

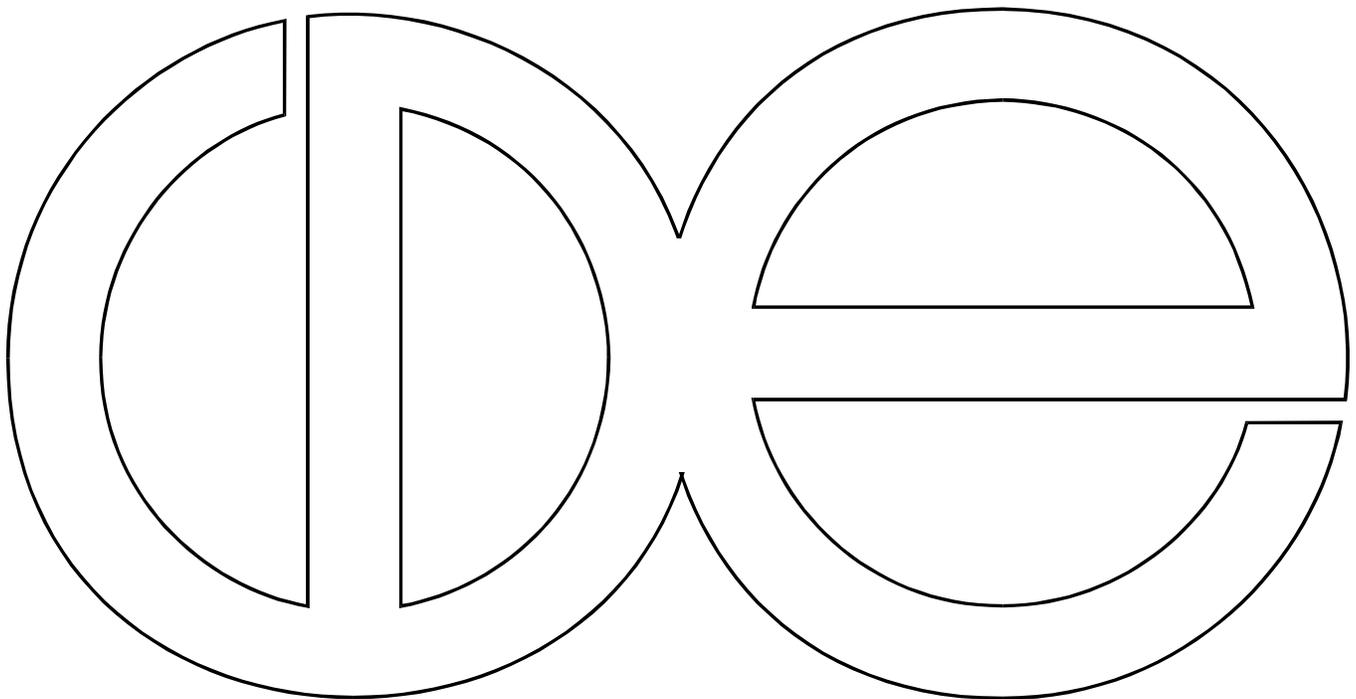
**Center for Demography and Ecology  
University of Wisconsin-Madison**

**The HIV/AIDS Epidemic, Kin Relations, Living Arrangements  
and the Elderly in South Africa**

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## INTRODUCTION

Although the effects of HIV/AIDS on individuals who contract it have been relatively well-known for sometime (Quinn et al. 1986), the understanding of the plethora of indirect effects and their pervasiveness in many realms of individual and social life is much less complete. Age selectivity together with the disease's relatively long periods of incubation and the associated morbidity and lethality may affect a number of social relations and social organizations that are either unique or distinctly more powerful than those observed for other diseases in Africa or anywhere else. In particular, the levels and age patterns of incidence of HIV and future increases in prevalence are likely to have a large impact on kin relations, residential patterns, household organization and the well-being of family members. Faced with the escalating burden of excess morbidity leading to the disruption of normal activities and functions, families and households are likely to adopt coping strategies to contain the damaging effects of the epidemics. An interesting issue is the magnitude and nature of costs borne by individuals and families as a consequence of the adoption of these strategies and whether or not these will be put into place without threatening the very fabric of family relations as we know it today.

Ten years ago, Palloni and Lee (1992) reviewed the potential effects on mortality levels at various ages that would affect household and family organization. The main idea is that when levels of widowhood and orphanhood rise as much as they could due to the HIV/AIDS effects on mortality alone (excluding effects on fertility and migration), the material basis of traditional kin relations (kin availability) and of household organization (residential patterns) will weaken or cease to operate. In their place, one could expect to see the emergence of new forms of social relations. In addition to projecting high levels of widowhood and orphanhood, the authors

anticipated the collapse of traditional family organization, kin networks, and the erosion of the foundations of typical household arrangements. They also predicted increasing prevalence of households where children live with grandparents in the absence of their parents and that this will have important implications for the well-being of children and the elderly alike.

In this paper, we update the work by Palloni and Lee and use a modified version of their model to calculate the demographic impact of HIV/AIDS on the elderly. Our evaluation rests on newly available data for South Africa as a whole and its provinces.

The AIDS epidemic is far worse in Southern Africa than it is in Central and Eastern Africa, where it first began. With its 5 million cases (UNAIDS 2002a), South Africa is currently the country with the largest number of people infected with HIV. The rapidity with which HIV has spread is exceptional. In less than a decade, adult HIV prevalence from antenatal surveys increased from 1 percent in 1990, 7.6 percent in 1994 to the current 26.5 percent (Department of Health 2003), a level which only follows levels of Zimbabwe and Botswana, the two countries with the world's highest prevalence (UNAIDS 2002b, 2002c). Based on a combination of vital registration data and estimates derived from AIDS modeling, Dorrington et al. (2001) attributed to AIDS a significant increase in mortality at young and middle adult ages since the late 1980s, and estimated that 40% of the adult deaths aged 15-49 in 2000 were from AIDS. Without treatment to prevent the progression from HIV to AIDS, they estimate that the cumulative number of AIDS deaths is expected to reach between 5 and 7 million by 2010.

Furthermore, in South Africa, like in much of the rest of Africa, the elderly have been until very recently primarily supported by intra- and intergenerational familial networks. In particular, coresidence with an adult child is a common form of living arrangement and a form of

exchange (Møller and Devey 1995). Thus what we observe in South Africa may be replicated in other countries with similar patterns of intergenerational relations.<sup>1</sup>

Strategies adopted by households and families to cope with the depletion of human and material resources induced by HIV/AIDS may range from changes in household structure, to reorganization of the division of labor in the domestic domain, shifts in norms regarding female, child and elderly labor force participation, and depletion of assets and cash reserves. The particular menu of strategies chosen will depend on the social group and some, though not all, of the changes in household structure introduced by HIV/AIDS will be reflected in observable shifts in living arrangements of the elderly. Increases in AIDS morbidity and mortality will reduce the availability of members of the young adult generation. Adult children will be sick or disabled for long periods of time and later die. They may lose the capacity to earn the income that would have been otherwise transferred to their aging parents. They may also require additional resources for their own support and medical care. Thus the elderly suffer a double burden with likely implications for their own health status and well-being: they become caregivers of the younger generations, first of their adult children and then of the AIDS orphans, and may find themselves without the income transfers from the middle generations, so that net resource flows may be *from* rather than *to* aging parents. Moreover, the physical and psychological well-being of older persons will be affected not only by the death of adult children and foregone transfers of income, goods and services, but also by the need to raise additional cash by diluting assets or deploying more hours of work to satisfy the increased burden entailed by the protracted nature of the illness. With its implied long-lasting health impairments on adult individuals, the disease jeopardizes households' ability to generate resources for the care of

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<sup>1</sup> The advent of a pension system in South Africa may be changing some of these patterns of generational transfers and may be consequential for the coresidence between elderly and some or all of their adult children.

households' most vulnerable members, namely, children and elderly, and thus aggravates the social and psychic costs of the illness (Ainsworth and Dayton 2001). This damaging sequel of the disease will start long before the time of death of those already infected. This phenomenon is what was referred to early on as the "bottom of the iceberg" (Palloni and Lee 1992:82). The effects of deterioration of the health status of adults on the well-being of children and the elderly is in all likelihood much larger than those implied by the direct effect via excess mortality.

Our central objective in this paper is rather modest since we only estimate the effects of HIV/AIDS on residential patterns of the South African elderly and evaluate observable changes in living arrangements of the elderly over less than a decade. We eschew assessment of other effects of the epidemic on the elderly but argue that these may be reflected, at least in part, in changes in residential arrangements. We use information from three data sources collected before and after the onset of the HIV/AIDS epidemic in South Africa: the 1991 census of the Republic of South Africa, the 10% sample of South Africa's first post-apartheid census conducted in 1996, and South Africa's 1998 Demographic and Health Survey (DHS).

We proceed in two steps. First, we evaluate macro models of the epidemic through backward and forward projections of HIV incidence and related mortality. These models yield estimates of expected availability of adult children for the elderly, lower bounds for the prevalence of sickness among the children born to elderly people, and ten to fifteen-year projections of changes in the availability of adult children and prevalence of sickness. Second, we contrast some of the epidemic's expected outcomes with observed changes in living arrangements of the elderly over time and across provinces. These contrasts depend on descriptions of observable patterns. We focus both on the impact of AIDS mortality as well as the burden of illness associated with the presence of sick adult children. A significant difficulty

made evident by these comparisons is that of identifying the direction and magnitude of changes in living arrangements of the elderly that can be unequivocally associated with the impact of HIV/AIDS.

## **PREVIOUS RESEARCH ON THE IMPACT OF EPIDEMICS ON FAMILIES AND HOUSEHOLDS**

Demographically speaking, the HIV/AIDS epidemic is not far removed from the large shocks suffered by pre-industrial populations. In fact, all evidence available to us seems to point to a catastrophe of much larger proportions. Although the parallel suggests that we could learn some lessons from the past by examining studies on the population impact of epidemics, famines and wars, this literature is in general devoid of systematic analysis of the complex effects on family and household organization. Attempts to assess the relation between past crisis mortality and the day-to-day operation of households, families and social relations are scarce. The effects most successfully examined are those directly related to global excess mortality, deficits in fertility, and increased regional displacement of individuals.<sup>2</sup>

Only rarely have extant studies of past population crises attempted to identify mechanisms translating raised levels of individual mortality or morbidity into shifts of the size distribution of households and the likelihood of fusion, fission, or outright disappearance of family units. An important exception is Livi-Bacci's (1978) assessment of the demographic effects of epidemics suffered by pre-industrial populations on the distribution of families by size.<sup>3</sup> We attempt to follow the lead contained in this handful of studies to understand the effects

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<sup>2</sup> For a summary of this literature, see Palloni (1990).

<sup>3</sup> This procedure was suggested by Livi-Bacci (1978) in his work on demographic crises. Livi-Bacci's original idea was to use this technique 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.

of HIV/AIDS on the living arrangements of the South African elderly, but adopt completely different assumptions to reflect the operation of a unique epidemic, with a distinct age pattern of incidence, protracted period of incubation and infectiousness, and singular lethality.

With the exception of a few studies on the direct economic costs for individuals and households (Ainsworth and Over 1999), most research on the effects of the African epidemic focus on particular members of families, such as mothers or children, and on the impact of adult male deaths which raise widowhood and orphanhood. Studies on the impact of HIV/AIDS on widowhood have focused on traditional behaviors such as widow inheritance in exposing women to HIV infection, and the changes in such traditional arrangements due to the epidemics in Uganda (Mukiza-Gapere and Ntozi, 1995). Ntozi et al. (1999a) found that stigmatization of AIDS widows in Uganda influenced their movements upon the loss of their spouse. Less healthy widows were more likely to leave their late husbands' homes and seek care in their natal villages, while healthier AIDS widows were more likely to remarry or form new sexual partnerships.

A review of a series of case studies on the impact of HIV/AIDS on orphanhood by Zaba and Gregson (1998) revealed that in areas with high HIV/AIDS prevalence, the prevalence of paternal orphanhood was higher than that of maternal orphanhood, and was attributed to polygynous unions whereby, at the father's death, all children born to his widows become orphans. In Tanzania, Urassa et al. (1997) found that 8 percent of children under 15 and 9 percent of children under 18 had lost one or both parents. In the region of Manicaland in Zimbabwe, the rapid increase in the number of parental deaths posed demands that exceeded the capacity of relatives to fulfill their traditional role of caring for orphans and triggered the emergence of child headed households (Foster et al. 1997). In the Kagera region of Tanzania

excess adult deaths not only implied higher levels of orphanhood but severely affected the nutritional status of orphaned children (Ainsworth and Semali 1998).

Community studies provide evidence for the effects of the epidemic on household organization (Barnett and Blaikie 1992; Boerma et al. 1999; Ntozi and Zirimenya 1999; Urassa et al. 1997). In Uganda's Rakai district, two or three generations with at least one orphan, and individuals living alone were more common in AIDS-affected households than unaffected ones, and in a significant fraction of households containing AIDS victims, grandparents cared for orphans (Barnett and Blaikie 1992). The burden of AIDS mortality and morbidity for households is shared by household members in a strict hierarchy. In Uganda, care of AIDS orphans was left to the surviving parent, then to grandparents, followed by older orphans, step-parents or members of the extended family such as uncles. Paternal orphans were more likely to be fostered by uncles than cared for by their mothers, because children belong to their father's lineage (Ntozi et al. 1999b). Grandparents were the main care providers to AIDS orphans in a study in Zimbabwe (Foster et al. 1995). Data from the Kisesa community study show that terminally ill people travel back to rural homes in seek of care by the extended family (Urassa et al. 2001). Elderly parents are the most likely caregivers of their infected children because parents are the most sympathetic and are most likely to be informed of their children's AIDS diagnosis first (Ntozi 1997). Strikingly, similar patterns of caregiving were found in Thailand (Knodel et al. 2001) where 27% of adults with "symptomatic" AIDS were cared for by a parent. Two-thirds of the adults who died of an AIDS-related disease had lived with or next to a parent by the terminal stage of illness, and a parent, usually the mother, had acted as a main caregiver for about half. For 70%, either a parent or other older-generation relative had provided at least some care. The vast majority of parents were aged 50 or more and many were 60 or older.

The foregoing summary identifies two important albeit weak regularities. **First**, most underscore transformations of living arrangements to accommodate AIDS orphans and widows, with an increased prevalence of households composed by the elderly with their widowed children and grandchildren, and households with grandparents and grandchildren but no member of the intermediate generation. **Second**, there is a rearrangement of the household to adjust to the needs to care for sick adult children. These changes may lead to increases in headship among the elderly and to a more influential presence of households composed by elderly parents, their adult children, and grandchildren.

Besides the somewhat elusive evidence connecting HIV/AIDS and concomitant changes in families and households, demographic models which attempt to identify the population level effects of HIV/AIDS have not succeeded in providing a benchmark against which to evaluate empirical evidence (Zaba and Gregson 1998). For example, although orphanhood is the most amenable outcome to modeling because it only requires assumptions about mortality and fertility, modeling the impact of HIV/AIDS on orphanhood is complicated by time lags between the onset of HIV and orphanhood and to difficulties to quantify pre-HIV/AIDS levels and patterns of orphanhood. Models predicting the impact of HIV on widowhood require additional conjectures about nuptiality and are more complicated to implement, especially in Sub-Saharan Africa where polygyny and re-marriage are frequent.<sup>4</sup>

Changes in household organization in general and in the living arrangements of the elderly in particular have proven to be even less amenable to modeling than orphanhood or widowhood. This is because, in addition to information on demographic determinants, one needs

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<sup>4</sup> The practice of levirate, or remarriage to members of the widow's former husband's family, is common in parts of Africa (Potash 1986). To the extent that it takes place rapidly after widowhood, levirate will conceal the impact of excess adult mortality. Similarly, child fosterage accompanied by a blurring of the distinction between biological and foster parents will lead to underestimates of the impact of adult mortality.

to assess the influence of propensities to co-reside and of internal migration flows, both of which may mimic the effects of HIV/AIDS on availability of kin and confound the epidemic's independent effect. Efforts to isolate the contribution of each of these factors are rare as they generally require the combined use of simulation and empirical observations. One study uses microsimulation, in combination with aggregate demographic analysis, to estimate how patterns of coresidence of elderly parents in Thailand would adjust in response to the HIV/AIDS epidemic (Wachter, Knodel and VanLandingham 2002). The authors project that 11.9% of the present generation of Thai elderly (50+) will lose one or more children to AIDS, and 13% of those who lose at least one child will lose two or more before death. They also estimate that, of the cohort of Thai men and women age 55 in 1995, one in 9 could expect to experience the loss of at least one child to AIDS, while one in 14 could expect to have lived with child during illness and have provided care.

The most important lesson emerging from this brief review of previous studies is that even the most direct effects, those working through augmented levels of orphanhood and widowhood, present themselves in a veiled form or not at all in aggregate data. Problems with identification of the proper time lags, imperfect knowledge of relations prevailing in the period preceding the epidemic, and the widespread use of norms typical of most African societies, such as those regulating fosterage and remarriage, tend to mask or dampen the observed effects of the epidemic. Some of these problems can be circumvented in studies focusing on individual households within a well-demarcated area and with well-designed follow-up protocols, such as those carried out by Ainsworth and Over or Urassa, Boerma and colleagues in Tanzania. But in most other cases we must resort to procedures that address head-on identification problems that conceal the unfolding of HIV/AIDS effects.

## THE IDENTIFICATION PROBLEM

### Minimum Identification Conditions

Efforts to estimate empirically the impact of HIV/AIDS on elderly residential arrangements can be successful only if a set of minimum identification conditions are satisfied at the outset. These conditions are associated with processes that either uniquely determine or loosely bound the observable patterns of the elderly living arrangements.

Living arrangements of the elderly are determined by two factors. The first is a function of purely demographic forces and influences the *availability of kin*. Pre-existing levels and patterns of mortality, fertility and migration limit the supply of kin that could reside with older people and, therefore, affect the ability to observe certain types of living arrangements. The second factor is the set of individual propensities to live with blood kin and other relatives. Residential propensities are a function of culturally bounded patterns of preferences and are likely to vary greatly across social classes and ethnic groups within the same society.

Thus the prevalence of living alone or with spouse but no children among elderly aged  $x$  at time  $t$ ,  $P(x, t)$ , is simply the product of  $D(x, t)$ , a measure of the supply of children available to the elderly aged  $x$ —the proportion of elderly who have surviving children to live with—and  $p(x, t)$ , a measure of the conditional probability of residing with one of the surviving children.<sup>5</sup> It could be that since excess mortality associated with HIV/AIDS affects  $D(x, t)$ , one could argue that the difference between estimates of demographic availability in contexts with and without HIV/AIDS is sufficient to identify the effects of HIV/AIDS on living arrangements of the elderly. But this line of thought ignores a number of difficulties. First, when examining changes in elderly's living arrangements, both  $D(x, t)$  and  $p(x, t)$  need to be identified simultaneously. While changes in  $D(x, t)$  can be assessed through a variety of procedures, including micro and

macrosimulations, estimation of  $p(x, t)$  is almost always problematic. This difficulty has already confronted scholars who have worked on microsimulation of households and families (Wachter, Hammel and Laslett 1978; Ruggles 1987) but has been met with no straightforward solution. Furthermore, the relationship between the observed distribution of living arrangements of the elderly, demographic availability of kin and individual propensities involves sources of misidentification which, if not properly neutralized, will bias inferences regarding the effects of HIV/AIDS on living arrangements of the elderly. While some of these sources of misidentification are unique to South Africa, others apply to broader contexts.

In the foregoing formulation, we assumed that the estimation of past levels and patterns of vital events (including nuptiality and migration) is unproblematic. While this may be so for fertility, mortality, and nuptiality, it is not so for migration. Migration exerts a severe drag on the supply of adult children, particularly when the operation of social networks as a force encouraging migration results in residential changes of several members of a household simultaneously or in rapid succession. In the absence of proper controls for outmigration flows, one can mistake a decline in  $P(x, t)$  for changes in other demographic determinants, particularly mortality. In the absence of direct estimates of the tug of migration, *a first minimum condition for the identification of the effects of HIV/AIDS is a comparison of measured effects across areas exposed to similar incidence of HIV/AIDS but experiencing different levels of migration.*

The second source of misidentification is peculiar to the nature of HIV/AIDS. Because the median duration from infection to full-blown AIDS and mortality in Sub-Saharan Africa is about 7.5 years (Boerma, Nunn and Whitworth 1998), one cannot expect to see large changes in patterns of living arrangements until sometime after the onset of the epidemic. In South Africa, the first AIDS cases were reported in 1984-1985 (Sher 1986), but the full force of the epidemics

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<sup>5</sup> To simplify, we ignore the possibility of age variation (across children and elderly) in  $p(x, t)$ .

could not have been felt before 1995. Using a data source for a year before 1995 is tantamount to choosing a baseline against which changes induced by the epidemic can be measured. The selection of a target date is also problematic for it should be sufficiently distanced from the benchmark to allow time for the effects to accumulate. *Thus, a second minimum identifying condition is to examine information on residential arrangements after 1995 relative to those prevailing sometime before this benchmark date.*

Third, the above formulation rests on a “whopper” assumption—to paraphrase Ruggles’s terminology (Ruggles 1987)—namely that changes in demographic forces do not significantly alter individual residential preferences. However, sudden changes in mortality levels could simultaneously shift preferences among kin by decreasing the propensity of elderly to live with a surviving adult child. If we are unaware of this, we will attribute a larger fraction of changes in  $P(x, t)$  to observed changes in mortality levels than we ought to, with an ensuing exaggeration of the effects of exogenous changes in mortality due to HIV/AIDS. Conversely, a sudden rise in adult morbidity may increase the propensity to live with a surviving (and possibly ill) adult child. The resulting increase in  $p(x, t)$  will offset the mortality-induced decrease in  $D(x, t)$  and yield an error in the opposite direction, namely an underestimation of the demographic effects of excess mortality. These examples ignore time lags and the precise mechanisms through which demographic availability influences residential preferences, but the main idea should be transparent: if our mission is to assess the impact of an external event on  $P(x, t)$  and to determine how much of this change occurs via changes in  $D(x, t)$  alone, identification will be problematic as long as we do not account for the impact of changes in  $D(x, t)$  on  $p(x, t)$ . It follows that *a third minimum condition for the identification of HIV/AIDS effects is the assessment of changes during*

*a period of time short enough to support the assumption that endogenous effects have not significantly altered residential preferences prevailing prior to the onset of HIV/AIDS.*

It should be noted, however, that if one is interested in the *total effect* of HIV/AIDS, the third identification condition is superfluous. Indeed, in this case all we need is a rough measure of change in  $P(x, t)$ —whether reflecting changes in  $D(x, t)$  or in  $p(x, t)$  induced by the epidemic itself. The only caveat is that inconsistent estimates of the effect of the epidemic will be obtained if any of the changes in  $p(x, t)$  are exogenous to the event of interest. Furthermore, if changes in  $D(x, t)$  and  $p(x, t)$  offset each other perfectly, no changes in  $P(x, t)$  will be observed, with the analyst's conclusion that the HIV/AIDS epidemic is inconsequential for living arrangements.

### **Identification Conditions in South Africa**

In South Africa, identification problems are exacerbated by the fact that the period of fastest growth of the incidence rates in HIV/AIDS coincided with a period of tumultuous social and demographic transformations that occurred just before and after the collapse of *apartheid*.

*Apartheid* and its associated system of *separate development* imposed restrictions on spatial mobility, education, and employment of black Africans, by forcibly resettling them to the homelands, four of which were made “independent states” in the 1960s and 1970s (Transkei, Bophuthatswana, Venda and Ciskei, or the TBVC states). This regime supported a migrant labor system, of circular character, which involved a large segment of the African adult population and affected almost every African household. Through the enforcement of influx control laws, African men working in the mining industries, on white farms, and in towns and cities were systematically denied the right to settle there with their families. Single sex hostels were built in all major cities to host rural African laborers. This system encouraged male out-migration but

kept families divided by forcing heavy restrictions on residential changes of migrants' wives, children and elderly relatives (Murray 1980, 1987; Russell 1998). What were once undivided rural households became "stretched households", that is spatially divided units connected by kinship and remittances (Spiegel, Watson and Wilkinson 1996). After the collapse of apartheid, migration involved broader age groups as well as women (Posel and Casale 2002; Collinson et al. 2003). The intensification of migration resulted in the rapid peri-urbanization of formerly rural areas bordering large metropolitan areas and in the swelling of the population of black townships living in backyard shacks (Kinsella and Ferreira 1997; Spiegel et al. 1996; Percival and Homer-Dixon 1995).

If death were the only reason for children to be cared for by grandparents, we would expect a higher proportion of the elderly living in skipped generation households, which are households composed by grandparents and grandchildren without members of the middle generation, in areas where the prevalence of HIV/AIDS is high. But in South Africa, children may "lose a parent" to migration as well as death (Bray 2003) and migration provides a condition for grandparents to take in and support their grandchildren (Smit 2001). Thus, an increase in prevalence of grandparents living with their grandchildren without the presence of an adult child may not be due to HIV/AIDS but to high rates of population mobility.

Moreover, in South Africa, there is a distinctive reason for alterations in the living arrangements of the elderly: a pension system that was extended to all South African elderly in 1993. With rising rates of unemployment, pension sharing with an elderly relative has become a reason for adult children to join their elderly parents' households and share the elderly pensions (Burman 1996; Møller and Sotshongaye 1996). Nearly 80% of age-qualified Africans reported receiving a social pension in 1996 (Case and Deaton 1998). Similar to the effects of HIV/AIDS

which may draw adult children back to their elderly parents' homes, the elderly pension system may affect propensities of adult children to co-reside with the elderly thus introducing another source of misidentification.

### **Operationalization of Minimum Conditions**

In order to partially satisfy the identification conditions, we utilize three different strategies. The first strategy is model-based and consists of estimating expected demographic impacts, that is changes in  $D(x, t)$  associated with mortality increases due to HIV/AIDS. These estimates are obtained through the application of simple multi-state models relying on estimated patterns of age-specific HIV incidence, incubation periods and HIV/AIDS related mortality. They provide us with a benchmark for the magnitude and direction of expected changes in living arrangements of the elderly ( $P(x, t)$ ) due to changes in availability of adult children ( $D(x, t)$ ) in the absence of changes in  $p(x, t)$ . They also provide a baseline to evaluate the burden of disease borne by their adult children. Observed data derived from data sources for the period 1991-1998 will then be compared with expected (model-based) values. This first strategy contributes toward the first and second identification conditions since it provides us with a sense of the magnitude of the expected changes in availability due to changes in demographic forces in the absence of endogenous or exogenous changes in residential propensities.

Second, for the comparison of model outcomes with empirical data, we choose the period 1991-1998 which brackets the sharp increase in the incidence of HIV/AIDS. Differences in patterns of living arrangements during this period will give us leverage to detect changes in living arrangements due to changes in  $D(x, t)$ . Although the interval between 1991 and 1998 is wide enough to capture time lags inherent in the progression of the epidemic and address the

second identification condition, it may not be short enough to contribute to the third identification condition. In fact, we may not be able to fend off the threats to identifiability originating in the reciprocal relations between demographic availability and propensities. However, our inability to distinguish changes in living arrangements due to changing propensities from changes due to demographic availability will be a less serious issue if we are interested in assessing the *overall* effect of HIV/AIDS. It will only be a problem if the propensities are changing due to exogenous factors (e.g., modernization).

Third, to address the first identification condition and distinguish the effects of HIV/AIDS on living arrangements of the elderly from those of migration, our analyses will compare conditions across South African provinces which differ in levels of HIV/AIDS prevalence and magnitude and direction of migration flows. By contrast, there is no similar strategy to reduce the confounding effects of the pension system on patterns of elderly coresidence. Fortunately, changes in pension laws are more likely to affect certain types of coresidential arrangements than others. While coresidence with adult children could be easily related to the issuance of pension receipts, changes in coresidence of the elderly with **grandchildren** (but not their parents) are less likely to be related to availability of pension payments to the elderly.

## **DATA ON LIVING ARRANGEMENTS**

### **Data Sources**

Our observation of changes in  $P(x)$  relies primarily on the analysis of the last apartheid census taken in 1991, the 10% public sample of the first post-apartheid census taken in 1996, and the 1998 South Africa Demographic and Health Survey.

The 1991 census of the population of the Republic of South Africa (RSA) is well known for its *apartheid*-induced distortions in coverage which produced significant underenumeration especially in the self governing territories (SGTs), i.e., the six homelands which remained part of the RSA after “independence” was granted to the four TBVCs (Orkin 2000).<sup>6</sup> Even after various adjustments, the underenumeration of the black African population was estimated at 17% (Zuberi and Khalfani 1999). The granting of “independence” to the TBVCs further complicates the comparability of data sources in the 1990s, because in 1991 the TBVCs conducted their own censuses.<sup>7</sup>

As the first post-apartheid census, the 1996 census counted the whole South African population. We use the 10% public sample of the 1996 census, based on a systematic sample of households stratified by district and province. The individual level data file includes all members of the selected households as well as an independent 10% systematic sample of people in special institutions (old age homes, hostels, prisons, schools, etc.) (Statistics South Africa 1998). Because different sizes of territory covered and different provincial boundaries complicate the comparison of the population enumerated in the 1991 and 1996 censuses, we use

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<sup>6</sup> In many urban townships, informal settlements, and rural areas that fell into the homelands (SGTs), where residents were overwhelmingly African, mapping was not uniformly available and many areas were not demarcated into census enumeration areas. Teams of enumerators “swept” through some of these areas without prior demarcation or lists. In other areas, considered inaccessible due to political unrest or other reasons, dwellings were counted using aerial photographs, and population characteristics imputed using household densities obtained from sample surveys. In perhaps the most extreme demonstration of the impact of apartheid on official statistics, when areas covered by aerial photographs and sample surveys were found with an unexpected number of women (wives and children of male migrant workers were barred from cohabiting with their husbands in areas of migration destination) women were reclassified as males. This resulted in the reclassification of 250,000 women in these areas (Orkin 2000). This suggests that 1991 census results for provinces containing former SGTs KwaZulu Natal, Limpopo and Mpumalanga should be regarded with caution.

<sup>7</sup> The TBVCs were reintegrated into South Africa between 1992 and 1993, and provincial boundaries redefined to yield nine provinces.

a version of the 1991 census which allots the RSA population to the same geographic areas of the nine South African provinces in the 1996 census.<sup>8</sup>

The 1998 Demographic and Health Survey is directly comparable to the 1996 census. The survey employed a two-stage sample using the 1996 Census Enumeration Areas as the sampling frame with sample numbers of households proportional to the number of households in the 1996 census (Department of Health 2002).

### **Information on Relationship to Household Head**

Our focus on household dynamics constrains the analyses to the population enumerated in households and excludes the population living in hostels and institutions. 6.6% of the population enumerated in the 1991 census and 4% of the population enumerated in the 1996 census lived in hostels or institutions.<sup>9</sup>

From the household roster of each data set which contains information on the relationship of each household member to the household head, we calculated the distribution of living arrangements of the elderly, widowhood and orphanhood rates and other indicators pertaining to elderly residential arrangements. However, not all data sets have the same level of detailed information on the relationship to household head. To ensure comparability, several adjustments had to be made. The 1991 census has the smallest number of relationships to household head

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<sup>8</sup> The former TBVCs are located in Eastern Cape (Transkei and Ciskei), Northwest Province (Bophuthatswana) and Limpopo (Venda), and correspond to 68 percent, 65 percent and 16 percent of the 1996 provincial populations respectively. Thus, comparisons between the provincial populations enumerated in 1991 and in 1996 should be drawn keeping in mind that the populations of Eastern Cape and Northwest province in 1991, and to a lesser extent of Limpopo, are much smaller than the 1996 populations enumerated in these provinces because of the absence in the 1991 census of the TBVCs populations.

<sup>9</sup> In the 1991 census, the population living in hostels and institutions was enumerated as individuals who, according to type of dwelling categorization, lived in hostels or retirement rooms. In the 1996 census, individuals living in hostels and institutions were administered an individual questionnaire, not a household questionnaire. We also excluded 32,973 cases (or 0.9% of the total 1996 census population) who were reported as living in households but who were administered an individual questionnaire and for whom the relationship to household head was missing.

(spouse, child, other family, unrelated), followed by the 1996 census (spouse, sibling, child, grandchild, grandparent, other family, unrelated), and by the richer 1998 DHS (head, spouse, child, child-in-law, grandchild, parent, parent-in-law, sibling, other relative, adopted/foster stepchild, not related). To increase our ability to capture key living arrangements of the elderly by successfully identifying grandchildren in the 1991 census, we imputed the relationship of grandchildren of head by estimating the proportion of “other family” who are grandchildren of head in a nationally representative survey conducted on a date close to 1991 and applying this proportion to 1991 data to obtain grandchild status.<sup>10</sup> We then degraded the 1996 and 1998 data by ignoring information unavailable in the 1991 census, so that, beside household head, the only relations to head used to construct household types were spouse, child, grandchild, other family, and unrelated.

## **A PROFILE OF SOUTH AFRICA’S PROVINCES**

Table 1 displays a profile of South African provinces for each year of observation. Based on these data, South African provinces can be assigned to one of two distinct groups of HIV prevalence defined by their level of severity and to one of two distinct regimes of migration defined by the level of urbanization, the sex ratio of their population and information gathered from the literature about whether a province is sending or receiving migrants. According to

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<sup>10</sup> The closest data source to the 1991 census is the South Africa’s Living Standard Measurement Survey (LSMS) conducted by the South Africa Labour and Development Research Unit at the University of Cape Town in association with the World Bank in 1993. The imputation procedure was carried out as follows: 1. We prepared a three-way table which cross-tabulates relation to head in 10-year age categories with 10-year age of head categories in the 1993 LSMS data. 2. For age-of-relation to age-of-head cells where the proportion of grandchildren was greater than 80% in 1993 data, a 100% grandchildren status was imputed to 1991 data. 3. For cells where the proportion of grandchildren was between 10% and 80%, logit regression models were fitted to 1993 data with the binary dependent variable 0/1 for grandchildren, and sex, marital status and age as independent variables. The parameter estimates thus obtained were used to calculate the probability of being a grandchild in 1991. Where probabilities were greater than 0.5, grandchild status was imputed. The success rate of imputation of grandchild

antenatal surveillance data, as of 1998, six provinces display adult HIV prevalence rates higher than 15% (Ubomba-Jaswa 2000). The most rapid increase in HIV prevalence over the 1990s has been experienced by KwaZulu Natal followed by Mpumalanga, Free State, Gauteng and Northwest Province, while the bulk of the increase of HIV prevalence in Eastern Cape occurred in the latter half of the 1990s. We regard these prevalence levels as high enough to have an observable impact on household organization, especially if compared with the, by Southern African standards, low to moderate levels of Limpopo, Western Cape and Northern Cape.

As for migration, Gauteng and Western Cape, with the highest average incomes and levels of urbanization, are major destinations for migrants. Since the discovery of gold in the late 19<sup>th</sup> century, Gauteng and Johannesburg have become beacons of attraction for migrant labor from other South African provinces as well as from neighboring countries. Today, due to its high concentration of industrial and commercial resources, Johannesburg continues to attract migrants from rural areas of Limpopo, KwaZulu Natal, Mpumalanga, and Eastern Cape. Western Cape, with its provincial capital Capetown, receives a large influx of migrants, especially from neighboring Eastern Cape and the former Transkei region (van der Berg et al. 2002). Free State contains some of the largest goldfields of South Africa, and, thanks to its dense network of roads, it receives substantial labor migration from Eastern Cape, as well as from neighboring provinces. Kwazulu Natal's major urban centers also receive migrants from Eastern Cape, especially from the former Transkei region (Percival and Homer-Dixon 1995). The province has a good transportation system which favors high rates of intra- and interprovincial population mobility. Mpumalanga and Northwest Province are predominantly rural, but have large peri-urban settlements near the border with Gauteng which attract large concentrations of

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status was evaluated by implementing this procedure based on 1993 data on 1996 census data where grandchild status is known. Success rate of imputation was 86.94%.

rural migrants from the provinces' more remote corners. Limpopo and Eastern Cape, with two of the former TBVC, predominantly rural and among the poorest, are historically two major sending regions of labor migration.

Despite the possible value of this categorization of provinces for our ability to identify the effects of HIV/AIDS on elderly living arrangements and to separate them these effects from those of migration, the association between provinces and household outcomes is fragile for at least one important reason. This is because we assign consequences of HIV/AIDS mortality for the living arrangements of the elderly to provinces with high HIV prevalence and neglect to consider the indirect effects on provinces with low to moderate prevalence. Yet some of the consequences of HIV/AIDS are likely to be experienced by the elderly in low prevalence regions as well as by those living in high prevalence regions. Consider, for example, the hypothesized increased proportion of the elderly living with grandchildren but no adult children in areas with high HIV prevalence. Because of the well established relation between mobility and the spread of high-risk sexual behavior leading to HIV infection (Hunt 1989; Pison et al. 1993; Quinn 1994; Nunn et al. 1995; Lurie et al. 1997), migrant husbands may pass on the infection acquired in urban and peri-urban areas or in mining towns to their wives in rural areas. The death of both parents will entrust children to the care of grandparents in migration sending areas. Under this scenario, HIV mortality would be as likely to increase the proportion of skipped generation households in low-prevalence areas which are also sending areas of migration, thus complicating our ability to obtain the first identification condition.

## THE DEMOGRAPHIC IMPACT OF HIV/AIDS: A SIMPLE MACROMODEL

### Model

We focus on an elderly woman aged  $x$  ( $x \geq 60$ ) who is alive in a target year, say 1995.<sup>11</sup> Hereinafter we refer to this woman as the target or target person, and to the year 1995 as the target year. Our approach consists in back-projecting women alive at the target year to the time when they started reproduction and then projecting them forward to reproduce their childbearing experience as well as their children's experiences with HIV/AIDS and mortality. Backward projection is carried out using estimates of AIDS-free mortality schedules and childbearing schedules for the period 1900 onward. Throughout we assume that childbearing and mortality are independent events for the subpopulation of target females.

Our goal is to calculate the following quantities: (a) the probability that her female children born alive when the target was aged  $x-y$  at time  $t-y$  have survived healthy to time  $t$ , or have not experienced HIV by time  $t$ ,  $SI(y,t)$ ; (b) the probability that they have survived to  $t$  but contracted HIV along the way and are alive but ill at time  $t$ ,  $QI(y,t)$ ; and, finally, (c) the probabilities that they have died due to HIV,  $QID(y,t)$ , or due to other causes,  $Q(y,t)$ . The time variable  $y$  varies from a minimum equal to  $x-50$  to a maximum equal to  $x-15$ , thus constraining the childbearing period of the target to be within ages 15 and 50.<sup>12</sup> The expressions for each of these functions are defined in the Appendix. With knowledge of the time distribution of children

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<sup>11</sup> We choose to work with female targets and her female children for convenience. However, as it happens, it is also a choice with non-trivial implications. First, relative to the male age pattern of HIV, the female age pattern is skewed towards younger ages. This simply means that our estimates of burden of illness have a younger than average age profile and may understate the problems associated with the oldest targets. Second, it is grandmothers who are more likely to be burdened with the care of grandchildren left by ill or dying adult children. In this sense the choice of female targets is justified for reasons other than computational convenience.

<sup>12</sup> We assume that none of the target persons could have contracted HIV prior to age  $x$ . Because we use a minimum value for  $x$  of 60, the assumption is sensible but not entirely accurate since some of these women could have been infected in the ten years prior to 1995. But since the incidence rates between age 49 (approximately attained the year the epidemic started) and 60 (attained in the middle of the census year 1996) are extremely low, the assumption is not at all limiting. Even if the assumption departs from reality, our calculations will be in error only if the

ever born (or the age pattern of fertility to which women aged  $x$  at time  $t$  were exposed during childbearing),  $N(x-y, t-y)$ , we can calculate the weighted probabilities of having a child in any of the four statuses defined above. This is achieved multiplying  $N(x-y, t-y)$ , for every permissible value of  $y$ , by each of the quantities defined above. These weighted values are the **average probabilities for an elderly woman aged  $x$  at time  $t$** . In particular,  $N(x-y, t-y) * QI(y,t)$  is the average fraction of all children born to the target person who are infected with HIV at age  $y$  at time  $t$ ;  $SI(y,t) N(x-y, t-y)$  is the average probability of having a child aged  $y$  at time  $t$  who is healthy; and, finally,  $N(x-y, t-y) * (QID(y,t) + Q(y,t))$  is the average probability of having lost a child to either HIV/AIDS or to mortality due to other causes.

### **Outcomes from the Model**

The main outcomes from the model track the history of illness and mortality experienced by the target person's children. Calculations can be fine-tuned to project forward or to assess the target person's status some years ahead of the initial date of calculation.<sup>13</sup> In particular, we estimate the aforementioned quantities for the cohort of elderly aged 60, 65 and 70 in 1995 and then project these forward ten years to 2005, thus assessing the experience of these cohorts when they are aged 70, 75 and 80 in 2005. We also estimate the quantities for those aged 60, 65 and 70 in the year 2000, and similarly project these forward to the year 2010. Thus, we are able to trace the experience of those aged 60, 65 and 70 in 1995, through the years 2000, when they are aged 65, 70, and 75 respectively, 2005, when they are aged 70, 75 and 80 respectively, and 2010, when they attain ages 75, 80 and 85.

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childbearing patterns to which targets not affected by HIV and the mortality and HIV incidence pattern of her children are different from those that apply to target persons who were infected prior to the target year.

<sup>13</sup> Throughout we start out with 1995 for convenience and for its closeness to 1996, the year of the first post-apartheid census.

Armed with knowledge of the distribution of mothers by survival status of children ever born for a period, we can estimate the probabilities of having a given number of children alive and healthy or a given number of children alive with HIV or dead due to HIV or other causes. This extension is straightforward and relies on the quantities defined before and on the estimated distribution of mothers by number of children ever born. If, for example, we are interested in estimating the probability of  $r$  children alive and not affected by HIV for a target aged  $x$  at time  $t$ , we use the following expression:

$$J_{xt}(r) = \sum_{\{0 \leq j \geq r\}} w(j) * [C(r,j) * (\sum_{\{0 \leq y\}} N(x-y,t-y) * SI(y,t))^r * (\sum_{\{0 \leq y\}} N(x-y,t-y) * (1-SI(y,t)))^{(j-r)}],$$

where  $C(r,j)$  is the quantity  $j!/(r!(j-r)!)$ ,  $N(x-y, t-y)$  is the standardized fertility rate (adding up to unity) at age  $x-y$ ,  $w(j)$  is the probability of having exactly  $j$  children ever born, and  $SI(x,t)$  is as defined before. Simple modifications of this expression lead to the probability of exactly  $r$  children alive and infected, and  $r$  children dead due to non-HIV/AIDS related causes or due to HIV/AIDS. Perhaps the most important quantity is the probability of having 0 children alive or 0 children alive and with no HIV. These are direct measures of demographic availability and potential burden of illness respectively.

### Required Inputs

Estimation of model outcomes depends of six pieces of information. The first and most important are the yearly HIV incidence rates from the onset of the epidemic until time  $t$ . The second is the incubation function that determines the waiting time in the infected state. The third is mortality of healthy individuals, of individuals infected with HIV, and of those with full-blown AIDS. The fourth is the time distribution of children ever born or, equivalently, the fertility function approximating the childbearing experience of the target population. The fifth is the time

distribution of targets by number of children ever born,  $w(j)$ . Finally, we need to have an approximation of the mortality schedule experienced throughout the childbearing period of the target persons. The nature of these inputs is described in the Appendix.

## RESULTS FROM THE MODEL

### Prevalence and Incidence at the National and Provincial Levels<sup>14</sup>

Figure 1a displays observed, fitted, and ‘adjusted fitted’ values of cumulated incidence for South Africa. The ‘adjusted fitted’ values are obtained after correcting the associated post-peak incidence using a model-based procedure outlined in the Appendix. Figure 1b shows the fitted and adjusted values of annual incidence rates consistent with estimated cumulated prevalence.<sup>15</sup> Two points are worth mentioning. First, the fitted cumulated incidence shows a peak of about 0.45, a value lower than those utilized by the United Nations and UNAIDS (Zlotnik, personal communication). However, after adjusting the post-peak incidence rates we obtain a ceiling of about .53, a value more consistent with those imputed by other researchers. Second, although fitted and observed values are hard to distinguish from each other, we downplay this feature since the ‘observed’ values are the result of operations that are of unknown nature to us.

Figure 2 shows adjusted incidence for South Africa and contrasts these with two provinces representing high and low HIV prevalence, KwaZulu Natal and Limpopo. Note that the incidence rates in all three settings peak around the same year but at different levels,

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<sup>14</sup> To simplify the illustration of the model results, we will discuss information for South Africa as a whole and for the provinces of KwaZulu-Natal and Limpopo only, to represent the expected patterns in a province with very high HIV prevalence and in a one with low or moderate levels.

<sup>15</sup> In all cases, and unless explicitly stated, we use the concept of incidence rates to refer to an occurrence-exposure rate, in strict analogy to a force of mortality. We discard the definition of incidence rate that contains in the

suggesting heterogeneity of ceilings and of stable incidence rates but not a different timing for the epidemic.

### **Households of the Elderly and HIV/AIDS Prevalence**

Estimates of HIV prevalence can be used in simple ways to calculate the prevalence of elderly households with at least one HIV-infected adult child. The estimates are calculated by province and obtained as follows:

$$D_k = \sum_{r} g_k(r) (1-p_k)^r,$$

where  $k$  denotes the province,  $r$  denotes the number of adult (15-49) members in the household,  $p_k$  is the observed HIV prevalence among adults in the province, and  $g_k(r)$  is the fraction of all households containing an elderly person that include exactly  $r$  adult members. The values of  $D_k$  are displayed in Table 2. Although the table is suggestive, the estimates rest on an assumption of independence that is likely to be violated. To the extent that infection of one adult member of the household is a marker for exposure for all other members of the same households (spouses and children), the quantities in Table 2 will overestimate the fraction of households with infected members. A bias in the same direction is possible due to the fact that the epidemic tends to cluster in social and ethnic groups. Since the expression overlooks such heterogeneity, it will generate overestimates of households' HIV/AIDS prevalence, more so in provinces where heterogeneity of social groups is paramount. Provinces with higher average adult household size will tend to show a higher probability of at least one member infected with HIV/AIDS even if the overall prevalence is relatively low. The figures in Table 2 show that in KwaZulu Natal one should expect about 37 percent of households (with at least one adult member) having at least

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numerator all the events of interest and in the denominator the entire population, exposed or not, as is frequently done in the literature.

one infected adult. This is a remarkably high value, even if upwardly biased. On the other hand, there are provinces that are hardly touched by the epidemic, as in the case of Western Cape.

### **The Children of the Elderly and their Experience with HIV/AIDS**

Figure 3a displays the functions  $SI(y,t)$  evaluated in 1995 and 2005 (prob\_healthy\_1995 and prob\_healthy\_2005). In addition, the figure includes the probabilities of surviving to age  $y$  in the absence of HIV/AIDS (prob\_survival\_1995). The two  $SI(y,t)$  curves trace the probabilities that adult children born  $y$  years before the target year (values of  $y$  in the x-axis: 10, ..., 55) are alive and healthy (uninfected) in years 1995 and 2005; the third set of plotted values represent the probabilities that the adult children will be alive in the absence of HIV/AIDS. Note that because all values of  $SI(y,t)$  are associated with **real cohorts of adult children**, they need not be monotonically decreasing. In fact, they should not be since they must reflect, on the one hand, the combined effects of mortality and of HIV incidence on the other. For example, in 2005 slightly more than 40 percent of the adult children aged 20 are expected to survive with no HIV infection, whereas about 50 percent of those aged 50 will do so. This is because the burden of HIV weighs more heavily among the younger cohorts than among the older ones. While in the absence of HIV/AIDS an elderly parent could expect that almost 80 percent of her adult children born 30 years before would have survived to target year 2005, only 48 percent will survive healthy, not infected, as a result of the epidemic. This is a formidable load of illness that could potentially translate into reduced transfers of assets and income to the elderly, additional labor, added burden to grandparents in the form of care for adult children and grand children and, finally, reorganization of residential arrangements.

Figure 3b shows another face of the impact of the epidemic: the cumulated incidence of HIV by cohort of children (cumulated\_hiv\_1995 and cumulated\_hiv\_2005) and the cumulated survival among adult children who experience HIV (survival\_hiv\_1995 and survival\_hiv\_2005). The **difference between these two sets of curves for each target year** is a measure of the bereavement load on the elderly, the cumulated mortality among their adult children due to HIV/AIDS. Note that, as it should be given the youth of the epidemic, the bereavement load is trivial in 1995 (the lowest pairs of curves) but it grows to be as large as 0.20 among those in cohorts aged 20-35 in 2005: this implies that the probability of an adult child dying of HIV/AIDS before attaining ages 20-35 in 2005 is of the order of 0.20 or, equivalently, that a fifth of all daughters belonging to these cohorts will experience mortality due to HIV/AIDS.

Figures 4a and 4b are analogous to Figures 3a and 3b but correspond to evaluations in 2000 and 2010 respectively. The cumulated impact of the epidemic is quite visible in Figures 4a and 4b: only 35 percent of the cohorts born 20-30 years before 2010 will reach the target year without having been infected by HIV/AIDS. The bereavement load for elderly associated with adult children aged 20-30 in 2010 grows from .20 in 2005 to a staggering .35 in 2010. This means that the probability of an adult child dying of HIV/AIDS before attaining ages 20-35 in 2010 is of the order of .35. More than a third of all daughters belonging to these cohorts will die due to HIV/AIDS.

Table 3 displays summary measures for South Africa nationwide and two provinces with stark contrasts in the HIV/AIDS epidemic, KwaZulu-Natal and Limpopo. These figures contain the probabilities that elderly aged 60, 65 and 70 will have children who are alive and healthy, infected, and dead to HIV/AIDS in selected target years. The numerical evaluation is for the pair of years 1995-2005 and for 2000-2010. The table sections associated with 1995 and 2005 trace

the experience of elderly aged 60, 65, 70 in 1995 over ten years up until 2005. A similar interpretation applies to the sections associated with the years 2000 and 2010.<sup>16</sup>

### **Constraints on the Availability of Healthy Adult Children Imposed by HIV/AIDS**

An outcome computable from the results of the model is the distribution of elderly by number of children alive and healthy, infected with HIV or dead due to AIDS. These estimates are indicators of demographic availability and of the potential burden of disease on the elderly. Figure 5 displays the estimated distribution of children surviving in 1995 in the absence of HIV/AIDS (alive1995) uninfected and alive in 1995 (healthy1995), and uninfected and alive in 2005 and in 2010 (healthy2005 and healthy2010) for elderly people aged between 60 and 70 in the target year. The impact of HIV is remarkable: the distributions of children healthy narrows down considerably and drifts toward much lower means. The fraction of the elderly with no surviving children (in the absence of HIV) is around 10 percent in 1995 but balloons to 18 and 20 percent in 2005 and 2010 respectively.

But the damage caused by the epidemic may be even larger than what these figures suggest. In fact, although the increase in the number of elderly persons with no surviving children is a key determinant of the probability of the elderly living alone (Wolf 1994; Palloni 2000), the fall in the mean number of children healthy may produce effects that are not captured by the results of the model. Indeed, while the model results indicate the magnitude and direction of demographic constraints placed on the elderly, they do not remove the uncertainty associated with endogenous effects whereby demographic constraints may shift and alter residential propensities. Thus, while in some social settings the drop in the number of healthy children may

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<sup>16</sup> Figures on these tables are calculated from the set of values  $SI(y,t)$ ,  $QI(y,t)$ , etc... and the time distribution of children ever born associated with elderly aged 60, 65, and 70 in the target years. The latter correspond to the values

increase the elderly's propensities to live with adult children, because joint residential arrangements are a mechanism to cope with a sick adult child, in other settings it may increase the propensity of grandparents will take in their grandchildren to ease the burden on their sick adult children. A third social reaction could be more perverse as elderly parents shy away from sick adult children to avoid social stigma and preference for coresidence is reduced. If the first mechanism were to prevail, we should expect an increase in the proportion of elderly living with adult unmarried and widowed children. If instead the second mechanism dominates, we should observe an increase in living arrangements involving skipped generation households. The third mechanism, together with the sheer pressure on the availability of surviving children produced by HIV/AIDS mortality, should lead to sharp increases in the proportion of elderly living alone or with their spouse.

Our model results provide only benchmark estimates of the constraints in demographic availability but cannot tell us anything about actual residential arrangements. In order to investigate these we turn now to results from the 1991, 1996 and 1998 data.

## **ANALYSIS OF CENSUS AND SURVEY DATA**

### **Orphanhood and Widowhood**

Before turning to an examination of the observed living arrangements of the elderly from the censuses and survey, we calibrate our ability to detect gross effects of HIV/AIDS from each data source. We do this by examining patterns of orphanhood and widowhood. If the empirical results we obtain are broadly consistent with expected results, we have *prima facie* evidence that the three data sources reflect the impact of HIV/AIDS and that they can be used to inquire about other outcomes, such as living arrangements of the elderly. Inconsistency between expected and

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of  $N(x-y,t-y)$  referred to in the expressions for the main functions.

observed results could mean one of two things: either that the epidemic has not yet gathered enough momentum to produce visible effects or, alternatively, that adjustment mechanisms (widow remarriage and child fostering) obscure what should be otherwise cleanly observed effects. If the first interpretation is correct, we should not expect to find large effects on residential arrangements either. If the second explanation is more appropriate, our ability to observe effects at the level of elderly residential arrangements will be a function of how effective are adaptive mechanisms, such as fosterage and remarriage in the case of orphanhood and widowhood, in offsetting the impact of the epidemic.

To facilitate comparisons with other research (Zaba and Gregson 1998), and because information on parental survival was not collected in the 1991 RSA census, we estimate linear regressions of the logarithm of the proportion orphans on the logarithm of prevalence by provinces from 1996 and 1998 data. The estimated regression coefficients can be interpreted as elasticities or, equivalently, as the proportionate change in orphanhood relative to a proportionate change in HIV prevalence. Each regression is based on nine observations, one for each province.

In each year, we focus on three types of orphanhood (maternal, paternal and double orphans) in the age groups 0-4, which we regress on contemporaneous HIV prevalence.<sup>17</sup> The R-squares fluctuate between 0.10 (for paternal orphans in 1996) and 0.42 (double orphans in 1998). The elasticities for maternal and paternal orphanhood are 0.19 and 0.17 in 1996, and 0.35 and -0.17 in 1998. Estimates of relative changes in maternal orphanhood obtained from data collected elsewhere in Africa suggest elasticities in the range between 0.10 and 0.90 (Zaba and Gregson 1998). Thus, our results fall within an expected, albeit fairly liberal, range. The

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<sup>17</sup> Estimations were carried out alternatively assuming parents identified as missing in a roster were alive and dead. The results presented here correspond to the conservative definition of orphanhood (when information on parent is missing, the parent is considered alive). Estimates corresponding to this more conservative definition are slightly lower in all cases.

estimated coefficient for maternal orphanhood for 1998 is, as expected, higher than for 1996. The elasticities of proportion double orphans in 1996 and 1998 are 0.30 and 0.61 respectively, and the change is in the expected direction. These estimates are 0.10 to 0.40 higher than estimates obtained from macro-simulation models (Palloni and Lee 1992).

Except for parental orphanhood, changes in the estimated coefficients for maternal and dual orphanhood between 1996 and 1998 are in the expected direction, as the epidemic progresses over time. The magnitude of the elasticity coefficients are also within the bounds of empirical or model based estimates. Differential fosterage practices, documented elsewhere in Africa (Ntozi et al. 1999b), may explain lower elasticities for paternal than maternal orphanhood.<sup>18</sup>

Because of the dynamics of HIV transmission and sex differences in the age-specific incidence curve of HIV/AIDS, the progression of the HIV/AIDS epidemic is expected to increase first the proportion of women who are widowed. To assess the responsiveness of widowhood to HIV/AIDS, we estimate the elasticity of widowhood among 15-49 year old women with respect to HIV prevalence between 1991 and 1998. This age constraint is partly practical as the information on marital status in the DHS is only collected for women of reproductive age. But the focus on women in young and mid-adult ages is also consistent with the age selectivity of AIDS mortality among men and the documented decline in mortality at the older ages over the 1990s in South Africa (Dorrington et al. 2001). Elasticities for 1991, 1996

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<sup>18</sup> In general, the level of paternal orphanhood is lower in the DHS than in the census. Also, the reversal of the direction of the relationship between paternal orphanhood and HIV between 1996 and 1998 may have to do with the formulation on the question of who is child of head and parental survival. In the 1996 census, biological children, step children and foster children are recorded as children of head, while in the DHS, the relationship to head is recorded separately for biological and fostered children. To ensure comparability with the census, in the DHS, we merged biological children and fostered children. This implies that we may end up counting more fostered children in the 1998 than in the 1996 census. If, according to the cultural norms observed elsewhere in Africa, foster fathers are reported as birth father, prevalence of paternal orphanhood in the 1998 DHS will be underestimated.

and 1998 are 0.037, 0.038 and 0.11 in 1991, 1996 and 1998 respectively. The change in the elasticities is in the expected direction, but their values are low suggesting a weaker relationship between HIV/AIDS and widowhood than between HIV/AIDS and orphanhood. This may be partly explained by remarriage patterns, but also by the fact that widowhood itself is likely to be of short duration, truncated by the death of the surviving partner, since the infectious status of one of the partners is highly correlated with the infectious status of the other (Palloni and Lee 1992). Paradoxically, the dynamics of HIV transmission censor the widowhood experience of the surviving partner and weaken one effect of the epidemic.<sup>19</sup>

In summary, these aggregate results reveal the signature of HIV/AIDS, though the observed effects of the epidemic may be dampened by socio-cultural responses especially in the case of paternal orphanhood and female widowhood.

### **Patterns of Living Arrangements of the Elderly**

The taxonomy of living arrangements of the elderly adopted here is suggested by the outcomes of the macrosimulation model. We focus on three main residential arrangements of the elderly: (1) Living alone or with spouse; (2) Living with unmarried or widowed adult children with or without grandchildren; and (3) Living with grandchildren but no adult children.

Similar to our previous analysis of orphanhood and widowhood, we explore the relationship between living arrangements of the elderly and HIV prevalence across the nine South African provinces by estimating a linear regression of the logarithm of each type of living

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<sup>19</sup> Levels of widowhood are particularly low in the 1991 and 1996 censuses. The low level in 1996 is especially surprising because the epidemic is more advanced in 1996 than in 1991 and, unlike the 1991 RSA census, the 1996 census enumerated the population of the former TVBCs where widowhood is expected to be higher due to worse socioeconomic conditions compounded by political violence. We strongly doubt the quality of the 1996 census data on marital status. A comparison of the female age-specific widowhood rates estimated from the 1991 census, 1996 census, the 1996, 1997, 1998 October Household Surveys and the 1998 DHS has shown lower widowhood rates in 1996 than in any of the other data sources, leading us to suspect underreporting of widowhood in this data source.

arrangement of the elderly in 1991, 1996 and 1998 on the logarithm of HIV prevalence. For each relationship, the data are shown by means of scatter plots of the values of the nine provinces together with the regression line that best fits the data, the estimated regression equation, and the associated R-square. Besides gauging the responsiveness of each type of living arrangement of the elderly to HIV prevalence, this approach allows identification of patterns across provinces grouped according to their shared level of HIV prevalence and migration characteristics. 1991 provides the baseline observation for the period before the onset of the HIV/AIDS epidemic in South Africa, while the observations for 1996 and 1998 are for a period when the impact of HIV/AIDS should already be felt.

We start by showing the relationship between living alone or with spouse and HIV prevalence in 1991, 1996 and 1998. We omit Western Cape, Northern Cape and Gauteng because they are outliers with very high proportions of the elderly living alone or with spouse, a condition associated with higher levels of economic development in these provinces and the concentration of white South Africans. With the inclusion of these three outliers, the effects of modernization on the elderly living arrangements would have offset those of HIV/AIDS.

Figure 6 shows the results for the remaining six provinces. The association between the proportion of solitary living among the elderly and HIV prevalence is positive and becomes stronger over time. The elasticity values increase from .23 to .67 and .34 as do the R-squares. Although the results for 1991 may be biased upward by the lack of coverage of the TBVCs populations among which prevalence of solitary living may have been lower, this pattern of change is generally consistent with model prediction. The latter half of the 1990s is a period when we expect the proportion of the elderly living alone or with spouse to increase as a result of

harsher constraints in the availability of children or preference for solitary living fed by stigma associated with AIDS.

In Figure 7, the relationship between the proportion of the elderly living with unmarried or widowed adult children and HIV remains weakly positive throughout the period, and there is a lot of variation around the regression lines. Similar to the poor health status of adults infected with HIV which may require assistance provided by their elderly parents in the form of coresidence, several factors may confound the relationship between HIV prevalence and this type of living arrangement. These factors all point in the direction of enduring preferences for extended living arrangements between elderly parents and their adult children over the 1990s, and could be attributed to the South African pension system which provided an incentive for coresidence between adult children and their elderly parents, or documented declines in marriage rates over the 1990s (Hertrich 2002; Hosegood and Preston-Whyte 2002), which reduced the fraction of married children and increase that of the unmarried living with their elderly parents. The effect of HIV on elderly's coresidence with adult children may be better assessed by examining the proportion of the elderly who live with a widowed child and taking advantage of the direct effect of HIV on widowhood.

Figure 8 shows the relationship between living with one widowed daughter age 15-49 and HIV prevalence. Although probable underreporting of widowhood in the 1996 census prevents us from determining the direction of change in this living arrangement between 1991 and 1996, the strength of the relationship in 1998 relative to 1991 is suggestive of a propensity of AIDS widows to join their natal homes upon the loss of their spouse.

The macromodel further predicted an increase in the number of grandparents who live with a young grandchild to relieve the burden off their sick children or to provide care for their

orphaned grandchildren. Figure 9 shows that this may be indeed a mechanism to cope with increasing numbers of sick adult children or fewer surviving children. The association between the proportion of elderly living with at least a grandchild younger than 15 in the absence of adult children becomes stronger over time, as suggested by the significantly higher elasticities in 1996 and 1998 relative to 1991. However, the strengthening of this association still does not confirm that it is indeed HIV/AIDS and not migration creating the conditions for grandparents to take in their grandchildren. KwaZulu Natal, Mpumalanga, Eastern Cape, which experienced the largest surge in HIV prevalence between 1991 and 1998, also experienced an intensification of internal (KwaZulu Natal and Mpumalanga) and interprovincial (KwaZulu Natal and Eastern Cape) migration.

Additional confirmatory evidence for the relationship between the level of HIV and the prevalence of skipped generation households is given in Figure 10 which shows a strong association between HIV and the share of the elderly who live with a double orphan grandchild under 15 in 1996 and 1998.<sup>20</sup> In 1996, a 1 percent increase in HIV prevalence is associated with a 0.37 percent increase in the proportion of elderly living with a double orphan grandchild, while in 1998 it was associated with a 0.51 percent increase in this proportion.

## **CONCLUSIONS**

The descriptive analysis of three consecutive data sets collected before and after the onset of HIV/AIDS in South Africa has revealed evidence which reflects the impact of HIV/AIDS on the living arrangements of the elderly. Our results suggest that the fall in the number of healthy children and the growing loss of children to AIDS experienced by South African elderly over the 1990s may have left the elderly with fewer or no surviving children to live with and may have

increased the propensities of grandparents to take in their grandchildren to ease the burden on their sick adult children or to care for their orphaned grandchildren.

Some of the outcomes we analyzed have changed in the way expected by models of availability, whereas some others have done so in accordance with what one would expect given hypothesized changes in preferences. Where the record is inconclusive, it may be because the epidemic has not worked its way through with sufficient force or, more importantly, because individuals and groups react in ways that conceal the trail left by HIV/AIDS, or because we may be unable to distinguish the effects associated with HIV/AIDS from those triggered by migration or the pension system, two phenomena that may lead to similar adjustments of living arrangements of the elderly as those triggered by HIV/AIDS.

We know, however, that the results from the macro model suggest a grim scenario not too far into the future, certainly before 2005 or 2010. It may be the case that the observed patterns emerging from more recent data, like the 2001 census, will show even greater consistency with model predictions. But this will depend on the quality of data and our ability to identify outcomes that undergo sufficiently large changes not offset by changes in propensities, outcomes that are sensitive to the significant heterogeneity of the epidemic and that are not overwhelmed by the effects of other unrelated phenomena.

Establishing benchmarks using model based approaches as we have done here is useful but insufficient. It is likely that if all the minimal identification conditions are not simultaneously met, one will need longitudinal studies, performed at lower levels of aggregation, and eliciting direct information on residential preferences, changes in availability, and changes in actual living arrangements in subgroups affected and not affected by HIV/AIDS.

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<sup>20</sup> Information on parental survival was not collected in the 1991 RSA census.

## APPENDIX

Assume we focus on an elderly woman aged  $x$  who is alive in the census year 1995. This will be the target or target person and the year the target year. The main objective is to derive expressions for the following probabilities applicable to the female children born alive when the target was aged  $x-y$  at time  $t-y$ : (a) that they have survived healthy (no HIV) to time  $t$ ; (b) that they survived to  $t$  but contracted HIV along the way, (c) that they have died due to HIV and other causes. In all these cases the index variable  $y$  varies from a minimum equal to  $x-50$  to a maximum equal to  $x-15$  thus bounding the childbearing period of the target between 15 and 50. We assume that none of the targets contracted HIV prior to age  $x$  or that the fraction that does is trivial and can be dismissed. Because we use a minimum value for  $x$  of 60, this assumption is sensible but not entirely accurate since some of these women could have been infected in the ten years prior to 1995. However, since the HIV incidence rates between ages 49 (attained the year the epidemic started) and 60 (attained in the middle of the census year 1996) are very low, the assumption is bound to be quite close to real experience. Furthermore, to the extent the assumption departs from reality, our calculations will be in error only if the childbearing patterns to which targets not affected by HIV and the mortality and HIV incidence pattern of her children are different from those that apply to targets who were infected prior to the target year.

To derive expressions for these probabilities we rely on the following simplified scheme: let  $\mu(z, t^*)$  be the force of mortality (in the absence of HIV) at age  $z$  at year  $t^*$ , the year when the target's child attained aged  $z$ ,  $\lambda(z, t^*)$  the rate of HIV infection at age  $z$  and time  $t^*$ ,  $\delta$  (d) the joint rate of incubation and mortality due to HIV/AIDS and other causes for individuals who have been infected  $d$  years. The set of all target's children born  $x-y$  years ago who could be aged  $y$  at time  $t$  is the union of several disjoint subsets: one containing individuals who will not reach

age  $y$  due to mortality in the absence of HIV,  $Q(y,t)$ ; another containing those who will attain age  $y$  at time  $t$  but are infected with HIV,  $QI(y,t)$ ; a third subset containing those who will die of HIV/AIDS related causes,  $QID(y,t)$  and, finally, a subset including those who will attain age  $y$  at time  $t$  as healthy individuals (not infected),  $SI(y,t)$ . The expression for the corresponding probabilities, referred to a target aged  $x$  at time  $t$ , are as follows:

$$SI(y,t) = \int_{\{0,y\}} \exp(-\int_{\{0,v\}} (\mu(v,t-v) + 4\lambda(v,t-v))dv)$$

$$QI(y,t) = \int_{\{0,y\}} [\exp(-\int_{\{0,w\}} (\mu(v,t-v) + 4\lambda(v,t-v))dv)] * 4\lambda(w) * \exp(-\int_{\{w,y\}} (\mu(z-w) dz) dw$$

$$Q(y,t) = \int_{\{0,y\}} [\exp(-\int_{\{0,w\}} (\mu(v,t-v) + 4\lambda(v,t-v))dv)] * \mu(w) dw$$

$$QID(y,t) = 1 - [SI(y,t) + QI(y,t) + QID(y,t)].$$

If one knows the time distribution of children ever born reflected in the age pattern of fertility to which women aged  $x$  at time  $t$  were exposed during childbearing,  $N(x-y, t-y)$ , we can calculate the weighted probabilities of having a child in any of the four statuses defined above. This is achieved by multiplying  $N(x-y, t-y)$ , for every permissible value of  $y$ , by each of the quantities defined above. These weighted values represent the **average probabilities for an elderly aged  $x$  at time  $t$** . In particular,  $N(x-y, t-y) * QI(y,t)$  is the average fraction of all children born to the target elderly woman who is infected with HIV at age  $y$  at time  $t$ ;  $SI(y,t) N(x-y, t-y)$  is the average probability of having a child aged  $y$  at time  $t$  who is healthy; and, finally  $N(x-y, t-y) * (QID(y,t) + Q(y,t))$  is the average probability of having lost a child to either HIV/AIDS or to mortality due to other causes.

In addition to the assumptions discussed above, we rely on the simplification that one can follow the progression of mortality of infected individuals combining the force of mortality due

to other causes, the incubation function, and AIDS-related mortality in a single synthetic super-function,  $\int_0^t \lambda(d) dd$ , which depends only on duration since infection and not at all on age.

### Estimation of the Model

Estimation of the model depends of five pieces of information. The first and most important are the yearly HIV incidence rates from the onset of the epidemic until time  $t$ . The second is the incubation function which determines the waiting time in the infected state. The third is mortality of healthy individuals, of HIV+, and of those with full-blown AIDS. The fourth is the time distribution of children ever born or, equivalently, the fertility function approximating the childbearing experience of the target population. Finally, we also need the distribution of targets by number of children ever born,  $w(j)$ . Below we briefly define the nature of these inputs.

#### Estimation of HIV incidence

A most difficult task is to derive estimates of HIV incidence for 1985-2010. We proceed in several stages. In a first stage we obtain a time series of prevalence estimates for each province during the period 1990-1999. These estimates were obtained from calculations made at Statistics South Africa using the estimates prepared by UNAIDS and independent estimates obtained from surveillance sites and gathered at the US Bureau of the Census. The second stage consisted in fitting a curve to the estimated cumulative prevalence,  $Pre(t)$ . We used a gamma function of the form

$$Pre(t) = \frac{k}{\Gamma(\alpha)} \int_0^t \exp(-\delta d) [d(t)]^{\alpha-1} dd(t),$$

where  $d(t)$  is the duration of the epidemic at year  $t$  and  $k$ ,  $\alpha$ , and  $\beta$  are parameters to be estimated.<sup>21</sup> Our motivation in using this expression is more a matter of adherence to convention than of logical reasoning or empirical judgment: there is no compelling reason to prefer this function over a virtually infinite set of equally plausible ones. Indeed, the utilization of this function leads to serious problems that require ad-hoc solutions. The most important of these problems is that, by its own nature, the associated incidence curve—the density function associated with the cumulative Gamma function—will tend to peak very early and taper off and drift to zero very rapidly. For starters, this type of behavior is inconsistent with the possibility that HIV/AIDS becomes endemic with a constant level of incidence, a possibility that is not only mathematically feasible but quite likely as well. Second, systematic comparisons with a number of simulated results using one detailed macro model of HIV/AIDS (Palloni, 1991) demonstrate that almost always, and regardless of the nature of input parameters, the Gamma incidence drops too fast and does not reflect at all the post-peak course of the epidemic.

To resolve this problem other researchers have adopted arbitrary solutions. They cannot be otherwise since there are no observations beyond the peak of the epidemic. Estimates exceeding that point are anybody's guess. In this paper we adopt a rather *sui generis* solution: we take a large set of simulated results using parameters that are deemed to represent well the situation in South Africa (regarding mortality, fertility, number of partners per person per year, etc...) and then recover the estimated incidence curves after the onset of the simulated epidemics. We then fit a Gamma function to the simulated cumulated prevalence, derive estimates of incidence and calculate the difference between the simulated and estimated incidence rates. These differences are tantamount to 'Gamma-adjustment factors'. To modify the post-peak estimated incidence for South Africa we search for the set of Gamma parameters

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<sup>21</sup> In all cases it was assumed that  $t(d)$  was equal to  $t-1985$ , that is that the epidemic started in earnest in 1985.

estimated in the simulated model that most closely resemble those observed in South Africa and adopt the corresponding ‘Gamma-adjustment factors’. We do not adjust the pre-peak estimates, which are heavily determined by the observed prevalence, but only the post-peak course of incidence rates. In all provinces and in South Africa nationwide, the adjustments apply to years after 2003, not before. In this sense, the uncertainty surrounding the estimates of HIV incidence is larger in the post 2003 period than before, when the estimates are at least more closely anchored to the trajectory of observed prevalence.<sup>22</sup>

### **Estimation of the Incubation Function**

We assumed that  $(d)$  follows a Weibull hazard function with parameters  $\alpha = 0.08$  and  $\beta = 3.2$  dictating a survival distribution with a median survival time of approximately 10 years. Given the fact that mortality among HIV infected individuals in Africa is likely to be much higher than normal even in the absence of full-blown AIDS, this assumption does not seem unrealistic. It is also consistent with reports suggesting that the median survival time of HIV infected individuals in Africa is on the order of 7.5 years (Boerma et al. 1998)

### **Estimation of Healthy Mortality Levels**

We use mortality levels estimated for South Africa by the United Nations for the period 1950-2000 and then the projected life expectancies through the year 2010 corresponding to the UN medium projections. For years before 1950 we estimate life expectancy linearly extrapolating backwards from 1960. In all case we use the North female pattern of mortality from the Coale-Demeny model life tables.

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<sup>22</sup> This is a generous statement, for the ‘observed’ prevalence is not so: it is estimated via procedures that are not always reproducible and rest on observed prevalence in small and selected samples of pregnant women.

### **Estimation of Fertility and of $N$ and $w$**

Estimates of  $N$  were obtained from the age pattern of fertility implicit in Coale-Demeny stable models. We made no extra efforts to approximate closely a fertility scheduled for South Africa since what mattered in our calculation was the experience of women who are now 60 and above, that is, the childbearing experience pertaining to years as late as 1980 and as early as 1945. The fertility pattern between 1945 and 1975 at least is a matter of guess work, and instead of deriving original estimates we chose to adhere to accepted age patterns that have been widely applied.

Estimates of  $w(j)$  were obtained directly from the observed distribution of children ever born to mothers aged 50 at the time of the 1996 census. Although this distribution may differ from the one that applies to mothers aged 60 and above in 1995, it is unlikely that the difference will be major since substantial fertility changes are unlikely to have been experienced by women younger than 30 or 35 in 1996.

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**Table 1. Percentage Distribution of Population by Province and Selected Characteristics, Republic of South Africa 1991, and South Africa 1996 and 1998**

	Western Cape	Eastern Cape	Northern Cape	Free State	Kwazulu Natal	Northwest Pr	Gauteng	Mpumalanga	Limpopo	Total
<b>Census 1991 (a)</b>	WC	EC	NC	FS	KZN	NW	GAU	MPU	L M	Total
African	16.6	57.3	29.8	83.3	81.7	75.5	61.7	88.3	96.4	70.2
Coloured	58.0	22.2	52.7	2.7	1.3	3.0	4.2	0.6	0.2	10.6
Asian	1.0	0.9	0.3	0.0	9.6	0.7	2.2	0.4	0.1	3.2
White	24.4	19.6	17.2	14.0	7.4	20.9	31.9	10.8	3.3	16.1
Urban	54.3	53.5	52.2	51.4	49.6	50.8	55.5	48.9	46.6	51.4
Non-urban	45.7	46.5	47.8	48.6	50.4	49.2	44.5	51.1	53.4	48.6
Sex ratio	1.01	0.97	1.02	1.12	0.90	1.21	1.18	1.02	0.85	0.95
N	271,654	113,463	57,214	142,398	499,295	88,582	331,959	187,819	313,555	2,005,939
% of total pop	13.5	5.7	2.9	7.1	24.9	4.4	16.5	9.4	15.6	100.0
<b>HIV%</b>	<b>0.1</b>	<b>0.6</b>	<b>0.1</b>	<b>1.5</b>	<b>2.2</b>	<b>1.5</b>	<b>1.1</b>	<b>1.2</b>	<b>0.5</b>	<b>1</b>
(a) Excludes TBVCs										
<b>Census 1996</b>	WC	EC	NC	FS	KZN	NW	GAU	MPU	L M	Total
African	21.1	86.4	33.1	84.5	81.8	91.2	70.2	89.4	96.6	76.7
Coloured	54.2	7.5	52.1	3.0	1.4	1.4	3.7	0.7	0.2	8.9
Asian	1.0	0.3	0.3	0.1	9.4	0.3	2.2	0.5	0.1	2.6
White	20.7	5.3	13.2	12.0	6.6	6.6	23.1	8.8	2.5	10.9
Unspecified	3.1	0.6	1.3	0.4	0.8	0.5	0.8	0.5	0.7	0.9
Urban	88.9	36.6	70.1	68.6	43.1	34.8	97.0	38.9	10.9	53.7
Non-urban	11.1	63.4	29.9	31.4	56.9	65.2	3.0	61.1	89.1	46.3
Sex ratio	0.96	0.86	0.96	0.97	0.89	0.97	1.04	0.91	0.84	0.92
N	361,735	563,816	70,974	240,179	735,832	304,384	660,722	246,319	437,240	3,621,201
% total pop	9.8	15.5	2.1	6.5	20.7	8.3	18.1	6.9	12.1	100.0
P.c. income 1996 (b) (in Rands)	17,880	5,479	13,398	10,628	8,070	7,944	25,281	12,921	3,159	11,421
<b>HIV%</b>	<b>3.1</b>	<b>8.1</b>	<b>6.5</b>	<b>17.5</b>	<b>19.2</b>	<b>13.8</b>	<b>15.5</b>	<b>15.8</b>	<b>8.0</b>	<b>14.2</b>
(b) Source: Statistics South Africa (1998a).										
<b>DHS 1998 ©</b>	WC	EC	NC	FS	KZN	NW	GAU	MPU	L M	Total
Urban	89.0	36.3	71.9	69.3	44.5	37.3	97.0	38.8	12.2	54.7
Non-urban	11.0	63.7	28.1	30.7	55.5	62.7	3.0	61.2	87.8	45.4
Sex ratio	0.97	0.86	0.90	0.90	0.86	0.91	0.89	0.88	0.81	0.88
N	3,992	14,409	4,261	3,925	7,919	4,259	4,450	4,661	5,018	52,894
% total pop	7.5	15.7	2.1	6.3	21.2	8.1	18.5	6.7	11.7	100.0
P. c. income 2000 (d) (in Rands)	20,777	7,792	12,481	12,334	10,592	9,693	25,988	11,088	6,021	13,502
<b>HIV %</b>	<b>7.2</b>	<b>15.9</b>	<b>9.9</b>	<b>22.8</b>	<b>32.5</b>	<b>21.3</b>	<b>22.5</b>	<b>30</b>	<b>11.5</b>	<b>22.8</b>

© The DHS only reports population group for the sample of women age 15-49, not for the sample of all household members.

(d) Source: University of South Africa, Bureau for Market Research (2000).

All percentages for 1996 and 1998 reflect sampling weights, but the N rows report the unweighted denominators.

Source: Authors' calculations from 1991 census, 1996 census and 1998 DHS data files.

**Table 2. Estimates of the proportion of households containing at least one elderly with at least one HIV infected adult member (by province, 1996)**

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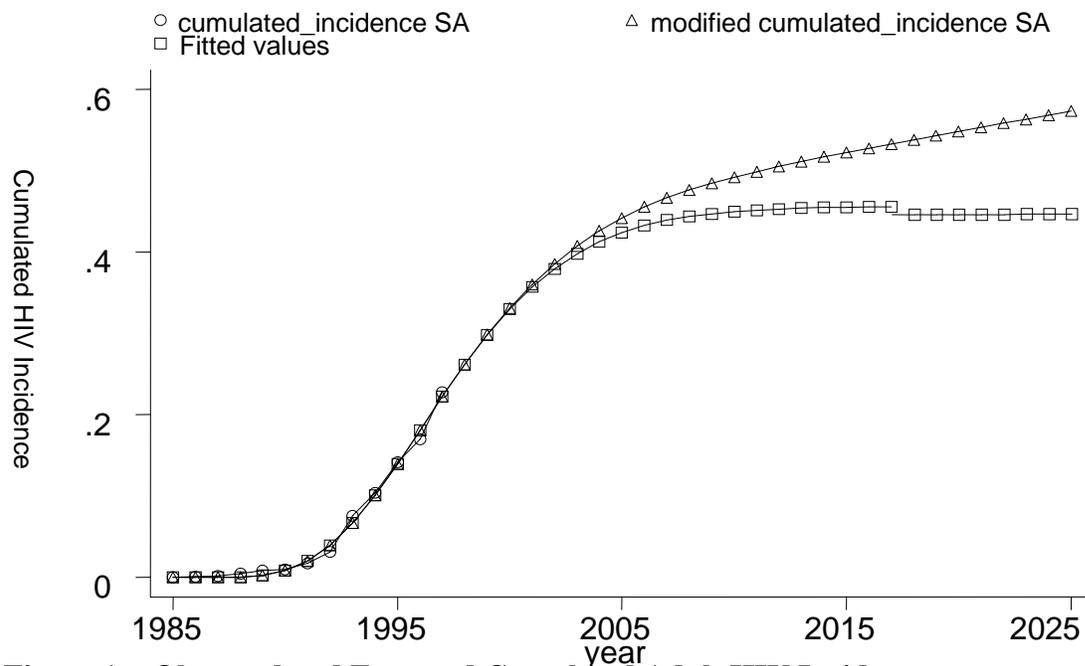
Province	HIV prevalence (1996)	Proportion
W. Cape	.031	.05
E. Cape	.081	.15
N.Cape	.065	.13
Free State	.180	.28
KwaZulu-Natal	.199	.37
NorthWest Pr.	.138	.27
Gauteng	.155	.24
Mpumalanga	.158	.29
Limpopo	.080	.14
Total	.142	.26

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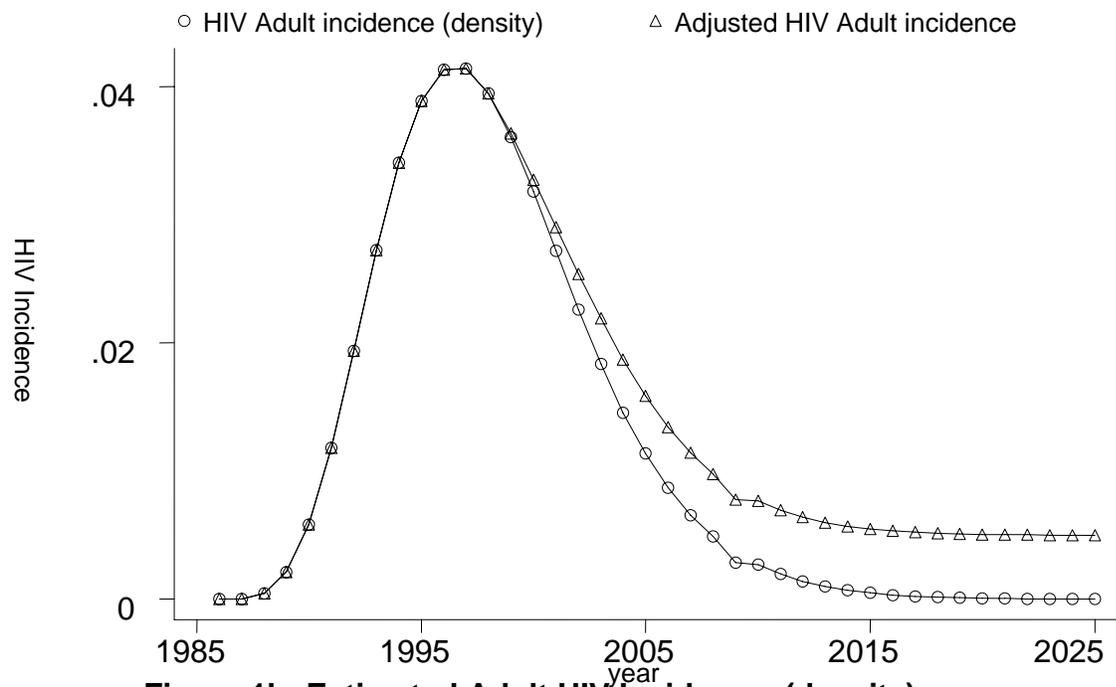
**Table 3. Proportion of elderly age 60, 65 and 70 who will have an adult child infected with HIV, or dead due to AIDS, 1995-2010**

<b>South Africa</b>				
<b>Age Elderly</b>	<b>Infected</b>	<b>Died</b>	<b>Infected</b>	<b>Died</b>
	<b>1995</b>		<b>2005</b>	
	60	.032	.01	.15
65	.026	.002	.12	.013
70	.019	.001	.08	.01
	<b>2000</b>		<b>2010</b>	
60	.14	.02	.28	.20
65	.11	.01	.20	.15
70	.08	.01	.13	.10
<b>KwaZulu-Natal</b>				
	<b>1995</b>		<b>2005</b>	
60	.040	.01	.28	.13
65	.032	.002	.19	.09
70	.022	.001	.12	.06
	<b>2000</b>		<b>2010</b>	
60	.19	.022	.34	.25
65	.14	.016	.25	.19
70	.10	.01	.16	.011
<b>Limpopo</b>				
	<b>1995</b>		<b>2005</b>	
60	.022	.002	.14	.02
65	.019	.001	.10	.01
70	.019	.001	.08	.01
	<b>2000</b>		<b>2010</b>	
60	.089	.012	.19	.13
65	.07	.01	.13	.10
70	.05	.01	.09	.06

*Note:* All calculations represent weighted averages of the functions  $QI(y,t)$  and  $QID(y,t)$  for elderly of the specified age. The weights are the time distribution of children ever born for elderly of the specified age.



**Figure 1a: Observed and Expected Cumulated Adult HIV Incidence**



**Figure 1b: Estimated Adult HIV Incidence (density)**

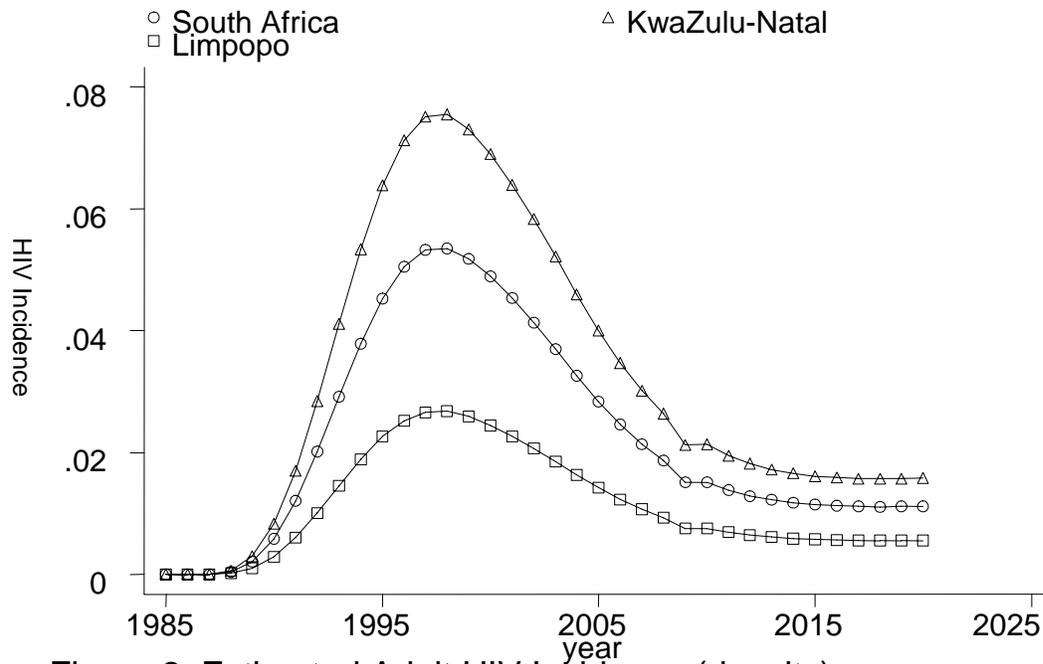


Figure 2. Estimated Adult HIV Incidence (density)

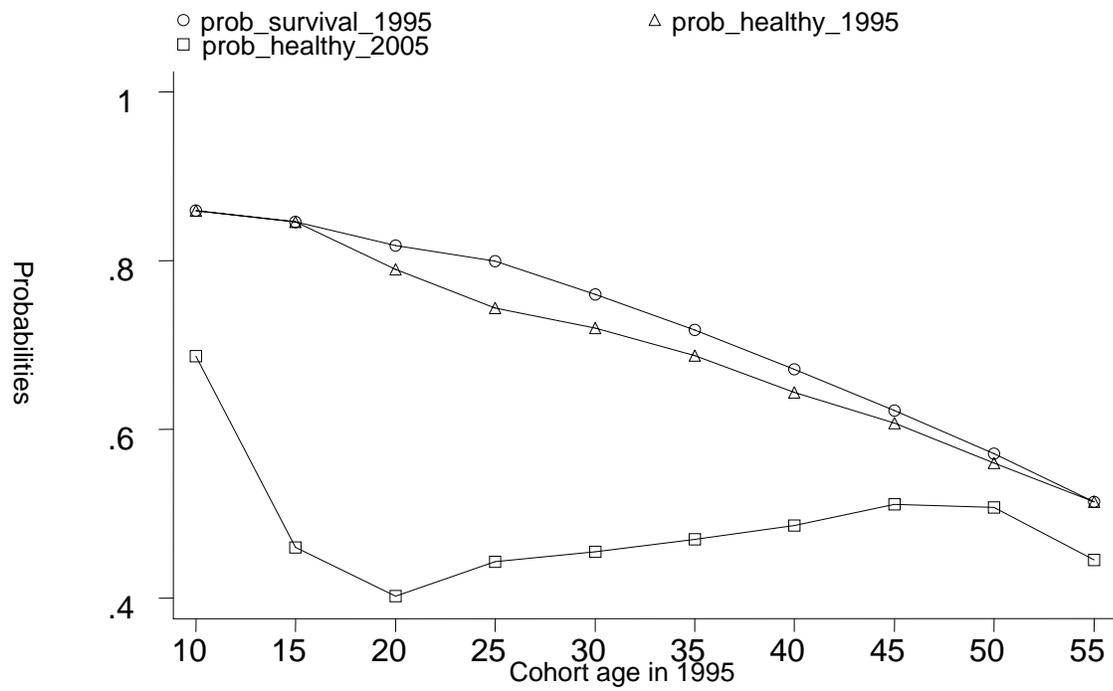
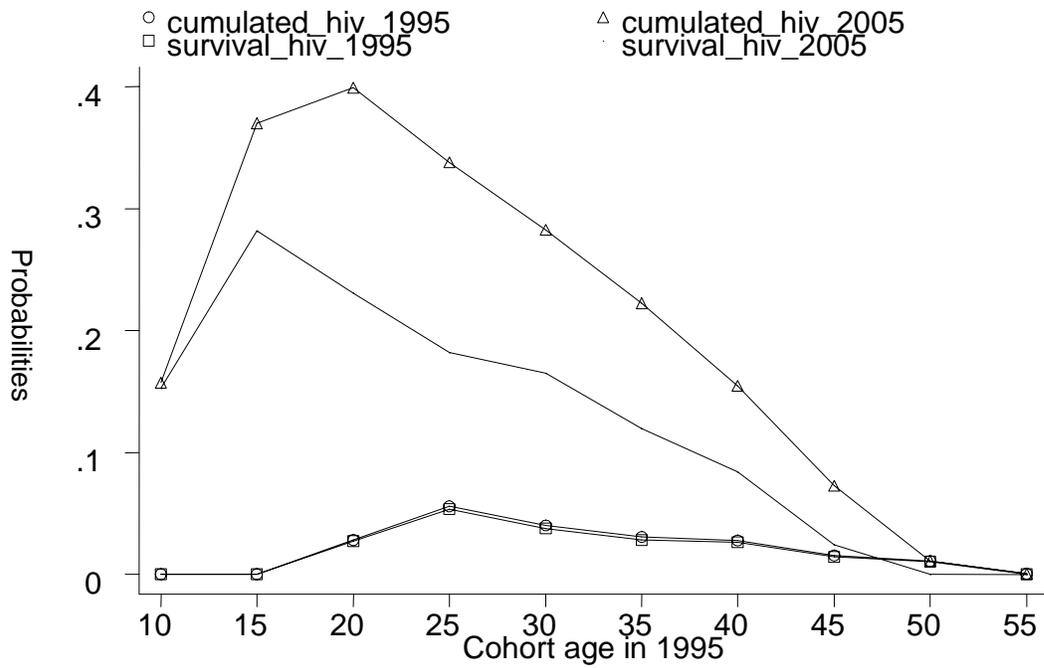


Figure 3a: Survival and Healthy Survival, South Africa 1995-2005



**Figure 3b: Cumulated HIV and HIV Survival, South Africa 1995-2005**

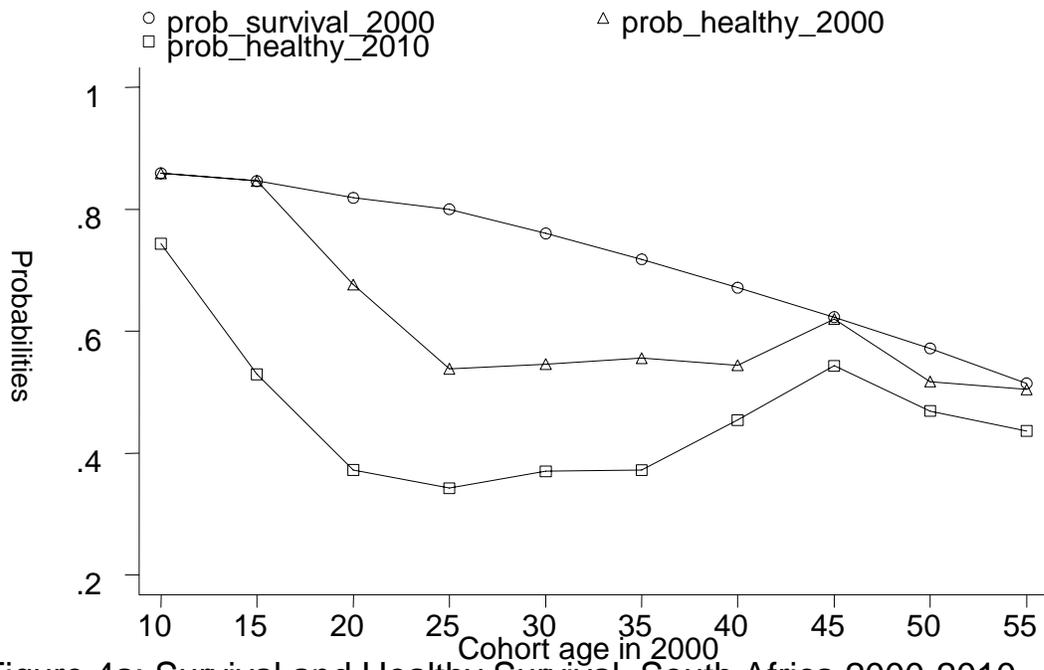
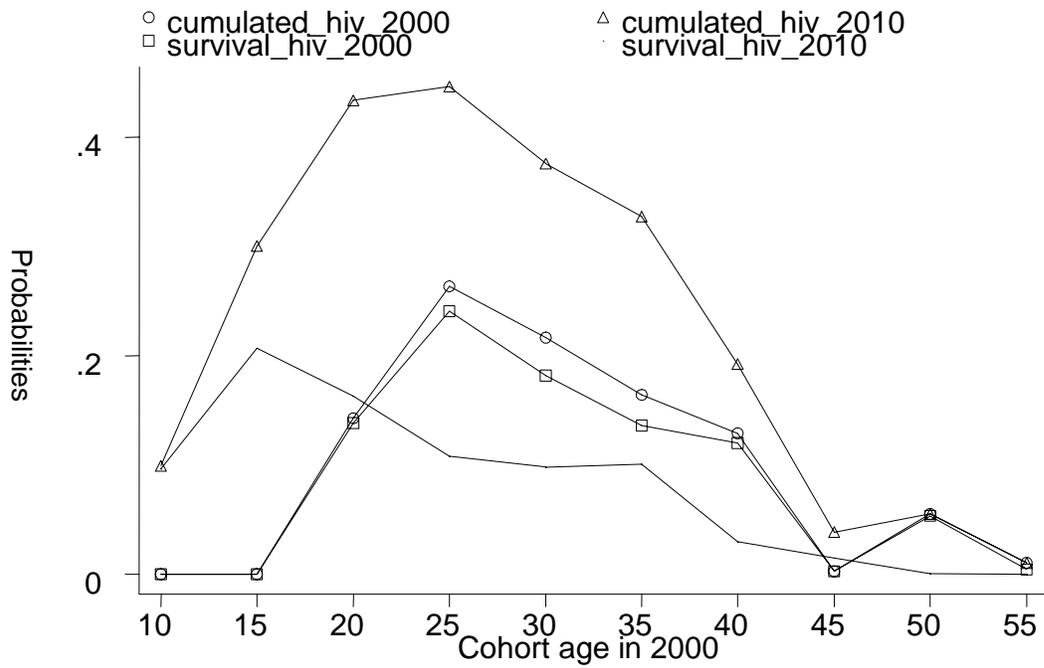
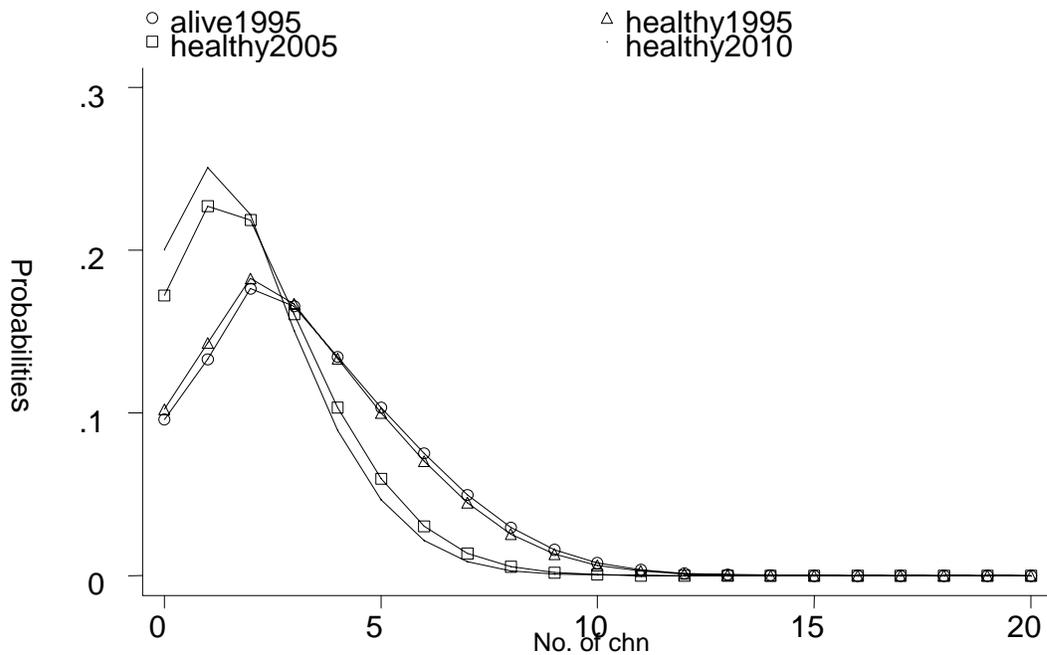


Figure 4a: Survival and Healthy Survival, South Africa 2000-2010

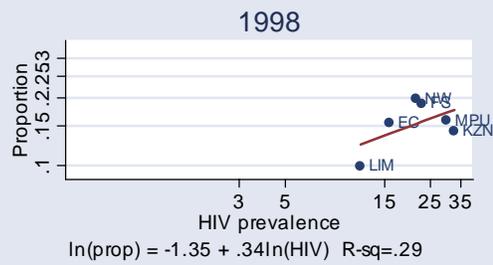
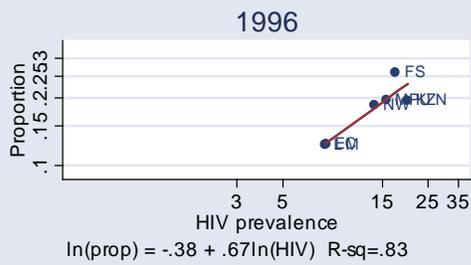
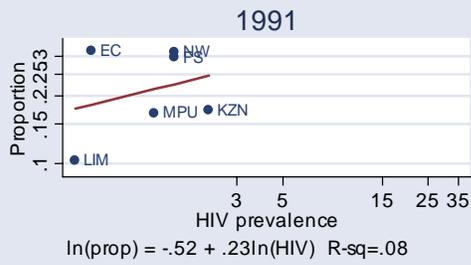


**Figure 4b: Cumulated HIV and HIV Survival, South Africa 2000-2010**



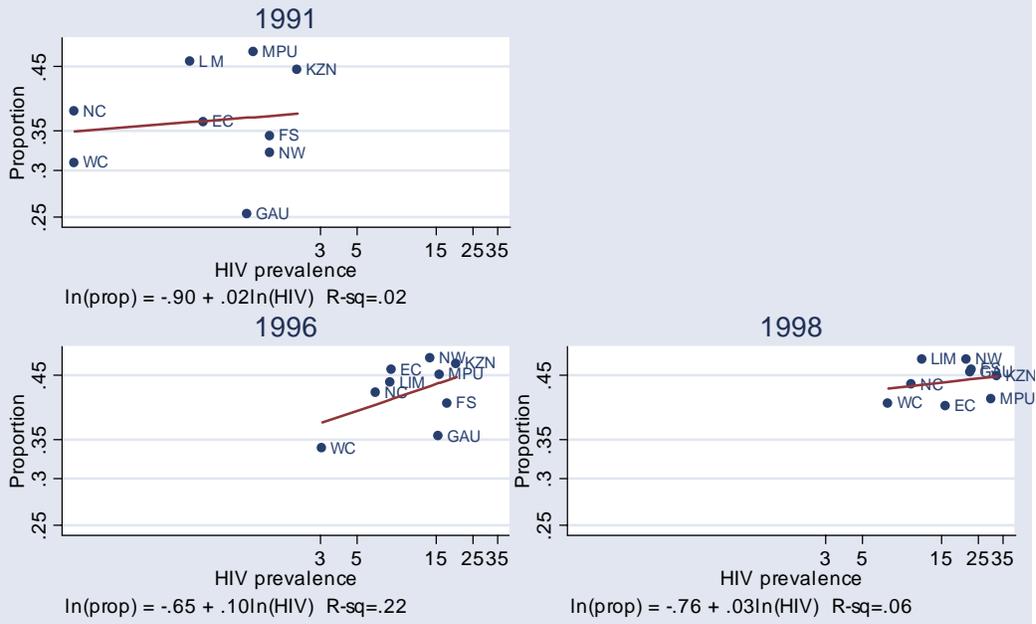
**Figure 5: Probs of adult children alive-healthy, South Africa 1995-2010**

Figure 6. Proportion elderly living alone or with spouse, and HIV prevalence



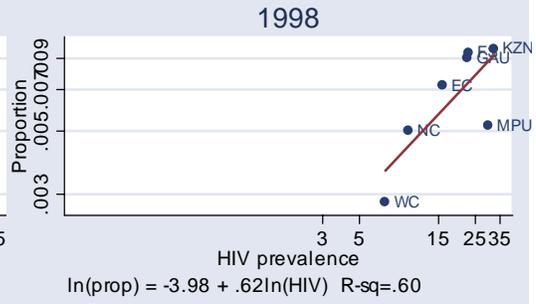
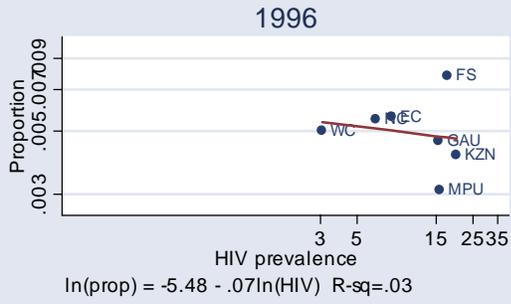
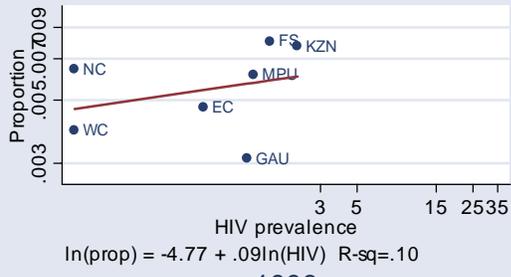
Sources: RSA 1991 census; SA 1996 census 10% sample; SA 1998 DHS  
 WC, EC, GAU dropped because outliers

Figure 7. Proportion elderly living with unmarried/widowed child 15 or older, and HIV prevalence



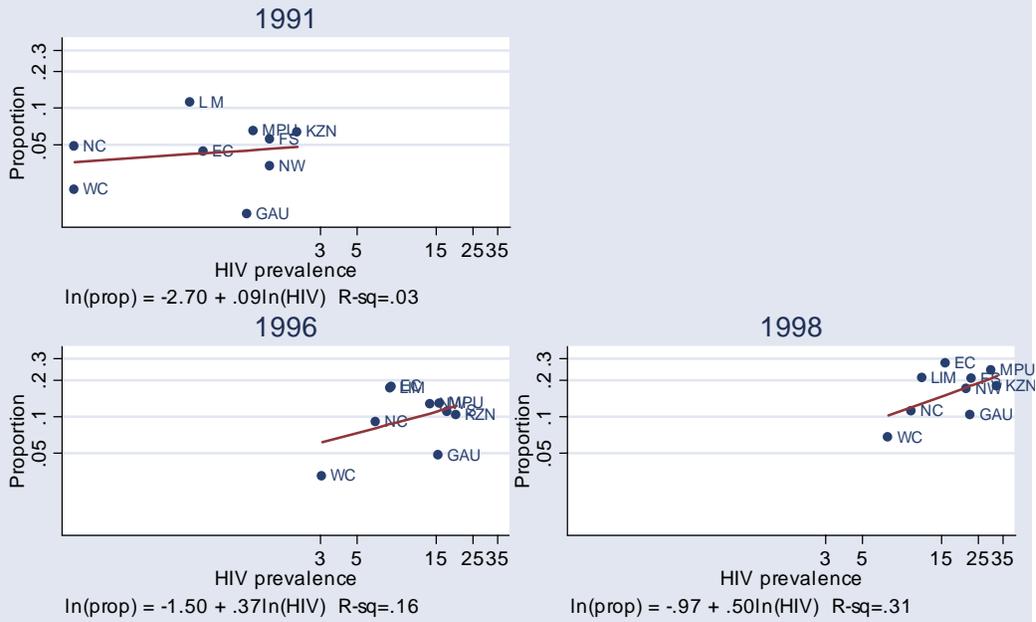
Sources: RSA 1991 census; SA 1996 census 10% sample; SA 1998 DHS

Figure 8. Proportion elderly living with widowed daughter age 15-49, and HIV prevalence 1991



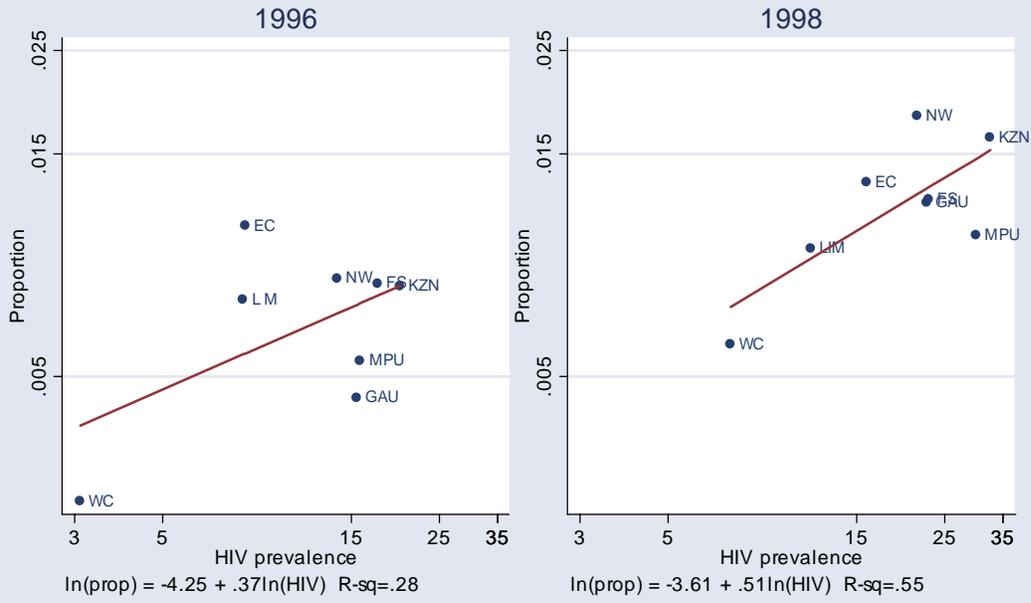
Sources: RSA 1991 census, SA 1996 census 10% sample; SA 1998 DHS  
Limpopo and North west Province were dropped due to no obs in 1998

Figure 9. Proportion elderly living with grandchild under 15, no adult children, and HIV prevalence



Sources: RSA 1991 census, SA 1996 census 10% sample; SA 1998 DHS

Figure 10. Proportion elderly living with double orphan grandchild under 15, and HIV prevalence



Sources: SA 1996 census 10% sample; SA 1998 DHS  
 Northern Cape dropped because no observations in 1998

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