

https://dspace.org/

Unimagined community: sex, networks and AIDS in Uganda and South Africa

Thornton, Robert

2007-06-01
University of California Press
http://hdl.handle.net/10539/3889

Downloaded from a DSpace repository. https://dspace.org/

2

COMPARING HIV-PREVALENCE TRENDS IN UGANDA AND SOUTH AFRICA: SEXUAL NETWORKS, FAMILY STRUCTURE, AND PROPERTY

INTRODUCTION

South Africa and Uganda have been at the centre of attention in the HIV/AIDS pandemic.[1] The first evidence of the HIV epidemic in Africa emerged in Uganda in the early 1980s.[2] It is the only African country that has shown an overall and sustained decrease in prevalence.[3] By contrast, South Africa was forced to deal with the epidemic only in the 1990s, and today has one of the world's highest HIV prevalence. Since 1992, HIV trends in the two countries have moved strongly and consistently in opposite directions. This cannot be due simply to levels of sexual activity since Uganda had a total fertility rate (TFR) of 6·9 in 2001[4] or 7.1 in 2005,[5] the highest total fertility rate in east and southern Africa and roughly stable since 1955 at least. South Africa's fertility has been relatively low and declining[6], with a TFR of 2.7 in 2005,[7] the lowest in Africa.

The explanation for this lies in differing *configurations of sexual networks* in the two countries. To demonstrate this, this chapter develops a mathematical model of prevalence trends and shows how this allows us to see differences in the topology of sexual networks. (A certain amount of mathematics is required here, but this is separated from the rest of the test and the reader who is not interested in the mathematics can skip over these sections.) The following chapter shows how sexual networks are embedded in and shaped by differing social contexts, especially with respect to marriage, reproduction, the development of households and the transfer of wealth and property. Together, these approaches help to explain the radical differences between HIV trends in Uganda and South Africa.

It is clear that sexual networks are responsible for transmitting the virus in almost all adult cases. It is also increasingly clear that the configuration and dynamics of sexual networks—such as their periodicity or timing, their density, their randomness or clustering—are as important as individual sexual behaviour in understanding the dynamics of HIV transmission, incidence

---

[1] Commission for Africa, 2005, Ch.6, para. 6

[2] Serwadda, Sewankambo,Carswell et al 1985. The origins of HIV, however, have been traced to areas farther to the west, in northern Democratic Republic of Congo (Zhu, Korber & Nahinias 1998).

[3] Stoneburner & Low-Beer 2004: 715; Assamoah-Odei, Calleja & Boerma 2004.

[4] Measure DHS 2002.

[5] UNICEF; online: http://www.unicef.org/infobycountry/uganda_statistics.html

[6] The South African total fertility rate (TFR) in 1970 was 5.6; in 1990, 3.6 and in 2005, 2.7 (UNICEF. Online http://www.unicef.org/infobycountry/southafrica_statistics.html)

[7] South African Institute of Race Relations 2003: 30.

and prevalence.[8] For instance, it has been estimated that newly infected people with high levels of HIV in their blood are up to 10 times more likely to infect others.[9] This estimate is based on research by Maria Wawer and her team on a sample of 10,000 people ages 15 to 49 who live in 44 villages near Uganda's border with Tanzania. If such people have sexual contact with multiple partners during this time, they become extremely efficient transmitters of HIV in the overall sexual network.[10] The efficiency with which HIV is transmitted in sexual networks is highly sensitive to its periodicity and to the number of links (sexual contacts) between participants.

The concept of *sexual networks*, as it is developed here, introduces a social dimension into research that has, for the most part, focused previously on the individual (knowledge and attitudes) and on behaviour and practices. This involves a change in the *scale of analysis*: large-scale social formations as compared to small-scale individual behaviour. It also involves a conceptual shift towards a social-epidemiological model that is able to accommodate society-level structures rather than simply statistical aggregates of individual behaviour in so-called 'populations'. If we take sexual relations to be *social relations* (not just 'behaviour'), and sexual networks to be types of *social structures*, we can see that HIV is transmitted at the population or national level by specific types of large-scale social structures, that is, sexual networks.

This approach constitutes a significant methodological departure from the standard statistical medical-epidemiological approach, and from those social-scientific approaches to HIV/AIDS that focus on the individual.[11] This can be seen by comparing the 'standard' epidemiological model shown in Figure 2.1, produced by Rand Stoneburner and Daniel Low-Beer[12] with the model based on power-law curves in Figure 2.3. In the first instance, Figure 2.1, Stoneburner and Low-Beer use a Gaussian or 'bell-curve' model to approximate the empirical data. This model probably represents the best fit between the observed data (all from Kampala, drawn from the principal antenatal sentinel surveillance sites at Rubaga, Nsambya and Mulago hospitals) and the standard epidemiological model.

---

[8] Morris, Sewankambo, Wawer, Serwada & Lainjo 1995; Morris 1997; Rothenberg, Potterat, Woodhouse, Muth Darrow & Klovedahl 1998; Rothenberg, Long, Sterk, Pach, Potterat, Muth et al., 2000; Aral 1999; Liljeros, Edling, Amaral, 2003; Wohlfeiler, Potterat, 2003; Low-Beer & Stoneburner, 2003.

[9] Brown 2005.

[10] I use the terms 'transmission' and 'transmitters' here rather than 'infectivity' or 'infective agents' in other to emphasise the differences between 'transmission' of pathogens through a connected network of susceptible person and the 'random' infection of populations by pathogens that travel through air, water or food resources as most other pathogens are.

[11] The standard epidemiological model is presented clearly and simply in Gouws & Abdool Karim 2005.

[12] Stoneburner & Low-Beer 2004.

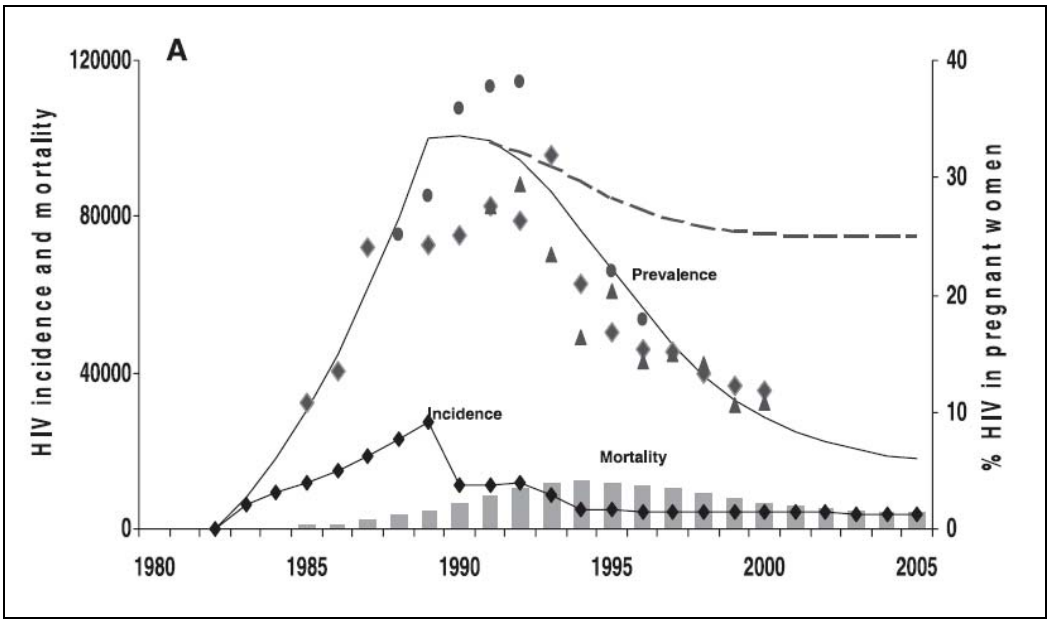


Figure 2.1. Stoneburner and Low-Beer's epidemiological model of HIV prevalence change in Uganda (Stoneburner & Low-Beer 2004: 717, fig. 3A)

Rand Stoneburner and Daniel Low-Beer explain their model as follows:

> Simulations of HIV incidence and prevalence in pregnant women, and mortality in the population aged 15 to 59 of Kampala, Uganda, from 1981 to 2005, showing simulated HIV prevalence under HIV incidence 'intervention' (solid line) and 'baseline' (broken line) scenarios compared with empirical HIV data from antenatal clinic sentinel surveillance sites in Nsambbya (diamond shapes), Mulago (circles) and Rubaga (triangles). The intervention scenario, in which incidence rates are reduced by 80% over a 2 year period among 15- to 24-year olds beginning in 1992-1993, produces declines in HIV prevalence consistent with empirical data. In the baseline scenario, incidence rates remained unchanged after 1990 and prevalence remains stable.[13]

Several problems are immediate apparent. In order to make the standard epidemiological model approximate a fit to the empirical data, the authors have had to assume that incidence (new infections) declined suddenly by 80% in 1989 to 1990.[14] This is extremely unlikely, and does not correspond to the recollection and stories that any Ugandans related about this period during research conducted in 2003. The reduction in incidence shown in this model is only hypothetical, introduced in order to make the standard epidemiological model achieve a closer fit to the data. In fact, the decline in prevalence is such that even if incidence had been zero—extremely improbable—it still would not account for the data.[15] Some researchers, however, have taken the hypothetical decline in incidence to be real. For instance, the authors of the

[13] Stoneburner & Low-Beer 2004: 717.

[14] See also Low-Beer and Stoneburner, 1997 (Figure 1), which shows a similar model with presumed incidence made to fall from 1985–1987, and then with an incidence reduction of 3% per annum. Neither model approximates the reality. This model is also presented in Stoneburner, Low-Beer, Tembo, Mertens and Asiimwe-Okorir, 1996 (Figure 5, p. 691), with more elaboration, and again, more briefly in Stoneburner, Carballo, Bernstein and Saidel 1998.

[15] Gray, Serwadda, Kigozi, Nalugoda, & Wawer 2006: 348.

influential 2002 USAID document, *What Happened in Uganda*[16] do this (repeating it again in 2006) when they argue that 'it is most probable that HIV incidence in Uganda peaked sometime during the late 1980s.'[17] In other words, to make the data (observed HIV prevalence) fit the model (a standard epidemiological curve) we have to assume that four out of five of all sexually active people suddenly stopped having unprotected sex for a couple of years.[18] Without this sudden, dramatic and improbable collapse in incidence, for which no confirming data exists, the dashed line wildly over-estimates the actual HIV prevalence.

Indeed, *incidence actually increased annually* from 0.9% 1993 to 2.3% in 2003, even as prevalence was falling for those who tested at voluntary counselling and testing centres.[19] The rise in incidence is consistent with a falling HIV prevalence only if incidence is rising in relatively small isolated networks. If incidence were in fact rising across a randomly selected population that is widely linked in a common sexual network—as the standard epidemiological model assumes—then prevalence would also necessarily rise. This is not the case in Uganda. Instead, incidence is stable or rising in relatively isolated sub-networks (lately, especially amongst women and middle-aged people) within which HIV prevalence might rise quite rapidly, but outside of which prevalence may remain stable or has fallen. This is also consistent with the fact that Uganda has a very high TFR.

Indeed, since there is no population-based estimates of HIV in Uganda or hard evidence of behaviour change before 1989, all must be conjectural.[20] Even so, at end of the Stoneburner & Low-Beer curve, the calculated, hypothetical trend line (solid line) considerably *under-estimates* the HIV prevalence which, in reality, levels off at a higher level than the standard epidemiological model would suggest. Further more, the curvature of the estimated (or fitted) curves tends in the opposite direction to the easily observable curvature of the actual data. The estimated curve in the period 1982 is concave or negative, while the actual data appears to trend in a convex way. In the period 1992-2002 the estimated curve shows a typical down-sloping logistic or sigmoid form tending towards zero, while the actual data suggests a concave downward trend that levels off well above zero. It appears that the epidemiology model is simply of the wrong form (see Figure 2.2 and Figure 2.3 below).

In Uganda, HIV remains endemic at higher levels that would other virus infections even after the collapse of a period of rising prevalence during the peak of an epidemic. This is because HIV does not confer immunity to infection on survivors (because there are none) as recovery from other viral infections does, and because the period of active viral infection (viraemia) is much longer in the case of HIV (and generally bi-modal or multimodal, rising at the beginning of infection and at the end). More importantly, however, the standard epidemiological model is based on the 'normal' (Gaussian) probability curve—the so-called 'bell curve' because of its shape[21]—that assumes that infection is randomly distributed in the population. While this is roughly true for most infectious agents that are dispersed by water, air, and food supplies, it is

---

[16] Hogle, Green, Nantulya, Stoneburner, & Stover 2002; Green, Halperin, Nantulya and Hogle 2006; quote is from the latter publication.

[17] This research involved 24 focus groups selected from different social strata and residential areas in three Ugandan cities, Mbarara, Kampala, and Mbale with fully transcribed records, and 30 intensive interviews with major social actors including educators, medical, military and government personnel, Christian priests, Islamic imams, traditional healers, journalists and others.

[18] Green, Halperin, Nantulya, & Hogle 2006: 337.

[19] Banyarama, Bunnell, McFarland & Hudes 2007.

[20] Gray, Serwadda, Kigozi, Nalugoda, & Wawer 2006: 347.

[21] Hart 2004.

not true of sexually transmitted pathogens which rely on 'wet' person-to-person genital contact. In other words, HIV is transmitted through a social network of a special kind, the sexual network. This error has had much greater consequences, however, than the difficulty it presents to those who seek to develop mathematical models of the epidemic.

This problem in modelling the HIV prevalence was noted in the early nineties by two molecular biologists formerly at the University of California.[22] Peter Duesberg and David Rasnick argued that because HIV epidemiology did not fit the normal epidemiological patterns characteristic of almost all other viral and bacterial epidemics, that it must not, therefore be a viral epidemic at all. Basing their argument at first on data from the US and Europe, they pointed out that

> AIDS is highly non-random with regard to sex (86% male); sexual persuasion (over 60% homosexual); and age (85% are 25-49 years old).

and

> From its beginning in 1980, the AIDS epidemic progressed non-exponentially, just like lifestyle diseases.[23]

They also pointed out that AIDS has no single defining disease[24] but is a syndrome, like obesity or illnesses caused by smoking, and that the vast majority of people who died of AIDS had a history of recreational drug use, or had taken anti-retroviral drugs. Since HIV infection in the population they had studied was also non-random, they concluded that HIV does not cause AIDS but was a product of toxicity arising from recreational drug use, lifestyle choices (especially ones that often accompanied the gay lifestyle in the US and Europe in the 1980s), and the drugs that were then used to treat it, especially AZT. If HIV was an infectious virus, they reasoned, it should infect people more of less 'randomly' (that is, there would be a Gaussian or bell-curve distribution of infections), and should grow exponentially at first. HIV infections did not do this. In a 1996 publication, he claimed—correctly at the time for US data, but ignoring African data entirely—that HIV was still not heterosexually transmitted 'outside of drug addicts' and that 'by any measure, the war on AIDS has been a colossal failure'.[25]

The core of their argument was the fact that the observed HIV trends in the data they examined did not conform to expected epidemiological patterns for viral pathogens such as flu virus, or poliomyelitis.[26] Instead, it looked much more like the linear progression of illnesses caused by

---

[22] Rasnick lost his affiliation with the University of California in 2002, but as late as 2006 he was still claiming to be affiliated with that university. He has been repeatedly warned by the University of California to cease using their name in his publications and pronouncements (See Treatment Action Campaign website, electronic document: ns09_05_2006.htm. Rasnick currently works in Pretoria with Matthias Raath (Farber 2006) selling high-dose vitamins that are alleged to cure AIDS or to be useful in treating it.

[23] Duesberg & Rasnick 1998.

[24] Duesberg and Ellison 1996; Farber (2006) provides a more recent but no less flawed restatement of this position. See Gallo, Geffen, Gonsaves et al (2006) for detailed refutation, and 'AIDS Truth' website, http://www.aidstruth.org/harper-farber.php.

[25] Duesberg and Ellison 1996: 5, 1.

[26] According to Duesberg and Rasnick (1998) diseases caused by viral agents have the following characteristics. "(i) They rise exponentially and then decline within weeks or months as originally described by William Farr in the early 19th century (Bregman & Langmuir, 1990). The rise reflects the exponential spread of contagion and the fall reflects the resulting natural vaccination or immunity of survivors. (ii) The epidemics spread randomly ("heterosexually" in the words of AIDS researchers) in the population. (iii) The resulting infectious diseases are highly specific reflecting the limited genetic information of the causative microbe. As a consequence the viral diseases are typically more specific than those caused by the more complex bacteria or fungi. It is for this reason

environmental factors such as pollution, illnesses caused by toxins, or life-style diseases.[27] Combined with their other observed differences, their conclusion published in 1996 and 1998 that HIV was not a cause AIDS seemed reasonable. Their conclusions were eventually shown to be wrong, but their critical observation about the shape of HIV prevalence trends was accurate.

Unfortunately, their findings reached the attention of the President of South Africa, Thabo Mbeki a year or so later.[28] This was to have tragic consequences. Their logic convinced President Mbeki. In October 1999, he told the National Council of Provinces that AZT was poisonous[29], and in early 2000 set up the 'Presidential International Panel of Scientists on HIV/AIDS in Africa' that included Duesberg and Rasnick. Then, in April, in a strongly worded and very personal letter to the UN Secretary General Kofi Anan, US President Bill Clinton, and British Prime Minister Tony Blair, Mbeki came close to saying that HIV did not cause AIDS, and urged world leaders to pay particular attention to the differences in the African HIV epidemic.[30] Mbeki's position was drawn fully and directly from Duesberg's work, although this was not directly acknowledged by the South African President. In a briefing paper for Mbeki that must have been received immediately before Mbeki penned his letter to world leaders, Duesberg stated the kernel of his argument that HIV does not cause AIDS, drawing earlier work with Rasnick and Ellison. Duesberg said,

> … the African AIDS epidemic is not following the bell-shaped curve of an exponential rise and subsequent sharp drop with immunity, that are typical of infectious epidemics. Instead it drags on like a nutritionally or environmentally caused disease.[31]

---

that the viruses and microbes are typically named for the specific disease they cause. For example influenza virus is called after the flu, polio virus after the poliomyelitis, and hepatitis virus after the liver disease it causes (iv) The microbial and particularly the viral epidemics are self-limiting and thus typically seasonal, because they induce anti-microbial and viral immunity and select also for genetically resistant hosts." All of this is certainly true.

[27] According to Duesberg and Rasnick (1998) diseases caused by environmental or toxic chemical factors have the following in common: "(i) They follow no specific time course, but one that is determined by the dose and duration of exposure to the toxin. (ii) They spread according to consumption or exposure to toxic agents, but not exponentially. (iii) They spread either non-randomly with occupational or lifestyle factors, or randomly with environmental or nutritional factors. (iv) They range from relatively specific to unspecific depending on the nature of the toxin. (v) They are limited by discontinuation of intoxication, but not self-limiting because they do not generate immunity." Duesberg correctly claimed in 1996 that medical researchers had the duty to consider the alternative hypothesis that 'some noninfectious cause such as poor diet or some toxic substance present in the environment or a toix consumed in an unusually large quantity' (Duesberg and Ellison 1996) had also to be considered for the science to be valid.

[28] Duesberg (2000). In 1996, Duesberg wrote: 'The single flaw that determined the destiny of AIDS research since 1984 was that AIDS was infectious. … The only solution is to rethink the basic assumption that AIDS is infectious and caused by HIV.' (Duesberg and Ellison 1996: 6) President Mbeki appears to have accepted this position completely.

[29] Mbeki was not alone in this, as Farber (2006) shows, and many, including Duesberg and Rasnick, among others, still assert this position.

[30] Van der Vliet 2004: 59. The letter can be found at www.washingtonpost.com for 19 April 2000.

[31] Duesberg 2000; see also Duesberg and Rasnick 1998. Duesberg refers specifically to two other supporting papers by Anderson (1996) that appeared in the influential volume edited by Jonathan Mann and D Tarantola. *AIDS in the World*, and to a paper by Seligman (1984) that appeared in the New England Journal of Medicine. One published six years earlier and the other published 16 years earlier, and both having been superseded by new more accurate research. Brookmeyer (1996) had already discussed the causes of such 'statistical' false reasoning in 1996, effectively refuting the 'denialists'.

Duesberg also specifically warned Mbeki in his briefing document about those who opposed his views. Citing Mbeki's own letter to world leaders that had been published by the *Washington Post* on 19 April, Duesberg wrote,

> President Mbeki must also be warned about Dr. Joe Sonnabend's answer to the president's question about the epidemiological discrepancy between the "heterosexual" AIDS epidemic in Africa and the non-random, 85%- male epidemic in the U.S. (Mbeki's letter to U.S. President Clinton, Washington Post, April 19, 2000). According to Sonnabend's hypothesis, Africans acquire HIV heterosexually, because they simultaneously suffer from a long list of diseases, including "tuberculosis, malaria, other protozoan infections, bacterial diarrhoeal infections, pneumonia, plasmodium, Leishmania" etc. However, the very low AIDS risk of an African HIV-positive, compared to an American, calls this hypothesis into question. If the Sonnabend-hypothesis were correct, African HIV-positives should develop AIDS much more readily than their American counterparts. But the opposite is true. In fact according to Sonnabend most Africans should already have AIDS by the time they pick up HIV "heterosexually".[32]

This is remarkable logic by any measure, and wholly tendentious. But, by this time, Mbeki was fully and personally committed to what came to be called the Dissident Position. Duesberg and Rasnick had convinced him through their topsy-turvy logic (a) the drugs used to treat AIDS were in fact the cause of it,[33] and (b) that because of parasites, other bacterial and viral infections and a generally higher disease load that Africans carried as a result of 'poverty', they probably already had AIDS by the time they contracted HIV!

Accordingly, in his address to the World AIDS Conference in Durban that month, Mbeki declared that poverty was the real cause of AIDS. Predictably, this unleashed a storm of protest and dismay, but up to the time of this writing (June 2007) Mbeki has yet to renounce any of these beliefs. All of the 'dissident documents' and his own dissident writings are still available on the Internet[34], some of them through official ANC websites. The controversy, however, has succeeded in stopping him from expressing his views publicly. It has not prevented a rearguard action from the President's office and from the Ministry of Health aimed at delaying provision of ARV therapy and mother-to-child-transmission protection.

The root of this tragedy lies originally in Duesberg and Rasnick's perfectly valid observation that the HIV prevalence curves are not typical of epidemics caused by other pathogens. They cited one example of an attempt to predict HIV prevalence that appeared in the Journal of the American Medical Association in 1990.[35] This lack of fit to the 'normal' curve is due to the fact that HIV is transmitted through sexual networks and is not 'randomly' distributed. As we shall see below, there are good reasons for this divergence from normal epidemic processes, reasons that do not mean that HIV does *not* cause AIDS, or that it is a disease of poverty or 'lifestyle'.[36]

---

[32] Duesberg 2000.

[33] Duesberg and Rasnick wrote: 'the long-term consumption of recreational drugs (such as cocaine, heroin, nitrite inhalants, and amphetamines) and prescriptions of DNA chain-terminating and other anti-HIV drugs, cause all AIDS diseases in America and Europe (1998: 86).

[34] See 'The Virus Myth' Internet site: http://www.virusmyth.net/aids/index.htm. According to its index page it contains over 1200 web-pages and 850 articles. It was last updated 7 July 2003 (accessed 31 May 2007).

[35] Bregman and Langmuir 1990. See Brookmeyer 1996: 784.

[36] In fact, poverty and HIV prevalence are slightly negatively correlated for large populations; see Stanton 2006; a PowerPoint presentation, unpublished. In other words, it can be shown that the greater the wealth, the higher the HIV prevalence; or, the less poverty, the greater HIV prevalence. The relatively high wealth of South Africa and

It simply shows that like environmental toxins and lifestyle diseases, HIV is spread through social structures (sexual networks) and is shaped by them.

Previous social research on HIV/AIDS has concentrated on the behavioural responses of individuals, especially their knowledge, attitudes beliefs, and practices (KAPB) with respect to sex and reproductive health. Work on sexual networks has previously focussed on identifying risk factors and tracing of contacts and, in any case, is virtually absent in Africa. By contrast, the approach taken here directs focus away from individual behaviour and 'risk' in order to focus on the sexual network as a form of 'social structure', not simply 'contacts' occurring as the result of 'behaviour'.

Unfortunately, while we have some data on sexual behaviour in Africa, we mostly lack specific data on the details of sexual networks.[37] As early as 1992, John Caldwell, Pat Caldwell and I Orubuloye (1992) noted that 'The AIDS epidemic in sub-Saharan Africa has revealed the inadequacy of our knowledge of the extent of sexual networking in the region.' This was still true in 2006: empirical data on sexual networks in Africa, with few exceptions,[38] does not exist. There are, however, ways to study the structure of networks in other indirect ways.

A NEW APPROACH TO NETWORKS

Recent discoveries—much of it in the last 5-10 years—in computer science and mathematics about the behaviour of networks provide new conceptual tools that can help us to illuminate the hidden processes in sexual networks.[39] Researchers have examined large-scale complex networks of many kinds, including the Internet (the physical infrastructure), the World Wide Web (the 'hyperlinks' that make the Internet work), networks of neurons, food webs in small ecosystems, secret communication networks between 'terrorists', citation networks among physicists, among many others. They have found that there are a number of invariant rules that govern how networks are configured and, more importantly, how they change and evolve. For instance, research has shown that these and other networks, irrespective of their size, have a 'small world' configuration in which all members of the network are linked with, on average, as few as 2-3 'links' between any two 'nodes' in the network, and at most 6 links—known as 'six degrees of separation' in social networks.[40]

These findings can be usefully applied to the study of sexual networks. Analysis of how HIV prevalence changes over time—the trend—can provide information about the gross structure of sexual networks for which we lack detailed empirical data. This paper uses these new models to examine the shapes of the trend lines based on least-square approximations to a power-law model of changing HIV prevalence in order to show that the shape of these trends suggest differences in the structure of sexual networks in the two countries. Findings from trend-line analysis are supported by analysis of differences in social structure that seem most likely to have

---

Botswana, both with extremely high HIV prevalence, tends to skew this correlation for Africa towards a stronger positive correlation between wealth and HIV than might exist elsewhere.

[37] The only detailed tracing of a significantly large sexual network that I am aware of to date is Kohler and Helleringer 2006. This is for an island, Likoma, in Lake Malawi.

[38] Jonathan Stadler's current work in the Bushbuckridge area of Limpopo Province, South Africa, is promising. See Kaufman, de Wet and Stadler 2001; Collins and Stadler 2001; Stadler 2003.

[39] Faloutsos, Falutsos & Faloutsos 1999, Barabasi 2002, Buchanan 2002, Watts 2003, Strogatz 2003, Leskovec, Faloutsos & Kleinberg 2005.

[40] Buchanan, 2002; Strogatz, 2003.

an impact on the configuration of sexual networks, including patterns of kinship, marriage, household structure, inheritance and wealth.

COMPARING HIV TRENDS IN UGANDA AND SOUTH AFRICA

HIV prevalence rose in Uganda very rapidly in the 1980s and early 1990s until 1992, when it began to decline very rapidly.[41] When it first began to be measured in 1985, it was already above 10% at ante-natal clinics (ANCs). It is likely that HIV had been endemic at a low level in Uganda for at least a decade before it was first tentatively measured in 1982, and possibly for generations.[42] By 2002, the decline in HIV prevalence had stabilised at around 7%, falling slightly then possibly creeping up again by 2007.[43] The direction of the trend after 2002 is still not clear.[44] On the other hand, South Africa has seen a steady rise in the prevalence from the beginning of the 1980s until the present, with as yet little evidence of a levelling off.[45] In 1992, the overall prevalence in Uganda was around 24%, with rates as high as 29% in Kampala, and 20% average in the four other major towns in Uganda.[46] The overall prevalence in South Africa nationally is similar today (2005) to that in Kampala, Uganda, thirteen years earlier. Prevalence in some age and sex groups and in some regions in southern Africa now exceeds 30%, a level never reached in Uganda.

The vast difference between the two countries is illustrated most starkly in Figure 2.2. This simply shows the temporal relationship of changing trends in the two countries.

---

[41] Asiimwe-Okiror, Musinguzi, & Madraa, 1996; Hogle, Green, Nantulya, Stoneburner & Stover, 2002; Uganda Ministry of Health 2002.

[42] Caldwell & Caldwell 1996, 563.

[43] See Chapter 1, note 9.

[44] At this date, May 2007, there was not sufficient data to suggest anything but fluctuating stability within a range of 5%–7%.

[45] Human Sciences Research Council, 2002; Dorrington, Bradshaw & Budlender 2002; Asamoah-Odei, Calleja & Boerma, 2004).

[46] These are Jinja, Mbarara, Tororo and Mbale.

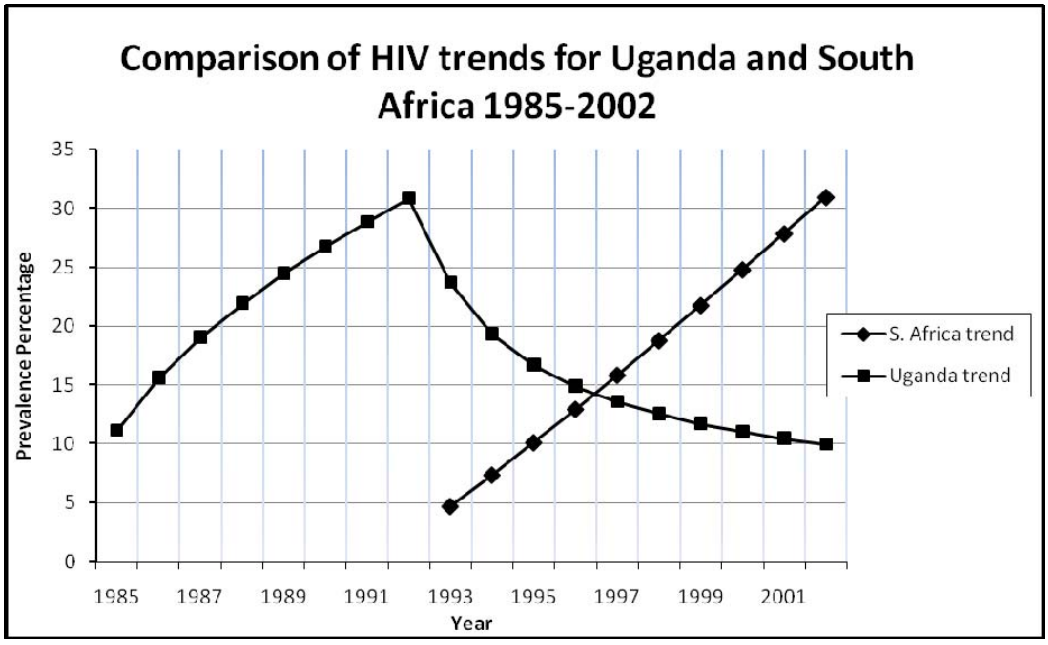


Figure 2.2. Stripped of data, the overall prevalence trends in Uganda and South Africa are starkly different

The data on which these trends are based is, of course, much 'messier'. There is, also, a large margin of error associated with each data point. Since we are concerned only with longer term trends, the margin of error associated with each point can be eliminated as a concern.[47] The fact that these trends are highly consistent with each other for different data sets within each country also increases the reliability of these findings. Since we are trying, first of all, to compare two countries in the broadest possible strokes, however, elimination of the 'noise' in the data allows us to see the overall relationships in time, and in the shape of these trends. In the following analysis, it is necessary to separate out different periods for close analysis.

We first compare these trends for the 11-year period (1992-2002 inclusive) when South African and Ugandan trends were moving in opposite directions. By plotting these two different national trends against each other, several significant patterns emerge during the decade 1992-2002.[48]

[47] Probable errors exist in all of the data points for many reasons. However, a trend line tends to even out the errors and show the overall 'trend' of the data, that is, it tendency to change in a particular direction at a particular rate. Since the entire data set is taken into account, the errors involved in each point are less important than the overall picture.

[48] South African Institute of Race Relations 2004: 297, table 'Antenatal Clinic Surveys: HIV infection rates of women attending public antenatal clinics by province, 1991-2002,' citing Department of Health, South Africa. 2002. Summary Report: National HIV and syphilis antenatal sero-prevalence survey in South Africa.

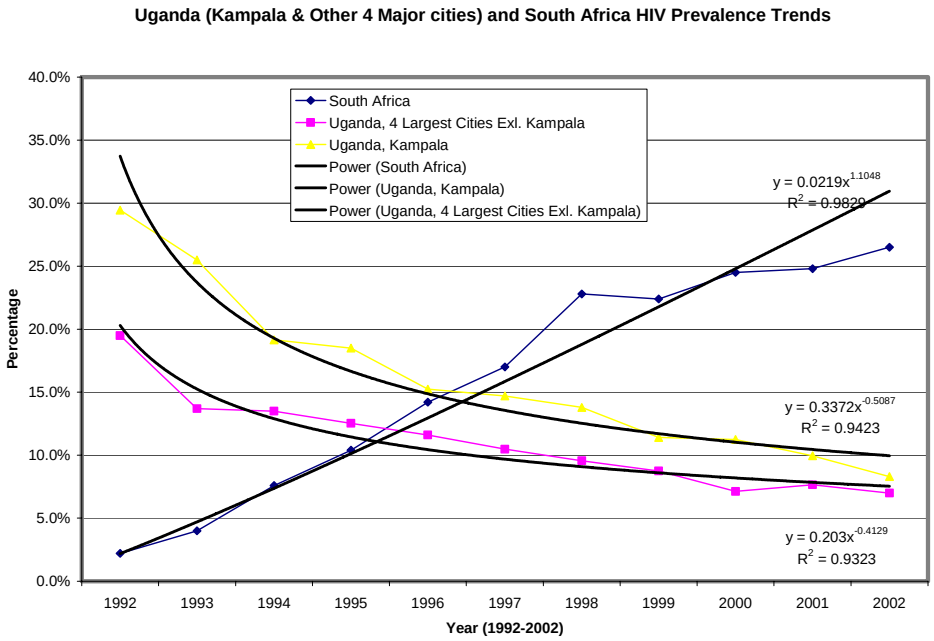


Figure 2.3. Comparison of HIV trends in Uganda and South Africa, 1992-2002

Figure 2.3 shows the trend for South Africa as a whole against two sets of data for Uganda.[49] The triangles represent aggregated data for Kampala (drawn from the same ante-natal HIV surveillance sites used in Stoneburner and Low-Beer's model above (Figure 2.1). In this plot, median values for these three sites have been used, because each set of data alone shows a broadly similar trend. Squares represent the data from Uganda's four largest cities with ANC sentinel sites, Mbarara in the southwest, Jinja in the south central area, Tororo and Mbale in the east. Diamond shape markers represent data points for ANC sentinel sites in South Africa.

For each set of data, a 'power' or 'power-law' trend line is calculated and plotted (see explanation boxes). Curves of this sort are associated with complex network systems.[50] The remarkable fact that the Ugandan data follows a power law trend much more closely than it does the standard epidemiological model, shown in Figure 2.1, suggest that efforts to understand the HIV epidemic in the same terms we use to model and visualise other epidemics is likely to fail. Something else is at work here. Since power law trends are closely associated with networks of all kinds, it suggests that we might gain insight by examining the social networks that transmit HIV. Power law trends are also associated with sudden changes in chemical and physical systems that are called 'phase changes', such as when liquid water suddenly turns to steam (a gas) at the boiling point, or when it turns to ice (a solid) at the freezing point. It turns out that this is also true in the changes in HIV prevalence in Uganda.

The mathematics of the trend line

[49] Asamoah-Odei, Calleja & Boerma, 2004; Kirby 2004.

[50] Leskovec, Kleinberg & Faloutsos, 2005; Hart, 2004.

The trend line (heavy dark line imposed over the data points) shows the closest fit of the data to a curve described by a 'power law' equation of the form $y=ct^a$. Here y is *HIV prevalence* expressed as percentage. The t is *time* in years, for this is how the available data is given to us. The symbol *'c'* is a proportionality constant that simply adjusts the scale of the graph, and a is the calculated exponent that allows the trend to fit the data.

The R^2 value estimates the closeness of fit, with values approximating 1 indicating a close fit. Here, the trend lines fit the data very closely with values approximating 1.

When $a = 0$, the curve is simply a horizontal line in which $y = c$, a constant value. When $a = 1$, we have a simple linear relation, that is, a straight line. The South African data shows and exponent close to 1, in which $y=ct$.

Fractional exponents where the absolute value of a lies between zero and one, $0 < |a| < 1$, however, yield curves like those seen in the Uganda case. If the exponent is negative, the curve has a 'concave' shape with what is called a 'fat tail': the prevalence, y, declines rapidly at first then 'tails off' very slowly without reaching zero, as in Figure 2.3. Positive values of the exponent yield the inverse 'growth' pattern, shown by the trend line from 1985 to 1992 in Figure 2.4.

This plot uses Microsoft Excel's built-in least-squares fit to the power-law model.

Where sexual networks are infected with the HI virus, HIV prevalence will be a function of the number of sexual contacts ('links') between people (network 'nodes') in a network of a specific configuration, and, over time, may behave in different ways depending on how the network is structured. For Uganda, it changes in a non-linear way that is characteristic of highly structured complex networks with hubs and/or clusters, and of networks that exhibit fractal structures. This is distinctly different from a linear growth that would be seen in simple 'diffusion' models of transmission, or exponential growth of unbounded biological systems, or the sigmoid curves of growing populations ultimately constrained by the 'carrying capacity' or 'saturation' of their environment. Specifically, the Ugandan curves lie half way between stability (a=0) and linear growth ($a > 1$) that would be characteristic of simple diffusion. This behaviour is the signature of complex network organisation that is 'fractal' or 'self-similar' at different scales.[51]

The two trends for Uganda—one for Kampala, the capital of Uganda (comprising median values for ANC clinics at Rubaga, Nsambya and Mulago hospitals), and one for the other four major cities for which we have good data—both show approximately equal declines during this period.[52] Both curves conform closely to one another, with the curve for Kampala reflecting higher HIV prevalence in the city than in the smaller regional cities. Trend curves for rural data and smaller towns (Moyo, Mutolere, Masindi, Hoima, Kilembe, Pallisa, Aber, Lwala, Soroti, Matany, Kagadi, Arua, Lacor, and Nebbi) are similar in shape, but lower in magnitude. Data for these sites is incomplete and therefore not presented here. The trends drop steeply for the first several years from 1992 to 1995, and then begin to level out towards an overall prevalence of around 6-7%. This is confirmed by the preliminary results of a new national survey of HIV prevalence in Uganda based on voluntary blood tests conducted on a representative statistical sample for the entire population that also shows an overall prevalence of about 7%.[53] Equally

[51] Eglash, 1999.

[52] Uganda Ministry of Health, 1992-2002.

[53] Wasswa, 2005.

significant is the fact that HIV levels stabilise but do not tend towards zero as ordinary viral epidemics would. This fact is accurately modelled by the power law, but not by the 'normal' (Gaussian) distribution curve.

The decline follows a period from the early 1980s to 1992 in which HIV prevalence rose rapidly in Uganda towards a peak in 1992. The transition from rapid escalation of HIV rates in Uganda from the early 1980s up to 1992 was as remarkable as its decline. The period of increasing prevalence is also closely modelled by a power law. The transition between increasing HIV prevalence and decrease is very rapid (See Figure 2.4).

By separating the period of increasing prevalence (1985-1992) from the period of decreasing prevalence (1992-2002), we can calculate two discontinuous trend lines that show similar but inverted patterns.

'Power law': the mathematical details

Unlike an *exponential* prevalence curve, the *power law* curve is simply a function of time, t, raised to a constant power. The exponent is a real number; that is, it may be a positive or negative whole number or fractional number. By contrast, the exponential curve expresses a situation in which the prevalence is a function of itself: that is, the higher the prevalence the faster the rate of increase, or the lower the prevalence, the lower the rate of increase, usually (in real life systems), up to some limit imposed by availability of resources.

The trend of the decline in Uganda conforms to a power-law decay from the peak reached in 1992. The decline in HIV prevalence (P_{HIV}) as a function of time (t, in years, in accordance with the data sets) is estimated by the expression

$$P_{HIV} = 0{\cdot}3372t^{-0{\cdot}5087}$$

for Kampala ANC test sites, and by

$$P_{HIV} = 0{\cdot}203t^{-0{\cdot}4129}$$

for the four other large cities (where t is time in years in accordance with the data). The two trends are very similar, and both have a negative non-integer exponent a, $0 < |a| < 1$. The trend for the period of *increasing* HIV prevalence, 1985-1992, for Kampala (Figure 2.4) is estimated by

$$P_{HIV} = 11{\cdot}09t^{0{\cdot}4912}.$$

All power law exponents (-0.5087, and -0.4129 for the period of decreasing HIV prevalence, and 0.4912, for the period of increasing prevalence) are approximately of the same magnitude, $|a| \approx 0{\cdot}5$. The similarity in these values suggests that we are dealing with the same form of network. In the first period (1985-1992) links were rapidly forming, making the sexual network an increasingly *efficient* transmitter of HIV. In the second period, links were rapidly breaking, resulting in a *decreasing efficiency* in HIV transmission in the sexual network .

The fact that these two trends were inverses of each other, with closely similar absolute values of the power-law exponent, suggests that whatever was happening in the overall sexual network to cause an increase in HIV, was also happening during the period of decreasing HIV prevalence, but in a way that was somehow 'inverted'. Moreover, this trend-line analysis suggests that *the changes in HIV prevalence were functions of the configuration of the overall sexual network, rather than simply statistical changes in human sexual behaviour*. In other words, we do not have to assume that 80% of the sexually active population stopped having sex between 1991

and 1992, as Stoneburner and Low-Beer are driven to assume, given the nature of their model. As we shall see, it is very likely that what was happening during the period of increasing HIV prevalence in Uganda was an increase in the number of links (sexual contacts) between persons of groups of people (such as within villages, around a nightclub or bar, at a funeral or ritual event), and especially an increase in the links between highly infected persons or clusters that were highly linked to other persons or clusters such as soldiers, transport workers, 'sugar daddies/mamas' and others. During the period of decreasing prevalence, overall connectivity decreased, but especially the links between highly infected persons or groups in the network and the general population decreased. This would explain the power-law shape of the trends since these are the 'signature' of networks connected in this way.

Previous work on networks has assumed that they are static, and that they can be represented adequately by graphs (drawings) that represent the links between nodes at some arbitrary time or over some arbitrary period of time.[54] This, however, is not adequate for our purposes. Sexual networks are dynamic, not static: they grow or shrink over time and they may increase the number of links, or the number of links may decline (decay). The configuration of networks also changes over time. Thus, we need a model of networks that takes this into account.

Fortunately, the power law describes just such changes in a network. Research on the evolution of the hard-wired Internet and software-based World Wide Web (WWW) has shown that

> The networks are becoming *denser* over time, with the average degree [the average number of links] increasing (and hence with the number of [links] growing super-linearly in the number of nodes). Moreover, the densification follows a power-law pattern.[55]

What this means is that empirical observation of very large networks such as the Internet and WWW shows that the number of links between nodes increases more rapidly ('super-linearly') than the number of nodes. This is caused by what has been called 'preferential attachment',[56] or a 'rich get richer' phenomenon.[57] According to this, the establishment of links to new nodes tend to favour those that are already highly linked. Nodes that are already popular, for instance, such as Yahoo.com or AOL.com, attract more links than other web-sites. This is also true of neurons in simple organisms, networks of Hollywood actors, or famous scholars whose works are most often cited. According to Barabasi (2002:80), these patterns, 'potentially present in most networks, could explain the power laws we spotted on the Web and in Hollywood', among many others. In the case of sexual networks that transmit HIV, clusters of intensive sexual contact produce higher likelihoods of HIV transmission. In other words, with respect to HIV, the 'rich' (highly infected clusters) get 'richer' (that is, prevalence increases more rapidly than less well sexually-connected clusters or categories of people.

The network power law

Leskovec, Kleinberg and Faloutsos (2005) have found that 'as the graphs [of networks such as the Internet and World Wide Web] evolve over time, they follow a version of the relation':

$$l(t) \approx n(t)^{a}$$

[54] Leskovec, Kleinberg & Faloutsos 2005.

[55] Leskovec, Kleinberg & Faloutsos 2005.

[56] Barabasi 2002: 91.

[57] Barabasi 2002: 80.

where $l(t)$ and $n(t)$ represent the number of links (l) and the number of nodes (n) at some time t, and a is the power law exponent. The expression '$l(t)$' simply states that the number of links is a function of time. This is a power law relation that means that the number of links in these networks increases in proportion to the number of nodes raised to some power represented by the exponent a. This expression describes the growth of a network. Leskovec, Kleinberg and Faloutsos (2005) call it the 'densification power law' or the 'growth power law'. Since their research concerns the Internet (the physical electronic infrastructure that connects computers) and WWW (the software 'links' connecting users that are 'followed' by browsers such as Netscape, Firefox or Internet Explorer), both of which are growing rapidly, they do not consider the case in which networks shrink by losing links. This is what happens when HIV prevalence declines. In this case, the inverse of the 'growth power law'—in other words, a 'decay power law'—exists when the exponent is negative, as follows

$$l(t) \approx n(t)^{-a}$$

This relation says that the number of links between nodes decreases more rapidly ('sub-linearly') than the number of nodes (persons). This relation closely models the period of decreasing prevalence in Uganda.

HIV prevalence in the population, then, will be proportional to the number of links (sexual contacts) that connect the nodes (persons) in the sexual network as a whole, itself a subset of the entire population. Thus HIV prevalence will vary according to the number of links, which in turn are proportional to the number of nodes raised to the power a, the 'densification exponent' that is characteristic of all such complex 'preferential attachment' networks. Accordingly, a power law that describes HIV prevalence trends emerges:

$$P_{HIV} \approx l(t) \approx n(t)^{a}$$

In the case of HIV, a decrease in the 'densification' of the network corresponds to any thing that stops the transmission of HIV between nodes, hubs or clusters, thus 'thinning' the density of links within the sexual network. This can be any form of behaviour change that decreases incidence, but can also be the result of changes in spatial mobility or social-structural changes that influences the frequency with which people have sex with each other. It can also be the result of morbidity and death, since the very sick and the dead do not participate in sexual networks. In other words, the number of relevant links—those representing relatively healthy people having unprotected sex—declines faster than the number of persons. Thus, using a condom, abstaining from sex, reducing the number of partners, or even death, all accomplish the same thing in this respect. Why does this result in a pattern of HIV prevalence that is closely modelled by a power law? This can be explained in the following way.

THE POWER-LAW MODEL AND HIV TRENDS

It is intuitively obvious that HIV spreads through 'links' in the sexual network. Thus all sero-positive people will be members of the sexual network in which some people overall, and *all* sero-positive people, necessarily have more than one sexual contact with another person. In fact, the set of HIV sero-positive people is a subset of the network of all sexually active people in the larger population. Infected individuals constitute a 'sample' or sub-set of the set of all members of the sexual network that has been 'selected' by HIV infection. All HIV+ people are necessarily linked by some set of links to all other HIV+ people, and to some HIV-negative people. Some members of the set of HIV+ people are likely to have many sexual contacts with others, especially during periods in which HIV prevalence is increasing. They may constitute

'hubs' in the network, and are efficient transmitters of HIV. In fact, there may be clusters of HIV+ people whose links with each other and with some HIV-negative people are quite dense. These persons or clusters can act as transmission 'centres' or hubs. Sexual networks, however, usually have long 'filaments' of contacts with cross-links to other filaments and to hubs. These 'filaments' can act as transmission lines in the way electrical power lines do. The efficiency with which HIV is transmitted overall, then, will depend on the specific configuration of the network.

This is what we find in the data for Uganda. The overall picture of increasing *and* decreasing HIV prevalence for Kampala data is shown in Figure 2.4.

It is important to note that the pattern observed here does not look like the patterns seen in other types of epidemics caused by 'normal' bacteria or viruses.

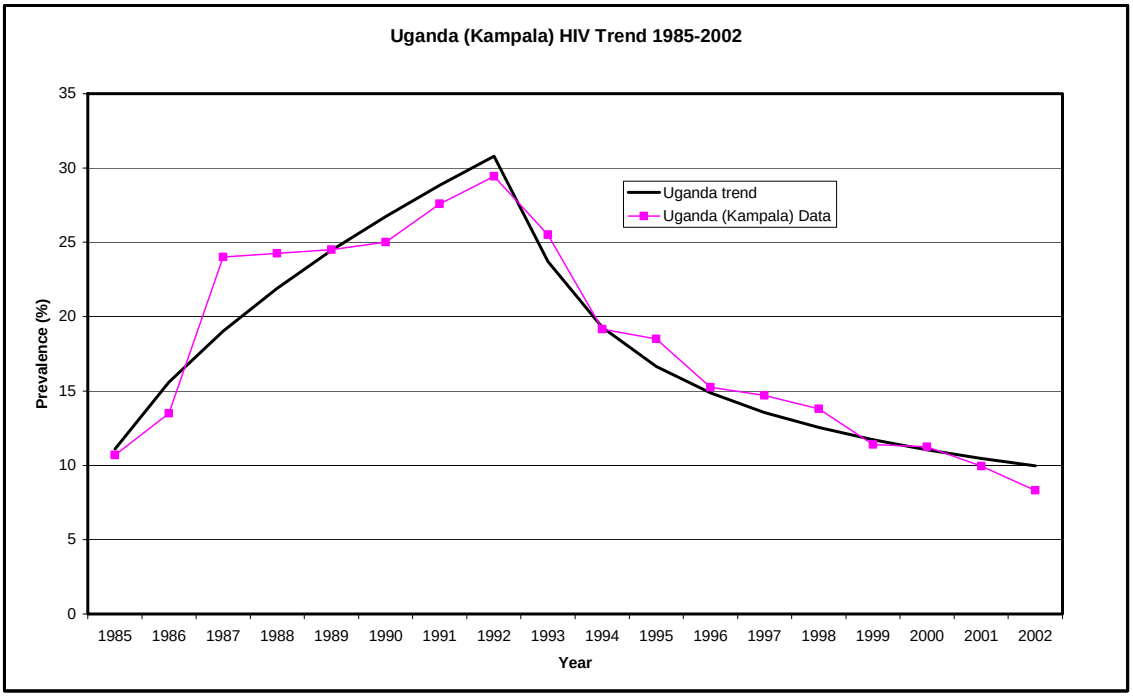


Figure 2.4. Trends for changing HIV prevalence for Kampala, Uganda, 1985-2002

This composite curve (Figure 2.4), which fits the data quite closely, shows a remarkable and sudden reversal. Such trends suggest that the dynamics of the system are unstable, and that something like a 'phase transition' or 'catastrophe event' occurred in 1992. A sudden shift of this sort—a 'tipping point'[58]— is most likely to be caused by a rapid re-organisation of the overall structure of the sexual network that transmits HIV rather than by incremental and local changes in HIV incidence caused by behavioural change, morbidity, or death alone. Much recent work in the mathematical analysis of networks shows that non-random networks (such as the Internet, but also social networks, scholarly citations, networks of neurons, and food chains in ecological systems) all show power law patterns. Steven Strogatz, one of the leading innovators in this field, remarks,

[58] Gladwell 2000.

> Power laws hint that a system may be organizing itself. They arise at phase transitions, when a system is poised at the brink, teetering between order and chaos. They arise in fractals, when an arbitrarily small piece of a complex shape is a microcosm of the whole.[59]

This analysis of the Ugandan HIV data strongly suggests that just such a 'phase transition' occurred. It also suggests that the structure of the network is 'fractal', that is, it exhibits a self-similar structure at different scales of analysis. This would be consistent with a highly clustered network in which different clusters of sexually active and therefore interlinked individuals are further grouped into larger clusters, and so on. In such a network, each cluster has fewer links to other clusters, both within the same order of magnitude (sections of a village) and at higher orders of magnitude (regions, language groups, age groups) than it does within itself. This structure permitted what amounted to the 'phase transition' that we see in Figure 2.4 in which the network suddenly became much more resistant to transmission of HIV virus around 1992. *This would be consistent with a model in which a highly clustered network with limited links between clusters suddenly lost those interlinking 'long distance' connections resulting in a phase transition of the network.* Through a process similar to water freezing and suddenly becoming 'lumpy' with ice crystals, then hard as it freezes to a solid, it seems that the Ugandan sexual network returned to a 'lumpy', clustered state. In other words, while people continued to have sex, possibly with multiple partners, persons or clusters that acted as hubs in the network largely disappeared. This is consistent with the data on changes in sexual behaviour in Uganda.

By contrast, the trend for South Africa (Figure 2.3) is approximated by a straight line. This would not immediately suggest that networks were involved, except for the fact that we know they are. What it does suggest is that the South African sexual network permits a more or less unimpeded flow of HIV through it, roughly like liquid paraffin diffusing through a wick in a lamp, or a strong smell diffusing through a room from its source. It takes a bit of time, but it moves at a constant rate. It would seem that something like this was happening in the South African sexual network.

Why a power law looks like a straight line in the case of South Africa

The trend line equation for the South African data is approximated by the power law equation:

$$P_{HIV} = 0{\cdot}0219t^{1{\cdot}1048}$$

where the exponent is close to 1.

As we have seen, where the exponent in the power law is 1 or close to one, the graph of the equation approximates a straight line because any value raised to the power of 1 is simply that value:

$$f(t) = t^1 = t.$$

In the South African case, this corresponds to a more or less straight line sloping upwards over time meaning that the increase in prevalence is simply *directly proportional to time*, with a small multiplication factor of 0·0219 that increases the slope ever so slightly upwards.

The South African data shows an approximately linear progression during the period 1992-2002. A linear progression would mean in this case that HIV prevalence (the *y*-axis) is simply a function of time (the *x*-axis), or, in other words, that prevalence is growing steadily with time

[59] Strogatz, 2003.

and that the *rate* of change was not itself changing. We must keep in mind that the data does not preserve spatial characteristics, so what it shows is a gradual trend towards saturation of the *entire sexual network* with HIV infection. This in turn suggests that the sexual network is relatively homogeneous and 'randomised', that is, that all members are likely to have contact with people both close and far, or within some category (linguistic, cultural, age) as well as with others outside of this category. It has been shown that people in multi-linguistic communities in Uganda are far more likely to have extra-marital sexual contact than those within mono-lingual communities.[60] Since virtually all South Africans live in multi-lingual communities and almost all speak several South African languages, this may predispose towards greater sexual contact in South Africa. It also suggests that most sexually active people have multiple partners, and that most have more or less the same number of partners (links) in the sexual network. The behaviour of the trend during this period suggests a nearly free diffusion of HIV through the population as if the growth of the epidemic was a function of time alone rather than what is usually the case in epidemics, namely that prevalence over time would be a function of the number of infected people already in the population. Furthermore, it suggests that the system (all sexually active people linked in the HIV transmission network) is not yet close to saturation or phase transition.

Normal exponential growth of epidemics

In their early stages, epidemics normally grow exponentially, that is, the rate of change in prevalence (of some infectious agent, say a flu virus) in the population is at first proportional to the number of persons infected: the greater the number of people already infected, the greater the rate at which more people will become infected. This relation can be expressed mathematically by the ordinary differential equation

$$\frac{dy}{dt} \approx y$$

meaning that the change in y with respect to time, t, is proportional to y. This has as its solution in y the simple exponential growth equation,

$$y = ce^{kt},$$

where c is the initial value (in this case 1), k is the proportionality constant and t is time represented on the x-axis. The e, a constant value, is a special type of number, called a transcendental number, like π; $e = 2{\cdot}7183\ldots$). It can be thought of as the limit on the returns from compounding interest (versus simple interest) as the number of periods of compounding interest are increased. It is thus a natural value for the base of the exponential function.

But the growth of HIV prevalence in South African did not suddenly begin as a linear increase. In its earliest stages, it grew exponentially, as the earliest periods of infection did in the US and elsewhere for which we have relatively complete data. (We do not know what happened in Uganda at first, because HIV and AIDS were unknown and not tracked before it was already infecting up to 10% of the population in parts of Uganda.)

Further analysis of South African data shows that the earliest period of the HIV epidemic is most accurately described as *exponential*. This shows that in its earliest stages, HIV prevalence increased freely as if it encountered no barriers, that is, as if it were like a virus that could be spread by and infect anyone. This could only be true if the sexual networks in which it was

[60] Bishai, Patil, Pariyo & Hill 2006.

propagated were multiply and randomly connected. Viruses and bacteria that are free to move easily, and where there is no previous immunity, grow exponentially in a population before they either evoke immunity in previously infected people, or die off as their hosts succumb to lethal pathogens (in which case, they decline exponentially to zero levels or nearly).

Exponential curves generally emerge in natural systems where growth is unrestricted, such as the growth of any population (dividing cells, free growth of viruses, bacteria or other organisms) that is not limited by resources (food, light, space). Such trends occur where the rate at which prevalence changes is a function of prevalence itself, that is, the more infections that already exist, the greater will be the increase, up to some limit such as death, recovery from infection, or lack of vulnerable individuals. The earliest South African data is precisely of this form (the R^2 value of 0·9935 shows a virtually perfect fit to the exponential trend line), as Figure 2.5 shows.

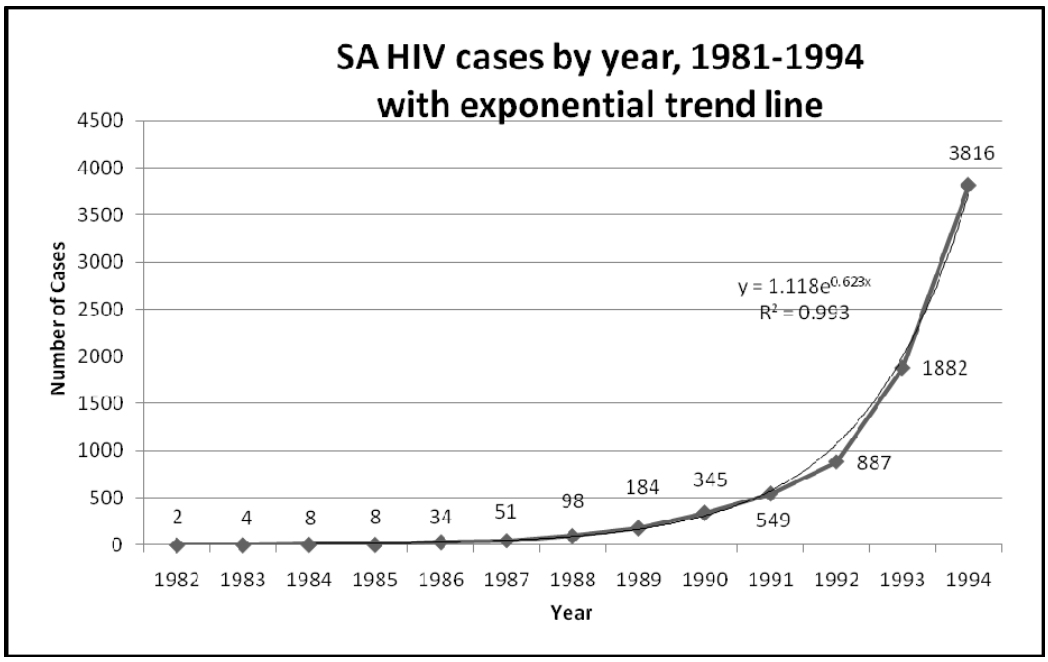


Figure 2.5. The earliest period of growing HIV prevalence in South Africa, 1984-1994 (data source: UNAIDS/WHO Epidemiological FactSheet 2000)

This trend, taken together with the later data describe an almost perfect 'growth curve' in which limits to growth have not been reached. It also strongly suggests that HIV transmission in South Africa occurs in a fully connected and randomised network without internal structural limits such as clustering or formation of 'core groups' of endemic but limited infection.

(Re)organisation of sexual networks and prevalence change

The patterns that we see in prevalence trends strongly suggest that sexual networks in Uganda are highly clustered or 'lumpy' with many locally-dense sub-networks that have fewer connections between them. In South Africa, networks appear to be randomised and highly interlinked. The 'catastrophe event' or 'phase transition' in the Uganda prevalence trend resulted from a fairly sudden re-organisation of the sexual network during which the density of links between local high-prevalence, high density networks—'clusters' or 'lumps' in the network—and the rest of the population were suddenly reduced. In South Africa, this has not

happened because the network is much more uniform with large numbers of people multiply connected to each other.

In Uganda, the centres of infection were either clusters of highly interlinked and infected persons around a bar or night club, for instance, or perhaps highly sexually active persons such as soldiers or truck drivers. People involved in culturally-sanctioned networks of sexual exchange (e.g. wife-sharing, or sexual cleansing after death of a spouse) are also likely hubs. In terms of this model, it does not matter whether these links were 'severed' by behaviour change (using a condom or reducing numbers and/or frequencies sexual contacts) or whether it was due to death or other factors. The result was the same: fewer links between highly infected hubs, clusters or filaments and the rest of the population. Since infected and highly sexually active hubs (whether individuals or groups/clusters) are highly efficient in spreading HIV, reducing links from these hubs creates a non-linear decline in the transmission of HIV. If some percentage reduction in sexual links occurs, say *r*%, then this will have little effect within densely connected sub-networks, but it will have a significant effect on linkage between clusters. In fact, *r*% reduction of cross-cluster links will effectively isolate some parts of the population from infection. The effect overall will be a re-organisation of the total sexual network in which prevalence will be non-linearly related to *r*. This can be described as a phase transition.

In other words, the rate of change in HIV prevalence was not directly proportional (linear) to the sum of changes in individual sexual behaviour or death but was instead a function of the change in the organisation of the overall network which had the effect of accelerating HIV decline.

The structure of the South African sexual network appears, from this analysis, to be quite different. In particular, it seems that the sexual network spans the nation (and probably also much of the southern African region, including, at least, Botswana, Lesotho and Swaziland which share languages, culture and population freely with South Africa). There appear to be few clusters or hubs in a relatively homogeneous network of sexual relations that ultimately incorporates most sexually active people (and certainly all those with more than one faithful partner). In such a network, HIV spreads according to normal growth curves ('logistic' or 'exponential') and has so far not reached a natural limit. In fact, we cannot, as yet, say what that natural limit might be.

Given the lack of actual empirical data on sexual networks, then, it appears that analysis of trends may serve as a proxy measure for the structure of sexual networks. Since we know that the configuration of sexual networks is critical to the transmission of sexually transmitted infections, it follows that such knowledge can be crucial in understanding the progress of the epidemic. This, in turn, can help us to create and implement interventions that might be more effective than what we have seen so far in Africa.

Perhaps the most important lesson that we may derive from this new understanding of networks and HIV transmission is that significant change in HIV prevalence is a property of the social network rather than of individual behaviour. We must re-direct our attention, then, from the scale of the individual—behaviour, psychology, risk assessment and so on—to the scale of the social. This represents a radical shift in perspective.