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THE EFFECTS OF HIV/AIDS ON FAMILY ORGANIZATION IN AFRICA

ALBERTO PALLONI
Center for Demography and Ecology
University of Wisconsin

YEAN JU LEE
Population Studies Center
University of Chicago

LUIS LAMAS (†)
Facultade de Ciencias Económicas e Empresariais
Universidade de Santiago de Compostela

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Abstract

In this article we examine the demographic effects of the spread of HIV/AIDS in countries of Central Africa. Our main goal is to show that the spread of the epidemic will have important effects on mortality and fertility but also on patterns of family organization. To understand well the nature of the impact on the family and on the capacity that these units will have to react and accomodate, it is necessary not just to gauge the effects on mortality but also on health status of adults. Even though the exact adaptations that will evolve as the epidemic spreads are difficult to predict, it is possible and useful to identify key areas where policies may be deployed with some efficay.

1. INTRODUCTION

The epidemiological characteristics of HIV/AIDS in countries where the epidemic is transmitted mainly through heterosexual contact are quite different from those found in societies where it is driven by homosexual contacts or ntravenous drug (IV) use. Indeed so sharp is the contrast that it has led to the adoption and widespread use of a classification of patterns of HIV/AIDS (Chin *et al.*, 1988). Equally important but much less studied is the fact that diversity in the pattern of the disease must necessarily be associated with non-trivial differences in the outcomes for individuals, families, and other social institutions. Although the exact mechanisms that lead to this heterogeneity may not always be transparent, one would indeed expect that at least some of the effects of HIV/AIDS in societies where its propagation is confined among homosexual men will differ from those where the virus is spread among adult females and males alike. Nowhere will these differences be more salient and visible than on family relations whose routine reproduction is more likely to be impaired when HIV/AIDS is broadly transmitted through heterosexual contacts.

Faced wiht a direct threat to the health and survival of some or all of its members, families

may choose from amongst a limited repertoire of coping strategies. Some of the adaptations may partially muffle damaging consequences of the epidemic without simultaneously undermining essential features of the family. Others, however, may do so only at the price of eroding the very foundation of familial relations as they are known today. Variability in the efficiency of adaptive strategies is in all likelihood related to the magnitude of the effects, the nature of the family organization, and the timeliness and adequacy of social interventions implemented by national governments or international relief organizations.

In this paper we explore the nature and assess the magnitude of selected effects of the HIV/AIDS epidemic on African families. We are only tangentially concerned wiht the most feasible or even the most likely adaptive strategies to accommodate to the new conditions. We are mainly interested in the *nature* of the stress exerted on the familiy organization and its day-to-day functioning and avoid forecasts about social and economic transformations that could reduce, alter or remove the stress. Just as social institutions and family organizations ultimately determine the pattern of heterosexually transmitted HIV/AIDS (Palloni and Jones, 1990) so do they strongly influence both the precise manner in which effects are experienced by individuals and social groups and the range of feasible adaptive strategies. Thus, to assess the nature of the stress created by HIV/AIDS —and more so to anticipate adaptations— it is necessary to differentiate the potential impact of *apparently* similar stresses on different type of families and social organizations. The conundrum is not new in demographic analysis and has been periodically raised in the literature on population crisis (Livi-Bacci, 1978). In section II of the paper we define a historical context for the discussion and briefly review the limitations of our knowledge about the social and economic impact of epidemics during pre-industrial times. In Section III we apply alternative procedures to evaluate the magnitude of the effects of mortality excesses due to HIV/AIDS under conditions approximating those found in Africa. We also examine the direction and magnitude of effects that are only indirectly related to excess mortality. In Section IV we assess health consequences that have been rarely highlighted in the literature but that, we argue, are of potentially formidable significance. In Section V we summarize our results and suggest a simple framework for the study of effects.

2. WHAT WE DO AND NOT KNOW ABOUT THE IMPACT OF PAST EPIDEMICS ON THE FAMILY

2.1. MACRO AND MICROLEVEL EFFECTS

By and large, the literature on the impact of epidemics and famines is virtually devoid of systematic analysis of the effects on family organization. To be sure, historians and demographic historians alike have gathered a fair amount of anecdotal evidence about the effects of recurrent crises on the daily lives of individuals and on the social morphology of pre-industrial villages and communities. In their rich study of Whickham for example, Wrightson and Levine (1989) combine historical documents and parish reconstitution to examine the cascading effects of excess mortality on family members and inheritance practices:

> "Christopher Arnold was buried on 24 July 1587 at the outset of the mortality crisis. In the normal course of events his holding would have passed to his son Richard, perhaps with a life interest to Christopher's widow Margaret. Richard, however, was buried on 27 July after making a deathbed will naming his brother Thomas and sister Margaret as principal heirs of his goods. His mother Margaret was buried on 3 August; his brother Thomas on 19 August. Ultimately, the holding passed to a third brother, Nicholas."

Although somewhat informal, their findings suggest that, contrary to expectations, the "terrible, repeated crisis mortality in Whickham at the turn of the sixteenth and seventeenth centuries (...) caused no dislocation on landholding patterns" (Wrightson and Levine, 1989). Their work is somewhat exceptional since in

general there have been only timid attempts to derive testable hypotheses and to minimally quantify the available evidence relating crisis mortality and the day-to-day operation of social relations. This is a very difficult task for, as already anticipated by Appleby, it entails the double burden of painstaking data collection at the local level and the application of sophisticated demographic tools (Appleby, 1977).

The effects that we have successfully examined are those directly related to global excess mortality, deficits in fertility, and increased regional displacement of individuals. In the process we have cultivated a strong fascination for homeostatic dynamics and long-run adjustments but have gathered weak leads about immediate reactions and the deployment of short-term coping strategies. Rarely has the analysis descended to disaggregated levels to, for example, identify the mechanism whereby raised levels of mortality translate into higher likelihood of fusion, fission, or outright disappearance of family units. We have only cursory knowledge about exactly how mortality, worsening health conditions or —at the more ethereal level of ideological representations— the socially constructed imagery of the disease affected the relations among family members, disrupted the flow of obligations and exchanges between families, eroded (or reinforced) prevailing systems of inheritance and property ownership, or even undermined the stability of the political and legal institutions[1].

Economic outcomes are also dealt with in general terms. While we know a great deal about the aggregate effects on prices and real wages and how these were associated with short and long run responses in the regimes of nuptiality, fertility and migration (Schofield, 1989; Weir, 1989; Dupaquier, 1989; Wrigley and Schofield, 1981), we know little about the details of transformations in the structure of individual opportunities and the labor market, differentials by social groups in the deterioration of standards of living, the changes in the practices of child and female labor force participation, and the re-organization of the division of labor in the domestic domain that mitigated the consequences of acute scarcity.

Finally, we have been able to trace some of the effects of epidemics on the ideological representations, political institutions and public health organizations. Historians have carefully examined social and political mobilization to implement health interventions (Cipolla, 1976; Bardet *et al.*, 1988; Slack, 1989; Biraben, 1975), have produced important insights about the nature of representations that provided palatable explanations of the disastrous events (Bardet *et al.*, 1988; Gilman, 1988; Biraben, 1975) and have even explored the possible connection between epidemics and the stability of political regimes (Evans, 1989).

We have been less successful in the description of less aggregate processes, those that cannot be readily understood or even discerned with recourse to general indicators. Except in cases where the epidemics wiped out entire family units, mortality excesses would have translated into higher levels of orphanhood, widowhood, and in household arrangements that could not be demographically sustained in the absence of fusion or some other adaptive change. Our analytic tools have enabled us to assess the long-term effects of changes on mortality levels (and patterns) on the levels of orphanhood, widowhood, and (feasible) household arrangements. But we know a lot less about the concrete impact of *sudden* swings in mortality and about how they paralyzed the domestic economy, weakened support systems for children and elders, disrupted the socialization and training functions of families, and altered or even temporarily suspended fundamental exchange relations among members of the same families and between families. By disabling a large and selected contingent of adults, the deterioration of the health of the population that accompanied an epidemic must have been influential in slowing down or stopping altogether the production of goods and services, displacing and idling workers, weakening local commercial and trade networks. How did individuals and families cope with these massive transformations that, for the most part, occurred suddenly, with little or no warning, and were recurrent and of variable duration?

In his study of the demographic effects of epidemics Livi-Bacci (1978) moved beyond a pure aggregate assessment of the consequences

of an epidemic and proposed to examine the effects of epidemic mortality on the distribution of families by size (see also Schofield, 1977)[2]. Live-Bacci's (and Schofield's) effort points in the direction that we would like to go in the analysis of HIV/AIDS but it also reflects the limitations that we face when dealing with these issues. To render his procedure more tractable, Livi-Bacci and Schofield introduced simplifying assumptions that are somewhat paralyzing if applied to the case of HIV/AIDS. First, the assumption was made that the relative excess of mortality was age-invariant (neutral) and, second, they represented family heterogeneity by size differences and not by composition by generations, sex, or kinship. Although the first assumption is probably harmless for the characterization of the impact of epidemics in the past and the second at worst only reduces the range of possible inferences, neither of them is adequate to capture the family consequences of the very selected mortality excess that accompany HIV/AIDS in the complex African social context. Neither can the increases in mortality be neutral, nor can the age, sex, and kin composition of families be immaterial since the HIV-/AIDS epidemic is mainly transmitted through heterosexual contact among age-selective groups. Furthermore, because HIV/AIDS is *selective with respect to ethnic group, class and region*, the effects on features such as the size distribution of families cannot be assessed in general but, instead, must be appropriately stratified.

2.2. HIV, THE PLAGUE OF THE XXth CENTURY?

The increases in the levels of mortality caused by the plague or other epidemics in pre-industrial societies varied significantly from place to place. However, sudden upward shifts of 20 to 30 percent were not uncommon. From what we currently know about the epidemic, it is unlikely that in the short run HIV/AIDS will inflate total mortality levels that much (Bongaarts, 1989; Palloni and Lamas, 1989; Auvert, 1989; Bulatao, 1989; Stanley *et al.*, 1989). Better-known diseases entreched among African population for long periods of time, such as malaria, tuberculosis, and schistosomiasis, already exert powerful disabling effects and have, at least for now, more potent, though also more neglected, socio-economic consequences than HIV/AIDS[3]. In view of these observations, one might well ask why should any special effort be devoted to assess the social and economic consequences of HIV/AIDS rather than that of other, better-know diseases or, alternatively, why aren't the effects of these diseases rather than those of HIV/AIDS likened to the impact of pre-industrial epidemics. The answer, we argue, is that the unique nature of HIV/AIDS, its natural history, epidemiological characteristics, and the social foundations of its transmission mechanisms are likely to have unprecedented repercussions on social organizations.

First, a crucial distinction between HIV-/AIDS and diseases that struck in the past is the age and sex differentials. The threat of the plague invoked by William Hendley's as one that brings "...Death [that will come] into our Windows, and enter into our Palaces, and cut off our Children from without, and the Young Men from the Streets..." (Text from William Hendley's **Loimologia Sacra**, 1721 cited in Slack (1989)) is a chilling description of an even-handed scourge that was recurrent in pre-industrial times. It is also a fair statement of the age-specific incidence of the plague which, as remarked by Pollitzer (1954) and Hirst (1953) and as confirmed empirically among others by M.F. and T.H. Hollingswort (1971) in their study of a London parish, affected mainly adolescents and young adults but spared the very young and the very old. In contrast, HIV strikes infants as well as adolescents and young adults. Second, although some epidemics were recurrent, the duration of the most frequent epidemics of the past never exceeded one or two years and they were more commonly born and exhausted within the span of a season. Instead, the prevalence HIV/AIDS cannot reach its peak until at least ten years after its onset and may actively linger for much longer before its incidence slows down significantly[4]. The peculiarly long incubation period that characterizes HIV's *natural history* not only explains its protracted sojourn in social groups but, more importantly, induces social and economic effects that could not possibly have occurred if the time

period elapsed between contraction of infection and final resolution was compressed down to a few weeks[5]. Third, the ravages of the plague, typhus, yellow fever, cholera, and even tuberculosis and smallpox, were partially if not totally blind with respect to social class and could strike the population fairly generally. Abrate's description of the 1630 plague in Carmagnola, a village in Piemonte, fits well with a general pattern:

> "(thus)... one can say that the plague struck absolutely at random, without respect for the rich and powerful that stayed behind. The true 'Equalizer' that numbers the graves. The case of the richest family in the Community, the well remembered Signor Ottavio Maghino is a good one: death not only took him but also his wife, his eighteen year old daughter and two servants..."
>
> (Abrate, 1972, p. 87)

Or, as verified by Gottfried (1978) in London during the epidemics in the fifteenth century:

> "The London elite were among the wealthiest, oldest, most frequently married and remarried... they had the highest replacement ratios of any urban body, yet on average, from 1430 to 1480 even they failed to reach a replacement level ratio equal to 1.00..."
>
> (Gottfried, 1978, p. 195)

Instead, HIV/AIDS in Africa surreptitiously invades social groups and communities when and if relations between men and women are organized according to some norms, but it spares communities where the organizing principles are different (Palloni and Jones, 1990). The great divide between 'high and low risk' communities could but need not coincide with social strata cemented on wealth, education, or the monopoly of authority and bureaucratic control. Thus, the inquiry about social and economic consequences of HIV/AIDS risks being a sterile one if it is neutral and posited only at the most aggregate levels.

Finally, although the plague, cholera and other epidemics that afflicted pre-industrial societies were accompanied by large increases in mortality, few if any of them were characterized by an absolute *individual* level of lethality as high as that of AIDS. The plague was about the most feared of these diseases but the probability of surviving it was far from nil. In fact, although the septicemic and pneumonic forms led to inevitable death, the other variations had survival probabilities between .40 and .60 and individuals frequently escaped death, albeit only after prolonged and somewhat risky periods of convalescence (Abrate, 1972; Gottfried, 1978). Under our current level of knowledge and technology, once AIDS sets in it leads to inevitable death. The only window of escape for an individual who contracts the virus is the small but nevertheless non-zero probability of never developing AIDS.

In summary, there are reasons to believe that HIV/AIDS may have effects on social organizations that are equally if not more powerful than the ones associated with diseases that are now endemic in Africa or those that from time to time castigated pre-industrial societies.

3. THE HIV/AIDS EPIDEMIC AND ITS EFFECTS ON THE FAMILY ORGANIZATION

Social scientists and epidemologists alike have already alerted us to some of the most visible consequences of the HIV/AIDS epidemic. With some important exceptions (Carballo and Carael, 1988; Fleming *et al.*, 1988; Miller and Rockwell, 1988; Rockwell, 1988; Torrey *et al.*, 1988; Brokensha, 1988), the focus has been almost exclusively on the potentially selective increase in (total) adult mortality and morbidity. A widespread and protracted presence of HIV-/AIDS will surely lead to non-trivial mortality increases, particularly among adults and young children. A more insidious consequence is that such increases may not occur across the board but could have a selective impact on social strata, partially or totally depleting the elites from which political leaders, intellectuals, professionals, and government administrators

are drawn. If so, HIV/AIDS may have economic and political implications that are even more devastating than those commonly atributed to the ravages inflicted by epidemics in the past.

A caveat, however, is in order. Although the claim that members of the elite will suffer a greater toll than the rest of the population may have some empirical validity, it has not been convicingly and unequivocally demonstrated due to lack of adequate information, free from selection effects. But even if it were empirically verified, it does not follow that the *prospective* course of the epidemic will continue to reproduce such class-selective impact. Indeed, what we know about the social conditions that facilitate an environment for the transmission of the disease, leads to a different conclusion (Caldwell and Caldwell, 1989; Larson, 1989; Palloni and Jones, 1990). When evaluating the likely consequences of HIV/AIDS, there is a danger of taking *current conditions* as a flawless reflection of conditions that will prevail in the future. If it exists at all, the class differential could turn out to be a purely transient feature of the epidemic, a result of differentials in the timing of the introduction of HIV among various social groups that will eventually fade away and not the outcome of different patterns of behavior that fuel different epidemics.

The HIV/AIDS epidemic will also have more subtle consequences for individuals and families than those implied by group-selective increases in mortality. First, in regimes where the HIV/AIDS epidemic is transmitted heterosexually, chances are that if one member of a relatively steady union or couple contracts the infection, so will the other and some of his (her) closest kin[6]. However, young children and the elderly who co-reside in the same household unit will not necessarily be exposed to increased risks merely as a secondary consequence of the infection of one or both members of the adult couple (s). There is a unique asymmetry in the spread of HIV/AIDS which spares some members of a family while threatening directly the health of others[7]. Also, as we show below, the asymmetry of health consequences results in important constraints on feasible household living arrangements. Second, unlike the illnesses that were most frequently transmitted and that devastated pre-industrial societies, HIV/AIDS leads to a gradual rather than sudden deterioration of the health of the infected individual. In the absence of behavioral modifications, a relatively long incubation period not only raises the potential for transmission of the virus but it also increases the economic, social, and psychic costs for those who are infected and for the members of their inmediate families. This is a unique feature of HIV/AIDS that, we argue, in combination with environments exposed to a high density of pathogens, is likely to completely overshadow the more easily observable increases in mortality. Third, the presence of HIV/AIDS has potentially important indirect effects on the health of women and children and may involve changes in fertility, breastfeeding, and families coping strategies.

We now assess the magnitude of some of these effects. Since our estimation procedures invoke assumptions about unknown quantities, in most cases we avoid point estimates and instead provide a *range* that corresponds to plausible scenarios for the epidemic in Africa. Even these ranges, however, must be taken as no more than illustrative markers of the scope of the actual effects.

3.1. ADULT MORTALITY EXCESSES AND THEIR SEQUELAE: ORPHANHOOD, WIDOWHOOD AND IMPROVISED CO-RESIDENCE ARRANGEMENTS

When HIV is mainly transmitted via heterosexual contact, the maximum levels of seroprevalence are generally found among young adults and very young children (Quinn *et al.*, 1986). In the absence of countervailing influences, the maximum mortality increase will invariably affect the same population, but at slightly older ages. The magnitude and age-patterns of increases in adult mortality will have three distinct consequences.

3.1.1. Orphanhood.

The levels of paternal and maternal orphanhood at young ages (between 0 and 10 or 15) will rise reflecting the increased mortality of parents. Estimates of the expected increase in the levels of orphanhood in Africa in general

and at local levels have been published in a few studies (Preble, 1990; Hunter, 1989). Although useful, these estimates are not calculated within a general framework for the simultaneous evaluation of several consequences for the family organization. Our estimates, which are based on a combination of observed statistics and results obtained from models for the spread of HIV/AIDS, enable us to assess the effects of the epidemic on multiple aspects of the family simultaneously. To calculate expected levels of orphanhood we use two alternative procedures that are described in more detail elsewhere (Palloni and Lee, 1991).

The first method involves a projection of a cohort of *healthy* couples who give birth to a healthy child at the onset of the projection. Couples and children are then subjected to attrition due to several sources: mortality due to normal causes, HIV infection, development of AIDS (if HIV positive), and mortality due to AIDS. Estimates for the force of mortality due to all causes except AIDS can be derived directly from corrected vital statistics. Estimates for mortality due to AIDS, rate of incubation, and age-specific rates of infection are obtained from a combination of empirical and model based procedures[8].

Calculations proceed as in conventional population projection. Ther are two important differences, however. First, we take into account the fact that at least one of the forces of attrition (infection) is changing over time since the prevalence of HIV/AIDS in the population is also changing. Secondly, our projection uses couples and not individuals as units of analysis. At any point after the beginning of the projection we calculate the distribution of couples according to the survival and health status of its two adult members and the survival status of the child born at the start of the projection. Changing the parameters to represent mortality due to AIDS, the incubation of HIV, and infectivity leads to alternative measures of the impact of HIV/AIDS.

Table I displays estimated values of the probabilities that a child who is born healthy to healthy parents will survive to exact ages 5, 10 and 15 and will experience maternal orphanhood, paternal orphanhood, or both. In all cases the estimates were calculated assuming that mortality of parents and children are independent but that the forces of mortality of parents are mutually dependent and that such dependency is only accounted for HIV/AIDS[9]. Comparing scenarios with infectivity equal to 0 to scenarios with infectivity equal to .01 and .10 provides an idea of the magnitude of the impact of HIV/AIDS. Under mortality conditions normally observed in Africa (absence of HIV), not more than 9 percent of children will experience maternal orphanhood before age 15. But its prevalence is increased to 18 and 24 percent under the two alternative scenarios for HIV. The levels of paternal orphanhood are only slightly higher than those of maternal orphanhood. Note that the probability of losing both parents simultaneously is more than trebled in regimes with HIV and grows to levels close to 11 and 26 percent when the children are aged 15.

The probabilities of becoming maternal or paternal orphans are uninformative of the timing of the relevant event and of the length of time that children are expected to spend as orphans during their first 15 or 20 years of life. Table II displays three sets of measures of the tempo of orphanhood. The first panel shows the median age at which children who eventually become orphans before age 20 experience orphanhood and the second panel shows the median ages at orphanhood according to the HIV/AIDS status os the surviving parent. The median ages at orphanhood are not greatly affected by HIV; if anything, they increase slightly in the intermediate HIV regime. This is because children who experience orphanhood in regimes with HIV are disproportionately drawn from among those whose parents become infected with HIV and who, therefore, must have survived long enough to contract the infection and develop AIDS. If infectivity is high enough, the waiting time to infection will be sufficiently short to 'anticipate' orphanhood. If infectivity is not high enough, orphanhood may be 'delayed' somewhat relative to an HIV-free regime. In the second panel we present separately the median times at orphanhood for those whose parents die of causes unrelated to AIDS (first and fourth columns) and for children whose parents succumb to

AIDS (remaining columns). This panel (columns 1 and 4) confirms the idea that orphanhood due to causes other than HIV will occur at progressively younger ages in regimes with HIV. Since these cases represent a decreasing proportion of all cases of orphanhood, the timing of the event tends to be delayed.

As could be expected, the contrast in the orphanhood experience between HIV-free and HIV regimes emerges more sharply when we examine the *total* fraction of time (TFT) that children spend in various states[10]. The third panel in Table II displays estimated fractional durations spent in the three orphanhood states[11]. In a HIV-free regime the fraction of time spent between age 0 and 20 as a maternal orphan is about 5 percent, whereas in an HIV regime this fraction rises to 8 and 12 percent when infectivity is intermediate and high, respectively. Perhaps more indicative of the effects of HIV is the fraction of time spent in *any one* of the 'orphanhood' states: about 13 percent when there is no HIV, 23 percent with intermediate infectivity and 37 percent with high infectivity. Note that the values of the estimates should be increased by about 1.54 if we want them to reflect time spent in various orphanhood states for *children who survive up to age 20*[12].

Although these estimates provide a good assessment of the sheer magnitude of the mortality-related impact of HIV/AIDS, they are not good estimates of the actual conditions that a particular society can experience. This is because healthy couples are only a subset of *currently observed* couples and also because most of them have already had other children who, just like the newly born, will also be exposed to the loss of parents. To incorporate the effects of the initial distribution of couples by health status and the age-distribution of surviving children on the levels of orphanhood, we apply a second procedure. This procedure is empirically based and seeks to estimate the levels of maternal orphanhood for children of mothers who, at the onset of the projection, have already attained childbearing ages. To the experience of children who have already been born at the onset of the projection we add the estimated experience of those who will be born within the time interval covered by the projection[13]. The quantities that we present here only assess levels of maternal orphanhood for children who are 10 years old or younger.

Table III displays estimates of the proportion of maternal orphans among those children who had not been born or who were aged between 0 and 10 at the start of the projection and who survive five and ten years into the projection period. There is one set of values for each of two possible HIV statuses of the mother (susceptible or HIV-infected) and two populations, one HIV-free and the other with HIV. The average values shown in the third and fourth rows of the table were obtained assuming two alternative levels of *current* HIV prevalence among women in reproductive ages. The first, .05, reflects the situation in West Africa —except perhaps Ivory Coast— whereas the second, .25, is more appropriate to represent the prevalence of HIV in East Africa. Thus, under conditions prevailing in East Africa we expect that a fraction of about .21 children below the age of 10 will be orphans over the next five years. Instead, during the same period of time, in countries of West Africa the fraction of orphans is expected to grow to about .11.

The quantities calculated before can be readily used to yield a maximum range for future probabilities of orphanhood. The lower bound of the range should equal the probabilities of becoming maternal orphans that would be observed if in the near future no additional HIV cases were verified. If current conditions of HIV prevalence approximate those found in East Africa, the lower bounds are .12 and .18 for the next five and ten years respectively[14]. If conditions are closer to those experienced in West Africa, the lower bounds are .05 and .07. The upper bounds are calculated assuming that in all cases the levels of future prevalence are those experienced in a steady state for a regime with infectivity equal to .01. These were already calculated and correspond to .37 and .56 for projections horizons of 5 and 10 years respectively.

To summarize: although Tables I and III are not directly comparable, both lead to the same conclusion, namely, that the presence of HIV/AIDS more than doubles the levels of orphanhood in less than ten years.

With a few exceptions, the spread of HIV/AIDS in Africa has been predominantly an urban phenomenon. Since household arrangements and kin relations acquire different forms in urban than in rural areas, the impact of orphanhood should also be experienced differently. Censal data from the decade of the seventies indicate that the proportion of female-headed families ranges from a low of about 5 percent in Burkina Faso to a high of 30 percent in Kenya. This is partially illustrated in Table IV, which shows that countries more severely affected by HIV tend to have higher proportions of female-headed households. The relation is not tight because high prevalence of female-headed household does not necessarily imply higher levels of HIV prevalence. Our argument, however, does not requiere a tight relation. Although based on information of admittedly low quality, we only claim that the expected growth in the prevalence of orphanhood will be higher in countries with household configurations where females are the central figure. The actual after-effects of maternal orphanhood should be, in theory at least, much stronger where mothers share an overwhelming part of the responsability of caring for and rearing of children. Indeed, in these cases maternal orphanhood is quite literally tantamount to paternal orphanhood.

A few considerations about Table IV are in order. First, the units of observation are somewhat inconsistent and perhaps inappropriate. The inconsistency arises since whereas the figures for HIV seroprevalence are for *urban* areas only, those for household arrangements are —with the exception of Ivory Coast— for the entire country. Yet even if the inconsistency were removed, the ideal unit of analysis is not the country but the social group. In the African case one should focus on ethnic groups by region of residence. Second, for obvious reasons Table IV cannot support any inference about the differential prevalence of HIV by type of household arrangements and hence cannot be used to assess the potential connection between HIV, orphanhood and household structure. Third, even the relation that we have invoked to support the argument of differential impact of orphanhood may be questionable. In fact, it is quite possible that a non-trivial fraction of all female-headed households are found in rural areas where males are absent due to permanent or temporary migration. Even if these women were also exposed to the risk of contracting HIV precisely because of their partners' residential displacements, the *social context* for orphanhood is radically different in the countryside where traditional kin relations, the strength of the lineage, and the influence of extended networks are more likely to have been zealously preserved.

A direct effect of orphanhood is an increased vulnerability of children. Whether or not increased vulnerability translates into actual deterioration of health or increases in mortality will depend on the nature of the social adjustments deployed to cope with the expected epidemic of orphanhood. In many areas of Sub-Saharan Africa, for example, the practice of fosterage is quite common. This is a system of social exchange that allegedly spreads the costs of childbearing (Page, 1988; Isiugo-Abanihe, 1983). Increased reliance on fosterage may be one of the many availiable resources to families directly affected by HIV/AIDS. The very sparse information that we have available for countries (rather than for ethnic groups) roughly indicates that fosterage practices are more prevalent precisely in areas that have been hardest hit by HIV/AIDS. Though the data are plainly insufficient, Table V contains preliminary indications about this association. Here again our argument does not rest on a causal connection between HIV/AIDS prevalence and fosterage system. Indeed it is likely that, if it exists at all, the association is spurious and that *both* the prevalence of fosterage and of HIV are the result of social institutions that control sexual relations, reproduction regimes and child rearing systems.

Fosterage arrangements do not seem to be entirely neutral in terms of mortality effects. First, although the finding has been confirmed only for Sierra Leone, children living with foster families could experience higher mortality than those living with their families of origin (Bledsoe and Brandon, 1989). Second, if the transactions through which fosterage is enacted involve strong sex preferences females (males) could be more likely to be fostered than males (females) and the net effects on survival will

favor children of the sex that is less demanded by foster parents. Thus, for example, among the Herero in Northern Botswana females are more likely to be fostered than males (Pennington, 1990). If the sex differentials in mortality according to fosterage status follow generally the same profile as among the Herero, we should expect that female children will experience higher mortality excesses as a consequence of increased fosterage.

But, besides changes in the levels and patterns of sex differentials in child mortality, fosterage could well result in *absolute increases* in child mortality (or morbidity). The pressure to foster out children may overwhelm the capacities of accommodation of the system that will be stretched to a point where child training, feeding, and health care deteriorates far below 'normal' levels.

It is unclear if and how the fosterage system can withstand the onslaught of the epidemic without collapsing. Essential for the operation of foster arrangements is a network of reciprocities which simply cannot be sustained when some of the nodes of the exchange system are forced to renege their obligations or simply disappear as a result of multiple and closely spaced adult deaths. In all likelihood, these societies will not be able to cope with the increased burden of orphanhood without the deployment of massive resources to either reinforce traditional social institutions or to generate new ones. In Uganda, for example, communities and villages have braced themselves to offer collective protection to adults affected by HIV/AIDS as well as to orphan children (New York Times, 1990; Kaleeba, 1990). The emergence of locally based organizations for risk devolution that perform their functions somewhat independently of the central state bureaucracy is probably one of a handful of accommodations that may alleviate the crisis in many of these countries[15]. If efficient adaptations are hard to find or if they are belatedly implemented, the African countries most affected by HIV/AIDS will experience significant increases in school drop out and child labor, widespread child abandonment, health deterioration among children, and outright increase in child mortality[16] (Boulos *et al.*, 1990; Kamenga *et al.*, 1990).

3.1.2. Widowhood.

Increases in adult mortality will also lead to a growing incidence of widow(er)hood. Depending on the patterns of spread of HIV and on the resulting sex differential in HIV prevalence, widowhood may increase more rapidly than widowerhood or vice-versa. When the HIV epidemic is 'male' driven, as it appears to be in Africa (Palloni and Jones, 1990), a typical sequence of events is as follows: an adult male becomes infected, develops AIDS and dies shortly thereafter. The spouse may or may not become infected and if she does, her expected survival time will be considerably shortened. Thus, the same forces that increase the incidence and anticipate the timing of widowhood will offset the tendency toward longer duration of widowhood. This is confirmed by the estimated duration of widowhood in the first panel of Table VI: the expected duration of widowing over a 20-year period following marriage is substantially reduced as a consequence of increased female mortality. The second panel of Table VI displays the estimated (cumulated) probabilities of being widowed at various ages for women who at age 25 were healthy and married to healthy husbands[17].

According to these estimates, in an HIV regime with intermediate infectivity, at least one-fifth of the women will experience widowhood before attaining age 35 and at least a third by age 35. Relative to normal mortality conditions, an intermediate regime of HIV virtually doubles the prevalence of widowing (compare first and second rows). Even though the experience of widowhood is shortened by HIV/AIDS itself, the sheer increase in the prevalence of widowing could create staggering problems since —at least among some social groups— they represent an added load to already overburdened women. How will different social groups (e.g., ethnic, classes) cope with it? Will re-marriage rates increase or will they decrease as stigmatization and the fear of contracting the disease spreads and minimizes the pool of available male partners? And for those who are stigmatized and shunted, will the extended kin network buffer the full impact of illness and

isolation? What will be the contrasts in responses between the polygamous and monogamous groups, between patrilineal and matrilineal systems, between urban and rural residents? Will the brunt of the burden fall on women's shoulders as neither the extended family, the fosterage system, informal organizations nor the central state can accommodate quickly and efficiently to the sudden change?

Admittedly, increases in widowhood will have potentially more serious consequences where women are more dependent on men for subsistence than where they retain considerable economic autonomy. The latter may be the case in rural areas and perhaps among women urban dwellers involved in trade and commerce. Moreover, as the traditional family organization based on patrilineality, polygamy and strong dependency on gerontocratic control is more resilient in rural areas, accommodation is more likely to occur there than in urban centers where the influence of and protection from the traditional extended family began to wane before the onset of the epidemic. And yet, as indicated before, it is the family in the urban areas that is most affected by the HIV/AIDS epidemic. Ironically, urbanization has increased the vulnerability of families to HIV/AIDS while simultaneously stripping them of one of the most efficacious means to cope with its effects.

3.1.3. Co-residence arrangements and the vulnerability of the elderly.

The third consequence of the increase in adult mortality follows from the other two: as 'young-adult' mortality tends to raise, a relatively powerful demographic constraint is imposed on family composition. Where the preferred arrangement is co-residence of three generations, an upward pressure in 'young-adult' mortality will increase the prevalence of or pressure for co-residence arrangements where some or all members of the *intermediate* generation are missing. Barring drastic changes in remarriage rules, the presence of the parental generation will be weakened and the relations between children and grandparents could become more dominant particularly but not only where grandparents are part of co-resident arrangements[18].

But children are not the only ones on the losing end. In fact, the older generation stands to experience heavy economic setbacks as well. In societies where joint residence of parents and grandparents is one of the adjustments that facilitates the support of the elderly, an increase in 'young-adult' mortality will lead to a deterioration in the efficacy of the residential arrangement or, equivalently, to the collapse of other mechanisms that insure support of the elderly by their children. Since normal sex differentials in mortality at older ages favor females, chances are that those who stand to lose most in the presence of HIV are elderly women: not only are they more likely to be burdened with the care of the very young, but by losing the support of their own children they will also experience a drastic deterioration of the control over the materials and social means to do so efficiently.

A precise quantitative assessment of this effect requires rather elaborate models. We have, however, utilized an approximate procedure that allows us to calculate the probability that a tree-generation family arrangement will be reduced to only two generations, children and grandparents (with or without grandfather present). Table VII, Panel A, displays the expected frequency of co-resident units where *both* parents died by survival status of the paternal grandparents. In Panel B we show the expected frequencies of co-resident units where *only the mother* survives by survival status of paternal grandparents. For example, in the next five years we expect that in a regime of HIV with intermediate infectivity (.01), there will be about 42 co-resident units (out of 100,000) with no parents and both paternal grandparents alive and 32 units with no parents and only the paternal grandmothers alive. The corresponding counts of co-resident units with a surviving mother are, respectively, 1,272 and 964. It is important to note that from the point of view of family needs, it is the prevalence of co-resident units with no parents or with only mothers alive that matters. Thus, for example, whereas under normal conditions, one would expect that within a five-year period 4,185 (147+180+1,733+2,125) out of 100,000 co-resident units will experience relatively high dependency, in a

regime with intermediate infectivity leads to a two-fold increase of these units (9,411)[19].

These figures illustrate from a different angle the effects of increases in young-adult mortality and point to a unique problem: whereas orphanhood must be placed in the context of *child care*, the loss of the intermediate generation also has implications that should, however, be placed in the context of *elderly care*. How acute the demand for elderly care will be depends both on the degree of dependency of elders on their children before the initiation of the epidemic, and on the extent to which they are called upon to deliver services to and care for their grandchildren and their surviving but possibly sick children. The worst combination of circumstances is one where the edifice of social relations erected prior to the onset of the HIV/AIDS epidemic relies on the intermediate generation to simultaneously support the youngest and the oldest. Wherever this occurs the vulnerability of elders will compound the vulnerability of the youngest.

We have very little information on the prevalence of various types of residential arrangements in Africa. Estimates retrieved from census around 1960 and 1970 and from surveys in 1975-1980 indicate that for Chad, Ghana, Congo, Nigeria and Cameroon the proportion of house hold with extended and stem family arrangements —possibly involving grandparents— hovered between 7 and 42 percent, with Cameroon at the lower and Congo at the upper end. And, at least in Chad, the prevalence of these arrangements was higher in urban than in rural areas. After reviewing some of the scanty available information, Locoh (1988) has concluded that "...judging from censal figures there appears to be a stronger propensity to live in large domestic units in West than in East or Central Africa...". If this inference is correct, it is precisely in areas where HIV prevalence is higher that co-residential arrangements are less conducive to a support system whereby grandparents supervise grandchildren but where grandparents are also less likely to depend on their own children for support. However, the validity of this statement depends entirely on an unverified proposition about the association between *flows of support*, on the one hand, and co-residence arrangements, on the other. Intergenerational flows of support may and indeed do occur even when co-residence of parents and grandparents is not a preferred or even the most feasible choice. The prevalence of co-residence between parents and grand-parents could understate the bond of dependency between generations should departures from the association between co-residence and intergenerational support be a common empirical occurrence.

3.2. INCREASES IN INFANT MORTALITY AND MOTHER'S HEALTH

In societies with a strong emphasis on descendance and a rigidly enforced norm of high fertility, a sudden upsurge of infant mortality may trigger and adaptive response toward even higher fertility. This can occur in two very different ways. First, an increase in infant mortality will, on average, shorten the period of post-partum amenorrhea and, in the absence of offsetting changes, it should result in a increase in the number of children ever born. This increase will be particularly visible where a variety of practices, such as breastfeeding and post-partum abstinence, ensure long interbirth intervals. The second mechanism takes the form of a volitional replacement response on the part of couples. If emphasis on large number of descendants implies a high though vaguely defined fertility target, detectable increases in infant and child mortality in the community at large and for a couple in particular could lead to fertility increases to insure the maximum of reproduction.

If it occurs at all, the potential increase in the natural rate of growth induced by fertility increase is likely to be short-lived and fully offset by increases in early child mortality due to HIV/AIDS, and the shortening of birth spacing and of the lactation period, and by the reduction in the *effective* reproductive span precipitated by higher female (and male) mortality. However, even a transient fertility increase may have potentially grave *health* consequences for individual women. Although fertility increases in areas where fertility is already high will occur at the expense of a deterioration of maternal health in general, those who will experience the brunt of the burden are women who are

already infected with HIV who are more likely to directly experience fetal or child losses (Lopita, 1990; Temmerman, 1989; Guay, 1990; Boulos, 1990; Kamenga, 1990). Maternal transmission of HIV alone should induce a correlation between HIV prevalence and early child mortality.

There are, however, other factors that could reinforce the fertility response to HIV. In several clinical studies in Africa and elsewhere, an association has been found between HIV risks and the presence of Sexually Transmitted Diseases (STD). Among the various mechanisms that could produce the relation the most plausible are a *direct linkage* through which STD enhances infectivity of the infected partner (or susceptibility of the uninfected partner) and a *spurious relation* due to a common dependency of both HIV and STD on patterns of sexual behaviors. Figure 1 is a plot of the (log) value of adult HIV prevalence and a proxy of STD prevalence (level of sterility). Although the linear relation is not tight (R-square=.20), it is strong enough to suggest a less than trivial association[20]. Consequently, it is in social groups with higher levels of HIV where the impact of infant mortality on fertility that is mediated by a volitional response could be greatest. Indeed, it is among these groups where sterility is higher and where child losses are more feared since they represent a higher fraction of the potential number of children that women can bear.

But, do these effects amount to much? First, barring an acceleration of the improvements in child health, the potential increase in early child mortality could be quite substantial. Table VIII displays the estimated increase in child mortality (below age 5) that will occur under a variety of scenarios about the probability of perinatal transmission, excess mortality of seropositive children, and the levels of seroprevalence among pregnant mothers. Conditions approximating those in some of the most seriously affected countries in Africa (Zaire, Uganda, Rwanda, Burundi, Tanzania, Congo) suggest that the levels of prevalence among pregnant mothers hover around 20 to 25 percent. Under the most conservative assumptions, these seroprevalence levels should result in an increase of about 8 percent in infant mortality. Under a worst case scenario the increase could be of the order of 50 percent.

Second, the magnitude of the short term increase in fertility depends on the average reduction in the post-partum amenorrhea period. An increase of child mortality of around 25 percent (average of the worst and best scenarios) should decrease the post-partum period by about 30 percent and the expected increase in fertility could be of the order of 9 percent (Jones and Palloni, 1990). Although this increase will not be high or sustained enough to bolster the long term natural rates of increase, it will inflict severe setbacks on the health of all but particularly HIV-infected women.

4. THE BOTTOM OF THE ICEBERG: THE HEALTH STATUS OF THE ADULT POPULATION

4.1. HEALTH STATUS AND THE HIV-/AIDS EPIDEMIC

Exclusive emphasis on the upward pressure on adult and infant mortality could divert attention from potentially serious effects on health levels. A relatively long incubation period combined with environments that predispose the population to repeated viral, bacterial, or parasitic diseases, could lead to health deterioration of the infected population on a scale not experienced in other geographic areas (WHO, 1987). Whether or not HIV interacts with other diseases to extract a higher *health* toll depends on a strongly suspected but not yet systematically verified link between seropositivity and health impairment induced by common infectious and parasitic diseases[21].

If in fact HIV and other diseases do interact to increase susceptibility to infections, aggravate their consequences if contracted, or lenghten recovery periods, the demand for health care will grow not just because of the expected increase of individual episodes of AIDS but also because of the gradual health deterioration that begins *before* the onset of full-blown AIDS and that may last for a protracted period of time. Productive individuals on whom families must rely for income-generating

activities will be disabled for periods of time that are longer than those implied by the expected duration of full-blown AIDS.

Thus, proper assessment of the economic losses attributable to HIV and the evaluation of the impact on the organization of the family should take into account not just the direct impact of mortality but also the stark shift in the distribution of members of the family by health status. The damage inflicted on the domestic economy, the effect on the efficient functioning of industries, services and bureaucracies, and the disruption on the support systems for children and elders are magnified if seroconversion is followed by more than trivial deleterious effects preceding the full-blown development of AIDS. Since the losses incurred by the family due to health impairment alone will extend over a relatively long period of time, the mortality impact of HIV/AIDS that we estimated before underrepresents the total potential losses.

To document the health consequences of HIV/AIDS at the *family level* we use the projection procedures referred to above to calculate the distribution of children by health of parents at several points in time. The first three panels of Table IX were constructed using the first projection procedure. The figures in these panels represent the experience of a cohort of children born to two healthy parents. The last panel was calculated using the second projection procedure and describes the experience of children now alive and of those who will be born to mothers according to their current HIV/AIDS status. The first panel of the table shows the median ages at whitch HIV and AIDS will affect *one* of the (susceptible) parents of a child just born. We always choose median value for the parent who is firts affected by either HIV and AIDS. The second panel of the table displays the fraction of total time between ages 0 and 20 that a child will live with healthy parents and several other combinations of parents HIV/AIDS status. The third panel in Table IX displays the expected number of years before a child just born experiences either the death or the illness (HIV-related) of one of the parents. The last panel shows the fraction of all children who will have an HIV mother five and ten years after the start of a projection. Like the figures in Table I and Table II, those in Table IX are statistics of the tempo and quantum of parental events. Unlike those in Tables I and II, however, the statistics in Table IX do not neglect the onset of HIV and AIDS. If HIV implies protracted deleterious health effects and harbors the potential for immediate economic losses, the figures displayed in Table IX are much more telling and ominous than those related to orphanhood alone: approximately 13 percent of all children born to a healthy female aged 25 will have a seropositve or AIDS mother within a five year period. The quantity is doubled if the time period is extended to ten (see Panel D of Table IX). Note that in countries with HIV regime similar to those found in East Africa the proportion of children who will have an HIV or AIDS mother could be as high as .25 (the weighted average of .63 and .13). Over a period of ten years the quantity increases to .32. Similarly, in an intermediate regime of HIV, only 40 percent of the first 20 years of life of a child will be spent with healthy parents. Instead, in an HIV-free regime the time spent with a living mother (but non necessarily healthy) is about 80 percent of the total possible within the first 20 years of life (Panel B of Table IX).

4.2. A PARADOXICAL EFFECT: CAN THE RISK OF HIV BE PASSED THROUGH GENERATION?

Societies seriously affected by HIV will improvise alternative adaptive responses to accommodate to the new conditions brought about by health deterioration, increased mortality, and possibly increased fertility. Although it is difficult to predict which adaptations are more likely to occur and where, some responses could involve the collpase of traditional practices and the emergence of new ones that will end up favoring conditions for the further entrenchment of HIV. We now discuss a particularly likely reaction that may have precisely this negative consequences.

As remarked before, fosterage is a pervasive institution in large parts of Africa involving the circulation of young children from parent to foster parents who become responsible for providing subsistence, instruction, and training (Page, 1989; Isiugo-Abanihe, 1983). And al-

though the exchange frequently involves the transfers of children from urban to rural localities, the flow in the opposite direction that occurs in return also forms an integral part of the exchange. Fosterage sustains high fertility regimes by spreading the costs of childrearing among several groups. Conceivably it could also absorb the rise in costs triggered by economic or population crisis.

However, it is unlikely that the system will respond efficaciously when faced whit massive and sustained pressures brought about by increases in the prevalence of orphanhood or, more generally, in the explosion of ill parents' demands for child care. The cracks in the fosterage system will develop at several levels. First, the potential demand for transfers of children in an HIV epidemic may be considerably more massive than the system is designed to absorb or accommodate. Searching costs will increase and the entire operation will become more cumbersome and taxing for the sending family and less rewarding for the family of destination. Second, since fosterage involves reciprocity, the partial or total disabling of the sending family by illness may render the exchange less attractive to the receiving end and may become ultimately impossible. Note that disabling a node in the system of exchange does not necessarily require the death of the members of a couple who foster children out. In fact, more frequent illness or the fear that surrounds the recognition of HIV or AIDS may be equally efficient deterrents to renew the exchange. As families affected or suspected to be affected by HIV are socially shunted, they are also eliminated rom generalized exchange systems that could have protected their children.

In the absence of well-defined social forms of collective care and protection of children —and particularly where the fosterage system is not part of the tradition or has fallen into disuse— the slack could be taken up by the extended family which abruptly must expand its boundaries and improvise new functions. However, the likelihood is that these ad hoc adaptations will be inappropriate or insufficient and could even accelerate the aggravation of the social, economic, and health conditions of children. They could also establish the ground for increased risks of HIV among the very young.

Families affected by HIV, where one or both parents are ill and incapable of sustained productive activity, will depend more heavily on support from young children. Children will be more likely to be withdrawn from school or may become frequent absentees, wherever possible their participation in informal markets will increase, and the parental incentives to foster some of them out could be considerably reduced[22].

As households struggle to preserve entitlements for subsistence, female children stand to be the biggest losers. In most African societies the control over females labor and reproductive capacities is of paramount importance for the preservation of the traditional (and sometimes the new) order. The articulation of a system for the eficient circulation of women and the enforcement of their subordination to males is a pillar of this social order. Females represent variable amounts of bridewealth or other obligations for their lineage of origin as they are married outside the lineage. The influence of urbanization and westernization coupled with increases in education may have altered traditional arrangements but has not completely destroyed their foundations. Women in urban areas can and are able to make a living, support higher education, and secure social and occupational advantages by engaging in informal relations with men who are not and may never become their husbands. Urban families economically strapped by the effects of paralyzing illness will find it to their advantage to encourage earlier engagement of daughters to older males. Incentives for increased participation in commercial sex —whether it is open prostitution or a more selective practice of formation of casual unions in exchange for economic compensation— will increase both for parents and daughters alike. If social and economic advancement of women today is made easier by the exchange of sexual favors, it is all the more likely that the survival of individuals and families will be made strongly dependent on it. The net result of intensification of commercial sex will be to reproduce the worst conditions of exposure to HIV and will make feasible the persistence of at least one necessary condition for HIV to become endemic —an age gap

between sexual partners—. If the sexual favors of younger girls can be exchanged for cash payments or other economic benefits by engaging them directly with protectors, 'sugar daddies' and various other assorted types of more or less casual remunerative relations, the practice could become a court of last resort precisely among those families affected by HIV/AIDS. Thus, paradoxically, the most salient of all social consequences of HIV may be that, in the absence of massive external interventions and left to themselves, families will adapt to the changed conditions by clinging to behaviors and social practices that sustain the continued reproduction and transmission of HIV.

5. CONCLUSIONS: HIV/AIDS, FAMILIES, SOCIAL ORGANIZATION, AND REPRESENTATIONS

The most evident *direct* effect of the HIV-/AIDS epidemic is an increase in adult and early child mortality. Whereas the increase in adult mortality predictably raises the incidence of orphanhood, widowhood and unconventional co-resident units, the sudden burst in child mortality will lead to short term fertility increases and very likely to further deterioration of the health status of women of reproductive ages.

A less evident *direct* effect of HIV/AIDS is the worsening health status of the 'young-adult' population which may totally overshadow the more readily visible increases in mortality. If the HIV virus turns out to interact with other pathogens to increase the overall frailty of the HIV-infected population, the mortality-effects of the epidemic will pale when compared to the blow inflicted on the health status of the most productive sector of the population.

The effects of increasing health disability and/or adult mortality will be felt most immediately by families but will inexorably reverberate throughout multiple social institutions. Making predictions about adapting strategies is far more difficult than, and premature without, identifying 'areas of impact' or, equivalently, those social relations and functions that will be most affected.

Regarding the family, there are three areas of primary interest. The first is the family as a *production unit*. The most important issue here is to what an extent the ability to appropriate entitlements is impaired as key members of the family are lost or partially disabled. Loss of income to preserve access to food, clothing, and health care is not the only entitlement that could be endangered. In patrilineal social groups inheritance rights may be automatically forfeited with the death of the father, claims over the use of land for cultivation or over the rights to exercise trade and commerce may be weakened or lost altogether. Potentially the most damaging effect is the erosion of human capital formation as children's formal education is truncated in exchange for compensation to subsidize urgent present needs. The actual magnitude of the loss induced by HIV/AIDS alone depends, of course, on whether or not dilution of human capital would have occurred anyhow in response to the severe economic (rather than health) crisis that the majority of African societies are undergoing.

The second area of relations perturbed by HIV/AIDS is related to the family as a *unit of reproduction*. Quite apart from the fact that disability and death will reduce the effective reproductive potential of a couple, HIV/AIDS could undermine the mechanisms through which the costs and benefits of reproduction are distributed. We have already mentioned the damage that the fosterage system can sustain. But the same applies to the division of labor within the co-resident unit, the extended family (if applicable) and even the division of labor between spouses and between parents and children. As the actual reproduction of families rests on intricate exchange networks involving members of the same and of other families and as these networks can collapse under the onslaught of the epidemic, so will the reproduction system itself be threatened. An integral part of the functioning of the family as a reproducing unit is the exchange of women and bridewealth. Incentives to sexually engage daughters earlier in exchange for compensation could lead not just to a 'family reproduction' of HIV but to the acceleration of transformations brought about by Wester culture whereby families lose their grip over partner selection. The institution of marriage will be

transformed by the changes in the rules of partner selection and by a squeeze in the marriage market, a spill-over effect of the increases in adult mortality.

Finally, the family as a *unit of training and socialization* will be severely impacted by HIV/AIDS. Caring, training and socialization of children are directly entrusted to parents or spread over several layers of relatives. Death and health impairment will weaken the accomplishment of these functions as those most closely connected with them are no longer able to perform them. Thus, as has been documented for other societies (but apparently for very different reasons), the new generation of children grows less attached to family and family members, and more to informal groups completely foreign to the familial realm.

The nature of the actual effects in these three areas will depend on three factors: a) the current organization of the family as a domestic unit of production, reproduction, and socialization; b) the organization and efficiency of *local* interventions to buffer the effect of the crisis; c) the organization and efficiency of *national and international* interventions. These three levels of social institutions that mediate the effects of HIV/AIDS can in turn be conceived as having two related domains. One is the material structure or set of social relations that constitute each level, and the other is the ideational structure or set of ideological representations about the disease and the individuals who contract the disease. Indeed, the influence of HIV on the *domestic domain* is not restricted to the fact that parents health will objectively limit their income earning capacities. Infected parents (and their children) could also be altogether precluded from participating in gainful economic activities as they are socially stigmatized, isolated, and discriminated against in response to the social construction of the disease. Whereas the first effect works through the material structure of the family as a unit of production, the second works through its ideational structure.

African societies offer a kaleidoscope of combinations of these three factors (and the corresponding economic and ideological domains) and it would not be surprising that adaptations to the effects of the crisis triggered by HIV/AIDS will be as variable as the regimes of HIV themselves. Adaptation and the nature of social interventions will be jointly determined by the severity of the crisis and by family organization, local and national institutions. The latter represent constraints and opportunities for the emergence of new relations. As traditional practices are subjected to extreme pressure, the crisis created by HIV offers an opportunity to witness and participate in the making of new social forms.

Uganda was the first African country to launch concerted, well targeted education campaigns; it is the country that has produced a fertile ground for the emergence of spontaneous, local based groups to help families and individuals cope with the tragic aftermath of HIV and AIDS. Although one could argue that the distinctive character of Uganda is simply the result of an accident —the different timing of arrival of HIV— and that similar responses will sprout elsewhere very soon, it is not farfetched to entertain the speculation that there is a deeper connection between, on the one hand, open, direct central interventions on a national scale and prolific local adaptations and, on the other hand, the civil chaos, social disorganization, and lack of strong central authority that Uganda experienced just as HIV was making its incipient appearance within the villages in the vicinity of Lake Victoria about ten years ago.

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ENDNOTES

1. For a relatively recent attempt to improve our knowledge in these areas see Bardet *et al.*, (1988).
2. The procedure suggested by Livi-Bacci (1978) was also suggested by Schofield (1977). Livi--Bacci proposed that the procedure be used to retrieve a measure of the intensity of the mortality crisis from observed statistics on the size distribution of families during the post-crisis period.
3. This point has been made also by Rockwell (1989).
4. Estimates of the median incubation period are variable and suggest a range between 5 and 15 years.
5. By all accounts the recovery by survivors of the plague took several months and was far from being risk-free. However, the entire episode of illness was undoubtedly shorter than that associated with HIV/AIDS. See Abrate (1972).
6. Spread of the infection among closest kin and partners is all the more likely in societies that adhere partially or totally to the practice of levirate and polygamy.
7. Of course, this feature of HIV/AIDS renders unusable all models and representations that rely on assumptions of neutral mortality increases.
8. The risks of mortality due to AIDS were assumed to be constant with a median survival time of one year. The incubation function was approximated with a mixture function of an exponential and a Gompertz function. It was parameterized so that the median incubation time is 10 years. The age specific rates of infection —the conditional probabilities that a man or a woman will become infected during a month— are taken from simulation models in a population exposed to HIV/AIDS for 10 to 15 years and with female-to-male infectivity rates of .00 (absence of HIV), .01 and .10. Infectivity rates are the conditional probability that an infected individual will pass on the infection to a susceptible individual in a single sexual contact. Male to-female infectivity is assumed to be three times as large as the baseline female-to-male infectivity. For more details, see Palloni and Lee (1991).
9. For reasons that we clarify later, it is unlikely that the assumption of independence of parents and children's mortality risks is a very good one. For one thing, we will be arguing that a tighter association between children and parents' health is precisely one of the distinctive consequences of HIV/AIDS. Thus, although the validity of this assumption can be debated in other applications, it is especially questionable in the presence of HIV/AIDS. By the same token, one could argue that the dependency between spouses' mortality is stronger than what is implied by their common associations with HIV/AIDS. This is a valid objection but the contribution of additional factors to an already strong dependency is numerically trivial.
10. It is important to note that, unlike the median times examined before, TFT is affected not just by the timing of the relevant events but also by the proportion of children who will experience them.
11. Unless explicitly stated otherwise, our estimates are derived from multiple decrement probabilities and hold when children are themselves exposed to mortality risks.
12. This is the reciprocal of the probability of surviving from birth to age 20.
13. A fuller description of the method can be found in Palloni and Lee (1991). The method requires the estimation of HIV-prevalence among adults. To obtain results compatible with those from the first method, we assumed estimated prevalence levels equal to those observed in the simulated population that was used as a baseline to retrieve age-specific conditional probabilities of becoming infected in the first method. To simplify the presentation of results we used only one simulated population with an infectivity rate of .01.
14. For societies in East Africa, for example, the lower bounds for the probabilities that a child between 0 and 10 years of age will be an orphan before the end of the next five years should approximately equal the weighted average of the probability for children whose mothers are currently seropositive and the probability of

becoming maternal orphans in the absence of HIV or .25*.30+.75*.031.

15. It may come as no surprise that there is also evidence of the emergence of similar institutions in pre-industrial societies undergoing epidemics (Abrate, 1978; Livi-Bacci, 1978). Social institutions for last-resort protection of individuals and families were also operative in normal times (Lesthaeghe, 1980). Born spontaneously out of sheer necessity and sustained on relatively fragile local support, it is unlikely that any of these organizations in African urban settings or villages will be able to perform their tasks effici-en-tly without the infusion of generous amounts of exogenous support.
16. Ironically, these are all effects that will be inevitably reinforced by the severe fiscal and monetary policies that are part of restructuring programs.
17. To be consistent with results presented earlier we assumed that the 'starting point' of the exposure to widowing occurs at age 25, approximately the mean age at childbearing. Under normal mortality conditions this assumption would result in an upward bias of the probability of becoming a widow within a fixed period of time. In regimes with HIV, however, the estimate will contain a slight downward bias.
18. This may not always be the case. When, in addition to grandparents, the dominant extended network includes aunts and uncles and other lateral kin, the bonds of dependency linking children to grandparents need not be reinforced upon the death of parents since responsibility for the care of children could be spread among uncles and aunts as much as among grandparents. However, since HIV/AIDS is likely to affect aunts and uncles as much as parents, this alternative adaptation may be foreclosed.
19. The calculations assume that we follow a 'co-resident cohort' consisting of two healthy parents aged 25 and 30 and at least one paternal grandparent. The cohort is then subjected to attrition due to mortality of parents and grandparents. It is further assumed that the mortality of children and grandparents is independent of the mortality of parents and that the propensity to co-reside does not change overtime.
20. Neither the association betwenn STD's and HIV implies a causal link nor our argument rest on its existence. What we argue only requires a high probability for the joint occurrence of sterility and high prevalence of HIV.
21. The nature of HIV's invasion on the immune system suggests powerful reasons to suspect that HIV positive individuals should be more prone and less able to efficiently combat a host of infectious and parasitic diseases. There is also significant evidence to support this conjecture. Badi and colleagues (Badi *et al.*, 1990) found increased incidence of tuberculosis among HIV positive women. Bacterial and parasitic infections were found in HIV positive patients in Central Africa (Taelmann *et al.*, 1990) and Germany (Weinke *et al.*, 1990). A variety of other abnormalities ranging from lipid irregularities (Blatt *et al.*, 1990) to increased energy expenditure (Melchior *et al.*, 1990) and hypovitaminemia B-12 (Riera *et al.*, 1990) have also been associated with HIV. We found no studies clearly confirming the hypothesis that seropositive individuals are more likely to experience longer and more healt-compromising recovery periods if a disease is contracted.
22. Paradoxically, the decline in incentives among those in the sending node of the fosterage system may relieve some of the pressure on the system and simultaneously contribute to make it increasingly irrelevant.

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TABLE I: PROBABILITIES OF BECOMING A MATERNAL ORPHAN (MO), A PATERNAL ORPHAN (PO), OR BOTH (OO) BY AGES 5, 10 AND 15 FOR CHILDREN BORN TO HEALTHY PARENTS

	AGE OF CHILD								
	5			10			15		
Regimes of HIV (Level of infectivity)	MO	PO	OO	MO	PO	OO	MO	PO	OO
.00	.030	.042	.001	.059	.085	.005	.088	.129	.014
.01	.038	.051	.003	.113	.134	.031	.180	.201	.110
.10	.067	.083	.006	.199	.225	.090	.239	.264	.264

Source: Projection of health status of parents of a newly born to healthy parents.

TABLE II: MEASURES OF THE TIMING OF ORPHANHOOD

Panel A: Median age of orphanhood for those who became orphans

Regimes of HIV (Level of infectivity)	Maternal orphanhood (MO)	Paternal orphanhood (PO)	Booth (OO)
.00	10.0	9.1	14.5
.01	10.9	10.8	14.6
.10	9.5	9.2	13.8

Source: See Table I.

TABLE II: MEASURES OF THE TIMING OF ORPHANHOOD

Panel B: Median age at orphanhood for those who become orphans by health status of parents at the time orphanhood occurred

	Maternal orphanhood (MO)			Paternal orphanhood (PO)		
Regimes of HIV (Level of infectivity)	Father healthy	Father HIV	Father AIDS	Mother healthy	Mother HIV	Mother AIDS
.00	10.0	—	—	9.1	—	—
.01	6.7	11.0	14.0	6.2	11.2	14.0
.10	2.0	8.5	12.5	1.5	8.0	12.5

Source: See Table I.

TABLE II: MEASURES OF THE TIMING OF ORPHANHOOD

Panel C: Fraction of time from age 0 to 20 lived by children according to survival status of parents

Regimes of HIV (Level of infectivity)	Maternal orphanhood	Paternal orphanhood	Booth
.00	.05	.07	.01
.01	.08	.10	.05
.10	.12	.14	.11

Note: See Table I.

TABLE III: PREVALENCE OF MATERNAL ORPHANHOOD AMONG CHILDREN ALREADY BORN AND BORN AFTER THE START OF THE PROJECTION

Mother's HIV status at the start of the projection

	HIV-projection period (years)		HIV+projection period (years)	
	5	10	5	10
Absence of HIV	.031	.044	—	—
With current conditions of HIV and infectivity equal to .01	.050	.086	.37	.56

Average proportion of orphans expected if current proportion of adult females infected with HIV is:	Projection period 5 years	10 years
.05	.07	.11
.25	.13	.21

TABLE IV: PREVALENCE OF HIV AND PREVALENCE OF FEMALE HEADED HOUSEHOLDS

Country	Proportion of heads of households who are females: 1970-80 [1]	HIV seroprevalence (% adults) [2]
Burkina Faso	.051	1.7
Ivory Coast (Abidjan)	.105	8.1
Ghana	.286	4.7
Mali	.151	1.6
Togo	.162	—
Burundi	.231	15.0
Kenya	.295	2.6
Reunion	.237	—
Rwanda	.251	20.1
Cameroon	.129	.5
Congo	.146	3.9
West Zaire	.289	4.5
Kenya	.295	2.6
Reunion	.237	—
Rwanda	.251	20.1
Cameroon	.129	.5
Congo	.146	3.9
West Zaire	.289	4.5

[1] Percent of children aged 0 to 14 who do not reside with another. From Page, 1988.

[2] Percent of adult seroprevalence. From Bongaarts *et al.*, 1989.

TABLE V: PREVALENCE OF FOSTERAGE AND HIV

Country	Prevalence of fosterage[1]	HIV prevalence[2] (% of adults)
Cameroon	17.94	1.5
Ghana	20.84	4.7
Ivory Coast	21.30	8.1
Kenya	12.39	2.6
Nigeria	11.50	.00
Sudan (North)	6.14	.39

[1] Percent of children aged 0 to 14 who do not reside with another. From Page, 1988.

[2] Percent of adult seroprevalence. From Bongaarts *et al.*, 1989.

TABLE VI: MEASURES OF THE EFFECTS ON WIDOWHOOD

Panel A: Expected duration of widowhood in a 20 year period for females who start their union at ages 15, 20 and 25

Regimes of HIV (Level of infectivity)	Age at start of union		
	15	20	25
.00	9.2	8.9	8.7
.01	5.8	5.1	5.1
.10	4.3	4.8	4.8

TABLE VI: MEASURES OF THE EFFECTS ON WIDOWHOOD

Panel B: Cumulative probabilities for being a widow in the age interval 30-45 for women who were healthy and married to a healthy husband at age 25

Regimes of HIV (Level of infectivity)	Age (duration of union)			
	30 (5)	35 (10)	40 (15)	45 (20)
.00	.046	.094	.143	.197
.01	.063	.191	.354	.455
.10	.112	.360	.564	.689

TABLE VII: PROJECTED DISTRIBUTION OF CO-RESIDENTIAL UNITS ACCORDING TO COMPOSITION BY GENERATIONS (PER 100,000)

Panel A: Co-residential units with no parents

Regimes of HIV (Level of infectivity)	Years since the start of the projection					
	5		10		15	
	Both grandparents	Only grandmother	Both grandparents	Only grandparent	Both grandparents	Only grandmother
.00	42	32	47	180	303	674
.01	127	96	909	1,117	2,385	4,417
.10	254	197	1,173	1,606	4,773	10,600

TABLE VII: PROJECTED DISTRIBUTION OF CO-RESIDENTIAL UNITS ACCORDING TO COMPOSITION BY GENERATIONS (PER 100,000)

Panel B: Co-residential units with mothers only

Regimes of HIV (Levels of infectivity)	Years since the start of the projection					
	5		10		15	
	Both grandparents	Only gramdmother	Both grandparents	Only grandmother	Both grandparents	Only grandparents
.00	1,272	664	1,733	2,125	1,591	3,533
.01	1,696	1,285	3,314	4,071	3,254	7,227
.10	2,840	2,153	5,837	7,170	4,321	9,596

TABLE VIII: PROPORTIONATE INCREASES IN CHILD MORBIDITY BELOW AGE 5

Levels of seroprevalence among pregnant women						
	.10		.25		.50	
Probability of perinatal transmission	.20	.50	.20	.50	.20	.50
Low HIV/AIDS mortality excess (1.50 times the normal force of mortality)	1.03	1.08	1.08	1.20	1.15	1.38
High HIV/AIDS mortality excess (5 times the normal force of mortality)	1.08	1.20	1.20	1.50	1.40	2.00

TABLE IX: THE HEALTH EFFECTS OF HIV/AIDS ON VARIOUS ASPECTS OF THE FAMILY

Panel A: Median age of a child at which a parent will be affected by HIV and AIDS

Regimes of HIV (Level of infectivity	HIV	AIDS
.00	—	—
.01	3.3	10.0
.10	1.7	7.0

Note: See footnote in Table I.

TABLE IX: THE HEALTH EFFECTS OF HIV/AIDS ON VARIOS ASPECTS OF THE FAMILY

Panel B: Fraction of time between ages 0 and 20 that a child just born is expected to live in various states according to health status of parents

Regime of HIV (Level of infectivity)	Both healthy	Both infected	Mother infected
.00	.68	—	—
.01	.39	.13	.24
.10	.14	.22	.41

Note: See footnote in Table I.

TABLE IX: THE HEALTH EFFECTS OF HIV/AIDS ON VARIOUS ASPECTS OF THE FAMILY

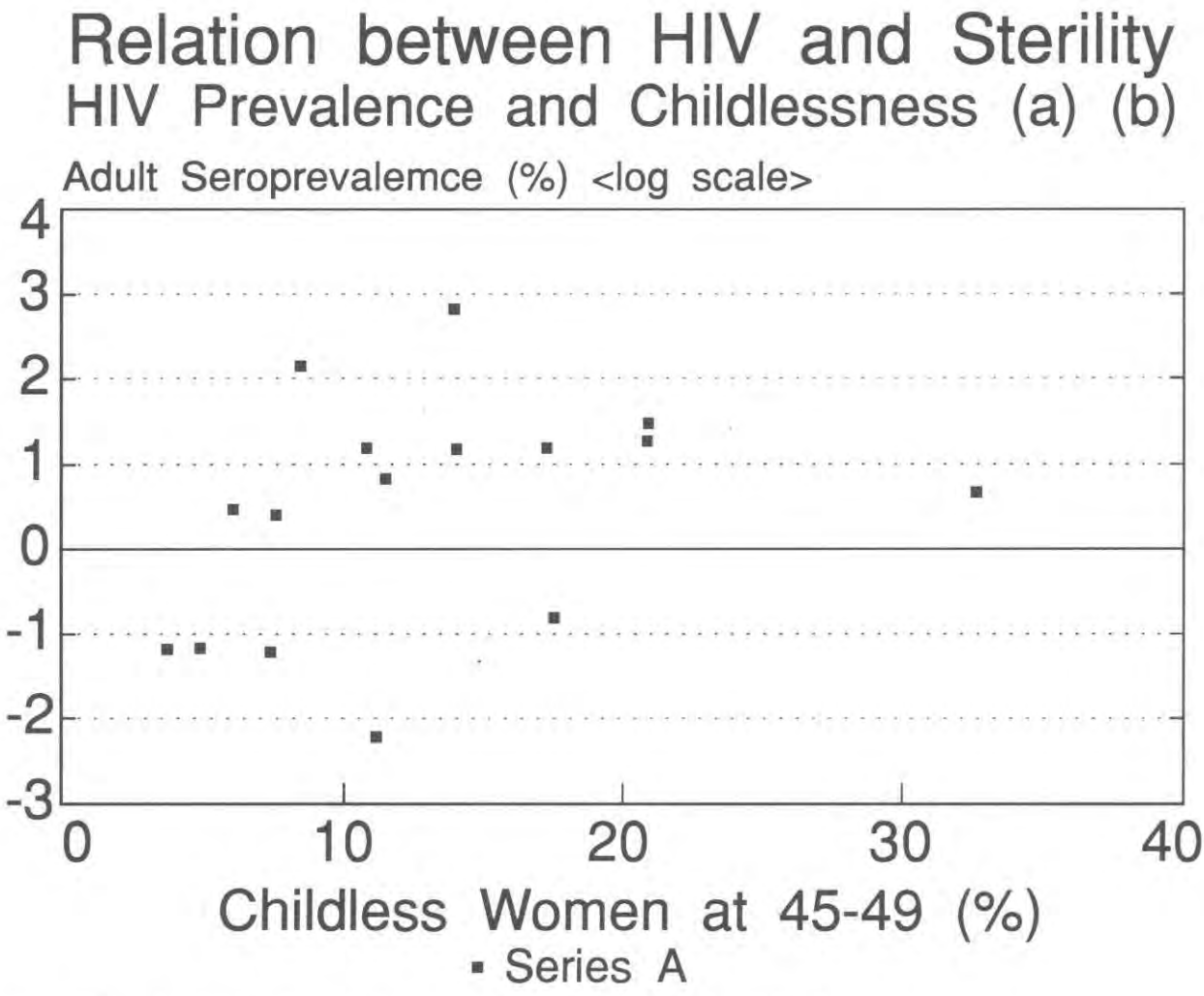
Panel C: Expected number of years before a child just born experiences the illness or death of one parent according to the mother's age at birth

Regimes of HIV (Level of infectivity)	Mother's age at birth		
	15	25	35
.00	14.2	13.6	12.8
.01	11.8	7.9	8.6
.10	7.9	2.7	2.7

TABLE IX: THE HEALTH EFFECTS OF HIV/AIDS ON VARIOUS ASPECTS OF THE FAMILY

Panel D: Projected proportion of children whose mo-thers are HIV/AIDS+ five and ten years the start of a hypothetical projection according to mother's current status

Years elapsed since the start of the projection			
5		10	
Mother's current status			
HIV/AIDS	Susceptible	HIV/AIDS	Susceptible
.63	.13	.46	.27



(a) Frank (1983); (b) Bongaarts [et al.] (1989)