Routes of Infection: Exports and HIV Incidence in Sub-Saharan Africa

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First Draft: March 25, 2007

This Draft: January 16, 2009

Abstract

This paper estimates the relationship between economic activity (specifically, exports) and the incidence of HIV in Africa. This relationship has implications for anti-HIV policy as well as for the consequences of trade increases in Africa. A challenge to this estimation is poor data on HIV infections in Africa over time. I address this by using two newly created datasets on incidence: one based on recent data from UNAIDS (UNAIDS, 2008) and the other based on inference from mortality data (Oster, 2008). I relate economic activity to new HIV infections in Africa and argue that there is a significant and large positive relationship between the two: a doubling of exports leads to approximately a doubling in new HIV infections. I argue that this relationship is causal and that the mechanism, in part, is through increased movement of people (trucking). I apply this result to study the case of Uganda, and argue that a decline in exports in the early 1990s in that country appears to explain between 35% and 50% of the decline in HIV infections. This suggests that the success of the Ugandan anti-HIV education campaign, which encouraged changes in sexual behavior, has been overstated.

1 Introduction

Economists and policy-makers have traditionally argued that there is a positive link between income and health (see, for example, Pritchett and Summers, 1996). This link has been taken as evidence that an increase in globalization and trade in the developing world will make people better off not only through increases in income but also through improvements in health (Dollar, 2001). In line with this, cross-country analysis has suggested that trade and health are positively linked (Owen and Wu, 2007; Levine and Rothman, 2006). Recent research, however, has called into question whether increases in income really do increase health (Case and Deaton, 2006; Ruhm, 2007). Focusing specifically on communicable diseases, historical episodes (the Black Plague, for example) suggest

*Ward Cates, Jane Fortson, Daniel Halperin, Norman Hearst, Emir Kamenica, Lawrence Katz, Michael Kremer, Steven Levitt, Kevin Murphy, Ben Olken, James Poterba, Jesse Shapiro, Andrei Shleifer, Rebecca Thornton and participants in seminars at the Gates Foundation, the University of Chicago, UCL, Northwestern, Ohio State and Berkeley provided helpful comments. Dwyer Gunn provided outstanding research assistance.

that trade in particular may decrease health by increasing the speed at which the disease is spread (Wilson, 1995; McNeill, 1976).

In this paper I explore the connection between economic activity and health in an important policy context: HIV in Sub-Saharan Africa. Significant policy emphasis has been put on the importance of trade in increasing income in Africa, following the lead of China and India (Arbache, Go and Page, 2008; Dollar, 2008). At the same time, roughly 5% of the adult population is infected with HIV, and if the historical evidence is a guide, increases in economic activity have the potential to increase the spread of the virus. This concern seems particularly worrying since individuals traditionally involved in trade-related activity (truckers and migrant workers) have very high HIV prevalence (Lurie et al, 2003a; Lurie et al, 2003b; Brewer et al, 1998; Brockerhoff and Biddlecom, 1999; Anarfi et al, 1997; Anarfi, 1993).

The primary analysis in this paper focuses on estimating the relationship between HIV and exports within countries over time and relating new HIV infections in a given year to the export activity in that year. I begin in Section 2 by briefly outlining the theory and empirical strategy motivating this argument. I set up a model describing the evolution of HIV prevalence as a function of sexual behavior and transmission rate, and model exports as affecting the extent of risky sexual behavior. This model implies a simple estimation strategy in which we estimate the relationship between HIV incidence rate and exports per capita.[1]

The significant challenge to this estimation is poor data on HIV infection rates, which make it difficult to calculate a time series of new infections.[2] To address this, I use two newly avaialble sources of data on HIV infections. First, I use recently produced data on trends in HIV infection from UNAIDS to estimate incidence in 35 countries (UNAIDS, 2008). Second, for a subset of 11 countries I also use estimates of infection rates based on inference from mortality data, described in Oster (2008). Both data sources are described in more detail in Section 3.

I use these two measures of HIV incidence to estimate the relationship between HIV and exports. I find a significant relationship between the two series: controlling for country and year fixed effects, I find that doubling exports leads to roughly a doubling in new HIV infections. The

[1] Disease incidence refers to number or rate of new infections; prevalence refers to the stock of infections.

[2] Before the early 2000s, virtually no testing was done in the general population. Testing was done among pregnant women, but the selection of the sample changed significantly between years in most counties, undermining our ability to use these data to estimates trends. In the last few years, excellent data based on testing of a random sample of the population have come out from the Demographic and Health Surveys (Halperin and Post, 2004). However, this does not address issues arising from a lack of consistent data over time. Further, in order to calculate new HIV infections in the current year it is necessary to have data going back into the past – new infections in year t is not simply the difference between number of infections in year t and year $t-1$; it is also necessary to adjust for deaths, which are affected by infection rates in earlier years.

relationship is larger and more precise in countries with higher HIV infection rates, likely due to more precision in the estimates of HIV incidence. I argue that the relationship between HIV and economic activity is causal. Reverse causality is unlikely to be a concern because of the contemporaneous time frame: there is no reason to expect new HIV infections this year (which would be asymptomatic) to drive exports this year. In addition, I argue that it is unlikely that an omitted variable (for example, government policy) is driving these results, based both on the fact that the estimates are robust to including country-specific trends and the fact that the relationship holds when I instrument for exports with world commodity prices. Finally, I test whether exports in future years impact HIV infections in the current year and find that, with a single exception, they do not.

There are a variety of possible mechanisms through which increases in exports could increase HIV infections, including through increases in income, since the data typically indicates that sex is a normal good. Fully teasing apart all of the possible mechanisms is beyond the scope of this paper, but as a first step I show several pieces of evidence supportive of a role for trucking, or people-movement, in mediating this mechanism. First, I demonstrate that volume of goods trucked increases with exports, and argue that this, in combination with existing evidence that truckers have more high risk sex, supports this mechanism. Second, I show that in countries with higher road density the relationship is much stronger. Finally, I find that in landlocked countries (those without a port city), which are likely to be more reliant on trucking to move exports, the relationship between HIV and exports is much stronger. It is important to note that these results do not *prove* the importance of trucking, or rule out a role for other mechanisms, but they do suggest this mechanism plays some role.

The final section of the paper applies the results on the relationship between exports and HIV to understanding prevalence declines in Uganda in the 1990s. There was a large drop in HIV prevalence in Uganda in the 1990s, which is unique among Sub-Saharan African countries; this drop is generally credited to educational programs, which promoted partner reduction ("zero grazing"), and to elements of ABC (Abstain, Be faithful and use Condoms) (Green, 2004; Green et al, 2006; Slutkin et al, 2006). This anti-HIV education campaign was among the first successful HIV prevention strategies, and this type of education campaign has become a central tenant of HIV prevention in Africa, with extensions to Kenya, Tanzania, Malawi and elsewhere. Both UNAIDS and PEPFAR have taken this as a major focus of their prevention approaches; in 2004, PEPFAR spent $100 million of their budget on ABC-related programs.

During this period in Uganda, however, there was also a very large decline in exports, due primarily to a decline in world coffee prices. The magnitude of the relationship between HIV and exports (estimated based on countries outside of Uganda) suggests that between 35% and 50% of the incidence decline in Uganda during this period can be attributed to changes in economic activity, which in turn suggests that the success of the education campaign has been overstated to some extent.

The Uganda results may have important policy implications, suggesting that education campaigns may be less effective than typically thought. In addition, the connection between exports and HIV in general also suggests specific avenues for HIV prevention. In particular, the implication that truckers and other migrants are an important driver of the overall epidemic supports targeting prevention activities at that group (similar to the targeting done of prostitutes in Thailand). Finally, the result suggests that, similar to recent findings on income and health more generally, increases in economic activity and trade may have a perverse effect on communicable disease, at least in this particularly important context.

The rest of the paper is organized as follows. Section 2 outlines a very simple model of the epidemic and presents the estimation strategy; Section 3 describes the data used. Section 4 estimates the relationship between HIV incidence and exports and Section 5 discusses evidence on the mechanisms. Section 6 discusses the case study of Uganda, and Section 7 concludes.

2 Epidemic Model and Estimation Framework

This section outlines a very simple model of the HIV epidemic and describes how changes in economic activity could impact epidemic growth. In addition to providing motivation, this suggests a particular functional form for estimation.

Assume every individual has either zero or one randomly chosen sexual partners, and denote p_{it} as the share of individuals with one partner in country i in year t. Assume the HIV transmission rate (the chance of becoming infected given one sexual partner) is β and the current HIV prevalence (i.e. infections divided by population) in country i in year t as h_{it}. The incidence rate (new infections divided by population) is denoted n_{it} and calculated as below.

$$n_{it} = p_{it}\beta h_{it} \tag{1}$$

Given this, we consider how economic activity may mediate this relationship. Based on the individual-level relationship between HIV infection and economic activity I posit that individuals involved in this activity (truckers, migrants) may have more sexual partners. Further, as mentioned in the introduction, if more exports mean more money in general, this may be used to buy more sex. Either argument suggests some relationship between p_{it} and exports. Denote exports per capita as y_{it}; I model $p_{it} = f(y_{it})$, where $f(.)$ is an increasing function.

One possibility is that p_{it} is just a linear function of y_{it}. This would be true, for example, if every unit of export needs one additional driver, so y_{it} is equal to the share of the population involved in trucking, and all truck drives have risky partners but no other individuals do. It is also plausible that p_{it} is a concave or convex function of y_{it}, for example if the availability of risky sex improves once the number of participant reaches a critical level (convex), or if initial recruits into economic activity have a higher taste for sex than later recruits (concave). To capture all of these cases, we model $p_{it} = \alpha y_{it}^{\gamma}$.

Under this assumption,

$$n_{it} = (\alpha y_{it})^{\gamma} \beta h_{it} \tag{2}$$

Taking logs of both sides, we come to

$$ln(n_{it}) = \gamma ln(y_{it}) + \gamma ln(\alpha) + ln(\beta) + \ln(h_{it}) \tag{3}$$

Note that $\frac{\partial n_{it}}{\partial y_{it}} = \gamma$ suggesting that we can recover the relationship between sexual behavior and exports by estimating the relationship between log new HIV infections and log exports per capita.

Given this framework, we note that the analysis becomes much more complicated if we do not observe incidence. Assume we observe only prevalence, h_{it}. We can express prevalence without using incidence in Equation 4.

$$h_{it} = (\alpha y_{it})^{\gamma} \beta h_{i,t-1} + h_{i,t-1} - d_{it} \tag{4}$$

where d_{it} is the death rate in country i in year t (this is a function of infections in all previous years). Given the additive structure we cannot take logs in this case, and $\frac{\partial h_{it}}{\partial y_{it}} = \alpha\gamma (\alpha y_{it})^{(\gamma-1)} \beta h_{i,t-1}$. In other words, the coefficient in this case is a function of both the export level and previous year's HIV prevalence; certainly less readily interpretable.

Estimation

The discussion above serves primarily to motivate the functional form of the estimation relationship. The primary analysis will estimate equation (5) below.

$$Ln(\text{Incidence Rate}_{it}) = \psi_0 + \psi_1(ln(\text{Exports PC}_{it})) + \psi_2(\text{HIV Rate}_{i,t-1}) + \tau_i + \pi_t + \epsilon_{it} \tag{5}$$

where τ_i is a full set of country fixed effects and π_t is a set of year fixed effects; standard errors in all cases are clustered at the country level. In all regressions we will control for HIV rate in the previous year. In addition, in the primary specifications I include controls for the country's GDP per capita and I will show results with controls for yearly temperature and rainfall.

3 Data

The analysis described above requires data on new HIV infections, as well as data on exports and country-level controls. These are discussed in turn below.

3.1 Data on HIV Infections

As discussed in the introduction, lack of good data on HIV incidence over time in Africa makes this analysis difficult. Until the early 2000s, most HIV testing in Africa focused on pregnant women or high risk groups (drug users, STI patients), meaning we know very little about the level of infection in the general population. More problematic, perhaps, is the fact that testing of even these non-representative groups is inconsistent (variations in the areas or populations tested over time), making the prevalence data very noisy and difficult to compare across years. Noisy data on prevalence, in turn, make it almost impossible to infer anything reliable about the new infection rate each year.

In recent years, population-based testing from the Demographic and Health Surveys (ORC Macro, 2006) have provided much better prevalence estimates. However, these estimates – even in the few cases in which we observe multiple years – are not sufficient to calculate incidence. To see why, note that, with the notation from Section 2, incidence can be written as below

$$n_{it} = h_{it} - h_{i,t-1} + d_{it} \tag{6}$$

To calculate incidence, we therefore must observe prevalence in the current year, and in a past year, *and* observe deaths in the current year. The number of deaths are, in turn, a function of infection rates in the past. Simply observing two years of prevalence is not sufficient; we must observe the whole time pattern of prevalence, over the course of the epidemic. Given this constraint, I therefore use two data sources which provide a full time series of HIV prevalence over the course of the epidemic and allow me to calculate incidence.

UNAIDS Data

The first dataset is from UNAIDS. UNAIDS is the United Nations organization responsible for reporting on, and addressing, the global HIV epidemic. UNAIDS has, for a number of years, put out estimates of HIV prevalence. However, they typically caution that these estimates are not comparable across reports (UNAIDS, 2008), making them difficult to use for calculating incidence. In 2008, UNAIDS announced a dramatic revision in their overall estimates of the magnitude of the HIV epidemic in Africa. Along with this revision, they released a full set of country-level HIV prevalence estimates, by year, for 1990-2007. These estimates are based on their best knowledge – from population-based testing, epidemic modeling and older antenatal clinic surveillance estimates – of levels and trends in the epidemic over time, and they provide the basis for the estimation of incidence.[3]

As noted above, to calculate incidence – even in recent years – it is necessary to know prevalence back to the start of the epidemic. For this reason, the UNAIDS data is not sufficient, since the epidemic was well underway by 1990. To fill in the gap between the apparent start of the epidemic in the mid-1970s and the start of the data in 1990, I use a combination of earlier trend data from UNAIDS and linear interpolation. More specifically, earlier UNAIDS reports from the countries in the sample typically included some time series information on infection rates at antenatal clinics through the 1980s. I use these data to calculate the trend in infection rates from the mid-1980s through 1990; the level is calibrated to match the 1990 data.[4] This data goes back to the early to mid-1980s; prior to that there was no test for the disease. To estimate rates in earlier years, I therefore calculate what epidemic growth rate would be necessary for the prevalence to increase from an assumed rate of zero in 1976 to the first observed year of data in each country. I use this growth rate to estimate prevalence.

The second step is calculation of incidence. As in Section 2, denote HIV prevalence in year t as h_t and incidence as n_t. Further, denote the chance of dying from HIV after τ years of infection as

[3] Another source for HIV data is the US Census HIV/AIDS Surveillance Database, which reports testing data from antenatal clinics and other sources over time. It would, in principle, be possible to put these data together and (with some smoothing assumptions) generate consistent estimates of prevalence. This would require a number of additional assumptions, however. Moreover, the data from this testing should be incorporated in the UNAIDS estimates, which take into account all testing done in the country.

[4] Typically the infection level in earlier reports is higher than in the new UNAIDS data. To address this, I calibrate the trend to the 1990 level. For example, if the earlier reports have a rate of 1% in 1982, 5% in 1985 and 10% in 1990, and the new data reports a rate of 5% in 1990, I assume the rate was 5% in 1990, 2.5% in 1985 and 0.5% in 1982.

d_τ. Given this, we can express incidence in year t as

$$n_t = h_t - h_{t-1} + \sum_{i=1}^{t-1} c_{t-i} n_i \tag{7}$$

That is, incidence is the difference between the stock of infections in year t and the stock in year $t-1$, with an adjustment for deaths. The number of people who die in year t is equal to the number of people infected in year $t-1$, multiplied by the chance of dying after one year, plus the number of people infected in year $t-2$, multiplied by the chance of dying after two years, and so on. This makes it clear that the other important input to calculating incidence is time from HIV infection to death from AIDS.

Detailed data on time to death are difficult to generate, particularly in developing countries, since it requires knowing (roughly) the time of infection. For this reason, the best available data are drawn from developed countries, from time periods before HIV treatment was available (Collaborative Group on AIDS Incubation and HIV Survival, 2000). Researchers modeling the epidemic in Africa (Stover, 2003; Statistics South Africa, 2004), have used the "fast" version of these paths, which assumes that the trend in time to death is similar to the developed world, but the average time to deaths is faster (due to lower nutrition, higher disease burden, etc). Figure 1 shows the time path to death used for these analysis; the values of c_τ used in the calculations are drawn from the data underlying this figure.[5] Of course, these data on time to death are an approximation, and in the robustness section I will explore how the results change if time to death is faster, or shaped differently.

The final output of this calculation is data on HIV incidence, by year, in 35 countries. The countries are listed in Appendix A, which also reports summary statistics on average prevalence and incidence in each country. One important note is that estimates from later years (particularly, the mid-1980s forward) are likely to be more accurate than early estimates, since they are based in larger part on observed testing results rather than interpolation. These estimates for later years do rely, of course, on the earlier years, but as we move forward in time this reliance gets less significant. For this reason I limit the analysis to 1985 and later; the central results are extremely similar if we include the earlier time period.

[5] These data on time to death are estimates for a period *without* HIV treatment. I argue this fits the data, at least for a very large share of the period here. UNAIDS(2008) estimates that as late as 2002, only 50,000 people in Sub-Saharan Africa were receiving anti-retrovirals, which is about .2% of the infected population in that period. By 2007, which is the end of the period here, this was up to 2.1 million, which is closer to 10% of the population, and could make more of a difference. In the robustness section I will also explore how the results change if we allow for changes in the time to death in this later period.

Mortality-Based Data

The second dataset includes estimates of incidence and prevalence based on inference from mortality data. The methodology and estimates are reported in detail in a companion paper (Oster, 2008). Here, I briefly outline how the estimates are produced.

The basic methodology is straightforward. Assume we observe deaths from HIV in a given year, denoted μ_t. Retaining the notation from above, with n_t as new infections in year t and c_τ the chance of dying τ years after infection, we can express μ_t as

$$\mu_t = c_1 n_{t-1} + c_2 n_{t-2} + ... + c_{20} n_{t-20} \tag{8}$$

Assuming that we observe μ_t and the elements of the $\mathbf{c}$ vector, the only unknowns in this equation are incidence values. Of course, with a single year of data on deaths we will not be able to solve for incidence in all years.

In order to solve for the full set of incidence data we again need to see data back to the start of the epidemic. Assume we observe some deaths in a year $t - x$ such that there is no HIV infection before year $t - x - 1$. In that case,

$$\mu_{t-x} = c_1 n_{t-x-1} \tag{9}$$

and from this it is possible to solve for n_{t-x-1}. If we then observe deaths in year $t - x + 1$, we can solve for n_{t-x}, since n_{t-x-1} is known:

$$\mu_{t-x+1} = c_1 n_{t-x} + c_2 n_{t-x-1} \tag{10}$$

Further backward induction yields the entire vector of incidence estimates.

These calculations require two pieces of data: estimates of time to death from HIV, which are again drawn from Figure 1, and estimates of death rates. As discussed in more detail in Oster (2008), deaths from HIV are estimated by comparing data on overall death rates (from Demographic and Health Surveys sibling mortality histories), and data on expected death rates (based on non-HIV countries with comparable development levels in the United Nations Demographic Yearbook).

The mortality-based prevalence and incidence data is available for 11 countries – the data are limited to countries with sibling mortality histories in the Demographic and Health Surveys. In Oster (2008) I present evidence suggesting that these data match well to population-based testing data, where the latter is available. These data are also highly correlated – correlation coefficient of 0.88 – with the UNAIDS prevalence estimates. The countries in these data are listed in Appendix A, with summary statistics on incidence and prevalence. As with the UNAIDS data, I limit the analysis to 1985 and after. This is done for many of the same reasons as with the UNAIDS data. Because the death rates rely on retrospective reporting, data from further back in time will be less reliable; again, robustness checks will be done using all of the data.

Both the UNAIDS data and the mortality-based estimates have drawbacks. In the case of the UNAIDS data, the exact methodology used to generate the 1990-2007 prevalence estimates is somewhat opaque, and some assumptions are necessary to generate incidence data from the prevalence measures. The methodology generating the mortality-based estimates is more transparent, but has more significant data requirements and can be used only for a smaller sample of countries. The hope is that by using both datasets and exploring, to the extent possible, robustness with respect to the assumptions generating the HIV incidence values, I can avoid the concern that the results are driven by some feature of the data generating process

3.2 Data on Exports

The other main data requirement is data on exports at the country-year level. The primary source for export data is the World Development Indicators (WDI) reports of export value; this is adjusted to be value per capita using WDI measures of population. In all cases, the measure of export used is an average of exports this year and in the previous year, which is helpful in dealing with some of the noise in the timing of the HIV estimates.

I also report results for several other auxiliary measures of trade. These include the value and volume of each country's one or two major exports, generated from the NBER-United Nations Trade Data from Feenstra et al (2004). I use 1998-2000 data to estimate each country's major exports, based on value (in most cases these exports are clear – copper for Zambia, coffee for Uganda, etc – and make up more than 50% of total exports). I focus on the single largest export in cases where the top export accounts for 50% or more of export value, and the top two exports otherwise. I then use these data to estimate value and volume of these exports for the available period (prior to 2001). Although following the major exports only excludes some of the trade in these countries, it is necessary in order to observe volume consistently over time, since volume is not well-tracked for many of the more minor exports. In addition to value and volume, I use import data from the WDI. Summary statistics on the trade measures are shown in Table 1.

3.3 Data on Controls

In the primary specifications, I control for country and year fixed effects, and for log of GDP per capita, which is drawn from the WDI. In addition, I explore the robustness of the results to controls for average temperature and precipitation by the country-year, which are taken from Dell, Jones and Olken (2008). Summary statistics for these measures are also provided in Table 1.

4 Results: HIV Incidence and Exports

This section presents the primary results in the paper, estimating the relationship between exports and new HIV infections. Subsection 3.1 below presents the basic results on HIV and exports, and Subsection 3.2 discusses issues of causality. Subsection 3.3 discusses the robustness of these basic results to the assumptions used to generate HIV incidence.

4.1 Primary Results

Table 2 show the primary results in the paper. Columns 1-3 focus on the UNAIDS data. Column 1 includes the minimum set of controls: lagged HIV rate, country and year fixed effects. The coefficient on exports is positive and significant: increasing exports increases the incidence rate. Column 2 presents the primary specification, which includes a control for GDP. The relationship between exports and HIV is still positive and highly significant, and of a similar magnitude. Finally, Column 3 also includes (with a slightly limited sample) controls for average yearly temperature and precipitation. The relationship remains large and significant; the coefficient actually gets larger, which is due entirely to the change in the sample. Columns 4-6 replicate these regressions using the mortality-based measure of incidence. As with the UNAIDS data, the coefficient on exports in these regressions is positive and significant; in this case, the coefficient is quite a bit larger, although the standard errors admit magnitudes close to the size seen with the UNAIDS data.

To give a visual sense of the data, Appendix B shows graphs of exports and incidence, over time, for all of the countries in the sample. In some cases we can see both UNAIDS incidence and mortality-based incidence; in others only the UNAIDS data is observed. In general, these figures show evidence consistent with what we see in Table 2 – exports and incidence are moving together. This is, of course, not universally true, but does seem to be evidenced in a large number of countries.

I noted in the discussion in Section 2 that, given the specification used, we do not expect the effect of exports to vary with previous year's HIV rate. This is clear from equation (5), and is true despite the fact that lagged prevalence is an important input in general in driving incidence. However, it is nevertheless interesting to consider how the coefficients differ across countries with different overall levels of HIV. Among other things, both methodologies for generating HIV data are likely to be more precise for countries with higher HIV rates, where the deaths will be more obvious and there is more testing done; this could lead to stronger effects for higher HIV countries. Table 3 replicates Columns 2 and 5 of Table 2, but splits the sample in half, into high and low HIV countries based on their average HIV rate over the entire period. For both measures of HIV rate the relationship in high-HIV countries is larger than in low-HIV countries consistent, perhaps, with less measurement error in incidence.

One important issue is to what extent individual countries are driving these results; this is likely to be a more significant issue in the analysis with the mortality data, where we have only eleven countries. Appendix Table 1 reports coefficients from regressions of the form in Columns 2 and 5 of Table 2 excluding each country in the sample. Even in the case of the mortality-based data, the coefficients and significance do not seem to be very sensitive to the exclusion of any of the countries in the sample.

Finally, before turning to discuss causality, we consider several auxiliary measures of trade. Table 4 reports results from regressions of HIV on the other trade measures – value and volume for major exports, and imports. Panel A show the results for the UNAIDS data; Panel B shows the results using the mortality-based data. These coefficients are consistently positive, and for the most part at least marginally significant. They are less precise than the results using the WDI data, perhaps due to the fact that when looking at major exports only we miss a substantial portion of export activity, perhaps the portion that is most variable. Nevertheless, the fact that these coefficients are positive and in many cases significant does suggest the results are not “special” to the WDI export data.

The relationship with exports estimated here is fairly large in magnitude and, hence, the overall effect of changes in exports on HIV may be big. Based on the coefficients in Table 2, I create a simple counterfactual: if exports in all years in the sample were lower by 25%, what effect would that have on HIV infections? Although the exact value depends on which measure of exports is used, on average the counterfactual suggests that a 25% decline in exports would mean a reduction of between 20% and 50% in the number of new infections.

4.2 Identification

The results above suggest that there is a reduced-form relationship between HIV and exports. This leaves open the question of whether the relationship here is causal. That is, if there is an increase in exports, will that lead to an increase in HIV? This issue is, of course, distinct from the question of what *mechanism* drives this relationship, which is discussed in the next section.

A first issue with identification is the possibility of reverse causality: higher HIV causing higher exports. This seems very unlikely. First, in the time frame we are considering – variations at the yearly level – it is hard to see how more new infections (which would be asymptomatic) could affect the economy. Second, even to the extent that HIV might affect exports in the long run (for example, by debilitating the workforce) the relationship would likely be in the other direction: more HIV would mean lower exports, not higher. For both of these reasons reverse causality should not be a major concern.

A similar timing-related logic applies to theories that the results are driven by a relationship between some other cause of death and exports. For example, if road accidents are an important source of mortality, they might go up during periods with more exports, when there is more trucking. However, this *cannot* drive the results, because what we are estimating is the relationship between new HIV *infections* this year and exports, not HIV deaths and exports. Additional mortality from other causes in any given year will be attributed to HIV infections an average of 10 years in the past, *not* to HIV infections in the current year. If anything, this type of story is likely to dampen the results.

A second issue lies in omitted variable bias. When considering this possibility, it is important to note first that the regressions Table 2 include country and year fixed effects. The inclusion of these controls means that any confounding factor will have to also vary within a country over time. We can take this a step further by estimating the regressions using these fixed effects *plus* country-specific trends. In some sense using such extensive controls with a limited sample size may be a concern, and make it somewhat harder to interpret magnitudes and understand where the identification is coming from. However, to the extent that the results hold qualitatively with these trends included, this excludes a large set of possible confounds. For example, one possible confounding factor is changes in government: if a new government comes in that cares a lot about the economy but not about health we could see an increase in exports but a decrease in HIV prevention. However, these policy changes are unlikely to happen quickly, and including country-specific trends should capture them.

Columns 1 and 2 of Table 5 report the relationship between exports and the two measures of HIV incidence, controlling for country-specific trends. These regression exclude GDP controls – the data is simply not sufficient to estimate all of these parameters together. Moreover, for the most part, reported GDP moves smoothly upward over time in these countries and is therefore largely captured by the additional controls. The coefficients remain positive and statistically significant for both HIV measures. This limits the set of omitted variables which could be driving the result.

As a second argument against omitted variables driving the results, I take advantage of an instrumental variables strategy, instrumenting for exports with world commodity prices. The countries used here export primarily commodities, so their exports are heavily influenced by commodity prices, but they are sufficiently small producers that the price is largely determined by events outside of their borders. The exclusion restriction – that world commodity prices do not drive HIV infections for reasons other than through their effect on exports – therefore seems reasonable. To perform the instrumental variable analysis, I generate a price measure for each country that is a weighted average of the world prices for each export major, with the weights determined by the share of the total exports accounted for by each commodity. Historical commodity prices are drawn from the IMF's International Financial Statistics database (year 1980-2001). I focus only on major exports, since identifying commodity prices for all exports is not possible. To adjust for the fact that these major exports make up a larger share of total export value in some countries than others, these regressions are weighted at the country-level by the share of exports accounted for by the commodity price.

Columns 3 and 4 of Table 5 show IV regressions where exports are instrumented with commodity prices (the instruments are commodity prices in the current year, previous year and year before that, to capture lags). For both measures of HIV the coefficient on exports is positive and statistically significant, and similar in magnitude to what we see in the OLS estimates. It should be noted, however, that the F-statistic on the instruments in the first stage is relatively small in both regressions, so these results should be taken with caution.

Finally, we can explore the causality issues broadly by looking at whether *future* exports drive current HIV rate. If true, this would suggest either some omitted variable influence or, in general, call into question the specification and data. To explore this, I regress each measure of HIV on current exports and future exports, with leads of different lengths (2-10 years).[6] Figures 2a and

[6]I begin with two years, rather than one year, since noise in the data is such that infections from next year might well

2b graph coefficients on present and future exports from these regressions. For both HIV measures the coefficient on current exports is consistently positive and significant in all specifications. We note that the coefficient varies some, particularly in the case of the UNAIDS data, simply because the sample becomes more restricted (i.e. to early years) when we include very long forward leads. In Figure 2a, focusing on the UNAIDS data, the coefficient on future exports is small and insignificant at all leads. In Figure 2b, focusing on the death data, this is largely true as well, although it looks like the coefficient on exports two years out is significant, albeit smaller. This may be due to issues of spill-over in "allocation" of HIV infections across years, or due to the use of the smoothing parameters, which effectively force some relationship in incidence rates across years. Overall, however, these figures are strongly supportive of the results. Exports this year matter; exports in the future do not.[7]

All of these analyses provide support for the claim that the relationship between HIV and exports is causal, at least in the sense that if exports increase, we expect to see HIV increase. The next subsection discusses the robustness of this result to the assumptions which generate incidence; I then move to discuss what mechanisms might drive this relationship.

4.3 Robustness to Assumptions Generating HIV Data

One important concern with the regressions above is that the relationship between HIV and exports is being driven by the assumptions that generate incidence, rather than by the underlying relationship. For both the UNAIDS data and, especially, the mortality data, assumptions go into converting either the prevalence data (for UNAIDS) or data on deaths (for the mortality-based estimates) into incidence rates. In this section I briefly consider how the relationship changes if I vary two of the central assumptions: the time path to death from infection and the epidemic start date.

To address the issue of time to death, I first generate both the UNAIDS incidence and the mortality-based incidence using two alterative paths of time to death. One of the alternative paths features a faster time to death: this faster time path assumes that everyone has the time to death profile of the oldest age group. The other alternative path models time to death as flatter but with similar speed: 8% of people die every year between 5 and 14 years after infection, rather than the more peaked shape observed in Figure 1 (both alternative paths are illustrated in Appendix Figure 1).

be attributed to this year, making this a less powerful test at very short leads.

[7] The coefficients here come from multiple regressions (one for each lead); the results look very similar if we run a single regression with leads of different lengths included.

In addition to these two alternatives, it may be important to consider how the availability of treatment could change these results. As noted in the data section, treatment levels before 2002 were extremely low – UNAIDS estimates only 0.2% of infected individuals in Sub-Saharan Africa were receiving treatment by 2002. This suggests that, for the mortality-based data, which is all from the pre-2002 period, this change is not important. The UNAIDS data, however, goes through 2007, at which point 10% of HIV-positive individuals were receiving treatment (UNAIDS, 2008). To explore this, as a third alternative I assume the standard time to death prior to 2002 and then assume no deaths after that. This is obviously an extreme change, but it should give a bound on how this change could affect the results.

The first three rows of Table 6 show the coefficients on exports in the primary specification (corresponding to columns 2 and 5 of Table 2, with country and year fixed effects and a control for GDP). The different assumptions on time to death make almost no difference for the results with the UNAIDS data. This is reflective of the fact that, for the most part, incidence in year t in this data is the difference between prevalence in year t and year $t-1$. The magnitude of the adjustments for death rates are small simply because in even late in the epidemic people who are dying were infected early in the epidemic, and early infection rates are lower. For the mortality-based data the results differ more in magnitude, which reflects the fact that this time to death is a much more important input to the calculation when we rely on death rates as a starting point. However, even with the different paths, the results are positive and significant (in fact, these results are larger in magnitude than the primary results).

I also consider how the results change if we change the assumed "start date" of the epidemic by four years in either direction (either assume the epidemic starts 4 years earlier or 4 years later). These estimates are shown in the third and fourth rows of Table 6. Again, these changes make little difference to the results, for either the UNAIDS data or the mortality-based data.

Overall, the results in Table 6 indicate that, while the *exact* magnitude of the relationship between HIV and exports can be somewhat affected by the assumptions about generating incidence, the general conclusions of this section are robust to changes in the important elements of the data generation.[8]

[8]It is worth noting that there are a number of other assumptions that go into generating the mortality-based data, which do not affect the UNAIDS data. For example, due to noise in the death data we do some smoothing over years, requiring the choice of a particular smoothing parameter. Oster (2008) discusses in more detail the robustness of the estimates to these assumptions.

5 Mechanism: Trucking and People-Movement

As discussed, there are a number of mechanisms by which exports could affect HIV infections. One channel is through the increased movement of people. More exports mean both more production, which may use migrant or other temporary labor, and more trucking is necessary to move things from the place of production to cities or ports. A second channel is through income; if more exports means more income, and sex is a normal good, this could also drive the effect. In this section I provide some preliminary evidence suggesting that trucking and transit may play a role in mediating this relationship. However, fully separating out what mechanisms are important, or making a strong causal argument that trucking or movement of people is a key element driving the relationship, is beyond the scope of the analysis in this paper.

Truckers and Sex; Trucks and Exports The first argument here is simply to note that truckers have more sex than individuals in the general population, and more exports means more trucking. The relationship between truckers and sex is well known in existing work and, in fact, existing evidence points to a number of ways in which trucking and HIV are linked. First, truck drivers and other migrants (i.e. those who spend time living or traveling away from home) tend to have more sexual partners than the average in the population (Lurie et al, 2003a; Brewer et al, 1998; Brockerhoff and Biddlecom, 1999; Anarfi et al, 1997; Anarfi, 1993; Orubuloye, Caldwell and Caldwell, 1993). Second, the sexual partnerships these people have away from home tend to be higher risk than those they have at home, largely because their partners are more likely to be infected: for example, they are more likely to be bar girls or commercial sex workers (Orubuloye et al, 1993). Finally, the partners (for the most part, wives) of those who travel may be more likely to have additional sexual partners while their spouses are away (Lurie et al, 2003b). The level of risky behavior is significant; in a survey in Nigeria, Orubuloye et al (1993) find that the truck drivers in the sample have an average of 12 non-marital partners a year.

This existing evidence suggests a link between trucking and sex. Panel A of Table 7 demonstrates the second step: more exports mean more trucking. This analysis uses data from the World Development Indicators and regresses the weight of goods trucked within a year on exports that year, with country and year fixed effects and a control for GDP. Column 1 includes only the counters in our sample for which this data is available; this is unfortunately a very small set (just 24 country-years). However, the coefficient is positive and strongly significant. Column 2 includes all low and lower-middle income countries with available data and, in this larger sample, also shows a significant positive relationship.

Taken together, these two facts suggest a role for trucking in mediating this relationship. If having more exports means there are more trucks, and truckers have more sex when they are away from home then not, these together imply that an increase in exports should increase risky sex. It is, of course, important to note that this does not prove the connection, and there are certainly models of behavior which would not be consistent with this. For example, if the *marginal* truck driver does not have a lot of risky sex, then the increased trucking would not obviously increase risk.

Variations across Countries: Roads As perhaps a more direct test, we can take advantage of variation across countries in our sample in road availability. To the extent that an important mechanism here is trucking or movement of people, we would expect the effect of exports to be stronger in areas where increases in economic activity will have a larger effect on these variables. This is likely to be more true in countries with more roads. Put differently, if we see an equally strong relationship between exports and HIV in countries with no roads, or very limited roads, this would suggest that trucking is *not* an important mechanisms. Conversely, if we see a stronger relationship in countries with extensive road networks, this could point toward the importance of transit and movement in mediating the export-HIV relationship.

Data on roads come from the World Development Indicators. I use two measures: total kilometers of roads divided by land area and total kilometers of paved roads, divided by land area. These measures do not vary much over time, so I use a constant value per country, the average of these variables over the entire period. I estimate the primary specifications (country and year fixed effects, GDP controls), but include an interaction between the road measures and exports, as well as the level of exports (the control for road level is captured in the country fixed effect).

These regressions are shown in Panel B of Table 7. Focusing first on the UNAIDS data, in Columns 1 and 2, we find that for roads overall, and for paved roads, the level effect of exports in these regressions is small and not significant, but the interaction is large, positive and significant. In other words, the relationship is much larger in areas where there are more roads, pointing to the importance of transit in this relationship. These results are similar if we simply divide the sample in half based on road density and estimate the relationship for the two halves of the sample separately – there is a positive and significant relationship for countries in the top half, and no relationship for those in the bottom (results available from the author). Columns 3 and 4 show this relationship in the mortality-based data; we see the same patterns, although the results are only significant for paved roads. This is likely due to the more limited set of countries in these data and, hence, the more limited variation in road density.

Variations across Countries: Ports Finally, in a test related to the above, I can explore whether the relationship between exports and HIV is stronger in countries that are more reliant on road transportation. In particular, we separate the countries in the sample into two groups: those with ports, which we posit are less reliant on roads for goods transport, and those without. Since most of the commodities produced by these countries will eventually be transported by ship out of Africa, for countries without a port there is significant additional trucking time necessary to export goods. To the extent that trucking is a mediating factor here, we would expect the export-HIV relationship to be larger in areas with no port.

Panel C of Table 7 shows these regressions, dividing the samples into countries with a port and those without. For both data sources the relationship is much larger, and more significant, for countries *without* a port, consistent with the theory above. Of course, there could be other factors which vary across coastal and non-coastal countries, and play into the export-HIV relationship. This evidence is supportive of a role for a transit mechanism but, again, not conclusive on its own.

The evidence in this subsection certainly does not prove that trucking is an important mechanism behind the HIV-export relationship. All of the evidence, however, is at least supportive of this case. In addition to shedding light on this mechanism, this evidence further supports the claim that the relationship between exports and HIV is causal. If an omitted variable like government policy was driving this result it seems unlikely we would see, for example, a difference between cities with ports and those without.

6 Application: Prevalence Declines in Uganda

The previous sections provides evidence that, at the macro level, new HIV infections and economic activity are linked. This section considers whether that link may be at least partially responsible for one of the more striking patterns in HIV prevalence in Africa over the 1990s.

HIV prevalence declined sharply in Uganda in the early 1990s, a decline unique among Sub-Saharan African countries (this is apparent in both the UNAIDS data and in the mortality-based estimates). This decline is generally attributed to anti-HIV educational efforts, including those by the Uganda National AIDS Program (NACP) and the ABC campaign, both of which encouraged changes in sexual behavior: partner reduction, “zero-grazing”, abstinence and condom use (Green, 2004; Green et al, 2006; Slutkin et al, 2006).[9] Among the more important components of the educational efforts were in-school education, communication by faith-based organizations, billboards and other forms of public advertising (for a good summary of the specific aspects of ABC, see Green, 2004; Slutkin et al, 2006 provide a summary of non-ABC partner reduction efforts like NACP). Recent work has argued that changes in concurrency as a result of this program were important in the decline in HIV (Epstein, 2007).

Uganda was among the first countries to address the epidemic head on and the subsequent decline in prevalence was seen as a signal of the success of the behavioral education approach. Subsequently, a number of other countries adopted this type of prevention strategy (Kenya, Tanzania, Rwanda, Malawi and others). Outside aid groups have pursued similar interventions. PEPFAR, in particular, has adopted this campaign as a major part of their anti-HIV strategy: roughly one hundred million dollars of their funding was spent on ABC programs in 2004.

However, in the early 1990s Uganda also saw a major decline in exports. This was likely due in large part to a decline in coffee prices and production.[10] Figure 3 demonstrates that the decline in export value lines up closely with the changes in incidence in Uganda; this is true in both the UNAIDS data and the mortality-based estimates.[11]

Given the relationship estimated in Section 4, the decline in export volume in Uganda during this period suggests that HIV incidence would have declined somewhat even without any education campaign. Using the coefficient estimates of the relationship between exports and HIV incidence, along with data on the magnitude of the decline in exports, we can calculate how large we would expect the decline in incidence to be based on the export decline alone. We can compare that with the actual magnitude of the decline, and estimate what share of the Ugandan "miracle" appears to be explained by exports.

The results of this calculation are shown in Table 8. Note that the coefficients I use in this calculation are based on regression estimates *without* Uganda, so this can be seen as an out of sample prediction. The share of the decline (from peak to trough) which is explained by exports

[9] Although there is a fair amount of agreement on the success of education in general in Uganda (although see Gray et al, 2006), there is less agreement on the relative success of the partner reduction elements, versus ABC specifically, and within the ABC on the various merits of A versus B versus C; for a summary and citations on this controversy, see Green et al, 2006.

[10] This change in exports did not reflect a collapse of the Ugandan economy in general. It was driven by a world coffee price shock, which was caused by factors outside of Uganda, and decreased exports.

[11] The UNAIDS data is somewhat noisy. This is driven by the fact that in the antenatal clinic data for the 1980s, UNAIDS reported a 13% HIV rate in 1986, but 24% in 1987 and then 25% in the next year. The increase during this period was surely much smoother than this, and if properly represented would likely look much more like the mortality-based data.

ranges between 35 and 50%, depending on which measure of incidence is used. It is worth noting that the changes in exports over time also match with the increase in incidence seen in the mortality-based data in the 1993-1995 period, an increase which is reflected in some other literature suggesting an increase in prevalence toward the end of the decade (Shafer et al, 2006). This increase is harder to explain with the education campaign, which continued at similar levels (if anything, increased) during this period.

It is important to note, of course, that the decline in exports certainly does not explain all of the decline in prevalence in Uganda in the 1990s; there is still a role for changes in the choices of sexual behavior. Further, in some sense the decline due to exports comes from the same underlying source as the decline due to an education campaign: a drop in sexual behavior. The crucial distinction between the two, however, is that the success of an education campaign lies in changing individual (unconstrained) *choice* of non-marital partners, whereas the "success" of a decline in exports lies in changing individual *opportunities* for such relationships.

This result may have important implications for overall HIV policy in Africa. If this education campaign was half as successful as previously thought, there may well be other polices which overtake it in terms of cost-effectiveness. In particular, policies that seek to change viral transmission rates (for example, male circumcision) may appear much more attractive in light of these findings. Funding for HIV in Africa is, unfortunately, too limited to make all types of interventions feasible simultaneously. Understanding the relative cost-effectiveness of various inputs is therefore crucial to formulating optimal policy.

7 Conclusion

This paper addresses the connection between HIV and economic activity, arguing that increases in economic activity significantly increase HIV incidence. This result – particularly the magnitude – may have important implications for HIV prevention. Interventions that target those involved in trucking, or those more generally targeted at migrants, may have an even larger effect than previously expected. Further, HIV is only one focus of development aid to Africa. Significant amounts of aid are also spent trying to increase trade. Policies like the African Growth and Opportunity Act (AGOA) are designed specifically to encourage exports of African textiles, and smaller efforts, like those made by Fair Trade, also aim to increase trade and contact with the West. The results here suggest that, while those policies may well improve trade, they may actually make the HIV epidemic worse. Combining other prevention efforts (male circumcision, for example) with trade policies may ameliorate some of these interaction concerns.

It is important to note in interpreting all of the results in the paper that I estimate the *short-term* effect of changes in exports on HIV incidence, within a given country. This is distinct from an estimate of the long-term effect of exports: in the long run, higher levels of economic activity are likely to translate into economic growth. As individuals become richer the epidemic may get worse (if money buys sex), or better (if richer people have more incentives to avoid HIV, or more information to do so (Oster, 2007)). Neither of these forces will be captured here. In addition, the estimate here is distinct from estimates of what determines cross-country patterns in HIV. Africa has, on average, lower levels of exports than much of the rest of the world, and higher HIV, and variations in HIV across countries within Africa do not always line up with aggregate variations in exports. This clearly points to some other factor – for example, circumcision or overall health – which drives HIV, and is not captured here. Although the estimates here may point to an important role for exports in explaining within country variations – they by no means explain all of the cross-country or cross-region variation.

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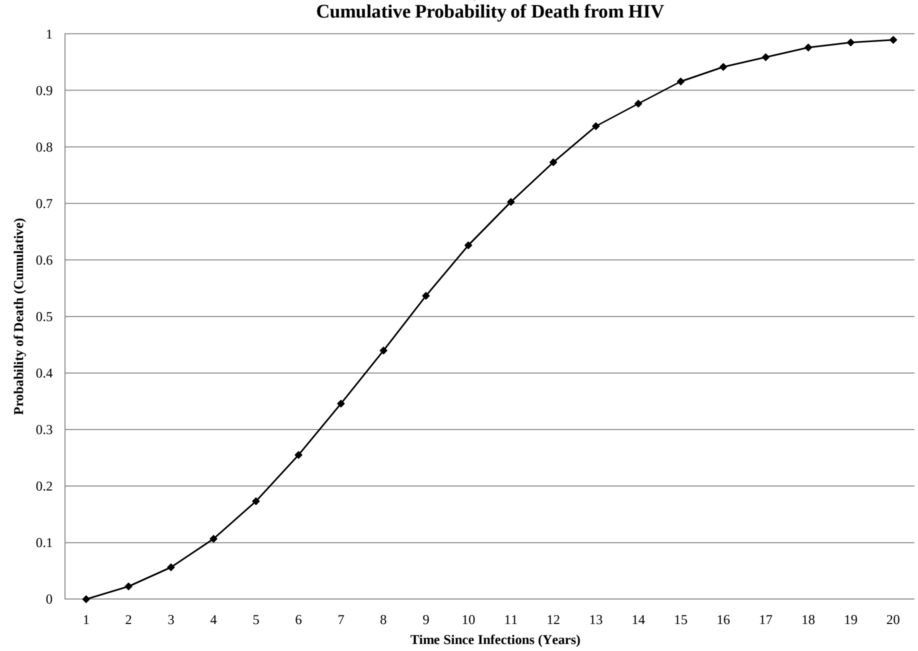
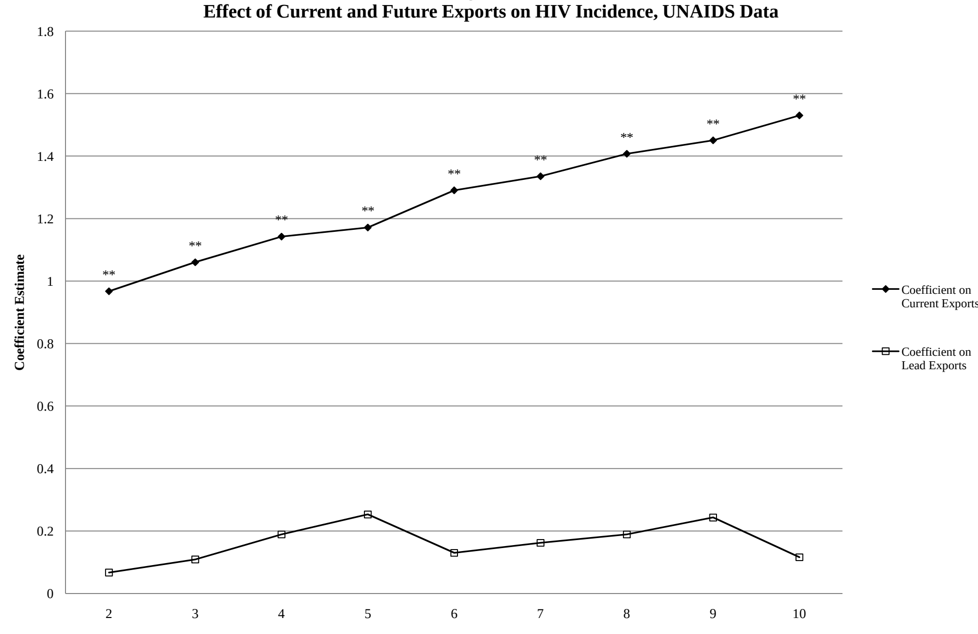
Figure 1:
Cumulative Probability of Death from HIV



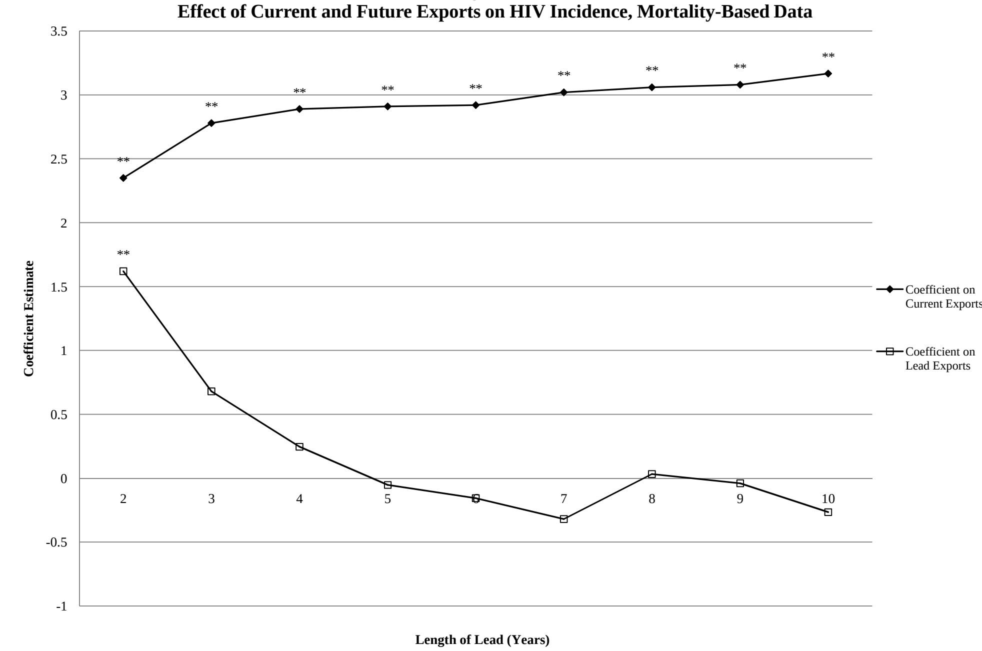
Notes: Figure presents the cumulative probability of death as a function of years from infection with HIV. sThe figure is based on data from Collaborative Group on AIDS Incubation and HIV Survival, 2000.

Figure 2a:
Effect of Current and Future Exports on HIV Incidence, UNAIDS Data



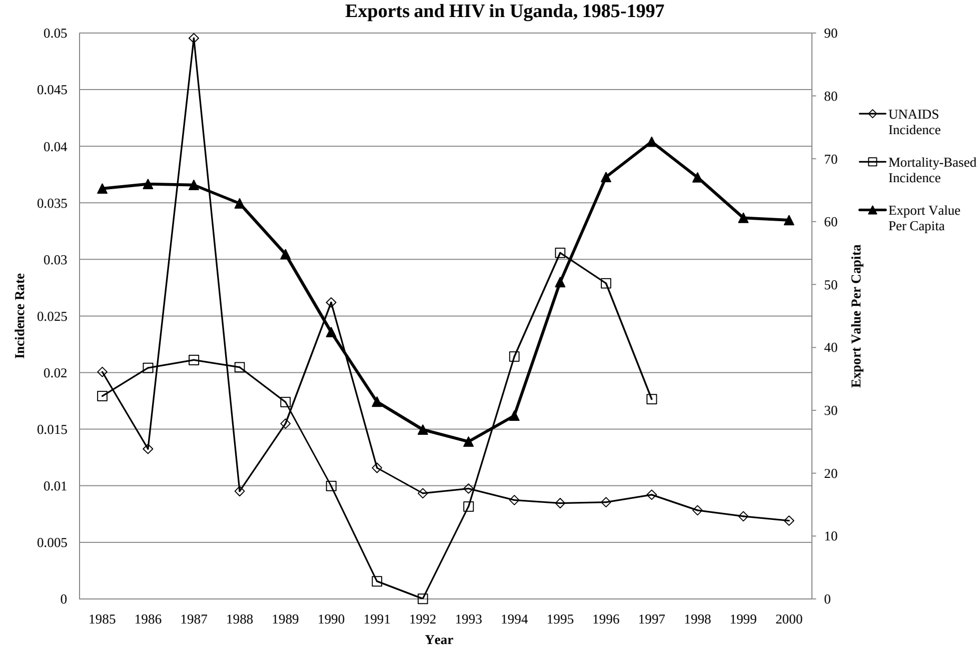
Notes: This table shows coefficients on current and future exports from regressions of the form in Column 2 of Table 1. The coefficients come from a total of nine regressions, each of which includes current exports and exports at a given lead length. ** significant at 5% level.

Figure 2b:
Effect of Current and Future Exports on HIV Incidence, Mortality-Based Data



Notes: This table shows coefficients on current and future exports from regressions of the form in Column 4 of Table 1. The coefficients come from a total of nine regressions, each of which includes current exports and exports at a given lead length. ** significant at 5% level

Figure 3:
Exports and HIV in Uganda, 1985-1997



Notes This figure shows exports and HIV incidence measures for Uganda for the period from 1985 to 1997.

Table 1. ***Summary Statistics on Trade, Controls***

Panel A: Trade Measures			
	Mean	**Std. Dev.**	**Number of Obs**
Export Value Per Capita, WDI	$609	$1102	766
Major Exp. Value Per Capita, NBER	$478	$1205	584
Major Exp. Volume Per Capita, NBER	2.22	9.65	457
Import Value Per Capita, WDI	$595	$814	916
Panel B: Controls			
	Mean	**Std. Dev.**	**Number of Obs**
GDP per capita	$704	$1010	766
Yearly Temperature	24.1	3.5	566
Yearly Rainfall	11.6	5.1	566

Notes: This table shows summary statistics on the trade measures and control variables. Summary statistics on HIV measures, by country, are in Appendix A.

Table 2. *HIV Incidence and Exports*

Dependent Variable:	*Log HIV Incidence Rate UNAIDS Data*			*Log HIV Incidence Rate Mortality-Based Data*		
	(1)	(2)	(3)	(4)	(5)	(6)
Explanatory Variables:						
Log Exports Per Cap. (Avg of year t, t-1)	.7765***	.7982***	1.2752***	2.6301**	2.8816***	2.7057***
	(.234)	(.277)	(.281)	(1.09)	(.631)	(.639)
UNAIDS Rate, year t-1	.6177	.6794	−2.5677			
	(2.411)	(2.602)	(2.802)			
Mortality-Based Rate, year t-1				−1.0765	−1.4737	−2.0576
				(7.825)	(8.121)	(10.604)
Log GDP Per Capita		−.0997	−.1384		−1.2725	−1.238
		(.787)	(1.007)		(2.771)	(2.878)
Year Avg. Temperature			.3873*			.2865
			(.23)			(.411)
Year Avg. Precip.			.0207			−.0193
			(.036)			(.086)
Country FE	YES	YES	YES	YES	YES	YES
Year FE	YES	YES	YES	YES	YES	YES
Number of Observations	752	752	552	160	160	151
R^2	.74	.74	.77	.64	.65	.65

standard errors in parenthesis, clustered by country

* significant at 10%; ** significant at 5%; *** significant at 1%

Notes: This table shows the relationship between HIV incidence and exports. Columns 1-3 use data on HIV incidence rate derived from UNAIDS data; Columns 4-6 use estimates based on inference from mortality data (Oster, 2008). Countries in each dataset are listed in Appendix A. The export measure is log exports per capita, from the World Development Indicators, averaged for the current year and previous year. The GDP measure is drawn from the World Development Indicators, and temperature and rainfall data are taken from Dell, Jones and Olken (2008).

Table 3. ***HIV Incidence and Exports, by Prevalence***

Dependent Variable:	*Log HIV Incidence Rate UNAIDS Data*		*Log HIV Incidence Rate Mortality-Based Data*	
	Low HIV	*High HIV*	*Low HIV*	*High HIV*
	(1)	(2)	(3)	(4)
Explanatory Variables:				
Log Exports Per Cap. (Avg of year t, t-1)	.7335**	1.1695**	1.9216***	4.2296***
	(.281)	(.457)	(.564)	(.827)
UNAIDS Rate, year t-1	−37.98*	4.75*		
	(21.95)	(2.728)		
Mortality-Based Rate, year t-1			45.7962**	−1.9323
			(18.841)	(6.135)
Log GDP Per Capita	−.5722	.3125	−1.1179	3.7616
	(.637)	(1.197)	(1.644)	(2.717)
Country FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
Number of Observations	375	377	84	76
R^2	.69	.64	.79	.66

standard errors in parenthesis, clustered by country
* significant at 10%; ** significant at 5%; *** significant at 1%
Notes: This table shows the relationship between HIV incidence and exports, separated by country-level HIV rate. Columns 1 and 2 use data on HIV incidence rate derived from UNAIDS data; Columns 3 and 4 use estimates based on inference from mortality data (Oster, 2008). Countries in each dataset are listed in Appendix A. The export measure is log exports per capita, from the World Development Indicators, averaged for the current year and previous year. Columns 1 and 3 include countries whose *average* HIV rate over the sample is low; columns 2 and 4 include countries with high average HIV rate.

Table 4. ***HIV Incidence and Exports: Alternative Trade Measures***

Panel A: UNAIDS Incidence			
Dependent Variable: Log HIV Incidence Rate, UNAIDS Data			
Explanatory Variables:			
Log Major Export Value Per Cap.	.718*		
	(.365)		
Log Major Export Volume Per Cap.		.331	
		(.242)	
Log Import Value Per Cap.			.722***
			(.207)
UNAIDS Rate, year t-1	−.062**	−.065*	.009
	(.031)	(.034)	(.027)
Log GDP Per Capita	.574	.625	.409
	(.513)	(.568)	(.763)
Country FE	YES	YES	YES
Year FE	YES	YES	YES
Number of Observations	461	421	752
R^2	.75	.73	.73

Panel B: Mortality-Based Incidence			
Dependent Variable: Log HIV Incidence Rate, Mortality-Based Data			
Explanatory Variables:			
Log Major Export Value Per Cap.	.9834		
	(.799)		
Log Major Export Volume Per Cap.		1.3816*	
		(.693)	
Log Import Value Per Cap.			1.9802*
			(1.079)
Mortality-Based Rate, year t-1	−1.3926	.8007	−8.0373
	(8.361)	(7.748)	(7.736)
Log GDP Per Capita	−.1478	−1.3185	−.1831
	(2.572)	(2.367)	(2.935)
Country FE	YES	YES	YES
Year FE	YES	YES	YES
Number of Observations	160	160	160
R^2	.59	.59	.59

standard errors in parenthesis, clustered by country

* significant at 10%; ** significant at 5%; *** significant at 1%

Notes: This table shows the relationship between HIV incidence and alternative measures of trade. Panel A uses HIV incidence derived from the UNAIDS data; Panel B uses estimates based on inference from mortality data (Oster, 2008). Major exports are defined by country as the top export only if that export accounts for more than 50% of trade volume; otherwise it is the top two exports. All trade measures are an average of the current year and previous year. Volume and value data come from the Feenstra et al (2004); import data is from the World Development Indicators.

Table 5. *HIV Incidence and Exports, Causality Tests*

	Country-Specific Trends		IV Regressions	
Dependent Variable:	*Log HIV Incidence Rate*		*Log HIV Incidence Rate*	
	UNAIDS Data	*Mortality Data*	*UNAIDS Data*	*Mortality Data*
	(1)	(2)	(3)	(4)
Explanatory Variables:				
Log Exports Per Cap.	.5922***	3.5864***	1.076*	2.675**
	(.168)	(1.097)	(.589)	(1.25)
UNAIDS Rate, year t-1	−8.5863***		−3.564	
	(2.509)		(2.798)	
Mortality-Based Rate, year t-1		−8.871		−4.087
		(13.06)		(7.132)
Log GDP Per Capita			−.4747	−1.283
			(1.485)	(2.794)
Country FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
Country-Specific Trends	YES	YES	NO	NO
Instruments F-Stat			4.94	5.39
Number of Observations	752	160	565	160
R^2	.88	.77	.69	.65

standard errors in parenthesis, clustered by country

* significant at 10%; ** significant at 5%; *** significant at 1%

Notes: Columns 1 and 2 of this table replicate Table 2 but include country-specific trends. Columns 3 and 4 run similar regression but instrument for exports with prices of the country's major commodities. The measure of exports is an average of the current year and previous year's exports per capita. The instruments are the world commodity prices of the country's major exports, from the IMF statistics; we instrument with price this year, last year and two years ago. These regressions are weighted by the share of export value made up by the major commodities. Major exports are defined by country as the top export only if that export accounts for more than 50% of trade volume; otherwise it is the top two exports.

Table 6. ***HIV and Exports, Robustness to Assumptions***

Change in Assumptions	*Result with UNAIDS Data*	*Result with Mortality-Based Data*
Faster Death	.7976***	3.169***
	(.274)	(.613)
Flatter Death	.7977***	3.311***
	(.277)	(.644)
No Death After 2002	.9038***	
	(.294)	
Earlier Start Date	.7748***	2.251***
	(.278)	(.670)
Later Start Date	.8493***	3.441***
	(.282)	(.899)

Notes: This table replicates Columns 2 and 4 of Table 2, using alternative assumptions in the generation of HIV incidence. We report here only the coefficients on log exports per capita (average of current and previous year); other controls include GDP, country fixed effects and year fixed effects. The assumptions about time to death can be seen visually in Appendix Figure 1. Earlier start date is four years before the primary start date; later start date is four years later.

Table 7. *Mechanism Tests: Trucking, HIV and Exports*

Panel A: Exports and Trucking, World Development Indicators				
Dependent Variable	*Goods Transported (Mill. Tons/Km)*			
	Sub-Saharan Africa	All Low Income		
Explanatory Variables:				
Export Value (in $Millions)	1.1959***	.1829***		
	(.129)	(.061)		
GDP (in $Millions)	−.734***	.2459***		
	(.088)	(.037)		
Country FE	YES	YES		
Year FE	YES	YES		
Number of Observations	24	87		
R^2	.99	.99		
Panel B: Export, Road Density Interactions				
Dependent Variable:	*Log HIV Incidence Rate UNAIDS Data*		*Log HIV Incidence Rate Mortality-Based Data*	
Explanatory Variables:				
Log Exports Per Cap.	.3563	.4662	1.8203	1.8113*
	(.351)	(.36)	(1.149)	(1.012)
Exports × Roads/Area	3.38***		6.6495	
	(1.238)		(4.296)	
Exports × Paved Roads/Area		17.454*		31.9262**
		(8.882)		(14.832)
Log GDP Per Capita	−.0523	−.0472	−.6981	−.5633
	(.817)	(.819)	(2.801)	(2.715)
Lagged HIV Rate	YES	YES	YES	YES
Country FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
Number of Observations	752	752	160	160
R^2	.75	.75	.66	.67
Panel C: Export Effect in Countries with Port, Without Port				
Dependent Variable:	*Log HIV Incidence Rate UNAIDS Data*		*Log HIV Incidence Rate Mortality-Based Data*	
	With Port	Without Port	With Port	Without Port
Explanatory Variables:				
Log Exports Per Cap.	.4074	.9638**	2.0739***	3.8201***
	(.383)	(.371)	(.298)	(.677)
Log GDP Per Capita	1.2102	−.5449	−.8221	2.9457
	(.994)	(.846)	(.811)	(2.88)
Lagged HIV Rate	YES	YES	YES	YES
Country FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
Number of Observations	421	331	56	104
R^2	.76	.8	.95	.71

standard errors in parenthesis, clustered by country; * significant at 10%; ** significant at 5%; *** significant at 1%
Notes: This table shows several tests of whether trucking or a transit mechanism is driving the results. In Panel A, poor countries include all low and lower-middle income countries that have this data in the World Development Indicators. In Panel B, road density is defined as kilometers of roads per square mile.

Table 8. ***Explaining the Ugandan Incidence Decline***

	Decline in Exports 1987 to Early 1990s (92-93)	Coeff. on Exports	Actual Decline in HIV 1987 to Early 1990s (92-93)	% Expl. by Exports
UNAIDS Data	0.930	0.672	1.647	**37.9%**
Mortality Data	0.930	2.415	4.259	**52.7%**

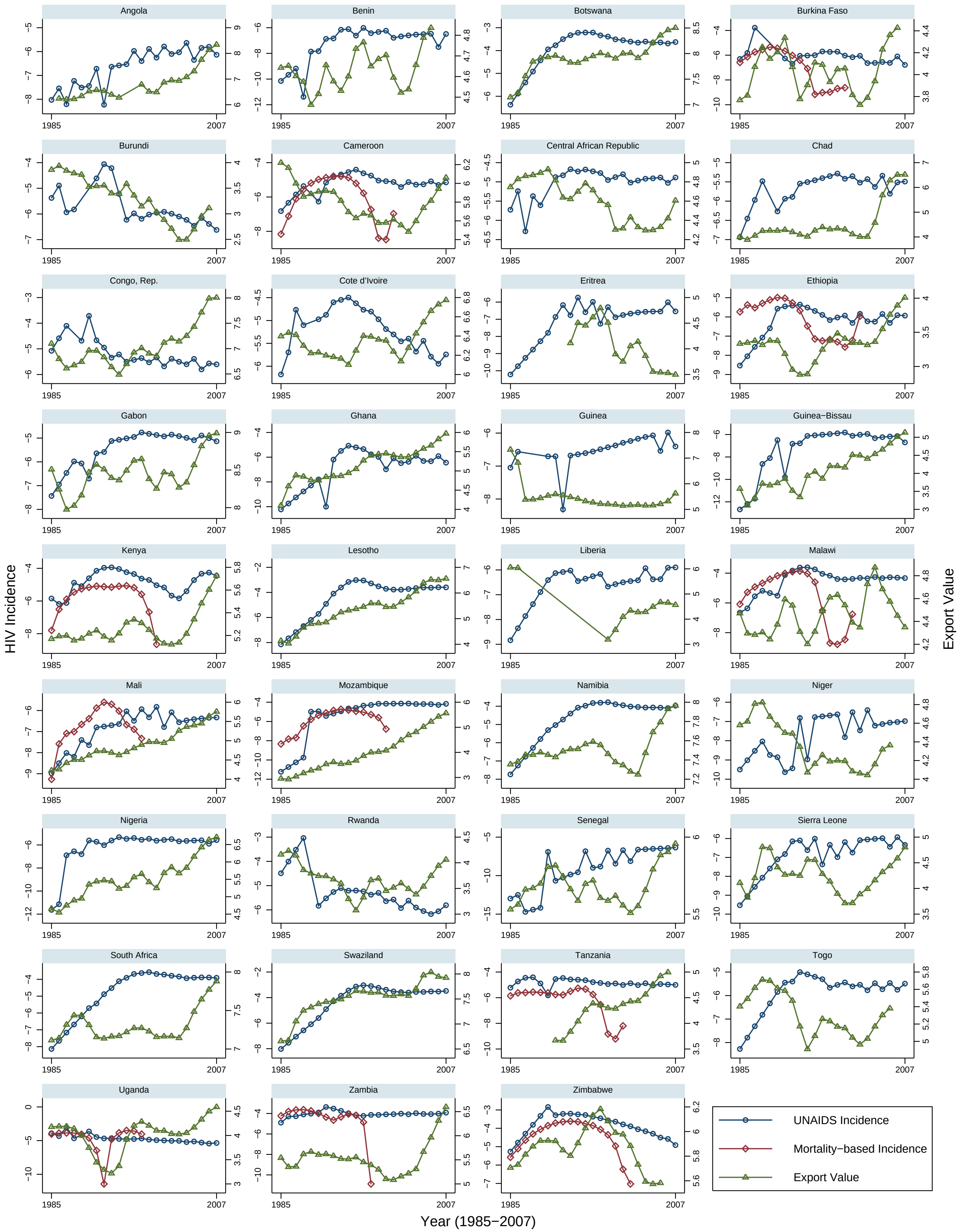
Notes: This table shows the share of the Ugandan incidence decline that can be accounted for by changes in export volume or value. The coefficient is from regressions like those in Table 2, but excluding Uganda from the analysis. % Explained is simply the product of the decline in exports and the coefficient on exports, divided by the actual decline in HIV.

Appendix A: Summary Statistics on HIV Measures

The table below reports summary statistics on average prevalence and incidence for each country in the sample, over the period covered in the data used for analysis (1985-2007 for the UNAIDS data; 1985-late 1990s or early 2000s for the mortality-based data).

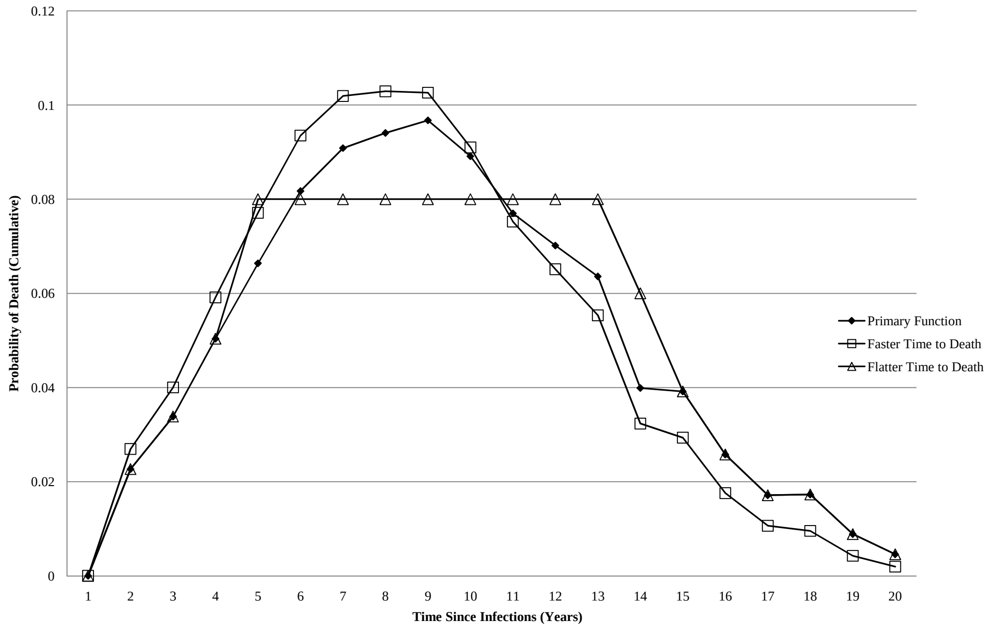
Country	**UNAIDS Avg. Prevalence**	**UNAIDS Avg. Incidence**	**Mortality-Based Avg. Prevalence**	**Mortality-Based Avg. Incidence**
Angola	0.0100	0.0017		
Benin	0.0076	0.0012		
Botswana	0.1635	0.0248		
Burkina Faso	0.0187	0.0025	0.0198	0.0019
Burundi	0.0321	0.0040		
Cameroon	0.0379	0.0056	0.0291	0.0041
Central African Republic	0.0443	0.0066		
Chad	0.0214	0.0033		
Congo, Rep.	0.0437	0.0056		
Cote d'Ivoire	0.0424	0.0057		
Eritrea	0.0076	0.0012		
Ethiopia	0.0167	0.0025	0.0392	0.0030
Gabon	0.0334	0.0053		
Ghana	0.0135	0.0020		
Guinea	0.0078	0.0013		
Guinea-Bissau	0.0098	0.0016		
Kenya	0.0657	0.0096	0.0265	0.0041
Lesotho	0.1351	0.0215		
Liberia	0.0101	0.0016		
Malawi	0.0879	0.0131	0.0810	0.0089
Mali	0.0086	0.0014	0.0138	0.0016
Mozambique	0.0594	0.0101	0.0287	0.0043
Namibia	0.0812	0.0132		
Niger	0.0042	0.0007		
Nigeria	0.0198	0.0031		
Rwanda	0.0594	0.0070		
Senegal	0.0030	0.0006		
Sierra Leone	0.0088	0.0015		
South Africa	0.0895	0.0149		
Swaziland	0.1414	0.0229		
Tanzania	0.0592	0.0082	0.0251	0.0029
Togo	0.0232	0.0035		
Uganda	0.0947	0.0113	0.1139	0.0170
Zambia	0.1263	0.0182	0.1182	0.0161
Zimbabwe	0.1895	0.0251	0.1063	0.0152

Appendix B: HIV Infections and Exports, by Country



Appendix Figures and Tables

Appendix Figure 1:
Cumulative Probability of Death from HIV, Alternative Assumptions



Notes: This figure presents the yearly probability of death under the primary time-to-death function and the two variations explored in the robustness section.

Appendix Table 1. ***HIV Incidence and Exports, Excluding Individual Countries***

Measure of HIV Incidence:	*UNAIDS Data*	*Mortality-Based Data*
Excluded Country		
Angola	.815*** (.287)	
Benin	.804*** (.277)	
Botswana	.778** (.294)	
Burkina Faso	.762** (.289)	2.802*** (.717)
Burundi	.750** (.284)	
Cameroon	.799*** (.280)	3.284*** (.536)
Central African Republic	.805*** (.283)	
Chad	.916*** (.265)	
Congo, Rep.	.887*** (.263)	
Cote d'Ivoire	.815*** (.275)	
Eritrea	.865*** (.286)	
Ethiopia	.807*** (.278)	2.811*** (.722)
Gabon	.807*** (.279)	
Ghana	.788*** (.278)	
Guinea	.827** (.337)	
Guinea-Bissau	.675** (.280)	
Kenya	.798*** (.277)	2.969*** (.626)
Lesotho	.774** (.293)	
Liberia	.805*** (.289)	
Malawi	.809*** (.282)	2.946*** (.598)
Mali	.805*** (.279)	2.876*** (.655)
Mozambique	.719** (.269)	2.588*** (.864)
Namibia	.820*** (.280)	
Niger	.819*** (.282)	
Nigeria	.771*** (.284)	
Sao Tome and Principe	.798*** (.277)	
Senegal	.875*** (.265)	
Sierra Leone	.801*** (.286)	
South Africa	.814*** (.279)	
Swaziland	.779*** (.282)	
Tanzania	.825*** (.277)	3.280*** (.383)
Togo	.807*** (.283)	
Uganda	.672** (.275)	2.415*** (.800)
Zambia	.812*** (.278)	3.027*** (.630)
Zimbabwe	.788 *** (.275)	2.794*** (.650)

standard errors in parentheses, clustered by country.
* significant at 10%; ** significant at 5%; *** significant at 1%
Notes: This table replicates Columns 2 and 4 of Panel A of Table 2, with each row excluding one country. Fewer countries overall are available in the mortality-based data.