevertheless, the community education variable measures the influence of other pathways. Both social influence of and social learning from other educated women in a community can improve child health practices (Kravdal 2004). High HIV prevalence could contribute to lower levels of education in the community since parental infection (and teacher mortality) make it harder to keep children in school (Grant

and Palmiere 2003), but the correlation between the two variables across the 18 province/residence areas is quite modest (-0.12). The wealth index based on housing quality and consumer durables owned by the household comes directly from DHS and is simply averaged for the cluster and for providence/residence areas. The distribution of both the community and individual-levels is presented in Table 2.

Table 2 about here

Individual Variables

The individual-level predictors of child's death also include socio-economic and biodemographic variables. The same wealth index that was averaged at the community level is also used for the specific household in which the child's mother was interviewed, provided that she was a usual resident of that household. We drop children whose mothers were visitors in the household at time of the interview. Mother's education is categorised as none, incomplete primary, complete primary, incomplete secondary, complete secondary, and higher: these categories are included as a continuous variable.[2]

Maternal age, child's birth order, length of the preceding birth interval, and child's sex are biodemographic variables with known relationships with mortality: We coded the dummy variables for these factors so that the reference category is lower risk and a "1" represents higher risk. Therefore, there are dummy variables for mothers under age 20 and over 35, for first born children as well as sixth or higher order children, children born less than 18 months after an older sibling, and boys.

Dependent variables

We perform separate estimates for infant, child, and under-five mortality. In each case, the outcome is dichotomous with living children coded "0" and dead children coded "1".

Therefore, higher coefficients represent high log-odds of childhood death. The period of observation is the four years before the survey. This cut-off was chosen because additional questions on the DHS woman's questionnaire pertain to children born in the last five years, and some births may be backwardly displaced in order to avoid interview questions. Children's ages are therefore least accurately reported if they were born about five years before the survey.

Infant deaths are deaths that occur before the first birthday, i.e., at less than twelve months of age. Children at risk of dying in infancy are those under age one four years before the survey and those born during the four years before the survey (n=6,594). That is, the risk set for this analysis is defined in exactly the same way as the standard infant mortality rate (IMR) where the numerator and denominator do not quite match (see following section on estimation issues). Children under twelve months of age and alive four years before survey have a left-censored mortality experience: they are selected into the observation set on the basis of not having died between birth and survey. Their counterparts with the same birth dates but who died before survey are not observed. However, those born during the year before survey have a right-censored infant mortality experience: they could still die under age one, but after the interview. Where fertility is constant, this undercounting and overcounting are of roughly the same magnitude. For the analysis of under-five mortality, all those contributing any person-months of observation (i.e., those aged 0 to 59 months at any point during the four years before the survey) are in the risk set (n=10,681), and we count all deaths under exact age five in the observation period.

The risk set for child mortality is defined in the same fashion as for the standard child mortality rate (CMR). Those over 12 but under 60 months four years before survey are initially at risk of child death, but drop out of the risk set when they reach 60 months of age. Those reaching twelve months of age in the four years before survey enter the risk set as of

their first birthday, and some of these are still at risk of child death at interview when the observation period ends. Sample size for the child mortality analysis is 10,164.

Estimation issues

We elected to use the standard definitions for infant, child, and under-five mortality when constructing my dependent variables in order to produce a set of easily interpretable estimates, but this could introduce problems where there has been change in mortality rates over time. This is less of a concern when analysing sub-national variation in mortality levels (as is done here) than it would be in analysing mortality trend. Nonetheless, even in a four-year time window some of the basic assumptions incorporated into the standard childhood mortality rates have not pertained in Zambia. Therefore, we also estimated discrete-time models with childhood death as the outcome variable and periods of exposure to childhood death on the right-hand side: neonatal (under one month, reference), postneotal (1-5 months), late postneonatal (6-11 months), early second year (12-17 months), late second year (18-23 months), third year (24-35 months), fourth year (36-47 months), and fifth year (48-59 months). All age ranges that could have been completed between four years before survey and survey were included except those in which the child's death occurred before the start of the age range. Neither deaths nor survivorship during age ranges that overlap the four-year time window were included, and each child could contribute multiple observation periods. In these models, interaction terms between key independent variables and periods of exposure measured age-specific effects of community characteristics. The results were remarkably similar to what is presented below, and therefore any bias introduced by using measures where the numerator and denominator are taken from different time periods seems to be minimal.

An additional issue is that estimates of community-level characteristics are even more likely to suffer from omitted variable bias than are those of individual-level characteristics, in part because appropriate controls are infrequently available or small sample size at the community level precludes implementing all theoretically desirable controls. With data from more than one point in time, community fixed effects can be controlled while still leaving enough degrees of freedom to estimate effects of some community characteristics. This is not an option for this study since earlier surveys in Zambia did not use the same sampling clusters. Therefore, we tested whether the estimated effects of cluster characteristics changed when a set of province/residence dummies was included to control for fixed effects among larger geographic units (following Kravdal 2004). These results are not presented below because such models necessarily omitted HIV that was measured at the province/residence level, but including province/residence fixed effects did not alter the significance of the other community characteristics.

Results

Descriptive statistics

Table 3 shows that childhood mortality rates are clearly higher where HIV rates are higher. For the purposes of this descriptive table, HIV rates were calculated not only on the basis of province and type of place of residence, but also the mother's age (grouped in ten-year intervals). If a province/residence/age group had fewer than 30 observations, either an adjacent age group (or groups) or men were included in the average. Forty-two per cent of children born during the four years before the 2001-2002 Zambia DHS had a mother belonging to a demographic group where HIV prevalence was less than 10 per cent: these are the lowest risk group shown in Table 3. The middle risk group is 10-20 per cent HIV positive

with 32 per cent of the sample, and the highest risk group comprises 26 per cent of the sample.

Table 3 about here

Despite the fact that mortality is higher where HIV rates are higher, other determinants of childhood mortality are more favourable to survival in the high HIV groups. Based on the data for infections (fever, diarrhoea, cough/rapid breathing) and nutritional status (child stunting and maternal BMI), lower mortality rates would otherwise have been predicted in areas with high HIV prevalence. Similarly, use of modern health care services is greater where HIV prevalence is higher, as are income and education. There are also fewer high-risk births by conventional indicators (birth order and preceding interval length) in high HIV areas. In sum, the epidemic is more advanced in areas that should otherwise have a mortality advantage: those that show evidence of socio-economic development in asset ownership, education, nutrition, use of health care, and fertility regulation. This is not surprising since the epidemic was first concentrated among relative mobile elites, but the disadvantage associated with being "more modern" has clearly persisted as the epidemic became widespread in Zambia. The effects of HIV on mortality levels are then likely to be much greater when controlling for socio-economic and health care context than is apparent in the bivariate relationship shown in the first rows of Table 3.

Multi-level models

The multivariate results show that HIV prevalence at the community level has a significant effect on child mortality, but that the effect on infant mortality is not statistically significant. While child mortality levels are lower than infant mortality levels since children are more vulnerable during infancy, the estimated impact of community HIV rates is of greater magnitude after infancy. This is consistent with other research showing that children in high

HIV areas are most likely to die during their second year of life (Hill et al. 2003). The coefficients for the community-level variables in Table 4 reflect estimated changes in the intercept in the individual-level logistic equations for childhood deaths. Therefore, since HIV prevalence is coded as proportion infected, the estimated effect of a ten percentage point increase in the HIV prevalence rate would move the intercept up by one tenth of the coefficient shown in Table 4. For child mortality, the intercept would increase 0.585, and therefore the odds child death are about 80 per cent greater ($e^{0.585}$=1.80) where HIV prevalence is ten percentage points higher. The actual range of HIV prevalence in the 18 areas of Zambia is from 0.047 to 0.261 (Table 2), and the odds of child death are over two and half times greater at the high end of that range than the low end. If all other variables were held at their means (or mode), then the chance of dying for the average child in the lowest HIV area would be 19 per thousand survivors to age one, while it would be 52 per thousand in the highest HIV area. These numbers are much smaller than the actual child mortality rates because the average child has a much lower probability of death than all children do: risk of death is concentrated among the most disadvantaged. The model predicts that if the area with the lowest HIV prevalence were populated solely with children with the highest mortality risk (children whose mothers have no education, whose households score low on the wealth index, etc.), that the child mortality rate would be 100 per 1000, whereas in the highest HIV areas these children would die at a rate of 269 per 1000.

Although the effect of HIV on infant mortality is not statistically significant, it is in the expected direction, and HIV does significantly elevate all deaths under age five (infancy included). The under-five mortality rate for the most disadvantaged children is predicted to increase from 95 per 1000 live births to 199 per thousand as HIV prevalence goes from 0.047 to 0.216.

Table 4 highlights other community-level predictors of childhood mortality besides HIV. Most notably, the probability of infant death is far lower in areas where greater proportions of children receive complete immunisation. Highest risk children in communities with low rates of immunisation (proportion of children fully vaccinated of 0.47) suffer 89 deaths per thousand more than children with the same individual characteristics in communities where all children are fully vaccinated. Higher community levels of education also augment infant survival chances, even net of health care inputs like immunisation and pregnancy care that would likely be used at higher rates by educated women. The estimated IMR is 103 in clusters where the lowest proportion of women are educated (0.17) compared to an IMR of 90 where almost all women have completed primary school (0.95). While this effect is not as large as that for immunisation, we stress that it is over and above the individual-level effect of education. Infants' chances of death decrease by 14 per cent if they have a mother who has completed primary school, and an additional 13 per cent decrease in the chance of infant death comes from living in communities where 95 per cent women have attained that level of schooling rather than 17 per cent as in the least educated communities.

Average levels of community wealth do not affect infant survival, though greater wealth augments child survival. Resources become more important as children make the transition from breastfeeding to the adult diet. A greater proportion of stunted children in the community also increases child mortality. Stunting is growth faltering, and occurs after long periods of nutritional deprivation (unlike wasting which represents acute nutritional crisis). Therefore, areas with much childhood stunting are those where many families cannot command adequate nutritional resources. Again, infants may be relatively protected from short food supplies because of breastfeeding, while slightly older children feel the brunt.

Discussion

The impact of HIV on national-level childhood mortality rates can also be crudely estimated from the results of the multi-level models. The actual child mortality rate and under-five mortality rate during the four years before the 2001/2002 Zambia DHS were 74 and 162, respectively (Table 3). In the absence of HIV (a reduction of 15.3 percentage points), child and under-five mortality could be as low as 30 and 83 per thousand. These numbers seem almost absurdly low by sub-Saharan African standards given that few countries have attained such low mortality (tiny island states like Cape Verde, Mauritius, Reunion, and Seychelles being exceptions; Population Reference Bureau 2004), but they are nonetheless believable. In comparison, Ghana is a West African state that enjoyed only a slight mortality advantage over Zambia in the early 1980s when Zambia's childhood mortality rates were at their lowest, but the national HIV prevalence in Ghana is only 2.2 per cent (GSS et al. 2004), and under-five mortality has fallen to 99 (United Nations 2002).

HIV levels are not going to fall to zero, and it is more instructive to compare the gains in childhood survivorship that could be expected given more modest drops in the infection rate compared with other interventions. The HIV/AIDS epidemic in Zambia is already generalised, and there are some indications that prevalence of infection may already have declined in the most recent years (Population Reference Bureau 2004; US Census Bureau 2003). Still, a one percentage point drop in the infection rate would be a remarkable achievement, and if it were accomplished the CMR would be expected to fall from 74 to 70. Achieving the same survivorship gain through reducing child stunting would require bringing down the per cent stunted from 46.1 per cent to 43.1 per cent.

Although the effect of community-level HIV prevalence was not found to be statistically significant in predicting infant mortality, the estimated effect is still large, and it is worth considering the relative weight of other interventions for infant survival as well. For

instance, a one percentage point drop in HIV prevalence would be expected to bring the IMR from 96 to 93. Only eight-tenths of a per cent more of Zambia's children would have to be fully immunised to effect similar improvement. The returns to primary schooling in the community are of lower magnitude, but considering that in the average community only 45 per cent of reproductive-aged women had completed primary school, there is much room for improvement. When the individual-level effects are considered in concert with the community-level effects, modest improvements in schooling could effect improvements in survivorship similar to those discussed here.

Conclusion

These estimates of the effect of HIV and other health care and socio-economic variables clearly identify factors that help explain sub-national variation in childhood mortality. The effects of HIV are sobering, while the positive effects of good health care practices and education provide some reason for encouragement. Although Zambia's mortality profile has been improving in recent years, it is clear that in the absence of HIV, the improvements could have been more dramatic. It will also be difficult to improve child health interventions in the regions where HIV is most prevalent since these are already the regions where other predictors of child survival are favourable (Table 3). Moreover, Zambia has experienced declining primary school enrolments (as evidenced by comparing cohorts in the 2001-2002 Zambia DHS), and these kinds of developments make it still harder to improve child survivorship.

It seems that HIV affects childhood mortality more strongly through direct than indirect mechanisms. Children born with the virus would likely survive their first year before succumbing to opportunistic infections, particularly if they were protected by extended breastfeeding as most Zambian children are.[3] Community HIV levels significantly increase

child mortality, while their estimated effect on infant mortality is smaller and not statistically significant. If indirect mechanisms like decreased production (from adult mortality) or deteriorating physical infrastructure (from diverted resources) were predominant causes of the effect of community HIV levels, the results would be more consistent across age of the child. This finding does not negate the importance of indirect mechanisms as much as it underscores the importance of reducing vertical transmission.

The current analysis highlights how other community characteristics also affect infant mortality and child mortality in distinct ways. Communities where maternal education is more common have lower infant mortality rates even when controlling for the individual-level effect of mother's own education, a result that points to the importance of social learning and social influence in spreading good health care practices. Use of preventative health care may operate in a similar fashion, though this is less clear because it could not be controlled at the individual level.

It is easy to call for greater access to food and other material resources given the findings that child mortality depends more on community characteristics like proportion stunted and mean wealth than on "simpler" inputs like vaccination and attended deliveries, but the HIV epidemic complicates advances in these areas. Labour supplies in agriculture and other productive industries are compromised by adult mortality. Better childhood nutrition and more household resources were estimated to improve survival chances even when controlling for HIV prevalence, but gains in these areas are difficult when the national economy is compromised by the epidemic.

Nonetheless, the bottom line is that Zambia could again achieve childhood mortality rates as low as in the early 1980s even without reducing the HIV prevalence rate. The upward trend in childhood death rates has already been reversed, and attainable interventions could make up further lost ground.

Notes

[1] There are larger numbers of missing cases on antenatal visits and birth attendant than on place of delivery. There is therefore measurement error on this variable in that if a woman delivered her child at home but did not answer the other questions, she is coded as not having used modern health care in relation to the pregnancy and birth, even though some of the information is missing. We chose to retain a composite pregnancy health care use variable despite this problem because more women availed themselves of antenatal care and used trained delivery attendants than delivered in medical facilities, and we did not want these modern health care inputs to go uncounted. Furthermore, delivery in a medical facility can be caused by problematic labor and thus, if used alone, could underestimate the contribution of health care use. When the models shown below were run using just medical facility births, the estimated survival-enhancing effects were weaker than for all forms of pregnancy health care.

[2] As there was no a priori reason for believing that the effect of education would be continuous across these categories, we also experimented with models that included the educational categories as a set of dummy variables. The effect of this coding change on the predicting probabilities of death was minimal, despite the fact that mother's education after completed secondary seemed to confer no additional survival advantage. We therefore present the more parsimonious model below.

[3] Children precluded from breastfeeding by their mother's death are not in the sample which was based on interviews with women.

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Appendix A

HIV prevalence rates in urban areas of Zambia 2001-2002

Province	Demographic and Health Survey: representative sample of women 15-49 and men 15-59	Central Board of Health: sentinel surveillance of pregnant women
Central	26.1	28.8
Copperbelt	21.7	22.7
Eastern	20.4	27.2
Luapala	21.6	22.2
Lusaka	23.1	27.2
Northern	24.8	18.4
North-Western	18.1	13.5[a]
Southern	22.1	31.6
Western	33.3	31.8

Note: the surveillance data were obtained from the US Census Bureau's AIDS Surveillance database

[a] Surveillance site is in a semi-urban area.

Table 1.
Per cent HIV positive by province and type of residence, Zambia 2001-2002

Province	Urban	Rural	Total
Central	26.1	11.8	15.1
Copperbelt	21.7	11.0	19.6
Eastern	21.7[a]	12.6	13.6
Luapala	21.6	9.4	11.1
Lusaka	23.1	16.7	21.8
Northern	24.8	4.7	8.0
North-Western	18.1	7.7	9.2
Southern	22.1	15.9	17.3
Western	25.4[a]	10.2	13.0
All Provinces	22.7	10.7	15.3

Source: Zambia Demographic and Health Survey 2001-2002, weighted data

[a] Indicates adjusted percentage because original HIV prevalence estimate was based on fewer than 50 cases. Details of the adjustments are in the data and methods section.

Table 2.
Variables in multi-level models for childhood mortality in Zambia, 1997-2002

Outcome	Mean[a]	Standard deviation	Minimum	Maximum
Individual-level characteristics				
Mother's education	1.49	1.08	0	5
Wealth index	-0.02	0.96	-0.73	3.98
First born	0.21		0	1
Sixth or higher order	0.24		0	1
Preceding interval <18 mo	0.05		0	1
Mother under 20	0.21		0	1
Mother over 35	0.13		0	1
Child is a boy	0.50		0	1
Community-level characteristics				
HIV prevalence	0.14	0.07	0.05	0.26
Urban	0.31		0	1
Pregnancy health care use	0.85	0.08	0.66	0.99
Complete immunization	0.71	0.08	0.47	1
Stunting	0.45	0.10	0.17	0.67
Completed primary school	0.45	0.21	0.17	0.95
Wealth	0.16	0.87	-0.64	2.33
Low BMI	0.16	0.05	0.05	0.27

Source: as for Table 1. All births in the four years before the 2001-2002 survey are included.

[a] Means for the individual characteristics are given for the sample at risk of under-five death.

Table 3.
Mortality and mortality determinants by HIV rates, Zambia 1997-2002

Indicator	National	<10 per cent HIV positive	10-20 per cent HIV positive	>20 per cent HIV positive
Childhood mortality (4 years before 2001/02 ZDHS)				
Under-5 mortality rate (per 1000 live births)	162	143	165	189
Infant mortality rate (per 1000 live births)	96	88	98	105
Child mortality rate (per 1000 survivors to age 1)	74	60	75	95
Maternal and child health indicators				
Per cent of children 6-35 mo with fever in last 2 weeks	54.1	56.2	56.2	47.7
Per cent of children 6-35 mo with diarrhea in last 2 weeks	30.2	30.6	29.7	30.3
Per cent of children < 12 mo with cough/rapid breathing	18.6	18.9	18.2	18.5
Per cent of children under 4 years stunted	46.1	47.0	49.8	39.8
Per cent of births to women with BMI<18.5	13.2	14.3	14.0	10.5
Use of MCH services				
Per cent of children 12-23 mo received DPT1 vaccination	94.3	92.4	96.2	95.1
Per cent of children 12-23 mo received DPT3 vaccination	79.5	75.5	81.8	83.5
Per cent of children 12-23 mo received measles vaccination	84.4	80.7	88.2	86.0
Per cent of children 12-35 mo fully vaccinated	70.8	68.1	71.3	74.7
Per cent of births with antenatal care	70.1	69.1	70.1	71.8
Per cent of births delivered by a skilled attendant	53.4	50.6	51.3	60.8
Per cent of births delivered in medical facility	40.2	34.6	36.1	54.6
Socioeconomic context				
Per cent of births to mothers with no education	15.5	15.2	21.7	8.2
Per cent of births to mothers with primary education	65.0	69.6	62.6	60.4
Per cent of births to mothers with secondary+ education	19.5	15.1	15.7	31.5
Per cent of births to households in lowest wealth quintile	25.3	23.8	32.6	18.6
Per cent of births to households in highest wealth quintile	10.9	4.7	9.1	23.4
Biodemographic context				
Per cent of births that are first order	21.5	30.2	20.6	8.4
Per cent of births that are sixth and higher order	23.1	16.6	33.4	21.2
Per cent of births following interval of <18 months	4.5	4.7	4.6	4.3

Source: As for Table 1.

Table 4.
Population averaged models with robust standard errors, Zambia 1997-2002

Outcome	Infant death	Child death	Under-five death
Individual-level characteristics: logistic coefficients			
Mother's education	-0.076	-0.137*	-0.099*
Wealth index	-0.246	-0.294**	-0.285**
First born	0.332**	0.113	0.241*
Sixth or higher order	-0.241	-0.256	-0.280**
Preceding interval <18 mo	0.934***	0.121	0.551***
Mother under 20	-0.096	-0.130	-0.117
Mother over 35	0.275	0.018	0.189
Child is a boy	0.019	0.449***	0.200**
Community-level characteristics: OLS coefficients predicting individual-level intercept			
Intercept	-2.507***	-3.353***	-2.447***
HIV prevalence	2.990	5.852*	4.369**
Urban	0.161	-0.116	0.057
Pregnancy health care use	-0.564	-2.836	-1.934
Complete immunization	-3.764***	-0.544	-2.431**
Stunting	-0.366	1.966*	0.551
Completed primary school	-0.172***	1.485	0.444
Wealth	0.182	0.118***	0.153
Low BMI	1.648	2.260	1.914

* significant at $p<0.05$; ** significant at $p<0.01$; *** significant at $p<0.001$

Source: As for Table 1.