HIV/AIDS and Vulnerability: Sundering the Bonds of Human Society?

Tony Barnett 12
Development Studies Institute
London School of Economics & Political Science

IDS/UNAIDS HIV/AIDS and Vulnerability Workshop

ABSTRACT

There is a disharmonious resonance between the length of infection in the individual human host and the length of a human generation. Put briefly, an infected person has children, these are orphaned and grow up to become infected, but not before they have themselves had children – who are orphaned in turn. Hence a basic unit of social structure in most human societies, the three generation bond between grandparents, parents and the current generation – and on into the future – is rent asunder: repeatedly in the absence of treatment, a vaccine or behaviour change. This process should be read against an ever present threat of growing viral resistance.

Children brought up in difficult circumstances develop a pragmatic and short term survival perspective. Furthermore, behavioural change messages about HIV/AIDS may have the unintended consequence of stigmatising the parental generation. Thus in Africa, where respect for elders is of central cultural importance, the intergenerational structural break appears as loss of respect for elders – at a time when rapid change is already contributing to this process. Combined with an un-moderated pragmatic orientation towards the world, children and young people are hard-pressed merely to survive from day to day. This may have substantial implications for social and political relations at the household, community, and ultimately the national level when these inadequately socialised people reach adulthood.

1 ESRC Professorial Research Fellow, London School of Economics
Email: a.s.barnett@lse.ac.uk
Website: http://www.lse.ac.uk/collections/DESTIN/whosWho/barnett.htm

2 This is not a finalised paper. It is a summary of some of the ideas presented at the meeting. Complete citations and references will be found in various published versions of the paper.
Introduction
The HIV/AIDS epidemic is one of a class of phenomena which can be described as long wave events\(^3\). Long wave events have the following characteristics:

1. Their effects are seen before their longer run significance and weight are appreciated
2. They form important backgrounds to and influences on shorter term intellectual and policy concerns
3. A central reason these events are difficult to halt is that it is enormously hard to get people to recognise them for what they are and to take appropriate action
4. Managing the consequences of long wave events makes novel demands and our existing experience is not necessarily a good guide to how we should respond
5. Most political and administrative capacities are not established to deal with such events
6. Their duration lasts decades and perhaps centuries
7. Their duration does not permit easy engagement by policy makers and politicians whose foci are unlikely to exceed 5-10 years – the “medium term.

Examples of such events include: the HIV/AIDS epidemic, climate change, long run population trends. This paper examines some aspects of the HIV/AIDS event as it presents itself in relation to unusual levels of orphaning.

A recent report by the South African Institute for Strategic Studies contains the following statement:

“South Africa is sitting on an AIDS orphan time bomb that could unleash a tidal wave of crime and civil unrest: up to three million children will be orphaned within the next 10 years. The burgeoning orphan population, which will grow up under extreme levels of poverty, will be sorely tempted - or even obliged for its physical survival - to turn to crime, drugs, gangs and the sex trade.”

There must be no doubt in our minds that the HIV/AIDS epidemic (or endemic as it might now be more accurately described) is an extremely serious one. However, its seriousness and our response to the complexities it presents raises many important questions with which we must engage when we

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consider (a) what we believe is actually happening (b) what evidence we have and (c) how we develop policies as part of that response.

Today’s intellectual milieu is profoundly marked by “post-modernism”. Briefly put, “post-modernism” tends to varying degrees to reject the progressive narratives of modernism, and in particular its assumptions as to (a) the right goals for social policy and (b) the link between scientific investigation and “evidence” which feeds into the formation of that policy. To put the situation in a simplified manner, there may be said to be two forms of post-modernism. One form deploys these insights to encourage a critical skepticism as to the evidence and goals which form the policy discourse; the other form takes these insights as a reason for the total rejection of the “truths” and goals of modernist discourses. To the mind of the present writer, the first is a sensible and indeed scientifically necessary therapy to counterbalance the misuse of the authority of scientific knowledge while the latter is a nihilist rejection of the possibility of knowing at all and therefore of acting and most seriously of acting morally.

This paper is situated firmly in the former framework and is extremely concerned about the process of knowing for the reasons that to know is to act and to act is to have the possibility of acting morally. The moral goal against which the paper is situated is one of saving and/or improving the welfare of fellow human beings as an absolute moral goal. In this case we are concerned with the lives and welfare of children. But as will become apparent, we are also concerned with questions about the long term welfare and fates of the societies in which people exist and find their identities.

At this point a personal aside is in order. The author’s grandmother was a relatively unlettered woman who frequently spoke in terms of mottos, metaphors and myths as a way of communicating her beliefs and insights. One that has particularly remained in the author’s mind is: Life is the stories we tell; stories are the lives we live.” A moment’s reflection will persuade the reader that my grandmother (who was born in the 1880s and died in the 1950s) was an early post-modernist. She saw that much of what we are, our
identities, our goals, our policies and our deployments of “truth”, all of these things, link to the stories we tell ourselves, each other and which are told to us. The point of this diversion is to underline for you that the statement from the South African Institute for Strategic Studies is a story; it is not a truth upon which public policy can safely be grounded. The same is true of much that is said around the AIDS epidemic, not least about the questions of AIDS orphaning and its implication. Against this framing insight, this paper does the following:

1. Describes the specificities of HIV/AIDS that make it a cause for particular concern among the myriad critical issues of our age;

2. Presents and briefly discusses what we know about the extent of HIV/AIDS related orphaning;

3. Speculates about some of the possible broader implications of these phenomena for public policy and for the welfare of children in the context of this particular long wave event.

1. The Specificities of HIV/AIDS

An epidemic is a social event par excellence. However, a clear understanding of the relation between orphaning and child welfare and the epidemic requires consideration of three levels:

- the life cycle of the infectious pathogen,
- the level of the individual body and
- the level of the human group, a community, a network, an organisation, a country or – as with HIV/AIDS – at a global level.

It is important to bear all of these levels in mind together with their interactions to fully understand the potential, possible and evidenced relation between the epidemic, actual and potential orphaning and the significance of these for present responses and future outcomes.

Level 1: the Pathogen

The pathogen inhabits the bodies of individuals but appears to have extensive, profound and difficult to understand social, economic and cultural implications.
To begin to understand these implications it is vital that we pay attention to the characteristics of the pathogen, its life cycle and its pathologies.

HIV is a retrovirus. This is a fairly small group of viruses where the core is composed of RNA (ribonucleic acid) rather than the more common DNA (desoxyribonucleic acid) found in other life forms. The significance of this feature of the virus is that their multiplication requires them to colonise host cells – in this case the cells of the human immune system – and convert these into “factories” for the production of more viral particles. This is achieved by converting viral RNA into DNA through an enzyme called reverse transcriptase. In the process the host cell is destroyed and many millions of viral particles are expelled into the body of the infected person. This process of viral insertion, transcription and particle expulsion is shown in Figure 1.

The human host’s immune system fights back and over a period of about 8 years on average there is a continuing battle between host immune system and viral population. The person has greatly varying viral load over this period and this influences their infectiousness to other people, their susceptibility to other infections and their state of health. This process has clear operational significance from the viewpoint of uniformed services, and also has implications for the relative balance of infectiousness of the services to the general population and for the susceptibility of uniformed personnel to other diseases common in their areas of operation. Opportunistic infections are a particularly important aspect of the disease syndrome for uniformed forces and those with whom they interact by virtue of their mobility and exposure.

A key feature of the virus, its translation of RNA to DNA, means that it is particularly liable to errors in transcription of the genetic code, resulting in high frequency of “mistakes”. These mistakes mean that within the host, the virus is mutating, these mutations accumulate, thus increasing the pool of viral variation which enables the virus to outwit the human immune response. Figure 4 shows the progress of this process in terms of a graph of viral population (the viral load usually measured in blood but in this case in semen).
and CD4 cell count, a measure of the number of active cells able to fight the virus per ml of blood. The typical period from initial infection to death is around nine years. Because of its slow progress from infection to death typical of this group of viruses, they are described as lentiviruses, slow acting viruses. Viral mutability and the further possibility of recombination of mutated viruses – both in individual people and in cases of re-infection of an already infected person, with a new viral clade – have serious implications, particularly in relation to the dangers of developing transmitted viral resistance to treatments where supplies of medication are interrupted and/or treatment compliance falls below 95% for other reasons.

This section has outlined some pathogen-specific characteristics.

**Figure 1  HIV Life Cycle**  
Source:  
http://www.wiley.com/legacy/college/boyer/0470003790/cutting_edge/aids_therapies/aids_therapies.htm

**Level 2: The individual**  
Infection with HIV is most commonly via sexual relation and globally the most frequent transmission route is via heterosexual relations. Mother to child
transmission is significant and of all children born HIV+, about 50 per cent will remain HIV+ after their first year and will go on to develop AIDS\(^4\).

The comparative efficiency of different modes of transmission is shown in Figure 2 while Figure 3 summarises some of the complexities and limitations associated with our understanding of the transmission process under different conditions.

<table>
<thead>
<tr>
<th>INFECTION ROUTE</th>
<th>RISK OF INFECTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>SEXUAL TRANSMISSION</td>
<td></td>
</tr>
<tr>
<td>Female-to-male transmission</td>
<td>1:700 to 1:3,000</td>
</tr>
<tr>
<td>Male-to-female transmission</td>
<td>1:200 to 1:2,000</td>
</tr>
<tr>
<td>Male-to-male transmission</td>
<td>1:10 to 1:1,600</td>
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<tr>
<td>Fellatio</td>
<td>0 to 6%</td>
</tr>
<tr>
<td>PARENTERAL TRANSMISSION</td>
<td></td>
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<tr>
<td>Transfusion of infected blood</td>
<td>95:100</td>
</tr>
<tr>
<td>Needle sharing</td>
<td>1:150</td>
</tr>
<tr>
<td>Needle stick</td>
<td>1:200</td>
</tr>
<tr>
<td>Needle stick/AZT PEP</td>
<td>1:10,000</td>
</tr>
<tr>
<td>TRANSMISSION FROM MOTHER TO INFANT</td>
<td></td>
</tr>
<tr>
<td>Without AZT treatment</td>
<td>1:4</td>
</tr>
<tr>
<td>With AZT treatment</td>
<td>Less than 1:10</td>
</tr>
</tbody>
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Adapted from Royce, Sena, Cates and Cohen, NEJM 336, 1072 (1997)

**Figure 2  Routes of Exposure and Transmission**

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\(^4\) This is because children are born with disease antibodies they have taken from their mothers. Thus, a child born to an HIV+ mother may be carrying antibodies to HIV because s/he takes them from the mother but does not in fact carry any virus, or because s/he takes them from the mother but does carry the virus. See Figure 2 for relative risk of MTC transmission as compared with other routes.
<table>
<thead>
<tr>
<th>Complex sexual behaviors with potential concomitant exposure of several different mucosal sites; anal intercourse in heterosexual couples may be fairly common.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexual history provided by study subjects limited by their memory. All studies depend on the reports of study subjects about the quantity and quality of sex, signs and symptoms of sexually transmitted diseases, use of medications, etc. Sexual diaries have proven cumbersome and sometimes inaccurate.</td>
</tr>
<tr>
<td>Lack of knowledge of the HIV status of the sexual partners (except in the case of discordant couples).</td>
</tr>
<tr>
<td>Unrecognized or undetected factors that can amplify transmission, especially STDs. Many STDs are asymptomatic yet might still increase the risk of HIV acquisition (2).</td>
</tr>
<tr>
<td>Long periods of follow-up between visits of people at risk, confounding accurate interpretation of risk. HIV-uninfected subjects probably suffer only very brief periods of high risk, but if they are studied infrequently a large number of low risk sexual encounters are included for consideration, reducing the calculated probability of HIV transmission.</td>
</tr>
</tbody>
</table>

**Figure 3  Routes of Exposure and HIV: Limitations of Estimations of Sexual Transmission**

The slow action of the virus at the level of the individual means that for much of the time an infected person is able to function at some satisfactory level socially and economically. They are also, of course, able to act sexually. The result is that, depending on the social, cultural and economic environment, the reproductive rate of each initial infection can be very high indeed. In most

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5 An epidemic is a rate of disease that reaches unexpectedly high levels, affecting a large number of people in a relatively short time. Whether and how an epidemic develops is linked to the reproductive rate of the pathogen. The gradient, final height and rate of decline of the curve is determined by the average number of secondary cases generated by one primary
cases, an infected person experiences an initial fever soon after contracting the virus. At this time the viral load rises and they are very infectious. Over time, viral load declines as does infectiousness (although people remain infectious during such periods) as the immune system combats the infection. Finally, the viral population overwhelms the immune system. In the absence of sustained anti-retroviral treatment, an infected person has periods of increasingly serious illness finally resulting in death. The process is shown graphically in Figure 4. It is important to note that the sexual nature of transmission and the long viral life cycle (combined with high mutation potential) means that infection is likely to occur at an early stage in an individual’s life, perhaps even soon after sexual debut, and not necessarily to result in illness until children have already been conceived. Thus, each individual infection has the possibility of keeping the host alive long enough to reproduce and leave behind offspring who can be infected in the future. Hence there is a pathological harmony between the viral and human life cycles. This pathological harmony may have significant social and economic results, particularly when it is realised that, because the wave of the epidemic curve is probably up to 50 and perhaps 120 years long, each succeeding generation is likely to be born into a higher prevalence environment than its parents – and because of its orphaned status, probably have an increased likelihoods of contracting HIV.

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case in a susceptible population and the period over which this takes place. This is also known as “the basic reproductive number” and represented by the symbol $R_0$ (Anderson and May, 1992). In order for an epidemic to be maintained, $R_0$ has to equal 1, in other words each person who gets better or dies has to infect one other person. At this point the disease is endemic but stable. When $R_0 > 1$, each person infects more than one other person, the number of cases will rise. When $R_0 < 1$, then the epidemic will be disappearing. The reproductive rate is the number of secondary cases resulting from each primary case. Where this is 0 or $<1$ infection does not become epidemic. Where it is $>1$ epidemic development is likely. The larger the $R_0$ number, the steeper the epidemic curve.
Figure 4  Sexual transmission of HIV: the relative risk of transmission over the course of disease as a function of viral load in semen\textsuperscript{6}

Source for the above: IAVI REPORT 8(3), September 2004-November 2004: Rethinking the risk of transmission, pp. 1-2, Myron S. Cohen, MD

Level 3: The Social Unit

The general situation with regard to the pathological harmony between pathogen and human society is outlined schematically in Figure 5. This diagram illustrates the ways in which a generalised epidemic adversely affects the potential and actual capacity for a society and economy to reproduce itself in a variety of ways, via transmission of knowledge and education, through maintenance of social and cultural patterns and via the peopling of institutions such as uniformed services in particular but government and community infrastructure in general. Briefly, this figure shows the following:

\textsuperscript{6} Orange indicates HIV RNA copies per ml semen, yellow indicates the reduced viral burden expected from host defences evoked by a vaccine or early use of antiviral therapy. The dashed line offers a theoretical viral burden threshold below which HIV transmission will not occur. Numbers provided at the different stages of disease represent the probability of HIV transmission/episode of heterosexual intercourse.
1. The resonance between the viral life cycle (in yellow) and the human generational cycle (in thick black). Here we see that generation 1 reproduces itself and acts as a host for the HIV pathogen. As this generation dies it leaves orphans.

2. These orphans enter a world where the risk of infection with HIV has increased as the general epidemic curve rises. In addition, as orphans, this generation is also possibly more socially, culturally and economically exposed to infection. Thus the pattern repeats itself, the generation reproduces, but so also does the pathogen, and a second generation of orphans is produced. This generation faces an increased risk of infection for the same reasons as did its parents – but the risk is increased as general seroprevalence rises and social exposure to sexually transmitted infections (including HIV) also increases as a result of less adequate socialisation, reflecting in part the decreased expectancy of adult life of parent generation.

3. In the background is the possibility of increased viral resistance to anti-retroviral medications as ARV roll out occurs under sub-optimal circumstances with poor compliance and inadequate health systems. We can only speculate about the significance of this development.
In addition to these processes of disruption, epidemic associated mortality affects the demographic structure of a society. These effects are not uniform and will vary from place to place. Thus, in parts of Africa we have evidence of what had happened to population in heavily affected regions of Uganda where the so-called “chimney effect” could be observed in Rakai District as long ago as 1993 when it was reflected in the census data as shown in Figure 6.

Figure 5  HIV/AIDS: Sundering the Bonds of Human Society?

In addition to these processes of disruption, epidemic associated mortality affects the demographic structure of a society. These effects are not uniform and will vary from place to place. Thus, in parts of Africa we have evidence of what had happened to population in heavily affected regions of Uganda where the so-called “chimney effect” could be observed in Rakai District as long ago as 1993 when it was reflected in the census data as shown in Figure 6.
Figure 6  The "chimney" effect of HIV/AIDS on population in Rakai, Uganda, 1993. 

Similar outcomes have been predicted for Botswana as shown in Figure 7.

Projected population structure with and without the AIDS epidemic, Botswana, 2020

However, the initial population structure upon which the epidemic impacts differs from place to place. Thus, Figure 8 shows a range of possible structures from Europe, Russia and the CIS, on each of which the effect of increased mortality will have different social and economic consequences.

**IMPACT WILL VARY DEPENDING ON THE DEMOGRAPHIC STRUCTURE OF DIFFERENT COUNTRIES**

**Figure 8** Impact will vary with base population structure - illustrations from Europe and Central Asia

2: **What we know/believe about the extent of HIV/AIDS related orphaning**

What we know about the extent of HIV/AIDS orphaning depends on how we know. The methods for knowing about HIV/AIDS related issues are very closely tied to modelling technologies because (a) in many of the most seriously affected countries vital registration is either not common or is not comprehensive when it exists (b) we are looking to a future event (c) some data are collected using qualitative methods and this may make it difficult to judge how generalizable are their findings.

In addition there have been some marked problems about HIV/AIDS statistics in general, revolving around the countervailing pulls between the need for simplicity in advocacy and precision and transparency in science and, it should be said, the uneasy place of the discourse of policy in relation to these two other ways of engaging with the world.

The best data are from national surveys of infection rates. These are expensive and logistically difficult to assemble. But we do have some
examples. As long ago as 1988 Uganda undertook a national sero-survey which covered as much of the country as was then accessible to government given local security problems – which meant some northern areas were not surveyed. Several more have been completed in other countries since and another half dozen are planned in the near future.

In the absence of a national sero-survey, governments collect information from antenatal clinic attenders – unique time series data on women who by definition have had unprotected sex. Are these representative of the wider population? Yes and no. This is the most accessible group on which to base surveillance under field conditions, but HIV+ women are less likely to become pregnant and so less likely to appear in ante-natal clinics; so this group may under-represent. How representative these surveys are can be questioned and they are, routinely, in order to be tested via peer reviews both of data collection methods and projection models - reviews freely found on the internet and in professional publications. So in sum, data collection methods, modelling assumptions and conclusions are all transparent; the results are then published and indicate the situation within boundaries of error. That is good science: constant peer review of methods, models and conclusions. Hence, in the 2004 UNAIDS report on the epidemic (published 6 July 2004), improved methods resulted in downward revision of estimates for 22 African countries and upward revision for 10 as compared to the data presented in the 2002 report. Such revisions reflect improving knowledge and techniques. To illustrate specifically: one of the biggest revisions is for Kenya. Estimates of national prevalence at 15% for the end of 2001 derived from ANC based surveillance are revised to 6.7% for the end of 2003, derived from a national population survey. But this more robust figure is fragile ground for rejoicing, especially if the trend line continues upwards - and next year’s figures suggest an increase on 6.7%. The most optimistic estimate is that it signals between 820,000 and 1.7 million adult Kenyans living with this dreadful disease. Sero-prevalence does not show epidemic effects: it provides a peek about eight years into the future, when these people will be dying – and when, unless prevention efforts are effective, there will be new cohorts of infected people.
The “only” 13% indicated in 2001 for Benue State in Central Nigeria\(^8\) - the country’s grain basket – suggests not only the immediate human suffering represented by 90% of the total of about 100 hospital beds in the Catholic Hospital in Makurdi being occupied by people with AIDS (according to the one doctor working there who was interviewed three years ago\(^9\)) but also that food production is being affected, as we know it to have been already in East/Central Africa, large numbers of orphans created and households left unable to manage already precarious livelihoods.

With these introductory remarks in mind, the following statements are widely believed to be “true” in relation to the question of orphans of the AIDS epidemic. They are derived from field observations, limited survey and also, it must be noted, modelling exercises of various kinds, most notably using the programme Epimodel\(^{10}\) produced (and frequently revised and updated) by The Futures Group. On this basis we know/believe the following:

- In 2003 there were 43.4 million orphans in Sub-Saharan Africa
- 12 million of them had lost one or both parents to HIV/AIDS
- By 2010 the ranks of children orphaned by AIDS will swell to 18.4 million\(^{11}\)
- There is a disproportionate number of orphans due to AIDS in Africa as compared to other world regions Figure 9.

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\(^{8}\) Netherlands Royal Tropical Institute Report for DFID – citation to be added.
\(^{9}\) Author’s fieldwork notes
\(^{10}\) Citation to be added
\(^{11}\) UNDP
Disproportionate Number of Orphans due to AIDS in Sub-Saharan Africa, 2001

Figure 9  Disproportionate numbers of orphans due to AIDS in Sub-Saharan Africa, 2001

- That in Africa, AIDS is the major cause of orphaning among children under 15 years of age and is projected to remain so by 2010 (Figure 10).

Percent of children under age 15 who are orphans by region, year, and cause

Figure 10  Per cent of children under age 15 who are orphans by world region, year and cause plus projection to 2010

- Projections suggest that by 2010, orphans will make up between 15 and 25 per cent of all children in Africa.
By 2010, orphans will account for 15% to over 25% of all children in 12 sub-Saharan African countries

All of these statistics are disturbing, but the last may have particularly serious social, economic and political implications and is the basis for the discussion in the next section.

3: Infectious disease and governance

Infectious disease morbidity and associated premature mortality have economic, social and political implications. In Africa, HIV/AIDS, tuberculosis and malaria generate substantial economic cost. HIV/AIDS in particular is a current and still relatively poorly-understood threat, concentrated as it is in the most productive and re-productive age groups.

The lack of understanding of the epidemic is disturbing, given we are so far into such a sustained long wave event. As yet, we do not understand the specifics of the epidemic’s effects on individual, household and business costs in highly affected countries. For example, our knowledge of its effects on rural communities remains based on a limited number of empirical studies, few of them very recent.

But of the many areas where our understanding is severely limited, nowhere is this more pronounced than in relation to issues of social cohesion and
governance. Some years ago the “Jaipur Paradigm”\(^\text{12}\) hypothesised that there was a relationship between levels of social cohesion, income distribution and the gradient and final peak of an epidemic curve.

**The Jaipur paradigm**\(^\text{13}\)

This proposed that susceptibility and vulnerability of a society to HIV, and thereby the profile of its epidemic, might be determined by two variables:

1. the degree of social cohesion
2. the overall level of wealth

The relationship of these two variables to the attributes of susceptibility and vulnerability is presented in Figure 12.

**Figure 12 The Jaipur Paradigm**

The idea of social cohesion was used strictly without value judgement to locate a society on a continuum from cultural and social heterogeneity to

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\(^{13}\) Entitled the Jaipur paradigm because these ideas were first developed at a policy research workshop in that city in the late 1990s.
social and cultural homogeneity. The assumption is that political stability and concerted social action are more achievable in circumstances of homogeneity. Effective action is possibly the product of good governance and a strong civil society. But it could also be seen as related to a prescriptive religious culture; or it could be the result of a controlling authoritarian political system or military dictatorship.

Whatever the source of a society’s cohesion and its role in determining that society’s susceptibility to HIV infection, the analysis cannot be turned around into a justification of oppression and social homogenisation. Neither are levels of social cohesion sufficient explanations. This concept has to be combined with some consideration of wealth and income in general and distribution in particular.

The degree of inequality of income and wealth distribution is important. The relationship between health, economic inequality, and social support has been explored extensively by Wilkinson\(^{14}\) and others, particularly in relation to the concept of “social capital”. For present purposes it is not necessary to consider inequality in wealth as a separate variable in determining susceptibility, because it is invariably reflected in the level of social cohesion: As economic inequality increases, social cohesion tends to deteriorate.

Combining the two variables allows the categorisation of groups and societies along two axes into four broad “types” each with a distinct epidemic pattern of HIV prevalence.

**Pattern 1**: High social cohesion and high wealth - most resource-rich industrialised societies.

**Pattern 2**: Low social cohesion and high wealth - societies in transition or societies with a predominant migrant labour economy.

**Pattern 3**: Low social cohesion and low wealth - countries experiencing civil war or economic collapse.

**Pattern 4**: High social cohesion and low wealth – countries with strong religious cultures, good governance, or highly controlling political systems.

\(^{14}\) Wilkinson, R.G., Unhealthy Societies: the affictions of inequality, 1996, Routledge and Kegan Paul, London. It is important to note that this important study does not concern itself with communicable diseases and that we know very little about the relation between these variables and communicable disease in general.
The four hypothetical HIV prevalence curves corresponding to each one of the patterns are presented in Figure 13 with the understanding that the patterns never exist in their pure form.

The first pattern is characterised by slow growth followed by a low peak and slow decline with low endemic prevalence. The second pattern, found in a society with low levels of social cohesion and high income, exhibits a sharp increase in prevalence followed by a relatively rapid decline as the society mobilises the means for an appropriate response. The third pattern, in a society with low levels of social cohesion and low income, shows an epidemic which may take some time to gain momentum, but once it does it rises rapidly and the prevalence of infection remains high. The fourth epidemic pattern observed in a society with high levels of social cohesion and low income is characterised by slow epidemic growth to a plateau of low HIV prevalence.

**Figure 13 Hypothetical epidemic patterns of HIV in different situations**

Societies can and do change. Social cohesion may break down or build up and countries may experience economic growth or decline. Social cohesion is particularly fragile if it is maintained by authoritarian social control. Analysis has to be performed with a historic perspective. The interpretation of the profile of the HIV epidemic in Uganda, for instance, has to take account of the transition from social chaos and economic decline in the 1970s and 80s to economic stability and greater order in the 1990s. It will be particularly important to follow and try to anticipate the epidemics in the countries of
Eastern Europe and Central Asia which are undergoing profound political, social, and economic transitions\textsuperscript{15}.

**HIV/AIDS and Population Structure**

Early consideration of the social and economic implications of the epidemic focused quite correctly on the demographic effects. Discussion mainly revolved around effect of the epidemic on rates of growth and total population size. Paradoxically, the question of population structure was less central to the discussion.

Early results from research from rural communities in Uganda in 1989\textsuperscript{16} did reveal that population structure was evidently affected adversely – as might have been assumed from the known age cohort specific incidence of illness and death. That this was in fact the case and that HIV/AIDS alters the structure of populations, increasing the number of dependents to producers and fully capable citizens was made very clear by Low-Beer, Stoneburner and Mukulu in 1997 in the study cited above\textsuperscript{17}. This showed that the effect was already apparent in the Ugandan census of 1993.

**The Governance Issue – the disharmonious resonance between viral and human life cycles**

HIV/AIDS has particular implications for governance because, as already noted, the infectious organism, HIV-1, is a group of slow acting viruses. The disharmonious resonance between the length of infection in the individual human host and the length of a human generation has also been noted. Inasmuch as an infected person has children, and these are orphaned and grow up to become infected, but not before they have themselves had children – who are orphaned in turn – so a basic unit of social structure in most human societies, the three generation bond between grandparents,


\textsuperscript{17} Low-Beer, D., Stoneburner, R.L. and Mukulu, A (May 1997), Empirical Evidence for the severe but localised impact of AIDS on population structure, Nature Medicine, Vol. 3 No. 5
parents and the current generation – and on into the future – is rent asunder: repeatedly in the absence of treatment, a vaccine or behaviour change. This process is illustrated in Figure 5 above – which, it should be remembered, must be read against an ever present threat of growing viral resistance.

Children brought up in difficult circumstances develop a pragmatic and short term survival perspective\(^{18}\). Marguerite Daniel’s careful study of orphaning in one of Africa’s richest countries shows the extent and nature of orphan related trauma\(^{19}\). Furthermore, behavioural change messages about HIV/AIDS may have the unintended consequence of stigmatising the parental generation who will be perceived to have failed morally and as parents\(^{20}\). Thus in Africa, where respect for elders is of central cultural importance, the intergenerational structural break appears as loss of respect for elders – at a time when rapid change is already contributing to this process. Combined with an un-moderated pragmatic orientation towards the world, children and young people are hard-pressed merely to survive from day to day. This may have substantial implications for social and political relations at the household, community, and ultimately the national level when these inadequately socialised people reach adulthood.

**Speculative futures**

Here are two broad possibilities. In one, the epidemic places so much pressure on state and elite survival that politicians take refuge in authoritarian responses and policies. A final dawning of the nature and extent of impact on tardy political leaderships results in desperate and ideologically syncretic responses which are above all pragmatic and attempt to discipline a population.

While the label “fascist” may seem odd, Karl Polanyi’s description of the harbinger ideas of fascism seems somehow apposite to describe this possibility: “spread of irrationalist philosophies, racialist aesthetics,

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\(^{18}\) Thanks to Dr Roland Msiska for drawing attention to this observation.


\(^{20}\) Thanks to Dr Elizabeth Marum, CDC Nairobi, for this observation.
anticapitalist demagogy, heterodox currency views, criticism of the party system, widespread disparagement of 'regime' or whatever was the name given to the existing democratic set up\textsuperscript{21}. Such a response is also possible within one of the future scenarios (called Africa Takes A Stand) developed within the recent UNAIDS-Shell Scenario Project\textsuperscript{22}. Poor countries are not immune to fascism and the Indian BJP movement and later government in India certainly trod a narrow line in this respect.

On the other hand, recent observations in rural Uganda indicate elements of a substantial and independent community response from among groups of first wave orphans now in their late twenties and early thirties who are working hard not solely for their individual and mutual welfare, but also for the welfare of their communities and in particular for the younger orphan generations\textsuperscript{23}. Measures include pressurising young men to relinquish migrant fishing livelihoods to return to complete school and similar pressure on young women working as bar-maids (for which read CSWs) – also to return to school.

The direction that particular states may take will depend on many complex factors and on pure chance. But we should be aware that such virtuous community involvement and commitment and desperate authoritarian responses are not at all mutually exclusive. The point is that “civil society organisations” and “social capital” can be bad as well as good; that the ideological frames provided by political leadership may influence the possibilities of action and thus directions taken by civil society organisations, and that a future generation of politicians marked by the HIV/AIDS epidemic might be predisposed to choose authoritarian rather than democratic directions and methods. The long term impact of the HIV/AIDS epidemic is hard enough to predict. This is just one of many possible stories we might tell. We do not know if it is true. There is unlikely to be a single truth. We must be careful not to stigmatise orphans of the AIDS epidemic, and for this and for many other reasons we need to be aware of our deep responsibilities in

\textsuperscript{22} Presented at a DFID Public Lecture by the UNAIDS Scenario team at LSE May 2004.
\textsuperscript{23} Barnett, observation from fieldwork October 2004.
describing and discussing possible implications of this long wave event on the lives of orphans…and on the orphans of orphans.