Reproductive Health and Behavior, HIV/AIDS, and Poverty in Africa

Prepared for the African Economic Research Consortium

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Abstract

This paper examines the complex linkages of poverty, reproductive/sexual health and behavior, and HIV/AIDS in Africa. It addresses the following questions: (1) what have we learned to date about these links and what are the gaps in knowledge to be addressed by further research; (2) what is known about the effectiveness for HIV prevention of reproductive health and HIV/AIDS interventions and policies in Africa; and (3) what are the appropriate methodological approaches to research on these questions. With regard to what has been learned so far, the paper pays considerable attention in particular to the evidence regarding the impacts of a range of HIV interventions on risk behaviors and HIV incidence. Other sections review the extensive microeconomic literature on the impacts of AIDS on households and children in Africa and the effects of the epidemic on sexual risk behavior and fertility decisions. With regard to methodology, the paper assesses the approaches used in the literature to deal with, among other things, the problem of self-selection and non-randomness in the placement of HIV and reproductive health programs. Data requirements for different research questions are discussed, and an effort is made to assess what researchers can learn from existing sources such as Demographic and Health Surveys.

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I. INTRODUCTION

Of all the issues touching on economics and demography in Africa, the AIDS epidemic is arguably the most pressing for research and policy. Sub-Saharan Africa is by far the region worst affected by the epidemic. An estimated 24.7 million adults in Africa[1] are infected with the human immunodeficiency virus (HIV), the virus that causes AIDS – accounting for almost two thirds of all adults with HIV globally (UNAIDS, 2006). Some 2.8 million adults and children in Africa became infected in 2006. Prevalence among adults – the share of the adult population estimated to be HIV positive – averages about 6% across the region but there is wide variation both in prevalence levels and in trends. Prevalence is generally stable and relatively low (under 5%) in West Africa and stable or declining in much of East Africa, but at higher rates (over 6% in Uganda, Kenya and Tanzania). In most countries of southern Africa, prevalence is increasing and extremely high – over 20% in Botswana, Lesotho, Swaziland, and Zimbabwe and close to that figure in South Africa.

This paper considers the complex linkages of poverty, reproductive health and behaviors, and HIV/AIDS in Africa. It addresses the following questions: (1) what have we learned to date about these links and what are the gaps in knowledge to be addressed by further research; (2) what is known about the effectiveness for HIV prevention of reproductive health and HIV/AIDS interventions and policies in Africa; and (3) what are the appropriate methodological approaches to research on these questions? With regard to the last question, an effort is made to assess what can be learned both through new data collection and from existing sources such as Demographic and Health Surveys, which have been carried out in many African countries.

First, a few definitions are in order. The WHO definition of reproductive health is "a state of physical, mental, and social well-being in all matters relating to the reproductive system at all stages of life" (WHO 2004). Corresponding to this broad definition of reproductive health, which was explicitly intended to incorporate sexual health, in this paper I will take a broad view as to what constitutes reproductive health *services* (RHS). This will obviously include traditional family planning and maternal and antenatal care. But it also will include programs and services such as control of non-HIV sexually transmitted infections (STIs); HIV prevention, testing, and treatment; condom distribution and promotion; and efforts to promote and provide circumcision to men. For the purposes of this paper we would hardly want to ignore these latter programs, which are all related in varying degrees to HIV prevention. Further, there is an ongoing debate over the advisability in the African context of integrating STI/HIV prevention and care into existing reproductive health services; for this reason, too, it is pertinent to consider the full range of programs related to reproductive and sexual health. In a similar vein, the relevant *behaviors* for this discussion must include not just behavior explicitly related to demographic decisions (fertility and contraception, age at marriage), but also, clearly, sexual risk behaviors.

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[1] Throughout this paper "Africa" is used synonymously with "sub-Saharan Africa".

With these basics out of the way, we turn to Figure 1, which provides an overview of the interactions among poverty, reproductive health (and reproductive health services and related behaviors and knowledge), and HIV/AIDS. The links are many and complex, with numerous possible feedback effects. To take one important example of the latter, patterns of sexual behavior such as unprotected sex with casual partners obviously affect HIV incidence and prevalence[2], but these behaviors may also change in response to recognition of HIV and the risks associated with it. Note as well that most of these processes and outcomes have both micro (individual) level and macro (population) level dimensions: individual HIV status and HIV prevalence rates, individual incomes or poverty and GDP growth or poverty rates, etc.

The rest of the paper is taken up with consideration of the key linkages in the figure: what we know about them, what we need to learn, and what is required for this learning to take place. I begin in Section II with the right hand side of Figure 1: the connections of reproductive health, reproductive and sexual health services (including HIV interventions), and behaviors, both to each other and to HIV/AIDS. A good deal of this section will consider the evidence regarding the impacts of HIV interventions on behaviors and HIV incidence. This in turn gives rise to a review of evaluation methodologies used in the literature and related data issues. The section also considers evidence for reverse linkages: the impacts of the epidemic on sexual as well as reproductive behaviors.

Section III considers linkages with poverty, that is, the relations connecting to the left hand side of Figure 1. First I discuss evidence from Africa on the pathways from poverty to HIV/AIDS, operating via reproductive health, reproductive/sexual behaviors and knowledge, and the use of services, as well as other through other possible routes. I follow this with a discussion of the reverse pathway, that is, the effects of HIV/AIDS on poverty. Two distinct literatures are considered: that concerned with micro (individual or household level) poverty impacts, and that concerned with macroeconomic or growth impacts. Econometric studies of micro level impacts are now quite numerous and have examined impacts on a wide range of outcomes, including household income and consumption, demographic structure, and children's health and schooling.

The emphasis throughout this paper is, in one way or another, on reproductive and sexual *behaviors* as well as knowledge: how they mediate the relationship between poverty and HIV/AIDS, what is known about how behavior responds to the epidemic or to interventions design to affect HIV risk or fertility, etc. These behaviors, of course, are what economists and demographers analyze. Consequently there is not a lot said here about medical or clinical research on HIV and fertility. Still, it will frequently be necessary to touch on these issues. One reason for this is that even 'purely' medical interventions may lead to changes in behavior that either enhance or compromise intended HIV prevention effects. It should also be noted that the research reviewed here as well as the discussion of research methodologies has a largely quantitative focus. This is hardly meant to imply that the techniques and findings of qualitative analyses by, e.g., anthropologists or social psychologists, are not important in the

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[2] HIV incidence refers to rate at which new infections occur and is defined as the share of initially uninfected people who become infected in a year.

study of poverty, reproductive health, and HIV/AIDS. Instead it reflects, again, the research agendas and approaches of economists and demographers.

II. LINKAGES OF REPRODUCTIVE/SEXUAL HEALTH, BEHAVIORS AND POLICIES TO HIV/AIDS

I begin with the right hand side of Figure 1—the connections of reproductive and sexual health, services (including HIV interventions), and behaviors to each other and to HIV/AIDS. Reflecting the importance of identifying effective prevention strategies, much of the discussion to follow will focus on what is known and not known about the effects of different HIV interventions, or more broadly 'reproductive health services', on risk behaviors and HIV incidence.

II.1 Links from reproductive and sexual health to HIV/AIDS

That is not to say that direct links from reproductive health *per se* (represented by the topmost circle in Figure 1) to HIV/AIDS are not important. They are: at least two such links have probably played a major role in the spread of the disease in Africa. They help to explain why so much of the continent has experienced generalized epidemics, with infections occurring in the overall adult population rather than concentrated within specific high risk groups such as sex workers and intravenous drug users as is the case elsewhere around the world.[3]

First, Africa has high rates of untreated non-HIV sexually transmitted infections (STIs) that are cofactors for HIV infection. STIs such as syphilis and herpes increase susceptibility to HIV via genital ulceration, which increases the likelihood of blood transmission during intercourse (Kapiga and Aitken 2003). Further, STI infection in HIV positive men is associated with greater viral load of HIV, which increases the likelihood of transmission to the partner (Cohen et al. 1997). Oster (2005) develops an epidemiological model incorporating plausible sexual behavior parameters for Africa and the U.S. that attempts to decompose the growth of HIV prevalence into behavior and transmission rate determinants. Her simulations suggest that the vast differences in US and African prevalence rates are due primarily to differences in the transmission rates of the virus, which would largely be a reflection of the high levels of untreated STIs in Africa.

A second important cofactor for HIV is male circumcision or more precisely, not having been circumcised. In East and Southern Africa, the regions in Africa with the highest prevalence, circumcision rates on average are significantly lower than in West Africa, where most men are circumcised and HIV rates are lowest. Non-circumcision may raise susceptibility to HIV infection directly[4] as well as indirectly by increasing susceptibility to cofactor STIs. A review of observational studies for Africa indicates that male circumcision is associated with a significantly reduced risk of HIV infection among men, with an adjusted relative risk of 0.42.

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[3] UNAIDS defines a generalized epidemic as one in which adult HIV prevalence among the general adult population is at least 1% and transmission is mostly heterosexual and a concentrated epidemic as one in which HIV is concentrated in groups with behaviors that expose them to a high risk of HIV infection. (See http://data.unaids.org/pub/GlobalReport/2006/2006_Epi_backgrounder_on_methodology_en.pdf)

[4] Among other factors, the tissue of the internal foreskin contains large concentrations of 'target cells' for HIV infection. See Bailey et al. (2001); Auvert et al. (2005).

(Weiss et al. 2000). These comparisons cannot account for all cultural and behavioral differences that distinguish circumcised and uncircumcised male populations, but recent experimental evidence discussed in section II.3.1 confirms that circumcision significantly reduces transmission risk.

Also with respect to physiological factors, there is evidence that pregnancy increases women's susceptibility to infection. Prospective studies of women from Malawi (Taha et al. 1998) and Rwanda (Leroy et al. 1994) indicate higher incidence of HIV among women who are pregnant. These comparisons did not control for differences in risk behaviors or partner characteristics. However, a more recent Rakia, Uganda study (Gray et al. 2005) did control for risk behaviors of the women and their male partners. The results show a (temporary) doubling of the risk of HIV acquisition during pregnancy. The increased risk may be due to the fact that pregnancy leads to a temporary reduction in CD4 count[5], or because of hormonal changes in pregnancy that lead to changes in genital tract conditions.

II.2 Links from reproductive and sexual behaviors to HIV/AIDS

A great deal has been written about the implications of patterns of sexual behavior for the spread of HIV/AIDS in Africa. The region is distinguished, as just noted, by the high prevalence of untreated STDs and in some areas, low rates of male circumcision, each a significant cofactor for HIV infection. But with respect to behavior, there are differences as well, in particular in terms of 'sexual mixing' patterns. Surveys of self-reported sexual behavior in different regions of the world indicate that Africans do not have more sexual partners over their lifetimes than people in other regions. What is different is that it is much more common in Africa than elsewhere to have two or more *concurrent* long-term partnerships. In the West and in Asia, in contrast, individuals are more likely to be serially monogamous, or else if they are not, concurrent partnerships are usually short term, e.g., involving a visit to a sex worker.

With concurrent partnerships that are long term, many more people at a given point of time are linked in sexual networks that in situations where serial monogamy is predominant (Morris and Kretzschmar 1997; Halperin and Epstein 2004). These networks allow the virus to spread rapidly in the population. The effects of concurrency are exacerbated by the fact that HIV viral load, and thus infectivity, is much higher during the initial weeks or months after infection. With serial monogamy, the virus will be trapped in a single relationship until that period passes, whereas concurrency has the potential to expose many people to the virus during the period.

The implications of concurrency for the growth of the epidemic can be demonstrated mathematically (see Morris and Kretzschmar). Empirically, one can learn about partnership behavior from standard surveys of sexual behavior in random samples of individuals, but it is

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[5] CD4 cells orchestrate the body's immune response to viral and other infections.

very difficult to deduce the structure of sexual networks from this information. What is required is information on the behavior of all people in a network and the tracing of the relationships between them. Gathering such comprehensive data is a difficult and resource-intensive proposition, and there appears to be only one example for Africa, the ongoing project in Malawi reported by Kohler and Helleringer (2006). All sexually active individuals age 18-35 in seven villages in an isolated rural area were surveyed for this study. Reflecting the concurrent nature of sexual partnerships, two thirds of the approximately 1000 surveyed individuals were connected to each other in a sexual network via relationships occurring within the previous three years.

Based on the work of Morris and others, the tendency toward concurrent partnerships in Africa is widely viewed as an important factor behind the region's uniquely severe AIDS epidemic. The question remains as to how important this behavioral phenomenon is relative to the physiological factors of low male circumcision and high rates of untreated STIs, but most observers would probably consider all of these to be important contributing factors (see Halperin and Epstein).[6]

Another aspect of sexual behaviors that has drawn a lot of attention is the prevalence in many African societies of sexual relationships of young women and significantly older men, whether through marriage or outside of it. Logically speaking, if all young people engaged in sex only with others in their age group from the time they became sexually active, the epidemic would not be able to continue beyond the current generation of older adults. Ultimately, for this to happen, younger people must engage in unprotected sex with older, infected, people. Sexual mixing via relationships of young women and older men thus provides the virus with a route from older to younger generations. It is reflected in the substantial gender imbalance in infection rates among young people aged 15–24 years—an estimated 4.6% for females and 1.7% for males across the region (UNAIDS 2006).

For fairly obvious reasons, fertility patterns and preferences can also encourage or inhibit the spread of HIV. Married women who, in an effort to limit their family size, reduce the frequency of unprotected intercourse via condoms or reduce the overall frequency of sex lower their risk of contracting HIV from infected partners. In high fertility societies where young women get married (usually to older men) and begin childbearing early, the risk of infection is cet. par. higher. At the same time, however, early marriage will mean less potentially risky premarital sex with non-steady partners, so the net effect on HIV risk is uncertain. Finally, the physiological link between pregnancy and HIV susceptibility noted in the last section suggest another way in which high fertility women are more vulnerable to the disease.

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[6] In contrast, the epidemiological modeling of Oster (2005), as noted above, indicates that the differences in HIV prevalence between Sub-Saharan Africa and the United States is attributable primarily to differences in transmission rates of the virus (reflecting the very high rates of untreated ulcerative STIs in Africa) , though sexual behavior and epidemic timing do help explain differences within Africa. Thus her results argue against sexual mixing patterns as a significant source of the difference between Africa and elsewhere. These results, however, may be sensitive to the way in which sexual networks are incorporated into the model. See Kohler and Helleringer (2006).

II.3 HIV prevention policies: Evidence and gaps in knowledge

The search for effective HIV prevention strategies undoubtedly forms the most crucial research agenda among the topics discussed in this paper. Solutions to the crisis have proved elusive. Until relatively recently, when declines in HIV prevalence were recorded in countries such as Kenya, Zimbabwe, and Zambia, only Uganda among African countries experiencing generalized epidemics could claim to have significantly reduced HIV prevalence and incidence. The success in Uganda has been at least in part attributable to reductions in risk behaviors (discussed further below), suggesting that that country's experiences should hold important lessons for other countries attempting to devise effective strategies for behavior change. However, there is less than complete agreement on what those lessons are. I will discuss these issues in some detail below.

First, however, it should be noted that HIV prevention policy is by no means limited to interventions designed to alter behavior. There are a number of potentially important interventions that are essentially medical in nature. As suggested above, however, several of these are in interest to behavioral scientists because they may have significant secondary effects on the epidemic through induced changes in risk behaviors.

II.3.1 Medical interventions

Antiretroviral drug therapy figures prominently among medical HIV interventions. There are two distinct applications of ARVs: to prevent mother to child transmission (MTCT) of HIV and to extend the lives of HIV positive adults. The former has been the subject of clinical trials in Africa, which have shown that even a single dose of the antiretroviral drug nevirapine given to the mother at the onset of labor and to the baby after delivery reduces by about half the rate of HIV transmission (See Guay et al. 1999; WHO 2006). ARV therapy for adults, long available in the developed world, is expanding rapidly in Africa, though still reaching only a fraction of those who need it. Several studies confirm their life-extending benefits in the context of very poor countries (Ferradini et al. 2006; Severe et al. 2005).

ARV therapy for adults is not a prevention intervention *per se*, but it nonetheless may have impacts on the spread of HIV, impacts which are mediated by behavioral responses. Patients receiving the drugs become much less infectious than before (the presence of the virus in their bloodstream drops precipitously), but if they had been very ill before treatment, they are also likely to become more sexually active—and of course, they will be alive and able to transmit the virus for a longer period of time. Perhaps more importantly, individuals who are not ill or knowingly HIV positive may experience 'treatment optimism': knowing that they can get treatment if they contract HIV encourages them to engage in more risky behaviors. This is an example of a negative externality, but there are also potential positive externalities, such as a reduction in AIDS stigma and a greater willingness of people to get tested if they know treatment is available (Moatti et al. 2002).[7] Informal evidence (see Glick 2005) indicates that

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[7] It should be pointed out that with HIV/AIDS, *any* intervention that alters the level of infectiousness or risk behavior of some individuals will have potentially significant externalities via sexual networks: the private

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the utilization of testing services surges when clinics begin offering ARVs. Still, it is not clear whether demand for testing increases primarily among those who are already ill with AIDS (hence less likely to be sexually active and in danger of infecting others) or also among the apparently healthy, including HIV positive but asymptomatic individuals who are still sexually active and therefore at risk for spreading the virus. From a prevention perspective this distinction is important. These and other behavioral impacts of ARV provision have yet to be considered rigorously. As discussed below, to do so requires the collection of population-based data, not just clinic-based data on individuals receiving treatment.

The discussion above of cofactors for HIV infection points to two other medical interventions which (via the link from 'services' to HIV shown in Figure 1) may have significant effects on the spread of the disease: STI control, and male circumcision. The recently concluded randomized ANRS trial in South Africa (supported by the Agence Nationale de Recherches sur le SIDA of France), using HIV infection as the endpoint, confirms the benefits of male circumcision in terms of transmission risk reduction: incidence was about 60% lower in the group of men getting the procedure relative to controls (Auvert et al. 2005).

Though this trial suggests potentially very significant prevention benefits to male circumcision, there is concern that male circumcision will lead to adverse behavioral reaction through 'risk compensation'(Cassell et al. 2006). It is hard to convey the concept of partial protection, which is what circumcision provides, so men may feel free to engage in more risky behavior than before. This can in theory result in a net increase in their infection risk. Indeed, in the ANRS study, most indicators of sexual risk behavior were higher in the treatment group than the controls following the start of the trial. Despite this, however, the estimate of the protective effect of circumcision was not sensitive to adjustment for participants' self-reported sexual behaviors. On balance, therefore, this study suggests that the benefits to circumcision will be large enough to overwhelm the negative behavioral responses, so this intervention remains very promising. More research needs to be done, however, on the endogenous behavioral responses to circumcision.

With respect to the effects of comprehensive STI treatment programs, findings from three rural community randomized trials, in Rakai and Masaka, Uganda, and Mwanza, Tanzania, are mixed in terms of HIV incidence outcomes, even though STI incidence fell in each case (Grosskurth et al. 1995; Wawer et al. 1999; Kamali et al. 2003). Only in the Tanzania case was there also a reduction in new HIV infections. Together these three studies suggest that STI control will yield HIV prevention benefits when (as in Tanzania) the epidemic is less mature, when transmission is still occurring primarily in specific high risk groups and before there has been significant behavior change in the population (Korenromp et al. 2005). Note that in this case, while there may not be significant effects of the intervention on risk behavior, understanding the nature of that behavior and of sexual networks is necessary in order to interpret the findings.

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decision of individuals to seek medical treatment, testing, purchase condoms, etc., will have benefits or costs to their partners by exposing them to reduced (or greater) risk of infection, and consequently to their partners' partners, and so on (see Over 1999). The example in the text in contrast refers to effects of an intervention on the behavior and attitudes of non-participants through the operation of social networks or other means.

11.3.2 Behavioral interventions

HIV prevention interventions that are explicitly designed to alter behavior include voluntary testing and counseling (VCT); provision and social marketing of condoms; public information campaigns emphasizing prevention behaviors, such as the 'ABC' approach (Abstinence, Be faithful, use Condoms); and a host of education programs aimed at youth to provide HIV knowledge and encourage safe behaviors, most commonly, later sexual debut. Note also that standard reproductive services will also potentially affect the rate of new infections via changes in behavior. For example, if women use condoms from family planning clinics for the purpose of birth control they will also reduce their risk of getting or giving the virus. Family planning services also generally provide HIV risk and prevention information to their clients, which can lead to changes in behavior and risk of infection.

Behavior change promotion: A, B, and C

Considering first behavior change promotion, Uganda remains the standard bearer for behavior change in high prevalence countries and its experience has inspired a great deal of discussion and debate. From the early 1990s to 2001, HIV prevalence in Uganda fell by two thirds, from 15% to around 5% of the population. Much of this reduction is attributable to the natural course of the epidemic, via rising mortality as the initial group of HIV infected persons began to succumb to AIDS; mortality also reduced incidence by removing infected individuals from sexual networks. However, it is generally agreed that reductions in risk behaviors also played a significant role. (Singh et al. 2003; Slutkin et al. 2006; Stoneburner and Low-Beer 2004).   Demographic and Health survey data indicated a 60% decline in sex with non-regular partners between 1989 and 1995 as well as increases among young people in age at sexual debut (Stoneburner and Low-Beer), although there is some concern over comparability of such surveys over time (Gray et al. 2006).

One can consider Uganda's (and other countries') experience along two dimensions: the broad policy stance, meaning the overall nature and intensity of the efforts of government and other actors in getting prevention messages across, and the content of the messages themselves. Uganda clearly indicates the importance of the former dimension. Under the very visible leadership of President Yoweri Museveni, the government attempted to counter the epidemic earlier (starting in the mid 1980s) and far more aggressively than in other countries.[8] This involved the use of mass media and the mobilization of community and church leaders as well as NGOs in education campaigns. Many observers have noted that the frequent open public discussion about AIDS served to destigmitize the disease. This is a somewhat difficult concept to quantify empirically, but it is noteworthy that by the mid 1990s the share of individuals indicating that they knew someone with AIDS or who had died of AIDS was substantially higher in Uganda than in similarly (or worse) afflicted countries where general awareness of

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[8] Senegal also is notable for an aggressive and early policy stance against AIDS. Unlike Uganda and most of Africa, these actions were able to contain the disease before it reached generalized epidemic stage; HIV in Senegal still appears concentrated among specific high risk groups such as sex workers (UNAIDS 2006).

AIDS was equally high: 91.5% of men and 86.4% of women (in 1995), compared with 68% to 71% in Zambia, Kenya and Malawi and below 50% in Zimbabwe. In South Africa, the share was below 50% as late as 2002 (Stoneburner and Low-Beer 2004). This suggests a widespread willingness to acknowledge the disease and presumably also, the risks that it poses.[9]

Exactly which behavior change messages were successful—and which should be used now in Uganda and elsewhere—is still being debated. Uganda is said to demonstrate the power of the 'ABC' approach (Abstinence, Be faithful, use Condoms) to prevention. As many observers have noted, however, condom promotion was not a major plank of Uganda's early national prevention strategy and condoms were not distributed or used widely enough to have played a significant role in the decline in HIV rates in the early 90s (Green et al. 2002). Nor did the early messages emphasize abstinence *per se*, though among the young, delayed sexual debut was encouraged (Slutkin et al. 2006). The strongest emphasis was on faithfulness to one's partner ('zero grazing'), or if not that, minimizing the number of casual partners.[10] The evidence cited above indicates success in reducing the incidence of sex with non-regular partners as well as more modest reductions in sexual activity among youth.

Condoms began to be promoted more heavily in Uganda in the early to mid 1990s using social marketing campaigns. This new emphasis seems to have affected the nature of prevention. Longitudinal data from Rakai district for the period 1994-2003 indicate that falling HIV prevalence was due to a combination of (primarily) rising AIDS mortality, on the one hand, and behavior change, on the other. This time, however, behavior adjustment took the form of increased condom use; levels of other self-reported risk behaviors did not change or even increased over the period (Wawer et al. 2005).

Therefore the Ugandan experience provides evidence for the efficacy of behavior change messages emphasizing partner reduction and, in a later period, condom use, though the latter success is clouded by the possibility that people who use condoms will feel free to increase or not reduce other risky behaviors. Abstinence apparently was promoted in the sense of messages to delay initiation of sexual activity, but not in the form of 'abstinence only' (until marriage) education.[11] This may have led to some movement in age at sexual debut among young people. A general point should be noted, however, to which I return below: when considering trends in behavior, it is very hard to distinguish between the effects of policies (and even more so, specific policies) and the responses people are likely to make in the face of the epidemic even without these policies. That said, the correspondence of aggressive mobilization

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[9] The responses from the DHS indicate either that people in Uganda were more likely to recognize other's illness and mortality as being caused by AIDS, or that others or their families were more likely to admit to having AIDS, or both. Since AIDS victims do not die from the HIV virus itself but from a variety of other infections that take advantage of weakened immune systems, it is easy (and very common in Africa) for families to claim that death was from some cause other than AIDS.

[10] Thus 'ABC', in the sense of an equal emphasis on each element, is not a totally accurate characterization of Uganda's early successful prevention campaign; indeed the phrase apparently came only into use only later, in the mid 1990s (Slutkin et al. 2006).

[11] The distinction is important, as abstinence (and especially, abstinence-only) messages for young people have become much more prominent in Uganda in recent years, encouraged by social conservatives, the US government, and First Lady Janet Museveni. For a discussion of the controversy see Epstein (2005).

efforts and substantial behavior change in Uganda from the late 1980s to the mid 1990s—and the comparative lack of both strong policies and behavior adjustment in other countries at similar stages of the epidemic[12]—offers strong support for the idea that policy played a significant role in changing behavior in Uganda.

With regard to condom promotion, few would argue with the efficacy of providing condoms to sex workers and other typically very high-risk groups such as truck drivers and the military. Such drives, beginning with Thailand's 100% condom use policy for brothels and subsequently copied elsewhere (UNAIDS 2000; Larivee 2002), have generally been quite successful both in achieving very high rates of condom use in the targeted populations and in bringing down infection rates in these populations. These successes, however, occurred in contexts of concentrated epidemics, where transmission was still occurring largely via these high-risk groups. In Africa, as noted, epidemics tend to be generalized, with transmission occurring throughout the general population. The issue then is whether condom promotion to the general population has been or can be an effective prevention strategy.

The African record on this to date is in from one perspective rather disappointing. Countries such as Kenya, Botswana, and South Africa had policies strongly promoting condoms for years with apparent success in increasing their acceptance and use but with little to show in terms of reduced prevalence (Hearst and Chen 2004). It is hard to draw conclusions from these simple cross-country comparisons because countries differ in how aggressively government in general pursued its AIDS education and prevention objectives.[13] Still, these experiences indicate that it is unlikely that condom promotion in the absence of successful promotion of other risk behavior reduction is sufficient to turn back the epidemic—there is no such "condom success story" (Green et al. 2006). One problem is that users of condoms tend not to use them consistently (Hearst and Chen). Further, it is possible that such intermittent use of condoms provides a false sense of security so that people feel comfortable persisting in high-risk behaviors (Ahmed et al. 2001); that is, they exhibit risk disinhibition. A second problem is that people are reluctant to use condoms in long-term partnerships as this implies a lack of trust.[14] Unfortunately, this is the context in which much or most HIV transmission occurs in Africa.

Nevertheless, in several countries (and in Rakai, Uganda as noted above) recent declines in HIV prevalence were associated with increasing condom use in casual partnerships. In these cases, unlike for Rakai, survey data show condom use increasing in step with reductions in other risk behaviors such the number of partners and adolescent sexual activity. This pattern was seen for Kenya (Cheluget et al. 2006), Zimbabwe (UNAIDS 2005; Gregson et al. 2006a), and urban Zambia (Fylkesnes et al. 2001) (we return to these country cases further below). Condoms probably contributed to declines in new infections in these cases. It is also

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[12] The epidemic appears to have started somewhat earlier in Uganda than in neighboring countries, fostered by years of civil war and dislocation that ended in 1986.

[13] Though Allen and Held (2004), as noted below, argue that policymakers in Botswana indeed pushed hard, but that the emphasis of policy—on condoms—was misdirected.

[14] Condom use appears to be high within serodiscordant couples who have been tested (see below in the discussion of VCT), but with testing rates still low the vast majority of serodiscordant couples in Africa are not aware of their HIV status.

important to point out that access to and use of condoms is essential when (as in Uganda today) half or more of new infections occur in serodiscordant couples (couples in which one partner is infected but the other is not). For many of these couples abstinence is presumably unattractive (Merson et al. 2000).

The evidence discussed above on condom promotion does not come from controlled studies or evaluations of specific programs. For policies promoting condom use among the general public (as opposed to specific high-risk groups) such evaluations unfortunately are uncommon. Evidence of effectiveness of condom promotion or social marketing programs often relies on the numbers of condoms distributed or sold, and occasionally, changes in self-reported condom use. While condom promotion in Africa does appear to work in this sense (Hearst and Chen 2004; Foreit 2001; Myer et al. 2001), the effect on prevention remains unclear without information on which groups (in terms of risk levels) use them, on whether they are used consistently, and on what happens with respect to other risk behaviors—and ultimately of course on changes in the rate of new infections.

A rare evaluation that does attempt to gather such data is the recent randomized trial in Kampala, Uganda (Kajubi et al. 2005). Recruited men in one poor community participated in a workshop that taught condom skills and encouraged condom use. Men in the control community received a brief informational presentation about AIDS. All participants received coupons redeemable for free condoms from distributors in both communities and completed questionnaires at baseline and six months later. It was found that men in the intervention group redeemed significantly more condom coupons than men in the control group, but they also increased their number of sex partners by 0.31 compared with a decrease of 0.17 partners in the control group. Thus the gains from increased condom use seem to have been offset by increases in the number of sex partners. This study provides evidence of a disinhibition or 'risk compensation' effect of condom use: individuals adopted one form of protective behavior and compensated by being less careful in other dimensions, possibly leading to an increase in net HIV risk (especially if condoms were not used consistently). As noted, the trends in behavior in Rakai since 1993 are also consistent with this process. Therefore questions remain about the overall impacts of condom promotion in generalized epidemics. Studies are needed, in particular, of the impacts of programs promoting condoms in conjunction with a strong emphasis on other risk behavior reductions. Ideally, the measured endpoints would include change in HIV incidence as well as behaviors.

With the exception of evaluations of voluntary counseling and testing and in-school interventions for youth (both discussed below), there are few other studies of this type in Africa that evaluate specific behavior change promotion programs, at least with respect to impacts on behavioral or biological endpoints. Williams et al. (2003) assess the effects of an intensive HIV intervention started in 1998 in a mining community in South Africa. The program included community-based peer education, condom distribution, syndromic management of sexually transmitted infections, and presumptive STI treatment for sex workers. Despite the intervention there was little evidence of significant behavior change over a two-year period and the prevalence of non-HIV STIs actually increased. It should be noted that there was no control community; the method was a simple pre- and post-test design using two cross-section

surveys. The authors suggest that the context was important in explaining the lack of response to the intervention: AIDS mortality was still low, and the South African government was not putting out broader messages about HIV risk and behavior change.

A rather different behavior change intervention, similarly evaluated in a simple pretest-posttest framework, was an AIDS prevention project implemented in Muslim communities in Uganda (Kagimu et al. 1998). This intervention trained religious leaders who in turn educated their communities about AIDS. After two years, there was a significant increase in the share of residents with correct knowledge of HIV transmission, methods of preventing HIV infection, and the risks associated with ablution of the dead and unsterile circumcision. Reductions in sexual risk behaviors were also recorded. However, the study design did not allow the disentangling of intervention effects from trends in these communities or in the country as whole.

It bears clarifying the differences between these program evaluations and the type of data discussed above. The latter consists essentially of trends observed in cohort or repeated cross-section data in a country or subnational region. As such, outcomes may reflect the influence of factors other than the policy of interest that have also changed over time, such as increasing knowledge of AIDS risk diffusing through social networks, endogenous responses to changes in prevalence and mortality (discussed below), and the operation of other policies and actors, for example, NGOs. The lack of a control or comparison group makes it impossible to distinguish policy impacts from these confounding factors (this is also a problem, of course, in project evaluations that do not have control groups). The best one can usually do is to compare trends in countries where policy approaches have differed, or as in the case of Uganda, consider different periods between which policy shifted emphasis from one prevention approach to another. Of course, may many other things may be different across countries or time periods.[15]

Programs aimed at youth

In Africa as elsewhere, many programs have sought to educate young people about HIV risk and reduce behaviors that expose them to risk: early sexual initiation, sex without condoms, or sex before marriage. Most commonly these programs operate through schools. The evidence of their efficacy in Africa is at best mixed. A recent comprehensive review of 11 school-based HIV education programs (Gallant and Tyndale 2004) indicates that while such interventions can be successful at improving young people's HIV awareness and attitudes, most did not produce sustained changes in behavior.

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[15] Moore and Hogg (2004), discussed further below, are able to control for many of these 'other things' by looking at trends in HIV prevalence in areas in Western Kenya and Eastern Uganda along the border of the two countries. For these two geographically proximal areas, there are few important differences in factors such as ethnic groupings or male circumcision rates, so differences in prevalence trends are more likely to be due to the major differences in policy stance of the two countries. This comparison comes closer to the ideal of intervention and comparison group study design than comparing countries as a whole.

Most of these evaluations did not employ randomized designs, and most used self-reported behavior outcomes as endpoints rather than HIV infections or other biological endpoints such as teenage pregnancy or STIs. However, several additional school-based prevention programs in Africa do make use of randomized controlled trials (RCTs) and in a few cases, the measured outcomes include HIV incidence or other biomarkers. These too have produced mixed results. In rural Tanzania, a community-randomized design was used to evaluate a project including in-school education, youth-friendly health services, and community-based condom promotion and distribution. The program led to improved knowledge, attitudes and self-reported behaviors in the ten intervention communities relative to the controls, but there was no consistent impact on biological indicators of HIV, other STIs, or pregnancy (DFID 2004). A Rakai, Uganda RCT of an extra-curricula education program (Kinsman et al. 2001) found no significant impacts on teenagers' self-reported behaviors; in this case poor implementation may be partly to blame.

A multi-arm randomized evaluation in Western Kenya (Dreyfuss et al. 2006) found that training teachers in Kenya for the HIV/AIDS curriculum did not lead to any reduction in teenage pregnancy but did increase the likelihood that teenage pregnancies occur within marriage. In-class debates over condoms and opportunities to write essays on ways of protecting oneself against HIV/AIDS led to increased self-reported use of condoms without an increase in self-reported sexual activity. Reductions in the cost of schooling led to reductions both in dropout rates and teen pregnancies. As Dreyfuss et al. note, in the absence of biomarker outcome measures, the implications of each of these program effects for HIV risk, while promising, are not clear. For example, an increase in teenage pregnancy within marriage at the expense of pregnancy outside of it may actually increase HIV risk if there is a greater tendency for the former to involve unprotected sex with older men.

On that score, a promising recent finding (Dupas 2006), from an extension of the same Kenya project, is that a program informing girls about the much higher HIV risk from older men relative to teenage boys led to a 65% decrease in the incidence of pregnancies by adult partners among teenage girls in the treatment group relative to the comparison group. Given these striking results, it would be of interest to replicate and evaluate this type of program in other contexts. Further, the generally disappointing findings for many other interventions aimed at youth point to the need for more careful design and evaluation of such programs. The need for rigorous evaluation would certainly apply to 'abstinence-only' programs, which are being heavily promoted in Uganda and elsewhere. There are no evaluations in Africa of this approach or comparisons of it with other programs for youth, but abstinence-only programs in the U.S. have by and large been found to be ineffective at delaying sexual initiation and reducing sexual risk-taking behaviors in the long term (Kirby 2001).[16]

The foregoing review has concerned school-based awareness and prevention programs. Evaluations of non-school interventions aimed at youth are less common, presumably a

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[16] A related controversy is whether advising young people about condoms encourages earlier sexual activity. As noted, Dreyfuss et al. for Kenya found that condom education increased condom use by teenagers but not rates of sexual activity (both behaviors self-reported).

reflection in part of the much greater ease with which evaluators can survey young people in their schools.[17] Agha (2002) reports on a quasi-experimental evaluation of adolescent sexual health interventions in four African countries (Cameroon, Botswana, South Africa and Guinea) in the mid to late 90s. These interventions combined to varying degrees mass media (radio messages), sponsored events, peer education and youth-friendly contraceptive services. Changes between baseline and follow-up surveys were compared for intervention towns or neighborhoods and selected comparison locales. Population impacts on perceptions and self-reported behaviors varied (and tended to be larger for young women), but one conclusion is that more intensive interventions using a variety of channels are needed to insure that a large share of adolescents is reached.

HIV testing

Currently only a small minority of adults in Africa are aware of their HIV status, many governments hope to change this by expanding access to testing and counseling services. Voluntary HIV Testing and Counseling (VCT) typically consists of a pre-test counseling session with a trained counselor, the serotest itself, and a post-test session in which individuals are counseled on behaviors to insure that they remain uninfected (if they test negative) or avoid infecting others (if positive). Those testing positive are also provided emotional support, and directed to services to provide palliative care and other forms of support. Testing is also the gateway to antiretroviral drug therapies for HIV positive individuals. This role of HIV testing is growing in importance as many African countries scale up the provision of ARV drugs.

Although there are a number of evaluations of VCT in Africa, most are based on simple single group pretest and posttest designs, whereby (self-selected) clients are interviewed before they receive the testing and counseling and are followed up some time later (See Glick 2005 for a review of this research). Without similar baseline and follow-up information for a control or comparison group, it is not possible to distinguish the effects of the intervention from general trends in behavior over time. Equally important is the problem of self-selection. Given heterogeneity in the population with respect to (for example) the motivation for behavior change and risk avoidance, those who choose to use VCT may be especially responsive to the information received about their serostatus and about HIV prevention. Hence they may adjust their behavior following VCT more than would individuals in the target population in general.

For these reasons, the first randomized controlled trial of VCT in Africa, conducted in urban Kenya and Tanzania as well as Trinidad (Voluntary HIV-1 Counseling and Testing Efficacy Study Group 2000), attracted significant attention both in the research community and the popular press. Volunteers interested in testing were randomly assigned to intervention and control groups; the latter were given general information about HIV/AIDS but not VCT. [18]

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[17] The survey by Bollinger et al. (2004), covering interventions in all developing countries published as of two years earlier, could not locate a single evaluation study of non-school based adolescent prevention programs that used as outcomes measures either self-reported condom use, number of partners, or age at first sex.

[18] However, as Glick (2005) notes with respect to this much cited study, even a rigorously conducted individualized randomized study may not provide meaningful estimates of program efficacy. Individuals are

Relative to controls, there were reductions in unprotected sex among testing serodiscordant couples (the study design insured adequate representation of couples testing together) and among HIV positive testers in general. Changes in behavior among those testing negative were much smaller. This is the same general pattern, in fact, that was found in many non-experimental studies. It suggests that the testing and counseling has some value in secondary prevention (i.e., preventing infection of the partners of those who are HIV positive), but less impact on preventing primary infection among those who are HIV negative.

The public health effectiveness of voluntary programs such as VCT depends not just on the response of those who participate, but on the extent of participation, or program coverage. For most countries in Africa, Demographic Health Surveys (DHSs) indicate very low numbers reporting having had an HIV test but high shares (about two thirds) saying they would like to lean their status (Glick and Sahn 2007). In Kenya, where the government in recent years has been rapidly expanding the number of testing sites, the overall numbers tested annually have increased dramatically, from 1,100 in 2000 to over a half million in 2005 (Marum et al. 2006), pointing to a strong demand for testing that had been constrained by a shortage of facilities. However, a formal analysis of the demand for HIV testing requires the use of population-based survey data collected in areas where the service is available.

The findings from several such studies provide a mixed picture. In a rural Rakai, Uganda study (Nyblade et al. 2000), VCT services were offered to all individuals, who could choose to receive the service in their homes or at a nearby clinic. Despite significant outreach, demand in the initial year of the program (1995/6) was not very high—32% of women and 35% of men agreed to receive their test results. However, this jumped to 65% for both sexes in 1999/2000 (Matovu et al. 2002).[19] In an urban Zambia study (Fylkesnes and Siziya 2004), uptake was much lower. Even where there was similar flexibility in setting (a clinic or at home) the probability of both indicating 'readiness' for testing and using the service was only about 18%.

Similarly, in a more recent study of rural Malawi (Thornton 2006) only about 40% of the individuals offered free testing chose to attend clinics to learn their HIV status. On the other hand, demand was very sensitive to monetary incentives: small cash payments (offered on randomly assigned basis) were enough to double the use of the testing service. This study not only randomized the testing incentive; it also used an objectively measured rather than self-reported behavioral outcome, namely, whether the individual decides to purchase condoms at the subsidized price. Consistent with studies of VCT outcomes discussed above, testing had no effect on condom purchases for those testing negative, but those who tested positive and had a

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randomly assigned to intervention and control groups only after recruitment into the study. Recruitment itself is not random but relies necessarily on volunteers and these individuals may be unrepresentative of the target population in terms of motivation or other factors influencing responsiveness to the intervention. The randomized trial does insure stronger internal validity, meaning that it provides reliable estimates of the effect of VCT on those who volunteer, the 'effect of treatment on the treated'. But external validity—the credibility of the findings as indicators of efficacy for the target population in general—may be weak.

[19] Though it should be noted that this is conditional on having agreed in the first place to give a blood sample (78% of respondents).

partner purchased significantly more condoms than non-testers. However, the average number of condoms purchased was small, so the overall cost-effectiveness of VCT in this setting was low.

The evident reluctance on the part of many to get tested may reflect a number of factors: significant stigma attached to AIDS, stress, and a strong reluctance to reveal a positive test result to a partner, particularly for women who may realistically fear domestic violence or divorce as a consequence. In view of these factors, some observers have recently questioned the usefulness of the VCT model for Africa (and elsewhere). Imported from developed countries, the VCT approach, as the word 'voluntary' implies, typically places a premium on privacy and personal choice in health care. In contrast, a policy of 'mandatory'—or perhaps more accurately, 'routine'[20]—testing would automatically test all individuals entering the health care system. Such a policy treats HIV as a public health issue in the same manner as other communicable diseases have been treated in the past in the West. Botswana and Lesotho have recently become the first African countries to initiate national policies of routine HIV testing.

Both the ethics and efficacy of mandatory testing have been hotly debated (see UNAIDS 2004 and Holbrooke and Furman 2004 for opposing views). The population level implications for risk behavior and the coverage of testing itself are unknown; with regard to the latter, it is possible that certain high risk or vulnerable groups will respond to a policy of automatic testing by deciding to stay away from the health system entirely. Assuming that coverage does increase significantly under routine testing, it is quite possible that the average behavioral response to testing and counseling will be different than what was observed in the samples studied in the VCT evaluation literature. This is because coverage will move beyond those who actively volunteer to be tested (presumably meaning those who perceive the greatest benefit from the information), to those who have less interest in knowing their status. Several plausible models of the demand for HIV testing and for behavior change predict that those who perceive less benefit to testing (and who therefore are not among those who test early) will adjust their behavior less in response to the intervention, though the opposite pattern is also possible (see Glick 2005).

This consideration applies as well to other policies designed to significantly expand coverage of testing or VCT, whether by offering cash incentives or by changing the mode of service delivery, e.g., replacing standard VCT clinics with mobile testing units. With respect to evaluation, it is obvious that to understand the demand (uptake) impacts as well as the behavioral effects of these policies, it will be necessary to analyze population-based data, not the clinical data used in most VCT studies. As noted in Section II.3.1, this applies also to assessments of the behavioral impacts (including impacts on testing demand) of making ARV therapy available to HIV positive individuals or those with AIDS.

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[20] 'Routine' means that an HIV test is given by default as part of any medical care, but the patient has the option of refusing the test; hence it is an 'opt out' approach to testing as opposed to the 'opt in' approach of VCT.

Integration of HIV prevention and care into existing family planning/reproductive health services

This issue is especially pertinent to a discussion that is concerned with the intersection of HIV/AIDS, reproductive health, and fertility. By and large throughout Africa, the current approach implements services for HIV (and STI) prevention, testing, and care separately from traditional family planning and reproductive health care, with the exception of interventions to reduce mother to child HIV transmission. Many, including UNAIDS and the United Nations Population Fund (UNFPA 2004 a, b) make an efficiency argument for integrating the two. Existing reproductive health infrastructures provide a ready-made conduit to supply HIV related services, and combining the provision of both kinds of services would enable the realization of economies of scale in delivery. It would also ensure that women in particular get access to HIV services.

Others are skeptical about integration in the African context, however (see Caldwell and Caldwell 2002; Foreit et al. 2002). The primary concern is that traditional family planning and maternal/infant health services, on the one hand, and HIV services, on the other, generally serve different clienteles with different needs. The main clientele for family planning and related services is married women. For many HIV services (testing, condom provision, etc.) the clienteles will consist of men, or of adolescents of both sexes. Concentrating HIV resources in family planning settings may alienate these groups, making them embarrassed or reluctant to use the services and possibly harming prevention efforts overall. Adolescents, for example, might be better served via separate 'youth-friendly' programs. It has also been pointed out that since (as inequitable as this situation may be) women often lack power in sexual decision-making, behavior change efforts targeted at the main clientele of family planning/reproductive health services may be ineffective.

The feasibility and benefits of providing a range of HIV/STI services to the typical female clienteles of family planning/reproductive health centers (as opposed to trying to reach other key groups via these means) is a somewhat different matter. Askew and Maggwa (2002) notes that even for this more limited objective there may be difficulties, citing unfavorable experiences in Africa with the detection and treatment of (non-HIV) STIs in family planning clinics. On the other hand, integration may be more feasible for certain activities, such as prevention of mother to child transmission and VCT. Ongoing research in Ethiopia and South Africa (Perchal et al., 2006; Homan et al. 2006) suggests that this can be cost-effective.[21] Integrating services such as VCT into family planning/reproductive health centers may also increase testing among women (if not men) by providing a more secure environment for them than stand-alone VCT centers.

It has also been pointed out (Reynolds 2006; Mphuru et al. 2006) that many potential clients of VCT—in particular adolescent girls—are also at risk of unintended pregnancy. Such

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[21] These and a number of other relevant studies were presented at a 2006 symposium titled "Linking Reproductive Health, Family Planning and HIV/AIDS in Africa." See http://www.jhsph.edu/gatesinstitute/policy_practice/leadership_forums/fp-hivmtg/agenda.html

pregnancies might be reduced if family planning information and contraceptives were offered in conjunction with HIV testing. In other words, family planning coverage and impacts may be strengthened by integration with HIV prevention programs. The notion that one can protect oneself against both HIV/STIs and unwanted pregnancy ('dual protection') may increase the attractiveness of condoms and other risk prevention methods (WHO 2003), especially for the young and unmarried. Indeed, a recent analysis of trends in DHS data from 18 African countries (Cleland and Ali 2006) documents a substantial rise in the use of condoms reported by young, sexually active single women, and at least 60% of those using a condom at last sex said they did so mainly or partly to prevent pregnancy. These findings suggest that among this group, promotion of condoms as a contraceptive device may be more effective than emphasizing the HIV/STI protection benefits. This is a further argument for 'integration' of family planning and HIV/STI prevention for this population—but not necessarily through existing family planning networks, which are not used by young single women.

At this time, the overall effectiveness (and cost-effectiveness) of service integration with respect to HIV prevention objectives, and in particular, the impacts on the use of HIV-related services by non-traditional clienteles such as men and adolescents, is not known. This would require more complex study designs than have been used so far to study the issue. For example, population-based surveys would be needed in both intervention and control communities to understand the impacts on uptake of both fertility/reproductive health services and HIV/STI services among different at-risk groups in the population.

II.3.3. Methodological issues in the evaluation of HIV and reproductive health interventions

There are many studies for Africa evaluating the effects of various interventions on behavior and (more rarely) HIV incidence or other biological endpoints such as STI infection and pregnancy. As the preceding discussion made clear, however, many questions remain as to which programs are truly effective. Conflicting or inconclusive results in the evaluation literature may reflect differences in study contexts as well as variation in the quality of programs or their implementation. But another factor that is probably also very important is variation in study design and evaluation methodology. There are many methodological challenges to evaluating behavioral HIV interventions and most existing evaluations do not meet these challenges completely.

The most serious challenge arises from the fact that, as alluded to above, people usually choose whether or not to participate in an intervention or use a service. Those who do so may not be representative of the overall population or the target population for the intervention. We might expect participants to be relatively responsive to the information or behavior change messages received. On the other hand, if participants in a prevention-related program do have a greater propensity to adjust their behavior, they may have made these adjustments prior to participating. In the first case, comparison of participants and non-participants will overstate the likely benefits of the program for the target population as a whole, while in the second case it will understate them.

Community-level assessments of program impacts, in which mean outcomes at the community level are measured, may suffer from a similar problem, because the placement of programs is usually not random. Placement may be related to a range of local characteristics, such as the demand for services or a greater perceived need for them in the eyes of policymakers (e.g., because of very high rates of risky behaviors or HIV prevalence in the community). These characteristics may easily be correlated with the outcomes to be influenced by the program, and thus will bias assessments based on simple comparisons with communities not receiving the intervention.

In what follows I discuss alternative approaches to dealing with these issues when assessing the effects of HIV and related reproductive health interventions. First, however, mention should be made of the types of outcomes measured in the evaluations of programs and policies, the variation in which was brought out by the preceding review of evidence. Typically for behavior change interventions the endpoints measured are self-reported risk behavioral correlates of HIV rather than HIV infections itself, though sometimes more objectively measured biological correlates such as STI infection or symptoms and pregnancy are used. There are good reasons for this. It is obviously of interest to understand effects on behavior, especially for interventions explicitly designed to affect behavior. Further, attempting to directly measure impacts on the rate of new infections can mean significantly expanding the scope and complexity of the evaluation, both in terms of sample size (large samples may be needed to detect statistically significant changes in incidence, especially where incidence is low) and procedures (the study must be able to test participants for HIV at different points in time, and ethical considerations dictate providing the existing standard of care to those testing positive).

A shortcoming of relying on behavior data, however, is that the link between changes in specific behaviors and the potential for reductions in HIV incidence—the ultimate goal of prevention interventions—is not very clear. It depends on a host of factors, including who (in terms of risk status) is changing behavior, the interactions of different behaviors that may be changing, and the stage of the epidemic (Garnett et al. 2006). There is, further, the well-known problem of biases in self-reported sexual behavior information. Although surveys like the DHS manage to achieve very high response rates, people may not be willing to answer personal questions about their behavior in a truthful way. In particular, comparisons of male and female responses suggest that women are prone to understate the number of partners they have; it is also possible that men, especially single men, overstate the number (see Gersovitz et al. 1998).[22] Data issues will be discussed further in Section II.4 below.

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[22] de Walque (2006) is able to address this issue more directly using recent DHSs with HIV testing. He finds a non-trivial number of cases of serodiscordant couples in which the female rather than the male is the HIV positive partner, meaning that the woman contracted the infection outside the partnership. These cases are at often at odds with self-reports about extra-marital partners by the woman and it can be inferred that a significant share of them are not due to infections received prior to the current relationship. Thus his analysis points to at least some underreporting by women of the number of current or recent sexual partners.

Experimental designs

Given the problems of selectivity in program participation and placement, the optimal approach to evaluation is usually to conduct randomized controlled trials (RCTs), or 'policy experiments', in which an intervention is randomly assigned to some individuals (or areas, or schools, etc) and not others. Generally in practice both groups are measured at baseline (pretest) and follow-up (posttest), though this is not strictly necessary if the randomization is done well, meaning that it results in essentially equivalent treatment and control groups.[23] The use of RCTs has been slow to gain ground in African HIV prevention research, and the number of examples remains fairly small. However, the approach is becoming more common. I note examples in what follows, and then discuss alternative approaches to measuring effects of policies.

Individual level randomization, the standard design in medical trials, has so far been used in only a small number of HIV prevention behavior evaluations in Africa. One prominent application of individual randomized controlled trials is the study of VCT conducted in urban Kenya and Tanzania (as well as Trinidad) mentioned earlier. Two other examples, also involving testing, are the studies reported by Thornton (2006) and Fylkesnes and Siziya (2004). In the former study, individuals in a rural Malawi sample were randomly assigned to receive cash incentives to get tested. As noted, demand for testing proved responsive to the monetary incentive. In Fylkesnes and Siziya's urban Zambia study, participants who had indicated "readiness" to be tested were randomly assigned to receive VCT at a clinic or in a setting chosen by the participant that included home counseling as one option. The mode of delivery mattered greatly: far more of those offered the choice of location used the service.

A different example is the RCT of male circumcision, the recent ANRS trial in South Africa described earlier. In this study, participants were men initially expressing a willingness to be circumcised to reduce their HIV risk. This group was then randomized into treatment and control groups (the latter were offered the procedure at the end of the trial). As noted in section II.3.1, there was a two-thirds reduction in HIV infection risk in the group of men getting the procedure.

In addition, several *community* (or more generally, *group*) level RCTs have been implemented in African settings. These have a number of important advantages for prevention research. Outcomes measured at the level of the community—whether they are biological endpoints or self-reported behaviors—capture the effects of local interactions and externalities that arise through social and sexual networks, learning, etc. The analysis at this level is intention to treat, since means of outcomes are measured over both program participants and non-participants, usually using random samples of the catchment area population. Hence population level estimates of program effectiveness are provided that reflect in part the uptake of the intervention. Examples of this approach include the three community-randomized trials of STI control, in Masaka and Rakai regions, Uganda, and in Mwanza, Tanzania. Another community-randomized trial currently underway in Tanzania, Zimbabwe, and South Africa will

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[23] It will be useful if randomization is not perfect, as noted below. Even when the randomization is valid there are several advantages to collecting baseline data, including greater statistical precision (see Duflo et al. 2006).

compare population level incidence and behavioral outcomes of community-based HIV voluntary counseling and testing to standard clinic-based VCT. Other examples of group randomization include the evaluations of school-based prevention education programs discussed above in Uganda, Kenya, and Tanzania. Here the randomization is fairly straightforward: the intervention is introduced in some schools and not others.

Community randomized experiments would be the ideal approach for investigating a number of other HIV programs that are hypothesized to generate significant external effects.[24] This would include the provision of antiretrovirals. As discussed earlier, the prevention effects of this intervention, operating through the demand for testing and risk behaviors in the community at large, are potentially significant but little is known about them. The introduction of testing sites providing the drugs is typically staggered, reflecting resource and logistical constraints: the drugs will initially be available in certain areas and not others. The latter can form natural 'late treatment' controls during the period for which ARVs are still unavailable in them. The implications of a policy of routine HIV testing could also be assessed using this approach, for example by implementing the policy in certain health districts and not others.

Randomized evaluations of fertility and reproductive health interventions that consider behavioral outcomes such as childbearing and contraceptive use are also rare in Africa.[25] This is true as well for quasi-experimental designs (defined below) that approximate the setup of randomized controlled trials. Those that have been carried out have focused on demographic or health outcomes and have not explicitly considered possible HIV-related effects. One quasi-experimental evaluation for Africa is the Navrongo experiment in Ghana, a community-level analysis which considered the effects on fertility and child health of interventions for training community health officers and for community mobilization (Phillips et al. 2005). Another community trial (again, not randomized) in rural Gambia (Luck et al. 2000) considered the effects on modern contraception use of a community-based mobilization intervention to increase the demand for contraceptives. A rare randomized community trial is the evaluation of the effects of community-based family planning outreach in Uganda (Lutalo et al. 2006) on modern contraceptive use and pregnancy.

Quasi-experimental designs

Implementation of randomized experiments is often difficult for financial, logistical or political reasons. Randomized studies can be complex and are potentially costly, especially community-randomized trials. It is often difficult to get governments or donors to agree to distribute a service or drugs on a strictly randomized basis to certain communities (or schools, or individuals) and not others. These factors in large part explain why randomized trials, outside of clinical settings for medical interventions, remain fairly rare in Africa.

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[24] As noted (see fn 7) this would characterize any HIV prevention intervention, given impacts on others in the population via transmission of risk though sexual networks. Beyond this, however, some interventions will also have impacts on behaviors or attitudes of non-participants that are important to understand.

[25] Clinical randomized trials of medical interventions related to fertility and reproductive health are more common, but as indicated, the focus here is on behavioral interventions or at least, on evaluations considering behavioral outcomes.

There are certain methodological concerns as well. While the results of randomized trials may have internal validity in the sense of accurately measuring the effects of the intervention on those in the study, external validity—the applicability of the results to the full population or target population for the policy—may not be assured. This is especially a concern for smaller pilot studies. The study population in such cases will be relatively small and homogenous, hence may not be representative of the target population overall. Another concern is that once a project is scaled up, the nature of the implementation of the intervention may differ significantly from the initial experimental study. For example, a small pilot project may be allocated more resources, have better supervision, and generate greater motivation on the part of service providers and beneficiaries than the same program would if expanded to a national scale. To some extent, experiments can be designed to minimize these problems, in particular by carrying out the trials on a larger scale and using more representative study populations. However, this will raise the cost and complexity of the study.

For these reasons, a quasi-experimental design may be necessary or preferred. These are research designs in which the selection of treatment and comparison groups occurs by some means other than random assignment, though the term is often used to refer to a range of statistical methods that attempt to establish causal effects of interventions that are not randomly assigned. Careful selection of comparison groups so that they are similar to treatment groups and the collection of baseline data (to control for remaining initial differences in the two groups) are essential. The two non-randomized community-level fertility interventions noted above adhere carefully to this approach, as does the study by Agha (2002) on adolescent sexual health interventions reported earlier. But many non-experimental evaluations, including evaluations of HIV interventions, do not. For example, a common design for VCT evaluations is simple pretest/posttest in which there is no comparison or control group; instead the treated (VCT clients) act as their own control group via their pre-test or baseline data, and the effect of the intervention is measured by the change over time in their behavior (see Glick 2005). As discussed, this approach fails to control for trends and shocks that may affect outcomes independently of the intervention.

The option of using well-conceived quasi-experimental designs clearly opens up many more possibilities for meaningful policy evaluation. This is perhaps easiest to see for community-level evaluations. As noted for ARV provision, most programs are not rolled out all at once. Even if the selection of communities that first receive the programs is not random, it may be possible to select appropriate comparison communities among those not first in line to get the programs for baseline and follow-up surveys.

Instrumental variable regression is another quasi-experimental approach and is one that is very familiar to economists. It is particularly useful for modeling individual level outcomes of programs. Exogenous factors, including proximity to a service or program, can be used to predict individual participation and thus the effects of the program. Also familiar to economists are regression discontinuity designs and propensity score matching techniques. In the latter, individuals with observed characteristics similar to those of individuals receiving treatment are used as controls. This approach would appear to have limits when it comes to

looking at interventions to change sexual behaviors (say, VCT), since participants are likely to differ from non-participants with respect not just to observed characteristics but also to unobserved propensities, attitudes, or risk behaviors that affect HIV-related outcomes. The propensity score matching approach may be valid, however, for considering the effects of HIV/AIDS on outcomes of interest that are relatively far removed from sexual behaviors and attitudes. An example is Donovan et al.'s (2003) analysis of the effect of AIDS-related adult deaths on Rwandan agricultural households' cropping strategies and incomes.

Finally, in some cases researchers can take advantage of 'natural experiments', whereby there is a change in the policy or natural environment that can be regarded as random or exogenous with respect to the outcome of interest. Major changes in legislation may sometimes qualify as exogenous to behavior, so that a causal effect of the new policy can be inferred. In demography a particularly strong example is given by the legislation passed by the government of the Romanian dictator Ceausescu in 1966 making family planning and abortion illegal (see Pop-Eleches 2006); the policy was clearly imposed from the top and led to sudden and large increases in fertility. A 'natural' natural experiment that has been exploited by researchers to estimate the effects of fertility on child health and other outcomes is the occurrence of a twins births, a (largely) exogenous event that yields an unanticipated increase in parity (Rosenzweig and Wolpin 2000).

In HIV research there do not appear to be examples of the exploitation of natural policy experiments, but the approach may yet find applications. For example, the implementation of a policy of routine or mandatory HIV testing for all individuals entering the health care system will lead to increases in testing that may reasonably be considered as exogenous to individual risk behavior, permitting the analysis of the effects of knowledge of serostatus that is uncontaminated by unobserved differences in preferences or behavior. (Note however that exogeneity this will not be assured if many people exercise their option of refusing the test). With respect to 'natural' natural experiments, one source of (plausibly exogenous) variation in exposure to HIV risk is in the distance to the epicenter of the AIDS epidemic in central Africa (Oster 2006), since the accident of location of individuals (or societies) should not be directly related to levels of risk behavior. As discussed further below, this makes distance a good instrumental variable for local HIV prevalence, incidence, or mortality.

The foregoing review of methodologies and studies for the most part involves evaluations of specific, fairly narrowly focused HIV interventions. However, understanding the reasons for success or failure in HIV prevention will inevitably also involve different kinds of assessments, especially when one is trying to understand the overall success or failure of a country's HIV/AIDS strategy. For example, while one may (and many have) debate which behavior change messages were most effective in Uganda in the early 1990s, the country clearly differed from its neighbors in several broader aspects of policy: the very visible involvement of President Museveni, an aggressive media campaign, and the effective mobilization of community leaders and churches in efforts to destigmatize the disease and promote safer behaviors. It is hardly feasible to set up experiments with all of these elements[26];

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[26] Though community-level experiments can assess some of them, such as modes of local involvement in mobilization efforts. Obviously this cannot be said of factors such as 'national leadership'.

instead, comparative country analysis is called for, with a careful eye for nuances of policy and country idiosyncrasies.

A thoughtful example of this type of analysis is the comparative study of Uganda and Botswana by Allen and Heald (2004). They note that the leadership of Botswana, like that of Uganda, responded to the emerging epidemic with significant levels of resources and public commitment. But the effort in Botswana was singularly unsuccessful in bringing down prevalence. Allen and Heald argue that this is because of two related factors: the inability or unwillingness of the central leadership to fully engage local and church leaders, and the strong emphasis of prevention policy on condoms, which contributed to the alienation of these traditional leaders from the mobilization.

A second example is the analysis of Moore and Hogg (2004) on trends in HIV prevalence in 1990-2000 in areas in Western Kenya and Eastern Uganda near the border separating the two countries. Sentinel surveillance sites in the Uganda study area showed declines in prevalence while no such changes were measured in Kenya. The two areas are geographically proximal and there are no obvious differences in terms of factors such as ethnic groupings or male circumcision rates. The authors thus conclude that the difference is due to Uganda's aggressive prevention policies contrasted with relative inaction of the government in Kenya during the same period.

II.4. Effects of HIV/AIDS on behavior[27]

II.4.1 Changes in risk behavior

Rising HIV prevalence is likely to feed back into behavior. When people see relatives and friends dying of a disease, it is expected that they will adjust their behavior provided they are aware of how the disease is linked to that behavior. Yet for most of Africa this response has been tragically slow (Caldwell 2000). This section considers the evidence for risk behavior change and the effects of these changes in turn on the epidemic. It is important to point out that it is very difficult to distinguish the effects of 'endogenous' responses of the population to evidence of the disease, on the one hand, and responses induced by policies such as education campaigns, on the other. In principle this can be done in cross-country analysis in which both local HIV measures (prevalence, incidence, or mortality) as well as prevention policy variables are included in regressions for behavioral outcomes. The policy variables would need to be appropriately standardized, which would be a challenging task. Less formally, as noted above some research has compared the experiences of countries at similar stages of the epidemic but with well defined differences in policies.

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[27] The focus here is on behavior change in the general population (i.e., not the knowingly HIV infected) in response to changes in actual or perceived HIV risk. The exception to this is the discussion of the effects of knowledge of own HIV status on fertility outcomes (effects of own HIV status on risk behaviors were discussed in section II.3.3.)

A second obvious methodological problem is that, as the term 'feedback' implies, there is simultaneity in the behavior-prevalence relationship. Estimation of the effects on behavior of HIV prevalence (or of incidence or mortality) will be biased because these variables are themselves functions of that behavior. Several authors have attempted to apply instrumental variable techniques to deal with simultaneity. Instruments to predict HIV prevalence or incidence in these studies include male circumcision rates, prevalence of cofactor STIs, and distance to the epicenter of the epidemic in central Africa (Breirova 2002; Werker, Abhuja and Wendell 2006, Oster 2006; Kalemi-Ozcan 2006). Of these candidates, only distance seems to have a strong claim to be a valid instrument for HIV. Both circumcision rates and STI prevalence, in contrast, are likely to correlate with country or cultural characteristics that are also related to the outcome behaviors.

One might also use lagged HIV prevalence as an instrument for current prevalence. If the lagged measure was collected in the first years of the epidemic, before any behavioral responses are likely to have occurred, reverse causality from behavior is not a problem (Kalemi-Ozcan 2006). On the other hand, there is still a potential problem with heterogeneity in terms of country-specific time-invariant factors that are correlated both with lagged HIV prevalence and measured current behaviors. This means that lagged HIV prevalence fails the exclusion requirement for instrumental variables, as it would be correlated with current behaviors via ways other than simply through its effect on current prevalence. Country-level fixed effects analysis—examination of within-country trends in HIV and behavior—has the opposite problem. It eliminates the effects of time-invariant country-level characteristics but is vulnerable to simultaneity bias through changes in behavior affecting the trend in HIV.

Oster (2006) uses distance to predict HIV prevalence in a cross-country regression analysis of risk behavior. She matches prevalence to self-reported behavioral data from DHSs for nine African countries. Estimated behavioral responses are very small. A one percentage point increase in the HIV prevalence rate decreases the share of women with multiple partners by 0.4 percentage points. For men, there seems to be no response at all of this behavior.[28] Oster argues that these results can be explained by the traditional high (non-AIDS related) mortality in Africa. Given the likelihood of dying early anyway from other causes, the cost of risky sex is relatively small: there is (relatively) little expected lifetime consumption and utility to lose. Consistent with this hypothesis, the response to HIV is larger among individuals with higher incomes, which is associated with greater life expectancy.[29] A key and perhaps surprising implication of this analysis is that a simple economic model of behavior appears to explain the variation in risk behavior both within Africa and between Africans and a very different group,

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[28] There is no reason to suppose that the effect of HIV prevalence is linear, as assumed in Oster's (and others') analysis; perhaps behavioral responses only emerge beyond some threshold level of (say) local death rates. Fitting a linear response will then underestimate behavioral response over the right hand portion of the HIV prevalence distribution.

[29] Although couched strictly in the terminology and assumptions of economic behavior, Oster's findings and hypothesis could be said to echo, in part, the cultural analysis of Caldwell (2000) who stresses the importance in Africa of attitudes toward death (including the pre-HIV possibility of early death) in explaining unwillingness to adjust behavior to avoid HIV risk. The anthropologist's notion of 'fatalism' in this case may jibe with the rational recognition of the possibility of early death.

gay men in the U.S. Observed differences between the two groups (that is, the much larger behavior adjustment among gay men in the face of AIDS) largely reflect differences in income and the price of risky sex as just defined.

In contrast to these pessimistic cross-country findings for Africa, in at least a few African countries there is evidence that significant behavior change has taken place. As always, Uganda stands out. As noted in Section II.3.2, comparison of 1995 and 1989 Ugandan Demographic and Health Surveys point to large changes after 1989, especially a decrease in casual or non-regular partners. This period corresponds to the timing of the country's major decline in prevalence and more relevantly, incidence (inferred from trends in infection among younger cohorts).

Uganda is no longer the sole exception. Also as noted earlier, behavior change has recently been documented in several other countries as well, and again, these changes correspond to declines in HIV prevalence or incidence. In Zimbabwe, where a general population cohort was followed from 1998-2003 in Manicaland province, prevalence declined slightly, mirroring antenatal site trends for the country overall showing declines in 2000-2004 (UNAIDS 2005). Sexually experienced men and women reported reductions in casual sex of 49% and 22%, respectively, over this period and younger cohorts reported delayed sexual debut. Data from multiple rounds of DHSs present a similar picture of behavior change for Zimbabwe overall, with a substantial increase in condom use with non-regular partners and an increase in fidelity. Zambia DHS surveys present a more mixed picture, showing declines in several risk behaviors to 1996, especially strong in the capital, but less movement afterwards. The changes in behavior are thought to explain declining incidence in the 90s in Zambia (Fylkesnes et al. 2001). In Kenya, antenatal sentinel surveillance data indicate a decline in national HIV prevalence from 10% in the late 1990s to 7% in 2004. DHS surveys from the same period show increases in age at first sex and condom use and a falling share of adults with multiple partners (Cheluget et al. 2006).

Whether these changes in (self-reported) risk behaviors are sufficiently large to have contributed to the observed declines in HIV prevalence is not immediately clear. To answer this question requires epidemiological modeling. One approach is to simulate the natural course of the epidemic, and see how closely the observed pattern in prevalence over time corresponds to the model predictions. Where there is a divergence such that prevalence is falling faster than predicted, it can be inferred that behavioral changes have also been at work, altering the natural course of the epidemic. Hallet et al. (2006) apply this approach to several African countries (see also UNAIDS 2005). Significantly, the simulations indicate that behavior change in Uganda, Zimbabwe, and urban Kenya contributed to reduction in HIV prevalence beyond that caused by the natural dynamics of the disease—that is, beyond the changes in mortality and incidence that occur as the disease progresses. In each of these countries, significant behavior change was in fact observed. Elsewhere (Côte d'Ivoire, Malawi, Rwanda, and Ethiopia) the model explains patterns in prevalence without behavior change.

To return to the heading of this subsection, is it possible to say to what extent these behavior changes reflect endogenous responses to the epidemic, as opposed to the impacts of

public awareness campaigns and other policies? To be sure, each of the countries just discussed experienced full-scale epidemics, with high levels of premature adult mortality providing populations with powerful evidence of HIV risks. This has been argued by many to have been a major factor behind changes in behavior (UNAIDS 2005; Caldwell 2000). However, the less favorable experiences of very high prevalence countries such as South Africa, Swaziland, and Mozambique make clear that endogenous behavioral responses to AIDS mortality are not the whole story: policy and possibly also cultural differences are important. But policy also varies considerably within the group of countries that have experienced changes in behavior and prevalence/incidence. In Uganda (especially), and Zambia, national governments were aggressive in education efforts against HIV/AIDS (Bessinger et al. 2003); in contrast, government joined the struggle on a large scale relatively late in Zimbabwe and Kenya (where initial emphasis had been on condom promotion, see Cheluget et al. 2006). Thus the precise role of policy in explaining recent changes in behavior (hence also in prevalence declines) is not clear.[30]

Since these cases of significant risk behavior change still appear to be the exception in Africa, they do not necessarily contradict Oster's cross-country findings of little overall behavior response to variation in HIV prevalence. However, they are not 'outliers' in the sense of being cases that are very atypical, hence uninteresting. Quite the contrary is true, as evidenced for example by the enormous efforts made to learn lessons from Uganda's recent history that might be applicable to other countries.

Can the changes in behavior observed in Uganda be explained by an economic model of risk behavior such as presented by Oster and others? As described above, risk behavior in this framework is a function of individual optimization subject to HIV risk knowledge, income, and the price of unprotected sex, the latter an increasing function of perceived transmission risk and income (the latter because greater future consumption and utility is lost for a wealthy person who contracts HIV and dies prematurely). We might surmise that behavior adjustment in Uganda was substantially greater than such a model would predict, given the low levels of income and education prevailing in that country in the early 1990s. Understanding of HIV risk also was probably not very much greater in Uganda then than in other countries where behavior changed far less in response to AIDS epidemics of comparable seriousness, as noted in section II.3.2. This suggests a strong role for public policy in Uganda's transition, as has already been argued. But it also brings out the notion that the role of policy is not just to provide information so that people accurately understand HIV risk—given low average incomes, that might not raise the cost of risky sex enough to significantly change behavior[31]—but to cause people to change their behavior more than they otherwise would given risk knowledge, incomes, and preferences.

One way this may have worked in Uganda and other settings is for government information campaigns to have been so successful in warning about HIV risk that they led

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[30] Of course, the strategy followed, not just the level of resources or commitment, is also likely to matter, as the comparative study of Botswana and Uganda (Allen and Heald 2004) discussed in section III.3.2 demonstrates.
[31] Oster's simulations of improving HIV risk knowledge suggests that even large improvements in knowledge will not lead to major changes in the mean levels of risk behavior or in incidence.

people to *overestimate* the true risk, hence to adjust behavior more than they would have if they had an accurate understanding of the risk. When a disease is characterized by massive negative externalities, as is the case for AIDS, this is by no means a bad outcome for public policy. There is evidence from developed countries that aggressive information campaigns do lead people to overestimate their individual risks of contracting certain diseases: a case in point is smoking and lung cancer in the U.S. (Cutler 2002).

Another possibility is that Uganda's policy of openly and aggressively discussing AIDS in multiple fora not only made individuals more aware of risk, but changed social norms regarding acceptable personal behavior. If changing social norms are internalized by individuals, preferences themselves have been changed (though one might say instead that the new norms raise the cost of certain behavior because individuals are afraid of incurring the social opprobrium of the community). If this is the case, reducing levels of HIV risk behavior involves a transformation more profound than merely altering the prices people face or providing them with information.[32]

Data issues in measuring trends in behaviors

Most of the analyses of trends in risk behavior discussed above rely on multiple rounds of nationally representative surveys such as the DHSs with questions on behaviors such as age at first intercourse, use of condoms at last sex, and number of partners in the past year. Researchers have also examined trends in antenatal site data on self-reported behaviors of clients, and occasionally, longitudinal data covering smaller populations. In principle, the analysis of repeated large, nationally representative surveys such as the DHS is the most appropriate approach to understanding behavior change in the population at large. However, it is important to be aware of potential pitfalls when using such data.[33] There are three primary sources of problems: (1) changes in samples; (2) changes in the questions posed; and (3) changes in how people respond to these questions, or, changes in reporting errors.

The first problem occurs when the samples drawn in two surveys differ in ways that are related to behaviors. For example, means levels of education and wealth may differ beyond what would be due to simple sampling errors. This is not supposed to occur in nationally representative samples—it obviously means that one or both samples are not representative— but can happen if, for example, the sampling frame for the survey changes, say because the national census has been updated and used for the later survey.[34] When the samples are different, differences in mean outcomes between earlier and later surveys may be spurious. With regard to (2), responses to questions about sexual behaviors may be sensitive to how they are asked, where in the survey they appear, and how they are ordered. Finally, as people

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[32] See Epstein and Kim (2007), who argue that changing values has been a key means by which behavior change has occurred in Uganda and elsewhere. They note in particular the role of Ugandan women's groups in promoting the notion that it is reproachful for men to engage in relationships with multiple partners.

[33] For several papers on this topic see the special supplement to *Sexually Transmitted Infections* Volume 80, Supplement 2 (December 2004).

[34] Even when the sampling frame is the same, sampling procedures in the field may lead to changes in sample composition (see Glick, Sahn, and Younger 2006 for an analysis of DHSs from Madagascar).

become more exposed to messages about HIV risk they are increasingly likely to believe that high-risk behaviors are viewed negatively by others. This may make them less inclined to tell interviewers about such behavior, i.e., there may be an increase in 'social desirability bias' from one survey round to the next. This will lead to a spurious trend in the data toward apparently lower levels of risky behavior.

Of these problems, (1) is the easiest to detect and correct. It is straightforward to determine from the data if sample composition has changed, by comparing the sample means of individual or household characteristics in the two data sets. One should pay particular attention to characteristics that should not be changing over time, such as the mean years of education of adults in a given cohort (i.e., born in the same year or several-year interval): if the sampled populations are the same in two surveys, these means should be statistically the same (see Glick and Sahn 2008). To adjust for changes in sample, one can stratify the data on characteristics such as education and location and examine changes in behavior within these categories, as in Zaba et al. (2004). A more flexible approach would be to estimate a regression (or probit, or other model as appropriate) for the behavior including as regressors indicators for survey round and controls for a range of characteristics (education, age, location, wealth, etc.). Provided that this is the only data problem and that the included covariates are adequate to account for the relevant differences in the samples, the coefficient on survey year captures the change in behavior between surveys controlling for between-sample differences in other factors that affect behavior.[35]

Changes in reporting error are more problematic: how can one determine if changes in self-reported behavior represent true changes or merely a shift in how people answer questions about HIV-related behavior? Assuming for the moment that the two samples are in fact representative so the first problem is not an issue, one could examine responses to the one standard question about HIV risk-related behavior that should always be the same across surveys for people in a given birth cohort: the age at which they first had sex, or alternately, if they had sex before marriage. That is to say, as long as the two samples are each representative, mean age at first sex as reported by individuals age (say) 25-34 in a given year should be the same statistically as that reported in a survey conducted five years later by individuals age 30-39 (the age of the cohort five years on). Inconsistencies in within-cohort responses signal a problem in the data; in particular, if mean age at first sex for the same cohort is higher in later surveys, this may be a sign that social desirability bias is increasing—people are becoming less willing to admit to engaging in high risk behaviors, even those occurring in the past.[36] If this is affecting responses to age at first sex questions, it plausibly biases responses

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[35] While this statement is strictly true, it must be recognized that not all of the difference in the control variables between two samples need be spurious, even for surveys spaced only several years apart. For example, average household wealth may have increased in the interval, and this could have led to actual changes in behavior. With these factors held constant, however, the regression estimate of the survey effect (the 'trend') does not capture this source of change. Nevertheless, the estimated effect is still informative, as it shows how behavior has changed over time for individuals with a given set of characteristics, thus capturing policy effects or the effects of the epidemic on behavior controlling for the effects, say, of economic growth.

[36] As Gersovitz (2005) point out, however, the inconsistency could be caused by other factors such as age bias, whereby as the cohort gets older individuals are more willing to be frank about youthful behavior, or alternatively, they become more conservative and hence less willing. Differential mortality of high and low risk individuals

about other risk behaviors as well, in the same direction. Note that changes in sample composition could also lead to differences in within-cohort responses between surveys, so where there is evidence of this, the within-cohort comparisons should stratify on key characteristics or use a regression framework with controls for these and other factors.[37]

Several authors have the examined within-cohort consistency of age at first sex responses on repeated DHS surveys from Africa (Gersovitz 2005; Glick and Sahn 2008; Zaba et al. 2004). Each of these authors reports at least some inconsistencies in responses. For example, Gersovitz (2005), considering DHS surveys from the mid-90s to early '00s for Kenya, Tanzania, Uganda, and Zambia, finds evidence of positive bias in age at first sex for men (reported age at first sex increasing over time for a given cohort) but less of a clear pattern for women. These results point to the need for caution in interpreting behavior trends from survey data; certainly, analysts should perform an investigation of these potential data problems.

II.4.2 Responses of fertility to HIV/AIDS

Fertility may also change in response to the epidemic, either through physiological changes in fecundity or through changes in fertility preferences and behavior. Among women who are infected with HIV, fecundity is reduced for several reasons, including higher rates of miscarriage and stillbirth (Strecker et al., 1993) and high rates of coinfection with other sexually transmitted infections, which may cause secondary infertility (Gray et al., 1998; Martin et al., 1991; Setel, 1995). Illness may also reduce the frequency of intercourse.

With respect strictly to behavior, the desire for children may fall because of women's fears (whether they are knowingly HIV positive or not) of passing on the virus to their children, of not being alive to care for their children, or of their children contracting the disease themselves and dying (Gregson et al. 1997; Setel 1995). Reductions in fertility could also occur as a side effect of prevention behavior, via reductions in unprotected sexual intercourse or increases in age at sexual debut.

On the other hand, other factors could lead to a positive fertility response to the epidemic. By slowing or reversing the trend toward lower child mortality and by increasing the risk that children will die once they become sexually active, the epidemic may serve to increase the desired number of births. For the population as whole (not just the infected) Kalemli-Ozcan (2006) and others argue that in response to uncertainty about child survival, parents will choose to have more children (the 'precautionary demand' motive), ultimately slowing or reversing the demographic transition in Africa. Among HIV positive women, there may be a desire to continue to deliver children in order to appear healthy or avoid stigma. Parents who are

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may also affect the comparison, though Gersovitz argues that this is not a major factor in his analysis of Kenya, Tanzania, Uganda, and Zambia.
[37] With regard to changes in question format, there is not much the researcher can do to assess whether this is leading to problems, though very large changes in responses would be a clue. The designers of DHS surveys, in particular, have generally attempted to keep formats as consistent as possible across years, though in various countries there have been some changes in HIV/AIDS related questions.

infected or fear they may be may try to speed up the pace of childbearing to meet reproductive goals that are socially or culturally important (Temmerman et al. 1994; Gregson 1994).

Turning to the evidence, many African studies, both clinic and cohort based, indicate that total fertility or childbearing odds are lower among HIV positive women (Allen et al., 1993; Batter et al., 1994; Ryder et al., 1991; Carpenter et al. 1997; Sewankambo et al. 1994; Hunter et al. 2003; Zambuko and Mturi 2005). The relative importance of physiological factors, on the one hand, and behavioral responses to being HIV positive, on the other, in explaining these differences are not clear. With respect to fertility intentions, in a number of qualitative studies in Africa, women diagnosed with HIV express their intension of curtailing further childbearing (Setel, 1995; Gregson et al. 1997, Ntozi and Kirunga 1998, Rutenberg et al. 2000) though one stud from Côte d'Ivoire points instead to a desire to continue childbearing despite being HIV positive (Aka-Dago-Akribi et al. 1999).

Several studies with HIV testing and follow-up of women use objective measures of fertility behavior rather than reported intentions: pregnancies, births, or contraceptive use occurring after diagnosis. This research generally finds that fertility and contraceptive behavior does not change after a positive HIV diagnosis (Heyward et al. 1993; Lutalo et al. 2000; Temmerman et al. 1990). It is possible that many women would have wanted to reduce their childbearing but were afraid to reveal their HIV status to their partners. Therefore the relatively few prospective studies looking at actual behavior following an HIV positive diagnosis are inconsistent with studies just noted suggesting that HIV positive women would like to reduce their childbearing. Note that they do not support the opposite hypotheses either, that fertility will increase.

To understand the impact of HIV/AIDS on fertility, it is necessary also to know if and how women who are not knowingly infected adjust their fertility in response to the risks associated with the epidemic. It is worth recalling that the vast majority of women and men in Africa—and of HIV positive women and men—have not been tested, so do not know whether they are infected or not.[38] In a Zimbabwe study (Gregson et al., 1997), very few women said they intended to increase the number of children or accelerate their childbearing in response to the AIDS epidemic, while almost half indicated they would like to have fewer children and the same share indicated they would like to wait longer before their next birth. Consistent with these findings, Noël-Miller (2003) using rural Malawi panel data, finds that women expressing significant worry about HIV risk were less likely than other women to have a birth in the 3-year interval between surveys.

Noël-Miller's study is unusual in that it links fertility behavior to prior subjective HIV risk perceptions. Also rare are studies that try to link fertility behavior to variation in HIV prevalence or incidence. The challenges to doing so are essentially the same as noted above with respect to risk behavior: first, the simultaneity of the HIV-behavior relationship, since

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[38] Without testing, the onset of severe symptoms such as weakness, weight loss, and opportunistic infections will indicate to some that they have AIDS though even then there is likely to be uncertainty (Setel 1995), and of course this typically occurs after some years of being HIV positive. Therefore most people with HIV who have not been tested do not know they are infected.

practices such as early marriage and frequency of unprotected sex that increase fertility also increase exposure to HIV, and second, the need to account for the mediating effects of policies. Two recent cross-country econometric exercises that examine this issue, Young (2005) and Kalemli-Ozcan (2006), reach precisely opposite conclusions. Young's estimates, using DHS data from 27 African countries, indicate a negative effect of HIV national prevalence rates on individual probabilities of a recent birth and on total fertility. Kalemli-Ozcan, using a different but overlapping sample of countries and using aggregate country-level estimates of total fertility rates, finds a positive effect on total fertility of (depending on specification) HIV prevalence or incidence.

Unlike Young, Kalemi Ozcan also attempts to instrument her HIV regressors. Since she finds the effects qualitatively similar to those using OLS as well as country fixed effects, it does not seem as if the different conclusions of these two studies are driven by the treatment of HIV prevalence as exogenous with respect to behavior in Young's regressions.[39] Differences in country samples, in the use of aggregate versus individual level data, or in the specifications used may be driving the disparity. These studies, it should be noted, estimate population level fertility impacts: since the data do not distinguish HIV positive from negative women, the estimates reflect a combination of biological and behavioral impacts, and outcomes for HIV positive women and for HIV negative women. Together these papers provide evidence at least that the issue of the effects of HIV/AIDS on fertility at the population level is far from being sorted out.

Therefore the extent (and even the direction) of feedback effects from HIV/AIDS to reproductive choices as well as risk behaviors remains an important area of future research, with broad implications for the future of the epidemic and, as noted below, for economic growth and poverty. Further, while the effects on fertility of expectations about *own* mortality risk and that of one's children have been considered and debated, research has not considered another factor: the huge number of AIDS orphans that are added to the households of relatives of deceased parents. Given the prevalence of fostering, men and women will anticipate that (if they themselves do not succumb) they may end up taking in the children of deceased siblings or other relatives. Therefore AIDS increases the expected number of surviving children in their households apart from effects via their own fertility responses, and one may expect this to reduce the desired numbers of own children. A key question is whether they consider the children they come to care for to be equivalent to their own, in terms for example of future resources transfers to them from these children. Given evidence (discussed below in Section III.2.2) that fostered-in orphans receive less schooling than children of household heads, perhaps such children are not complete substitutes for own children. There still may be fertility effects, however, and this possibility needs to be considered.

Data requirements to appropriately address the impacts of the epidemic on fertility and sexual risk behaviors are fairly strong. To capture changes in behaviors, researchers need either longitudinal or repeated cross-section data with information on fertility or sexual activity.

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[39] However, the instruments Kalemi-Ozcan uses to predict HIV rates, STI incidence and shares of young women reporting premarital sex, are certainly questionable. For the exclusion restriction to be met it must be assumed, implausibly, that these factors influence fertility only through their effects on HIV rather than directly.

These data, which as discussed above should be subject to reliability checks, must be matched to information on local HIV prevalence, incidence, or mortality. Repeated DHS are a logical source of information on changes in behavior, though a number of studies cited above also use smaller cohort samples. These can be matched to local antenatal surveillance site information on changes in HIV prevalence, or to other sources of HIV data. It is anticipated that the latter will include in the future repeated rounds of DHS or related national surveys with HIV testing modules.[40]

Even with such data, of course, the researcher has to confront the simultaneity problem: are observed behavior changes the result or cause of changes in HIV prevalence? Several possibilities for instruments were noted above, though the validity of some of them is questionable. It is likely to be easier to predict the level of prevalence, as in these studies, than changes in prevalence over time. One is then measuring response to changes in HIV using spatial rather than intertemporal variation. In principle this can be done at the subnational, not just country, level, provided there is adequate variation in HIV data and instruments.[41]

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[40] Comprehensive longitudinal data collected in single areas (e.g., a district), such as those collected in Rakai and Masaka, have many advantages, but a disadvantage is that there may be little meaningful spatial differences in HIV prevalence/incidence or in changes in prevalence/incidence. If so, analysis must rely solely on intertemporal variation to discern relationships between behavior and local HIV risk.

[41] This may be the case for the 'distance to the epicenter' instrument. Note that variables such as distances to towns or to transportation networks, while likely correlated with local HIV rates, are not valid instruments. These factors also likely affect levels of risk behavior directly by altering the nature of sexual networks.

III. LINKAGES WITH POVERTY

III.1 Pathways from Poverty to HIV/AIDS

Next I turn to the poverty side of Figure 1: the effect of poverty on HIV/AIDS operating through reproductive health, behaviors, and the use of services, as well as through other pathways. I follow this with a discussion of the reverse pathway, that is, the implications of the AIDS epidemic for poverty.

First, what is the overall association of HIV/AIDS and poverty? At a global level, the association is positive. The countries most burdened by the disease—most of them in Africa— are among the poorest. This is assumed to reflect the association of a country's level of income with the quality of its health infrastructure and HIV awareness. At the micro level poverty potentially increases vulnerability to HIV via compromised reproductive health or reduced HIV/AIDS awareness (links highlighted in the figure). The poor are less likely than the wealthy to know about prevention behaviors and less likely to have had an HIV test. Glick and Sahn (2007) examine these associations using recent DHSs from nine African countries. Echoing earlier studies, they find that the effects of household wealth on both prevention knowledge and testing, estimated from probit models, tend to be positive, if not always statistically significant. The poor also have less access to, or ability to pay for, condoms, and may also be more likely to have untreated cofactor STIs that increase susceptibility to HIV infection. Another potential pathway from poverty to HIV is via overall poorer nutrition and health status. It has been hypothesized that these factors compromise immune defenses or increase receptivity (via weakness in epithelial cells) to HIV infection (See Stillwagon 2002).

Yet in spite of these factors, when one looks either within or across countries in Africa, HIV infection tends to be associated with greater wealth or income, not less. HIV prevalence is highest in the relatively wealthy countries of Southern Africa (e.g., Botswana, South Africa). Since there are many potential confounding factors at the national level, micro evidence is more compelling. A new and valuable (because nationally representative) source of information are several DHS surveys and AIDS Indicator Surveys (AIS) which gather HIV serodata linked to information on individual characteristics.[42] These population (as opposed to clinic or antenatal site) HIV surveys have been made possible by the development of simpler testing technologies. Mishra (2006) reports on an analysis of eight such surveys (in Burkina Faso, Ghana, Cameroon, Uganda, Kenya, Tanzania, Malawi, and Lesotho). His findings as well as some earlier micro level studies (See Wojcicki 2005) indicate generally positive associations of HIV infection and wealth, the latter measured by the level of physical assets.

Why is a positive association of wealth and HIV observed? For one thing, the better off tend to have more concurrent sexual partners. Since such relationships (whether casual or not)

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[42] Assuring representivity in these surveys remains a concern due to selective non-response in testing, which is shows substantial variation across countries (see García-Calleja et al. 2006). Even given these issues, these new population-based data have led to a significant downward revision in prevalence estimates in most countries compared with prior estimates obtained from antenatal surveillance sites, confirming long held concerns that the latter were not representative of the population at large.

have a financial cost, this is not surprising: in economic terms, having multiple partners is a 'normal' good. Glick and Sahn (2007) estimate ordered probits for number of partners for women and men in a number of African countries using DHS data. Among men, the estimated effects of wealth (controlling for education and several other factors) are not always significant, but where they are, they are usually positively signed. The same goes for the effects of level of education. For women the impacts of both wealth and education are more ambiguous, though the likely underreporting of the number of partners for women (See de Walque 2006) makes these estimates harder to interpret.

The wealthy also tend to be more geographically mobile than the poor—for example, the better off within rural populations are more likely to travel regularly to urban areas—and such mobility is associated with the holding of multiple concurrent sexual partnerships. Less causally, HIV prevalence is higher in urban areas, which are also wealthier. Finally, the wealthy with HIV tend to survive longer than the poor. Their better nutrition keeps their immune systems stronger, and their superior access to health care also helps them deal with opportunistic infections. Hence they are more likely than poor individuals who are infected to show up in surveys and be recorded as HIV positive. This implies that the survey data may be overstating the actual association of wealth and HIV infection. On the other hand, to the extent that being HIV positive leads to reductions in work and income, the association will be underestimated.

In the analysis reported by Mishra, alternate logistic models of HIV odds were run with controls for factors such as occupation, rural/urban residence and self-reported risk behaviors. The positive association between wealth status and HIV becomes statistically insignificant in most cases when these potential confounders and mediating factors are controlled, though even then, wealthier adults in most of the country samples are no less likely than poorer adults to be HIV-infected. The reduction or elimination of the wealth effect is not surprising; as the previous discussion implies, we would not expect there to be much difference in HIV probabilities once we control for the factors, including sexual behaviors, that differentiate the wealthy and the poor.

The foregoing suggests several key research questions on the relationship of poverty and HIV/AIDS. First and most broadly, is there at some level a conflict between the goals of economic development and poverty reduction, on the one hand, and HIV prevention, on the other? The dilemma is that the process of growth and concomitant poverty reduction is typically associated with a range of factors—increased geographic mobility, income, and urbanization—that increase HIV susceptibility. Geographical mobility, for example, is not merely associated with, or an outcome of, wealth: it is often precisely the means by which individuals and families escape poverty, by moving for example to cities where economic opportunities are more plentiful. The evidence thus should cause us to doubt the popular view that 'the best way to reduce AIDS to is to reduce poverty' (see Fenton 2004 and Shelton et al. 2005 for discussion).

Related to this, should prevention efforts be targeted more carefully at somewhat better off groups in the population? To what extent do the associations of wealth and seropositivity

reflect direct 'wealth effects' on the demand for partners as opposed to structural confounders such as urbanization and access to transportation networks?

In terms of dynamics, how does the wealth-HIV (or poverty-HIV) relation evolve over time as epidemics mature? It is noteworthy that even in Uganda, with possibly the oldest epidemic and the highest level of HIV awareness, one still observes a positive association of wealth and HIV infection. This does not mean, however, that this relationship is static. There are indications, including for Uganda, that the association of wealth and HIV weakens over time. This may be because of the natural spread of the epidemic beyond the initial (wealthier) affected population, or because the wealthier are in a better position to access or understand prevention messages and to act upon them, e.g., by being able to purchase condoms. The advantage of the better-off with respect to accessing and understanding information about health risk would be derived in part from their superior education. In line with this hypothesis, a micro level analysis in Masaka, Uganda by de Walque (2004) indicates that while there was no significant association of education and HIV seropositivity in 1990, by 2000 the association was negative and significant. For Zambia, a similar pattern is reported by Michelo et al. (2006). These findings suggest that the educated (and by extension, the better-off) are the first to receive prevention messages or to understand them, or both.

To address this dynamic issue requires panel or cohort data (repeated observations on a group of individuals) or else repeated cross-section data such as those used by de Walque from Masaka. The data must link individual characteristics such as education and assets to information on HIV status. Panels in particular could also address the simultaneity problems inherent in the wealth-HIV association mentioned above. But even without panel data, thoughtful statistical analysis using cross-section surveys with linked HIV data will be able to clarify the links of poverty and HIV. This will be the case especially if these survey data can be further matched to data on local environmental factors such as access to transportation routes, population density, migration, and industry. This would help illuminate the extent to which the association of wealth and HIV infection is causal in the sense of being due to behavior that is a direct function of wealth (e.g., an increased demand for partners), or instead is largely a reflection of the correlation of wealth with structural economic factors such as residence and population density (which affect, among other things, the density of sexual networks and the supply of sexual partners).

The opportunities for such analysis should increase in the coming years as second round DHS/AIS cross-section data with HIV testing become available for many countries. This will permit analysis of changes over time, if not of the individual level dynamics captured in panels.

III.2. Pathways from HIV/AIDS to poverty

III.2.1 Macro-level perspectives

The implications of the AIDS epidemic for African economic growth, and by extension, poverty, are not clear, especially with respect to the long term. There are certainly many mechanisms through which the epidemic may have deleterious macroeconomic effects. Striking adults in the prime of life, AIDS depletes the ranks of workers, and especially (given the evidence on the association of income and HIV) relatively educated or skilled workers. There are many accounts of this process occurring in specific industries, as well as in the education sectors of hard hit African countries. The latter implies a reduction in the supply or quality of schooling. On the demand side in the education sector, private investments in children's schooling by households hit with AIDS illness or death are expected to fall (discussed in the next section). These demand and supply side effects in education imply negative intergenerational trends in human capital, reducing future growth. Faced with escalating turnover and training costs as well as surging health care expenditures for sick employees, both local and foreign firms may reduce investment or pull up stakes altogether. Public sector budgets will be strained by the need to increase spending on care for those with AIDS-related illnesses, reducing allocations for development-enhancing expenditures in health and other areas.

Despite these considerations, cross-country regression analysis on data through the mid or late 1990s (e.g., Bloom and Mahal 1997; Dixon et al. 2001) found either no statistical effects of HIV prevalence on (per capita) economic growth or ambiguous effects. However, future impacts may be very different. Here, predictions based on simulation modeling diverge considerably. Initial studies yielded what might be considered to be relatively 'optimistic' predictions, on the order of perhaps a 1% annual reduction in growth (summarized in Bell et al. 2003)—far from trivial in the context of Africa's already typically very low growth rates, but not devastating. Further, such 'first generation' models tend to show little decrease, or even an increase, in *per capita* GDP growth, because HIV-related declines in GDP are offset by increased mortality and population decline. Moreover, in a traditional Solow-type growth framework, reductions in growth are dampened by the fall in the supply of labor relative to capital, since this increases the productivity of labor. A recent similarly optimistic set of projections has been put forth by Allywn Young (2005), who as noted earlier emphasizes a different mechanism, that of reduced fertility in response to the epidemic. The reduction in the dependency ratio increases per capita consumption as well as savings, potentially increasing economic growth as well as providing the resources to care for those with AIDS.

Others are far less sanguine, however. The important work by Bell et al. (2003, 2004), for example, yields a much grimmer picture. Their model emphasizes a different behavioral response: a large reduction in investment in the human capital (schooling) of children as a result of AIDS-related illness and mortality, which occurs both because households are poorer and because the returns to such investments have fallen. The cumulative result of reductions in human capital investments is a large decline in GDP.

Other pessimistic projections, including many less formal assessments, often emphasize less tangible implications of the epidemic. These include effects on children's socialization and the fraying of social safety nets; the disruption of firm and government operations through high turnover and the thinning of workforces; consequent reductions in profitability and in the incentives of firms and households to invest and to save; and more generally, the deterioration in the functioning of a range of public and private institutions. Especially in Southern Africa, there is evidence that some of these effects are already occurring. At least, personnel losses of magnitudes that are potentially devastating have taken place, as documented by Husain and Badcock-Waters (2002).

In his review of a recent collection of papers on the subject (Haaker 2004), Arndt (2006) notes several important gaps in the topics addressed that could equally apply to the literature as a whole. One is the lack of attention to how macroeconomic impacts may vary depending on the level of development and economic structure—for example, whether the agricultural economy is land or labor constrained, and whether the economic infrastructure and supply of key services such as education are well developed or not. A second is a lack of modeling directed at understanding the implications of expanding ARV provision. While there is little direct evidence on how long the drugs will prolong life in African contexts, evidence from a (less) poor, non-African country, Brazil, suggests a median time of perhaps four years (Marins et al. 2003).[43] Even this relatively limited delay may have significant implications for family incomes, children's schooling and health, and hence national income. On the cost side, implementation of scaled up ARV programs will also imposes significant burdens on governments, even if the drugs themselves are financed by donors. An exception to this gap in the literature is Young (2005), whose macro modeling does incorporate the effects of ARV provision on life expectancy.

However, the main problem with efforts to model the macroeconomic implications of AIDS in Africa is that projections hinge crucially on assumptions made about micro-level behavioral responses of individuals as well as firms, with respect for example to fertility, schooling investments, and sexual risk behavior. This is brought out clearly by the comparisons of the widely divergent outcomes of several of the analyses. This is often not merely a matter of whether different parameters values are imposed for specific responses, but instead whether these channels are modeled at all. As Bell et al. (2003) note, earlier efforts based on a simple Solow-type framework emphasize the aggregate growth of labor and physical capital; such models ignore responses of human capital investment and hence the reduction in the transmission of human capital across generations. Most models do not incorporate changes in fertility and in investments in physical capital by households. Excluding specific channels or responses, of course, is equivalent to assuming zero response. Ultimately learning more about these behaviors is essential, which takes the focus back to the micro level.

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[43] This is the estimated survival time in on 'first-line' drug combinations, which eventually stop working. In developed countries individuals are expected to live longer than this on first-line therapies, and then are able to switch to second-line or even third-line approaches. These are much more expensive and unlike the first-line drugs, generics have not been developed for them. Hence it seems a long way off before they will be available to the poor in Africa.

III.2.2 Micro/household level perspectives

        Many studies, both qualitative and quantitative, have investigated the effects of AIDS on poverty and other outcomes at the household level. The main areas of focus for this research have been the impacts of an AIDS illness or death on household income and consumption, on household structure (or dissolution), and on children, in particular, children's schooling.

Effects on household consumption, production, and demographic structure

Methodological concerns

        Pressure on the consumption levels of AIDS-afflicted households may come through reductions in labor supply, incomes, and farm production as well as through the burdens of health care expenditures for the ill and funeral costs for the deceased. Most of the studies examining these impacts have used panel data (data on the same households collected at different points in time).[44] However, several rely on retrospective questions about mortality in cross-sectional data and use them to make simple comparisons of afflicted and non-afflicted households. In at least one case only AIDS-afflicted households are identified for interview.

        Panel data are far superior for examining this issue. Some studies have made use of such data to condition the estimates of mortality impacts on baseline characteristics of the households, in particular, income or assets. As noted above, AIDS mortality in Africa appears to be selective, on wealth in particular. Including wealth and other baseline characteristics in regressions of household outcomes on mortality allows the estimation to control for differences in these factors between households experiencing deaths and other households. However, households may also be heterogeneous in terms of unmeasured factors that affect both adult mortality and consumption or other outcomes of the household. For example, it may be that, conditioning on wealth and education, adults who are more likely to become infected with HIV (because they lack health and prevention knowledge, because the male spouse visits sex workers, or because they are less concerned about the future) are also less likely to allocate resources toward consumption or children's health and schooling. AIDS illness and mortality are then correlated with outcomes of interest even after we control for observable characteristics, so their inclusion in the regression is not sufficient to eliminate biases.

        Therefore many analysts using panel data employ differencing or fixed effects methods. Here, one regresses *changes* across periods in household consumption (or some other outcome) against *changes* in the right hand side variables; for mortality, the change over time is simply an indicator of whether someone has died in the interval. With this approach the influence on the outcome of any unobservable factor that might be correlated with mortality subtracts out. This is, however, subject to the assumption that the unobservables affect the *level* of the

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[44] Studies for Africa of the effects of AIDS illness or death on household incomes and expenditures (using varying methodologies) include Bachmann and Booysen (2003, 2004), Bechu (1998), Bollinger et al. (1999), Gregson et al. (2006b), Menon et al. (1998), Ngalula et al. (2002), Over et al. (1996), and Steinberg et al. (2002).

outcome, but not changes in the outcome over time; the household fixed effect, in other words, must be time-invariant.

This assumption may not always be valid. Some of the more likely violations have to do with investments in children's human capital, the subject of the next subsection, but the concern is also relevant to the outcomes considered here. It is possible that households that experienced an AIDS death would have experienced different growth paths for assets or consumption than other households in any case, or would have undergone different changes in family structure. These differences will likely not be very important when considering short term mortality impacts, but will be a source of bias, if present, for fixed effects estimates of longer term impacts.

One other point about panel data is that the appropriate baseline information pertains to the period, not immediately prior to death, but prior to when the individual become ill with AIDS. One requires an adequate interval in the panel to capture this change. There is also a standard problem of attrition in panel data (see Alderman et al. 2001). Households and individuals are mobile so many may be lost to follow-up. Given the possibility that AIDS deaths leads household to migrate or dissolve, selection on mortality may seriously bias many of the estimates in the literature that do not have information on those who leave the sample. For analysis of the effects on children the concern is especially important given the possibility that a child is sent away to live in other households following the death of a parent. I return to these problems below.

A very different methodological challenge is distinguishing, in the absence of clinical information, deaths due to AIDS from deaths due to other illnesses. The simplest and most common approach is not to try: one can simply compare households experiencing an adult death from any cause with households not experiencing a death. This is clearly not the ideal method but it requires the least information and can be used with many standard surveys. Further, overall adult morality rates from surveys appear to correlate well with regional AIDS prevalence (Mather et al. 2004a), and where AIDS mortality is very high, it will likely account for most prime age adult deaths. Still, the effects of AIDS illness (which is of long duration) and death are likely to be more severe than for other sources of mortality, raising the possibility that analysis that aggregates the two will underestimate the impacts of AIDS deaths specifically. Therefore some researchers use proxies such as duration of illness to assign AIDS deaths. Many others opt for a more thorough (and skill-intensive) approach known as the 'verbal autopsies' method (see Kamali et al. 1996; Garenne and Fauveau 2006) whereby interviews with family members or caregivers about the illness and symptoms of the deceased are used to ascertain the cause of death.

Finally, a problem with empirical analysis of the effects of prime age adult mortality is the relative rarity of these events. In areas of very high HIV prevalence, mortality will also be high, but in other setting the small number of recent deaths recorded in the data may mitigate against finding statistically significant effects. This needs to be kept in mind when reviewing this literature. Considerations of statistical power therefore point to a significant advantage of large (and expensive) surveys over smaller more focused ones, unless the sampling procedure

was such that households experiencing a death are oversampled. A rare example of this is the Kagera Health and Development Survey (KHDS) in Tanzania.

Evidence on the effects of mortality on households

Most studies of the effects of AIDS mortality find that overall household income or expenditures fall in afflicted households while at the same time the composition of spending shifts sharply toward medical care and funerals. The latter burdens are typically very significant. Among Tanzanian households suffering an AIDS illness and death, the sum of medical and funeral costs on average exceeded the estimated annual household income per capita in the population (Ngalula et al. 2002). This burden is not atypically high. A study of South African households, for example, found annual AIDS-related medical costs and funeral expenses each comprising about a third of household annual income (Steinberg et al. 2002). The burden of health care expenditures from AIDS tends to be larger than for other terminal illness or injury because, as just noted, of the duration of illness is long (Menon et al. 1998; Ngalula et al. 2002). Some studies document reductions in household physical assets as well as incomes (Menon et al. 1998; Mushati et al., 2003).

While these studies document significant burdens associated with adult illness and death, the effects on per capita household income and consumption from the loss of the adult, especially in the long term, are not as clear. In an analysis of the Tanzania KHDS (World Bank (1997) it was found that households with a prime-aged adult death in the last year had 7.5% lower expenditures per adult equivalent durables) than households without an adult death. This is perhaps a surprisingly modest reduction in income (though consumption would have fallen by more since expenditures had to be allocated to funerals or medical care). It should be noted that the comparison does not control for initial household income, which may have been higher in AIDs afflicted households given the associations noted above of wealth and HIV infection.

Beegle at. al. (2008) are able to take advantage of subsequent waves of the KHDS panel to control for differences among households in adult mortality. They consider the effect of a prime-age adult death on subsequent growth in household per capita expenditures using data covering the period 1991 to 2004. Their specification is differenced across time, i.e., they use household fixed effects to control for fixed unobservables affecting mortality over the period. The estimates indicate that a death leads to 7% lower consumption growth during the first five years after the death, with recovery after that though not to pre-death levels.

As Strauss and Thomas (2008) point out with reference to this study, a 7% decline in consumption is not trivial, but is small in comparison with the household impacts of some macroeconomic shocks in developing countries. They note as well that such relatively modest impacts at the micro-level seem to belie the claims of many that Africa is in danger of massive economic failure due to AIDS. To some extent, however, these claims relay on general equilibrium or institutional effects that are not captured in micro data, or perhaps, that are not yet realized. For example, extreme negative consumption impacts on surviving family members may be avoided through transfers from the extended family or community. But if

AIDS mortality reaches a certain threshold in the community, this support network may eventually fail. There is little evidence on these issues, but what research has been done is discussed briefly below.

Perhaps the firmest conclusion one can make from the research conducted to date is that there is considerable heterogeneity in the severity of the effects of an adult death. Among other things, the position in the household of the deceased will strongly condition the welfare consequences. Mather et al. (2004a), summarizing five East and Southern African country studies, note that it is not always the case that the individual who dies is either the household head, the primary breadwinner, or the wife of the household head. Most females who died were not wives of household heads (or household heads themselves), and even for males the patterns were mixed. Where the deceased did not have one of these roles, the income impacts are relatively small. In other cases, such as poorer families headed by HIV widows, the negative consequences may be quite severe. In contrast to these studies, Beegle et al. (2008) do not find a relationship between a household's initial level of welfare and the consumption impacts from an adult death.

Along with consumption patterns, household production patterns may change as a result of an AIDS illness or death. It was initially widely assumed that among agricultural households, labor shortages from AIDS mortality would lead to a shift to less labor-intensive crops (Topouzis and du Guerny 1999). However, empirical research, again primarily using longitudinal data, indicates that responses vary widely, depending on the agricultural or regional context, the initial level of household wealth, and who in the household has died (See Gillespie and Kadyala 2005 and Mather et al. 2004a for a discussion of the literature).[45] Barnett et al. (1995) in Uganda evidence present evidence of households shifting farming activity away from high value cash crops toward crops for own consumption. For rural Kenya, Yamano and Jayne (2004), using household fixed effects, find no change in area devoted to food crops after an adult death but a large reduction in area allocated to cash crops such as coffee and tea if the death is of a male head of household.

In other settings, however, changes in cropping patterns are less evident. Beegle (2003) found that there were few changes in the labor supply of surviving individuals in rural Tanzanian households a year after a prime-age adult death. Nor did these households shift cultivation towards subsistence food farming or reduce their diversification. Similarly, for Rwanda, Donovan et al. (2003) find that death-affected households have few significant differences in crop production patterns from similar (based on propensity score matching) non-affected households, though the amount produced tends to be smaller in afflicted households.

This literature also indicates that production responses are tightly wound up with changes in household structure resulting from an AIDS death. This is not surprising, since such changes will determine what happens to total household labor supply and its composition. Findings are varied here as for other responses. In the Kenya study of Yamano and Jayne mentioned above, deceased adult members were not replaced: households and total labor

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[45] The Food Security Group at Michigan State University maintains a website with links to many research papers on this topic (http://www.aec.msu.edu/fs2/adult_death/index.htm).

supply got smaller. The same was found in rural Mozambique (Mather et al. 2004b). In contrast, Rwandan households were able to maintain their labor supply through the addition of new members via marriage or through the addition of young relatives (Donovan et al. 2003).

The most dramatic—and clearly, not necessarily voluntary or desirable —change in household structure is the dissolution of the household unit itself, as survivors move in with relatives or (in fewer cases) become destitute. Understanding this process, and in particular, the effects on the welfare of persons in households that dissolve, raises challenges for empirical researchers. In cross-section household survey data, one obviously does not observe such households; one only observes existing units that reflect post-death reallocations of family members among other households. Indeed, this is a potential problem for much of the analysis reviewed in this section—and longitudinal studies face a similar problem. Panel studies of the effects of an AIDS death on surviving family members typically measure these outcomes only for individuals in families that remain intact and in the panel. It may well be that individuals from dispersed households are worse off.

To learn about household dissolution when only cross-section data are collected, some studies have used retrospective interviews with individuals about their recent histories. This will provide information on, for example, the share of persons who have experienced AIDS deaths in their families and subsequently suffered the breakup of their households. It will not provide information on associated changes in consumption and nutrition, though as discussed in the next section, one could fairly easily gather information on children's previous and current school status. With longitudinal data, changes in household composition and existence can be traced from the baseline roster information on households. For AIDS or mortality-affected households that 'disappear' by the time of follow-up, it will not be evident whether they dissolved or moved away, but typically, at least in rural areas, this information can be obtained from neighbors or community leaders. However, it will be difficult or impossible to learn about changes in consumption, health and schooling experienced by the individuals in such households.

Turning to the evidence on the sources of household dissolution, a large scale longitudinal study of rural South Africa (Hosegood et al. 2004) found that households in which an adult had died from AIDS (5% of the sample during the one-year study period) were three times more likely to dissolve than those where no deaths had occurred. Households that were initially poor were more likely to dissolve following a death. In another large panel survey, in eastern Zimbabwe (Mushati et al. 2003), 10% of households dissolved between 2000 and 2003 and 24% had moved; again the probability of dissolution was higher for households experiencing an adult death. The threat of dissolution (and the likelihood of migration) is often larger for women whose spouses die rather than for men whose wives die, both because the loss of income may be greater and because of legal or traditional barriers to women's right to inherit land and other assets (Urassa et al. 2001; Shah et al. 2001). In some studies household dissolution following an adult death was relatively uncommon. In panel data from Kenya, Malawi, and Rwanda relatively few such households were observed to break up, reflecting the fact that, as noted above, in these contexts adult deaths from AIDS did not usually involve the main family breadwinner (Mather et al. 2004b).

Effects on children and investments in human capital

A great deal of attention has been given to impacts of AIDS illness and mortality on children, both because of direct concern about their welfare and because AIDS-related reductions in child schooling and health investments have negative implications for future economic growth. It is estimated that about 9 percent of children under 15 in Africa have lost one or both parents to AIDS, and that one out of every six households with children in the region is caring for at least one orphan (UNAIDS 2006). 'Double orphans'—children who have lost both parents—are almost always taken in by their extended families (Monasch and Boerma 2004). By and large it appears that this traditional safety net mechanism has performed very well in the AIDS epidemic, holding at bay the outright destitution of countless children. This does not mean, however, that orphaned children or children in AIDS-afflicted households do not suffer serious negative consequences.

Methodological concerns

Researchers attempting to understand these impacts face methodological challenges similar to those described above for other micro-level outcomes. Much of the initial work on this topic used cross-section data and compared the nutritional status and (more often) school enrollment of orphans to that of non-orphans[46](Lloyd and Blanc 1996; Ainsworth and Filmer, 2002; Bicego et al., 2003). There are several issues to be aware of regarding such analyses. The counterfactual for understanding the effects of AIDS mortality is the outcomes that would be faced by orphaned children in the absence of their parent(s)' illness and death. Most analysis using cross-section data compare the mean nutrition, schooling, or per capita household consumption of orphans and non-orphans, that is, the outcomes for latter are used as the counterfactual.

With a nationally representative survey, this comparison would accurately portray differences in current outcomes for the two groups, information that may be valuable from a targeting perspective.[47] But it does not indicate the *effect* of orphanhood—say, the loss in welfare measured by nutritional status or household per capita consumption—unless being orphaned is random with respect to other factors contributing to welfare. This is a very unappealing assumption; for one thing, as discussed earlier, in Africa AIDS mortality is generally associated with greater wealth.[48]

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[46] "Orphans" rather than "AIDS orphans" since the cause of parental death is usually not known.

[47] Though as Case et al. (2004) note, one should compare children of comparable ages (and possibly gender as well) to obtain meaningful estimates.

[48] Note that adding controls for household characteristics in a regression framework does not overcome the problem of non-randomness of orphan status and leads to results that, unlike the purely descriptive comparisons of mean outcomes, are not interpretable. Consider the simple regression $H_i = \alpha_1 O_i + \alpha_2 X_i + \lambda_i + e_i$ where H_i is a human capital (health or schooling) indicator for child i, O_i is an indicator of orphan status, and X_i is a control for current (receiving) household income. $\{\lambda_i + e_i\}$ is a compound error term composed of unobserved (pre-parental illness) determinants of child human capital such as cumulative nutrition or prior school investments (represented by λ_i) and a random disturbance term (e_i). For the coefficient on O_i to represent the causal effect of orphanhood, orphan status would have to be uncorrelated with the error term, hence with all elements of λ_i. Note that one of

Several authors using cross-section data therefore difference within households so as to control for unobservables at the level of the household (Case et al. 2004; Yamano et al. 2006). These household fixed effects[49] estimates compare outcomes such as health and schooling of orphans and non-orphans in the same household. Yet even if non-orphans fare better than orphans on these measures, the difference does not indicate the change in outcomes for latter due to the loss of the parents. This problem can be seen most clearly for children who have been fostered into new households after their parents or parent has died. The characteristics of originating households prior to the death of the parent(s) may differ systematically from those of receiving households in ways that affect human capital outcomes. There is some evidence, for example, that orphaned children tend to be sent to live with relatively affluent relatives who can most easily bear the added burden; Ksoll (2006) shows this to be the case for Tanzania. Children in those families would then tend in any case to have more human capital than the incoming children, so do not form the appropriate comparison group.[50] The assumption that they do underlies the household fixed effects approach, which therefore may yield misleading findings with respect to the impacts of orphanhood.

On the other hand, the within-household comparisons may indicate whether or not orphans are discriminated against relative to other children in the household, who will be more closely related to the household head (Case et al. 2004). If 'discrimination' is taken strictly to mean caregivers' preferences for their own or more closely related children over orphans, this too requires certain assumptions. There may be lagged effects on children's human capital as a result of deprivation or emotional scarring from the illness and death of the parent(s), so that orphans have lower health status or cognitive ability to start with. If this reduces the returns to schooling investments, caregivers may provide less education to orphans than to more closely related children even if they would not otherwise tend to favor the latter.

Panel data avoid the central limitation of cross section analysis: they permit observation of actual changes in individual children's health, schooling, or other outcomes after

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these elements is originating household income, which is not observed. As discussed in the text, these assumptions are not plausible. Even without these problems, observed current household income X_i may be affected by the presence of an orphan (especially if resources are represented by per capita consumption); the effect of orphanhood as measured by α_1 would be net of this effect hence would not capture the total impact.

[49] Despite this use of this term, note that this within household estimator is different from 'household level fixed effects' described in the last section, whereby one differences household level outcomes and determinants cross time.

[50] The foregoing considers the situation of orphaned children being taken into new households. In other cases orphans remain in the same household but are deprived of the care and resources of their parent(s). The problem for the within household estimators noted in the text—that the counterfactual of the orphan's pre-mortality welfare is not represented by the current welfare of non-orphans in the same household—still applies under a range of assumptions about intrahousehold distribution. First, if resources are not completely pooled among household members (in particular, if parents and their children form semi-autonomous units within the household), orphans and non-orphans in the same household would likely have differed even in the absence of parental death. If these differences are systematic due to differential AIDS mortality among adults, the non-orphans are not appropriate as a control group. Second, to the extent that resources are at least partially pooled among household members, the welfare of the non-orphan children will be affected by the death of any adult income earner or care provider in the household, so current measures for this group again are not representative of pre-death conditions, for either orphans or non-orphans.

a parent or other adult becomes ill and dies. Baseline data permit the researcher to control for wealth and other initial characteristics of the orphan's household, something that is not possible in cross-section data on orphans. Alternatively, as with the panel analysis of the impacts of mortality on consumption, fixed effects—in this case, child-level fixed effects—can be used to eliminate the impacts of observed as well as unobserved factors that influence both adult mortality and children's outcomes. Yamano and Jayne (2005), Evans and Miguel (2004), and Case and Ardington (2005) take this approach, which in essence compares changes in outcomes for children who are orphaned with changes for other children to yield a difference-in-difference estimate of orphanhood effects. If the panel is long enough, it is possible to model the effects both of parental illness and subsequent parental death, both relative to the appropriate baseline of the pre-illness period. Cross-section comparisons of orphans and non-orphans, whether descriptive or using household fixed effects techniques, only show (imperfectly) the impact of parental death.

Researchers using panel study designs do face the problem of the loss of information at follow-up for orphans who are fostered out or whose households dissolve or migrate. It will usually be impractical to locate all such children, though for children fostered out of intact households one may still learn from remaining household members about their current schooling, if not their nutritional status or receiving household income. This is the case for the study by Yamano and Jayne (2004), whose panel of rural Kenyan households collects information on schooling of children who have left the household, though information is lost on children in households that move or dissolve (these events, however, are relatively rare). Beegle et al. (2005) report on a follow-up to the KHDS survey, in Kagera, Tanzania, that ambitiously attempts to trace children initially surveyed some 10 to 13 years earlier, even if the children had left their baseline villages. Failing this rather substantial effort, one can attempt to bound the effects on the estimates caused by attrition due to household dissolution, migration, or other factors (Manski 1995; Evans and Miguel, 2004, apply this idea). Of course, if attrition is significant the range in the estimates will be correspondingly large.

Another problem, noted in the last subsection, is that the assumption of time-invariant fixed effects may not be valid. Differencing eliminates bias from unobservables that affect both mortality and the levels of child outcomes such as schooling and health. It is only valid if changes in child outcomes—for example, changes in school enrollment status—are not similarly correlated with unobservable factors that affect HIV risk and mortality. Yet it is not hard to imagine how this correlation can occur. Suppose, for example, that conditional on observed characteristics, parents whose behaviors put them at higher risk of HIV/AIDS (because of lack of health knowledge or concern about the future) also tend to invest fewer years in their children's schooling. Then even in the absence of a death, changes in the schooling status of their children would be different from that of children in other households: they will be more likely to have left school. Rather than differencing away, these factors influence the change in the outcome, and the negative effect of mortality on schooling will be (in this example) overstated.

The more general problem is that both schooling and health status are realistically characterized as outcomes of cumulative processes. Current school enrollment (or academic

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performance) depends in part on prior school status and performance; the latter, for example, will determine whether a child is promoted and perhaps also the willingness of the parents or child to continue. Similarly, certain measures of child health, such as height-for-age, capture the cumulative impacts of prior nutrition and heath stresses. This means that unmeasured factors affecting initial levels of these outcomes will also affect how they evolve over time. If these factors are associated with mortality, differencing will not eliminate the bias in the estimates of mortality effects.[51] As with the estimation of the effects on household income and structure, this will be less of a concern when considering short-term mortality impacts than when estimating longer-term impacts.

Evidence of the effects of parental illness and mortality on children

Turning to the results from this literature, cross-section analyses for Africa (often using DHS data) generally find lower school enrollments and poorer nutritional status among orphans than among non-orphans (Monasch and Boerma, 2004; Case et al. 2004). Based on their household fixed effects estimates, Case et al. conclude that lower orphan schooling does not represent merely an association of orphanhood with low (current) household income, as several earlier researchers argued (e.g. Lloyd and Blanc 1996). Rather, orphans are discriminated against in favor of children in the same household who are more closely related to the household head. Therefore they represent a particularly vulnerable group potentially needing targeted assistance. The degree of schooling disadvantage is a function of how distant the relationship is between the orphan and his or her caregiver, suggesting that 'true' (preference-based) discrimination based on relatedness is at work, not lower returns to investments in orphans arising from prior deprivation (or at least, not only lower returns).

Yet cross-section studies do not always find negative associations of orphanhood and welfare outcomes. Ainsworth and Filmer (2002) find diversity in orphan/non-orphan differentials across countries and argue that generalizations about orphan disadvantage are not warranted.[52] Chatterji et al. (2005) find for Rwanda and Zambia that among children 6-12 years old at the time of the survey, orphans actually have a higher point estimate of the probability of being in school than other children, although the difference is not statistically significant, while among 13-19 year olds, orphans are less likely to be in school, but this difference too is not significant. Lindblade et al. (2003) find similar health status of orphans and non-orphans under age 6 in western Kenya.

Given the difficulties in interpreting orphanhood effects from cross section data noted above, studies using longitudinal data are of particular interest. Evans and Miguel (2004) find negative schooling effects of orphanhood in their 5-year panel data of children in Kenya.

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[51] The cumulative nature of health or schooling outcomes can, subject to certain assumptions, be captured by including prior (lagged) values of the outcomes in the regression explaining current outcomes. This yields a more appropriate specification, but it is well known that differencing this model does not remove bias from unobservables (see Strauss and Thomas 1995). It could be estimated consistently using instrumental variable fixed effects, but this requires finding exogenous variables in the data that can predict prior schooling or health.

[52] Case et al. (2004) argue, however, that Ainsworth and Filmer's comparisons of orphans and non-orphans are misleading because they fail to adjust for differences in mean age and sex across the two groups.

Impacts are very large for maternal deaths (a 9 percentage point fall in enrollment probability relative to non-orphans). Negative effects, but smaller, were also found for maternal illness preceding death. These child fixed effects estimates of orphanhood are substantially larger (more negative) than estimates obtained on the same data without accounting for individual fixed effects, i.e., cross-section estimates. This suggests that previous cross-section studies tend to understate the impacts.

Using different panel data from Kenya, Yamano and Jayne (2005) are also able to study impacts on individual children over a five-year period. Without controlling for mortality selection, they find significant negative impacts of adult death on school enrollment among poor children and for both boys and girls, but when child-level fixed effects are used, there is only a negative effect on girls. Their data, it should be noted, do not allow them to distinguish between parental deaths and other adult deaths in the household, and we might expect the impacts of the former to be stronger. Case and Ardington (2005), using a 2-3 year panel survey from South Africa and child-level fixed effects and other methods, report a negative effect of mother's death on children's education outcomes but no significant effect of father's death.

Ainsworth et al. (2005) use the first several years of KHDS data from Kagera, Tanzania to estimate the effects of a recent parental death. They use standard OLS or probit models to estimate impacts on school enrollment and child fixed effects to estimate impacts on hours attending school. They find that enrollment was delayed for maternal orphans and for children in poor households where a parent died, but children 7-14 already in school who lost a parent were not more likely to drop out than other children. However, enrolled girls sharply reduced their hours in school after losing a parent.

Finally, Beegle et al.'s (2005) unique study using subsequent waves of the KHDS (see above) finds long term, that is, essentially permanent, negative effects of the loss of parents. By the time they reach adulthood, maternal orphans are on average two centimeters shorter and schooled one year less than they would have been had they not been orphaned. Unlike the panel studies just mentioned, Beegle et al. do not use individual fixed effects to deal with household or child level unobservables affecting both orphanhood and schooling and health; instead they include a range of baseline covariates to control for (observable) factors that may correlate with later orphan status. While this leaves their estimates vulnerable to contamination from unmeasured factors, they show that subsequent orphanhood is not correlated with initial schooling and health of the children. This suggests that unobservables correlated with health and education are not correlated with becoming an orphan in the future.

These panel data studies therefore do suggest significant negative consequences of orphanhood, and that losing a mother may be more harmful to child's health and education than losing a father. The latter result is consistent with a greater role for mothers than fathers in various aspects of child development. Some evidence also suggests that school age daughters are harder hit than sons. We can sum up these findings by saying that informal social safety nets in Africa, as essential as they are for insuring the welfare of children suffering parental illness and death from AIDS, do not appear to fully insure against these shocks.

Other impacts

There are several related issues that research has not addressed or has only just begun to examine. One is whether, as many have worried, informal safety nets will be stretched to the breaking point as adult mortality from AIDS rises, as it will continue to do in many countries in Africa. The rural Kenya study by Evans and Miguel (2004) mentioned above is one of the few to try to address this question statistically. They find that the proportion of children who have been orphaned in the community does not affect the likelihood of school participation, either for orphans or non-orphans, implying that in this setting mortality and orphan-hood have not reached such proportions as to weaken traditional support systems.

Evans and Miguel's finding for non-orphans addresses in part the issue of the effects of parental death on *other* children, in particular, children in households that care for orphans. One might well expect negative externalities: an orphaned child represents a negative resource shock to the receiving household that would be expected to reduce per capita consumption and schooling and health investments for all members, even if it is the case that orphans are allocated fewer resources than own children. Note that if only cross-section data are available, to assess this impact one is essentially comparing children in households that have not suffered an AIDS death and have not taken in orphans, on the one hand, with non-orphaned children in households that are caring for an AIDS orphan, on the other. Hence familiar issues arise with respect to the equivalence of two groups of households, specifically, the problem of non-randomness of the selection of caregiving households.

To date there appear to be only two studies that have considered these impacts. Evans (2005) conducts a careful cross-section analysis of DHS surveys from 26 African countries. He attempts to deal with the selection problem in several ways, including instrumental variable techniques (using reported death of a woman's sibling of as an instrument for her household receiving an orphan) and matching techniques. The estimates indicate, contrary to what many would expect, that there are essentially no negative health or schooling spillover effects on recipient household children as a result of caring for a double-orphan child. The health of adult women in such households (measured using body mass index) similarly is not negatively impacted. Possible reasons for these findings are that caregiving households tend not to be particularly vulnerable economically to begin with, and further, that they tend not to have children of the same age as the incoming orphans.

The other study, a longitudinal analysis of Uganda by Deininger et al. (2005), comes to rather more pessimistic conclusions. Deininger et al. consider the effects of a household receiving a foster child during the period covered by their long panel (from 1992 to 2000). More than 15% of households received a child during the period. The authors find that the addition of a foster child resulted in significant reductions of per capita consumption, income, and household investment (measured as the change in physical assets) and these effects were more pronounced for the poor. Fostered-in children themselves were found to be disadvantaged relative to other children in the household, but in terms of school enrollments, this disadvantage eventually disappeared with the implementation of Uganda's Universal Primary Education program during this period, which largely eliminated enrollment fees. Like

many other issues, the nature and extent of spillover effects of AIDS mortality and orphaning requires additional research, especially using panel data.

Even less is known about the implications for children of providing antiretroviral drug therapy to adults with AIDS. The impacts of ARV therapy on household incomes and other microeconomic outcomes are also largely unresearched. To date the only econometric analysis of these issues comes from an ARV program in rural Western Kenya (Goldstein et al. 2005, 2006). ARV patients were followed from the time they began the regimen to up to one year afterwards. Large improvements in patients' health indicators as well as their labor supply were seen. In sync with these changes, children in the households of the treated individuals experienced large and significant increases in school attendance (an increase in hours per week of 20 percent) as well as in an indicator of short-term nutritional status. In lieu of the appropriate control group (which would be children of AIDS-afflicted parents who did not get treatment) the authors use orphans from a random sample of households in the catchment area, who presumably more or less represent what the situation would eventually be for the children of treated parents if they did not receive the drug therapy. Difference-in-difference estimates using these children as the control group suggest even larger gains for the children of treated parents than the estimates not controlling for trends.

This initial research therefore indicates that there are substantial beneficial externalities to households and children from supplying ARVs to adults with AIDS. However, the study has (so far) only reported on short-term benefits. As noted earlier, first line drug therapies, which are all that is widely available now in Africa, are currently expected to extend life of AIDS sufferers for several years. The question is whether the gains for children evaporate after that time. To some extent, the benefits, with respect for example to the health of young children, are cumulative so will have lasting effects even if households later suffer from declines in living standards. Still, the long run benefits of scaling up ARV distribution remain unknown. This is hardly surprising in view of the recent introduction of these drugs, but it is important that the question be given a high priority in future research.

Finally, the literature on the health and schooling of AIDS orphans (and the impacts on households that care for them) does not deal with broader possible effects of Africa's AIDS epidemic on human capital investment. What are the effects in families not (or not yet) directly affected by AIDS? Analogous to the discussion of fertility impacts in Section II.3.4, changing expectations about one's own mortality and that of one's children should condition decisions about investing in the children's health and schooling, even among households not currently afflicted by AIDS. A standard economic model of human capital investments would predict negative effects of the epidemic. A lower expected lifespan for children reduces the return on investments in their education: each additional year of schooling yields a smaller increment in total lifetime earnings compared to someone with a normal (i.e., pre-AIDS) expected lifespan. Further, since parents themselves are more likely to die young, the old-age security motive for schooling one's offspring (that better educated children will earn more and thus provide more transfer income) is diminished.

Research approaches to this issue will necessarily be different than those used to estimate schooling effects on individuals or households directly affected by AIDS; they will look more like the analyses of fertility responses to the epidemic discussed above. In fact, the authors of the two existing cross-country econometric studies of the effects of HIV prevalence on fertility, Young (2005) and Kalemli-Ozcan (2006), also analyzed school enrollment effects. Just as with fertility, Young finds a positive effect and Kalemli-Ozcan a negative effect. As with fertility, the question is important but the answer remains elusive.

IV. CONCLUSION

This paper has considered the linkages of poverty, reproductive health and behavior, and HIV/AIDS in Africa. It has presented evidence on what research to date has shown about these links, identified gaps in knowledge requiring further research, and discussed methodological approaches to these questions. Given the diversity of the subjects covered and the sheer size of the empirical literature investigating these issues, it would be very difficult to sum up the discussion in any concise way. Instead, in view of the emphasis on practical guidance for researchers, I will highlight several salient points with respect to conducting research in these areas.

The first is that using an appropriate methodology and study design is crucial. A main theme of this paper has been the need to account for the phenomenon of selectivity or non-randomness: in the use of services, in the placement of programs, in HIV infectivity, in fertility and sexual risk behaviors, in orphanhood and orphan caretaking. Where individual behavior and preferences are at issue, selectivity looms large. Much of the discussion in this paper has dealt with how researchers can overcome the problem, and the limits of different kinds of data and techniques for doing so. With regard to data, there are many good reasons to collect more panel data in Africa. In particular, they offer more ways of dealing with the selection issues that are a barrier to better understanding of the impacts of HIV/AIDS at the household level. Panel data sets are more common in Africa than a decade or two ago; at least four have been carried out in South Africa alone. Elsewhere in Africa, however, panel data collection is less common, if increasing.[53]

Secondly, it is relevant to note that enormous resources are being allocated to HIV programs in Africa, from prevention interventions to ARV provision. This of course is a reflection of the seriousness of the problem and the commitment of donors and governments to finding solutions. It provides opportunities for researchers that may be lacking in other areas. Certainly, few individual African (or for that matter, Western) researchers or social science research groups would have access to the resources necessary for evaluation of many of the interventions discussed in this paper. Further, many HIV-related interventions also involve the provision of medical services that obviously are beyond the scope of what social scientists do. However, many such interventions are underway or planned in African countries, by ministries of health or education, by NGOs, and by donors. Social science researchers potentially could link up with these efforts to contribute to the evaluation of outcomes, and even better, to help in the design of these evaluations. The inclusion of economists or demographers in this process would help to expand the range of behavioral outcomes to be considered. Economists and demographers are also particularly well placed to undertake the population-based sampling and statistical analysis required to understand the demand for and population level outcomes of various HIV or reproductive health care services.

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[53] South Africa features the KwaZulu-Natal Income Dynamics Survey, the Cape Area Panel Study, the South African Labour Force Survey, and the Africa Centre Demographic Information System (ACDIS) KwaZulu Natal rural panel. Panel data from other African countries have been mentioned throughout this paper.

Third, it nevertheless remains the case that many or most researchers will not be involved either in evaluating specific programs, conducting policy experiments, or collecting original data. Instead they will come to existing data sources with specific research questions. This report therefore has also emphasized how researchers can exploit secondary data sources to address important questions on reproductive health, HIV/AIDS, and poverty. In particular, the range of subject areas that have been considered and analytical methods used with the Demographic and Health Surveys is close to astonishing. There is plenty of scope for imaginative and informative work with these data, including by matching them to other sources of information on the policy and economic environments. These data and related surveys like the AIDS Indicator Surveys will become even more valuable in the future as more of them include nationally representative information on HIV status.

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Figure 1

Interrelationships of Poverty, Reproductive Health, and HIV/AIDS